EVALUATION OF TRANSVERSE, BODILY TOOTH MOVEMENT AND ITS EFFECTS ON THE SURROUNDING HARD TISSUE

A Thesis

by

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ABSTRACT

Orthodontic expansion has been associated with uncontrolled tipping and alveolar bone loss. Recent research evaluating orthodontic expansion has shown osteoblastic activity on the buccal cortical bone apical to the dehiscence. We hypothesize that the negative effects seen during orthodontic expansion is a result of tipping rather than expansion. The aim of the present study was to produce buccal translation, with little or no coronal tipping, and evaluate hard tissue changes of the dental-alveolar complex.

A prospective, randomized, split-mouth study was conducted with 11 patients (average age 14.1 years, range 12.5-16.9 years) requiring maxillary first premolar extractions for comprehensive orthodontic treatment. Pre and post-treatment records included models, photographs, and small field of view cone beam computed tomographic images (FOV CBCT) of the right and left maxillae. One maxillary first premolar was randomly chosen and moved to the buccal with 50 grams of force applied approximately at the tooth's center of resistance. The other premolar served as the control tooth. Forces were re-activated every 3 weeks for approximately 9 weeks of active movement, after which the tooth was held in place for 3 weeks to allow for maturation of the surrounding tissue. Pre and post treatment records were analyzed and superimposed to evaluate changes in the dental-alveolar complex.

The results showed significant movement (0.96 mm, $p=0.008$) of the experimental premolar occurred 3 mm apical to the CEJ. There was minimal buccal

tipping (2.2°, p=0.003). Maximum and minimum buccal bone heights decreased 0.60 mm (p=0.003) and 0.25 mm (p=0.262) respectively. The distribution of the maximum bone height measurement was bimodal, with 6 patients showing 0.42 mm (IQR -0.25 mm to 0.52mm) and 5 patients showing 8.3 mm (IQR 7.15 to 10.05) of vertical bone loss. Buccal bone thickness at the midline 3 mm apical to the CEJ decreased 0.63 mm (p=0.016). Based on direct measurements and CBCT superimpositions, buccal bone grew 0.46 mm (p=0.005) and 0.51 mm (p=0.036), respectively.

Using light continuous forces, it is possible to produce buccal tooth movement with only limited amounts of tipping. With such movements buccal bone growth occurs, but there are potential limitations.

DEDICATION

I would like to dedicate this thesis to my parents, Jon and Donna, and to my wife, Heather. Their unwavering support and inspiration has been critical the last three years. I would have never started this journey if it was not for Heather, and I would not have had the courage to succeed without my parents.

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CHAPTER I

INTRODUCTION AND

LITERATURE REVIEW

A tooth size to arch length discrepancy (TSALD), also known as crowding, is one of the most common problems facing the clinical orthodontist today. In fact, 39% of American adults have lower incisor crowding that is considered to be moderate to extreme (>4 mm).[2] By definition, crowding is a discrepancy between the combined mesio-distal width of the teeth and the space available on the underlying alveolar bone. Consequently, in order to correct a TSALD, one must either remove tooth structure, through extractions, or increase space with expansion. In recent years nonextraction treatment has become increasingly popular. New techniques and materials for expansion have led to a marked reduction in the percentage of orthodontic treatments featuring premolar extractions.[3, 4]

Expansion allows the clinician to increase space. There are two basic types of expansion in orthodontics: orthopedic and orthodontic. Orthopedic expansion focuses on moving the underlying skeletal base, primarily the maxilla, and requires that the patient possesses remaining growth potential. Orthodontic expansion, on the other hand, creates space by moving the dentoalveolar complex and can be accomplished regardless of growth.

The predominant theme in all forms of expansion is a loss of torque control. The loss of torque control leads to uncontrolled tipping of the crowns in the buccal/labial

direction. Several appliances claim to control this movement through a variety of methods, including the utilization of soft tissue pressures, passive self-ligation, and light force nickel titanium archwires. However, both clinical and animal studies have shown that all current, non-surgical, forms of transverse expansion produce some degree of tipping. And tipping appears to have deleterious effects on the surrounding tissues.

Biomechanically uncontrolled tipping occurs when a force is applied away from the tooth's center of resistance without an additional moment force or couple. A moment to force ratio approaching 10:1 is necessary to achieve translation without tipping. Cantilevers with force application at the center of resistance have been used to accomplish bodily movement. In fact, such force systems are often used for the mesial-distal movements that occur during space closure with sliding mechanics. Since this force system has not been used routinely in the transverse dimension, its effects on both the tooth and surrounding tissues are not well documented.

The goal of the present study is to move premolars in a buccal direction utilizing a cantilever force, applied near the center of resistance. The effects on both the teeth and surrounding tissues will be evaluated with pre and post movement cone beam computed tomography (CBCT), as well as study models. After expansion, the tooth will be extracted for surface evaluation with micro computed tomography (microCT). This information will further our understanding of lateral movement and provide insights concerning the changes that occur.

Problem

From 1988-1994 the Third National Health and Nutrition Examination Survey (NHANES III) collected data representing the entire United States population. The oral health component of the survey evaluated a range of clinical features including occlusal characteristics and orthodontic treatment. Their findings highlighted the significant need for orthodontics in the United States population. By applying the Index of Treatment Need (IOTN)[5] to the NHANES III data it has been estimated that 57%-59% of the population has at least some degree of need for orthodontic treatment.[2] Irregularity was found to be severe enough in 15% of the population to create significant effects on appearance and function and thus require major arch expansion or extraction of teeth.[6] In fact, the most common orthodontic problem is crowding or tooth size to arch length discrepancy. According to the NHANES III the percentage of the population with crowding greater than 3mm, which is considered moderate to extreme, was 29.6% in the maxilla and 36.1% in the mandible.[2] When evaluated further 23% of the individuals 15-50 years of age had a mandibular incisor irregularity index (II) of 4-7 mm and 17% had an II $\geq$ 7 mm.[7]

The treatment options for solving a tooth size to arch length discrepancy include reducing the tooth mass, through extraction[8, 9] or interproximal reduction[10, 11], and arch expansion.[12] While orthodontics has seen many advances over the last century, the choice between extraction and non-extraction remains one of the most significant decisions in treatment planning. Both extraction and non-extraction treatments have

ardent supporters. Edward Angle, regarded as the father of modern orthodontics,[13] was an advocate for buccal expansion to resolve any tooth size to arch length discrepancy and thus accommodate a full compliment of teeth.[14, 15] Although the majority of clinicians today are not as dogmatic as Angle, recent trends indicate that there has been a marked reduction since 1968 in the percentage of orthodontic treatments featuring premolar extraction.[3, 4, 16] A national survey of orthodontists in the United States performed by O'Conner in 1993 found that the extraction rate had declined from 37.74% to 29.28% over the previous five years. [3]

Extraction Treatment

Historically, critics of extraction treatment have claimed that removal of teeth causes detrimental effects on facial aesthetics.[17, 18] Some believe that extraction treatment causes over retraction that leads to excessive flattening of the face and in some instances even a concave facial appearance. Although it is possible for this to occur when there is a poor diagnosis and treatment, numerous studies provide evidence indicating that this fear is unfounded.[19-22] Others argue that there is a difference in the facial profile of patients treated with extractions versus those treated non-extraction. Boley et al. in 1998 had 192 general dentists and orthodontists evaluate the photographic and cephalometric profiles of 25 consecutively treated cases. These cases included patients with both extraction and non-extraction treatment. The results showed that general dentists and orthodontist could not determine whether they had been treated

nonextraction or extraction by looking only at the face.[23] Rushing et al.[24] and Johnson et al.[25] showed similar results. It has also been noted that when cases are treated to a similar mandibular incisor position, there was no significant difference between extraction and nonextraction treatment in the long-term profile changes.[26]

The literature supports the idea that satisfactory results can be achieved by extraction or nonextraction treatment.[23-25] In fact, extraction treatment can offer significant advantages with mechanics, as well as the opportunity to place the incisors in an ideal position and inclination. However, extracting teeth is an irreversible decision that is often difficult to make. Many cases fall on the border between extraction and nonextraction. For numerous reasons, including a conservative approach, public perception and attempting to avoid litigation, an increasing number of clinicians are tending toward nonextraction treatment for such cases.[3]

Expansion Treatment

McNamara described three fundamental methods of expansion: passive expansion, orthopedic expansion, and orthodontic expansion.[27] Passive expansion occurs when the buccal or labial musculature are removed from the teeth, allowing the forces produced by the tongue to move the teeth. The FR-2 designed by Frankel and the lip bumper are appliances that are capable of producing passive expansion. Passive expansion produces dentoalveolar changes rather than bone deposition at the midpalatal suture.[28] Orthopedic expansion, the second method of expansion described by

McNamara, must be utilized in order to achieve expansion at the midpalatal suture. The best example of an orthopedic expansion appliance is a rapid palatal expander (RPE). Orthopedic expansion forces are focused on the underlying skeletal structures so that changes primarily occur in the underlying basilar bone, rather than by movement of the teeth through the alveolar bone.[27, 29, 30] Since orthopedic expansion targets the suture of the maxilla, it is restricted to the time period when these structures are readily influenced. Sutural patency and remaining growth potential are critical factors when attempting orthopedic changes. Due in large part to these confounding factors, orthopedic expansion is a viable option for only a limited number of patients. Orthodontic expansion, the third method of expansion, focuses primarily on the lateral movement of the dentoalveolar complex as its primary means of expansion. It is not dependent upon growth or maturational status.

History of Expansion

Orthodontic expansion is a well-established practice that has been utilized since the inception of modern orthodontics. The E arch, Edward Angle's first appliance introduced in the late 1800's, was among the first appliances used for orthodontic expansion. In the E arch system, all of the teeth were banded and a heavy archwire was inserted from molar tube to molar tube. The remaining teeth were then ligated to the expanded archform. The archwire force moved the teeth labially into alignment, correcting any crowding or irregularities.[31]

Angle progressed from the E-arch, system to the Pin and Tube, and then to the Ribbon Arch.[32] As with the E-arch all three systems relied in large part on tipping and had minimal root control.[14] In 1928 Angle introduced the Edgewise system.[33] By reorienting the slot from vertical to horizontal and utilizing rectangular wire this system, was able to gain better root control and reduce tipping as the arches were expanded.[33]

By the first decade of the twentieth century companies began to manufacture standardized appliances which made the concept of banding each tooth much more realistic. The appliances were made as sets of various kinds and were placed on cards to be sold to orthodontists. Orthodontists were able to utilize soldering techniques to fit the appliance to the individual patient.[34] At that time however, clinicians were limited to the use of precious metals as the only available archwire material.

Stainless steel was introduced during World War I. In the late 1930s, the process of drawing the material was refined to the point that it could be used to form archwires.[35] The properties of stainless steel allowed for better control in three planes of space while maintaining a moderate force level. Greater control during expansion meant further reductions in uncontrolled tipping. However, the amount and type of force that was biologically appropriate had not been determined.

In 1956, Raymond Begg introduced the Begg appliance to the United States. Begg had been taught the ribbon arch appliance at the Angle school prior to returning to Australia in the 1920s.[14] Begg kept the ribbon arch bracket but turned it upside down, he also added auxiliary springs to help with root control, and replaced the rectangular archwire with a small 0.016 inch stainless steel round wire.[36, 37] With his technique,

Begg used the light continuous forces of the small, round stainless steel wire to tip and expand the teeth. He then utilized the auxiliary spring for root control to upright them. He was one of the first to introduce the concept of light continuous forces in orthodontics.

In the 1960s there was an increase in the understanding of the tissue response to tooth movement and the efficiency of light forces.[38, 39] Research in bone physiology also made significant breakthroughs that provided a more complete picture of the surrounding tissue's response to tooth movement.[40] With advances in material science that allowed orthodontist more control over tooth movement and the amount of force, it became evident that the orthodontist should abide by the newly discovered biological principles. Most of the established names in the era, including Begg, Jarabak, and Burstone, adopted the concept of physiologic, light, continuous forces.[41-43]

With small, round, stainless steel archwires orthodontists have been able to create lighter forces as they expand and unravel the dentition. However, small archwires do not provide the same control as the large rectangular wires, resulting in significant tipping. The introduction of nickel titanium archwires to the straightwire system allowed for the next step in the evolution of expansion. In the 1960's the Office of the Navy was actively studying new types of alloys that exhibited a shape memory effect (SME).[44] Nickel titanium alloy showed a SME and was given the name nitinol, an acronym for nickel-titanium Naval Ordinance Laboratory. In comparison to other archwire materials being used at the time, early nitinol had a very high load deflection rate, allowing it to be springy.[35] In fact, it delivered only one-fifth to one-sixth the force

per unit of deactivation that other materials delivered.[45] These unique properties allowed nitinol to better approach the light, continuous forces needed for orthodontic expansion.[35] It also allowed the clinician to apply light forces with a rectangular wire that provided some tip control.

More recently, improvements in orthodontic materials, such as copper NiTi and brackets with passive self-ligation, have led to the development of the Damon System. By utilizing the low friction environment created by passive brackets and the light forces of copper NiTi, Dr. Dwight Damon has created what he has termed a "biological force" for orthodontic expansion. It has been suggested that, as the arches widen, the biological forces stimulate the tongue to lift and reposition anteriorly, "awakening" its own intrinsic forces.[46, 47]

Expansion and Arch Perimeter

Extensive studies have been performed on the relationship between arch width and crowding. Brunelle et al. utilized the NHANES III data to show that transverse deficiencies were related to maxillary alignment.[6] Subjects with fair (3-5 mm) or poor (>6 mm) irregularity index demonstrated much higher prevalence of posterior crossbite than subjects with good (1-2 mm) or excellent (0-1 mm) alignment.[6] Howe et al. compared the dental casts of patients with severe crowding to those of untreated individuals classified as having an ideal or near ideal occlusion.[48] They found that the two samples had significant differences in both arch width and arch perimeter.[48]

Germane et al. created a mathematical study model that was able to quantify the amount of arch perimeter gained as a result of expansion.[1] Their conclusions are listed in Table 1.

Adkins et al. evaluated 21 consecutively treated orthodontic patients who required arch expansion. A Hyrax (OSI, Wilmington, Del.) expansion appliance was used to treat each patient. Pre and post treatment measurements were performed on dental casts that were digitally marked and measured.[49] They found that molar width increased 6.5 mm, premolar width increased 6.1 mm, and arch perimeter increased 4.7 mm. They concluded that for every 1 mm of width gained at the maxillary first premolar an increase of 0.7 mm can be expected in maxillary arch perimeter.[49]

Expansion and Crossbites

Orthodontic expansion also helps to correct transverse discrepancies and crossbites. According to the NHANES III data, 9.1% of the United States population has a posterior crossbite.[2, 31] Godoy et al. when evaluating the efficiency of orthodontic expansion (quad helix) versus orthopedic expansion (removable rapid palatal expander), found that orthodontic expansion was successful in 100% of the 33 patients in the orthodontic expansion (quad helix) group. Over half of those patients showed cross bite correction within three months of treatment.[50] Minimal literature is available regarding correction of crossbites using orthodontic expansion via archwires alone. However, there is convincing literature describing the transverse effects of archwire expansion.

Vajaria, et al. evaluated 27 patients treated with the Damon System. The inter-premolar and inter-molar changes were significant. Width increased 2.87 mm and 2.79 mm at the maxillary first premolar and maxillary first molar, respectively.[51] These transverse changes appeared to have had a positive effect on the aesthetic outcome.

Expansion and Aesthetics

Moore et al. evaluated the influence of buccal corridors on smile aesthetics, as assessed by laypersons.[52] In the study, the maxillary posterior dentition of 10 smiling subjects was digitally altered to fill in varying amounts of the buccal corridor. The results showed that a broader smile was judged to be more attractive than a narrow smile.[52] In a similar study, Roden-Johnson et al. described how laypersons, general dentists, and orthodontists perceived buccal corridors. They found that both general dentists and orthodontists rated broader arch forms as more aesthetic than untreated arch forms.[53]

Martin et al. also evaluated the impact of buccal corridors on smile attractiveness.[54] They digitally altered a smiling photograph of one female to produce smiles that filled varying amounts of the oral aperture. Eighty-two orthodontists and 94 laypeople evaluated the photos. The findings revealed that both groups rated smiles with small or no buccal corridors as being significantly more attractive than those with large buccal corridors.[54]

Negative Effects of Expansion

Although orthodontic expansion can improve aesthetics, crowding, and transverse discrepancies, it is not universally accepted as a viable treatment modality. One of the first critics of orthodontic expansion was Dr. Charles H. Tweed. In 1933, Dr. Tweed brought attention to some of the negative effects of orthodontic expansion. He explained that buccal expansion to resolve a TSALD often resulted in a protrusive, unaesthetic, profile and an unstable dentition.[8, 14, 55] Tweed strictly practiced Angle's philosophy of the full complement of teeth for six and one-half years. After being disappointed with some of the results he saw, Tweed decided to perform a thorough evaluation of his completed cases. He was able to take models, x-rays, and photographs of 70% of all cases that he had ever treated. He classified them as successes or failures based on four orthodontic objectives: stability, healthy tissue, functional occlusion, and facial aesthetics.[55] He found that over 80% of the cases were judged to be failures based on at least one of the four objectives. Tweed evaluated the records, and compared them to published norms. For one of his measurements, he made sagittal sections of the models in an attempt to calculate the inclination of the mandibular incisors relative to the basilar bone. Margolis, using Broadbent's cephalometric data acquired from the Bolton study, previously demonstrated that lower incisor angulation ranged from 85 to 93 degrees.[56] Tweed consistently found that the incisors in the group of failures were beyond this range. Excessive proclination was problematic for stability and detrimental to the facial aesthetics. He decided to retreat many of those patients with extraction

therapy to bring the incisors within the appropriate range.[55] In 1940 he presented 100 of the cases who had been treated first without extraction and then again with extraction therapy at the American Association of Orthodontist meeting in Chicago. The photographs and plaster models that he presented supported his criticism of excessive proclination when utilizing buccal expansion to correct a tooth size to arch length discrepancy.[57]

Expansion Effects on the Dentition

While the materials and techniques associated with orthodontic expansion have shown significant improvements since Dr. Tweed presented his cases in 1940, many of the negative effects remain. One of the negative aspects of orthodontic expansion is uncontrolled tipping of the clinical crowns. Lundgren et al. in 1996 utilized light forces (50cN) in a human model to expand the maxillary premolars.[58] They noted movement in all three planes of space. In fact, the apices of the premolars moved palatally in 49 of 56 cases, and they tipped between 0.2 to 22.9 degrees. In 2009, Paventy et al. evaluated the effects of comprehensive treatment with the Damon System.[59] Nineteen patients with moderate to severe crowding (5 mm or more) were treated following the published Damon System protocol. Treatment in the transverse dimension was limited to expansion with the normal archwire sequence used in the Damon System. The study showed effective expansion of the dental arches with increased arch perimeter. However, the increase in arch width was in part due to tipping of the crowns. On

average, the first and second maxillary premolars expanded more at their cusp tips than at their lingual gingival margins. For the maxillary first premolar, the difference was 1.7 mm, and for the maxillary second premolar the difference was 1.6 mm. In 2011, Cattaneo et al. evaluated transverse movements and buccal bone modeling in humans after orthodontic archwire expansion.[60] Sixty-four patients were randomly assigned to treatment with either active (In-Ovation R) or passive (Damon 3MX) brackets. Outcomes were evaluated with digital models and pre and post-treatment cone beam CT radiographs. They found that in all but one patient, transverse expansion was achieved through buccal tipping. Specifically, the Damon group showed 11.7 degrees of tipping at the first premolars and 13.5 degrees of tipping at the second premolars. The In-Ovation group had 11.8 degrees and 13.0 degrees of tipping of the same teeth.[60] Kraus in 2012 used foxhound dogs to evaluate archwire expansion using mechanics similar to those used with the Damon system.[61] Over eight weeks he saw on average of 3.5 mm of tooth movement. With the buccal movement there was a significant amount (15.8 degrees) of tipping.

Expansion Effects on Surrounding Tissues

Poor tooth position and excessive tipping are not the only undesirable effects of orthodontic expansion. Buccal or labial crown movement may be producing deleterious amounts of stress on the surrounding hard tissue. Steiner et al., who evaluated labial tooth movement in monkeys, found decreases in the connective tissue and the buccal

marginal bone levels after labial movement.[62] Kraus‘ experimental model mentioned before showed similar results with slow, controlled buccal movement in foxhounds.[61] He found on average 2.9 mm and 1.2 mm of bone height loss, at the mesial and distal roots, respectively.

In Paventy’s thesis, the human clinical trial described previously, a loss of buccal bone height after expansion with the Damon system was also noted.[59] Statistically significant buccal bone height loss occurred at the maxillary first premolars, mandibular first and second premolars, and mandibular first molars. Also, statistically significant facial bone width loss was evident 3 mm apical to the bony crest of the maxillary first and second premolars and first molars, as well as the mandibular right first premolar, second premolars, and first molars. In 2010, J. Paventy re-evaluated some of the same subjects 6-12 months post-treatment (5 of the 19 subjects could not be contacted).[63] He noted that all teeth except one showed a small amount of recovery of facial bone height and width, but none of the improvements were statistically significant. In the randomized clinical trial described earlier by Cattaneo et al., loss of buccal bone was also seen.[60] They found that the buccal bone area lateral to the second premolar decreased on average 23% and 18% (right and left sides) with Damon and 17% and 12% (right and left sides) with In-Ovation. Cattaneo and coworkers also found that the bone loss that occurred with the inter-premolar expansion was positively associated with buccal tipping.

In 1985 Quinn published a review of the literature on force magnitude in orthodontics. When evaluating different tooth movements, Quinn noted that with

tipping, forces were concentrated on the crestal bone.[64] These forces could surpass physiological levels and become deleterious. To more fully understand the detrimental affects associated with orthodontic expansion, a better understanding of the dentoalveolar anatomy and biology of tooth movement is necessary.

Dentoalveolar Anatomy

Dentoalveolar is a term used to describe the complex of tissues including the tooth and the tissues that house the tooth root. The tooth root is comprised of three tissues: the inner pulp tissue, dentin that immediately surrounds the pulp and an outer encasing layer of cementum. Both the dentin and the cementum are considered hard or calcified connective tissues, much like the surrounding alveolar bone, with a fibrous matrix as its principal constituent. Unlike the surrounding bone, cementum contains no blood vessels and has no innervation. It is not clear whether physiologic resorption or remodeling of the cementum occurs under normal circumstances. However, during tooth movement, resorptive and reparative processes occur on the cemental surface.[65]

The tooth root is suspended in the alveolar socket by the periodontal ligament (PDL). The cells responsible for creating and maintaining the PDL are fibroblasts. Fibroblasts make ground substance, as well as extracellular fibers of connective tissue such as collagen and elastin. PDL fibroblast cells originate from the dental follicle tissue during tooth development. During physiologic remodeling, as well as tooth movement, fibroblasts come from both mitotic replication and local mesenchymal cells.[66]

The periodontal ligament is an uncalcified connective tissue whose fibrous matrix is primarily collagen. The PDL attaches directly from the cementum of the tooth root to the alveolar bone of the surrounding socket. The uncalcified collagen fibers of the PDL extending into the calcified bone of the surrounding socket are called Sharpey's fibers.[67] The PDL is responsible for resorbing and distributing the forces of mastication. Due to the heavy multidirectional forces produced during normal physiological conditions, turnover is much higher for collagen than most other tissues.[68]

There are three different types of bone that make up the dentoalveolar complex. Each of the three types can be differentiated into two common maturational categories, woven bone and lamellar bone. Woven bone is found during the early stages of life, in ligament and tendon insertion areas, and in regions undergoing significant remodeling due to pathology or fracture. Woven bone is therefore immature and poorly developed.[69] Bone strain introduced by mechanical stress perpetuates the production of woven bone which ultimately remodels into the more mature lamellar bone.[70] Lamellar bone is the primary component of both trabecular and cortical bone. It is organized based on the mechanical load placed on the bone and is highly mineralized.

The first of the three types of dentoalveolar bone creates the socket immediately adjacent to the tooth root and PDL. This bone is known as cribriform plate or alveolar bone proper. As the name cribriform suggests, it has numerous small openings that allow communication from the trabecular bone to the PDL via blood vessels, interstitial fluid, and nerves. Despite its numerous openings, the cribriform plate is comprised of relatively compact, lamellar bone.[67, 71]

Surrounding the alveolar bone proper is a layer of spongy, trabecular bone. It is comprised of lamellar and bundle bone with sparse trabeculae running through bone marrow. The marrow contains blood vessels, nerves, and fatty tissue. If the alveolar complex is narrow, as is often seen when the tooth root approximates the cortical plate, the spongy, trabecular, portion becomes very thin or non-existent. When this occurs, the cribiform plate fuses directly to the cortical plate, which is the final type of bone in the dentoalveolar complex.[71, 72]

The cortical plate is a dense, compact, lamellar bone that makes up the outer layer of the complex. It is referred to as the buccal or lingual cortex, depending on the orientation. In a healthy environment it is covered by the periosteum and attached gingiva.

Dentoalveolar bone, just like all bone tissue, has two major cell types responsible for development and remodeling. The first cell type is the bone forming cells known as osteoblasts. Osteoblasts derive from local osteoprogenitor cells known as preosteoblasts. Ultimately, the preosteoblasts come from mesenchymal stem cells located in the deep layers of the periosteum, as well as in the bone marrow.[66] The second type of bone cells is the bone resorbing cells known as osteoclasts. Osteoclasts are multinucleated cells that derive from hematopoietic stem cells in the bone marrow. Osteoclasts form by the fusion of precursor cells. They are commonly referred to as being derived from the monocyte/macrophage lineage since the hematopoietic stem cells responsible for their creation also gives rise to monocytes in the peripheral blood and tissue macrophages.[73]

Osteoblasts and osteoclasts combine with communication and supporting cells such as osteocytes, bone lining cells, and the capillary blood supply to construct what is known as the "Basic Multicellular Unit" or BMU.[74] The BMU is responsible for physiological remodeling in both cortical and trabecular bone. Although both bone types utilize the BMU during remodeling, the actual remodeling process is uniquely different between cortical and trabecular bone. Trabecular bone as mentioned previously is highly vascular and less dense. This allows for the BMU to move more freely, consequently acting on an entire region of bone termed the resorptive front. The BMU is forced to have a different method of action in cortical bone due to its increased density. The BMU forms what is known as a "cutting cone", made up of osteoclasts, which creates a cylindrical canal through the cortical bone in the direction of the load.[75] Following the osteocytes and cutting cone are the osteoblasts. The osteoblasts fill the newly created tunnel and produce an osteon of new bone.[76] This difference in physiological remodeling allows us to better understand the disparity in speed and efficiency of tooth movement through cortical and trabecular bone.

Biology of Tooth Movement

Orthodontic tooth movement has been described as the biological effect of an external force on the dentoalveolar complex. The external force disrupts the physiologic equilibrium of the tooth that is created by the surrounding hard and soft tissues and encourages movement, until stability is attained in the new force system.[14] This response

is complicated due to the involvement and interaction of numerous tissues, including the tooth root, the surrounding periodontal ligament, and the alveolar bone. Each tissue has its own respective cell populations and thus different mechanisms of remodeling and stress adaptation.[77] Due to this complexity, various theories have been developed to describe the events associated with orthodontic tooth movement. All of these theories are based on bone studies performed in the middle of the 19th century. At that time, von Meyer and Wolf found that mechanical stimuli played a significant role in the maintenance and structural development of skeletal tissue.[78, 79]

Sandstedt in 1904 and 1905 applied this early knowledge specifically to orthodontic tooth movement using a dog model.[80, 81] He moved maxillary incisors lingually 3 mm and noted that bone was deposited on the alveolar wall of the tension side and was resorbed on the pressure side by numerous multinucleate osteoclasts in Howship's lacunae. In these studies, he also described the detrimental effects of heavy forces. Sandstedt explained that heavy forces occluded capillaries, causing thrombosis, cell death and the production of localized cell-free areas.[81, 82]

This classic research, combined with that of Oppenheim in 1911 and 1930, and Schwarz in 1932, laid the groundwork for the development of the first theory proposed to explain orthodontic tooth movement.[83-85] This theory, called the pressure-tension theory, implies that differential pressures within the dentoalveolar complex created by orthodontic forces are responsible for tooth movement. The pressure-tension theory has been supported through histological data that shows fiber disorganization and diminution in the PDL on the side of compression.[86] These changes result in circulatory differences,

which start a cascade of molecular events. In instances of light pressure, osteoclasts can be observed actively removing bone on the bone margin adjacent to the PDL, known as frontal resorption. In cases of heavy pressure thrombosis occurs, followed by cell death and the development of a "hyalinization" or a cell free zone.[38, 87] This zone does not allow for the invasion of osteoclasts onto the bone adjacent to the PDL. Therefore osteoclasts must take a more circuitous route and arrive from undamaged areas in the medullary bone removing bone back toward the area of hyalinization. This inefficient form of movement has been termed "undermining resorption".[88, 89] On the tension side, Sandstedt witnessed bone apposition as a result of stress placed on the PDL fibers. The tension causes an increase in cell replication and differentiation of progenitor cells into osteoblasts that lay down the new mineral matrix.[90, 91]

A second theory, known as the piezoelectric theory, of orthodontic tooth movement, was proposed in the early 1960's. Bassett and Becker suggested that electric potentials are generated in stressed tissues as a response to mechanical forces.[92] It has long been understood that crystalline structures, when deformed, produce an electric charge. The piezoelectric theory argues that the deformation of crystalline structures within the bone, such as hydroxyapatite, collagen, and fibrous proteins, also give off a charge when deformed, that in turn stimulates bone formation.[93, 94] Studies by Basset and Becker in 1962, as well as Zengo et al in 1974, show that an electric potential is in fact generated when the bone is stressed. During stress the concave side of bone is associated with an electronegative force favoring osteoblastic bone forming activity, while the convex side is associated with an electropositive force that favors osteoclastic

activity and increased bone resorption.[92, 95] Davidovitch et al. proposed that mechanical and electrical stimuli are interrelated during orthodontic tooth movement. They demonstrated an increase in cellular activity in the alveolar bone and PDL, and an increase in tooth movement, when an externally applied electrical current was combined with orthodontic forces. Their findings suggest that a streaming electrical potential could play a roll in molecular communication during tooth movement.[96, 97]

Although electrical potentials appear to play a role in tooth movement, there are two reasons why the likely source is probably not the piezoelectric force alone. First, the electric potential generated when the crystalline structure of bone is stressed is not continuous. The electric potential increases when the initial force is placed and quickly dissipates. Second, as the force system is removed there is an electrical potential generated that is equal and opposite of the original.[14] If the piezoelectric force were the primary factor, this reversal would negate any changes initially created. In fact, Masella and Chung described the piezoelectric phenomena as brief and effete.[98] They stated that it should not be conflated with the longer lasting, apparently more influential, ion flux that occurs simultaneously in tissue fluid.

Tissue fluid flow is the focal point of another, more progressive explanation for orthodontic tooth movement. Henneman et al. in 2008, described a four-step theoretical model for tooth movement.[99] The first step involves fluid flow in both the alveolar bone and the PDL surrounding the tooth root. This fluid flow is an immediate result of external forces applied to the tooth and results in the second phase, strain of the PDL cells and osteocytes. In the PDL there is both positive strain, tensional deformation due

to a stretching of the collagen fibers, and negative strain, compressive deformation allowing the fibers to become relaxed. Studies by Melsen support the innovative idea that the changes on the side with negative strain are a result of relaxation or unloading of the PDL fibers instead of the classical idea of compression.[100] It is believed that the unloading of the PDL fibers or negative strain reduces the amount of fluid flow in the canaliculi. The canaliculi are tiny channels connecting the lacunae within the bone. The lacunae house osteocytes and when fluid flow to the lacunae is reduced, apoptosis of the osteocytes can occur. Osteocyte apoptosis in turn attracts osteoclasts, which results in bone resorption. Conversely, on the side with positive strain, fluid flow through the canaliculi increases. The increase causes shear stress on the osteocytes resulting in an escalation in their activity.[101-103] The increase or decrease in cellular activity and differentiation makes up the third step in the model and is followed by the fourth step, remodeling.[99]

An alternative theory for apoptosis of osteocytes on the resorption side is based on the principal of microdamage. When an external force is placed on a tooth the force is dispersed throughout the dentoalveolar complex. Baumrind found that bone deflection occurred via forces that were lower than the forces necessary to produce consequential changes in PDL width. He proposed that forces are transmitted to the PDL, bone, and tooth, causing deformation in all three tissues. Deformation of the particular tissue is dependent on its material composition and elastic properties.[90] Muhlemann and Zander,[104] who evaluated Macaca mulatta (rhesus) monkeys, and Grimm,[105] who evaluated 12 year old human patients as well as cadavers, reported

similar findings. Both groups noted significant dentoalveolar displacement when orthodontic forces were placed. It was theorized that tooth displacement leads to material fatigue and microcracks. Verna et al. evaluated buccal movement of the lower right first molar in 25, 3-month-old, male Danish pigs produced with forces of 130 cN.[106] They reported microcracks on the resorptive side of the alveolus during the initial phase of tooth movement. Microdamage invokes a cellular response that ultimately leads to osteocyte apoptosis.[107] Crack displacements could tear osteocyte cell processes, which directly secrete bioactive molecules capable of triggering an osteoclastic response.[108]

Frost's mechanostat theory provides the most comprehensive explanation of how microstrain affects the bone. According to the mechanostat, bone loss and bone growth are stimulated by the elastic deformation of bone. This is an adaptive process that allows bone to modify its mass and geometry based on the functional needs of the organ.[109] If stress levels are too high, apposition will outpace resorption during remodeling and the bone will become more stress resistant. If stress levels are too low, the available bone strength is not needed and resorption occurs. In vivo studies support this theory and have shown that active bone modeling increases cortical bone mass with local stress that causes microstrains in the range of 1500-3000 με. If, however, the microstrain range is between 100-300 με, the stress is not sufficient to cause adequate strain and remodeling removes cortical-endosteal and trabecular bone mass.[109, 110] Between these two ranges is a microstrain level that allows for remodeling to occur in equilibrium, where the amount of apposition equals the amount of resorption. The bone's adaptive capabilities, however, are finite. Microstrains that are greater than 3000

με are considered pathologic. At this point, microcracks and microdamage begins to accumulate.[109] Beyond this range, the fracture strength is reached and pathologic fracture occurs.[111, 112]

Force Amount and System

The amount of force, as well as the type and direction of the force are critical components of successful tooth movement. Schwarz in 1932 stated that "… biologically the most favorable treatment is that which works with forces not greater than the pressure in the blood capillaries. This pressure in humans as well as in most mammalians is 15-20 mm Hg; about 20-26 g for 1 sq cm of surface." In 1963, Jepsen combined this theory with the average premolar volume to estimate the ideal force at 54cN (~54 grams).[113] Other studies evaluating the optimum force level in relation to rate of movement or efficiency and have come to similar conclusions about the ideal force. Owman-Moll et al. evaluated 32 maxillary premolars after application of a continuous force of either 50cN or 100cN.[114] Their data supports the idea that for tooth movement to occur a threshold force must be met (~50cN). After this threshold is surpassed, increasing the force level does not increase the rate of tooth movement. Ren et al. in 2004 developed a mathematical model to determine the optimum force magnitude for orthodontic tooth movement.[115] Their conclusion was that the higher forces often used in orthodontic practice do not necessarily produce a more efficient tooth movement. Data by Pillon et al. in 1996 further supports this concept.[116] They evaluated rate of tooth

movement with forces of 50, 100, and 200 grams and found no difference. Although research regarding optimal force levels for orthodontic tooth movement is limited,[117] most orthodontist agree that light forces (for premolars circa 50g) are appropriate.[118] However, the quantity of force is not the only significant factor in the force system.

As early as 1985, Quinn et al. indicated that after the threshold level is reached, the amount of force is not as important as the type and location of the force.[64] Quinn et al. described the stress/strain model in relation to force magnitude and its location on the teeth.[64] They explained that similar force magnitudes do not necessarily result in similar stress/strain patterns. They showed that tipping produces high compressive forces concentrated in the cervical and apical thirds of the tooth root. Even when a relatively light external force is exerted on the crown of a tooth, if it is concentrated in a relatively small area on the root surface, it could exceed physiologically appropriate levels. The result can be detrimental to both the tooth and the surrounding tissue.

This suggests that the critical component during orthodontic expansion may be the control of torque and buccal tipping. If the forces can be distributed over the entire buccal surface of the root, instead of being concentrated at the crestal region, the adverse effects of orthodontic expansion could be minimized. True translational forces could potentially stimulate bony apposition along the entire buccal cortical plate lateral to the tooth root. The experimental work of Kraus[61] and Ruso[119] support this concept. Their histological evaluations of teeth that had been tipped showed bone loss at the crestal region, where the forces were at their highest level. Apical to this area, where force levels dissipated, osteoblastic activity was noted on the periosteal surface of the buccal

cortical bone while osteoclastic activity was noted on the endosteal side. This pattern continued apically to a reversal line, where the endosteal activity transitioned into bony apposition. This reversal line coincided with the center of rotation of the tooth, delineating the area of the tooth that moved buccally from the area that moved palatally.[61, 120] The osteoblastic activity that was seen proves that bony apposition on the buccal cortex is possible during lateral tooth movement. The areas of resorption observed stress the critical role of torque control. Controlling the torque however, is more difficult clinically than it appears conceptually.

Numerous studies have evaluated the type of force necessary to create translational or bodily tooth movements.[121-123] If the force is applied at the level of the bracket an appropriate moment needs to be created to move the center of rotation to an infinite position beyond the apex. The moment to force ratio required for translational movement is 10:1. To obtain such a moment in the straight wire system one must utilize a rectangular archwire that fills a substantial portion of the bracket slot. The problem is that the light flexible wires that have been developed to provide appropriate forces over a large range do not provide the adequate moment to counteract the force produced. The resulting forces lead to uncontrolled tipping.

Cortical Plate Encroachment During Tooth Movement

In 1982, Karring et al. performed a study on a beagle dog model to evaluate the effect of facial tipping on the maxillary second and third incisors.[124] Orthodontic appliances were used to tip the incisors on the left side of the maxilla in a facial direction through the alveolar bone plate. After five months of this movement the appliance was reversed so that the teeth were brought back to their original position over the subsequent five-month time period. At the same time the incisors on the right side of the mouth were tipped out to a position corresponding to that attained on the left side. Both sides were retained in these positions for 5 months at which time the animals were sacrificed. Meticulous care was taken so that all teeth during the study were free of plaque and gingival inflammation. In all dogs, including the controls, the apical termination of the junctional epithelium corresponded to the cement-enamel junction (CEJ), indicating that soft tissue migration was not evident. The average distance between the CEJ and bone crest in the control group was 2.2 mm ($\pm$ 0.5 mm). The average distance between CEJ and bone crest in the test group in which the incisors were retained in a tipped position was 4.1 mm ($\pm$ 2.1 mm) and in the group in which the incisors were moved back after tipping the measurement was 1.8 mm ($\pm$ 0.4 mm). Although the angulation of tip and the amount of force placed on the incisors was not recorded, the study highlights two important issues. First, a dehiscence can be produced in the alveolar bone by tipping the teeth in a facial direction. Second, bone has the

capacity to regenerate in such defects when the teeth are moved back to their original position.

Steiner et al. in 1981 used a monkey model to evaluate dehiscence formation during labial tooth movement.[125] They moved the central incisors of five Macaca nemitrina monkeys a mean distance of 3.05 mm over 13 weeks. This movement was accomplished through the use of the edgewise appliance and a 0.016 in. by 0.022 in elgiloy wire delivering approximately 50 grams of force. Afterwards, the teeth and surrounding tissues were evaluated with periodontal flap surgery. Their results indicated that there was a significant amount of recession at the gingival margin, connective tissue level, and marginal bone height. Eight months later, Engelking and Zachrisson took the same animals and moved the teeth lingually back into position with fixed appliances.[126] The incisors were retracted a mean distance of 1.8 mm and then retained for 5 months. The animals were sacrificed and clinical and histological examinations were performed. The marginal bone levels recovered, relative to their original levels, an average 2.5 mm and 3.1 mm for the maxillary incisors and mandibular incisors, respectively. Bone histomorphometric analysis with tetracycline labels demonstrated significant osteogenesis in the periodontium of the retracted teeth. This study supports the findings of Karring et al that labial tooth movement can create a dehiscence and that the defect can be repaired if the tooth is moved back into position.[124]

Thilander et al. performed a study similar to that of Karring and coworkers with similar results.[127] They postulated that, even though bone loss was observed as the tooth tips labially, a bone matrix remained in the soft tissue.[128] This bone matrix maintains the

capacity to remineralize following repositioning of the tooth back into the alveolar process. They further speculated that the dimensions of the alveolar process might be under a genetic control or at least have some limitation or boundaries within the skeletal housing. They supported this hypothesis with the findings of a study performed by Lindskog-Stokland et al. in 1993.[129] This group utilized a beagle dog model to evaluate the dentoalveolar tissues after mesio-distal movement of premolars into areas with markedly reduced bone height. The tooth was moved bodily toward the area of reduced bone (half the height of the premolar root) with light forces for a period of 6 months. After movement, the tooth was retained for 2 months before biopsies were sampled. The results showed that that none of the teeth experienced loss of connective tissue attachment. The CEJ to bone margin distance was greater on the pressure side of the teeth that were moved into the defect compared to the contralateral control side. However, the bone level was significantly more coronal than the reduced bone level in to which the teeth were moved. Thilander and colleagues argued that Lindskog-Stodland et al. were able to show bone growth because their movement was confined to the established boundaries of the jaw. They also discussed the use of light forces. They suggested that the light forces utilized by Lindskog-Stodland et al. might allow for only the inorganic component of the alveolar bone to be lost. Since the organic component was maintained, it is likely to result in remineralization of the bone.[128] Thilander and coworkers pointed to the type of movement that occurred as a significant difference between studies. The studies on labial movement showed significant tipping while the study on mesio-distal movement showed bodily movement. As previously discussed,

the two movements create entirely different stresses and strains on the bony interface between the tooth root and surrounding bone. Although Thilander and coworkers mentioned these differences, they did not propose them as the reason for bone loss in lateral movement instead of the postulated genetic boundary of the jaw.

Mesio-Distal vs. Lateral Tooth Movement

Lateral tooth movements are significantly different than mesio-distal tooth movements. Disparities are seen in both the biology of the movements and the mechanics behind the movements. During normal mesio-distal tooth movements, the tooth root travels briefly through the cribiform plate and then exclusively through medullary bone. During lateral movements, the tooth travels through the cribiform plate and then a very short distance through medullary bone, if any at all, before contacting the cortical bone of the buccal plate. As mentioned when discussing dentoalveolar anatomy, trabecular bone and cortical bone have significant remodeling differences. Medullary bone has a rich vascular supply and is less dense than cortical bone. In fact, cortical bone derives the majority of its nutrients from the overlying periosteum. These differences allow a tooth root covered in very dense cementum to move through medullary bone with minimal resorption. The honeycomb nature of medullary bone allows for the BMU to act on a resorptive front easing tooth movement. When the tooth root contacts the buccal plate in lateral movement the response from the cortical bone is much different. The increased density of the buccal cortex creates more resistance to

movement. The response to the strain on the cortical plate is not fully understood. However, it is known that the BMU essential for remodeling of the cortex are only present in the periosteum and underlying medullary bone and must enter the cortical bone through cutting cones.

Typical mechanics and orthodontic systems for mesio-distal movements differ significantly from that of lateral or transverse movements. Although both forces are usually directed through the same point on the tooth, they are not the same. During mesio-distal and lateral tooth movement the force, typically at the bracket, is located above the center of resistance. Since the force is away from the center of resistance it will create a rotational movement, or moment, in the same direction of the force. The tooth will tip in this direction until the wire engages in the bracket slot and creates a separate force, known as a couple, that counteracts the tipping. This couple provides the moment, or rotational force, necessary to counteract the moment created from applying a force above the center of resistance. During mesio-distal tooth movement this couple is created by the width of the bracket slot. In lateral movement, the depth of the slot creates the counteracting moment. Although brackets slots vary in width most have a depth of 0.028 inches. Often the width is greater than three times the depth, which creates a large advantage in producing the counteracting moment. Consequently, it is much more difficult to control tipping during lateral movements than during mesio-distal movements.

Cemental Resorption

In 1927 Ketcham first identified root resorption as a significant problem related to orthodontic tooth movement.[130] Although there have been significant improvements in orthodontic treatment since 1927, cemental resorption remains a significant problem. In 1991 Linge and coworkers evaluated standardized intraoral radiographs of 485 consecutively treated orthodontic patients.[131] They found that 16.5% of the patients had loss of root length of more than 2.5 mm in one or more maxillary incisors. Makedonas et al. in 2013 utilized cone beam computed tomography to evaluate 156 patients treated with fixed appliances and four first premolar extractions.[132] They found severe root resorption, greater than 2 mm, in 25.6% of the patient population.

A variety of factors have been indicated as the cause of resorption during tooth movement. Linge et al. used a correlation matrix to evaluate their data and found several risk factors associated with resorption including: history of trauma, overjet, time of treatment in rectangular wires, time of treatment with class II elastics, lip/tongue dysfunctions, impacted cuspids, and history of finger habits persisting beyond the age of 7. Kaley and Phillips attempted to identify more factors in 1991, when they evaluated 200 consecutively debonded patients treated with the edgewise appliance.[133] They found approximation of the maxillary incisor to the cortical plate (odds ratio of 20), maxillary surgery (odds ratio of 8), and root torque (odds ratio of 8.5) to be significant factors related to root resorption. Although there is some debate as to the predisposing factors the literature is clear that comprehensive orthodontic treatment causes root resorption.[134]

The literature also suggests that root resorption follows a pattern that mirrors the force system affecting the tooth. Casa et al. in 2001 investigated the effects of lingual root torque on premolars with scanning electron microscopy.[135] They observed resorption on the lingual side at the apex as well as the buccal side at the cervical region. The areas of resorption corresponded to the regions of compression. Owman-Moll and colleagues noted a similar effect and pattern when evaluating tipping movements of human premolars.[136] As one would expect during buccal tipping, the forces are greatest at the cervical region on the buccal and the apical region on the lingual. Owman-Moll and colleagues found that this is precisely where the majority of resorption was found. The link between areas of compression and areas of cemental resorption has been supported in human and animal models, with continuous and intermittent forces, and with conventional orthodontics as well as clear aligners.[61, 137-139]

Although resorption appears at any point of compression between the root and alveolus, the magnitude and duration of the force appear to play a significant role in the extent of resorption. Casa et al., in the study mentioned previously, showed with electron microscopy that resorptive lacunae increased in severity with increases in force magnitude and duration.[135] In 2006 the same group used histochemistry to visualize osteoclastic cells on the cemental surface.[140] Their findings, as well as those of numerous other studies, support the positive relationship between resorption and force magnitude and duration.[141-143]

In 2010 Weltman and colleagues performed a systematic literature review of root resorption associated with comprehensive orthodontic tooth movement.[134] After

evaluating the current literature, they suggested that clinicians should use as light a force as possible during orthodontic movement. This suggestion has merit because the magnitude of force is not merely associated with resorption; it has a biologic cause and effect relationship. Kvam in 1972, and Rygh in 1973, provided a biological explanation for orthodontically induced cemental resorption.[144, 145] Kvam and Rygh showed that root resorption is a side effect of cellular activity associated with the removal of necrotic hyalinized tissue. As mentioned in a previous section, necrotic tissue is created when compression forces within the bone exceed capillary pressure. At this point blood supply is reduced and necrotic tissue begins to accumulate. Brudvik and Rygh in 1995 showed that tartrate-resistant acid phosphatase (TRAP)-positive macrophages and multinucleate giant cells are responsible for the removal of the necrotic hyalinized tissue.[65] TRAP positive cells arrive at the necrotic tissue from the medullary bone opposite of the tooth root. They remove the necrotic tissue until they reach the adjacent root surface. The function of the TRAP positive cells continues when the cemental surface is reached. Subsequently, cementum and often the underlying dentin are removed, producing resorptive lacunae on the root surface. The literature is clear that orthodontic forces will result in root resorption, however with proper control a clinician can minimize the amount of damage created during treatment.

Conclusions

Buccal expansion is a common treatment modality in current orthodontic practice. Expansion is utilized to solve a variety of dental maladies including crowding, aesthetic problems, and functional problems. Although expansion is a common and relatively accepted practice, it has significant side effects.

All current forms of expansion cause some degree of dental tipping. Excessive tipping not only places the tooth in a poor position from a functional and aesthetic perspective, it also decreases stability and jeopardizes the health of the tooth root and surrounding tissues. Uncontrolled tipping concentrates forces in the dentoalveolar complex at the bucco-cervical area as well as the apico-lingual area. The deleterious effects of these forces have been well documented. The two most significant effects are buccal bone loss and an increase in cemental resorption.

Many critics of expansion believe that expansion is synonymous with tipping and cite the deleterious effects of uncontrolled tipping as reasons to avoid expansion entirely. These critics often seek extraction therapy as an alternative to orthodontic expansion. Supporters of buccal expansion either accept tipping as a side effect of treatment or claim that their system is able to control tipping during expansion. However, to date there have been no studies that produced buccal orthodontic expansion with pure translational movement. Our goal is to establish a system that produces buccal expansion free of buccal tipping. In the present study we will utilize light continuous forces with the appropriate torque control to create translational movement in maxillary

first premolars. The effects of this movement on the tooth root as well as the surrounding tissue will be documented with photography, digitized models, cone beam computed tomography, and micro computed tomography. The information that is gathered will provide insight on the true effects of orthodontic expansion free of tipping.

CHAPTER II

BACKGROUND

A tooth size to arch length deficiency is one of the most common problems facing the clinical orthodontist.[6, 146-148] It occurs when the combined mesio-distal width of the teeth exceed the space available in the underlying alveolar bone. To treat such cases, clinicians must either remove tooth structure or increase space with expansion. In recent years, the non-extraction approach has become more popular as new techniques and materials for expansion have been developed.[3, 4]

Despite improvements, expansion continues to have unintended and problematic consequences; one of the most notable is uncontrolled tipping.[58-61, 136, 149] The type of force system necessary to avoid uncontrolled tipping and create translational or bodily tooth movement has been established.[121-123] A force applied at the level of the bracket requires a moment to force ratio of 10:1 to create translation. Although our understanding of optimal force levels for orthodontic tooth movement is limited,[117] most orthodontists agree that light forces (for premolars circa 50g) are appropriate.[113, 118] Considering the 10:1 moment to force ratio, a 50 grams force would require a 500 gram moment to produce translational movement. To obtain such a moment with a straight wire system, a rectangular archwire that fills a substantial portion of the bracket slot must be used. Conversely, the light flexible wires that provide appropriate forces over a large range lack the size necessary to fill the slot and produce an adequate moment. The large archwires that are able to fill the slot create forces well beyond 50-grams.

Therefore, orthodontic expansion is usually accomplished with either an inappropriate force level or, more often, uncontrolled tipping.

Tipping produces highly compressive forces concentrated in the cervical and apical thirds of the tooth root.[64] Even when a relatively light external force is exerted on the crown of a tooth, tipping concentrates the resulting strain onto a relatively small area of the alveolus. Strains that exceed 3000 με are considered pathologic, producing microcracks and microdamage that accumulate and eventually lead to failure.[109] Such damage provides an explanation for the link between dental tipping associated with expansion and alveolar bone loss.[59-61, 125, 150]

This suggests that the critical component during orthodontic expansion may be the control of torque. If the forces can be distributed over the entire buccal surface of the root, instead of being concentrated at the crestal region, the adverse effects of orthodontic expansion could be minimized. True translational forces could potentially stimulate bony apposition along the buccal cortical plate lateral to the tooth root. Recent histological evaluations of teeth that were tipped buccally showed bone loss at the crestal region, where the forces were concentrated.[61, 120] However, periosteal osteoblastic activity was noted on the buccal cortex apical to the dehiscence while osteoclastic activity was noted on the endosteal side. This pattern continued apically to a reversal line, where the endosteal activity transitioned into bony apposition. This reversal line coincided with the tooth's center of rotation, delineating the portion of the tooth that moved buccally from the portion that moved palatally.[61, 120] The osteoblastic activity on the periosteal side demonstrated that bony apposition on the buccal cortex is possible

during lateral tooth movement. The areas of resorption (i.e. dehiscences) stress the critical role of torque control.

The aims of the project were to produce buccal translation of the maxillary first premolars and to determine whether bone forms on the buccal surfaces. It is hypothesized that the negative effects of buccal expansion are the result of tipping rather than expansion. As such, a force system was designed to minimize tipping and produce buccal translation with light, continuous forces.

CHAPTER III

MATERIALS AND METHODS

All of the subjects were patients seeking treatment in the graduate orthodontic clinic of Texas A&M University Baylor College of Dentistry. The project was approved by the institution's IRB and informed consent was obtained from all of the patients. The patients were selected based on 1) having previously accepted a comprehensive orthodontic treatment plan that included the extraction of at least one maxillary first premolar, 2) being between 11 and 17 years of age, 3) having at least one fully erupted maxillary first premolar that was amenable to buccal expansion, and 4) having fully erupted maxillary first molars. Based on estimates of buccal tooth movement,[151] a power analysis indicated that 12 subjects would be needed to establish a difference of 1.2 mm in buccal tooth movement between sides, assuming a power of 0.95, an alpha of 0.05 and a correlation of 0.5. A total of 13 subjects were enrolled in the study. Two of the subjects were not included in the analyses because their premolars had not moved sufficiently to evaluate bony changes.

The remaining 11 patients (5 females and 6 males) ranged between 12.5-16.9 years of age, with a mean age of 14.1 years. Prior to the start of treatment, records were taken, including plaster models, limited field of view cone beam computed tomographic (CBCT) images, and digital photographic images. The CS 9000 3D (Carestream Dental, Atlanta, GA) CBCT unit was chosen based on its small voxel size (0.076 mm, isotropic) and minimal average radiation dose (9.8 μSv).[152] Four images (pre/post and

study/control) were taken on each patient. The maxillary first premolar was centered in the field of view to maximize the accuracy of reconstruction for the volumetric data.[153] The field of view was 3.75 cm x 5.00 cm. Settings for the CBCT images were 70 kV, with 10mA, at 10.8 seconds.

Upon completion of the records each patient was treated with an appliance designed to produce buccal translation of one maxillary first premolar. The experimental side was randomly selected in 10 of the patients. Two of the patients had only one maxillary first premolar planned for extraction. The teeth on the control side were not banded and did not receive any form of treatment.

The appliance was adapted from previous studies investigating the effects of buccal forces on premolars.[118, 136, 138, 143, 151, 154] It was first fabricated on the study models, and consisted of bands on the maxillary first molars and the first premolar (Dentsply GAC, Islandia, NY). A transpalatal arch (0.036 inch stainless steel wire) was soldered to the molar bands to maintain molar position and provide a framework for a bite plane made with Triad acrylic (Dentsply GAC, Islandia, NY) (Figure 1-A). On the facial surface of the premolar band, a 0.040 inch stainless steel wire was soldered to serve as a power arm (Figure 1-B). The solder joint was positioned so that the point of attachment was in the cervical third of the premolar, which is thought to aid in the production of bodily tooth movement when utilizing a power arm.[155] The power arm extended in to the vestibule to the premolar's center of resistance, which was estimated to be 40% from the apex, measured between the alveolar crest and the apex of the root (Figure 1-C).[42] This distance was calculated from the CBCT image to be 17.4 mm (IQR

15.0 to 18.0) from the buccal cusp tip. Due to limitations in vestibular depth the actual power arm distance was 16.7 mm (IQR 13.9 to 17.7) from the buccal cusp tip.

The bands, the cantilever on the premolar and the transpalatal arch were then transferred to the patient and bonded using a dual cured, resin modified, glass ionomer cement (Reliance Orthodontics, Itasca, IL). The occlusal bite plane was checked prior to and after cementation to ensure appropriate bite opening. Triad acrylic (Dentslply GAC, Islandia NY) was added to, or removed from the bite plane so that the first premolar was free of interferences during buccal movements. The palatal premolar cusp was smoothed to ensure unimpeded tooth movements.

A 50 gram force was applied to the maxillary first premolar on the experimental side with a b-titanium alloy 0.021 in x 0.025 inch sectional wire (3M Unitek, Monrovia, CA), anchored in the auxiliary slot on the first molar band (Figure 1-D). The wire was bent vertically so that the point of attachment on the power arm was located at the estimated center of resistance (Figure 1-B and C). It was bent buccally to create a 50 g lateral force (Figure 1-D). The force was verified with a Correx (Haag-Streit, Berne, Switzerland) gram force strain gauge. The activated b-titanium wire was ligated to the premolar cantilever with a 0.0010 inch stainless steel ligature tie (Figure 1-B and C). A small amount of flowable composite (3M ESPE, Irvine, CA) was placed over the ligature to minimize vestibular irritation.

The buccal force was checked and reactivated to 50 grams every three weeks. After six weeks a second set of study models was taken and compared to the pretreated models to determine first premolar movement. A second evaluator verified all

measurements. Two millimeters of movement was confirmed in two patients and a 0.021 in x 0.025 in stainless steel wire was bent to passively hold the teeth in the new position. This force was approximately 10-15 grams, similar to the forces exerted by the buccal musculature after 2 mm of lateral tooth movement.[156, 157] The passive force was held for three weeks to allow the surrounding tissue to mature.

In the remaining 11 subjects, 2 mm of movement had not occurred after the initial six weeks. For them, the 50-gram force was maintained for an additional three weeks, after which their teeth were held passively for three additional weeks, as previously described. Active tooth movement did not exceed nine weeks for any patient, regardless of the amount of tooth movement present. The nine-week cut off was established so that patients could proceed with their scheduled orthodontic treatments.

After the three-week maturation period the appliance was removed and post treatment records were taken, including plaster models, limited field CBCT images, and digital photographs. Table 2 provides the average duration and variability for each phase of treatment.

Evaluations

The pre- and post-treatment plaster study models were digitally scanned using an Ortho Insight 3D model scanner (Motion View Systems, Hixson, TN) and evaluated using the Motion View Software (Motion View Software, Hixson, TN). Four width measurements were taken to evaluate the amount of buccal movement of the first

premolars and first molars. Two bilateral width measurements were taken between the buccal and palatal cusp tips of the first premolars (Figure 2). Molar width measurements were taken between the mesial buccal cusp tips and central fossae (Figure 2). Each measurement was taken twice by a single blinded investigator and averaged. Replicate analyses of seven randomly selected sets of models showed interclass correlations of 0.98 ($p<0.001$) and 0.94 ($p<0.001$) for the inter-palatal cusp and inter-buccal cusp measurements, respectively. The interclass correlations for the maxillary molar were 0.43 ($p=0.19$) for the measurements taken between the central fossae was and 0.34 ($p=0.25$) for those taken between the mesial buccal cusps.

The amount of tipping of the experimental premolar was evaluated on the digital models using the Motion View Software (Figure 3). The angulation of the experimental premolar was measured from three points, including the cervical of the control premolar on the palatal side, the cervical of the experimental premolar on the palatal side, and the palatal cusp of the experimental premolar. All measurements were taken twice by a single investigator and averaged. Based on replicate analysis of five randomly selected sets of digital models, the interclass correlation for the tipping was 0.88 ($p=0.03$).

Tooth movements were also evaluated on the limited field of view CBCT images using the Kodak 3D imaging software (Kodak Carestream, Atlanta, GA). To evaluate buccal bone height and thickness the images were oriented so that all three planes of space bisected the first premolar, as previously described.[158] Three buccal bone thickness measurements were taken at the mesio-distal midpoint of the first premolar, including maximum buccal bone thickness, minimum buccal bone thickness, and buccal

bone thickness 3 mm apical to the CEJ (Figure 4). All thickness measurements were taken as the shortest distance from the buccal surface of the tooth root to the buccal surface of the buccal cortex. Two height measurements were taken from the cemento-enamel junction (CEJ) to the crestal bone. They were taken as the investigator moved through the coronal slices from the mesial to the distal aspect of the premolar. The smallest and largest measurements were recorded, representing the maximum and minimum distance of the crestal bone to the CEJ. A single, blinded investigator performed each measurement twice, and the measurements were averaged. Six randomly selected CBCT images were evaluated a second time to estimate reliability. Interclass correlations ranged from 0.92 (minimum height from CEJ to crestal bone) to 0.99 (maximum height from CEJ to crestal bone).

Pre and post movement CBCT images were superimposed with Invivo5 software (Anatomage, San Jose, CA) (Figures 5, 6, and 7). A voxel superimposition was performed aligning the data to best fit the regions that exhibited stability over the treatment period. Thickness measurements were taken as previously described to estimate changes in root position and in buccal bone thickness (Figure 4). Both measurements were taken 3 mm apical to the cemento-enamel junction at the buccal surface of the cementum and the buccal cortical plate, respectively. Measurements were taken three times by a single investigator and changes in buccal bone thickness were averaged. The interclass correlations for root movement and buccal bone thickness were 0.95 and 0.99, respectively.

Change in buccal bone thickness was also calculated indirectly using the following formula: bone change = [final buccal bone thickness – (initial buccal bone thickness – root movement)]. Bone thickness was derived from the CBCT measurements, while root movement was derived from the superimpositions.

Statistical Analysis

SPSS version 22 (SPSS Inc., Chicago IL) was used to analyze the data. The skewness and kurtosis statistics indicated that the distributions were not normal. As such, the central tendencies and dispersions were described with medians and inter-quartile ranges. Single-sample Wilcoxon Signed Rank Tests were used to determine whether statistically significant changes had occurred. Wilcoxon Signed Rank Tests were used to compare the control and experimental side measurements, as well as to compare changes in buccal bone and tooth movement evaluated from the superimposed CBCT images.

CHAPTER IV

RESULTS

Of the two patients who were dropped from the study, one (#9) had interproximal contacts that impeded premolar movement in the buccal direction (Table 3). The second patient (#10) had greater than 2 mm of movement, but the tooth relapsed almost the entire 2 mm distance during the three-week maturation period. One of the patients (#8), who had active orthodontic treatment performed on his maxillary central and lateral incisors, was not included in the superimpositions because movements of these four teeth made it impossible to accurately superimpose the pre and post movement CBCTs. Therefore, superimpositions were performed on 10 patients.

At the end of the first three-week time period, the active force had dissipated from 50 grams to 40.4 $\pm$ 4.9 grams. After the second and third three-week time periods, the force had dissipated from 50 grams to 41.4 $\pm$ 4.6 grams and 43.1 $\pm$ 4.2 grams, respectively.

Model Analyses

There were statistically significant movements of both the experimental premolars and the molars (Figure 8). The inter-premolar distance measured from the lingual and buccal cusp tips increased 1.56 mm (IQR 1.38 to 2.52 mm, p=0.003) and 1.82 mm (IQR 1.48 to 2.61 mm, p=0.003), respectively. Inter-molar widths increased

0.85 mm (IQR 0.08 to 1.32 mm, p=0.008) between the mesio-buccal cusp tips and 0.55 mm (IQR 0.08 to 0.83, p=0.01) between the central fossae.

Comparisons of the pre-and post treatment models showed slight but significant (p=0.003) buccal crown tip of the experimental first premolar. It tipped approximately 2.2 degrees (IQR 1.26 to 4.01 degrees), with a range of 0.95 to 5.37 degrees (Table 4).

CBCT Radiographic Analysis

Buccal bone thickness showed significant ($p < 0.05$) reductions on the experimental, but not on the control side (Figure 9). The maximum thickness of the buccal bone on the experimental side decreased 0.45 mm (IQR 0.20 to 0.60 mm, p=0.006), the minimum thickness decreased 0.35 mm (IQR 0.15 to0.43 mm, p=0.004), and bone thickness 3 mm from the CEJ decreased 0.63 mm (IQR 0.17 to 0.79 mm, p=0.008)(Table 5).

The maximum vertical distance measured from the cemento-enamel junction to the crestal bone increased 0.60 mm (IQR 0.40 to 8.30 mm) on the experimental side and showed no significant changes on the control side (Figure 10). The difference between sides was highly significant (p=0.002)(Table 5). The maximum vertical distances exhibited a bimodal distribution. Six subjects had a median loss of 0.42 mm (IQR -0.25 to 0.53 mm) while 5 subjects had a median loss of 8.54 mm (IQR 7.15 to 10.05 mm) (Figure 11).

The minimum distance measured from the cemento-enamel junction to the crestal bone on the experimental side increased 0.25 mm (IQR 0.10 to 0.55 mm), but this increase was not significantly greater than the -0.05 mm (IQR -0.40 to 0.20 mm) change measured on the control side (p=0.262) (Table 5).

Analysis of 3 Dimensional Superimpositions

Movements of the experimental first premolar measured from the superimposed CBCT images were also statistically significant (p=0.008). At the level 3 mm apical to the cemento-enamel junction, the root moved 0.96 mm (IQR 0.65 to 1.30) on the experimental side. Premolar movement on the control side was minimal (-0.03 mm; IQR -.06 to 0.08) and not statistically significant (p=0.95) (Table 4).

Direct measurement of buccal bone growth, at a level 3 mm below the cemento-enamel junction, showed median increases of 0.46 mm (IQR 0.29 to 0.94), which was statistically significant (p= 0.005). All of the patients added bone 3 mm below the CEJ at the midline (Figure 12). The median growth indirectly measured was 0.51 mm (IQR -0.4 to 1.0), which was also statistically significant (p= 0.036) (Table 4). The difference between the direct and indirect measurements was not statistically significant (p=0.767).

There was a negative correlation (-0.674, p=0.033) between the initial buccal bone thickness measured on the CBCT images and the amount of bone growth measured from the superimpositions. Also, there was no significant correlation found between the initial and final thickness of bone (0.202, p=0.551).

CHAPTER V

DISCUSSION

Lateral movements of the maxillary first premolar were produced with the application of light continuous buccal force near the center of resistance. Although no optimal force level for efficient orthodontic tooth movement has been established,[117] 50 grams has been accepted as an appropriate force for both bodily and buccal tooth movements.[116, 159]

The rates of tooth movement were slightly less than previously reported, and they decreased over time. The appliance produced 1.6 and 1.8 mm of movement at the lingual and buccal premolar cusps, respectively, after nine weeks. Another human study using a similar appliance design with a 50-gram buccal force located at the level of the bracket produced 3.7 mm of buccal premolar movement after seven weeks.[151] However, they demonstrated over 12 degrees of uncontrolled tipping. Mesiodistal tooth movements occur at rates of approximately 1 mm per month.[160-164] The center of resistance during mesiodistal movement is located more coronal than the center of resistance during buccal movement.[165] Therefore studies evaluating mesiodistal movements likely have less tipping and are more comparable to the present study. The slightly lesser rate between mesiodistal movements and the present study could be due to the buccal cortex, which might be expected to respond differently to forces than medullary bone. Since cortical bone is denser, one would expect it to remodel at a

slower rate. Therefore, buccal tooth movements might be expected to be slower than mesiodistal movements.

The reduced rate of movement observed in the present study could also have been due to relapse during the "passive" force phase, which would have negated some of the buccal tooth movement (Figure 13). Although the amount of relapse was not recorded, the photographic images clearly show palatal tooth movement between the end of active forces and removal of the appliance. Based on the paucity of literature describing buccal forces,[156, 157] it was estimated that a 10-15 gram force was sufficient to create a passive environment and allow for bone maturation. In retrospect, a 15-20 gram force may have been necessary to ensure stability during the maturational phase.

It is clinically possible to produce lateral translation with minimal tipping. In the present study, there was 2.2 degrees of buccal crown tip. Similar force loads located at the middle of the crown (level of the bracket) have shown significantly more tipping.[59] For example, Cattaneo et al.[60] reported 11.7 and 13.5 degrees of buccal tipping in the first and second premolars, respectively; Lundgren et al. noted 12.6 degrees of tipping when applying a buccal force to first premolars;[151] and Weiland found 9.3 degrees using super-elastic archwires.[166] By moving the force apically toward the center of resistance the amount of tipping was reduced 5-8 fold, in the present study, with movements approaching pure translation.

Although tipping was significantly reduced, it was not entirely negated. It is possible, due to the depth of the vestibule, that the force was not placed apically enough. The center of resistance of single rooted teeth has been estimated to be 24% to 60% of

the distance between the root tip and the alveolar crest.[42, 122, 167-169] Meyer et al. found that the center of resistance was located more apically with buccal-lingual than mesio-distal movements.[165] Since the maxillary first premolar is usually bifurcated, it was estimated that the center of resistance was located 60% of the distance between the tooth root tip and alveolar crest, approximately 17.4 mm from the buccal cusp tip. After adjusting for soft tissue limitations, the power arm length in the present study was 16.7 mm from the buccal cusp tip. Although this discrepancy was small and not statistically significant (p=0.445), moving the force any distance from the center of resistance could have produced some tipping.

Perhaps most importantly it is possible to form buccal cortical bone during lateral tooth movements. The superimpositions in the present study showed approximately 0.5 mm of buccal bone growth. The initial buccal bone thickness 3 mm below the CEJ on the experimental side was 1.4 mm. At the same location the tooth root moved 0.96 mm. If no osseous changes had occurred, then the final buccal bone thickness should have been 0.44 mm. However, the final thickness was 0.85 mm, which was twice the expected thickness. The difference (0.51 mm) is consistent with the amount of growth observed on the superimpositions. Direct measurement at the midline of the tooth on the CBCT superimposition showed that all teeth had a measurable amount of buccal bone growth (Figure 12). Sarikaya et al.[170], who evaluated lingual orthodontic tooth movement in cases with four premolar extractions, noted that either some bony apposition or plastic deformation of the cortical plate had occurred. The mechanism for such cortical changes may be the strain created by tooth movement. In vivo studies have

shown that cortical bone mass increases when strain increases.[109-111] Evaluations of the dental-alveolar complex have shown that increasing stress levels increases the thickness of the alveolar cortex.[171-174] Histologic examinations of the buccal cortex after lateral tooth movement have revealed the presence of osteoblastic activity and new bone on the buccal cortex.[61] The present study is among the first to demonstrate apposition of bone on the buccal cortex of patients.

Cortical growth appears to occur when the tooth root approaches the cortical plate. In the present study it appeared that the tooth root moved buccally through the medullary bone until it reached the cortical plate, at which point bone growth or apposition at the cortical plate occurred. This contention is based on the negative correlation (-0.674 p=0.033) observed between the initial buccal bone thickness and the amount of bone growth. Such a relationship indicates that there was less buccal bone growth in patients with greater amounts of initial buccal bone (trabecular and cortical), probably because the tooth had to travel further before encountering the cortical plate. As expected, there was no correlation (0.202 p=0.551) between the initial and final thickness of the buccal bone. The literature supports the idea of tooth movements through medullary bone,[77, 99] with the movements having little effect on the alveolar width until the tooth approaches the cortex.[175] While there are no studies investigating the amount of strain on the buccal cortex produced with orthodontic expansion, it appears that the effects are limited to the PDL and immediately adjacent bone. Grimm suggests that a force as light as 50 grams may initially create measurable bone strain of less than 5 microns of deformation.[105] Finite element analyses indicate that a buccal,

translational force of 50 cN produces a light uniform distribution of forces in the periodontal ligament on the compressive side of the tooth.[176] These forces are even further decreased in the adjacent alveolar bone, attaining maximum values of approximately 0.001 mPa (~1 gram/mm^2). A force this small will likely affect the cortex only when the tooth root is in close proximity. We estimate this distance to be less than 1 mm, since buccal bone thickness in the present study, after tooth movement, 3 mm below the CEJ was 0.86 mm (IQR 0.73-0.96) (Table 6).

Cone beam computed tomographic imaging is a reliable method for evaluating hard tissue changes of the dentoalveolar complex.[153, 158, 177, 178] When compared to caliper measurements, no differences have been reported.[153, 158, 179] However there are limitations with CBCT images, due to voxel size and the partial volume averaging effect.[177, 180, 181] When a voxel lies on two objects of different densities, the resulting voxel will reflect the average density of both objects, rather than the actual density of either object. Therefore when the alveolar bone thickness or periodontal ligament space is below or equal to the voxel size, it will assume the average density of the alveolar bone and the periodontal ligament. This averaging effect causes thin layers of bone to become indistinguishable from the surrounding tissues. As such, bone height and thickness can often be underestimated, making it falsely appear as though there is bone loss.[177, 182, 183] With a voxel sizes of 0.38 to 0.40 mm, a minimum thickness of 0.6 mm of alveolar bone is required for distinguishing the bone from the cementum.[177, 184] When the voxel size decreases from 0.4 to 0.25 mm, there is a significant increase in the accuracy of linear alveolar measurements.[177] In addition to voxel size, milliamperage

and the capture time settings play a critical role in image quality.[185] In the present study, image quality and spatial resolution were maximized by using a small field of view (FOV) scan at a voxel size of 0.076 mm, current at 10 mA and exposure time at 10.8 seconds.

While it is clear that bony cortical apposition is possible during buccal translation, many unknowns remain. Five subjects in the present study exhibited significant vertical defects (dehiscences) in the buccal bone mesial to the midline. The maximum vertical bone heights exhibited a bimodal distribution, (Figure 11) with two distinct clusters of individuals showing either 0.42 mm (IQR -0.25 to 0.53 mm) or 8.30 mm (IQR 7.15 to 10.05 mm) of vertical bone loss. In other words, some patients had clinically insignificant amounts of bone loss, while others had dehiscences extending along the entire root. Similar differences were noted in a study evaluating movements toward the lingual cortex.[170] It is also worth noting that the dehiscences were typically located mesial to the midline; the bone distal to the midline showed very little vertical change. While the samples were small, there were no differences in tipping or in the amount of tooth movement between patients with and without vertical bone loss. However, the group showing dehiscences had an initial buccal bone thickness of 0.90 mm, while the group without dehiscences had a thickness of 1.45. The lack of power best explains the lack of statistical significant differences.

The appearance of dehiscences may have been a result of the teeth moving through the buccal cortex. This assumes that apposition did not occur on the periosteal surface of the buccal cortex, or that it occurred at a slower rate than the resorption

associated with the tooth movement. As such, the root would eventually reach the buccal limit of the cortex and present as a dehiscence. The location of the dehiscences lends support to the idea that the rate of tooth movement can surpass the rate of bony apposition. In the present study the mesial and distal halves of the experimental premolar did not move at the same rate. A cantilever originating from the first molar generated the force responsible for buccal movement. The experimental premolar therefore, moved laterally along an arc (Figure 14). We attempted to mitigate the effects of the arc by extending the cantilever from the distal aspect of the molar rather than the mesial, thus increasing the length or radius and decreasing the angle of the arc. However, a slight mesial to buccal rotation was observed and as a result the mesial aspect of the experimental premolars moved further and at a faster rate than the distal aspect. This difference could explain the presence of a dehiscence on the mesial aspect of the 5 subjects.

It is also possible that the bone was actually present, but not evident on the CBCT images. As mentioned previously, the partial volume averaging effect can make two separate objects indiscernible. In order for both objects to be apparent they must be separated by more than two voxels.[186] Since the voxel size was 0.076 mm in the present study, the alveolar buccal bone would not have been evident if it was less than 0.152 mm thick, or closer than 0.152 mm to the cemental surface. Also, for two objects to be discernable on radiographic images, there must be a 40-60% difference in mineral density between them.[187] During tooth movement woven bone is formed in the direction of displacement.[100] In addition, tooth movements produce a regional acceleratory

phenomenon (RAP),[100, 188] which causes a loss in mineralization. If the alveolar cortex approached the density of the surrounding soft tissue, it would have become indistinguishable. Interestingly, alveolar bone becomes radiographically apparent 6-24 months after treatment, not because of latent growth or healing, but because of the RAP effect and the decreased density of the alveolar bone associated with tooth movement.[189] Since the final CBCT images were taken three weeks after tooth movement was discontinued, it is possible that there was not enough time for mineralization of newly formed bone to have taken place in the present study.

One significant limitation of the current study was the inability to perform histological evaluations. Histology was not performed because removal of the buccal cortex during premolar extraction could have produced a bony defect. Although such a defect might be expected to resolve during space closure, it was considered to be an unjustifiable risk. While CBCT imaging has proven to be a reliable method of assessing the tooth root and surrounding hard tissue, it does not allow the investigator to confirm the presence of osteogenic activity. Future studies should use a similar appliance in an animal model in order to better understand the effects of buccal translation at the cellular level. Also, a longer maturational period would have allowed for better radiographic visualization of any bone that may have been present.

Orthodontic buccal expansion cannot be justified based solely on the findings of the present study. Although the present study showed that it is possible to design a force system that will translate teeth and stimulate buccal bone formation, the limitations and long-term effects of such movement were not addressed. Until it can be confirmed that

the dehiscences we observed can recover, or are really covered by bone, this approach should not be utilized clinically.

Furthermore, the present study did not take into account the stability of orthodontic expansion. While orthopedic expansion is relatively stable,[30, 190-194] the stability of orthodontic expansion with translation is unknown. If the tooth is not stable after buccal translation, then the ability to stimulate buccal bone growth becomes inconsequential. Future studies are needed to ensure that clinicians properly understand and manage this critical component of orthodontic treatment.

CHAPTER VI

CONCLUSIONS

The present study found that clinically significant amounts of lateral translation can be obtained over time with minimal tipping. The rates of lateral, translational, tooth movement against the buccal cortex were slower than previously reported mesiodistal tooth movement through medullary bone. During lateral movements, formation of buccal bone does occur. However, cortical bone growth with lateral translation has limitations that are not fully understood and there is a possibility of creating vertical bony defects.

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APPENDIX A

FIGURES

Figure 1. Photographic images of the appliance: **A.** Occlusal view of the supporting trans-palatal arch with acrylic bite plane. (Note no forces on the control premolar.) **B.** Buccal view of cantilever arm and attachment to premolar at the center of resistance. **C.** Frontal view of the lever arm soldered to the study premolar band and the attachment of the cantilever. **D.** Occlusal view of active cantilever arm attached to the study premolar.

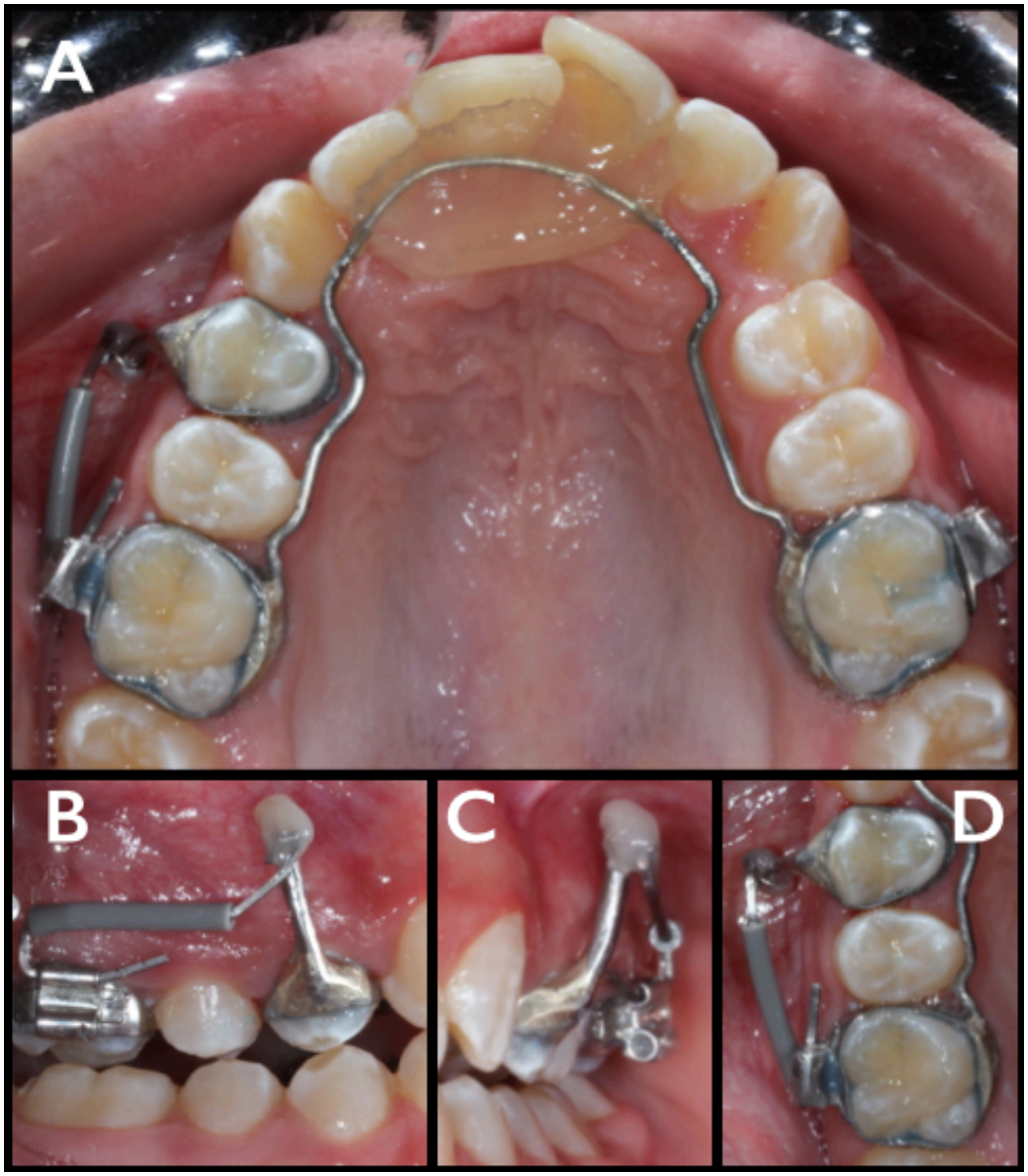


Figure 2. Measurements taken from the digital models to evaluate premolar and molar movement: The inter-premolar distance was measured between the buccal cusp tips as well as the palatal cusp tips. The inter-molar distance was measured between the mesiobuccal cusp tips as well as the central fossae.

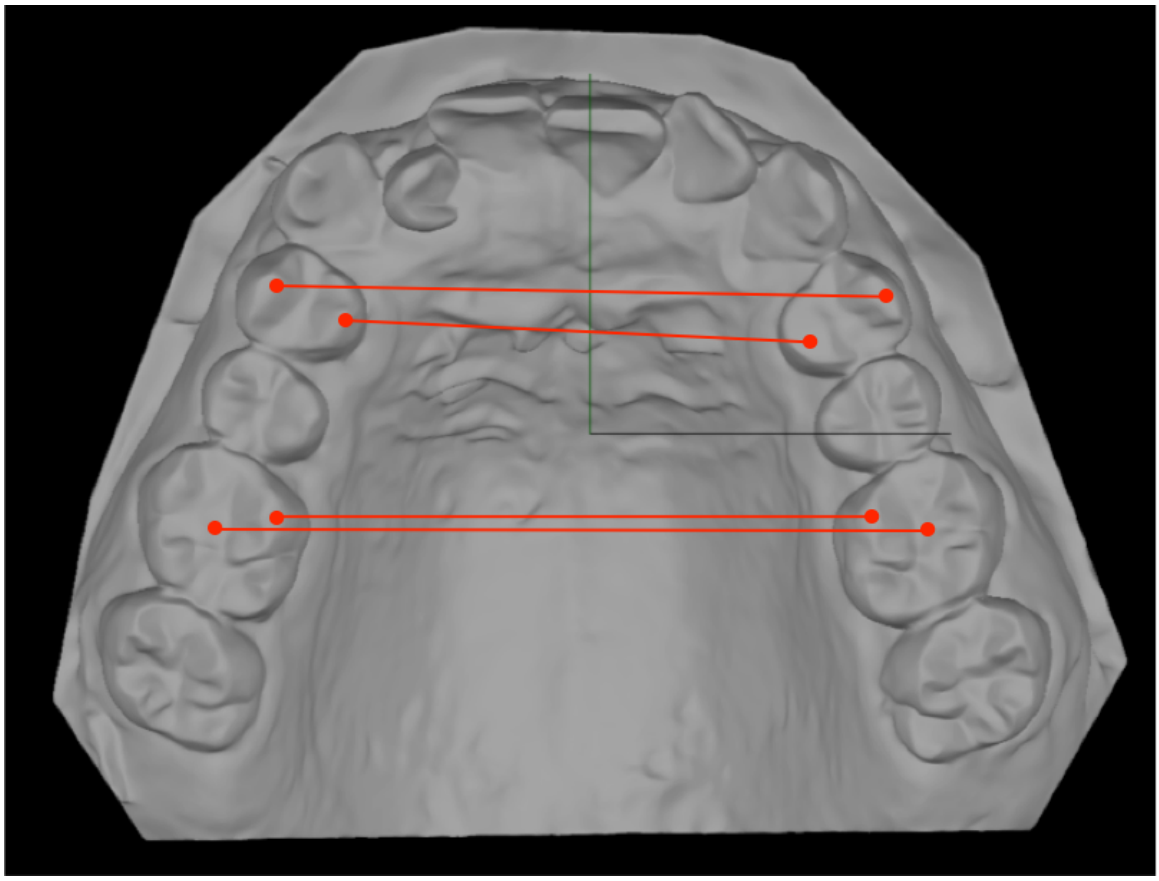


Figure 3. Measurement taken from the digital model to evaluate the amount of tipping in the first bicuspid: The angle was measured at the midline from the experimental first premolar cusp tip, to the cervical margin on t he palatal side of the experimental first premolar, and then the cervical margin on the palatal side of the control first premolar.

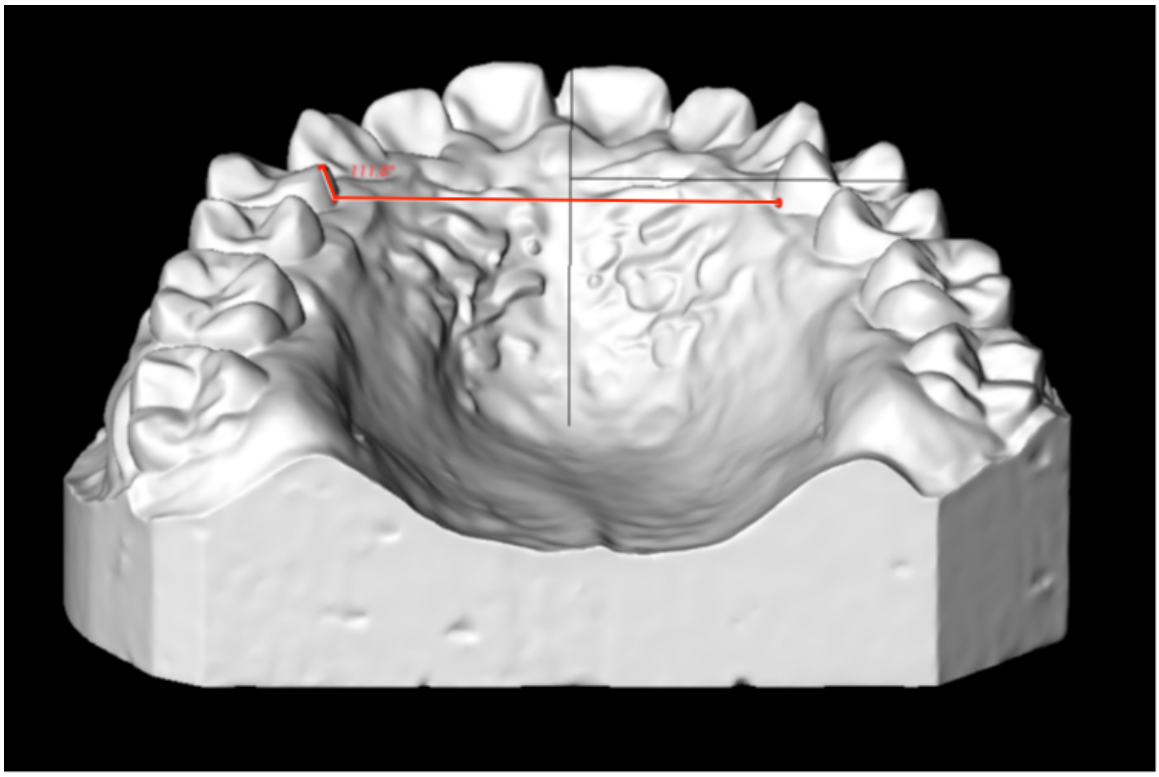


Figure 4. Image depicting location of buccal bone measurements taken from the CBCT images: **1.** Buccal bone maximum width **2.** Buccal bone minimum width. **3.** Width of buccal bone 3 mm apical to the CEJ **4.** Buccal bone height from CEJ to crestal bone, a maximum and minimum was taken for this measurement as the viewer moved through the slices in both a mesial and distal direction.

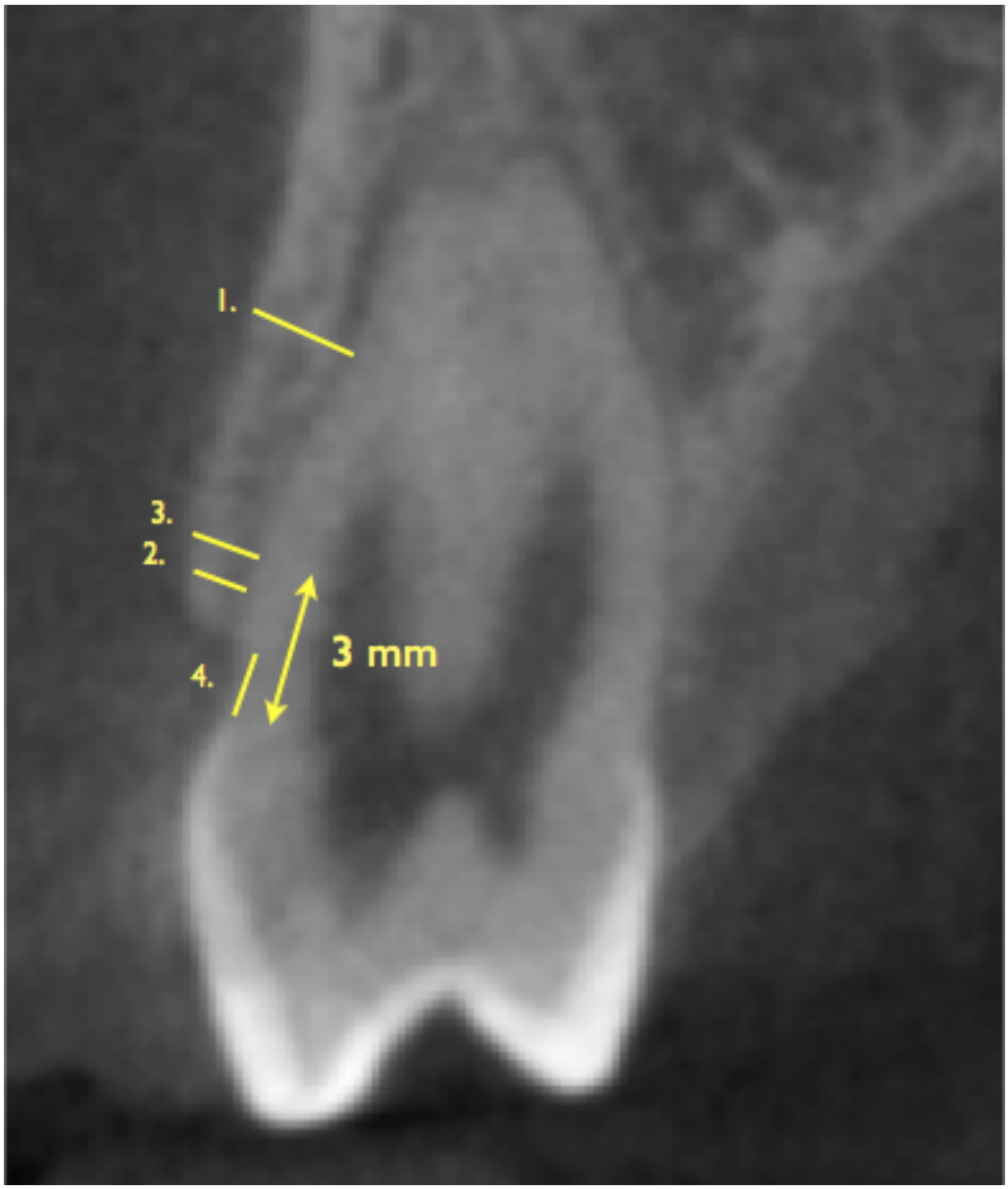


Figure 5. Images from the 3D superimposition of patient 6. Patient 6 represents a patient with a minimal amount of tooth movement. White is the final position and green is initial. **A.** Coronal view of pre-treatment CBCT images. **B.** Coronal view of post-treatment CBCT image. **C.** Frontal view of superimposed 3D CBCT images.

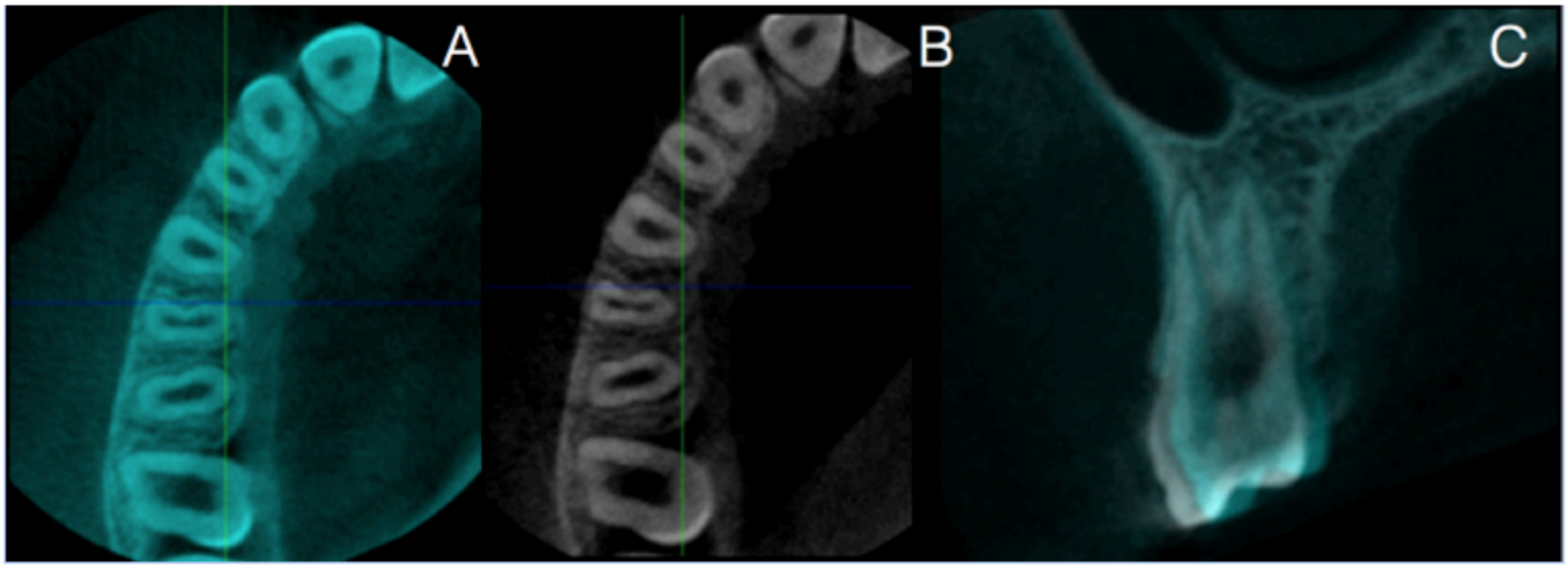


Figure 6. Images from the 3D superimpositions of patient 13: Patient 13 represents a patient with an average amount of tooth movement. White is the final position and green is initial. **A.** Coronal view of pre-treatment CBCT images. **B.** Coronal view of post-treatment CBCT image. **C.** Frontal view of superimposed 3D CBCT images.

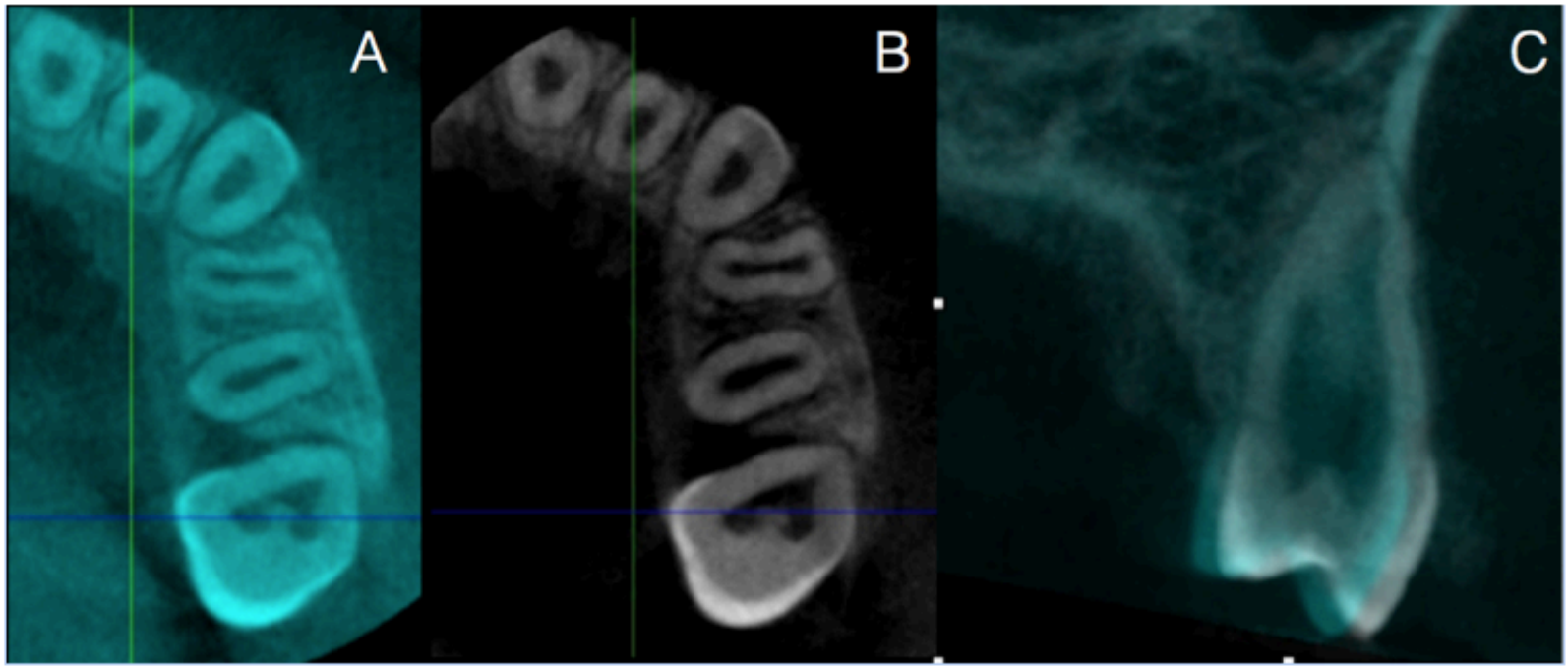


Figure 7. Images from the 3D CBCT superimpositions of patient 12: Patient 12 represents one of the patients with a substantial amount of tooth movement. White is the final position and green is initial. **A.** Coronal view of superimposed 3D CBCT images. **B.** Frontal view of superimposed 3D CBCT images.

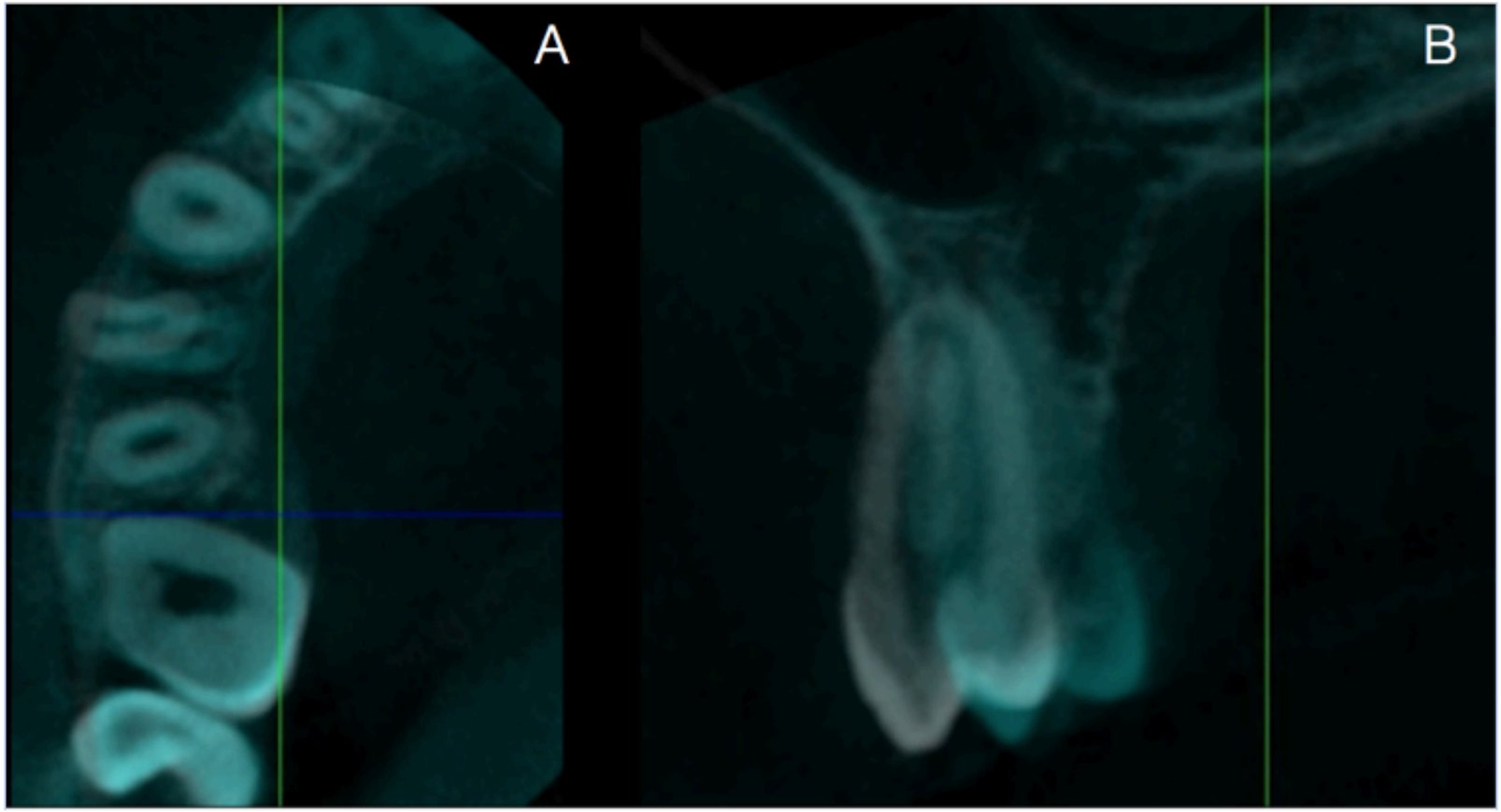


Figure 8. Interdental width changes (medians and interquartile ranges) evaluated on plaster models: First premolar measurements were taken between the lingual cusps (U4s L-L) as well as between the buccal cusps (U4s B-B). First molar measurements were taken between the central fossae (U6s CF-CF) and between the mesial-buccal cusp tips (U6s MB-MB).

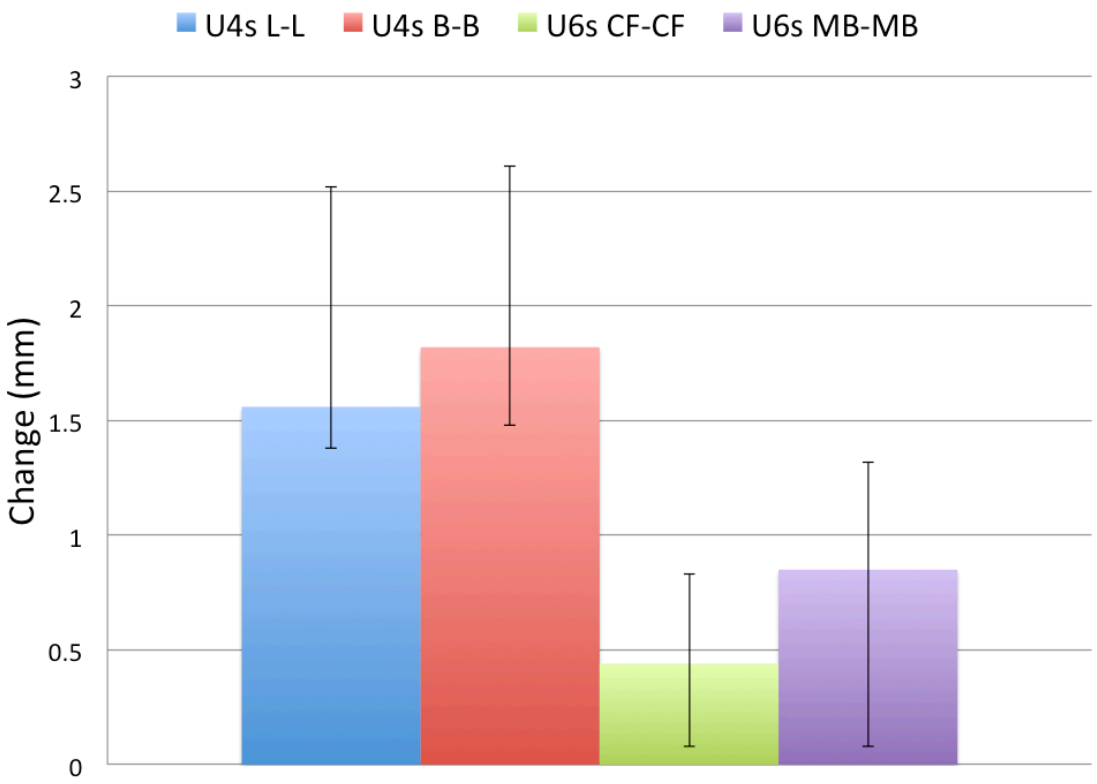


Figure 9. Buccal bone thickness changes measured on the CBCT images: The 3-D measurements were made on both experimental and control teeth from the most lingual to the most buccal aspects of buccal bone at three heights (3 mm below the CEJ, at the maximum thickness along the root surface, and the minimum thickness along the root surface.

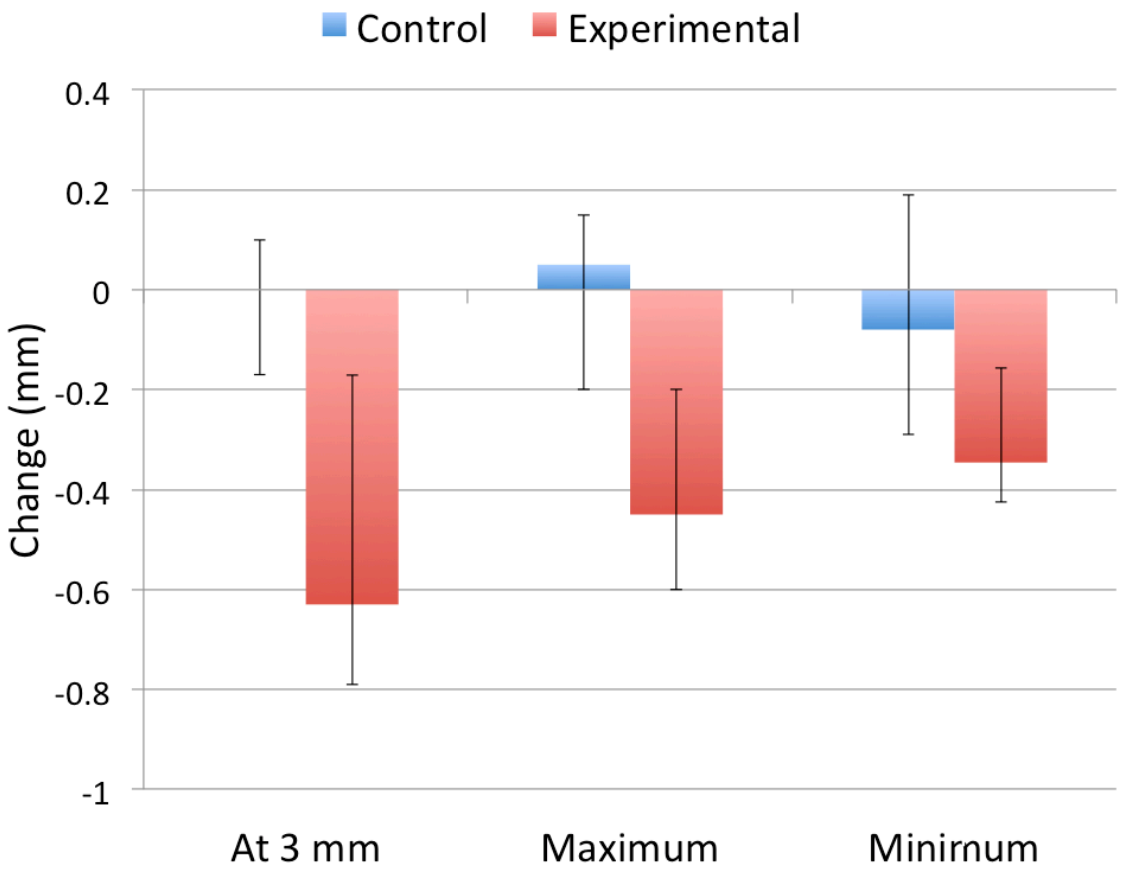


Figure 10. Buccal bone height changes taken from the CBCT images: the 3-D measurements were taken from the CEJ to the most apical and most coronal extents of the crestal bone on the facial surface of the experimental and control teeth.

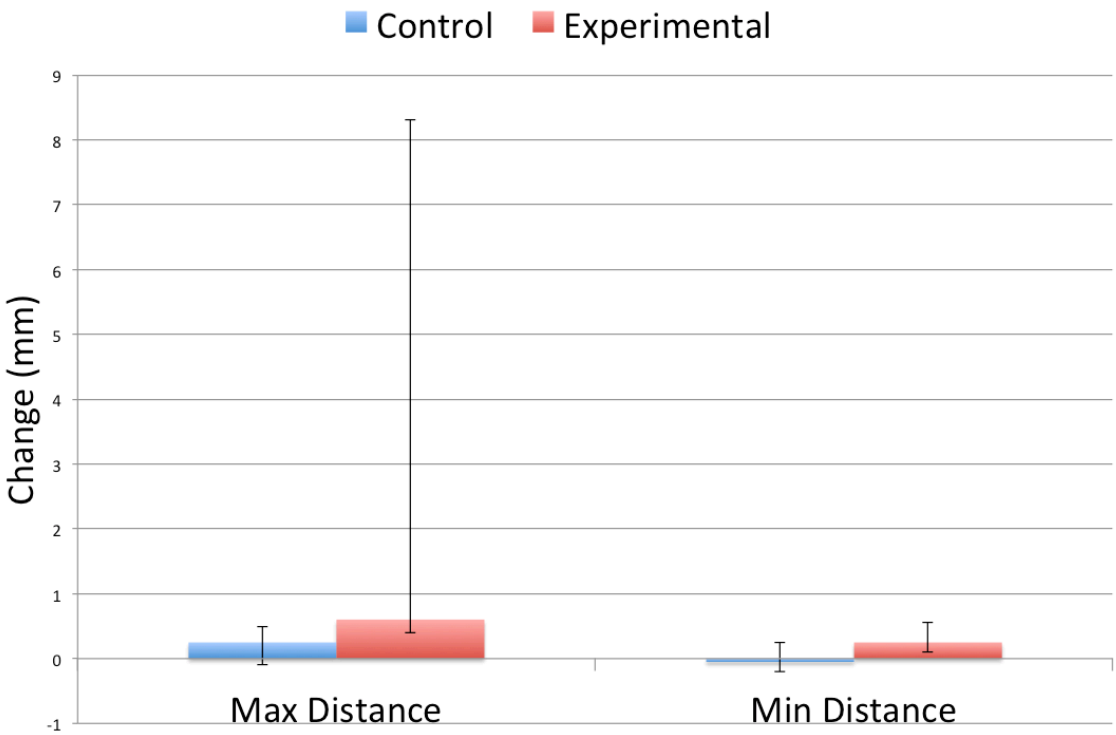


Figure 11. Frequency and dispersion for the change in maximum vertical bone height on the experimental side taken from the CBCT images: Maximum vertical bone height corresponds to the greatest distance from the CEJ to the crestal bone at any point along the buccal surface of the tooth root. Note the bimodal tendency with six subjects concentrated around no change in vertical height while five subjects displayed vertical height loss greater than 7 mm.

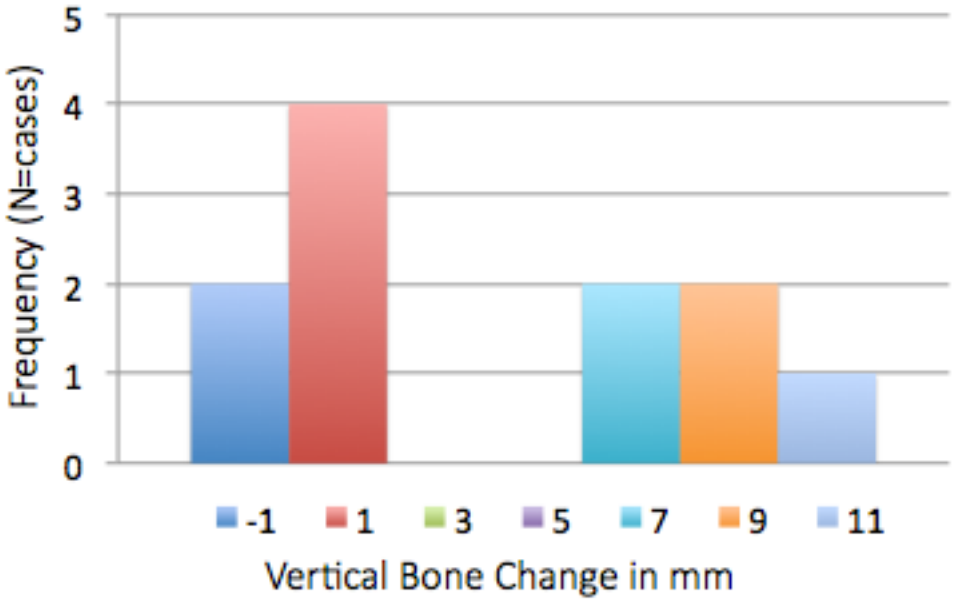


Figure 12. Amount of buccal bone growth directly measured from the 3D superimpositions: Buccal bone growth was measured 3 mm below the CEJ at the midline of each tooth. The measurement was taken from the most buccal aspect of the buccal cortex on the intial CBCT image to the same position on the final CBCT image.

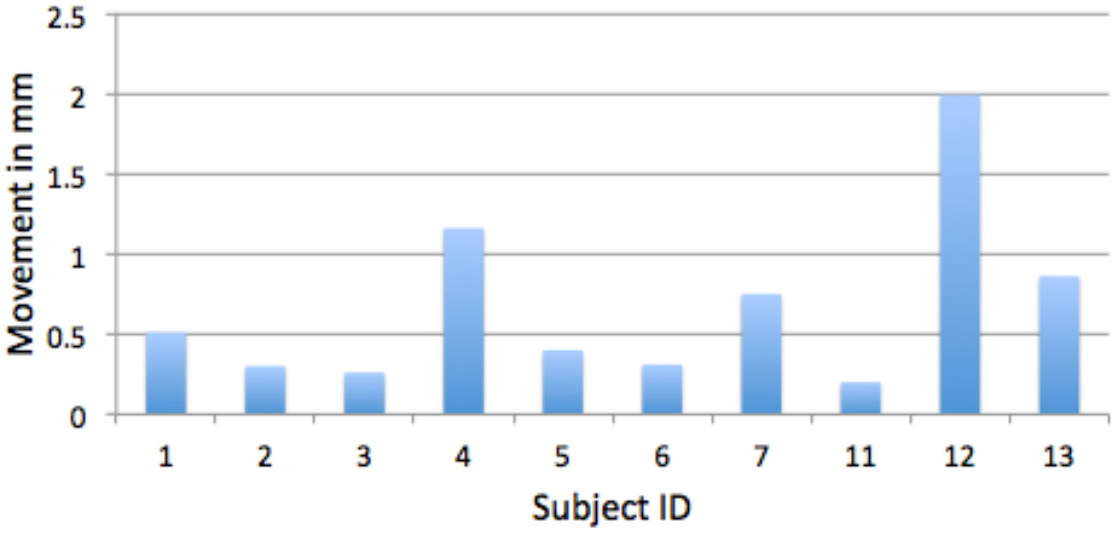


Figure 13. Photographic images depicting relapse during the passive force phase for patient #11 and #5, respectively: **A.** Image taken at the end of active forces and beginning of the passive force phase. **B.** Image after removal of passive force. Note palatal movement (relapse) of the first premolar.

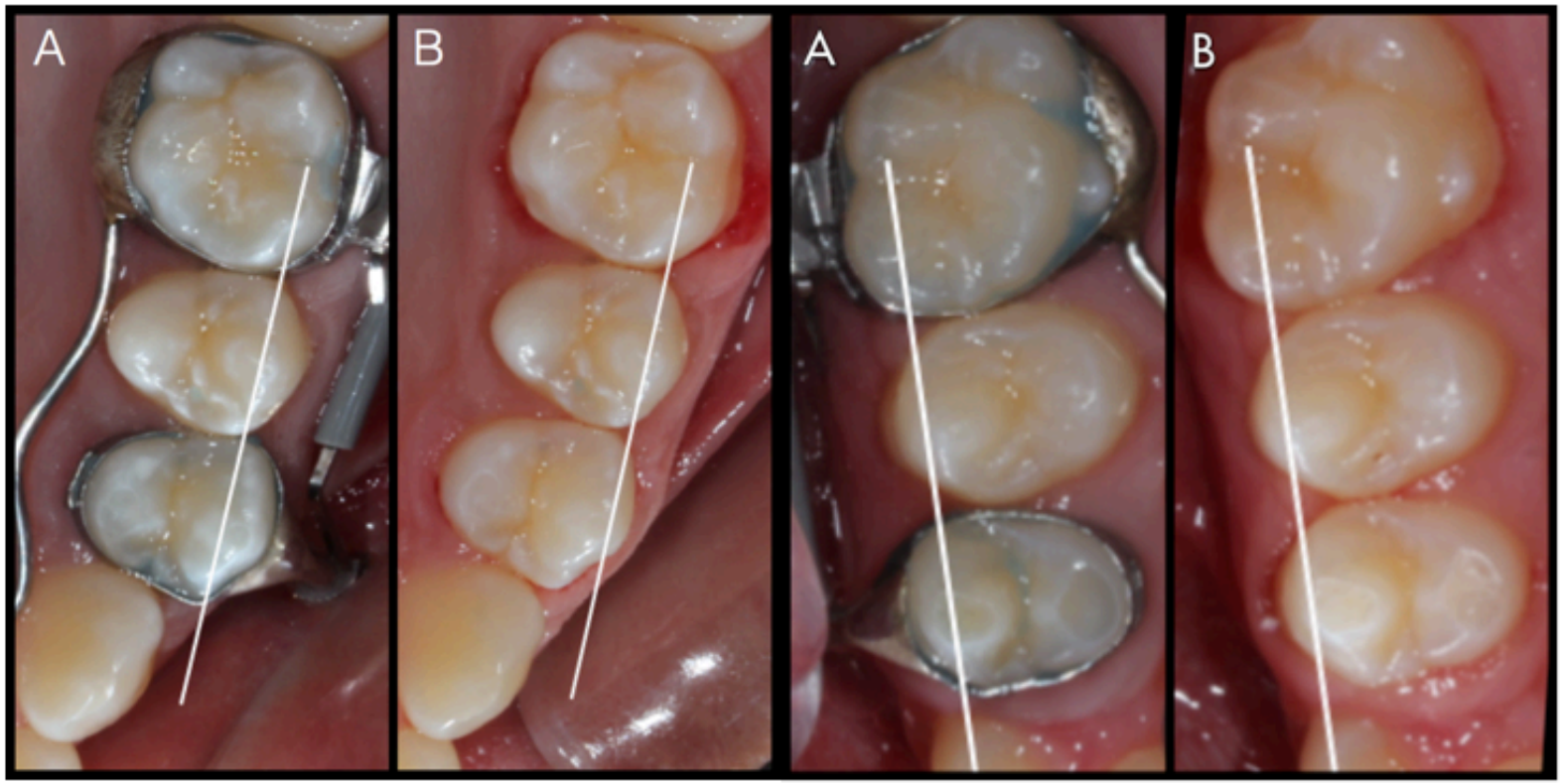
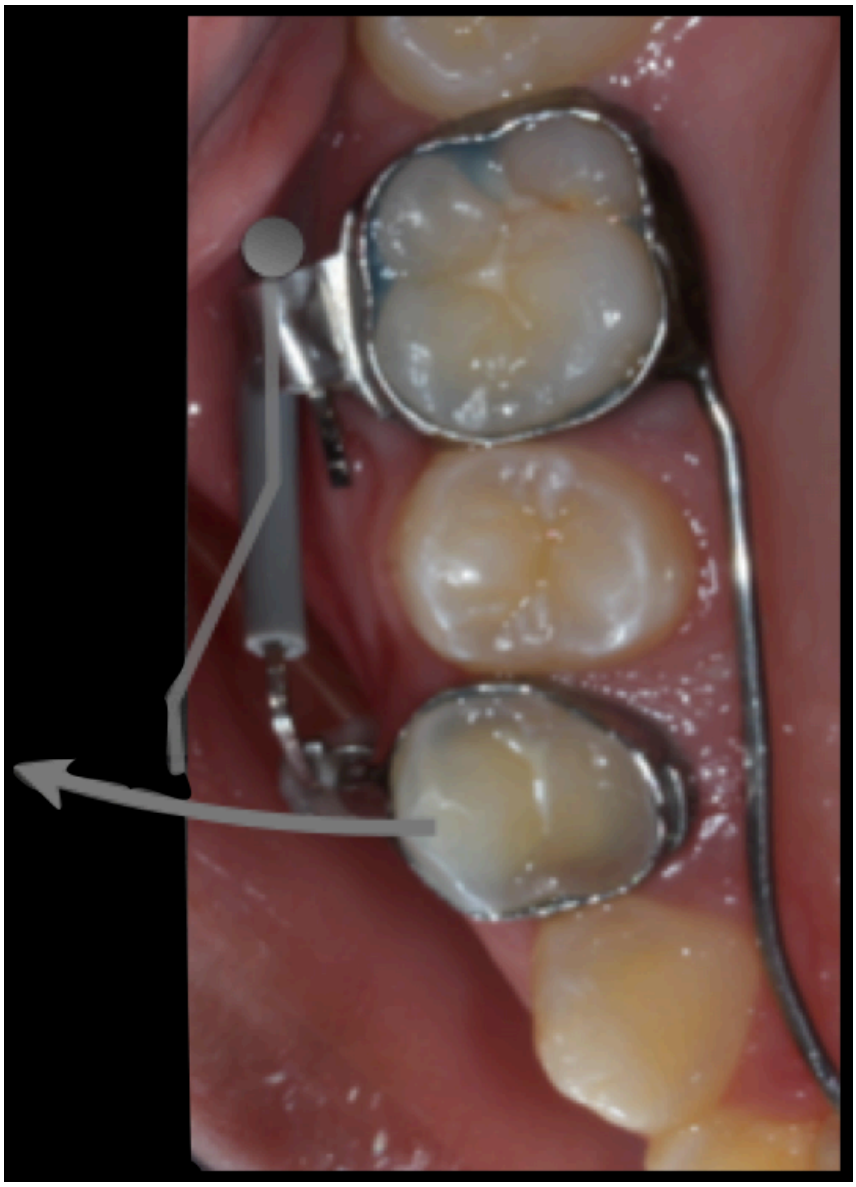


Figure 14. Occlusal photograph displaying rotational movement of the experimental premolar: The cantilever responsible for producing the buccal force was anchored at the auxiliary tube of the maxillary first molar. The cantilever was bent to exit from the distal aspect of the tube in order to reduce the effect of the arc. However, a mesial to buccal rotation was still produced in the experimental premolar.



APPENDIX B

TABLES

Table 1. Table adapted from Germane et al[1] depicting the amount of arch perimeter increase (mm) based on the amount of expsansion (mm).

Amount of expansion/flaring	Amount of increased arch perimeter
1st molar expansion of 1mm	0.27 mm
1st molar expansion of 5mm	1.72 mm
Canine expansion of 1mm	0.73 mm
Canine expansion of 5mm	5.34 mm
Incisor flaring of 1mm	1.04 mm
Incisor flaring of 5mm	6.03 mm

Table 2. Summary of average time duration (days) between appointments. At delivery the appliance was activated to 50 grams. The force was re-activated to 50 grams at the subsequent appointments until 2 mm of tooth movement was measured. At that time a passive force of approximately 15 grams was placed until the appliance was removed. The target time between appointments was 21 days.

	Delivery and 1st Re-Activation	1st and 2nd Re-Activation	2nd Re-Activation and Maturation	Maturation and Removal
Days	21.3 $\pm$ 2.1	20.4 $\pm$ 2.0	17.7 $\pm$ 4.3	20.5 $\pm$ 2.6

Table 3. Data describing the amount of tooth movement (mm) measured on the models of patients #9 and #10. These patients were excluded due to the lack of significant movement.

	1st Premolar Intercuspal Distance		Intermolar Distance	
ID Number	**Buccal Cusps**	**Palatal Cusps**	**Central Fossae**	**Mesial Buccal Cusps**
9	0.13	0.25	0.6	0.74
10	0.28	0.57	-0.25	0.43

Table 4. Median, distribution, and probability for measurements of tipping (deg), root movement, and bone growth for the experimental premolar.

	Quartile			
Variable	50th	25th	75th	Probability
Tipping	2.2°	1.26°	4.01°	0.003
Experimental Root Movement 3 mm apical to CEJ	0.96 mm	0.65 mm	1.30 mm	0.008
Experimental Buccal Bone Growth Measured	0.46 mm	0.29 mm	0.94 mm	0.005
Experimental Buccal Bone Growth Calculated	0.51 mm	-0.40 mm	1.00 mm	0.036

Table 5: Median, distribution, and significance of each buccal bone measurement for the control and experimental sides. Note all experimental measurements are significantly different than the control except for the change in minimum vertical distance measured from the CEJ. Also note the variability in the measurement for change in maximum vertical distance from CEJ.

	Experimental			Control			
Buccal Bone Measurements	50th	25th	75th	50th	25th	75th	Probability
Change in thickness 3mm apical to CEJ	-0.63	-0.79	-0.17	0.00	-0.17	0.10	0.016
Change in maximum thickness	-0.45	-0.60	-0.20	0.05	-0.20	0.15	0.011
Change in minimum thickness	-0.35	-0.43	-0.15	-0.08	-0.29	0.19	0.041
Change in maximum vertical distance from CEJ	0.60	0.40	8.30	-0.05	-0.45	0.15	0.003
Change in minimum vertical distance from CEJ	0.25	0.10	0.55	-0.05	-0.40	0.20	0.262

Table 6. Central tendency and distribution for buccal bone thickness measurements taken from the CBCT images: Initial and final thickness measurements were taken 3 mm apical to the CEJ for both the control and experimental sides.

	Experimental			Control		
Buccal Bone Thickness in mm	50th	25th	75th	50th	25th	75th
Initial measurement 3 mm apical to CEJ	1.40	0.85	1.50	1.45	1.20	1.65
Final measurement 3 mm apical to CEJ	0.86	0.73	0.96	1.50	1.00	1.60