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RESPONSE OF ENDODONTICALLY TREATED TEETH TO ORTHODONTIC FORCES

BY

RITCHIE MAH

A THESIS

SUBMITTED TO THE FACULTY OF GRADUATE STUDIES AND RESEARCH

IN PARTIAL FULFILLMENT OF THE REQUIREMENTS FOR THE DEGREE

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IN

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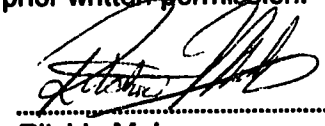

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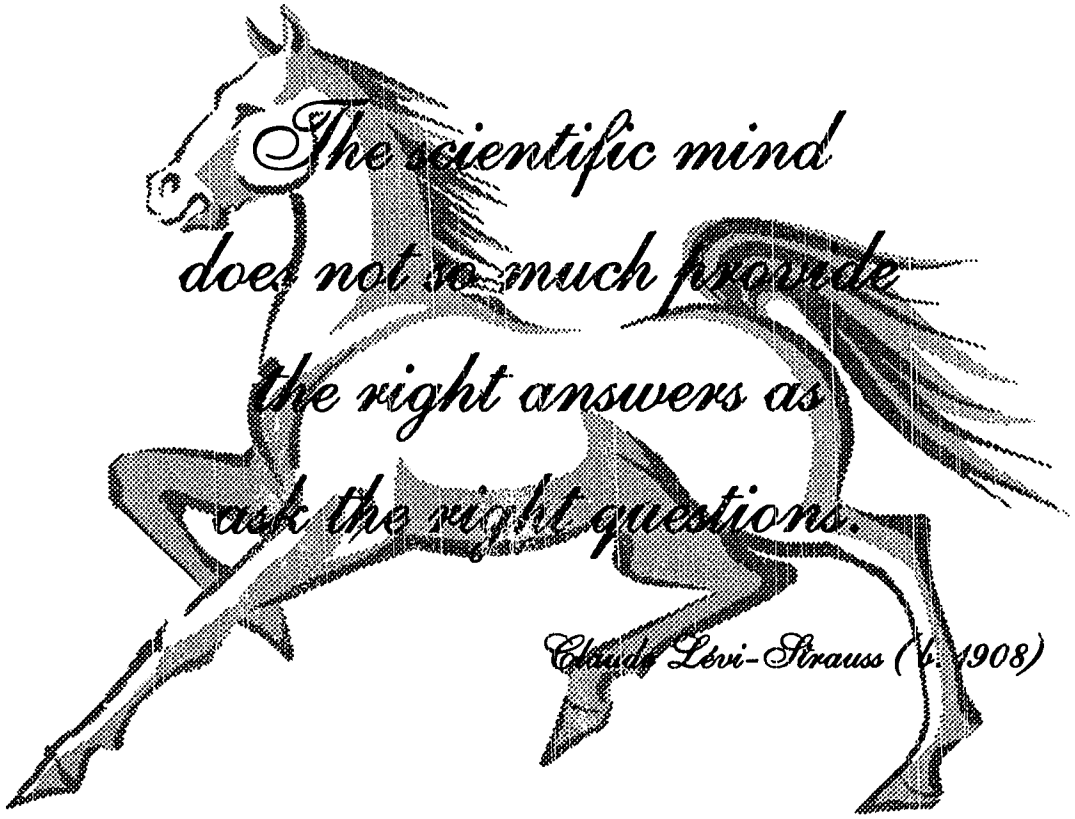
The scientific mind
does not so much provide
the right answers as
ask the right questions.
Claude Lévi-Strauss (b. 1908)

University of Alberta

FACULTY OF GRADUATE STUDIES AND RESEARCH

The undersigned certify that they have read, and recommend to the Faculty of Graduate Studies and Research for acceptance, a thesis entitled:

"RESPONSE OF ENDODONTICALLY TREATED TEETH TO ORTHODONTIC FORCES"

submitted by Ritchie Mah in partial fulfillment of the requirements for the degree of Master of Science in Clinical Sciences.

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To My Parents

For giving me the education
that they could not have.

ABSTRACT

This study evaluates differences in tooth movement and root resorption between vital and root filled teeth. Mandibular canine root canal therapy was performed unilaterally in 12 young male ferrets. After removal of mandibular premolars and maxillary canines, bilateral tooth movement was induced with orthodontic springs (150 - 175 g). Procion red dye was injected one week pre-surgically to mark bone and cementum deposition. Three months later, the ferrets were perfused with a fixative solution under GA. Mandibles were removed, radiographed, and then sectioned for evaluation. Tooth movement was assessed from pre- and post-treatment mandibular casts and bone deposition was assessed histologically. Root resorption was evaluated by radiographic and histologic root length, resorption lacunae number, and deposition of cementum. The presence and extent of periapical inflammation in root filled teeth that were moved and those that were not did not differ significantly ($p > 0.05$). Root filled teeth showed greater loss of cementum after tooth movement than vital teeth ($p < 0.05$), but without noticeable differences in radiographic root length. Orthodontic movement of endodontically treated teeth results in loss of tooth substance greater than similar movement of vital teeth, but the difference is small and only detectable microscopically. No significant difference is found in amount of tooth movement between vital and root filled teeth.

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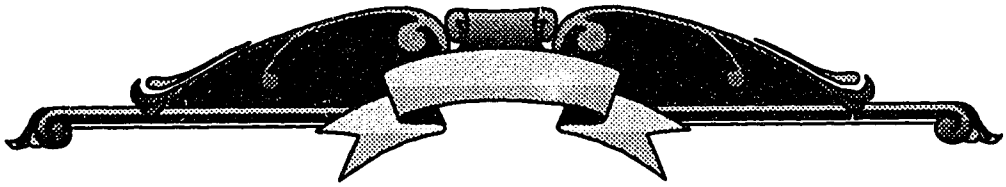


Chapter One

Introduction

And

Literature Review

1.1 - INTRODUCTION

Orthodontics is the oldest specialty in dentistry, as it has long been recognized that controlled movement of teeth is possible by the application of controlled forces. Evidence of attempted tooth movement as early as 1000 BC has been found [1]. In later centuries, many orthodontic appliances were designed, based largely on empirical knowledge. In the 19th century, dentistry became documented, with publications such as Norman Kingsley's *Oral Deformities* [2]. Edward H. Angle's classification of malocclusion in the 1890's pioneered a rational approach to orthodontics [3]. Most early attempts to correct malocclusions were based on esthetics alone whereas Angle's interest in prosthodontics emphasized the occlusion of the dentition and its function. Angle designed and implemented various orthodontic appliances, which have subsequently been modified and improved to the point where today's pre-angulated, pre-torqued, and pre-adjusted appliances offer orthodontists a very efficient method of controlled tooth movement. Although the design and function of various appliances differ, the biological response of the teeth and surrounding tissues to applied forces remains the same. This allows each of these appliances to function, whether an edgewise type appliance, a Begg appliance, a removable appliance, or some other variation of these appliances.

The biological basis of tooth movement, although extensively studied, is incompletely understood. An intact periodontal ligament (PDL) must be present around the teeth to be moved as tooth movement is primarily a PDL phenomenon [4]. The response to applied forces is thought to be initiated in the PDL and propagated through a series of regulatory cascades

[5]. Nerves and periosteal receptors of the PDL account for only a portion of the sensation experienced by a tooth. Neural and cellular receptors within the pulp also detect stimuli applied to the tooth and "it is likely that all teeth moved with fixed appliances will undergo certain pulp alterations" [6]. This is shown by canal calcification occasionally accompanying orthodontic tooth movement, although the most characteristic tissue alteration is vacuolization of the odontoblast layer [7,8]. As well, pulpal extirpation is recommended to stop internal root resorption, indicating some role of the pulp in response to orthodontic forces [9,10]. Teeth which have been subjected to endodontic therapy may thus respond differently to orthodontic forces than vital ones, which will be investigated in this study [11,12]. In addition, many endodontically treated teeth, although clinically successful, show evidence of periapical inflammation [13]. Based on these observations, endodontically treated and non-endodontically treated teeth would be expected to exhibit differences in response to orthodontic forces. The magnitude of these differences, however, is not clear.

There is some difference of opinion among orthodontists as to whether endodontically treated teeth respond differently to applied forces. Many consider that there is little difference between vital and non-vital teeth [11,12]. Others suggest that there is a higher incidence of ankylosis, root resorption, and root fracture in non-vital teeth [14]. Conflicting studies have also shown that some root filled teeth are easier and others more difficult to move than their untreated controls [4,11]. Other studies disagree as to whether root filled teeth are more or less likely to experience root resorption [14,15,16,17]. Susceptibility to ankylosis in root filled teeth undergoing orthodontic treatment has also been found to be greater than in their untreated

counterparts [16,17,18]. The literature to date has clearly not established whether significant differences exist between vital and non-vital teeth in orthodontics. The controversy, present both clinically and experimentally, warrants further investigation.

1.2 - STATEMENT OF THE PROBLEM

Orthodontic patients occasionally present with one or more endodontically treated teeth, either prior to initiation of treatment or during the course of orthodontic treatment. Past clinical reports and experimental investigations have failed to come to a consensus as to whether endodontically treated teeth respond favourably or unfavourably to orthodontic treatment [11,12,14,15,16,19]. Without sound scientific data on which to base treatment, objective judgments are difficult and prognosis is uncertain.

If normally used orthodontic forces are ineffective in moving non-vital teeth or induce unacceptable levels of root resorption, clinical techniques may require modification. The problem that this project addresses is the lack of objective data on the effect of orthodontic forces on endodontically treated teeth.

1.3 - PURPOSE

The purpose of the present study is to determine in an animal model whether there is a difference between endodontically versus non-endodontically treated teeth in response to orthodontic forces. If a difference does exist, the study will attempt to quantify this difference. The two parameters of interest are the degree of tooth movement and the extent of root resorption in response to applied forces.

1.4 - RESEARCH QUESTIONS

1. Do endodontically treated teeth move to a different extent than vital teeth when the same orthodontic forces are applied to them?
2. Are endodontically treated teeth more susceptible to external root resorption than non-endodontically treated teeth in response to orthodontic forces as measured histologically and radiographically?
3. Does orthodontic treatment alter the degree of periapical pathology detected in some endodontically treated teeth?

1.5 - HYPOTHESES

Primary Hypothesis: H_o: There is no difference in linear, tipping tooth movement between endodontically versus non-endodontically treated teeth.

Secondary Hypotheses: H_o: There is no difference in root resorption between endodontically and non-endodontically treated teeth following application of orthodontic forces as assessed by root length, number of resorptive lacunae, and cemental resorption.

H_o: There is no difference in extent of periapical pathology around endodontically treated teeth that have been moved compared to ones that have not.

1.6.1 ORTHODONTIC TOOTH MOVEMENT - RATIONALE

Active treatment of malocclusion is desirable for a number of reasons. According to Proffit, malaligned or maloccluded teeth can cause three types of problems: (1) psychosocial difficulties related to impaired dentofacial esthetics [4,20-24]; (2) oral function handicaps associated with mastication, swallowing, or speech, or temporomandibular joint disorders [25]; and (3) increased periodontal disease and/or caries [26,27]. Societal values dictate that the benefits of treatment far outweigh the risks. Preventive dentistry today includes the prevention and treatment of malocclusions. With an increasing proportion of our population retaining their dentition into late adulthood, the demand for treatment later in life is high [28,29].

Orthodontics is "the area of dentistry concerned with the supervision, guidance and correction of the growing and mature dentofacial structures, including those conditions that require movement of teeth or correction of malrelationships and malformations of related structures by the adjustment of relationships between and among teeth and facial bones by the application of forces and/or the stimulation and redirection of the functional forces within the craniofacial complex. Major responsibilities of orthodontic practice include the diagnosis, prevention, interception and treatment of all forms of malocclusion of the teeth and associated alterations in their surrounding structures; the design, application and control of functional and corrective appliances; and the guidance of the dentition and its supporting structures to attain and maintain optimum relations in physiologic and esthetic harmony among facial and cranial structures." [30] The ability to achieve controlled tooth movement thus plays a crucial role in realizing the goals of orthodontic practice.

Orthodontic tooth movement carries some risks as well. Some of these are preventable with patient cooperation and adequate supervision; some are unavoidable and irreversible. The patient needs to be informed of these prior to the initiation of orthodontic treatment [31,32].

Damage to the supporting tissues of the dentition can have several causes. Orthodontic appliances themselves are somewhat irritating to the buccal mucosa, cheeks, and lips, as their profile and shape protrude from the tooth surfaces. Although most patients are able to adjust to this chronic irritation, some can develop further complications such as pneumoparotitis, which has been reported in some cases [33].

The gingival tissues are very prone to irritation during orthodontic treatment. Poor oral hygiene is probably the single most causative factor in these cases. The problem of plaque accumulation and periodontal inflammation is significantly greater in adolescents than in adults undergoing orthodontic treatment [34]. However, periodontal problems rarely become a concern in adolescents because periodontal disease is uncommon at this age and adolescents' tissues seem somewhat more resistant to irritation than their adult counterparts. Conversely, periodontal considerations become more significant in older patients, regardless of whether existing periodontal disease is detected. As a general rule, any patient over 35 years of age will have some periodontal problems that could affect orthodontic treatment [4]. Periodontal pocketing exhibits a direct relationship to age, whereas the prevalence of mucogingival problems seems to peak in the twenties [35]. In extreme cases of gingival irritation, hyperplastic tissue can develop, or worse, lead to lesions such as peripheral giant cell granulomas,

complicating treatment [36].

Mucogingival attachment, especially in the lower anterior region, is often cited as a treatment limitation since the movement of the lower anterior teeth may be limited by this zone of tissue. In cases where stripping of minimal attached gingiva is inevitable or probable, a gingival graft may be considered as a treatment option [37]. Treatment plans to avoid this may involve extraction of teeth and/or different choices of mechanics to achieve tooth movement.

The process of orthodontics is thus fraught with risks and complications, however, these are usually minimal compared to the benefits. As the public becomes more cognizant of this and continues to raise its collective dental awareness, coupled with the increase in preventive dentistry, orthodontic demand rises dramatically and will likely continue to do so. Recent surveys of the long-term effects of orthodontic treatment reveal that the vast majority of past orthodontic patients feel that they benefited from their treatment and are pleased with the result. Of these, many noted dramatic changes in dental and facial appearance, but almost all recognized an improvement in both dental and psychologic health.

1.6.2 - BIOLOGIC BASIS OF TOOTH MOVEMENT

The tissue reaction accompanying physiologic tooth movement was first described by Stein and Weinmann in 1925 [38]. They observed the gradual migration of molars mesially in adults, corresponding to the wear on the occlusal surfaces. Björk later used radiographic evidence to demonstrate mesial migration of upper molars as they erupted while lower molars were found to erupt slightly distally [39]. It was recognized that the final position of the teeth could be controlled by the application of forces to guide teeth. From this observation came the concept of controlled tooth movement, and orthodontics developed.

Orthodontic tooth movement is based on the principle that force applied to a tooth will result in changes in the periodontium and surrounding alveolar bone, allowing tooth movement to occur. Bone remodels by apposition and resorption simultaneously in selective areas [6]. This allows the tooth to move through the bone as force is applied [4]. The periodontium thus plays an important mediating role in the process of tooth movement, as forces to the tooth are conveyed to the bone by the neural, vascular, cellular, and fibrous elements of the periodontal ligament (PDL) [6]. The cells responsible for bone and cementum resorption and deposition are also present in the PDL [40].

Under normal circumstances, each tooth root is surrounded by the highly collagenous periodontal ligament which has fibers inserting into the cementum of the root surface at one end and into a compact bony plate, the lamina dura, at the other [6]. The orientation of these fibers is such that, under normal function, they resist tooth displacement, but absorb occlusal forces. The PDL also contains many cell types, including fibroblasts, cementoblasts,

osteoblasts, and resorptive cells, including osteoclasts and cementoclasts [40]. There is also a rich blood and nerve supply. All these elements are surrounded by a connective tissue matrix. Each of these elements plays an important role in tooth movement as well as normal function.

The mesenchymal cells are thought to be particularly important in their ability to differentiate into fibroblasts and osteoblasts [41]. Remodelling and recontouring of the bony socket and root structure also occurs with normal function with bone and cementum removal carried out by multi-nucleated osteoclasts and cementoclasts [6]. These are thought to be derived from monocytes in the blood and lymphatic systems rather than from the local osteoprogenitor cells [42].

The blood vessels and cells of the vascular system and the nerve endings within the PDL provide for the nutritional maintenance of the PDL as well as the ability to feel pressure, pain, and position. The fluid within the PDL space, combined with the porous bone of the socket, allows the PDL to function as a natural shock absorber, protecting the tooth from fracture. Under relatively heavy forces of several kilograms, as in normal masticatory function, this incompressible tissue fluid transmits the force to the alveolar bone, which then flexes. These bending stresses are spread over significant distances, generating piezoelectric currents that stimulate skeletal changes. This is thought to be one mechanism by which the form of bone adapts to functional demands [4].

Masticatory forces may be very heavy, up to 50 kg, although this pressure is intermittent and sustained for only fractions of a second [4]. If pressure against a tooth is high, the tissue fluid can escape through the porous walls of the socket, compressing the PDL and

resulting in pain. This normally will occur only after 3 - 5 seconds of heavy force application. The brief forces of mastication, lasting for 1 second or less, occur without pain.

With sustained forces, biological adaptation occurs that results in tooth movement. Thus, prolonged forces from natural elements such as the lips, tongue, and cheeks, have the potential to alter tooth position [43]. This is also the basis of orthodontics, where the application of controlled, sustained forces induces tooth movement. Tissue reactions occurring in response to orthodontics are the same as those observed in physiologic tooth movement. The teeth and supporting structures move in response to external forces; they do not differentiate between the source of these forces. As teeth are moved more rapidly during orthodontic treatment, tissue changes are more pronounced and extensive. It has even been suggested that tissue damage is a prerequisite to the stimulation of bone resorption in orthodontic tooth movement with a positive correlation between magnitude of force, tissue injury, and the degree of bone resorption [44].

The application of continuous, light forces achieves the most optimal tooth movement [4]. This is because lighter forces allow cells within the PDL to survive and participate in "direct" resorption, remodelling the bone directly adjacent to the PDL. "Direct" resorption has also been described as "frontal" resorption since the osteoclasts within the PDL resorb bone from the tooth side of the socket. This occurs in the initial phase of tooth movement and continues as long as force levels are kept low [40]. Application of heavy forces is thought to cause hyalinization of the PDL, compressing it to such an extent as to obstruct blood flow [6]. This results in necrosis of many of the PDL cells, including the osteoclasts which had been

involved in "frontal" resorption. Resorption of alveolar bone then occurs just from the bony side of the socket, in the process of "indirect" resorption. "Indirect" resorption is synonymous with "undermining" resorption, describing the remodelling of bone from the underside of the lamina dura [6]. As underlying bone is resorbed, the bone adjacent to the hyalinized zone of the PDL will abruptly cave in, resulting in a sudden, jarring movement of the tooth. In addition to being a more painful process, the movement of teeth by this method is less predictable and occurs more intermittently than by "direct" resorption [6]. In practice, orthodontic mechanics are aimed at maximizing "direct" resorption and minimizing "indirect" resorption [4]. In reality, both processes occur simultaneously.

Since it is practically impossible to achieve a perfect light and continuous force, resulting in "direct" resorption, the goal is to avoid forces which will encourage "indirect" resorption. Use of heavy continuous forces has been found to be the least desirable, as this does not allow time to repair and regenerate, permitting ongoing "indirect" resorption [4]. If heavy forces cannot be avoided, their intermittent use will at least allow some repair and regeneration to occur, with fewer deleterious effects [4]. This means that appliances should not be activated too frequently as it is unnecessary with light, continuous forces, and if the force is heavier, intermittent force application is desired over constant application regardless [4].

Orthodontic forces are thus dependent on magnitude as well as duration to effect the desired changes physiologically. Depending on the type of tooth movement desired, the ideal force values range from 15 to 150 grams [4]. Burstone and Groves found that the "optimal rate of tooth movement" was observed in children when 50 to 75 grams of force was applied [45].

The optimal force level should be just enough to partially compress the PDL without occluding the blood supply [4]. Due to the shape of the tooth root, the area that undergoes compression will vary depending on the direction of movement [6]. It is thus necessary to consider not only the amount of force applied, but also the force per unit area, or pressure. Intrusion necessitates the lightest force as most of the force is concentrated in the apical region which is very small and cannot be subjected to excessively heavy forces [4]. Bodily movement of teeth, or translation, theoretically places pressure against the whole root surface, allowing the use of heavier force as this is spread out over a larger area [4]. The resultant force per unit area is then equal to other movements such as tipping, rotation, and extrusion which will require force magnitudes somewhere between intrusion and translation [4]. It is also important to note that the size of the tooth, the type of tooth, and the shape of the root will change the values as the area of the root surface varies with each of these factors [6].

With the use of continuous arch wires, it is difficult to obtain absolute force values for each tooth in isolation as they are joined by the arch wire. The use of a stress and tension gauge to measure the force applied to a tooth can serve as a guide, but ultimately, it is the clinical judgment of the orthodontist that determines the "ideal" force.

Applying a continuous orthodontic force would theoretically produce ideal tooth movement. In practice, this is impossible [4]. Fortunately, the duration of force application to effect tooth movement has a threshold that is attainable clinically, somewhere in the range of 6 hours per day, with effective tooth movement increasing proportional to force duration beyond this threshold. Clinically, the goal is to apply as close to a sustained force as possible.

This is most closely approximated by fixed appliances [4]. These produce forces without relying on patient compliance, thus tending to be most effective. The frequency and amount of activation will also affect the appliances' effectiveness. Removable appliances, including bite plates, headgears, cross-bite appliances, etc., can be almost as effective if worn continuously, but their usefulness decreases considerably with non-compliance [4]. If the duration of wear drops below the threshold level, no effective tooth movement will occur [4].

The process of tooth movement has been described only by what has been observed to occur, not how it happens. It is not entirely understood how force induces tooth movement. Hypotheses from the 19th century revolve around one of two possible mechanisms: the application of pressure and tension to the PDL, and bending of the alveolar bone, producing an electric current [46,47]. Experimentally, both occur simultaneously with cellular elements, as well as extracellular components of the PDL and bone, responding to the external forces, leading to osseous remodelling [46].

Research with cultured cells demonstrated that shape distortion may lead to cellular activation, either by opening plasma membrane ion channels, or by crystallizing cytoskeletal filaments [46]. The piezoelectric current generated by mechanical distortion of collagenous matrices has also been described as a possible stimulus of cells by altering their membrane potential and beginning a cascade effect [48]. Isolated human PDL cells have been shown to respond biochemically to mechanical and chemical signals, including endocrines, autocrines, and paracrines [6,46].

Histochemical and immunohistochemical studies demonstrate that early tooth

movement involves PDL fluid movement and cellular and matrix distortion [46]. Vasoactive neurotransmitters are released from PDL nerve terminals, causing leukocytes to migrate out of surrounding capillaries [46]. Cytokines and growth factors are also secreted, stimulating differentiation of mesenchymal cells into fibroblasts, osteoblasts, and other remodelling cells [5]. These cells in turn can affect the adjacent alveolar bone and tooth movement occurs. Many cells of the nervous, immune, and endocrine system play essential roles in tooth movement, as do local cells of the PDL and alveolar bone. Research today continues into each of these areas, attempting to explain fully the phenomenon of tooth movement.

1.6.3 - PERIODONTAL LIGAMENT RESPONSE TO ORTHODONTIC FORCES

The periodontal ligament (PDL) separates and attaches each tooth to the adjacent alveolar bone, occupying a space of about 0.5 mm around the entire root surface. It is comprised mainly of heavy collagenous fibers, with many of the principal fibers arranged obliquely to withstand pressure during mastication. Towards the gingival margin some fibers are anchored to the alveolar crestal bone. As well, the free gingival fibers of the supraalveolar tissue constitute another group, anatomically distinguished as dentogingival, dentoperiosteal, transseptal, alveologingival, and circular fibers [49]. After orthodontic tooth movement, fibers on the tension side of the tooth may appear histologically to be stretched [50]. There is a reduction in the number of cells of the supraalveolar fibers as cellular elements are compressed by stretched fiber bundles [50].

Several types of extracellular fibers are found within the PDL. The collagen fibers are the most significant and dominate the periodontal space [6,40]. These fibers are made of fibrils of indefinite length [51]. They consist of type I collagen and type III collagen arranged into bundles of different orientation (the principal bundles) [52]. The collagen of the PDL is constantly being remodelled and regenerated during normal function [52]. This process is accelerated by induced orthodontic forces. Fullmer observed the presence of oxytalan fibers within the collagenous tissue of the PDL, anchored to the cementum [53]. Sims described the oxytalan fiber system of the PDL as "a three-dimensional fiber meshwork" that "underwent reconstruction and adaptation to extensive metabolic and anatomic changes within the periodontium" during tooth movement [54]. Oxytalan and collagen fibre bundles have been

found to be intricately interwoven [55]. Oxytalan has been implicated in both tooth eruption and support mechanisms, as well as influencing blood flow, but these functions have yet to be proven [56]. It was postulated that oxytalan fibres had elastic properties and may be important in orthodontic relapse, as "the apparent increase in oxytalan fibres during orthodontic treatment might indicate that the oxytalan fibres have some anchoring effect which would prevent overstretching of the tissue in certain areas" [57]. However, more recent studies have not supported this, as "no evidence was found to sustain the claim that oxytalan fibres...provide any anchoring effect" and "these fibres seem highly unlikely to be responsible for relapse of tooth movements" [58]. "Reconstitution of the oxytalan system provided evidence against the concept that oxytalan fibers are stretched by orthodontic movement and subsequently contribute to relapse by elastic rebound" [54].

The collagen fiber bundles of the PDL are stretched as a tooth moves. It is believed that there is a proliferation zone in the PDL where an intermediate plexus exists, with connective tissue cells producing collagen. As this is incorporated into fibrils, elongation of PDL fibers continues as the tooth moves [59].

The fibrils of the PDL are embedded in a ground substance, containing glycosaminoglycans (GAGs), proteoglycans, salts, various other substances, and water [6]. The ground substance provides a medium into which the connective tissue cells can produce collagen that subsequently aggregates to form the fibrils [52]. The fluid content of the ground substance allows the PDL to act as a shock absorber against forces applied to the teeth [4].

The other major constituents of the PDL include the cellular elements such as

mesenchymal cells, vascular and neural components. The undifferentiated mesenchymal cells provide a source of fibroblasts and osteoblasts as needed [60]. Fibroblast-like cells comprise 50% of the PDL by volume [61]. In rat PDL, approximately half of these then contribute to osteoblast formation, while the rest are involved in PDL maintenance [60,62]. Fibroblasts are essential for collagen synthesis and degradation associated with PDL remodelling, both physiologic and orthodontic [63]. Transmission electron microscopy has demonstrated that "a portion of the population of PDL cells which proliferate in response to orthodontic force represent functional ligament fibroblasts" [64]. Significantly more fibroblasts have been found in appositional areas of the PDL than in resorptive areas ($p < 0.05$) [65]. The derived cells can apparently function as both fibroblasts, forming new collagen, and fibroclasts, removing existing aged collagen fibrils [63,66]. In addition to secreting and resorbing collagen, proteoglycans, and glycoproteins, fibroblasts can contract and migrate [67-70]. Fibroblasts seem to produce oriented fiber systems *in vitro*, and new cells from one part of the ligament can migrate to another [71]. These cells migrate in the healing of injury to the PDL and in response to orthodontic stimulation [63]. It has been shown that stretching of the principal fibers and widening of the PDL space prompts an increase in cellular elements [72].

The PDL reacts to forces on the teeth by mediating the response of the surrounding structures. Different stages are encountered depending on the stimulus. Under normal masticatory function, heavy, but intermittent, forces are transmitted to the teeth and PDL. With tooth contacts of fractions of a second and force magnitudes up to 50 kg, the incompressible fluid within the PDL transmits the force to the alveolar bone while preventing

tooth displacement [4]. This in turn causes the bone to bend, producing bioelectric currents, thought to be important in stimulating osseous remodelling and regeneration [47]. If heavy pressure against the teeth is maintained for greater than one second, the PDL fluid is squeezed out the pores in the alveolus, displacing the tooth within the PDL space. This results in compression of the PDL against adjacent bone, which is perceived as pain after 3 - 5 seconds of force application [4]. As long as forces are light and/or less than one second duration, i.e. in normal mastication, tissue fluids prevent this undesirable response [4].

If a force of greater duration than 5 seconds is applied to a tooth, the PDL loses its ability to act as a shock absorber [4]. As tissue fluid is squeezed out under heavy pressure, compression of the PDL leads, not only to pain, but also to necrosis of the cellular elements. "Indirect" resorption of adjacent alveolar bone is the result [40]. If prolonged light forces are used, cellular elements of the PDL survive and begin the process of "direct" resorption of the alveolar bone, which is relatively painless [4].

Cells of the PDL and alveolar bone differentiate and begin remodelling in response to cellular activity of the nervous, immune, and endocrine systems [46]. Studies of each of these areas has become the focus of understanding more fully the process of tooth movement.

The neural response is one area which has received significant attention with recognition of different types of nerve endings and recent identification of new neurotransmitters [73]. Molecules such as substance P (SP), calcitonin gene related peptide (CGRP), vasoactive intestinal peptide (VIP), neurofilament protein (NFP), among others, have been found in the PDL [73]. The activity of each of these substances is likely related to

their concentration, which varies with different stimuli. However, their role is as yet not fully understood, particularly in relation to tooth movement. Nerve growth factor (NGF) is one such substance as it is known to be essential for growth, differentiation, and maintenance of sympathetic and sensory neurons. Its distribution and concentration is altered in tooth movement, and is likely to be associated with the bone remodelling [74].

Kato *et al.* looked at the distribution of vasoactive intestinal polypeptides and calcitonin gene related peptide in the PDL of mouse molar teeth, noting that CGRP fibers were most prevalent in the lower third of the root [75]. In the furcation area, some VIP-containing fibers, but no CGRP fibers were detected [75]. Increases in levels of CGRP were noted in response to pulpal inflammation and periapical formation in rat molars, as well as after replantation of teeth [76,77]. Depending on the degree of injury to the pulp and periodontium, progressively increasing levels of CGRP were noted, possibly corresponding to clinical symptoms experienced by dental patients [78].

As the neural response of the PDL appears to play an essential role in tooth movement, changes in different neuroactive peptides during tooth movement may be expected. CGRP-positive nerves increased after the first three days of tooth movement in rat molars, only to return to normal levels by the seventh day [79]. Since this corresponded to the increase and decline of blood vessels in the PDL in the same time frame, it likely indicates that CGRP is involved in blood flow regulation of the PDL. An increase in CGRP-immunoreactive nerves with tooth movement was found to be reproducible; the majority of teeth moved showed increases in the coronal pulp and periapical tissues [79]. This supports the

suggestion that CGRP plays some role in orthodontic tooth movement [80]. CGRP-immunoreactive nerve fibres increased in concentration around blood vessels during tooth movement initially, then decreased after seven days [81]. These changes in CGRP-immunoreactive nerves of the PDL may induce pain [81]. CGRP is known to have vasoactive, sensory, and neuro-modulating functions [80]. Due to their wide distribution, CGRP type nerves are likely involved in the diverse regulatory, reparative, and maintenance functions of the PDL [82].

Other neuropeptides have shown increases in response to tooth movement also, such as substance P, a neuropeptide found in high concentration in the posterior root of the spinal cord [83]. Substance P has been implicated in the transmission of painful stimuli and in the mediation of inflammation [83]. Its presence in tissues with high remodelling potential, such as the PDL and in bone periosteum, suggests that it may accelerate remodelling through mediation of an inflammatory response, in addition to its role in pain transmission [84]. Orthodontic tooth movement increased the levels of SP-immunoreactivity in the pulp rapidly, then later in the PDL in areas of compression [83]. The early peak in the pulp is consistent with SP's role in pain perception, while its later increase in the PDL may suggest its participation in alveolar bone remodelling [83]. The orthodontic forces may have induced the release of SP, which functions as an initial trigger for a biochemical cascade involving the activation of various cells of the PDL [83].

The importance of identifying the presence of different types of nerves related to tooth movement in beginning to understand their role is obvious. In the PDL of mouse molars,

morphometry indicates that approximately 95% of all periodontal axons are unmyelinated, with the greatest proportion of myelinated axons in the gingival third of the ligament [85]. Loescher and Holland found only 61% unmyelinated axons in lower cat canine teeth at 1 mm from the tooth apex, with the number of axons decreasing towards the crown [86]. Immunohistochemistry was used to identify nerve endings reactive for neurofilament protein (NFP) and S-100 protein within the human PDL [87]. Four types of nerve endings were identified: Ruffini-like endings mainly around the apices; coiled nerve endings in the mid-region of the PDL; spindle-shaped nerve endings, and expanded nerve endings, both rarely found near the tooth apices [87]. Animal studies have contributed much of the knowledge regarding the types and distribution of nerves in the PDL. Thick NFP-immunoreactive nerves were found to enter the PDL of monkeys through the bottom of the alveolar socket, while thinner NFP-immunoreactive nerves infiltrate the ligament from the lateral walls [88]. The PDL of the monkey molar was further thought to be innervated by both the trigeminal ganglion and mesencephalic nucleus [88]. Other studies have used NFP and S 100 protein to look at the distribution pattern of Ruffini endings in rodent PDL, showing that the pattern was similar for all rodents examined [89,90]. In another study, it was found that pulpectomy in ferrets does not affect the incidence or distribution pattern of nerves in the PDL [91]. This is significant as it demonstrates that pulpal innervation or denervation would likely have no effect on PDL response.

1.6.4 - PULPAL RESPONSE TO ORTHODONTIC FORCES

Traumatized teeth undergo a variety of injuries from coronal and/or root fracture to complete avulsion. Depending on the severity of the injury, the pulp too is subjected to varying amounts of insult, occasionally resulting in pulpal necrosis [92]. If the pulp survives, secondary dentin is invariably formed, sometimes leading to complete obliteration of the pulp canal or internal resorption [92,93]. Orthodontic forces are regarded by many as a form of controlled micro-trauma to the teeth, although this may not be completely accurate as the forces applied are for a much longer duration than most traumatic injuries [6].

Certain changes are known to occur in the pulp of teeth undergoing orthodontic treatment. The application of orthodontic forces on a tooth results in a transient inflammatory response in the pulp, which may account for some of the discomfort patients experience at the beginning of treatment [94]. Some suggest that a depressed pulpal respiratory rate accompanies force application, with greater significance in older patients, as there was a direct correlation between increased age of patient and degree of respiratory depression [95]. This was shown to be related to the apical opening size, which decreases with age. Thus, as patients age, the blood flow and subsequent respiratory recovery after orthodontic force application is reduced [96]. Other studies have shown an increase in the blood flow to the pulp with tooth movement, which may accompany the increased cellular activity surrounding the tooth as it moves through bone. Kvinnsland *et al.* used fluorescent microspheres to demonstrate a significant increase in blood flow in the pulps and PDLs of mesially tipped rat molars [97]. Similar findings in vascular response were reported by Stanley *et al.*, who suggested there

were capillaries present that only became functional when the pulp was traumatized [98]. A histomorphometric study by Nixon *et al.* not only demonstrated a significant increase in vascular supply to the pulp, but further found that this increase was correlated to time and amount of force applied [99]. These variances in the literature may be related to differences in experimental subjects, type of tooth moved, method of measurement, and even type of tooth movement used. In any case, clinically, this mild pulpitis has little significance as it quickly dissipates and rarely becomes problematic [4].

In addition to the inflammatory response, vacuolization of the odontoblast layer is characteristically observed in the pulps of teeth subjected to orthodontic forces [8]. These changes were found to be less with intermittent forces than with a conventional fixed orthodontic appliance, and almost non-existent when a removable appliance was used [6]. The duration of force application was also found to affect the blood flow of the pulp [99]. This may be important in treatment planning to decrease the risk of devitalizing teeth.

The type of tooth movement also appears to have varying effects on the dental pulp. Some studies suggest that intrusive forces can restrict pulp circulation. An initial decrease in blood flow was shown in rats by Guevara and McClugage using *in vivo* microscopy [100]. A depressive force that is excessive has been implicated in impeding the arterial supply in the apical region, resulting in devitalization of the pulp [99].

Characteristic pulpal changes have also been found with extrusive movements. Using human premolars, Mostafa *et al.* placed extrusive forces on teeth and showed circulatory disturbances with congested and dilated blood vessels, odontoblastic degeneration,

vacuolization, and edema of the pulp [101]. It was suggested that these changes were again the result of a compromised blood flow, but the results indicated the need for further studies in this area.

Another method of examining changes in the pulp following orthodontic treatment is to use radiographs. Popp *et al.* took radiographs of patients prior to orthodontic treatment, after retention, and approximately 5 years out of retention and compared these to patients who had not had orthodontic treatment [102]. Their findings showed occasional radiographic changes in the pulp and PDL of orthodontically treated teeth similar to those expected in traumatized teeth. With time, a narrowing of the pulp canal was seen in both treated and untreated teeth. This was attributed to aging. No other significant differences between the two groups were noted.

Although minimal pulpal changes are shown by most studies, there are still occasional reports of loss of tooth vitality during orthodontic treatment [7,103]. Poor control of orthodontic forces is usually responsible, often due to use of excessive continuous forces [4]. This will result in several stages of "indirect" resorption, with abrupt, relatively large increments of tooth movement [40]. These movements would be highly traumatic, possibly severing the blood vessels entering the root apex. The same is true for root apices that must be moved great distances, as the likelihood of compromising the blood supply increases with amount of movement. Movements such as the root torquing of incisors, bringing an impacted maxillary cuspid into occlusion, or any movement where the tooth apex must be relocated excessively will have increased risk of losing pulp vitality [8,99,100].

The literature regarding pulpal changes in response to orthodontic forces appears to justify the clinical impressions of many orthodontists. That is, changes within the pulp are known to occur histologically, but are transient in nature and pose minimal risk to the vitality of most teeth [4,97,99]. The level of trauma induced by orthodontic treatment is slight and very rarely exceeds the physiologic limits of healthy dental tissues [4,99].

1.6.5 - BONE RESPONSE TO ORTHODONTIC FORCES

The PDL and pulp responses to orthodontic forces have been discussed as they are tissue mediators of the effects of these forces [4]. The actual remodelling of the alveolar bone constitutes the most significant results of these mediators, as this is how tooth movement per se actually occurs. If it were not for the remodelling ability of the bone, orthodontic treatment would not be possible [4,6]. The observation of bony remodelling has long been recognized, but it is only with time and the refinement of research techniques that this process is beginning to be understood.

A generally accepted rule in tooth movement is that alveolar bone is resorbed whenever the PDL is compressed for a sustained period. Alveolar bone deposition occurs wherever there is a tensile force on the bone [104]. As previously discussed in 1.6.2, factors such as force direction, magnitude, and duration have significant effects on this general principle. In addition to the compressive and tensile forces relayed to the bone through the PDL, bioelectrical changes induced by bending of the alveolar bone have been described [47,48,105]. Currently, the two hypotheses that attempt to explain the control mechanism behind bone remodelling centre around these events.

Differing areas of pressure and tension in the PDL cause alterations in the vasculature, with blood flow variations resulting. As pressure is sustained on a tooth, it shifts position within the PDL. The ligament is thus compressed in some areas, decreasing blood flow, while it is stretched in others, increasing blood flow [97,99]. Presumably, this would cause a reduction in oxygen levels on the pressure side while an increase would occur on the tension side [97,99].

These changes in PDL cellular respiration would be effected quickly, which in turn could affect cellular metabolism. As the metabolism changed, chemical alterations and subsequent cellular differentiation could result, initiating tooth movement [106]. Justus *et al.* suggested that the linkage between the compressive and tensile forces on the PDL could be mechanochemical in nature, with strain on the bone causing a change in the solubility of the hydroxyapatite crystals and acting as a chemical mediator [107].

The piezoelectric theory relates tooth movement to alterations in bone metabolism produced by the generation of electrical currents as the bone bends or flexes [47,48,105]. The piezoelectric effect was described by Fukada and Yasada, who found that the bending of bone mechanically resulted in the generation of electric potentials in areas of compression and tension [108]. Studies by other researchers have confirmed this, not only in long bones, but also in alveolar bone, with electrical currents associated with widening of the PDL [109,110]. This formation of piezoelectric currents is hypothesized to stimulate osteoblastic and osteoclastic activity within the PDL and adjacent alveolar bone, remodelling the tooth socket and allowing tooth movement to occur [47,48,105]. There is no question that piezoelectricity is necessary for the maintenance of skeletal bones [48,109]. Bone atrophy through loss of mineral content is known to occur without these signals [105,109]. Piezoelectricity is only one form of bioelectric potential formation. A second, possibly more important source, is described by Borgens, who detected endogenous ionic currents that were attributed to streaming potentials rather than to piezoelectricity [111]. He suggested that the source of current in mechanically stressed bone is cells rather than matrix. Otter *et al.*, in examining wet and dry bovine tibia, concluded that

while in the dry state the current is primarily piezoelectric, in wet bone the dominant mechanism is streaming potentials [112]. Whichever is the source of electric potentials in bone, it seems that these endogenous bioelectric currents are involved in bone repair, remodelling, and even growth [46]. The importance of this as it relates to tooth movement and its extent in alveolar bone remodelling remains unclear.

Since the formation of electrical potentials occurs simultaneously with the application of pressure and tension to the alveolar bone, it is difficult to determine which is responsible for the initiation and control of osseous remodelling [47,48,105]. As the two theories are not mutually exclusive, both mechanisms may play a role [4].

Regardless of the mechanism responsible, once bone formation has been initiated, it passes through three stages: osteoid, bundle bone, and lamellated bone [49]. As a precursor to bone formation, there is a noticeable increase in the number of fibroblasts and osteoblasts through mitosis [69,113]. These come solely from the undifferentiated local precursor cells of the PDL [62]. New osteoblasts are often seen arranged in a chain, known as a proliferation zone. From this zone, the osteoblasts begin to form osteoid, which, unlike calcified bone, is not resorbed by osteoclasts. It has been demonstrated that the formation of osteoid takes longer in adults than adolescents, possibly due to a latency in initiating osteoblast differentiation [114].

Mature or lamellated bone consists of a matrix of collagen fibrils and hydroxyapatite crystals in a cementing substance [49]. These collagen fibrils must have a periodicity of 640 to 700 Å in order for bone to form, in growth or remodelling [6]. The uncalcified osteoid forms from fairly thick collagen fiber bundles, present on the lamina dura during tooth movement.

As this tissue begins to calcify, it appears radiographically as a wide opaque line along the lamina dura [115]. As this layer thickens, the earliest formed portions begin to calcify, with the newest layers remaining uncalcified. This newly calcified tissue is known as woven or bundle bone, with a relatively high content of the cementing substance, known to be mostly polymerized connective tissue polysaccharides [116].

The bundle bone then begins to mature as osteoblasts become incorporated as osteocytes and more fiber bundles are added. At a certain thickness and maturity, portions of the bundle bone are reorganized into lamellated bone, with finer fibrils in its matrix [6]. A demarcation known as a reversal or resting line is seen between old bone and bundle bone [6]. It has been shown that recently calcified bundle bone is more readily resorbed than mature bone [114]. The importance of mature, lamellated bone formation, along with PDL reorganization, is that relapse tendencies are lessened as the periodontium matures [4,6].

Changes seen on the pressure side of the PDL involve compression of its cellular, vascular, and fibrous elements. The degree and duration of compression determines which type of resorption, direct or indirect, occurs [4,40]. Direct or frontal bone resorption results from light compressive forces, as osteoclasts form directly along the bone surface in the area of PDL compression, removing bone adjacent to the PDL and beginning tooth movement. Osteoclasts are thought to form from monocytes in the PDL in response to a localized reduction in blood flow as the vasculature becomes compromised [117,118]. This means that the cellular elements of the PDL must survive the compressive forces in order to differentiate in this region, which is generally the goal of applying only light, continuous forces [4,6,40]. As these

forces become excessive, hyalinization of the PDL is seen in the area of compression, meaning that the zone becomes avascular and cells become necrotic and disappear, resulting in a cell-free zone of compressed collagenous fibres [6]. Without precursor cells, osteoclasts cannot form in this region, temporarily ceasing bone remodelling. Consequently, direct bone resorption can only occur when compression of the PDL is light and does not cause hyalinization [6,57].

Once the necrotic hyalinized tissue has formed, bone remodelling can only occur from the undamaged areas adjacent to the zone of necrosis [4,6,40]. This means that cellular recruitment from cancellous bone beneath the hyalinized area and other areas of the PDL must ensue, in the process of indirect or undermining resorption [4,40]. This process occurs several days later, characterized by the differentiation of osteoclasts within adjacent bone marrow spaces which begin to resorb the underside of the bone next to the necrotic PDL area. There is an inevitable delay in the tooth movement with hyalinization and indirect resorption for several reasons [4,40]. First, the cells within the marrow spaces are somewhat distant from the bioelectric and chemical stimulus of the compressed PDL, thus their differentiation is delayed. Secondly, the time it takes to remove and undermine the thick cortical bone of the socket is greater than with direct resorption. As this occurs, the tooth will not move for a period until the bone neighbouring the hyalinized region has been eliminated. The length of this delay is directly proportionate to the extent of the hyalinized area which in turn increases with excessive forces. Once the cortical bone has been undermined sufficiently, it gives way suddenly, resulting in an abrupt movement of the tooth. Not only is this sudden movement

somewhat painful, it also risks tearing the neurovascular bundle entering the pulp through the apex [4,40]. This in turn increases the chances of devitalizing the pulp, an undesirable sequela of tooth movement [4,7,103].

The region of the PDL which is compressed and potentially subjected to hyalinization depends on the type of tooth movement attempted. In tipping, the hyalinized area will be located near the alveolar crest; in a bodily movement, it will be closer to the middle of the root [119]. In rotational movements, there are almost always two compressed zones, depending on the cross-sectional shape of the root [50,57]. With excessive tipping forces or with torquing movements, a second hyalinized area can form in the apical region [6]. The extent of these areas will depend on the magnitude of the force applied.

The avoidance of heavy, hyalinizing forces should be the goal of orthodontic mechanics. Despite the best efforts of well planned treatment, it is virtually impossible to avoid indirect resorption completely [4,40]. By applying a light initial force, cellular activity in the PDL can be increased without causing excessive ligament compression. However, even with light forces, small areas of PDL necrosis are inevitable, resulting in areas of indirect bone resorption [4,6,40]. In reality, it is a combination of direct and indirect resorption that accompanies most tooth movements; the key is to maximize the amount of direct resorption and minimize indirect resorption through the controlled use of light forces to move teeth efficiently.

The nature of the chemical mediators that initiate osteoblastic and osteoclastic differentiation is an area of interest in further understanding the mechanism of tooth

movement. Yamasaki *et al.* used rats to show that indomethacin, a non-steroidal anti-inflammatory which inhibits prostaglandin production, prevented the formation of osteoclasts, delaying bone resorption [120]. Administration of imidazole and verapamil also inhibited osteoclast formation [121]. Conversely, prostaglandin solutions injected near the experimental teeth caused the appearance of osteoclasts and bone resorption [120]. Administration of theophylline and ouabain were also found to enhance osteoclast formation [121]. Other studies have found increases in cAMP (cyclic adenosine monophosphate) and enzymes such as alkaline phosphatase and succinic dehydrogenase in conjunction with increased osteoclast activity [122,123,124]. The role of these chemical mediators, among others, is being investigated further, however, their function at present is still not clearly understood [73].

1.6.6 - ROOT RESPONSE TO ORTHODONTIC FORCES

Tooth movement requires resorption and apposition of bone adjacent to the roots of the teeth. As previously discussed, this occurs through the activation of cellular elements of the PDL and alveolar bone, initiating bone remodelling. It was once thought that the roots of the teeth were not subjected to this remodelling process as they are much harder than bone and are acellular [4]. However, recent findings have made it obvious that the roots too are prone to resorption, which increases with application of orthodontic forces [4,15,16,49,104].

Root resorption on extracted permanent teeth was first described in the nineteenth century by Bates and Schwarzkopf [125,126]. By the early twentieth century, this phenomenon was well recognized and began to be studied more extensively. Ottolengui was the first to relate root resorption to orthodontic treatment, which was later confirmed radiographically by others, such as Ketcham and Rudolph, looking at root shape and resorption lacunae respectively [127,128,129]. These initial observations have led to a plethora of research on root resorption and orthodontic treatment, although much remains to be learned.

The process of root resorption is a normal physiological process in deciduous teeth, allowing eruption of the permanent dentition. In fact, some deciduous teeth show evidence of root resorption even with congenitally missing succedaneous teeth [130]. This suggests that there may be inherent factors that influence root resorption unrelated to the eruptive force of permanent successors. For example, children with cleidocranial dysplasia often have developed succedaneous teeth, yet the overlying bone and roots of the deciduous teeth fail to resorb, impeding eruption. Moreover, in some cases where deciduous teeth do resorb and

exfoliate, the permanent teeth still do not erupt, termed "primary failure of eruption", believed to be a defect in the eruption mechanism [4]. These phenomena have yet to be explained or understood entirely, as the control process of eruption remains as controversial as ever. It is obvious that the resorption potential of deciduous teeth is higher than that of permanent teeth. Whether or not resorption of permanent teeth progresses by the same fashion as that of deciduous teeth is not clear. The processes may be similar in effect, but without a thorough understanding of the controlling mechanism of either, it is impossible to state unequivocally [4,130]. Physiological resorption and its control mechanism are clearly not simple issues and with the addition of external factors such as orthodontic force, the picture is clouded further.

Root resorption can be divided into internal resorption, which is of pulpal origin, and external resorption, which proceeds from the PDL towards the pulp. External root resorption is subdivided into three types: *surface resorption*, a self-limiting process which involves small areas and undergoes spontaneous repair from adjacent intact parts of the PDL; *inflammatory resorption*, where initial root resorption has reached dentinal tubules of an infected necrotic pulpal tissue or an infected leukocyte zone; and *replacement resorption*, where resorbed tooth structure is replaced by bone, leading to ankylosis [18,92]. Of these, root resorption related to orthodontic treatment is of the surface type, with most defects undergoing repair and not appearing on radiographs [18].

Although not to the same extent as their deciduous counterparts, permanent teeth have also been found to resorb physiologically. In a radiographic study of normal and

orthodontically treated individuals, Massler *et al.* determined that all people "showed some evidence of periapical resorption in... permanent tooth roots" and 86.4 % of teeth examined had some evidence of root resorption [131]. In most cases, there were no obvious reasons for the resorption to occur (eg. periapical infection, trauma, endodontic treatment, etc.). Histologically, Henry *et al.* found similar results, with 90.5 % of untreated teeth showing microscopic changes associated with resorption, with most occurring near the root apices [132]. Upon examination of orthodontically treated patients, Massler *et al.* found that 93.3 % of teeth showed evidence of resorption, which was only slightly higher than the untreated teeth [131]. However, "although the number of teeth involved by resorptions was increased only slightly after orthodontic treatment, the severity of these resorptions was very obviously and markedly increased" [131]. It is suggested that earlier investigations by Rudolph (1936) - 74 %, Becks (1939) - 73.6 %, and Hemley (1941) - 3.5 % may have only reported the more severe instances of resorption, resulting in lower reported frequencies [129,133,134]. Additionally, the quality of radiographs available may not have allowed detection of smaller lesions, and the subjectivity of the observations vary the results greatly.

It was noted by Becks, through the course of several studies, that orthodontic treatment was not the sole factor in the production of root resorption in patients [133,135,136]. He believed that certain patients have a predisposition to root resorption and would experience significant resorption when exposed to orthodontic forces. Those that were not prone would show little evidence of root resorption [133]. This opinion is shared by many other authors, although the reason for the susceptibility varies with each [11,12,15,16,137]. The predisposing

factors that appear to alter an individual's susceptibility to root resorption can be categorized as either biologic or mechanical. Biologic influences including individual susceptibility, hereditary predisposition, systemic, local, and anatomic factors associated with orthodontic treatment are commonly listed as potential risks [137].

Individual susceptibility to root resorption varies not only among people, but also in the same person at different times. Physiological changes induced by fluctuating hormone levels, metabolic rate alterations, and growth period variations can modify specific cellular metabolisms [19,137]. In turn, a person's reaction to disease, trauma, and aging would change, resulting in differing responses to tooth movement, including degree of root resorption. This would potentially explain different individual tendencies to root resorption as a result of orthodontic treatment [4,6,19,137].

Other factors that alter a person's normal physiological response could potentially affect the response of teeth to orthodontic forces also. This includes any systemic disturbance that can modify metabolic activity, such as hypothyroidism, hypopituitarism, hyperpituitarism, among other endocrine disorders [19,137]. Hyperparathyroidism, hypophosphatemia, and Paget disease have also been implicated as potential risk factors related to increased root resorption [138,139,140]. Although the role of hormones in tooth movement is not clearly understood, it has been suggested that an imbalance may not cause but rather only influence root resorption [141].

Normal metabolism is dependent on adequate nutrition. Hence, it is reasonable to assume that malnutrition would result in a modification of metabolic activity which in turn would affect root resorption [142]. Specifically, calcium ions are thought to play an important

role in mediating the effects of external stimuli, including hormonal and mechanical influences, on target cells [143]. Studies have been inconclusive, with some demonstrating increased root resorption with dietary deficiencies in calcium and vitamin D while others found little influence of diet on this phenomenon [135,141]. Tooth movement in rats has also shown controversial results in root resorption when a low calcium diet was implemented [143,144].

Chronologic age plays an important role in the activity level of all tissues, including those involved in root resorption [137]. The vascularity and pulp size of older teeth are substantially reduced from that of their youthful counterparts, resulting in wider dentin and smaller pulp chambers. The PDL narrows, becoming less vascular and less adaptable while the alveolar bone becomes denser, avascular, and aplastic. There appears to be a positive correlation between the progression of these changes and the susceptibility to root resorption, as evidenced by the increased incidence of root resorption in adults [49]. Some authors found that the degree of root resorption increased with age even without orthodontic treatment [131,137]. Older teeth will also have greater exposure to micro-trauma and macro-trauma. This often results in small areas of cemental resorption with subsequent repair [18]. Whether or not there is a cumulative effect of this continuous remodelling is not known. Thus, although there appears to be some correlation between age and root resorption, there is no clear explanation for this observation [137].

In cases where the macro-trauma is significant, the involved teeth may exhibit external root resorption without orthodontic treatment [18]. The movement of these teeth orthodontically increases the potential for further resorption of root material [18,145]. One study

showed that the average loss of root length in orthodontically treated traumatized and untraumatized teeth was 1.07 mm and 0.64 mm respectively [141]. However, in traumatized teeth without signs of resorption prior to orthodontic treatment, this increased risk is not found [146]. Conversely, untraumatized teeth that show signs of root resorption prior to orthodontic treatment are prone to further resorption [137]. There is a high correlation between the amount and severity of root resorption present before treatment to the root resorption observed after treatment, both in traumatized and untraumatized teeth [131]. One study found that the incidence of root resorption increased from 4 % before orthodontic therapy to 77 % after treatment [147]. Further, root resorption detected during the first 6 to 9 months of treatment indicates an increased risk of further resorption [148]. On the other hand, if no resorption is detected in this initial time period, little will be expected with further treatment [137,148].

It is known that the ease and degree of orthodontic tooth movement depends on the anatomy of the root surface [4,6,40]. Larger, multi-rooted teeth are obviously more difficult to move than teeth with single, smaller roots. As the force applied to the root surface varies with its area and volume, it is logical to assume that the amount and severity of root resorption would also be determined partially by the root structure. Root forms that deviate from normal have been found to be more susceptible to orthodontic root resorption [149,150]. Teeth with blunt or pipette-shaped roots were found to experience significantly higher levels of root resorption than teeth with normal roots; the pipette-shaped root was especially susceptible [148]. Another study found that teeth with convergent apical root canals were also

prone to a greater degree of root resorption with orthodontics [151]. In incompletely formed roots with very wide apices, Rosenberg reported a decreased incidence of root resorption over those with completely formed roots [152]. This observation was reproduced in another study in which an average loss of 0.5 mm root length was found in developed teeth that were orthodontically treated over undeveloped counterparts [141].

Even with complete root formation, it is obvious that different types of teeth have different root forms. This affects their susceptibility to resorption, although all orthodontically treated teeth will show some. Most studies have found that maxillary teeth are more susceptible than mandibular teeth with the maxillary incisors showing the greatest degree of root resorption [128,131,145,153,154]. This is partially explained by the fact that these teeth usually undergo the most movement due to malocclusion, function, and esthetics. One study suggested that due to their root structure and relationship to the bone and PDL, forces on these teeth tend to be transferred to the apical region, causing increased root shortening [155]. Other studies have found the mandibular incisors to be more susceptible to resorption than the maxillary ones, but these are the exception [134,156]. In either case, it is believed that if there is no apical root resorption seen in the maxillary and/or mandibular incisors, significant apical resorption in other teeth is unlikely [147,157]. In terms of incidence of root resorption, from most to least susceptible, the teeth are ranked as follows: maxillary lateral incisors, maxillary central incisors, mandibular incisors, distal root of mandibular first molars, mandibular second premolars, and maxillary second premolars [49,134,145,147,156,157,158]. Teeth that are not mentioned have insignificant amounts of resorption compared to the listed ones.

The movement of teeth through bone would suggest that bone density plays a significant role in orthodontics, affecting rate and extent of movements as well as amount of root resorption. Several researchers have found that denser alveolar bone resulted in increased root resorption during orthodontic treatment [6,143,159,160]. In less dense bone, more marrow spaces will be present, from which will come more osteoclasts, increasing bone resorption and facilitating tooth movement. A strong continuous force on less dense alveolar bone will cause the same degree of root resorption as mild continuous force on very dense alveolar bone [161]. A further observation is that direct contact between roots and cortical bone increases the stress at the apex, precipitating root resorption [159]. This is especially a risk in torquing movements, usually involving the maxillary incisors [162].

Conflicting evidence has been found, however, suggesting that bone density may affect the rate of tooth movement, but not the extent of root resorption [163]. Experiments with calcium deficient rats have found decreased bone density associated with decreased root resorption in one case and increased root resorption in another [143,144]. Confounding factors such as incidental hyperparathyroidism and lactation may have been responsible for these differing observations [137]. It is clear that further studies in this area are required.

Other researchers have looked at gender as a possible predisposing factor to root resorption. Some studies have found no correlation between gender and root resorption between treated and untreated teeth while other studies have shown females to be more susceptible to root resorption [131,142,149,151,153,154,162]. One study suggested an idiopathic root resorption ratio of 3.7:1 females to males, respectively [149]. Apical root length shortening was

found to be greater in treated females (0.73 mm) than in treated males (0.67 mm) [141]. However, this difference may reflect the increased root development and dental maturity of the females over their male counterparts rather than an actual gender difference. Even in cases where a discrepancy was found, hormonal differences were not advocated as being the cause of the disparity. The relationship between gender and susceptibility to root resorption appears weak at best [137].

Teeth which have had previous endodontic treatment can be moved orthodontically without much difficulty [11,137]. It is controversial whether these teeth are more susceptible to root resorption than their vital counterparts in response to orthodontic forces [10-12,14-16,137]. This argument constitutes one of the main reasons for this study and will be discussed at length in the next section. Suffice it to say that the literature is inconclusive as to whether endodontically treated teeth are more or less susceptible to root resorption with orthodontics.

In addition to all the biologic variations that change the root resorption potential, there are numerous mechanical factors that have been implicated. The most obvious mechanical influence on the biologic system from orthodontics is the force applied to move the teeth. The level of this force has been found to have profound implications with respect to root resorption. Higher stress associated with greater forces is one of the most significant causes of increased root resorption [4,164]. According to Schwartz, orthodontic forces exceeding the optimal level of 20 to 26 g/cm^2 result in PDL ischemia, often preceding root resorption [115]. Consistent correlations between increased levels of force and increased degree of root resorption, in addition to the efficiency of using gentler forces, has led to the use of

progressively lighter forces in modern orthodontic appliances [4]. The use of heavy, continuous forces is to be avoided as much as possible, since this method is not only painful, but unpredictable and biologically damaging [4].

In discussing force levels, it is necessary to consider not only magnitude, but duration as well. One study looked at root resorption resulting from fixed and removable appliances, which apply continuous and intermittent forces respectively [141]. It concluded that the use of fixed appliances is more detrimental to root structure than removable ones. Ketcham claimed that the splinting effect of using a fixed orthodontic appliance disturbed the normal function of the teeth and PDL's, resulting in greater root resorption [128]. Conversely, Stuteville thought the jiggling forces associated with the use of removable appliances were more harmful to the roots [165]. This controversy has not been of great interest since both fixed and removable appliances have limitations and each is used in different types of treatment. The desired final result will justify the mode of treatment, as the increased risk of root resorption in either case is minimal [4,6].

Of greater interest has been the debate over Begg versus edgewise fixed appliance therapy. The classical view on this has been that practitioners using light wire Begg mechanics were gentler to the tissues than edgewise practitioners, who had been using heavier rectangular wires. The Begg appliance was thought to cause less root resorption than edgewise, but other authors have found no difference, or even an opposite result with less resorption using edgewise mechanics [146,151]. With new arch wire alloys and more efficient appliances, the edgewise appliance has been able to reduce force levels dramatically, thus

enabling it too to be a light wire technique [4]. Nevertheless, significant maxillary root resorption using both techniques has been found so the controversy as to which technique is gentler may be irrelevant [137,162,163]. Greater differences are probably found depending on the practitioner's skill rather than the appliance system employed.

Orthodontic tooth movement in general increases the incidence of root resorption regardless of the way the tooth is moved [6,11,137]. However, certain movements have more deleterious effects on root structure than others. Intrusion is probably the most damaging movement as the force applied is concentrated at the root apex due to its small area [8,154,166]. However, tipping, torquing, bodily movement, and palatal expansion have also been implicated, with varying degrees of influence [49,134,141,145,165]. Some authors believe that bodily movement is less damaging as the stress is distributed over the entire root surface rather than at one point, however, this too is controversial [49].

Various studies have found that different treatment modalities can affect root resorption also. The use of elastics has been suggested to increase root resorption due to a jiggling effect, while utilizing magnets to close spaces can stimulate a more physiological response, lessening this potential [141,167,168]. Occlusal trauma on poorly aligned teeth was also thought by some to increase root resorption, but others dispute this [105,148,169]. It is very difficult to show a definitive correlation with most of these variables as individual differences would likely have more variation than the influence of these factors. For this reason, the risk from most of these sources is considered minimal.

An exception to the preceding statement is found when examining the duration of

treatment. Most studies found that the severity of root resorption is directly related to treatment duration [8,105,141,157,158,161]. Rudolph found that the incidence of root resorption at 40 %, 70 %, 80 % and 100% after 1, 2, 3, and 7 years of active treatment, respectively [129]. Goldin reported an average loss of root length of 0.9 mm/year, again showing its progression with time [162]. A few studies did not support these findings, but these were the minority [145,170]. A logical treatment goal is to move teeth as efficiently as possible and minimize treatment time.

Most authors agree that root resorption induced by orthodontics ceases once active treatment is terminated [10,132,145,160,161,170,171,172]. This is expected since tissue pressure is required for root resorption to progress, thus once appliances are removed, so is the source of the pressure [10]. Some authors claim that this resorptive process continues for about a week after appliance removal, followed by 5 to 6 weeks of cemental repair, possibly accounting for slightly more root shortening [49,172]. However, this too is highly variable. Progressive root resorption beyond this time likely originates from sources other than orthodontic forces, such as occlusal trauma, active retainers, or other stimuli [105,169]. Active root resorption following appliance removal can be treated through a combination of splinting the teeth and selective endodontic treatment [173]. Fortunately, cases of this severity are unusual and rare.

In summary, root resorption represents a very real albeit unpredictable phenomenon. Its occurrence in the normal population has been established, however, its severity and incidence appears to increase with orthodontic treatment [11,19,131-138]. The literature indicates there are certain factors which certainly contribute to increased root resorption [4,19,137]. These

include long treatment times, previous root resorption experience, use of heavy forces, and radiographic evidence of susceptible root forms [15,16,19,137]. Other factors are correlated, such as familial predisposition, type of tooth movement, amount of tooth movement, endocrine imbalances, among others [19,137]. However, these influences are controversial and cannot be definitively implicated in cases of increased root resorption. Endodontically treated teeth fall into this category and need to be examined more closely to determine the extent of this correlation, if any. This is the focus of the next section of the literature review. In any case, many authors believe that root resorption has little or no detrimental effect on the life span or function of the dentition [12,145].

1.6.7 - ORTHODONTIC TREATMENT OF ENDODONTICALLY TREATED TEETH

Wickwire *et al.* stated "there is a paucity of documented information concerning the prognosis of the endodontically treated tooth undergoing orthodontic movement" [11]. This statement was made over twenty years ago, yet the literature today has little more to offer than that of the 1970's. The controversy over the response of endodontically treated teeth to orthodontic forces has existed for decades, with the likes of Steadman suggesting that roots of these teeth act as foreign bodies which "melt away as the years pass" [14]. Additionally, Baranowskyj reported delayed bone healing in a dog when intrusive forces were applied [174]. However, others found no difference between bone, PDL, or cementum in orthodontically moved vital and non-vital teeth [175]. This agrees with the clinical impression of most orthodontists who feel that endodontically treated teeth can be moved as readily as their vital analogues. Subsequent studies too have found that endodontically treated teeth can be moved effectively by orthodontics without excessive deleterious effects [11,176].

Root resorption in endodontically treated teeth undergoing orthodontics has also been found to be controversial. Steadman's finding that non-vital teeth responded poorly to orthodontic forces was partially supported by the work of Wickwire *et al.*, who found "a greater frequency of root resorption in the endodontically treated teeth as compared with the control group" of vital teeth [11,14]. Many of the root filled teeth in Wickwire's study had a history of prior trauma, which in itself may have predisposed the teeth to root resorption [11]. It is also possible that these findings were biased by including some teeth that had

unsuccessful endodontic treatment [176].

Conversely, Spurrier *et al.* showed that "endodontically treated incisors resorb with less frequency and severity than vital control teeth" [15]. An earlier study by Huettner and Young on monkeys also found no difference between normal and endodontically treated teeth with respect to orthodontic movement [175]. These results have been substantiated by other researchers, comparing orthodontically treated vital and non-vital teeth in cats and also in a long term follow-up of orthodontic patients [16,177]. It is suggested that the decreased amount of resorption in these patients may be due to an increased density of the dentin following endodontics, providing some resistance against root resorption [6,16]. A histologic investigation was conducted by Satoh using rats as an experimental model [178]. He too found that the "occurrence of external root resorption ... with experimental tooth movement in pulpectomized teeth were lesser and later than those of vital teeth" [178]. Although the difference in root resorption between vital and non-vital teeth in these studies was statistically significant, clinical differences are minimal when endodontically treated and vital teeth are compared [4,15,16].

With such clearly opposing evidence in the literature, it is difficult for clinicians to know whether vital and endodontically treated teeth respond comparably to orthodontic forces. This in turn affects treatment decisions which are based upon predictable tooth movement with minimal root damage. For the most part, the degree of root resorption is minimal and rarely significant clinically, thus orthodontic treatment of endodontically treated teeth is readily instituted [4]. This study has been undertaken to shed more light on this

controversial topic. Hopefully, this will serve to clarify the situation and give orthodontists a further basis for their treatment decisions.

BIBLIOGRAPHY

1. Corrucini RS, Pacciani E. "Orthodontistry" and dental occlusion in Etruscans. Angle Orthod 1989; 59: 61 - 4.

2. Kingsley NW: Treatise on oral deformities as a branch of mechanical surgery, New York, 1880, Appleton.

3. Angle EH: Treatment of malocclusion of the teeth and fractures of the maxillae, Angle's system, ed 6, Philadelphia, 1900, SS White Dental Mfg Co.

4. Proffit WR.: The biological basis of orthodontic therapy. In: Contemporary orthodontics, second edition. St. Louis, 1993, Mosby - Year Book, Inc.; pp. 266 - 88.

5. Melcher AH.: An overview of the anatomy and physiology of the periodontal ligament. In: Norton LA, Burstone CJ, eds. The biology of tooth movement. Boca Raton, Florida, 1989, CRC Press, Inc.; pp. 1 - 7.

6. Reitan K, Rygh P. Biomechanical principles and reactions. In: Graber TM, Vanarsdall RL Jr., eds. Orthodontics: current principles and techniques, second edition. St. Louis, 1994, Mosby - Year Book, Inc.; pp. 96 - 192.

7. Delivanis HP, Sauer GJR. Incidence of canal calcification in the orthodontic patient. Am J Orthod 1982 July; 82(1): 58 - 61.

8. Stenvik A, Mjør IA. Pulp and dentine reactions to experimental tooth intrusion: a histologic study of the initial changes. Am J Orthod Dentofac Orthop 1970; 57: 370 - 85.

9. Chivian M. In: Cohen S, Burns DC, eds. Pathways of the pulp. 4th ed. St. Louis: CV Mosby, 1987.

10. Tronstad L. Root resorption - a multidisciplinary problem in dentistry. In: Davidovitch Z., ed. Biological mechanisms of tooth eruption and root resorption. EBSCO Media, Birmingham AL. 1988.; pp. 293 - 301.

11. Wickwire NA, McNeil MH, Norton LA, Duell RC. The effects of tooth movement upon endodontically treated teeth. Angle Orthod 1974; 44: 235 - 42.

12. Mattison G, Gholston L, Boyd P. Orthodontic external root resorption - endodontic considerations. J Endod 1983 June; 9(6): 253 - 6.

13. Brynolf I. A histological and roentgenological study of the periapical region of human upper incisors. Odontol Rev 18 (Suppl. 11), 1967.

14. Steadman SR. Résumé of the literature on root resorption. Angle Orthod 1942; 12: 29 - 36.

15. Spurrier SW, Hall SH, Joondeph DR, Shapiro PA, Riedel RA. A comparison of apical root resorption during orthodontic treatment in endodontically treated and vital teeth. Am J Orthod Dentofac Orthop 1990 Feb; 97(2): 130 - 4.

16. Remington DN, Joondeph DR, Årtun J, Riedel RA, Chapko MK. Long-term evaluation of root resorption occurring during orthodontic treatment. Am J Orthod Dentofac Orthop 1989 July; 96(1): 43 - 6.

17. Hines FB Jr. A radiographic evaluation of the response of previously avulsed teeth and partially avulsed teeth to orthodontic movement. Am J Orthod 1979 Jan; 75(1): 1 - 19.

18. Andreasen JO. Review of root resorption systems and models. Etiology of root resorption and the homeostatic mechanisms of the periodontal ligament. In: Davidovitch Z., ed. Biological mechanisms of tooth eruption and root resorption. EBSCO Media, Birmingham AL. 1988.; pp. 9 - 21.

19. Brezniak N, Wasserstein A. Root resorption after orthodontic treatment: part 2. Literature review. Am J Orthod Dentofac Orthop 1993 Feb; 103(2): 138 - 46.

20. Klima RJ, Wittemann JK, McIver JE. Body image, self-concept, and the orthodontic patient. Am J Orthod 1979 May; 75(5): 507 - 16.

21. Jacobson A. Psychology and early orthodontic treatment. Am J Orthod 1979 Nov; 76(5): 511 - 29.

22. Albino JE, Lawrence SD, Tedesco LA. Psychological and social effects of orthodontic treatment. J Behav Med 1994 Feb; 17(1): 81 - 98.

23. Jenny J. A social perspective on need and demand for orthodontic treatment. Int Dent J 1975; 25: 248 - 56.

24. Macgregor FC. Social and psychological implications of dentofacial disfigurement. Angle Orthod 1979; 40: 231 - 33.

25. Ingervall B, Mohlin B, Thilander B. Prevalence of symptoms of functional

disturbances of the masticatory system in Swedish men. J Oral Rehab 1980; 7: 185 - 97.

26. Sadowsky C, BeGole EA. Long-term effects of orthodontic treatment on periodontal health. Am J Orthod 1981; 80: 156 - 72.

27. Polson AM. Long-term effect of orthodontic treatment on the periodontium. In: McNamara JA, Ribbens KA, eds., Malocclusion and the periodontium, Ann Arbor, The University of Michigan Press, 1987.

28. Data from 1989 survey of orthodontic practices, AAO Bulletin 8, No. 3, Winter 1990, p. 7.

29. McKiernan EX, McKiernan F, Jones ML. Psychological profiles and motives of adults seeking orthodontic treatment. Int J Adult Orthodon Orthognath Surg 1992; 7(3): 187 - 98.

30. Hester CH *et al.* Glossary of dentofacial orthopedic terms. St. Louis, 1981, American Association of Orthodontists.

31. Thilander BL. Complications of orthodontic treatment. Curr Opin Dent 1992 Dec; 2: 28 - 37.

32. Vanarsdall RL. Complications of orthodontic treatment. Curr Opin Dent 1991 Oct; 1(5): 622 - 33.

33. McDuffie MW, Brown FH, Raines WH. Pneumoparotitis with orthodontic treatment. Am J Orthod Dentofac Orthop 1993 Apr; 103(4): 377 - 9.

34. Boyd RL, Leggott PJ, Quinn RS, Eakle WS, Chambers D. Periodontal implications of orthodontic treatment in adults with reduced or normal periodontal tissues versus those of adolescents. Am J Orthod Dentofac Orthop 1989 Sept; 96(3): 191 - 8.

35. Moriarty JD, Simpson DM. Incidence of periodontal problems to patients with dentofacial deformities. J Dent Res 1984; 63: IADR abstract 1249.

36. Wolfson L, Tal H, Covo S. Peripheral giant cell granuloma during orthodontic treatment. Am J Orthop Dentofac Orthop 1989 Dec; 96(6): 519 - 23.

37. Newman GV, Goldman MJ, Newman RA. Mucogingival orthodontic and periodontal problems. Am J Orthod Dentofac Orthop 1994 Apr; 105(4): 321 - 7.

38. Stein G, Weinmann J. Die physiologische Wanderung der Zahne. Z. Stomatol 1925; 23: 733.

39. Björk A. Bite development and body build. Dent Rec 1955; 75: 8.

40. Graber LW, ed. In: Orthodontics, state of the art, essence of the science. St. Louis, 1986. The C. V. Mosby Company.

41. Ten Cate, AR. The role of fibroblasts in the remodeling of periodontal ligament during physiologic tooth movement. Am J Orthod 1976; 69: 155 - 68.

42. Bonucci, E. New knowledge on the origin, function and fate of osteoclasts. Clin Orthoped 1981; 158: 252 - 69.

43. Proffit WR. Equilibrium theory revisited: factors influencing position of the teeth. Angle Orthod 1978; 48: 175 - 86.

44. King GJ, Fischlschweiger W. The effect of force magnitude on extractable bone resorptive activity and cemental cratering in orthodontic tooth movement. J Dent Res 1982; 61: 775 - 9.

45. Burstone CJ, Groves MH. Threshold and optimum force values for maxillary anterior tooth movement. J Dent Res 1961; 39: 695.

46. Davidovitch Z. Tooth movement. Crit Rev Oral Biol Med 1991; 2(4): 411 - 50.

47. Davidovitch Z, Finkelson MD, Steigman S, Shanfeld JL, Montgomery PC, Korostoff E. Electric currents, bone remodeling, and orthodontic tooth movement. II. Increase in rate of tooth movement and periodontal cyclic nucleotide levels by combined force and electric current. Am J Orthod 1980 Jan; 77(1): 33 - 47.

48. Norton LA. Stress-generated potentials and bioelectric effects: their possible relationship to tooth movement. In: Norton LA, Burstone CJ, eds. The biology of tooth movement. Boca Raton, Florida, 1989, CRC Press, Inc.; pp. 349 - 57.

49. Reitan K. In: Orthodontics-Current Principles and Techniques, ed. Graber TM and Swain BF. St. Louis, 1985, The C. V. Mosby Company; p. 101.

50. Reitan K. Tissue rearrangement during retention of orthodontically rotated teeth. Angle Orthod 1959; 29: 105.

51. Selvig KA. An ultrastructural study of cementum formation. Acta Odontol Scand

1964; 22:105.

52. Sodek J. Collagen turnover in periodontal ligament. In: Norton LA, Burstone CJ, eds. The biology of tooth movement. Boca Raton, Florida, 1989, CRC Press, Inc.; pp. 157 - 81.

53. Fullmer HM. Observations on the development of oxytalan fibers in the periodontium of man. J Dent Res 1959; 38: 510.

54. Sims MR. Reconstitution of the human oxytalan system during orthodontic tooth movement. Am J Orthod 1976 Jul; 70(1): 38 - 58.

55. Simpson HE. A three-dimensional approach to the microscopy of the periodontal ligament. Proc R Soc Med 1967; 60: 537 - 42.

56. Sloan P. Structural organization of the fibres of the periodontal ligament. In: Berkovitz BKB, Moxham BJ, Newman HN., eds. The periodontal ligament in health and disease. Oxford, 1982, Pergamon Press; p. 68.

57. Edwards JG. A study of the periodontium during orthodontic rotation of teeth. Am J Orthod 1968; 68: 441 - 61.

58. Bowling K, Rygh P. A quantitative study of oxytalan fibres in the transseptal region and tension zones of rat molars following orthodontic movement. Eur J Orthod 1988; 10: 13 - 26.

59. Sicher H. Tooth eruption, the axial movement of continuously growing teeth. J Dent Res 1942; 21: 201.

60. Yee JA, Kimmel DB, Jee WS. Periodontal ligament cell kinetics following orthodontic tooth movement. Cell Tissue Kinet 1976 May; 9(3): 293 - 302.

61. Berkovitz BKB, Shore RC. Cells of the periodontal ligament. In: Berkovitz BKB, Moxham BJ, Newman HN., eds. The periodontal ligament in health and disease. Oxford, 1982, Pergamon Press; p. 25.

62. Roberts WE, Chase DC. Kinetics of cell proliferation and migration associated with orthodontically induced osteogenesis. J Dent Res 1981; 60: 174 - 81.

63. Ten Cate, AR. The role of fibroblasts in the remodeling of periodontal ligament during physiologic tooth movement. Am J Orthod Dentofac Orthop 1976; 69: 155 - 68.

64. Yee JA. Response of periodontal ligament cells to orthodontic force: ultrastructural identification of proliferating fibroblasts. Anat Rec 1979 Aug; 194(4): 603 - 14.

65. Nemeth E, McCulloch CA, Melcher AH. Coordinated regulation of endothelial and fibroblast cell proliferation and matrix synthesis in periodontal ligament adjacent to appositional and resorptive bone surfaces. Anat Rec 1989 Apr; 223(4): 368 - 75.

66. Ten Cate AR, Deporter DA. The degradative role of the fibroblast in the remodelling and turnover of collagen in soft connective tissue. Anat Rec 1975; 182: 1 - 14.

67. Sodek J. A new approach to assessing collagen turnover by using a microassay: a highly efficient and rapid turnover in rat periodontal tissues. Biochem J 1976; 160: 243 - 6.

68. Limeback HF, Sodek J, Brunette DM. Nature of collagens synthesized by monkey periodontal-ligament fibroblasts *in vitro*. Biochem J 1978; 170: 63 - 71.

69. Bellows CG, Melcher AH, Aubin JE. Association between tension and orientation of periodontal ligament fibroblasts and exogenous collagen fibers in collagen gels *in vitro*. J Cell Sci 1982; 58: 125 - 8.

70. Pitaru S, Melcher AH. Orientation of gingival fibroblasts and collagen fibers *in vitro*: resemblance to transseptal and dentogingival fibers. J Periodont Res 1983; 18: 483 - 500.

71. Gould TRL, Melcher AH, Brunette DM. Migration and diversion of progenitor cell populations in periodontal ligament after wounding. J Periodont Res 1981; 17: 70 - 9.

72. Reitan K. The initial tissue reaction incident to orthodontic tooth movement as related to the influence of function. Acta Odontol Scand 1951; (suppl. 6).

73. Davidovitch Z, Nicolay O, Alley K, Zwilling B, Lanese R, Shanfeld JL. First and second messenger interactions in stressed connective tissues in vivo. In: Norton LA, Burstone CJ, eds. The biology of tooth movement. Boca Raton, Florida, 1989, CRC Press, Inc.; pp. 97 - 129.

74. Saito I, Hanada K, Maeda T. Alteration of nerve growth factor-receptor expression in the periodontal ligament of the rat during experimental tooth movement. Arch Oral Biol 1993; 38(11): 923 - 9.

75. Kato J, Ichikawa H, Wakisaka S, Matsuo S, Sakuda M, Akai M. The distribution of

vasoactive intestinal polypeptides and calcitonin gene related peptide in the periodontal ligament of mouse molar teeth. Arch Oral Biol 1990; 35(1): 63 - 6.

76. Kimberly CL, Byers MR. Inflammation of rat molar pulp and periodontium causes increased calcitonin gene related peptide and axonal sprouting. Anat Rec 1988 Nov; 222(3): 289 - 300.

77. Kvinnsland I, Heyeraas KJ, Byers MR. Regeneration of calcitonin gene related peptide immunoreactive nerves in replanted rat molars and their supporting tissues. Arch Oral Biol 1991; 36(11): 815 - 26.

78. Byers MR, Taylor PE, Khayat BG, Kimberly CL. Effects of injury and inflammation on pulpal and periapical nerves. J Endod 1990 Feb; 16(2): 78 - 84.

79. Saito I, Ishii K, Hanada K, Sato O, Maeda T. Responses of calcitonin gene related peptide immunopositive nerve fibres in the periodontal ligament of rat molars to experimental tooth movement. Arch Oral Biol 1991; 36(9): 689 - 92.

80. Kvinnsland I, Kvinnsland S. Changes in CGRP immunoreactive nerve fibres during experimental tooth movement in rats. Eur J Orthod 1990 Aug; 12(3): 320 - 9.

81. Saitoh I, Ishii K, Kobayashi M, Hanada K, Maeda T, Sato O. An immunohistochemical study on the response of nerve fibers in the periodontium of rat molars during experimental tooth movement. Nippon Kyosei Shika Gakkai Zasshi 1990 Oct; 49(5): 466 - 74.

82. Silverman JD, Kruger L. An interpretation of dental innervation based upon the pattern of calcitonin gene related peptide (CGRP) immunoreactive thin sensory axons. Somatosens Res 1987; 5(2): 157 - 75.

83. Nicolay OF, Davidovitch Z, Shanfeld JL, Alley K. Substance P immunoreactivity in periodontal tissues during orthodontic tooth movement. Bone Miner 1990 Oct; 11(1): 19 - 29.

84. Gronblad M, Liesi P, Korkala O, Karaharju E, Polak J. Innervation of human bone periosteum by peptidergic nerves. Anat Rec 1984; 209: 297 - 9.

85. Freezer SR, Sims MR. Morphometry of neural structures in the mouse periodontal ligament mesial to the mandibular first molar. Aust Orthod J 1989 Mar; 11(1): 30 - 7.

86. Loescher AR, Holland GR. Distribution and morphological characteristics of axons

in the periodontal ligament of cat canine teeth and the changes observed after reinnervation. Anat Rec 1991 May; 230(1): 57 - 72.

87. Maeda T, Kannari K, Sato O, Iwanaga T. Nerve terminals in human periodontal ligament as demonstrated by immunohistochemistry for neurofilament protein (NFP) and S 100 protein. Arch Histol Cytol 1990 Jul; 53(3): 259 - 65.

88. Maeda T. Sensory innervation of the periodontal ligament in the incisor and molar of the monkey, Macaca fuscata. An immunohistochemical study for neurofilament protein and glia specific S 100 protein. Arch Histol Jpn 1987 Oct; 50(4): 437 - 54.

89. Maeda T, Sato O, Kobayashi S, Iwanaga T, Fujita T. The ultrastructure of Ruffini endings in the periodontal ligament of rat incisors with special reference to the terminal Schwann cells (K cells). Anat Rec 1989 Jan; 223(1): 95 - 103.

90. Sato O, Maeda T, Iwanaga T, Kobayashi S. Innervation of the incisors and periodontal ligament in several rodents: an immunohistochemical study of neurofilament protein and glia specific S 100 protein. Acta Anat Basel 1989; 134(2): 94 - 9.

91. Holland GR. The effect of pulpectomy on the longitudinal distribution of nerve fibres in the periodontal ligament of the ferret. Arch Oral Biol 1991; 36(2): 161 - 4.

92. Andreasen JO. Luxation of permanent teeth due to trauma: a clinical and radiographic follow-up study of 189 injured teeth. Scand J Dent Res 1970; 78: 273 - 86.

93. Jacobsen I, Kerekes K. Long-term prognosis of traumatized permanent anterior teeth showing calcifying processes in the pulp cavity. Scand J Dent Res 1977; 85: 588 - 98.

94. Anstendig HS, Kronman JH. A histologic study of pulpal reaction to orthodontic tooth movement in dogs. Angle Orthod 1972 Jan; 42(1): 50 - 5.

95. 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 Dentofac Orthop 1980 Apr; 77(4): 368 - 78.

96. Unsterseher RE, Nieberg LG, Weimer AD, Dyer JK. The response of human pulpal tissue after orthodontic force application. Am J Orthod Dentofac Orthop 1987 Sep; 92(3): 220 - 4.

97. Kvinnsland S, Heyeraas K, Ofjord ES. Effect of experimental tooth movement on periodontal and pulpal blood flow. Eur J Orthod 1989; 11: 200 - 5.

98. Stanley HR, Weisman MI, Michanowixa AE, Billizzi R. Ischemic infarction of the pulp: sequential degenerative changes of the pulp after traumatic injury. J Endod 1978; 4: 325 - 35.

99. Nixon CE, Saviano JA, King GJ, Keeling SD. Histomorphometric study of dental pulp during orthodontic tooth movement. J Endod 1993 Jan; 19(1): 13 - 16.

100. Guevara MH, McClugage SG. Effects of intrusive forces upon the microvasculature of the dental pulp. Angle Orthod 1980 Apr; 50(2): 129 - 34.

101. Mostafa YA, Iskander KG, el-Mangoury NH. Iatrogenic pulpal reactions to orthodontic extrusion. Am J Orthod Dentofac Orthop 1991 Jan; 99(1): 30 - 4.

102. Popp TW, Årtun J, Linge L. Pulpal response to orthodontic tooth movement in adolescents: a radiographic study. Am J Orthod Dentofac Orthop 1992 Mar; 101(3): 228 - 33.

103. Dougherty HL. The effect of mechanical forces upon the mandibular buccal segments during orthodontic treatment. Part I. Am J Orthod 1968; 54: 29 - 49.

104. Roberts WE, Ferguson DJ. Cell kinetics of the periodontal ligament. In: Norton LA, Burstone CJ, eds. The biology of tooth movement. Boca Raton, Florida, 1989, CRC Press, Inc.; pp. 55 - 69.

105. Baumrind S. A reconsideration of the propriety of the "pressure-tension" hypothesis. Am J Orthod 1969; 55: 12.

106. Giannelly AA. Force-induced changes in the vascularity of the periodontal ligament. Am J Orthod 1969; 55: 5 - 11.

107. Justus R, Luft J. A mechanochemical hypothesis for bone remodeling induced by mechanical stress. Calif Tissue Res 1970; 5: 222 - 35.

108. Fukada E, Yasada I. On the piezoelectric effect of bone. J Phys Soc Jap 1957; 12: 1158.

109. Bassett CA, Pawluk RJ, Becker RO. Effects of electric currents on bone *in vivo*. Nature 1964; 204: 652.

110. Roberts WE, Smith RK, Cohen JA. Change in electrical potential within periodontal ligament of a tooth subjected to osteogenic loading. Prog Clin Biol Res 1982; 101: 527 - 34.

111. Borgens RB. Endogenous ionic currents traverse intact and damaged bone. Science 1984; 225: 478.

112. Otter M, Shoenung J, Williams WS. Evidence for different sources of stress-generated potentials in wet and dry bone. J Orthoped Res 1985; 3: 321.

113. Macapanpan LC, Weinmann JP, Brodie AG. Early tissue changes following tooth movement in rats. Angle Orthod 1954; 24: 79.

114. Reitan K. Tissue reaction as related to the age factor. Dent Rec 1954; 74: 271.

115. Schwartz AM. Tissue changes incident to tooth movement. Int J Orthod 1932; 18: 331 - 52.

116. Schubert M, Hamerman D. A primer on connective tissue biochemistry. Philadelphia, 1968, Lea & Febiger.

117. Tuncay OC, Shapiro IM. A possible role for hypoxia in cellular energy metabolism of calcified tissue. In: Norton LA, Burstone CJ, eds. The biology of tooth movement. Boca Raton, Florida, 1989, CRC Press, Inc.; pp. 249 - 67.

118. Baron R. Mechanisms and regulation of osteoclastic bone resorption. In: Norton LA, Burstone CJ, eds. The biology of tooth movement. Boca Raton, Florida, 1989, CRC Press, Inc.; pp. 269 - 73.

119. Reitan K. Tissue behaviour during orthodontic tooth movement. Am J Orthod 1960; 46: 881.

120. Yamasaki K, Miura F, Suda T. Prostaglandin as a mediator of bone resorption induced by experimental tooth movement in rats. J Dent Res 1980 Oct; 59(10): 1635 - 42.

121. Yamasaki K. The role of cyclic AMP, calcium, and prostaglandins in the induction of osteoclastic bone resorption associated with experimental tooth movement. J Dent Res 1983 Aug; 62(8): 877 - 81.

122. Davidovitch Z, Shanfeld JL. Cyclic AMP levels in alveolar bone of orthodontically-treated cats. Arch Oral Biol 1975 Sept; 20(9): 567 - 74.

123. Takimoto K, Onishi K, Nakagawa H, Kawata T. Change in alkaline phosphatase activity in alveolar bone during the experimental tooth movement. Nippon Kyosei Shika Gakkai Zasshi 1965; 24(2): 182 - 6.

124. Takimoto K, Deguchi T, Mori M. Histochemical detection of succinic dehydrogenase in osteoclasts following experimental tooth movement. J Dent Res 1966 Sept - Oct; 45(5): 1473 - 6.

125. Bates S. Absorption. Br J Dent Sci 1856; 1: 256.

126. Schwarzkopf E. Resorption der Zahnwurzeln bei Regulierung. Dtsch Monatschr f Zhk 1887; 5: 180.

127. Ottolengui R. The physiological and pathological resorption of tooth roots. Item of Interest 1914; 36: 332 - 62.

128. Ketcham AH. A preliminary report of an investigation of apical root resorption of vital permanent teeth. Int J Orthod 1927; 13: 97 - 127.

129. Rudolph CE. A comparative study in root resorption in permanent teeth. J Am Dent Assoc 1936; 23: 822 - 6.

130. Kronfeld R. The resorption of the roots of deciduous teeth. Dental Cosmos 1932; 74: 103.

131. Massler M, Malone AJ. Root resorption in human permanent teeth. Am J Orthod 1954; 40: 619 - 33.

132. Henry JL, Weinmann JP. The pattern of resorption and repair of human cementum. J Am Dent Assoc 1951; 42: 270.

133. Becks H. Orthodontic prognosis: evaluation of routine dento-medical examinations to determine "good and poor risks". Am J Ortho and Oral Surg 1939; 25: 610.

134. Hemley S. The incidence of root resorption of vital permanent teeth. J Dent Res 1941; 20: 133 - 41.

135. Becks H. Root resorptions and their relation to pathologic bone formation. Int J Orthod 1936; 22: 445 - 82.

136. Becks H, Cowden RC. Root resorptions and their relation to pathologic bone formation. Part II: classification, degrees, prognosis and frequency. Am J Ortho and

Oral Surg 1942; 28: 513.

137. Brezniak N, Wasserstein A. Root resorption after orthodontic treatment: part 1. Literature review. Am J Orthod Dentofac Orthop 1993 Jan; 103(1): 62 - 6.

138. Goultschin J, Nitzan D, Azaz B. Root resorption. Oral Surg Oral Med Oral Pathol 1982; 54: 586 - 90.

139. Tangney NJ. Hypophosphatasia: a case report and literature review. J Irish Med Assoc 1979; 72: 530 - 1.

140. Smith NHH. Monostotic Paget's disease of the mandible, presenting with progressive resorption of the teeth. Oral Surg Oral Med Oral Pathol 1978; 46: 246 - 53.

141. Linge BO, Linge L. Apical root resorption in upper anterior teeth. Eur J Orthod 1983; 5: 173 - 83.

142. Marshall JA. A comparison of resorption of roots of deciduous teeth with the absorption of roots of the permanent teeth occurring as a result of infection. Int J Orthod 1929; 15: 417.

143. Goldie RS, King GJ. Root resorption and tooth movement in orthodontically treated, calcium-deficient, and lactating rats. Am J Orthod 1984; 85(5): 424 - 30.

144. Engström C, Granstöm G, Thilander B. Effect of orthodontic force on periodontal tissue metabolism. Am J Orthod Dentofac Orthop 1988; 93: 486 - 95.

145. Phillips JR. Apical root resorption under orthodontic therapy. Angle Orthod 1955; 25: 1 - 22.

146. Malmgren O, Goldson L, Hill C, Orwin A, Petrini L, Lundberg M. Root resorption after orthodontic treatment of traumatized teeth. Am J Orthod 1982; 82: 487 - 91.

147. Goldson L, Henrikson CO. Root resorption during Begg treatment. A longitudinal roentgenologic study. Am J Orthod 1975; 68: 55 - 66.

148. Levander E, Malmgren O. Evaluation of the risk of root resorption during orthodontic treatment: a study of upper incisors. Eur J Orthod 1988; 10: 30 - 8.

149. Newman WG. Possible etiologic factors in external root resorption. Am J Orthod 1975; 67: 522 - 39.

150. Oppenheim A. Human tissue response to orthodontic intervention of short and long duration. Am J Orthod 1942; 28: 263 - 301.

151. Kinsella P. Some aspects of root resorption in orthodontics. N Z Orthod J 1971: 21 - 5.

152. Rosenberg HN. An evaluation of the incidence and amount of apical root resorption and dilaceration occurring in orthodontically treated teeth, having incompletely formed roots at the beginning of Begg treatment. Am J Orthod 1972; 61: 524 - 5.

153. Massler M, Perreault JG. Root resorption in the permanent teeth of young adults. J Dent Child 1954; 21: 158 - 64.

154. McFadden WM, Engström C, Engström H, Anholm JM. A study of the relationship between incisor intrusion and root shortening. Am J Orthod Dentofac Orthop 1989;96: 390 - 6.

155. Oppenheim A. Biologic orthodontic therapy and reality: part III. Angle Orthod 1936; 6: 69 - 116.

156. Sjolien T, Zachrisson BU. Periodontal bone support and tooth length in orthodontically treated and untreated persons. Am J Orthod 1973; 64: 28 - 37.

157. DeShields RW. A study of root resorption in treated Class II Division 1 malocclusion. Angle Orthod 1969; 39: 231 - 45.

158. Sharpe W, Reed B, Subtelny JD, Polson A. Orthodontic relapse, apical root resorption, and crestal alveolar bone levels. Am J Orthod Dentofac Orthop 1987; 91(3): 252 - 8.

159. Ten Hoeve A, Mulie MR. The effect of anteroposterior incisor repositioning on the palatal cortex as studied with laminography. J Clin Orthod 1976; 10: 804 - 22.

160. Remmelnick HJ. The effect of anteroposterior incisor repositioning on the root and cortical plate: a follow-up study. J Clin Orthod 1984; 18: 42 - 9.

161. Reitan K. Initial tissue behavior during apical root resorption. Angle Orthod 1974; 44: 68 - 82.

162. Goldin B. Labial root torque: effect on the maxilla and incisor root apex. Am J Orthod Dentofac Orthop 1989; 95: 208 - 19.

163. Wainwright WM. Faciolingual tooth movement: its influence on the root and cortical plate. Am J Orthod 1973; 64: 278 - 302.

164. Harry MR, Sims MR. Root resorption in bicuspid intrusion: a scanning electromicroscopic study. Angle Orthod 1982; 52: 235 - 58.

165. Stuteville OH. Injuries of the teeth and supporting structures caused by various orthodontic appliances, and methods of preventing these injuries. J Am Dent Assoc 1937; 14: 1494 - 507.

166. Dermaut LR, De Munck A. Apical root resorption of upper incisors caused by intrusive tooth movement: a radiographic study. Am J Orthod Dentofac Orthop 1986; 90: 321 - 6.

167. Kawata T. A new orthodontic force system of magnetic brackets. Am J Orthod Dentofac Orthop 1987; 92: 241 - 8.

168. Blechman AM, Smiley DH. Magnetic force in orthodontics. Am J Orthod 1978; 74: 435 - 43.

169. Dougherty HL. The effect of mechanical forces upon the mandibular buccal segments during orthodontic treatment. Part II. Am J Orthod 1968; 54: 83 - 103.

170. VonderAhe G. Postretention status of maxillary incisors with root-end resorption. Angle Orthod 1973; 43: 247 - 55.

171. Rygh P. Orthodontic root resorption studied by electron microscopy. Angle Orthod 1977; 47: 1 - 16.

172. Copeland S, Green LJ. Root resorption in maxillary central incisors, following active orthodontic treatment. Am J Orthod 1986; 89: 51 - 5.

173. Gholston L, Mattison G. An endodontic-orthodontic technique for esthetic stabilization of externally resorbed teeth. Am J Orthod 1983; 83: 435 - 40.

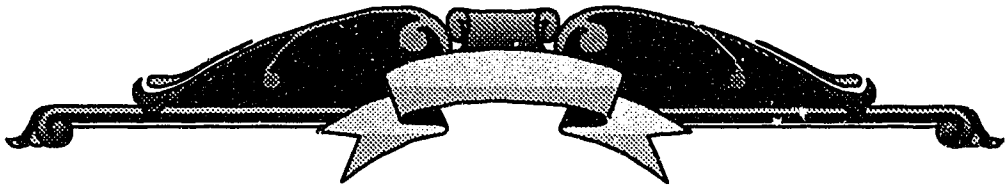
174. Baranowskyj GR. A histologic investigation of tissue response to an orthodontic intrusive force on a dog maxillary incisor with endodontic treatment and root resection. Am J Orthod 1969; 56: 623 - 4.

175. Huettner RJ, Young RW. The movability of vital and devitalized teeth in the Macacus rhesus monkey. Am J Orthod 1955; 41: 594 - 603.

176. Kaffe I, Tomas A, Littner MM, Schwartz I. A radiographic survey of apical root resorption in pulpless permanent teeth. Oral Surg 1984; 58: 109 - 12.

177. Mattison GD, Delivanis HP, Delivanis PD, Johns PI. Orthodontic root resorption of vital and endodontically treated teeth. J Endod 1984 Aug; 10(8): 354 - 8.

178. Satoh I. Root resorption of vital and endodontically treated teeth in orthodontic movement. Kanagawa Shigaku 1990 Mar; 24(4): 601 - 17.



Chapter Two

Research Study And Results

2.1 - INTRODUCTION

Orthodontic movement of endodontically treated teeth has become more common due to the increased restoration of pulpally involved teeth and elevated demand for adult orthodontics. However, as Wickwire *et al.* stated, "there is a paucity of documented information concerning the prognosis of the endodontically treated tooth undergoing orthodontic tooth movement" [1]. While it is clearly possible to move non-vital teeth, some doubt remains as to how easily this is achieved and what degree of root resorption it induces.

In most cases, root resorption is part of a normal physiologic process of cemental remodelling and repair, albeit an unpredictable one [2]. It is widely accepted that orthodontic treatment increases the likelihood and severity of root resorption in all teeth, with certain teeth being more susceptible than others [3,4]. Many variables have been implicated as predisposing factors to root resorption, including individual susceptibility, genetic influences, endocrine imbalances, dietary changes, anatomic factors, and mechanotherapy techniques [5]. However, the extent to which each of these factors is involved in the resorptive process is controversial, with different studies finding varying degrees of influence [5,6]. Prolonged treatment, use of heavy forces, and unfavourably shaped roots are implicated in increased root resorption associated with orthodontic treatment [5,7]. These are well recognized risks which are avoided, as much as possible, in clinical orthodontics.

Previous studies are inconclusive as to the response of endodontically treated teeth to orthodontic forces [1,5,8]. Most authors agree that there is little difference in the movability of these teeth relative to their vital counterparts [1,7,8]. The exception to this is the previously

traumatized tooth with root canal therapy, which may be more susceptible to ankylosis, especially in cases of complete avulsion [9]. Further, there is conflicting evidence as to whether non-vital teeth are more or less likely to experience root resorption with orthodontic force. Wickwire *et al.* found that endodontically treated teeth were subject to greater root resorption than vital teeth during orthodontics [1]. This supported the traditional view of Steadman, who suggested that endodontically treated teeth were likely to "melt away" as they would be regarded as foreign objects by the body [10].

In a radiographic study, Spurrier *et al.* found less susceptibility to root resorption in endodontically treated teeth than in vital ones [8]. Huettner *et al.* found no histological differences between the cementum in orthodontically treated vital and non-vital teeth [11]. With experimental orthodontic movement on feline teeth, Mattison *et al.* confirmed this histologic finding, with no significant difference between external root resorption of endodontically treated and vital teeth [12].

These apparently contradictory findings in the existing literature leave considerable uncertainty as to the susceptibility of endodontically treated teeth to root resorption. The study reported here was designed to determine whether non-vital teeth move as readily as vital teeth when subjected to comparable forces and secondly, whether orthodontically moved, root filled teeth showed more apical root resorption than vital teeth.

2.2 - MATERIALS AND METHODS

2.2.1 - Animals

Twelve young male ferrets (*Mustela furo*), previously descented and castrated, were used in this study. They ranged in age from 3 to 4 months and in weight from 1.20 to 1.92 kg. Six ferrets were randomly selected to receive active orthodontic appliances while the other six had inactive appliances placed.

2.2.2 - Animal Preparation

Under general anaesthesia (ketamine hydrochloride, 100 mg/mL, and xylazine, 20 mg/mL, IM) each ferret was injected with 80 mg/kg body weight of vital fluorescent stain (Procion brilliant red H8B-chloro-s-triazine compound, Polysciences Inc., Warrington, PA). An ophthalmic ointment (Lacri-Lube®, Allergan Inc., Markham, Ontario, Canada) was applied as needed to prevent desiccation of the eyes. Following every general anaesthetic procedure, each ferret received a single dose of antibiotic (ethacillin, 1 mL, IM) and analgesic (buprenorphine, 0.3 mg/mL, IM).

One week later, under general anaesthetic, each ferret had one randomly chosen mandibular cuspid treated endodontically. Access was obtained using a #557 carbide bur on a high speed handpiece. The pulp was extirpated with a barbed broach and the canal was instrumented using files (K-flex® endodontic files, Kerr® Brand, Romulus, MI) and a # 2 Gates Glidden drill. Obturation was completed using lateral condensation with gutta percha and endodontic sealer (Sealapex®, Kerr® Brand). The access opening was then sealed with

glass ionomer restorative material (Ketac-Silver®, ESPE Premier Sales Corp., Norristown PA).

In the same surgery, all mandibular premolar teeth were extracted bilaterally, as were the maxillary cuspids, to avoid interferences with the orthodontic appliance and facilitate tooth movement of the experimental teeth. At this stage, a polysiloxane impression (Coltoflax®, Coltène®/Whaledent, New York NY) of the mandibular arch was taken to record the initial position of the cuspids.

An orthodontic appliance was then placed bilaterally on the mandibular teeth, consisting of custom pinched 0.005" thick stainless steel bands (Permachrome, Unitek®, Monrovia CA) with a soldered loop made of 0.020" stainless steel round wire (Fig. II-1). These were cemented to the mandibular cuspids with a dual cure glass ionomer orthodontic band cement (Band-Lok™, Reliance Orthodontic Products, Inc., Itasca IL) and light cured for 60 seconds. The band was placed so that the loop faced distally, [illegible] distance between the cuspid attachment and its ipsilateral molar. At this point, a small bi-cortical hole was made in the mandible, through the furcation of the first molars bilaterally, passing from the buccal to the lingual. A ligature wire (0.009" stainless steel) was passed through this hole and used to attach the distal eyelet of a closed coil nickel titanium spring (Sentalloy™, GAC International Inc., Central Islip, NY). A ligature wire was used to attach the mesial eyelet of the spring to the band loop. Depending on whether the subject was to have inactive or active appliances, springs were either left at their initial rest length of 3 mm, giving 0 g of force, or activated to 15 mm, which gave 150 - 175 g of force assessed using a stress and tension

gauge (28 - 450 g, Dentaurum, Fed. Rep. Germany).

One ferret died shortly after the first general anaesthetic due to pulmonary failure and was replaced. Each animal was kept under close observation until recovered from the anaesthetic and monitored on a daily basis thereafter. For several days after the preparation, the animals were offered a soft diet as well as standard laboratory chow. Except for the animal mentioned above, all animals recovered well. The average weight before preparation was 1.50 kg (± 0.18). Some appliances became detached and were repaired immediately to minimize variation in application of orthodontic forces.

Twelve weeks after appliance placement, under general anaesthesia (as above, plus 40 mg/kg sodium pentobarbitone IP), each animal was perfused via the descending aorta with a physiologic saline prewash (250 mL) followed by 4 % paraformaldehyde (500 mL). The average weight taken prior to perfusion was 1.38 kg (± 0.14). The mandibles were removed, appliances detached, and polysiloxane impressions taken to record the new positions of the teeth. The two sides of the mandible were separated and radiographed. The mandibular halves were then decalcified, frozen, and stored at -70° C. Serial sections of the mandibular canine apices and surrounding tissue were made at 16 µm intervals. Using the pulp canal space as a guide, the most central section was stained with hematoxylin and eosin. Adjacent sections were left unstained to allow examination under fluorescence microscopy.

2.2.3 - Measurements

There were four groups of teeth. One consisted of vital teeth with inactive appliances;

the second, also vital, had active appliances. A third group contained endodontically treated, non-vital teeth with inactive appliances while the fourth had non-vital teeth with active appliances. Since there were twelve subjects in all, each with two experimental teeth, twenty-four teeth were assessed, with six in each group.

Tooth movement - casts

Tooth movement was measured from casts of the initial and post-mortem impressions. The distance between each mandibular cuspid and the ipsilateral molar was measured at the gingival margin with a digital caliper (Mitutoyo Digimatic CD-6", Mitutoyo Corporation, Japan). By subtracting the final distance from the initial distance, the amount of tooth movement was determined. Most of the tooth movement expected was distal tipping of the cuspid. As the centre of resistance is closer to the tooth apex, any tipping movement would be expected to register significantly close to the tooth crown. Comparisons between movement of vital and endodontically treated cuspids were then performed from these figures.

Tooth movement - histologic

Bone deposition and hence tooth movement was also assessed on the unstained sections using fluorescence microscopy (Zeiss Photomicroscope III, Carl Zeiss Inc., Thornwood NY). The Procion dye fluoresced when observed with the appropriate filter (BG 38 red-attenuation filter, Carl Zeiss Inc.) under mercury lighting (Fig. II-2). The Procion dye was incorporated into new bone at the beginning of the experimental period and it was thus

possible to determine the extent of bone deposition during this time by measuring on the tension side the distance from the fluorescent line to the edge of the PDL. This line was traced via a camera lucida, measured at three points within the middle third of the root, and averaged. This could only be appraised on the tension side of the PDL as bone was resorbed on the pressure side.

Root resorption - radiographic

The degree of root resorption was assessed both histologically and radiographically. Measurements of root length were taken from the radiographs using a digital caliper, from cemento-enamel junction (CEJ) to apex. By comparing the post-treatment root lengths of vital and nonvital teeth, the comparative extent of root shortening was determined. This was based on the assumption that left and right cuspids were of equal pre-treatment length.

Root resorption - histologic (root length)

Histologically, root lengths were evaluated on the stained hematoxylin and eosin sections by measuring the length of the root from apex to the crest of the alveolar bone (Fig. II-3). Using the measurements from vital and nonvital teeth, both inactive and active, the difference in root loss between the four groups was determined. Linear measurements under the light microscope were determined using calibrated markings in the eyepiece, taking into account the magnification factor. The actual length was calculated using a standardized ruler.

Root resorption - histologic (resorption lacunae)

From each stained section passing through the centre of the root canal the number of resorption lacunae in the cementum of each root was determined (Fig. II-4).

Root resorption - histologic (cementum resorption)

Through remodelling, Procion dye was incorporated into the cementum. A fluorescent line could be seen under the microscope using the appropriate filters and mercury lighting, demarcating the initial position of the cementum. Where cementum was resorbed, this line was lost. In an unstained central section of each cuspid, the length of the root surface not exhibiting fluorescence was traced via a camera lucida and measured using a digitizing pad (Summasketch® Plus, Summagraphics® Corporation, Fairfield CT) and morphometric software (Bioquant II, R&M Biometrics Inc., Nashville TN) (Fig. II-2).

Periapical Lesion Size

Most of the root filled teeth in this study exhibited some degree of periapical inflammation (Fig. II-5). The area of each periapical lesion was measured to determine whether the size of the lesion was affected by orthodontic tooth movement. Lesions were traced via a camera lucida under light microscopy (x37), then measured using a digitizing pad and morphometric software. This allowed comparisons of lesion size between endodontically treated teeth with active and inactive appliances.

2.3 - RESULTS

All data are summarized in Table II - 1. Four comparisons were made: i) vital inactive teeth vs. vital active teeth, ii) non-vital inactive teeth vs. non-vital active teeth, iii) vital inactive teeth vs. non-vital inactive teeth, and i) vital active teeth vs. non-vital active teeth. All comparisons were made using the Student t-test for paired and independent groups, with correction for small sample size (SPSS® 6.1 for Windows™, SPSS® Inc., Chicago IL).

Tooth movement - casts

The reduction in canine to molar distance was greater in the vital active group than in the vital inactive group ($p < 0.05$) and also greater in the non-vital active group than in the non-vital inactive ($p < 0.05$). There was no statistically significant difference ($p > 0.05$) between vital inactive versus non-vital inactive teeth nor between vital active and non-vital active teeth.

Tooth movement - histologic - (Fig. II-2)

Teeth loaded with active appliances showed greater bone deposition and thus greater movement than those with inactive appliances in the vital group ($p < 0.05$). In comparing non-vital active to non-vital inactive, no significant differences ($p > 0.05$) were found. The same was true between the vital inactive vs. non-vital inactive teeth and between the vital active vs. the non-vital active teeth.

Root resorption - radiographic

Statistically significant differences ($p < 0.05$) were found only between the root

lengths of non-vital active and non-vital inactive teeth, with root shortening more prominent on the active teeth. The remaining comparisons between vital active vs. vital inactive, non-vital active vs. vital active, and non-vital inactive vs. vital inactive, showed no statistically significant difference ($p > 0.05$).

Root resorption - histologic (root length / resorption lacunae / cementum resorption) - (Fig. II-2, II-3, II-4)

Comparisons of histologically determined root length showed no statistically significant ($p > 0.05$) differences between any of the four groups. A statistically significant difference ($p < 0.05$) in the number of resorption lacunae was found between vital inactive teeth and non-vital inactive teeth. The non-vital group showed greater incidences of resorption bays. Linear measurement of cemental resorption showed statistically significant differences between non-vital inactive and non-vital active teeth as well as between vital active and non-vital active teeth ($p < 0.05$). In both cases, the non-vital active group showed the greatest degree of cemental resorption.

Periapical Lesion Size - (Fig. II-5)

No lesions were found in any of the vital teeth, while most of the non-vital teeth exhibited some degree of periapical inflammatory response. There was no significant difference ($p > 0.05$) in lesion size between non-vital inactive teeth and non-vital active teeth.

2.4 - DISCUSSION AND CONCLUSION

The orthodontic springs used in this study induced significant tooth movement ($p < 0.05$). There was no significant difference ($p > 0.05$) in tooth movement between vital and non-vital teeth subjected to the same force whether the tooth movement was measured by the comparison of pre- and post-treatment casts or by the amount of bone deposition. This supports the clinical observation that endodontically treated teeth move as readily as vital teeth with orthodontic therapy. It also agrees with existing literature [1,7,8]. Radiographic evaluation of tooth movement in past studies has usually involved root filled teeth that were previously traumatized, which may have influenced their results [1,3,8]. This factor was eliminated in this study, leaving endodontic treatment as the only variable.

Huettner and Young reported similar findings in monkeys, with "no difference between orthodontically moved vital and devitalized teeth in their gross and histologic aspects, provided that the orthodontic forces are carefully regulated and that the root canals have been sterilized, endodontically treated, and the periodontal membrane kept intact" [11]. This indicates that denervation of the pulp has little effect on the response of the periodontal tissue to orthodontic forces. It appears that "the pulp and the attachment apparatus are two separate entities, thus when the pulp of a tooth is effectively removed and the root canal space properly sealed, the periodontal ligament remains unaffected" [12].

Root resorption was assessed both radiographically and histologically. Gross radiographic examination showed only root shortening of active non-vital teeth compared to inactive non-vital teeth. Comparisons between vital and non-vital groups, whether active or

inactive, showed no significant difference. Radiographic measurements of root resorption are of low resolution and small changes may not be detected. The same conclusion was made by Mattison *et al.* in a study on movement of feline cuspids, finding that the "forces of movement are the factor responsible for root resorption and not the vitality or lack of vitality of the tooth" [12]. This differs from the findings of recent studies by Spurrier *et al.* and Satoh, who found that vital teeth resorbed earlier and more than endodontically treated ones [8,13]. Remington *et al.* also noted there was a decreased amount of resorption observed in root filled teeth, speculating that increased dentin density in these teeth may provide some resistance to root resorption [2,14]. The findings of the present study also contrast the opinion of Steadman, who suggested that orthodontic movement of endodontically treated teeth would result in their "melting away" due to excessive resorption, unlike their vital counterparts [10]. Wickwire *et al.* also concluded that "there appeared to be a greater frequency of root resorption in the endodontically treated teeth as compared with the control group" [1]. The non-vital teeth they examined were previously traumatized, increasing their susceptibility to resorption. Remington *et al.* criticized these findings also, stating that Wickwire *et al.* may have biased findings by including teeth with unsuccessful endodontics [1,14]. The greater resorption of endodontically treated teeth may be related more to the presence of periapical inflammation than to the lack of vitality. Another explanation suggested by some authors is that orthodontic treatment initiated too early after endodontics may cause increased resorption, thus a postponement of orthodontic therapy for some time after root canal therapy is recommended [12,15]. The present study did not address this question.

No statistically significant differences ($p > 0.05$) in root length were detected histologically between any of the groups. Although apparently of greater resolution than radiographic measurements, this approach is subject to a number of variables which may mask differences. The apex of the crestal bone was used as a fixed point, but its location may, in fact, vary. Variation in the angulation of the plane of section would also result in differences in measured root length not related to resorption.

Non-vital teeth showed more resorption lacunae than vital teeth but the small difference in incidence between non-vital active and non-vital inactive teeth was not statistically significant ($p > 0.05$). This suggests that the incidence of resorption lacunae is related to non-vitality and probably the presence of periapical lesions rather than to orthodontic forces. A similar finding was reported by Mattison *et al.* on orthodontic movement of vital and non-vital feline cuspids, concluding there was "no significant difference between external root resorption of endodontically treated and vital teeth when both were subjected to orthodontic forces" [12].

Cemental resorption is greater in teeth undergoing orthodontic tooth movement than those that are not, but the difference is only statistically significant when the teeth are non-vital ($p < 0.05$). This is a much more sensitive technique for the measurement of resorption than either the radiographic or histological measurements of root length.

Most of the non-vital teeth showed some degree of periapical inflammation. But there was no significant difference between inactive non-vital and active non-vital teeth. These findings suggest that the endodontic treatment of the teeth resulted in periapical lesions, but

that the size of the lesions is not affected by orthodontic movement. This contrasts a study by Baranowskyj which found that the healing process after endodontics was delayed if orthodontic force was applied too soon [15].

Under the conditions of this study, the conclusion is that non-vital teeth move as readily as vital teeth when subjected to orthodontic forces. Root filled teeth show more apical resorption than vital teeth after tooth movement, though the difference is small and probably related to the presence of periapical inflammation.

Variable	Measurement	Inactive Vital	Active Vital	Inactive Non-vital	Active Non-vital
Tooth Movement	Casts mm (S.D.)	**1.09** (± 0.91)	**2.76** (± 1.17)	**0.48** (± 0.94)	**2.00** (± 1.14)
	Bone Deposition µm (S.D.)	**140.67** (± 52.55)	**254.17** (± 108.97)	**182.17** (± 96.22)	**236.80** (± 141.70)
Root Resorption	Root Length-radiograph mm (S.D.)	**10.95** (± 0.37)	**10.54** (± 0.34)	**11.04** (± 0.26)	**10.44** (± 0.51)
	Root Length-histologic mm (S.D.)	**9.68** (± 1.35)	**8.75** (± 2.33)	**9.65** (± 1.07)	**9.29** (± 0.93)
	Resorption Lacunae # (S.D.)	**0**	**0.20** (± 0.45)	**3.50** (± 3.51)	**3.83** (± 4.92)
	Cemental Resorption µm (S.D.)	**596.50** (± 924.40)	**995.80** (± 1592.53)	**766.00** (± 579.13)	**3189.00** (± 1473.61)
Lesion Size	Area mm^2 (S.D.)	**0**	**0**	**5.15** (± 3.82)	**5.83** (± 5.66)

Table II-1: Summary of Variable Measurements

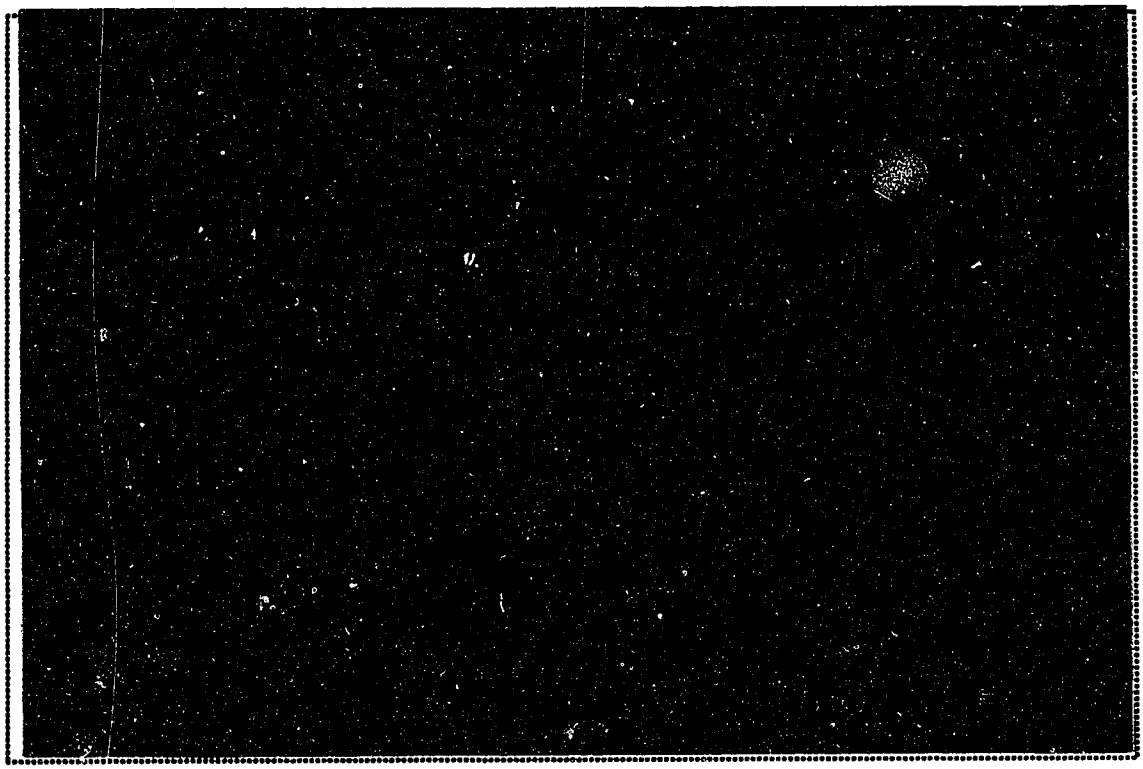


Figure II - 1. Orthodontic appliance in place. (B = band on canine, S = nickel titanium orthodontic spring, L = steel ligature)

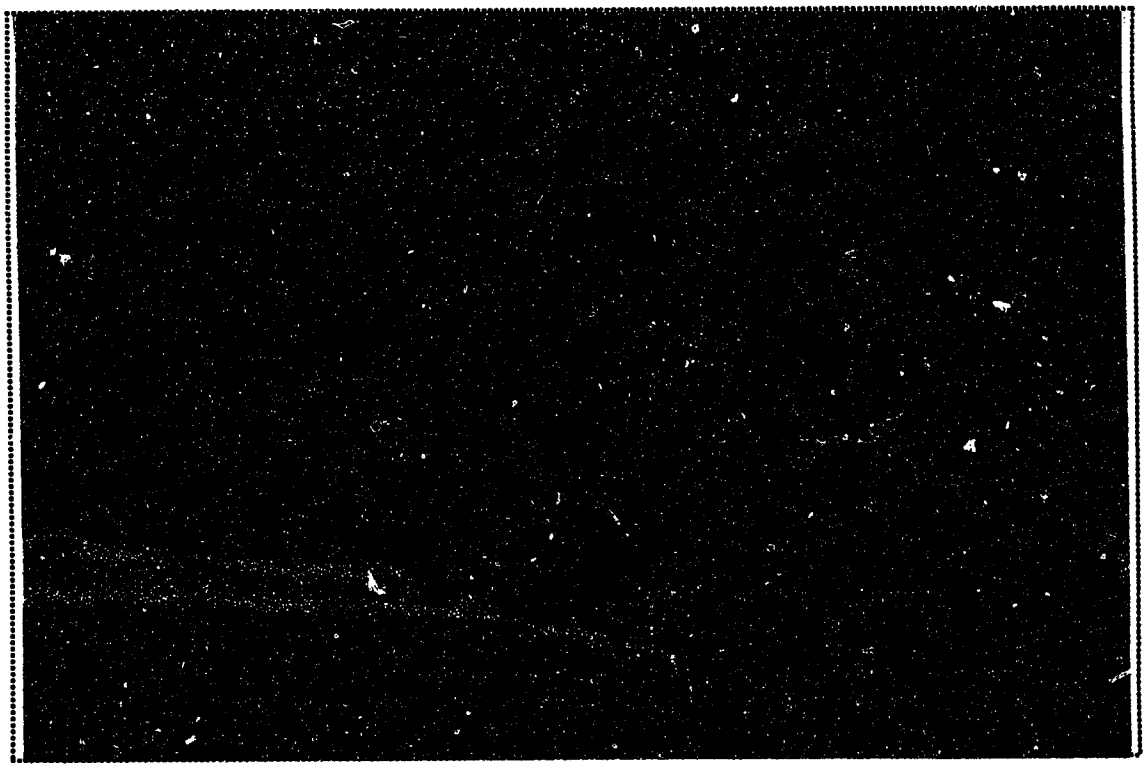


Figure II - 2. Fluorescent micrograph of mid-root region of tooth subjected to orthodontic forces. Orange fluorescence denotes deposition of Procion dye. (B = bone, C = cementum) (x63 magnification)

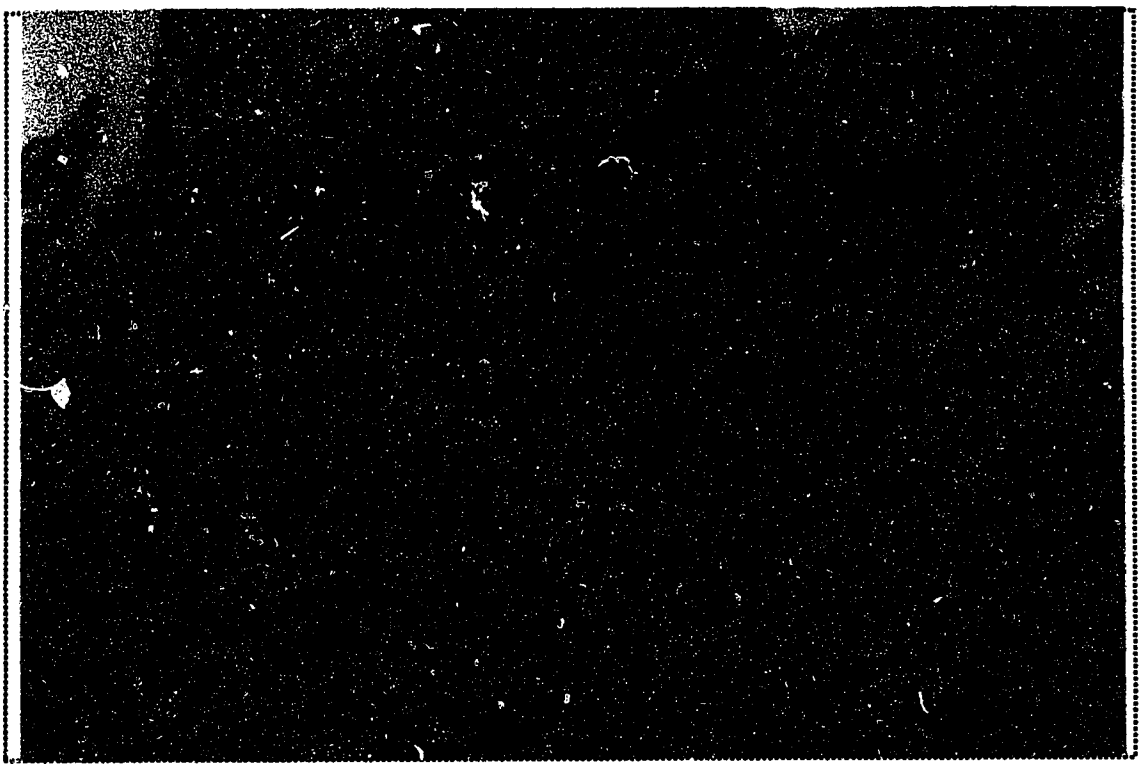


Figure II - 3. Root length assessed histologically. (C = crestal bone, A = root apex) (x5 magnification)

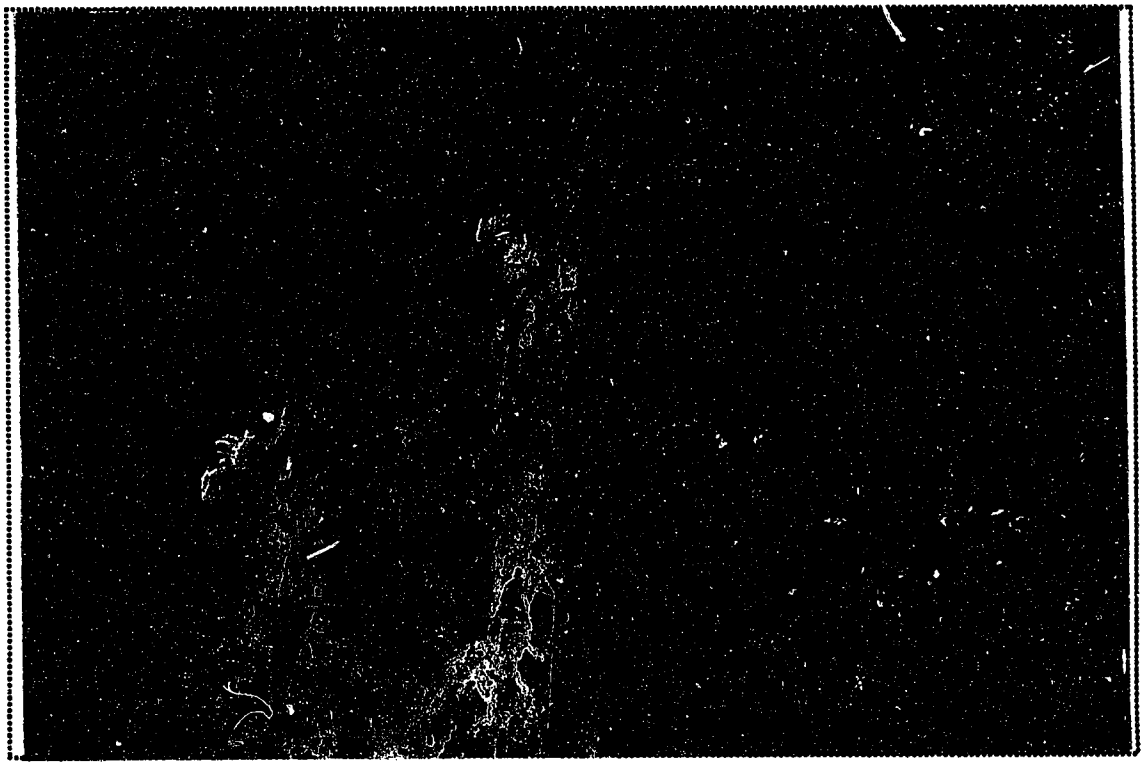


Figure II - 4. Apical resorption lacunae from a non-vital tooth subjected to orthodontic forces. (Arrows) (x25 magnification)

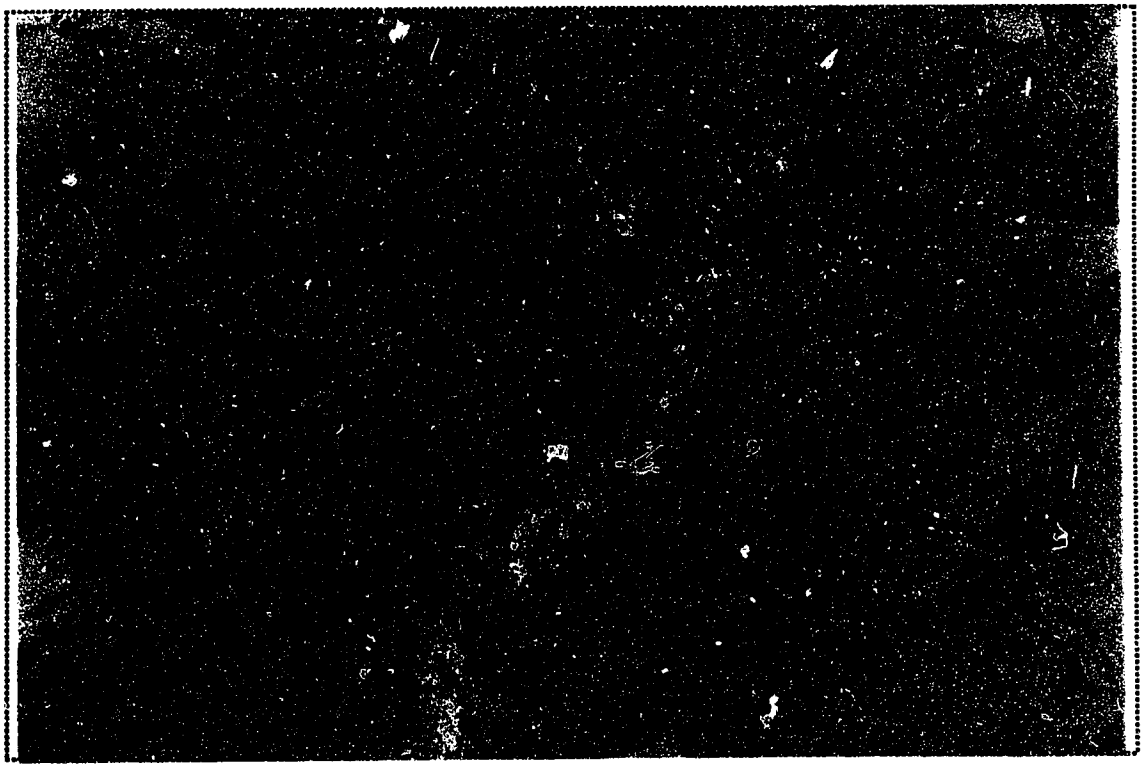


Figure II - 5. Periapical inflammation around a non-vital tooth subjected to orthodontic forces. (x10 magnification)

REFERENCES

1. Wickwire NA, McNeil MH, Norton LA, Duell RC. The effects of tooth movement upon endodontically treated teeth. Angle Orthod 1974; 44: 235 - 42.

2. Reitan K. Biomechanical principles and reactions. In: Graber TM, Swain BF. Orthodontics-current principles and techniques. St. Louis, CV Mosby, 1985: 101-92.

3. Massler M, Malone AJ. Root resorption in human permanent teeth. Am J Orthod 1954; 40: 619-33.

4. Hemley S. The incidence of root resorption of vital permanent teeth. J Dent Res 1941; 20: 133-41.

5. Brezniak N, Wasserstein A. Root resorption after orthodontic treatment: part 1. Literature review. Am J Orthod Dentofac Orthop 1993 Jan; 103(1): 62-6.

6. Newman WG. Possible etiologic factors in external root resorption. Am J Orthod 1975; 67: 522 - 39.

7. Proffit WR.: The biological basis of orthodontic therapy. In: Contemporary orthodontics, second edition. St. Louis, 1993, Mosby - Year Book, Inc.; pp. 266 - 88.

8. Spurrier SW, Hall SH, Joondeph DR, Shapiro PA, Riedel RA. A comparison of apical root resorption during orthodontic treatment in endodontically treated and vital teeth. Am J Orthod Dentofac Orthop 1990 Feb; 97(2): 130-4.

9. Andreasen JO. Traumatic injuries of the teeth. St. Louis, 1972, The C. V. Mosby Company, ch. 7: 211.

10. Steadman SR. Résumé of the literature on root resorption. Angle Orthod 1942; 12: 29 - 36.

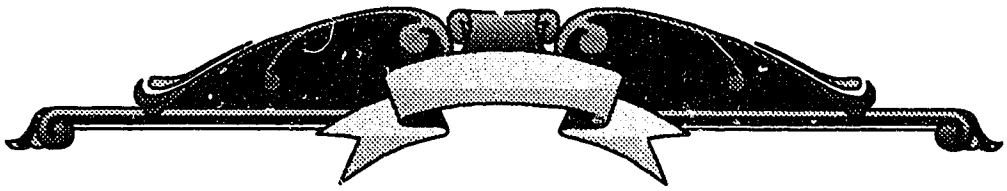
11. Huettner RJ, Young RW. The movability of vital and devitalized teeth in the Macacus rhesus monkey. Am J Orthod 1955; 41: 594 - 603.

12. Mattison GD, Delivanis HP, Delivanis PD, Johns PI. Orthodontic root resorption of vital and endodontically treated teeth. J Endod 1984 Aug; 10(8): 354 - 8.

13. Satoh I. Root resorption of vital and endodontically treated teeth in orthodontic movement. Kanagawa Shigaku 1990 Mar; 24(4): 601 - 17.

14. Remington DN, Joondeph DR, Årtun J, Riedel RA, Chapko MK. Long-term evaluation of root resorption occurring during orthodontic treatment. Am J Orthod Dentofac Orthop 1989 July; 96(1): 43 - 6.

15. Baranowskyj GR. A histologic investigation of tissue response to an orthodontic intrusive force on a dog maxillary incisor with endodontic treatment and root resection. Am J Orthod 1969; 56: 623 - 4.



Chapter Three

Discussion

And

Recommendations

3.1 - GENERAL DISCUSSION

Orthodontic movement of endodontically treated teeth has become a fairly routine procedure in clinical practice today. This movement seems to pose little difficulty clinically, with the exception of possible ankylosis of previously traumatized teeth, thus research into this area has been limited. The few studies that have looked specifically at this area have mostly been retrospective radiographic studies with general conclusions made on few observations. Many criticisms can be made about the studies of the past concerning their questionable methodology, inaccurate measuring techniques, and retrospective nature. Despite this, these studies constitute the bulk of knowledge in this area. Wickwire *et al.* published their study over two decades ago, yet it remains the basis for the beliefs of many clinicians today [1]. Until definitive research is conducted and verified, the findings of past studies will continue to guide our treatment. This study has further investigated this area, attempting to clarify this controversial topic.

The orthodontic springs used in this study induced significant tooth movement ($p < 0.05$). This was determined by comparing the active groups, both vital and non-vital, to their respective inactive counterparts. As expected, the active springs caused significantly more tooth movement than inactive ones when tooth movement was measured by the comparison of pre- and post-treatment casts ($p < 0.05$) (Appendices 9, 10). More importantly, there was no significant difference ($p > 0.05$) in tooth movement between vital and non-vital teeth subjected to the same force. This appears to support the clinical observation that endodontically treated teeth move as readily as vital teeth with orthodontic therapy.

However, with such a small sample size and large standard deviations, this finding should be regarded cautiously to avoid a type II statistical error whereby an insignificant finding at this level may prove to be significant in a larger study.

Despite this, several sources of error exist in the methodology. Firstly, it is difficult to determine exactly how much of this movement was due to movement of the cuspids and how much was due to movement of first mandibular molars. In order to minimize the mesial movement of the molars, the distal ligature of the nickel titanium springs was attached intra-osseously in the first molar furcation area, as this was thought to minimize movement of the molar. Undoubtedly, there was still some movement of the molar, however, this was thought to be minimal. A second source of error may have been caused by the interruption of force application at various times throughout the experimental period. As the ferrets ate, they occasionally dislodged the bands on the cuspids or broke the ligature wires. In either case, the force of the orthodontic spring was eliminated until the appliance could be repaired, which was usually the next day. During this inactive period, the potential for error was introduced as forces were no longer uniform within a given group. Again, these errors were thought to be minimal as the appliances were monitored closely and repaired as quickly as possible when needed. A third source of error may have been in the tooth movement itself. From the mechanics of the appliance placed, distal tipping of the cuspid would be expected rather than bodily movement. Depending on the root size and shape, bone support, force of occlusion, etc., each tooth would move slightly differently even under identical circumstances. Thus, although randomly distributed in one of four groups, individual teeth would already be

predisposed to differences based on individual variation. A fourth, and possibly most significant source of error, may have been introduced by the presence of periapical lesions around most of the non-vital teeth. These represent an inflammatory response of the periodontal tissues to the insult of the root canal treatment. Presumably, with the presence of these lesions in the periapical region, bone density would be decreased around these teeth apices, allowing tooth movement to occur more easily. However, as will be discussed later, these periapical lesions were evenly distributed between active and inactive groups, thus their error should have been equal in both groups. From the data, it is impossible to determine whether or not a difference in quantity of tooth movement would have occurred between vital active and non-vital active groups had the periapical inflammation not been present. Further studies will be needed to determine this. A fifth source of error may have been an inadequate time frame for any differences to become evident. For practical purposes, assuming that teeth generally move about 1 mm per month in humans, we set a 3 month experimental period. Judging from the small mean amounts of movement with relatively large standard deviations, perhaps a longer experimental period would have shown some difference in tooth movement not detectable in the short duration of this study (Appendices 5-8). Further error may have resulted from the fact that tooth movement comparisons were made from pre- and post-treatment casts, which were subject to distortion and land marking errors in determining the CEJ of each tooth. Other minor sources of error include the inaccuracy of the digital calipers, intra-operator measurement error, differences in spring activation, and individual variation in the ferrets.

A second method used to assess tooth movement was through the examination of bone deposition on the mesial side following the experimental period. Procion dye that was injected one week prior to the start of the experimental period fluoresced under mercury light, using the appropriate filter. The resultant fluorescent line demarcated the initial uptake of the dye; bone seen between the tooth and this line was deposited during the experimental period. In the mid-root area, where the fluorescence was clearest, a linear measurement was made from the tooth to the line, representative of the amount of bone deposition seen histologically. Using bone deposition as an indicator of tooth movement, comparisons were made between the four groups. The only significant difference in bone deposition was found between the vital active and vital inactive groups, which would be expected from the cast measurements ($p < 0.05$). Comparisons between vital and non-vital teeth, whether active or inactive, showed no significant difference ($p > 0.05$). The only unexpected finding was that no significant difference was found in bone deposition between non-vital active and non-vital inactive groups ($p > 0.05$), although their movements differed significantly according to cast measurements ($p < 0.05$). Several sources of error could account for this observation. The location of the measurement was within the mid-root region, taken at the point of clearest fluorescence. Obviously, this point was not exactly the same in each sample, thus error was introduced. Another error occurred as the fluorescent line was not always well defined, with differing thicknesses in certain regions. This could account for some variation depending which part of the line the measurement was taken from. Further, the metabolic rate of each ferret would also have affected the amount of bone deposition, since remodelling of the bone

is dependent on many factors other than external force. Variations between animals would thus be expected as each responds differently to similar stimuli. Finally, as the tooth was tipped, the centre of resistance would be somewhere in the middle to apical third of the root. At this point, rotation would occur, thus little bone would be deposited. Had the bone deposition been measured closer to the crestal bone, more remodelling may have occurred, with greater deposition evident. However, due to the lack of clarity of many fluorescent lines in this area, this was not possible in this study. Other sources of error such as the inaccuracy of the digitizing pad, intra-operator error, and possible metabolism of some of the Procion dye, become trivial in comparison to the greater sources of error.

Despite the inherent weaknesses of this study, its findings of orthodontic movement of non-vital teeth, both histologic and radiographic, are similar to those reported in existing literature. Huettner and Young examined monkeys (*Macacus rhesus*), which they thought responded closely to humans in terms of tooth movement, and found that "there is no difference between orthodontically moved vital and devitalized teeth in their gross and histologic aspects, provided that the orthodontic forces are carefully regulated and that the root canals have been sterilized, endodontically treated, and the periodontal membrane kept intact" [2]. This was thought to indicate that denervation of the pulp had little effect on the response of the periodontal tissue to orthodontic forces. Their study too was fraught with methodological errors, such as a sample of only three, use of different orthodontic mechanics, and conclusions based mostly on qualitative observations. However, the findings have yet to be proven incorrect, and are supported by this study. As stated in a later study by Mattison

et al., it appears that "the pulp and the attachment apparatus are two separate entities, thus when the pulp of a tooth is effectively removed and the root canal space properly sealed, the periodontal ligament remains unaffected" [3].

The finding of similar movement of vital and non-vital teeth also agrees with existing literature using radiographic measurements [1,4]. Wickwire *et al.* used cast and radiographic measurements to evaluate movement of endodontically treated teeth, in a retrospective study. Their conclusion was that "teeth with root canal therapy move as readily as vital teeth" [1]. Radiographic evaluation of tooth movement in past studies has usually involved root filled teeth that were previously traumatized, which may have influenced their results [1,4,5]. This factor was eliminated in this study, leaving endodontic treatment as the only variable.

The second portion of this study examined root resorption associated with movement of non-vital teeth. Root resorption was assessed both radiographically and histologically. Gross radiographic examination showed only root shortening of non-vital active teeth compared to non-vital inactive teeth. Comparisons between vital active vs. non-vital active groups, vital active vs. vital inactive groups, and vital inactive vs. non-vital inactive groups, showed no significant difference. However, radiographic measurements of root resorption are of low resolution and small changes may not be detected. The same conclusion was made by Mattison *et al.*, in a study on movement of feline cuspids, finding that the "forces of movement are the factor responsible for root resorption and not the vitality or lack of vitality of the tooth" [3]. This differs from the findings of recent studies by Spurrier *et al.* and Satoh, who found that vital teeth resorbed earlier and more than endodontically treated ones [4,6].

Remington *et al.* also noted there was a decreased amount of resorption observed in root filled teeth, speculating that increased dentin density in these teeth may provide some resistance to root resorption [7,8]. This findings of this study also contrast the opinion of Steadman, who suggested that orthodontic movement of endodontically treated teeth would result in their "melting away" due to excessive resorption, unlike their vital counterparts [9]. Wickwire *et al.* also concluded that "there appeared to be a greater frequency of root resorption in the endodontically treated teeth as compared with the control group" [1]. The non-vital teeth he examined were previously traumatized, increasing their susceptibility to resorption. Remington *et al.* criticized these findings also, stating that Wickwire *et al.* may have biased findings by including teeth with unsuccessful endodontics [1,7]. The greater resorption of endodontically treated teeth may be related more to the presence of periapical inflammation than to the lack of vitality. Another explanation suggested by some authors is that orthodontic treatment initiated too early after endodontics may cause increased resorption, thus a postponement of orthodontic therapy for some time after root canal therapy is recommended [3,10]. Huettner and Young suggest that a "delay between the completion of the endodontic therapy and the initiation of orthodontic treatment ... would be sufficient for the periodontal membrane to repair itself after endodontic instrumentation" [2]. The present study did not address this question.

No statistically significant differences ($p > 0.05$) in root length were detected histologically between any of the groups. Although possibly of greater resolution than radiographic measurements, this approach is subject to a number of variables which may mask

differences. The apex of the crestal bone was used as a fixed point, but its location may, in fact, vary. Variation in the angulation of the sectioning plane would also result in differences in measured root length not related to resorption. Further, depending on the exact location of the section measured, it may not have represented the longest portion of the tooth. Other sources of error again include measurement error, intra-operator error, and small sample size.

Another histological method of evaluating root resorption is the number of resorption lacunae. Non-vital teeth showed more resorption lacunae than vital teeth but the small difference in incidence between active non-vital and inactive non-vital teeth was not statistically significant ($p > 0.05$). This suggests that the incidence of resorption lacunae is related to non-vitality and probably the presence of periapical lesions rather than to orthodontic forces. A similar finding was reported by Mattison *et al.* on orthodontic movement of vital and non-vital feline cuspids, concluding there was "no significant difference between external root resorption of endodontically and vital teeth when both were subjected to orthodontic forces" [3].

The last histologic variable used to quantify root resorption was the resorption of cementum. Procion dye was incorporated into cementum prior to the experimental period. As cementoclasts resorbed portions of the cementum, the fluorescent line in cementum became discontinuous, representing areas of cementum removal. By linearly measuring these areas, a cumulative amount of resorption could be determined for each tooth. From this, it was found that cemental resorption is greater in teeth undergoing orthodontic tooth movement than those that are not, but the difference is only statistically significant when the

teeth are non-vital ($p < 0.05$). This is a much more sensitive technique for the measurement of resorption than either the radiographic or histological measurements of root length. Sources of error exist in this method also, however. Depending on which section of the tooth was examined, portions of resorption could have been missed due to the third dimension of depth, which was not accounted for. Inaccuracies in measurement, intra-operator error, and lack of clarity of the fluorescence would account for smaller sources of error.

Possibly one of the greatest factors affecting the quantity of root resorption was the presence of periapical lesions. Most of the non-vital teeth showed some degree of periapical inflammation, which could have caused variations in root resorption. It is impossible to determine the extent of this factor in this study, but there was no significant difference between inactive non-vital and active non-vital teeth. These findings suggest that the endodontic treatment of the teeth resulted in periapical lesions, but that the size of the lesions is not affected by orthodontic movement. This contrasts with a study by Baranowskyj which found that the healing process after endodontics was delayed if orthodontic force was applied too soon [10].

The conclusion is that non-vital teeth appear to move as readily as vital teeth when subjected to orthodontic forces. Root filled teeth show more surface resorption than vital teeth after tooth movement, though the difference is small. No difference was found in apical resorption between vital active vs. non-vital active and vital inactive vs. non-vital inactive groups. The difference seen in the non-vital active vs. non-vital active group is probably related to the presence of periapical inflammation.

3.2 - RECOMMENDATIONS FROM PRESENT STUDY

1. The results of this study regarding orthodontic treatment of endodontically treated teeth show that vital and non-vital teeth show no significant difference in tooth movement when subjected to similar forces ($p > 0.05$). This finding is in agreement with Wickwire *et al.* that "teeth with root canal therapy move as readily as vital teeth" [1]. Clinically, orthodontists should expect the same degree of predictable movement in non-vital teeth as in vital teeth. The one exception to this rule may be the previously traumatized tooth that other literature suggests may be more prone to ankylosis [11,12].

2. Root resorption assessed by gross radiographic and histologic length showed no significant difference between vital and non-vital teeth ($p > 0.05$). This suggests that the clinician should not be overly concerned with excessive root resorption of endodontically treated teeth, confirming the findings of Spurrier *et al.*, who suggested that vital and non-vital teeth can be treated with the same level of confidence [4]. There appears to be very little clinical difference in the amount or severity of root resorption between vital and non-vital teeth.

3. Histologic evaluation of root resorption by assessing resorption lacunae displayed no significant differences between active vital and active non-vital teeth ($p > 0.05$). The only significant difference was found between inactive vital and inactive non-vital groups, suggesting that the process of root canal therapy may predispose to minute degrees of root resorption ($p < 0.05$). However, this implication was not supported

by other variables. Cemental resorption analysis showed a statistically significant difference between active vital and active non-vital groups, suggesting that tooth movement of endodontically treated teeth may induce some root resorption detectable by this means ($p < 0.05$). This was supported by a significant difference between active non-vital and inactive non-vital teeth ($p < 0.05$). Caution should be taken on the interpretation of these findings based on the numerous sources of error cited previously. Although root filled teeth showed a greater loss of cementum after tooth movement than did vital teeth ($p < 0.05$), this did not result in root length differences, either histologic or radiographic. As these results are not supported by gross evaluation, it is likely that little or no difference would be detected clinically. Orthodontic movement of endodontically treated teeth results in a loss of tooth substance greater than similar movement of vital teeth, but the difference is small and only detectable microscopically.

4. Periapical lesions were detected around most of the endodontically treated teeth, both active and inactive. The nature of these lesions suggests they were an inflammatory response to the root canal therapy. However, the incidence and extent of periapical inflammation around endodontically treated teeth that were moved did not differ significantly ($p > 0.05$) from those that were not. As suggested in previous studies involving endodontically treated teeth, it is probably prudent to delay orthodontic treatment for some time after root canal therapy to allow repair of the periodontal tissue [2,3,10]. Vital teeth, whether moved or not, showed no evidence of inflammation.

3.3 - RECOMMENDATIONS FOR FUTURE STUDIES

The results of this study were not as definitive as hoped, due to a number of limitations and sources of error. A subsequent study employing many of these techniques could be performed with improvements on areas of weakness. By implementing some or all of these recommendations, a more clear-cut result would likely be achieved.

1. There are obvious limitations in an animal study as the observations are not directly transferable to the human situation. Differences in any or all of the following variables make direct applications to humans questionable: teeth size and shape; occlusion; periodontal assembly; force magnitude; response to force application; diet; and response to inflammation. There are thus numerous problems with any animal model, which may be eliminated by using human subjects or reduced by using an animal model more similar to man, such as monkeys.
2. A larger sample size is desirable to improve statistical significance and reduce the effect of random chance on the results. This should also lower the standard deviations, resulting in more conclusive findings. From a larger sample, only animals with "successful" root canal treatment (ie. no periapical lesions) could be used, eliminating periapical inflammation as a variable.
3. Most of the non-vital teeth in this study exhibited periapical inflammation, introducing a source of significant error. In a future study, the animals should be allowed a delay period following endodontic therapy, allowing recovery of the periodontal structures from instrumentation trauma. Huettner and Young suggested a three week recovery

period as being adequate, but a longer period may be desirable to further reduce this source of error [2].

4. The experimental period in this study was 3 months, which may not have been adequate to allow differences between the groups to become evident, as only small amounts of tooth movement were noted. A longer experimental period would possibly allow move significant results to be observed, as greater changes would occur.

5. The orthodontic appliance design could be improved to allow a standardized, uniform application of forces. Although the force levels were approximated in this study using a stress and tension gauge (28 - 450 g, Dentaurum, Fed. Rep. Germany), slight variation was still present. This could be reduced with a more finely calibrated instrument. More importantly, as several of the appliances came loose during the experimental period, force levels were interrupted in these cases. A more permanent cement or composite, such as Concise® (Unitek, Monrovia CA), could be utilized to prevent band displacement and allow more constant force application.

6. One obvious problem in measuring tooth movement was that pure distal movement of the mandibular cuspids was not possible as the first mandibular molars moved mesially somewhat also, despite the fact that the distal attachment of the appliance was in bone, not on the molar. It would be desirable to bolster the anchorage of the distal attachment, possibly by attaching the appliance to more teeth or using a more permanent anchor, such as an osseointegrated implant. The result would be greater

movement of the cuspids, presumably with increased significant tissue changes.

7. The impressions taken in this study were taken with a polysiloxane material (Coltoflax®, Coltène®/Whaledent, New York NY), without impression trays. A more detailed and accurate impression may be obtained if a custom tray is made for each animal and a two-paste polyvinylsiloxane impression material employed. This would reduce some of the error in distortion of the impressions.

8. Linear measurements of tooth movement were assessed from the casts using the respective CEJ's of the mandibular cuspids and first molars. Error associated with visually determining these landmarks may be reduced by using another landmark less subject to change. This could be in the form of a metallic implant, which could be detected radiographically, or a marker in the tissue, such as osseointegrated implant, which could be used as a stable reference point from which to make measurements.

9. Radiographic root length was compared in this study only from post-treatment radiographs, as the pre-treatment ones were not standardized. This was based on the assumption that contralateral cuspids would be roughly equivalent in length initially, which may be erroneous. A more accurate method would have been to construct a head holder and take radiographic images using a standardized set-up to account for magnification, both pre- and post-treatment. Individual teeth could then be compared to themselves at two different times as opposed to their contralateral counterparts, obviating the need to assume their respective initial lengths were equal.

10. Another source of error encountered in this study was the oblique sectioning of some

teeth, due to varying root curvatures. Perhaps a more standardized method could be employed to ensure the sections passed through each tooth at the same angle, minimizing this error. As well, the centre of each tooth could then be located consistently, allowing measurements to be made on comparable sections from different teeth.

11. Fluorescence from the Procion dye used in this study was not always as clear as expected. A more appropriate dye or different filters may have increased the clarity of the fluorescent lines, both in bone and cementum. Research would need to be conducted to determine the "ideal" combination of fluorescent dye and filter to examine both bone and cemental deposition. As well, a different dye injected post-treatment may have enabled a more accurate measurement of the deposition of bone and cementum during the experimental period by comparing with the initial dye.

12. Measurement techniques to determine bone deposition and cemental resorption do not take into account the third dimension of depth from the sections. A more representative measurement of each of these variables may be gleaned from area measurements, using multiple sections for evaluation. In essence, this would create a three dimensional model of the tooth and periodontium, portraying a more accurate depiction of the situation. As well, the location that the bone deposition was taken at was in the mid-root region, which may have experienced very little movement. A more significant difference may have resulted if the gingival region of the root had been assessed, but this was not possible due to the inconsistent fluorescence.

13. A relatively recent method of assessing bone remodelling activity has been the use of tartrate-resistant acid phosphatase (TRAP), an enzyme that has been found to selectively stain osteoclasts [13,14,15,16]. This technique may provide a more representative model of bone remodelling than trying to measure bone deposition in one area, as was done in the present study. Research would need to be done to determine if this marker is appropriate to assess bone remodelling in orthodontic tooth movement.

It is clear that much room for improvement exists in pursuing another study of this type. Practical and financial limitations prevented many of the above changes from being implemented, but this study serves as a viable model upon which further research into this area can be continued. It is hopeful that future research along these lines would provide more definitive answers and finally resolve the controversy that exists regarding the orthodontic movement of endodontically treated teeth.

BIBLIOGRAPHY

1. Wickwire NA, McNeil MH, Norton LA, Duell RC. The effects of tooth movement upon endodontically treated teeth. Angle Orthod 1974; 44: 235 - 42.

2. Huettner RJ, Young RW. The movability of vital and devitalized teeth in the Macacus rhesus monkey. Am J Orthod 1955; 41: 594 - 603.

3. Mattison GD, Delivanis HP, Delivanis PD, Johns PI. Orthodontic root resorption of vital and endodontically treated teeth. J Endod 1984 Aug; 10(8): 354 - 8.

4. Spurrier SW, Hall SH, Joondeph DR, Shapiro PA, Riedel RA. A comparison of apical root resorption during orthodontic treatment in endodontically treated and vital teeth. Am J Orthod Dentofac Orthop 1990 Feb; 97(2): 130-4.

5. Massler M, Malone AJ. Root resorption in human permanent teeth. Am J Orthod 1954; 40: 619-33.

6. Satoh I. Root resorption of vital and endodontically treated teeth in orthodontic movement. Kanagawa Shigaku 1990 Mar; 24(4): 601 - 17.

7. Remington DN, Joondeph DR, Artun J, Riedel RA, Chapko MK. Long-term evaluation of root resorption occurring during orthodontic treatment. Am J Orthod Dentofac Orthop 1989 July; 96(1): 43 - 6.

8. Reitan K. Biomechanical principles and reactions. In: Graber TM, Swain BF. Orthodontics-current principles and techniques. St. Louis, CV Mosby, 1985: 101-92.

9. Steadman SR. Résumé of the literature on root resorption. Angle Orthod 1942; 12: 29 - 36.

10. Baranowskyj GR. A histologic investigation of tissue response to an orthodontic intrusive force on a dog maxillary incisor with endodontic treatment and root resection. Am J Orthod 1969; 56: 623 - 4.

11. Andreasen JO. Luxation of permanent teeth due to trauma: a clinical and radiographic follow-up study of 189 injured teeth. Scand J Dent Res 1970; 78: 273 - 86.

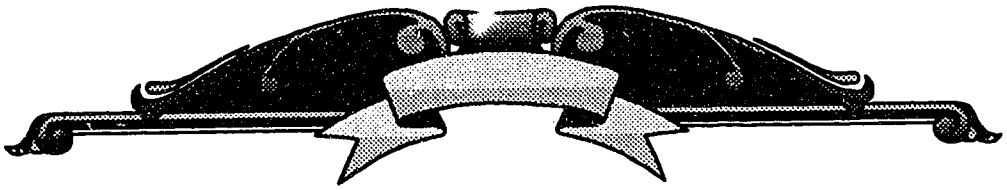
12. Hines FB Jr. A radiographic evaluation of the response of previously avulsed teeth and partially avulsed teeth to orthodontic movement. Am J Orthod Dentofac Orthop 1979 Jan; 75(1): 1 - 19.

13. Minkin C. Bone acid phosphatase: tartrate-resistant acid phosphatase as a marker of osteoclast function. Calcif Tissue Int 1982; 34: 285 - 90.

14. Bianco P, Ballanti P, Bonucci E. Tartrate-resistant acid phosphatase activity in rat osteoblasts and osteocytes. Calcif Tissue Int 1988; 43: 167 - 71.

15. Lilja E, Lindskog S, Hammarstrøm L. Histochemistry of enzymes associated with tissue degeneration incident to orthodontic tooth movement. Am J Orthod Dentofac Orthop 1983; 83: 62 - 75.

16. Farrell L, Yen EHK, Brudvik P, Rygh P, Suga DM. Identification of orthodontically induced root resorptive cells using TRAP stain. J Dent Res 1990; 69: 201.



Appendices

APPENDIX 1

Experiment No. (Animal No.)	Pre-treatment weight (kg)	Post-treatment weight (kg)
F155 (612)	1.56	1.20
F156 (607)	1.40	1.22
F157 (614)	1.48	1.34
F158 (611)	1.40	1.46
F159 (610)	1.38	1.54
F160 (615)	1.92	1.58
F161 (617)	1.60	1.46
F162 (618)	1.62	†
F163 (608)	1.50	1.38
F164 (609)	1.50	1.36
F165 (613)	1.66	1.46
F166 (616)	1.44	1.14
F167 (2343)	1.20	1.38

† = Animal died following initial general anaesthetic; not included in study

Ferret Pre- and Post- Treatment Weights

Variable	Mean (kg)	Standard Deviation	Minimum (kg)	Maximum (kg)	Number (n)
Pre-tx. Weight*	1.50	0.18	1.20	1.92	12
Post-tx. Weight	1.38	0.14	1.14	1.58	12

* = Does not include F162, which died prematurely

Summary of Means for Weights

APPENDIX 2

Experiment No. (Animal No.)	Pre-treatment measurement (mm)	Post-treatment measurement (mm)	Net movement (mm)
F155 (612)* - R†	11.36	10.09	1.27
F155 (612)* - L	11.64	9.14	2.50
F156 (607) - R†	11.59	10.34	1.25
F156 (607) - L	11.70	9.83	1.87
F157 (614) - R†	11.00	10.96	0.04
F157 (614) - L	10.48	11.06	-0.58
F158 (611)* - R	11.39	8.70	2.69
F158 (611)* - L†	11.82	10.05	1.77
F159 (610)* - R†	11.96	11.32	0.64
F159 (610)* - L	11.67	9.34	2.33
F160 (615)* - R†	11.71	8.90	2.81
F160 (615)* - L	11.90	7.27	4.63
F161 (617) - R†	13.70	11.93	1.77
F161 (617) - L	12.60	11.27	1.33
F163 (608)* - R†	11.31	7.49	3.82
F163 (608)* - L	11.56	8.27	3.29
F164 (609) - R†	11.28	10.48	0.80
F164 (609) - L	11.34	9.60	1.74
F163 (613) - R*	11.73	12.19	-0.46
F165 (613) - L	12.17	11.43	0.74
F166 (616)* - R†	11.10	9.42	1.68
F166 (616)* - L	10.81	9.71	1.10
F167 (2343) - R	11.29	9.84	1.45
F167 (2343) - L*	10.02	10.55	-0.53

* = active orthodontic springs
† = endodontically treated cuspid

Cast Measurements for Tooth Movement

APPENDIX 3

Experiment	Animal No.	Block No.	Pulp Status	Appliance Activity	Tooth Movement Cmb (mm)	Histologic Bone Description (μm)	Periapical Lesion (mm²)
F155	612	3717	V	A	2.50	386	None
F155	612	3718	NV	A	1.27	465	3.57
F156	607	3715	V	I	1.87	166	None
F156	607	3716	NV	I	1.25	151	7.78
F157	614	3711	V	I	-0.58	217	None
F157	614	3712	NV	I	0.04	66	10.91
F158	611	3713	NV	A	1.77	266	None
F158	611	3714	V	A	2.69	192	None
F159	610	3709	V	A	2.33	368	None
F159	610	3710	NV	A	0.64	203	14.24
F160	615	3719	V	A	4.63	264	None
F160	615	3720	NV	A	2.81	†	7.93
F161	617	3721	V	I	1.33	63	None
F161	617	3722	NV	I	1.77	279	3.21
F162	618	Died	§	§	§	§	§
F163	608	3723	V	A	3.29	101	None
F163	608	3724	NV	A	3.82	150	9.25
F164	609	3725	V	I	1.74	135	None
F164	609	3726	NV	I	0.80	138	None
F165	613	3727	V	I	0.74	155	None
F165	613	3728	NV	I	-0.46	140	3.52
F166	616	3729	V	A	1.10	214	None
F166	616	3730	NV	A	1.68	100	None
F167	2343	3731	NV	I	-0.53	319	5.47
F167	2343	3732	V	I	1.45	108	None

V = Vital; NV = Non-vital; A = Active; I = Inactive

† = missing value due to poor slide; § = not applicable

Tooth Movement & Periapical Lesion Measurements

APPENDIX 4

Specimen	Block No.	Pulp Status	Appliance Activity	Radiographic Tooth Length (mm)	Radiographic Root Length (mm)	Histologic Root Length (mm)	Resorption Bays (No.)	Cumulative Resorption (µm)
F153	3717	V	A	16.94	11.20	10.14	†	†
F153	3718	NV	A	16.03	10.61	9.26	0	436
F156	3715	V	I	17.29	11.11	9.83	0	0
F156	3716	NV	I	16.19	11.09	10.40	3	757
F157	3711	V	I	17.28	11.19	9.71	0	0
F157	3712	NV	I	17.22	11.13	8.97	4	1150
F158	3713	NV	A	16.67	10.52	8.11	0	4017
F158	3714	V	A	16.68	10.48	4.29	0	0
F159	3709	V	A	16.21	10.31	8.91	0	0
F159	3710	NV	A	16.05	10.47	10.91	0	4531
F160	3719	V	A	16.56	10.57	10.51	0	3653
F160	3720	NV	A	17.29	11.10	8.80	10	2807
F161	3721	V	I	17.32	11.17	7.03	0	0
F161	3722	NV	I	17.63	11.38	10.11	10	564
F162	Died	§	§	§	§	§	§	§
F163	3723	V	A	16.11	10.29	8.40	0	0
F163	3724	NV	A	15.64	9.52	9.43	10	3371
F164	3725	V	I	16.71	10.30	10.57	0	1827
F164	3726	NV	I	17.10	10.70	9.71	0	0
F165	3727	V	I	16.22	11.00	10.46	0	0
F165	3728	NV	I	17.05	10.88	7.89	1	1665
F166	3729	V	A	16.41	10.41	10.23	1	1326
F166	3730	NV	A	16.37	10.44	9.20	3	3972
F167	3731	NV	I	‡	‡	10.82	3	460
F167	3732	V	I	‡	‡	10.47	0	1752

V = Vital; NV = Non-vital; A = Active; I = Inactive

† = missing value due to poor slide; § = not applicable; ‡ = no radiograph available

Root Resorption Measurements

APPENDIX 5

Variable	N	Mean	Std. Dev.	Minimum	Maximum
Tooth Movt-casts (mm)	6	2.00	1.14	0.64	3.82
Bone Deposition (μm)	5	236.80	141.70	100.00	465.00
Periapical Lesion (mm^2)	6	5.83	5.66	0	14.24
Tooth Length (mm): X-ray	6	16.34	0.58	15.64	17.29
Root Length (mm): X-ray	6	10.44	0.51	9.52	11.10
Root Length (mm): Slide	6	9.29	0.93	8.11	10.91
Resorption Bays (no.)	6	3.83	4.92	0	10
Cemental Resorption (μm)	6	3189.00	1473.61	436.00	4531.00

Descriptive Statistics for Nonvital Active Teeth

APPENDIX 6

Variable	N	Mean	Std. Dev.	Minimum	Maximum
Tooth Movt-casts (mm)	6	0.48	0.94	-0.53	1.77
Bone Deposition (μm)	6	182.17	96.22	66.00	319.00
Periapical Lesion (mm^2)	6	5.15	3.82	0	10.91
Tooth Length (mm): X-ray	5	17.04	0.53	16.19	17.63
Root Length (mm): X-ray	5	11.04	0.26	10.70	11.38
Root Length (mm): Slide	6	9.65	1.07	7.89	10.82
Resorption Bays (no.)	6	3.50	3.51	0	10
Cemental Resorption (μm)	6	766.00	579.13	0	1665.00

Descriptive Statistics for Nonvital Inactive Teeth

APPENDIX 7

Variable	N	Mean	Std. Dev.	Minimum	Maximum
Tooth Movt-casts (mm)	6	2.76	1.17	1.10	4.63
Bone Deposition (µm)	6	254.17	108.97	101.00	386.00
Periapical Lesion (mm^2)	6	0	0	0	0
Tooth Length (mm)-X-ray	6	16.49	0.31	16.11	16.94
Root Length (mm)- X-ray	6	10.54	0.34	10.29	11.20
Root Length (mm)- Slide	6	8.75	2.33	4.29	10.51
Resorption Bays (no.)	5	0.20	0.45	0	1
Cemental Resorption (µm)	5	995.80	1592.53	0	3653.00

Descriptive Statistics for Vital Active Teeth

APPENDIX 8

Variable	N	Mean	Std. Dev.	Minimum	Maximum
Tooth Movt-casts (mm)	6	1.09	0.91	-0.58	1.87
Bone Deposition (µm)	6	140.67	52.55	63.00	217.00
Periapical Lesion (mm^2)	6	0	0	0	0
Tooth Length (mm)-X-ray	5	16.96	0.49	16.22	17.32
Root Length (mm)- X-ray	5	10.95	0.37	10.30	11.19
Root Length (mm)- Slide	6	9.68	1.35	7.03	10.57
Resorption Bays (no.)	6	0	0	0	0
Cemental Resorption (µm)	6	596.50	924.40	0	1827.00

Descriptive Statistics for Vital Inactive Teeth

APPENDIX 9

Variable	Mean Difference	Standard Error	Degrees of Freedom	p value	Significance (p < 0.05)
Tooth Movt-casts (mm)	1.52	0.605	10	0.031	S
Bone Deposition (µm)	54.633	71.822	9	0.466	NS
Periapical Lesion (mm^2)	0.683	2.787	10	0.811	NS
Tooth Length (mm): X-ray	-0.696	0.337	9	0.069	NS
Root Length (mm): X-ray	-0.593	0.254	9	0.045	S
Root Length (mm): Slide	-0.365	0.577	10	0.541	NS
Resorption Bays (#)	0.333	2.465	10	0.895	NS
Cemental Resorption (µm)	2423.000	646.391	10	0.004	S

Comparative Statistics for Non-vital Active vs. Non-vital Inactive Teeth

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APPENDIX 10

Variable	Mean Difference	Standard Error	Degrees of Freedom	p value	Significance (p < 0.05)
Tooth Movt-casts (mm)	1.665	0.603	10	0.020	S
Bone Deposition (µm)	113.500	49.389	10	0.044	S
Periapical Lesion (mm^2)	†	†	†	†	†
Tooth Length (mm): X-ray	0.479	0.241	9	0.078	NS
Root Length (mm): X-ray	0.411	0.214	9	0.088	NS
Root Length (mm): Slide	0.932	1.100	10	0.417	NS
Resorption Bays (#)	-0.200	0.181	9	0.297	NS
Cemental Resorption (µm)	-399.300	766.398	9	0.615	NS

† = analysis cannot be performed as standard deviations of both groups is 0

Comparative Statistics for Vital Active vs. Vital Inactive Teeth

APPENDIX 11

Variable	Mean Difference	Standard Error	Degrees of Freedom	p value	Significance (p ≤ 0.05)
Tooth Movt-casts (mm)	-0.758	0.666	10	0.281	NS
Bone Deposition (µm)	-17.367	75.440	9	0.823	NS
Periapical Lesion (mm^2)	-5.832	2.309	10	0.030	S
Tooth Length (mm): X-ray	-0.143	0.268	10	0.604	NS
Root Length (mm): X-ray	-0.100	0.251	10	0.699	NS
Root Length (mm): Slide	0.538	1.025	10	0.611	NS
Resorption Bays (#)	-3.633	2.226	9	0.137	NS
Cemental Resorption (µm)	-2193.20	925.013	9	0.042	S

Comparative Statistics for Non-vital Active vs. Vital Active Teeth

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APPENDIX 12

Variable	Mean Difference	Standard Error	Degrees of Freedom	p value	Significance (p ≤ 0.05)
Tooth Movt-casts (mm)	-0.613	0.535	10	0.278	NS
Bone Deposition (µm)	41.500	44.759	10	0.376	NS
Periapical Lesion (mm^2)	-5.148	1.561	10	0.008	S
Tooth Length (mm): X-ray	-0.074	0.321	8	0.823	NS
Root Length (mm): X-ray	-0.082	0.203	8	0.697	NS
Root Length (mm): Slide	0.028	0.702	10	0.969	NS
Resorption Bays (#)	-3.500	1.432	10	0.035	S
Cemental Resorption (µm)	-169.500	445.327	10	0.711	NS

Comparative Statistics for Non-vital Inactive vs. Vital Inactive Teeth