Dimensional Changes in the Palate Associated with Early Treatment of Posterior Crossbite

by

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Abstract

Introduction: Unilateral posterior crossbite with functional shift is a relatively common condition. Spontaneous correction occurs in ~17% of the cases and mixed dentition slow maxillary expansion demonstrated 84% stability in permanent dentition. The purpose of this study was to evaluate palatal symmetry, dimensions and molar angulation changes following slow maxillary expansion during the mixed dentition compared to untreated controls.

Methodology: 30 subjects treated with a Haas-type slow maxillary expansion appliance for unilateral posterior crossbite with a functional shift were compared to 30 control subjects matched for dental age, gender and dental classification. Records were taken at T1= before expansion and at T2= after expansion. Palatal width, surface area, volume and molar angulations measured on digitized models. Palatal surface area and volume divided into anterior, middle and posterior parts then split into crossbite and non-crossbite sides by the midpalatal plane to measure symmetry. Student's t-test assessed the differences between the groups.

Results: Mean intercanine width increase from T1-T2 was 4.65mm, while the intermolar width increase was 4.76mm. The mean increase in the surface area from T1-T2 was 127.05 mm2, while for the controls it was 10.35mm2. The mean palatal volume increased by 927.55mm3 for the treated sample while for the controls it increased by 159.89 mm3. At T1, significant differences between the crossbite and noncrossbite sides were only present in the anterior halves of the controls in surface area and volume. At T2, difference was only present in the surface area of the treated sample in the middle halves. The first permanent molar showed an increase in the buccal and distal inclination after treatment while in controls it showed a decrease. The increase in the buccal inclination was greater on the treated crossbite

side.

Conclusions: Expansion across the canines and first permanent molars was almost similar. Palate was symmetric before expansion and became asymmetric after expansion in the middle halves. The first permanent molars tipped buccally and distally following expansion with significant tipping on the crossbite side buccally. Our method of molar angle measurement does not require radiation and allows measurement of each individual molar's angulation.

Preface

The treated sample was collected from Dr. Kennedy's practice. The input and refinement of proposed methodology was done by committee members Dr. Ed Yen, Dr. Alan Hannam and Dr. David Kennedy. The work on methodology on Rhinoceros 5.0 software was performed by Dr. Alan Hannam. Data was collected and analyzed by Dr. Abdulkadir Bukhari who prepared the manuscript with advice from Dr. David Kennedy, Dr. Alan Hannam, Dr. Jolanta Aleksejuniene and Dr. Ed Yen. All statistical analyses were performed by Dr. Jolanta Aleksejuniene. The study was approved by the University of British Columbia office of Research Services, Humans Research Ethics Board (Certificate Number: H14-02193).

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List of Abbreviations

IC: Intercanine

ICW: Intercanine width

IM: Intermolar

IMW: Intermolar width

RME: Rapid maxillary expansion

SME: Slow maxillary expansion

TMD: Temporomandibular joint disorders

TMJ: Temporomandibular joint

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Dedication

I dedicate this thesis to my parents and family who have supported and encouraged me throughout my education.

Chapter 1: Introduction

1.1 Overview

Posterior crossbite is a relatively common condition which occurs when there is a transverse discrepancy between the arches. The prevalence of posterior crossbite was found to be between 8% and 23% (Thilander & Lennartsson, 2002; Kurol & Berglund, 1992; Kutin & Hawes 1969).In the deciduous dentition, this can be bilateral with a 3-5% prevalence. A unilateral posterior crossbite with more than two posterior teeth involved occurs >95% of the cases and about 33% have a lateral incisor involved (Lindner & Modéer, 1989). 80%-97% of the crossbite cases have a functional shift of the lower jaw towards the crossbite side (Kurol & Berglund, 1992; Thilander, Wahlund, Lennartsson, 1984; Lindner & Modéer, 1989). Posterior crossbite with functional shifts occur when there are interferences in centric relation causing the mandibular lateral forced guidance into maximum intercuspation (Björk, Krebs, Solow, 1964). This interference is usually caused by the primary canines (Proffit, Fields, Sarver, 2006). The lower midline shift to the crossbite side is associated with the functional shift (Hesse, Årtun, Joondeph, Kennedy, 1997; Kutin & Hawes, 1969). Associated with the functional shift is chin deviation toward the crossbite side in maximum intercuspation causing facial asymmetry (Marshall, Southard, Southard, 2005; Hesse, Årtun, Joondeph, Kennedy, 1997). Asymmetry in the antero-posterior dental classification was found with the crossbite side having a Class II relationship while the non crossbite side having Class I relationship (Hesse, Årtun, Joondeph, Kennedy, 1997). Posterior crossbite affects girls more than boys; this could be attributed to the thumb sucking habit (Johnson & Larson, 1993).

Deciduous dentition Crossbites left untreated will result in the permanent dentition crossbite (Kutin & Hawes, 1969); spontaneous correction has been reported to be about 17% (Lindner, 1989) .

One of the indications of expansion is to treat posterior crossbites of skeletal origin. Another indication is to relieve mild anterior crowding cases (Binder, 2004) as each 1mm of expansion in the inter-premolar width provides 0.7mm of arch circumference increase (Adkins, Nanda, Currier, 1990). Early treatment by expanding the maxilla corrects the posterior crossbite, allowing permanent teeth to erupt into normal occlusion; it eliminates interferences providing favorable dental and skeletal changes during growth (Bell, 1982; Kurol & Berglund, 1992). However between 16-40% of the crossbite cases treated in deciduous dentition needed further expansion in the mixed dentition to correct the permanent first molar crossbite (Schröder & Schröder, 1984; Tsarapatsani, Tullberg, Lindner, Huggare, 1999). On the other hand, correction of posterior crossbite by slow maxillary expansion in the mixed dentition demonstrated 84% stability in the permanent dentition (Huynh, Kennedy, Joondeph, Bollen, 2009). Successful and long term stable results of early treatment of unilateral posterior crossbite has been reported (Petrén, Bjerklin, Bondemark, 2011).

As part of normal development maxillary and mandibular permanent molars express changes in the crown torque and intermolar width. These changes were evaluated from the eruption of the permanent first molar to early adulthood; the mandibular first and second molars erupt with lingual crown torque then upright buccally by 5.0° and 7.5° as a result the intermolar width increase by 2.2 and 0.78 mm, respectively (Marshall, Dawson, Southard, Lee, Casko, Southard, 2003). Maxillary first and second molars erupt with buccal crown

torque then upright lingually by 3.3° and 5.9° associated with intermolar width increase by 2.8 and 2.0 mm, respectively (Marshall, Dawson, Southard, Lee, Casko, Southard, 2003).

1.2 Etiology

There are several causes of posterior crossbite which can be either due to dental or skeletal transverse problems indicative of a narrower maxilla. These causes include: Genetics, short lingual frenum with low tongue posture, habits such as digit or pacifier habits, mouth breathing which might be due to airway problems (Melink, Vagner, Hocevar-Boltezar, Ovsenik, 2010; Kutin, & Hawes, 1969; Proffit, Fields, Sarver, 2006). 23% of posterior crossbites were associated with sucking habits (Kurol & Berglund, 1992). Pacifier sucking habits have also shown association with unilateral posterior crossbite especially when the habit exists for more than 3 years (Melink, Vagner, Hocevar-Boltezar, Ovsenik, 2010). Bishara et al. (2006) determined the effects of pacifier or digit habits in a longitudinal study. When these habits persisted for less than a year, there were no occlusal changes. However, if the habit extended for more than four years, it was associated with posterior crossbite, anterior openbite and increased overjet. Posterior crossbite showed a higher incidence with the pacifier habits, while overjet of more than 3mm was more associated with digit habits. There was no significant difference between the two habits on the incidence of anterior openbite and Class II canine relationships (Bishara, Warren, Broffitt, Levy, 2006).

1.3 Diagnosis

Early diagnosis and intervention is helpful to prevent several side effects; it is important because the transverse growth is the earliest to be completed (Allen, Rebellato,

Sheats, Ceron, 2003). An accurate clinical diagnosis is made with the patient biting in centric relationship to determine whether there is a functional shift. The crossbite location can be unilateral or bilateral, and may be dental or skeletal origin or a combination of both. Usually the crossbite is caused by upper arch constriction but occasionally both arches are involved (Binder, 2004). Many of the unilateral crossbite cases are actually bilateral with a functional shift into maximum intercuspation (Binder, 2004). If the etiology is due to sucking habits, usually it will be associated with proclined maxillary incisors, anterior open bite, narrowing of the upper arch with high palatal vault and lingual tipping of mandibular incisor (Proffit, Fields, Sarver, 2006). Most of the changes associated with sucking habits resolve only if the habit is stopped before the eruption of permanent incisors (Proffit, Fields, Sarver, 2006). An accurate habit history should be taken regarding the frequency, duration and when the habit has stopped (Binder, 2004). In addition to the clinical examination, diagnostic records such as dental models and radiographs should be taken to develop a treatment plan (Binder, 2004). The transverse skeletal discrepancies are mostly evaluated on the dental casts and radiographs (Allen, Rebellato, Sheats, Ceron, 2003; Proffit, Fields, Sarver, 2006). Dental models are also useful to evaluate the Curve of Wilson; buccal or lingual tipping of molar teeth are compensatory changes assessed from models (Binder, 2004). This may determine if the crossbite is of dental rather than skeletal origin. In a true skeletal maxillary deficiency, the upper posterior teeth will tip buccally and the lower posterior teeth will tip lingually (Proffit, Fields, Sarver, 2006). PA cephalograms are useful radiographs to determine transverse skeletal discrepancies (Allen, Rebellato, Sheats, Ceron, 2003) and to evaluate dental midlines relative to the skeletal midlines (Cross & McDonald, 2000).

1.4 Consequences of Leaving Posterior Crossbite Untreated

Posterior crossbites with functional shifts should be treated as soon as they are noticed as this shift might have effects on both hard and soft tissues (Binder, 2004). These include changes in the TMJ, skeletal and dental symmetry, soft tissue and loss of tooth structure by attrition (Proffit, Fields, Sarver, 2006; Nerder, Bekke, Solow, 1999; O'Byrn, Sadowsky, Schneider, BeGole, 1995; Cross & McDonald, 2000; Hesse, Årtun, Joondeph, Kennedy, 1997).

1.4.1 Skeletal Asymmetry

The lateral shift affects the symmetrical growth of the mandible (Schmid, Mongini, Felisio, 1991) by causing changes in the mechanism of bone development and the direction of growth resulting in mandibular asymmetry (Vadiakas, & Roberts, 1991; ; O'Byrn, Sadowsky, Schneider, BeGole, 1995). Unilateral posterior crossbite with functional shift is associated with chin deviation to the crossbite side (Ishizaki, Suzuki, Mito, Tanaka, Sato, 2010; Van Keulen, Martens, & Dermaut, 2004; Hesse, Årtun, Joondeph, Kennedy, 1997). There is sometimes a canted occlusal plane which is higher on the crossbite side (Ishizaki, Suzuki, Mito, Tanaka, Sato, 2010), negatively affecting facial esthetics and symmetry. The human face always has some degree of physiologic asymmetry (Bishara, Burkey, Kharouf, 1994) but when the asymmetry becomes highly visible and can be easily detected, then that is not considered normal. Primožič et al. compared the facial asymmetry of 30 children with posterior crossbite to 28 children without posterior crossbite before and after treatment by using 3D laser scans; the crossbite group had facial asymmetry before treatment, which was obvious mainly in the lower third of the face (Primožič, Ovsenik, Richmond, Kau, Zhurov,

2009). This asymmetry resolved following treatment (Primožič, Ovsenik, Richmond, Kau, Zhurov, 2009).

1.4.2 Temporo-Mandibular Joint Asymmetry

The mandibular functional lateral shift is also associated with asymmetry in the condylar position with the condyle on the crossbite side displaced upward (Myers, Barenie, Bell, Williamson, 1980) and the condyle on the opposite side is displaced downward and forward (Myers, Barenie, Bell, Williamson, 1980; Hesse, Årtun, Joondeph, Kennedy, 1997). Histological studies show that remodeling of the TMJ area on both crossbite and non-crossbite sides occured in cases of untreated crossbite cases with functional shift (Pinto, Buschang, Throckmorton, Chen, 2001). Kecik et al. found that in children with a functional posterior crossbite before treatment, the crossbite side had larger joint spaces in the glenoid fossa (Kecik, Kocadereli, Saatci, 2007). Other studies have reported similar findings about asymmetrical joint spaces in functional posterior crossbite, which was corrected after early treatment of the crossbite (Schmid, Mongini, Felisio, 1991; Sandikçioğlu & Hazar, 1997; Petrén, Bondemark, Söderfeldt, 2003; Lippold, Hoppe, Moiseenko, Ehmer, Danesh, 2008; Ishizaki, Suzuki, Mito, Tanaka, Sato, 2010). Hesse et al. evaluated the condylar position in children with mean age of 8.5 years with functional posterior crossbite before and after treatment using tomograms; the condyle on the non-crossbite side was displaced forwards before treatment and the condyle moved backwards and upwards following crossbite correction, which corrected the condylar asymmetry (Hesse, Årtun, Joondeph, Kennedy, 1997). However, the condyle on the crossbite side did not show any changes in the position before and after treatment (Hesse, Årtun, Joondeph, Kennedy, 1997). These findings were

similar to an early study by Myers et al. using standardized transcranial TMJ radiographs; the temporomandibular joint spaces regained symmetry following the correction of the functional posterior crossbite (Myers, Barenie, Bell, Williamson, 1980). By contrast, Leonardi et al. found that there was symmetric condylar position on both crossbite and non-crossbite sides pre and post treatment in children with functional posterior crossbite (Leonardi, Caltabiano, Cavallini, Sicurezza, Barbato, Spampinato, Giordano, 2012). Thilander et al. reported that subjects between 5 and 17 years old with untreated unilateral posterior crossbite had TMJ symptoms and headaches (Thilander, Rubio, Pena, de Mayorga, 2002). However, other studies reported no association between posterior crossbite and temporomandibular joint disorders (TMD) (Farella, Michelotti, Iodice, Milani, Martina, 2007; Pahkala, & Qvarnström, 2004). In addition, posterior crossbites cause functional asymmetry which is evaluated through jaw movements and muscular activity (Abekura, Kotani, Tokuyama, Hamada, 1995; Ferrario, Sforza, Colombo, Ciusa, 2000).

1.4.3 Functional Asymmetry

In the normal chewing cycle, closing the mouth is associated with the mandible movement towards the bolus side (Ben-Bassat, Yaffe, Brin, Freeman, Ehrlich 1993). Unilateral posterior crossbite patients have shown the movement of the mandible to the opposite direction of the bolus, which is called the reverse chewing sequence (Ben-Bassat, Yaffe, Brin, Freeman, Ehrlich 1993). Jaw movements were recorded in patients with unilateral posterior crossbite using kinesiographs; patients had a high prevalence of reverse sequencing chewing pattern on the crossbite side before treatment, which was significantly reduced following the treatment (Piancino, Talpone, Dalmasso, Debernardi, Lewin, Bracco,

2006). Brin et. al showed the same results although they were not statistically significant (Brin, Ben-Bassat, Blustein, Ehrlich, Hochman, Marmary, Yaffe, 1996). Family members with bilateral posterior crossbite had a higher prevalence of reverse sequencing chewing pattern, which was not present in the father who had his crossbite treated (Piancino, Talpone, Vallelonga, Manera, De Lama, Altamura, Bracco, 2010). Also, patients with functional crossbite chew for a longer period of time compared to the normal occlusion population (Gaylord S. Throckmorton, Buschang, Hayasaki, Pinto, 2001). Six months after crossbite correction chewing became faster closer to the normal population (Gaylord S. Throckmorton, Buschang, Hayasaki, Pinto, 2001). During the early mixed dentition, unilateral posterior crossbite was found to be associated with less occlusal contacts than the normal occlusion group (Castelo, Gaviao, Pereira, Bonjardim, 2007).

1.4.4 Dental Asymmetry

Other side effects caused by the unilateral posterior crossbite with lateral shift is the asymmetry in antero-posterior dental relationship resulted from the crossbite side having ½ cusp Class II molar tendency while the non-crossbite side having Class I molar relationship and mandibular midline shift (Hesse, Årtun, Joondeph, Kennedy, 1997). This molar asymmetry and midline deviation showed improvements following expansion in primary or mixed dentition (Brin, Ben-Bassat, Blustein, Ehrlich, Hochman, Marmary, Yaffe, 1996; Bishara, Burkey, Kharouf, 1994; Hesse, Årtun, Joondeph, Kennedy, 1997; Ishizaki, Suzuki, Mito, Tanaka, Sato, 2010; Kecik, Kocadereli, Saatci, 2007; Lindner & Modéer, 1989; Sandikçioğlu & Hazar, 1997). However, the midline shift improvement has shown unpredictable results in the long term follow-up (Petrén, Bjerklin, Bondemark, 2011).

1.4.5 Muscular Asymmetry

Posterior crossbites have also shown to affect the symmetry in the thickness and activity of the muscles; this asymmetry can be evaluated by several methods. Muscular activity is usually detected by the surface electromyography (Dahlström, 1989). Ultrasound is another method used to visualize muscle movement (Emshoff, Bertram, Strobl, 1999; Kiliaridis & Georgiakaki, 2003) and determine the thickness of the superficial masticatory muscles (Emshoff, Bertram, Br, lmaier, 2002). Also, computerized tomography (Katsumata, Fujishita, Ariji, Ariji, Langlais, 2004; Van Spronsen, Weijs, Valk, Prahl-Andersen, Van Ginkel, 1989) and magnetic resonance imaging (Raadsheer, Kiliaridis, Van Eijden, Van Ginkel, Prahl-Andersen, 1996; Zanoteli, Yamashita, Suzuki, Oliveira, Gabbai, 2002; Van Spronsen, Weijs, Valk, Prahl-Andersen, Van Ginkel, 1989) are used for diagnosing and evaluating the volume and sections of masticatory muscles. However, computerized tomography has the disadvantage of expressing the effects that have accumulated over time; the issue with magnetic resonance imaging is that it is not available in every clinic (Castelo, Gaviao, Pereira, Bonjardim, 2007). Muscle thickness of the anterior temporalis at rest was greater on the normal side than the crossbite side during the early mixed dentition. In normal early mixed dentition occlusion subjects, there were no significant differences in the thickness of the masseter or the anterior temporalis at rest or in maximum intercuspation (Castelo, Gaviao, Pereira, Bonjardim, 2007). That the maximum biting force was lower in the early mixed dentition crossbite subjects than the same dental stage normal occlusion group (Castelo, Gaviao, Pereira, Bonjardim, 2007). This bite force difference in children with functional posterior crossbite was reported by Sonneson et al. (L Sonnesen, Bakke, Solow, 2001). For primary dentition children, there were no differences in the maximum biting force

between the crossbite and normal occlusion groups (Castelo, Gaviao, Pereira, Bonjardim, 2007). Sonnesen and Bakke found that the bite force in children on the crossbite side increased to about the same level as children with normal occlusion after crossbite correction (L Sonnesen & Bakke, 2007). Long face pattern population subjects had thin masseter muscles and light biting force (Kiliaridis, Georgiakaki, Katsaros, 2003; Kiliaridis & Kälebo 1991). However, Castelo et. al found that the anterior face height did not correlate with masseter and temporal muscle thickness or the bite force in either the normal occlusion or posterior crossbite groups (Castelo, Bonjardim, Pereira, Gavião, 2008). People with normal occlusion have some normal degree of asymmetry in the activity of the anterior temporalis muscle; there was increased activity on the right side at rest (Alarcón, Martín, Palma, 2000; Ferrario, Sforza, Miani, D'Addona, Barbini, 1993). However, asymmetric muscular activity beyond the normal range have been associated with posterior crossbite with functional shift, with the shift attributed to the cause of the problem (Vadiakas & Roberts, 1991). The posterior temporalis was more active on the crossbite side at rest and during maximal intercuspation, while the anterior temporalis had less activity on the same side (Troelstrup & Møller, 1970). Alarcon et.al found that the posterior temporalis was more active on the non crossbite side while the anterior temporalis was more active on the crossbite side at rest (Alarcón, Martín, Palma, 2000). This finding was the same according to Tecco et al. and Kecik et al. for the anterior temporalis muscle only (Tecco, Tete, Festa, 2010; Kecik, Kocadereli, Saatci, 2007). The EMG activity of anterior temporalis was also higher on the right posterior crossbite group compared to the normal occlusion group (Andrade, Gavião, DeRossi, Gameiro, 2009). There was no difference between the left posterior crossbite group and the normal occlusion group (Andrade, Gavião, DeRossi, Gameiro, 2009). Alarcon et al.

reported in the crossbite subjects that the anterior temporalis on the non crossbite side and the anterior digastric on both sides were more active compared to subjects with normal occlusion during swallowing (Alarcón, Martín, Palma, 2000). During chewing, the masseter muscle on the crossbite side had less activity than the normal group, while the anterior digastric muscle on the crossbite side was more active than the normal occlusion population (Alarcón, Martín, Palma, 2000). Piancino et al. found that in unilateral posterior crossbites the masseter muscle activity is reduced during chewing on the crossbite side while it remains unchanged on the non-crossbite side (Piancino, Farina, Talpone, Merlo, Bracco, 2009). However, the masseter muscle was more active at rest on the crossbite (Kecik, Kocadereli, Saatci, 2007). Andrade et al. found that the masseter muscle was more active at maximum clenching on the crossbite side (Andrade, Gavião, DeRossi, Gameiro, 2009). During maximum clenching, the EMG showed that sternocleidomastoid and posterior cervical muscles were more active in patients with either unilateral or bilateral posterior crossbite than the normal occlusion population (Tecco, Tete, Festa, 2010). The muscular activity returns to normal and becomes more symmetrical after posterior crossbite treatment (Kecik, Kocadereli, Saatci, 2007; Tsarapatsani, Tullberg, Lindner, Huggare, 1999; Yawaka, Hironaka, Akiyama, Matzuduka, Takasaki, Oguchi, 2003).

1.5 Treatment

In order to determine which treatment plan is suitable for posterior crossbite treatment, the underlying cause should be identified (Allen, Rebellato, Sheats, Ceron, 2003). Crossbite cases of dental origin can be treated with a heavy labial expansion arch, expansion with a lingual arch or cross elastics just to move the teeth out of the crossbite (Proffit, Fields,

Sarver, 2006). Skeletal posterior crossbites require an increase in the transverse arch dimensions (Binder, 2004). Functional crossbites in the primary dentition caused by dental interferences can be sometimes corrected permanently by eliminating the interferences or premature contacts by selective grinding of the teeth involved (Kurol & Berglund, 1992; Thilander, Wahlund, Lennartsson, 1984). This grinding technique is effective only if the maxillary intercanine width is greater than the mandibular intercanine width by 2-3mm (Lindner, 1989). If this condition is not met, maxillary expansion is required (Lindner, 1989). Implant studies determined that transverse growth of the maxilla occurs at the same rate as body height changes (Björk, 1966; Björk & Skieller, 1977). The transverse width increase in the maxillary arch with expansion can be from either skeletal or dental changes. Ideal orthopedic treatment timing of posterior crossbite depends on the stage of mid palatal suture maturation as evaluated by histological studies (B. Melsen, 1975). Four stages have been described by Melsen; the first "Infantile" stage is until age 10 years old where the suture is broad with no interdigitations. The second "Juvenile" stage extends between 10 and 13 years of age and is characterized by some overlapping of the suture. The third "adolescent" stage occurs between 13 and 14 years of age where the suture has more interdigitations. The last "adult" stage characterized by "synostoses and numerous bony bridge formation across the suture" (Baccetti, Franchi, Cameron, McNamara, 2001; Melsen, 1975; Melsen & Melsen, 1982). It is therefore more difficult to achieve orthopedic expansion as the patient ages (Baccetti, Franchi, Cameron, McNamara, 2001). The orthopedic effect can be accomplished when the maxillary expansion occurs before (Hicks, 1978; Proffit, Fields, Sarver, 2006) or during the pubertal growth spurt (Haas, 1970; Hicks, 1978; Proffit, Fields, Sarver, 2006).

However, it is difficult to get an orthopedic effect after the pubertal growth spurt (Haas, 1970; Hicks, 1978; Proffit, Fields, Sarver, 2006).

1.5.1 Rapid versus Slow Maxillary Expansion

The rate of maxillary expansion is the variable most studied to determine the maxillary changes (Bell, 1982). A fixed jackscrew appliance is usually used for rapid maxillary expansion; the appliance is usually activated at a rate of two turns/day (0.25mm of expansion per turn) and active treatment time takes up to 2-3 weeks (Proffit, Fields, Sarver, 2006). The rate of expansion varies between patients because it depends on the patient's tolerance and the amount of expansion required (Bell, 1982). A single turn can produce a force magnitude of 10 pounds; so multiple turns per day can produce high amounts of forces which results in skeletal effects by transmitting the force from the teeth to the sutures (Proffit, Fields, Sarver, 2006). The skeletal and dental contribution from rapid palatal expansion was found to be equal by the 10th week of treatment (Proffit, Fields, Sarver, 2006). In order to improve stability and minimize relapse of the posterior crossbite correction, over expansion is required until the maxillary lingual cusps contact the lingual surface of the mandibular buccal cusps and the appliance should be used as a retainer (Proffit, Fields, Sarver, 2006) . Inadequate retention time results in relapse of the transverse width (Storey, 1973). Three months of retention time following rapid expansion is recommended to allow physiological adaptation of the sutures (Agarwal & Mathur, 2010; Proffit, Fields, Sarver, 2006). One effect of rapid palatal expansion is nasal cavity width increase which has been associated with reduction in airway resistance and improved breathing (Gray, 1975; Oliveira De Felippe, Da Silveira, Viana, Kusnoto, Smith, Evans,

2008). Another effect is the development of a midline diastma during expansion, as the suture opens, which closes spontaneously later on mainly by tooth tipping (Agarwal & Mathur, 2010; Proffit, Fields, Sarver, 2006).

Rapid maxillary expansion is usually done in the early permanent dentition. However, if earlier treatment during the primary or mixed dentition is desired, it can be accomplished by using either fixed or removable slow expansion appliances. These also increase the transverse dimensions by opening the mid-palatal suture (Bishara & Staley, 1987; Proffit, Fields, Sarver, 2006). The rate of activation of a fixed slow expansion appliance with a fixed jackscrew is about 2-3 turns/week which equals to approximately 0.5-1mm/week (Bell, 1982; Proffit, Fields, Sarver, 2006). If a removable appliance is used then the rate will be 1 turn/week or the appliance will be dislodged. The force magnitude produced by slow expansion is about 2 pounds. Slow maxillary expansion occurs at a rate that allows the suture physiological adaptation and remodeling (Proffit, Fields, Sarver, 2006). The net result over a 10 week period with slow expansion is 5mm of dental and 5mm of skeletal expansion which is identical to rapid expansion (Proffit, Fields, Sarver, 2006). However, slow maxillary expansion is not associated with the development of midline diastema unlike rapid maxillary expansion (Proffit, Fields, Sarver, 2006). Also, slow maxillary expansion is associated with less relapse and better stability (Bishara & Staley, 1987) if followed by adequate retention which is usually about 2-3 months (Bell & LeCompte, 1981; Hicks, 1978; Proffit, Fields, Sarver, 2006). Animal studies have compared the effects of rapid and slow maxillary expansion; it showed that slow expansion was associated with less tipping of the abutment teeth, less root resorption of the deciduous teeth (Ohshima, O., 1972), greater sutural stability (Ohshima, 1972; Storey, 1973) and less relapse potential than rapid expansion (Storey,

1973). Another consideration in minimizing relapse is the type of retention (Hicks, 1978). Following slow maxillary expansion and 8 weeks of fixed retention skeletal changes were stable. Removable retention had 10-23% loss of the increased arch width 2-6 weeks following expansion. The greatest relapse of 45% in arch width was without any retention 3 weeks following expansion (Hicks, 1978). However, the sample size for this study was only 5 patients (Hicks, 1978).

According to Petren et al. the success rate of posterior crossbite treatment during primary and mixed dentition varies according to the modality used (Petrén, Bondemark, Söderfeldt, 2003). The success rates of selective grinding during primary dentition ranged between 27 and 79% (Thilander, Wahlund, Lennartsson, 1984; Tsarapatsani, Tullberg, Lindner, Huggare, 1999; Kurol & Berglund, 1992; Lindner, 1989). The success rate using Quadhelix during both primary and mixed dentition is almost 100% (Bell & LeCompte, 1981; Bjerklin, K., 2000; Erdinç, Ugur, Erbay, 1999; Sandikçioğlu & Hazar, 1997; Tsarapatsani, Tullberg, Lindner, Huggare, 1999); the success rate of rapid maxillary expansion (Hyrax) during the mixed dentition is almost 100% too (Sandikçioğlu & Hazar, 1997); the lowest success rates of 51-100% were with the use of removable appliances (Bjerklin, K., 2000; Erdinç, Ugur, Erbay, 1999; Sandikçioğlu & Hazar, 1997; Thilander, Wahlund, Lennartsson, 1984).

1.5.2 Treatment Effects on the Maxilla

Skeletal maxillary sutures have different strength (Wertz, 1970) which result in not uniform expansion. Superior-inferiorly, the orthopedic expansion is greater towards the teeth inferiorly and less towards the nose superiorly (Proffit, Fields, Sarver, 2006; Wertz, 1970).

Antero-posteriorly the mid-palatal suture opening is greater at the anterior region compared to the posterior region (Proffit, Fields, Sarver, 2006; Wertz, 1970).

Studies have determined the effects of expansion on the dentition, arch form and palatal dimensions during primary, mixed and permanent dentition. A comparison was made between the untreated individuals with narrow arches to controls with average arch forms; patients with narrow arch forms became average arch forms following expansion (Spillane & McNamara, 1995).

Skeletal effects measured on PA cephalometric radiographs following expansion showed an increase in the facial and maxillary widths during treatment and retention (Brin, Ben-Bassat, Blustein, Ehrlich, Hochman, Marmary, Yaffe, 1996). This expansion was greater than the normal growth effect and was statistically significant; post retention, the maxillary and facial width in the treatment group was significantly below the average widths (Brin, Ben-Bassat, Blustein, Ehrlich, Hochman, Marmary, Yaffe, 1996).

Bacceti et al. compared the short and long term effects of rapid maxillary expansion with a Haas expander between groups of patients treated before or after the growth spurt determined by the Cervical Vertebral Maturation (CVM) method (Baccetti, Franchi, Cameron, McNamara, 2001). The late treatment group involved mainly dentoalveolar structure changes, while the early treatment group had skeletal changes with increased maxillary width and improved long term stability (Baccetti, Franchi, Cameron, McNamara, 2001). Bartzela et al. compared the long term effects of slow and rapid maxillary expansion on the treatment of crossbite in the early and late mixed dentition (Bartzela & Jonas, 2007). The late treatment groups with slow or rapid maxillary expansion had the highest amount of increased arch width (Bartzela & Jonas, 2007). The ideal posterior arch width, according to the modified

Pont's Index, was present in the both slow expansion treatment groups at the end of the active treatment; but it was stable only in the late slow expansion group at the 2 year follow up (Bartzela & Jonas, 2007). The arch width expansion in the late slow expansion treatment group was about 3.1+/- 2.3 mm (Bartzela & Jonas, 2007). The early rapid expansion treatment group had the highest relapse rate of about 24% (Bartzela & Jonas, 2007). Brin et al. found that treatment with a slow removable expander and 6 months of retention resulted in a mean increase in the intermolar width of about 3mm; the width of the treated group was similar to the untreated controls (Brin, Ben-Bassat, Blustein, Ehrlich, Hochman, Marmary, Yaffe, 1996). However, Hicks found a range of arch width increase from 3.8-8.7mm in 10 and 15 years old patients treated with slow maxillary expansion (Hicks, 1978).

Wong et al. reported the effects of early mixed dentition slow maxillary expansion, using three devices (Haas type, Hyrax and Quadhelix); they studied arch circumference, arch length, intermolar width, intercanine width and molar angulation and compared it to controls with the same age and gender (Wong, Sinclair, Keim, Kennedy, 2011). Measurements were made at three time intervals (T1= pre-treatment ~7 years, T2= Post treatment ~8 years, T3= 4 years after expansion ~12 years with no treatment performed between T2 and T3). Arch circumference increased by 1mm from T1 to T3, arch length slightly decreased from T1 to T3. The arch width increased at T2 and became wider than the controls but at T3 the intermolar width was similar to the controls with 80% intermolar expansion stability. The intercanine width remained significantly wider than the controls at T3 with 98% stability. The intermolar angulation showed an increase at T2 then decreased at T3 with molar uprighting but this was not compared to controls (Wong, Sinclair, Keim, Kennedy, 2011).

1.5.3 Palatal Shape and Dimensions Following Expansion:

Primožič et al. studied the effect of expansion on palatal dimensions in primary dentition patients with unilateral posterior crossbite and compared to controls with normal occlusion (Primožič, Ovsenik, Richmond, Kau, Zhurov, 2009). Before treatment, the palatal volume in the crossbite group was smaller than the control group; after treatment, the crossbite group had an increase in the palatal volume (339.5 mm^3) which was significantly greater than the increase in the untreated control group (19.4 mm^3) (Primožič, Ovsenik, Richmond, Kau, Zhurov, 2009). This change increased the similarity in the palatal volume between the two groups (Primožič, Ovsenik, Richmond, Kau, Zhurov, 2009). A randomized clinical trial on unilateral posterior crossbite patients treated by RME during the late deciduous or early mixed dentition were compared to untreated patients with the same condition (Lippold, Stamm, Meyer, Végh, Moiseenko, Danesh, 2013). Maxillary transverse dimensions, palatal width, maxillary arch length and inclination, palatal depth, mandibular transverse dimensions, midline deviation, overbite and overjet were measured. Significant differences in the transverse width between the two groups were found. The transverse palatal width increased in the treatment group; the palatal depth showed significant reduction following treatment in the region of the first primary molars and a significant difference remained between the two groups at one year follow up. For the treatment group, the inclination of the maxillary arch showed significant increase. Compared to controls, there was a significant increase in the bite depth and significant reduction of the midline deviation of the treatment group (Lippold, Stamm, Meyer, Végh, Moiseenko, Danesh, 2013). Marini et al. evaluated transverse palatal volume changes following RME in early mixed dentition between three time intervals T1 (before treatment), T2 (after expansion and retention for 3

months) and T3 (six months follow up without any orthodontic intervention) (Marini, Bonetti, Achilli, Salemi, 2007). At T3, the transverse dimensions decreased significantly however the increase in palatal volume was stable (Marini, Bonetti, Achilli, Salemi, 2007). Muchitsch et al. reported that following treatment of mixed dentition posterior crossbite by rapid maxillary expansion, using cemented acrylic splints in children, there was a significant increase in the palatal widths as well as the cross sectional areas of the primary canines, primary second molars and permanent first molars (Muchitsch, Winsauer, Wendl, Pichelmayer, Kuljuh, Szalay, Muchitsch 2012). There were significant minute reductions in the palatal height in the area of the second primary molars and decreased palatal length (Muchitsch, Winsauer, Wendl, Pichelmayer, Kuljuh, Szalay, Muchitsch 2012).

Oliveira et al. studied late mixed dentition children with posterior crossbite and comparing the effects of rapid palatal expansion with the Haas and Hyrax appliances on the palate and dentition (Oliveira, Da Silveira, Kusnoto, Viana, 2004). They found that the Haas appliance had more skeletal effect by expanding the palate while the Hyrax appliance caused more dental expansion (Oliveira, Da Silveira, Kusnoto, Viana, 2004). The palatal total area showed an increase in both groups, and intermolar width was increased more in the Haas group as was palatal flattening which had a small insignificant frequency (Oliveira, Da Silveira, Kusnoto, Viana, 2004). Oliveira De Felippe et al. also reported the effects of rapid expansion in 13 years old children on nasal cavity size, airway resistance, palatal surface area, palatal volume, intermolar distance, palatal inclination and palatal flattening (Oliveira De Felippe, Da Silveira, Viana, Kusnoto, Smith, Evans, 2008). The palatal area and intermolar distance increased post expansion but it decreased in the long term follow up (Oliveira De Felippe, Da Silveira, Viana, Kusnoto, Smith, Evans, 2008). By contrast the

palatal volume increased and remained the same in the long term follow up (Oliveira De Felippe, Da Silveira, Viana, Kusnoto, Smith, Evans, 2008). Gohl et al. measured the changes in the palate following rapid maxillary expansion in subjects with mean age of ~13 years and compared them to controls undergoing orthodontic treatment using CBCT. Significant increase in palatal volume of the expansion group was mainly due to increase in the intermolar and intercanine widths, but there were no significant change in the palatal height or length (Gohl, Nguyen, Enciso, 2010). Handelman et al. reported the effects of rapid maxillary expansion on the palate and dentition in adults and compared them to children treated with rapid maxillary expansion and non-treated controls (Handelman, Wang, BeGole Haas, 2000).

To summarize only one study evaluated changes in the palate after slow maxillary expansion and was done in the primary dentition stage (Primožič, Ovsenik, Richmond, Kau, Zhurov, 2009). All the other studies were done with rapid maxillary expansion during late deciduous, mixed and permanent dentition stages (Lippold, Stamm, Meyer, Végh, Moiseenko, Danesh, 2013; Marini, Bonetti, Achilli, Salemi, 2007; Muchitsch, Winsauer, Wendl, Pichelmayer, Kuljuh, Szalay, Muchitsch 2012; Oliveira, Da Silveira, Kusnoto, Viana, 2004; Oliveira De Felippe, Da Silveira, Viana, Kusnoto, Smith, Evans, 2008; Gohl, Nguyen, Enciso, 2010; Handelman, Wang, BeGole, Haas, 2000); some of the rapid maxillary expansion studies that were done on mixed dentition lacked controls (Muchitsch, Winsauer, Wendl, Pichelmayer, Kuljuh, Szalay, Muchitsch 2012) and only one study had a short term follow up without post expansion orthodontic treatment (Marini, Bonetti, Achilli, Salemi, 2007). Finally, during the mixed dentition stage the focus was on palatal changes following rapid maxillary expansion, so the literature lacks studies evaluating the palatal dimensional

changes following slow maxillary expansion during mixed dentition. All previous studies that measured the molar angle on models used either the intermolar angle or the buccal inclination of each molar using 2 cusp tips.

1.6 Objectives

The purpose of this study is to evaluate initial palatal dimensions, symmetry and molar angulation changes following slow maxillary expansion during the early mixed dentition compared to untreated controls.

1.6.1 Research Questions

Does slow maxillary expansion with a Haas type appliance in the early mixed dentition change palate dimensions and first permanent molar angulations?

Are the palatal halves symmetric in unilateral posterior crossbite before treatment?

Are the palatal halves symmetric in unilateral posterior crossbite after treatment?

1.6.2 Study Hypotheses

Palatal dimensions (width, surface area and volume) and first permanent molar angulations will change following slow maxillary expansion during the early mixed dentition.

The palatal halves are asymmetric before treatment.

The palatal halves are asymmetric after treatment.

1.6.3 Null Hypotheses

No changes will occur in the palatal dimensions and first permanent molar angulations following slow maxillary expansion during the early mixed dentition.

The palatal halves are symmetric before treatment.

The palatal halves are symmetric after treatment.

Chapter 2: Methodology

2.1 Materials and Methods

This retrospective case control study was reviewed and the approval was acquired from the clinical Ethics Board at the University of British Columbia, Vancouver, Canada (H14-02193).

2.1.1 Sample Size Calculation

A power calculation was performed regarding palatal volume assuming a mean difference of 339.52 mm^3, SD of 427.80 as per Primozic et al. (Primožič, Ovsenik, Richmond, Kau, Zhurov, 2009), 80% power and α=0.05 a sample of 25 subjects was required in each group.

2.1.2 Subjects

The treatment sample consisted of 30 subjects who were treated for unilateral posterior crossbite with a functional shift with a Haas-type appliance at a private dental office. Records were taken at two time intervals T1= before expansion (mean dental age of 8 years old) and T2= after expansion (dental age of 9 years old). The untreated control sample consisted of 30 subjects from the Oregon Health and Sciences University. The untreated control sample and treated sample were matched for gender, dental classification and dental age using the method described by Demirjian & Goldstein (Demirjian & Goldstein, 1976). Inclusion criteria for the treatment sample were pretreatment unilateral posterior crossbite with functional shift, slow maxillary expansion done in the mixed dentition and non-

extraction cases. Exclusion criteria were Growth modification appliances: HG, facemask, etc., ectopic palatal lateral incisors, attrition affecting first molar cusps, craniofacial anomalies, eruption of permanent successors (canines and premolars) and poor quality models. The expansion protocol for SME was 1 turn every 2 days (0.25 mm every 2 days) until the posterior crossbite was overcorrected with the buccal inclines of the maxillary lingual cusps contacting the lingual inclines of the mandibular buccal cusps. Then the expander was left passively for retention for a minimum of 6 months. The principal investigator was blinded for the crossbite and non crossbite sides until the whole data was collected.

Table 2.1 Matching of Study Groups

Groups	**N**	**Gender^**	
		Males	Females
Control Sample	30	11	19
Treatment Sample	30	11	19
Significance		P=1.00	
	Dental Age#		
	T1 Mean ± SD		**T2** Mean ± SD
Control Sample	8.30 ± 0.75		9.42 ± 0.92
Treatment Sample	8.36 ± 0.70		9.47 ± 0.94
Significance	P=0.890		P=0.860

^ Chi Squared test; # Independent sample t test

2.1.3 Model Analysis

The maxillary models were scanned using the Ortho Insight 3D™ scanner (Figure 2.1). Measurements of palatal width, surface area, volume and molar angulation were done on digitized models using Rhinoceros 5.0 software (Figure 2.2).

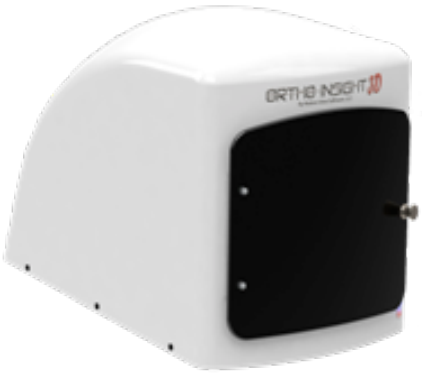

Figure 2.1 Ortho Insight 3D™

Figure 2.2 Rhinoceros 5.0 Software

Points were placed at lingual gingival margin of the greatest convexity of the teeth from canines to first permanent molars, on the four first permanent molar cusp tips, on the mid distal surface of the incisive papilla and along the mid palatal raphe which was determined by the anatomical landmarks in the mid palatal region (Figure 2.3). These

midline landmarks were usually between the rugae anteriorly and more posteriorly by either one or two lines running along the midline. In the case of one line, the points were placed on it, if there were two lines then the points were placed between these two lines.

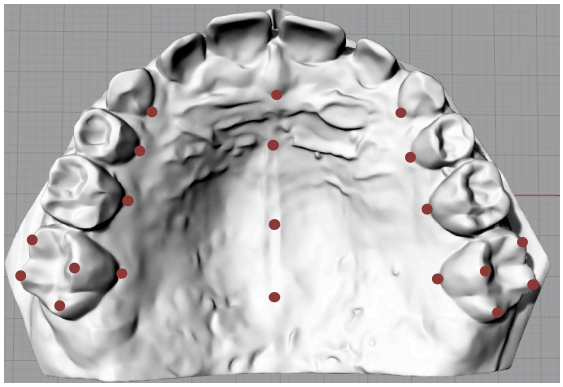

Figure 2.3 Landmark Identification on the Digitized Models

These points were used to construct the lines, surfaces and planes which were used for the analyses.

2.1.3.1 Palatal Widths

Widths were measured from a straight line at the canine and first permanent molar areas (Figure 2.4).

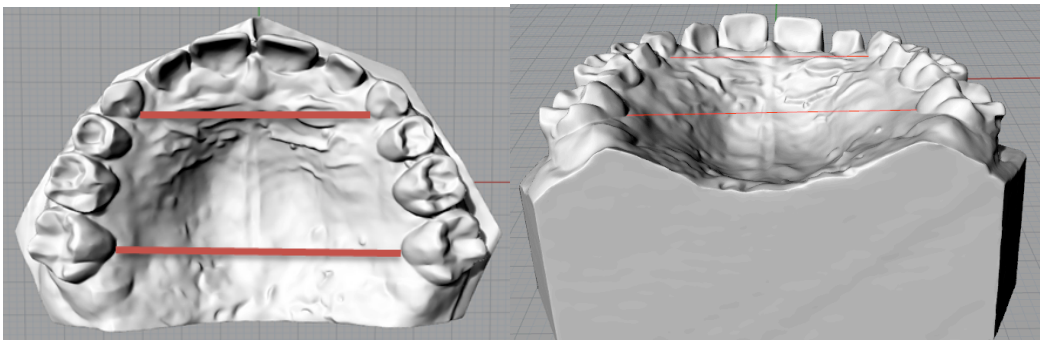

Figure 2.4 Intercanine and Intermolar Widths

2.1.3.2 Palatal Surface Area

The palatal surface area was assessed by constructing curves through the points; these curves were rebuilt by the software to 30 points. The total surface area was measured. Then the surface area was divided by inter 1st primary molar and inter 2nd primary molar planes into three areas: anterior, middle and posterior. These areas were divided by the midpalatal plane into halves to measure symmetry between the halves (Figure 2.5).

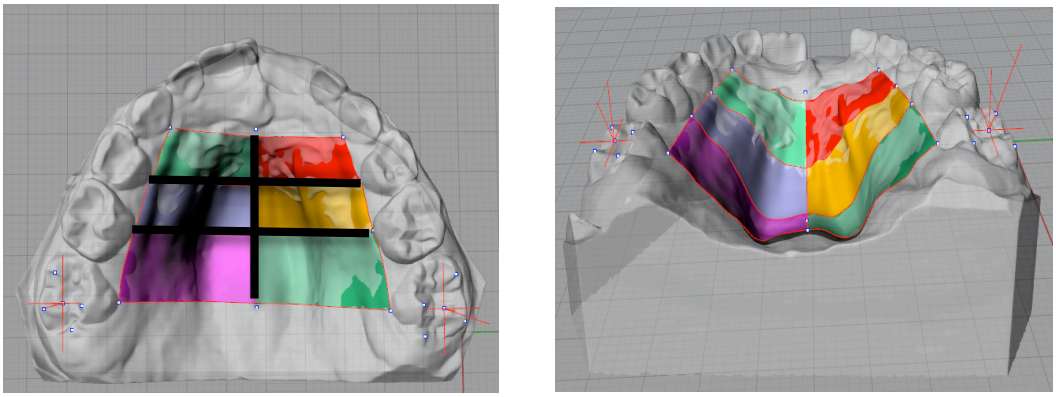

Figure 2.5 Palatal Surface Area Divided into Six Halves

2.1.3.3 Palatal Volume

Palatal volume was measured from the area enclosed between the palatal surface and a horizontal plane passing through the points at the gingival margins of the teeth. The total volume was measured; then the constructed volume was divided into six volumes similar to that described for the palatal surface area. The volume of each six areas was individually measured (Figure 2.6).

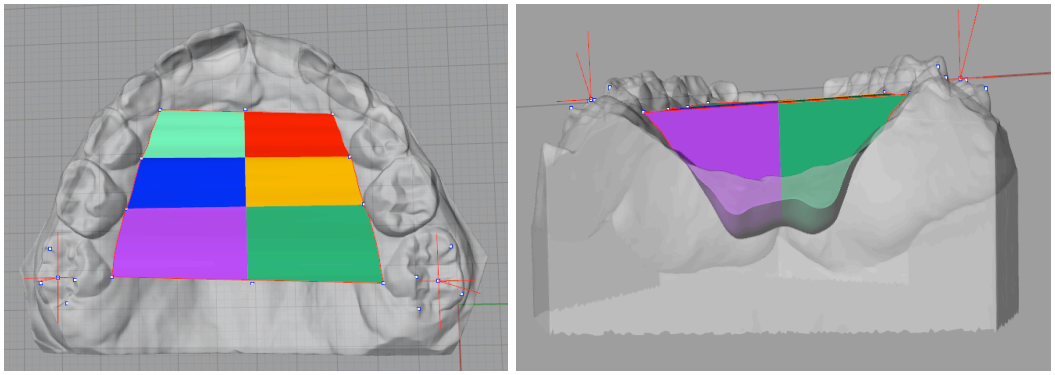

Figure 2.6 Palatal Volume Divided into Six Halves

2.1.3.4 Molar Angulation

The 3D angulation of each permanent first molar was expressed by constructing the normal to a rectilinear planar surface fitted through the cusp tips, and by measuring its relationship to the normal of a plane fitted through the points describing the palatal gingival margins of the maxillary teeth. The orientations of these normals were expressed relative to a common origin placed at the centre of the molar planar surface (Figure 2.7). To measure the angular differences between them, both normals were projected onto lateral and frontal reference planes. The lateral plane was oriented perpendicular to the horizontal plane, and parallel to the midpoints between the canine and molar gingival points. The frontal plane was perpendicular to the lateral plane, and placed anterior to the molars. The projections on these two planes thus allowed each molar's 3D inclination to be expressed as two angles, i.e. mesiodistally and buccolingually. In the lateral plane, the angle between the molar normal and the palatal normal was signed positively if the molar normal was tipped distally. In the frontal plane, it was positive if the molar normal tipped buccally (Figure 2.7).

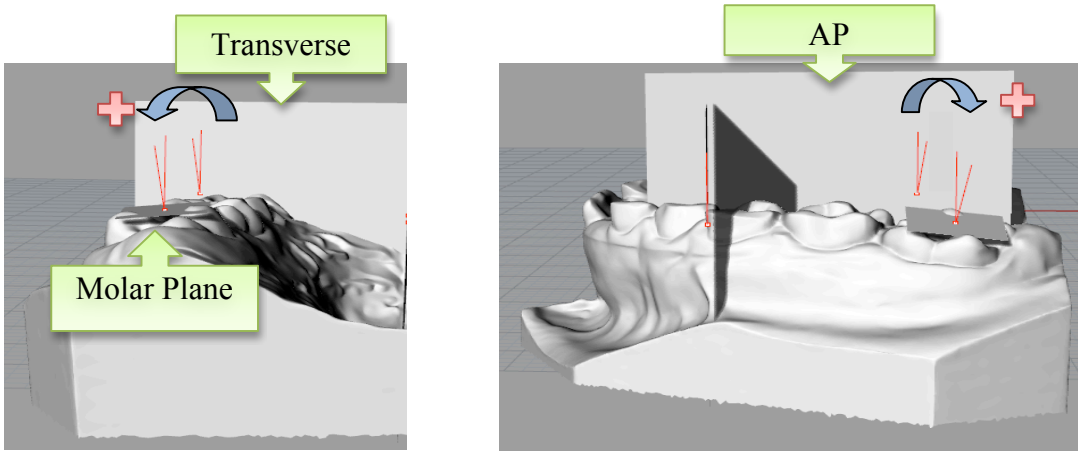

Figure 2.7 Buccolingual and Mesiodistal Molar Angulation Measurements

2.1.4 Assessment of Intra-Examiner Agreement

Intra-examiner agreement was done by repeating the measurements on 15 models 1 week apart. Correlation coefficient was used to determine the agreement and independent t-test was used to confirm that by determining significance ($P<0.05$).

2.1.5 Statistical Analysis

In order to choose appropriate statistical tests the results were assessed for normality. The skewness and kurtosis values did not exceed ±1.96, therefore parametric statistics were used.

All statistical analyses were conducted using SPSS version 21.0 statistical software. Student's t-test was performed to assess the differences between the two groups: two-tailed

paired used to compare between sides and within the groups while unpaired used to compare between the groups with a threshold for statistical significance level of $P< 0.05$.

Chapter 3: Results

3.1 Intra Examiner Agreement

The range of intra-class correlation coefficient was from 0.7-1 which meant good to excellent intra examiner agreement. This was also confirmed by a t-test, which showed no significant differences between the first and second measurements.

3.2 Variables Measured

3.2.1 Palatal Width (mm)

Intercanine and Intermolar widths are shown in Table 3.2, Figures 3.1 and 3.2.

Figures show range, median and outliers. The treatment group is represented in red and control group is represented in green at both time intervals (T1 & T2).

3.2.1.1 Intercanine Width

The mean intercanine width (ICW) in the treatment group at T1 was 22.74mm $\pm$ 2.15 while for the control group it was 24.35mm $\pm$ 2.04; this difference was statistically significant (P= 0.004). After treatment, the mean ICW was 27.39mm $\pm$ 2.05 and for the control group it was 24.92mm $\pm$ 2.17; this difference was statistically significant (P< 0.001). The mean increase in ICW from T1-T2 in the treatment group was 4.65mm (Range= 3.56 - 5.73) which was statistically significant (P< 0.001). The mean ICW increase from T1-T2 in the control group was 0.58mm (Range= (-0.51) - 1.67) which was not significant (P= 0.294).

3.2.1.2 Intermolar Width

The mean intermolar width (IMW) in the treatment group at T1 was 30.32mm ± 2.8 while for the control group it was 30.75mm ± 2.20; this difference was not statistically significant (P= 0.51). After treatment, the mean IMW was 35.08mm ± 3.03 and for the control group it was 31.22mm ± 2.40; this difference was statistically significant (P< 0.001). The mean IMW increase from T1-T2 in the treatment group was 4.76mm (Range= 3.25 - 6.27) which was statistically significant (P< 0.001). The mean increase in the control group was 0.47mm (Range= (-0.72) - 1.66) which was not significant (P= 0.432).

Table 3.1 Palatal Width

Palatal width (mm)	**Treatment Sample**		**Control Sample**		**P Value#**
Intercanine Width	N	Mean ± SD	N	Mean ± SD	
T1	30	22.74 ± 2.15	30	24.35 ± 2.04	P= 0.004
T2	30	27.39 ± 2.05	30	24.92 ± 2.17	P< 0.001
P Value^		P< 0.001		P= 0.294	
Intermolar width		Mean ± SD		Mean ± SD	
T1	30	30.32 ± 2.80	30	30.75 ± 2.20	P= 0.510
T2	30	35.08 ± 3.03	30	31.22 ± 2.40	P< 0.001
P Value^		P< 0.001		P= 0.432	

^ Paired sample t test; # Independent t-test

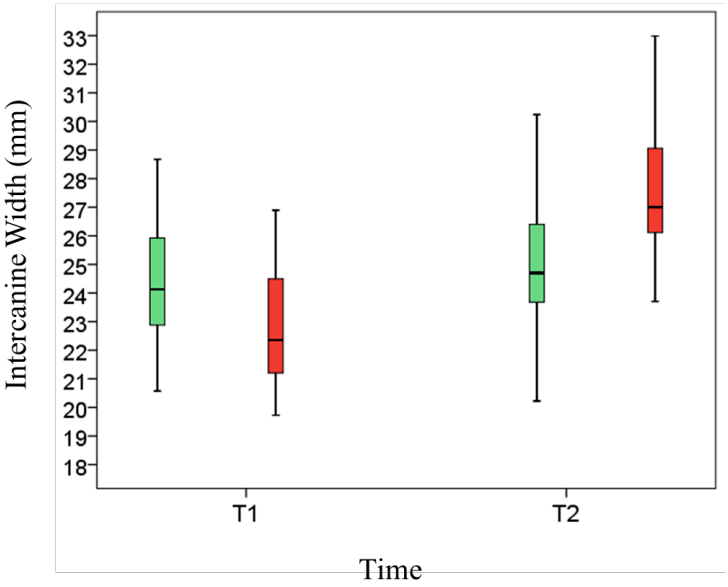

Figure 3.1 Intercanine Width - Comparison between the study group at T1 and T2. Treatment Sample (Red) and Control Sample (Green).

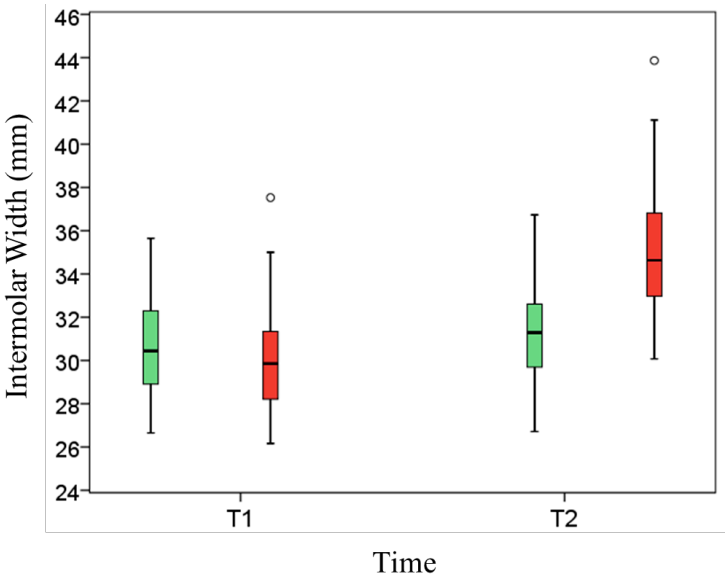

Figure 3.2 Intermolar Width - Comparison between the study groups at T1 and T2. Treatment Samples (Red) and Control Sample (Green)

3.2.2 Palatal Surface Area (mm^2)

Measurements of palatal surface areas are shown in Tables 3.3-3.8 and Figures 3.3-3.9.

Figures show range, median and outliers. The treatment group is represented in red and control group is represented in green at both time intervals (T1 & T2).

3.2.2.1 Overall Comparison Treatment Sample vs. Control Sample at T1/T2:

The control sample lost 3 cases at T2 due to a large bubble in the palate in one case and first premolar eruption in the other two cases. The mean total surface area in the treatment group at T1 was 832.98mm^2 $\pm$ 88.90 while for the control group it was 858.33mm^2 $\pm$ 70.60 and difference was not statistically significant (P= 0.227). The treated group showed a mean surface area of 960.03mm^2 $\pm$ 97.62 while the mean for the control group was 869.19mm^2 $\pm$ 84.23, with this difference being statistically significant (P< 0.001). The mean increase from T1-T2 in the treatment group was 127.05mm^2 (Range= 78.8 - 175.31) which was statistically significant (P< 0.001). The mean increase from T1-T2 in the control group was 10.86mm^2 (Range= (-30.72) - 52.44) which was not significant (P= 0.602).

Table 3.2 Total Palatal Surface Area

Total Surface area (mm^2)	**Treatment Sample**		**Control Sample**		**P Value#**
	N	Mean $\pm$ SD	N	Mean $\pm$ SD	
T1	30	832.98 $\pm$ 88.90	30	858.33 $\pm$ 70.60	P= 0.227
T2	30	960.03 $\pm$ 97.62	27	869.19 $\pm$ 84.23	P< 0.001
P Value^		P< 0.001		P= 0.602	

^ Paired sample t test; # Independent t-test

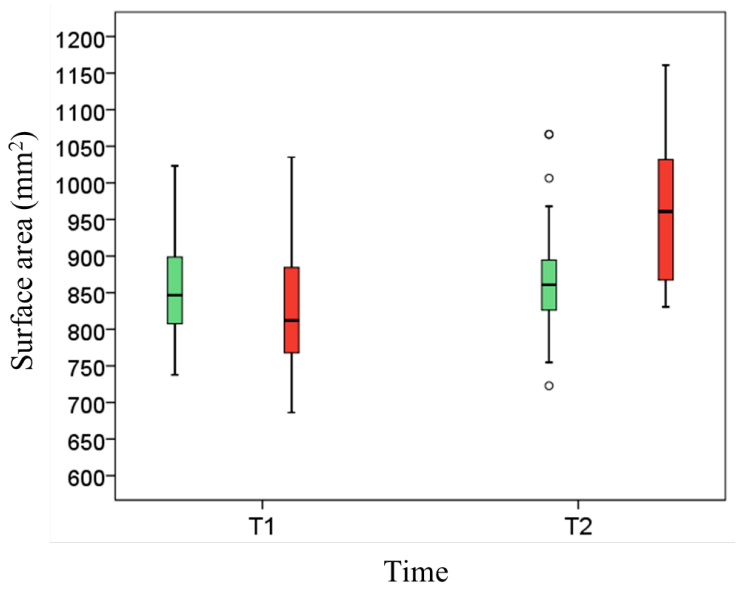

Figure 3.3 Total Palatal Surface Area - Comparison between the study groups at T1 and T2. Treatment Sample (Red) and Control Sample (Green).

3.2.2.2 Anterior Palatal Halves Treatment Sample vs. Control Sample:

The mean surface area of the anterior halves on the crossbite side at T1 in the treatment group was slightly smaller than the corresponding side in the control group but not statistically significant (P= 0.400). On the non-crossbite side no significant difference between the treated and control groups were noted (P= 0.152). After expansion the mean surface areas on the crossbite and non crossbite side were significantly greater in the treatment group than the control group (P= 0.001 and P= 0.003, respectively). The mean increase from T1-T2 in the treatment group on the crossbite side was 23.56mm^2 (Range= 13.69 - 33.43) and on the non crossbite side it was 22.16mm^2 (Range= 12.70 - 31.62) and both were statistically significant (P< 0.001). The mean change in the control group from T1-T2 on the side corresponding to the crossbite side in the treatment group was 0.41mm^2

(Range= (-9.94) - 10.76); on the other half it was -1.18mm^2 (Range= (-11.64) - 9.28) neither change was statistically significant (P= 0.937 and P= 0.822, respectively).

Table 3.3 Palatal Surface Areqa Anterior Halves

Surface Area Anterior Halves (mm^2)	**Treatment Sample**		**Control Sample**		**P Value #**
Crossbite side	N	Mean ± SD	N	Mean ± SD	
T1	30	103.3 ± 15.74	30	107.1 ± 18.77	P= 0.400
T2	30	126.86 ± 21.88	27	107.5 ± 20.05	P= 0.001
P Value^		P< 0.001		P= 0.937	
Non-Crossbite side	N	Mean ± SD	N	Mean ± SD	
T1	30	103.83 ± 15.85	30	110.35 ± 18.80	P= 0.152
T2	30	125.99 ± 20.42	27	109.17 ± 20.42	P= 0.003
P Value^		P< 0.001		P= 0.822	

^ Paired sample t test; # Independent t-test

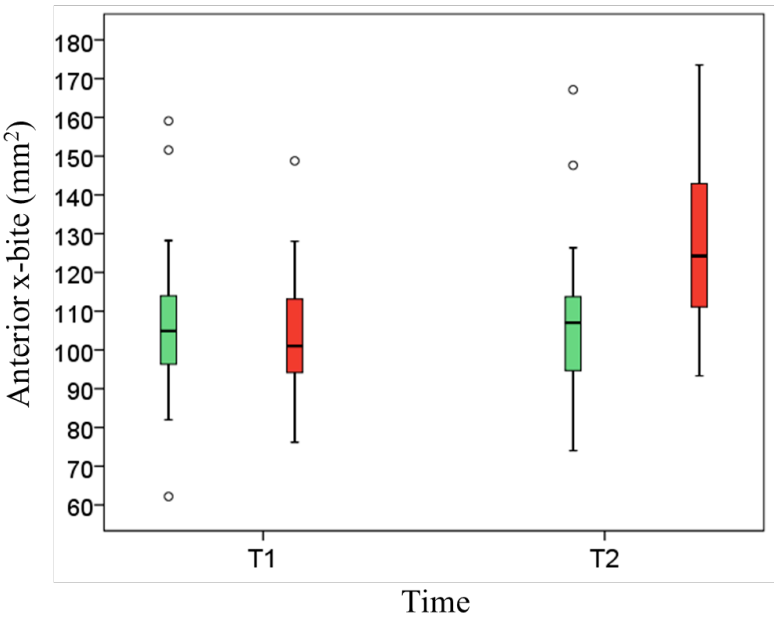

Figure 3.4 Palatal Surface Area Anterior Halves (X-Bite Side) - Comparison between the study groups at T1 and T2. Treatment Sample (Red) and Control Sample (Green).

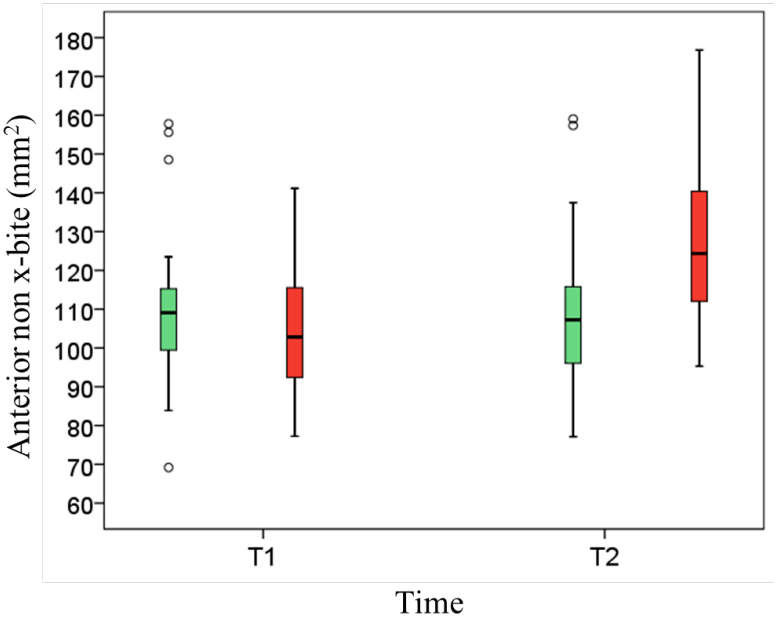

Figure 3.5 Palatal Surface Area Anterior Halves (Non X-Bite Side) - Comparison between the study groups at T1 and T2. Treatment Sample (Red) and Contrl Sample (Green).

3.2.2.3 Middle Palatal Halves Treatment Sample vs. Control Sample:

For the middle halves the mean surface areas on the crossbite side and non crossbite side at T1 in the treatment group were less than but not statistically significant different than the corresponding sides in the control group (P= 0.517 and P= 0.168 respectively). After expansion the mean surface areas on the crossbite side and non crossbite side were greater in the treatment group than the corresponding sides in the control group; the difference was significant on the crossbite side (P= 0.013) but not significant on the non crossbite side (P= 0.116). The mean increase from T1-T2 in the treatment group on the crossbite side was 15.13mm^2 (Range= 5.76 - 24.49) and on the non crossbite side it was 14.37mm^2 (Range= 5.35 - 23.39) and both were statistically significant (P< 0.001 and P= 0.002, respectively). The mean increase in the control group on the side corresponding to the crossbite side was 0.52mm^2 (Range= (-8.36) - 9.40), on the other half it was 0.83mm^2 (Range= (-8.29) - 9.94) with neither side statistically significant (P= 0.907 and P= 0.856, respectively).

Table 3.4 Palatal Surface Area Middle Halves

Surface Area Middle Halves (mm^2)	Treatment Sample		Control Sample		P Value#
Crossbite side	N	Mean ± SD	N	Mean ± SD	
T1	30	131.85 ± 18.43	30	134.83 ± 16.84	P= 0.517
T2	30	146.98 ± 17.81	27	135.35 ± 16.59	P= 0.013
P Value^		P< 0.001		P= 0.907	
Non-Crossbite side	N	Mean ± SD	N	Mean ± SD	
T1	30	129.09 ± 17.22	30	135.40 ± 17.8	P= 0.168
T2	30	143.46 ± 17.66	27	136.23 ± 16.52	P= 0.116
P Value^		P= 0.002		P= 0.856	

^ Paired sample t test; # Independent t-test

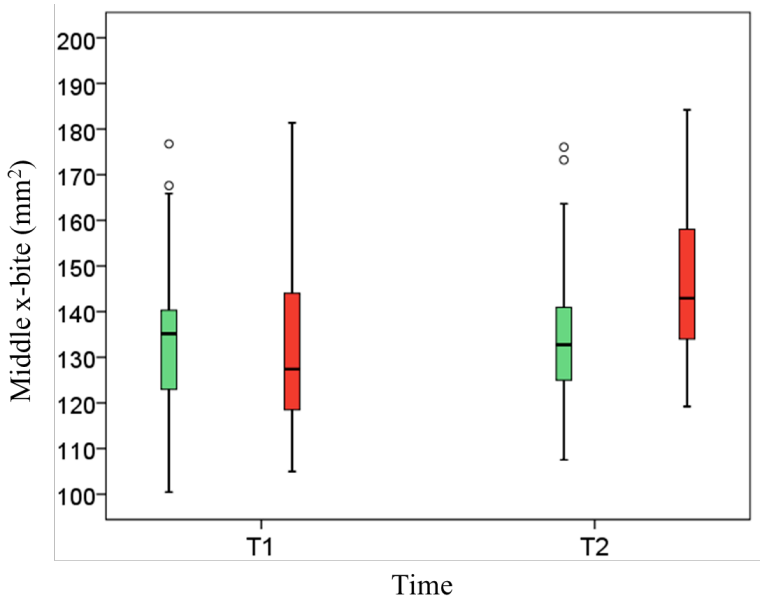

Figure 3.6 Palatal Surface Area Middle Halves (X-Bite Side) - Comparison between the study groups at T1 and T2. Treatment Sample (Red) and Control Sample (Green).

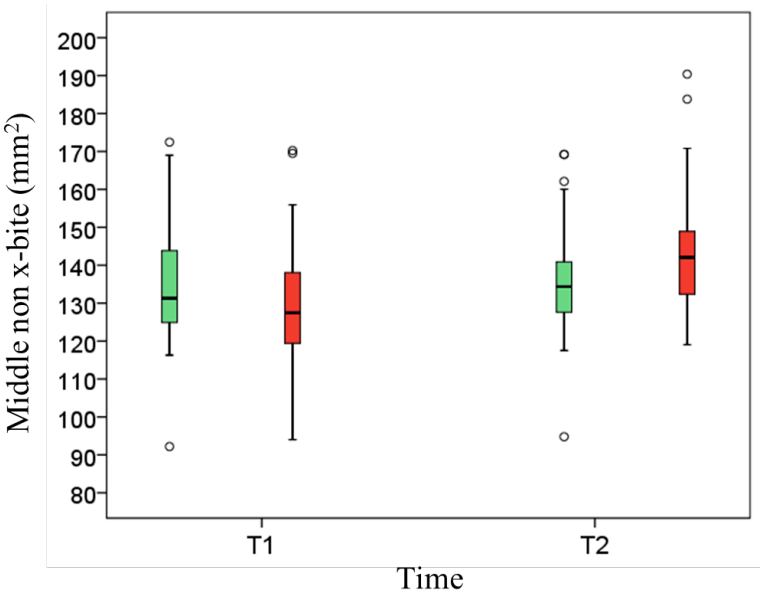

Figure 3.7 Palatal Surface Area Middle Halves (Non X-Bite Side) - Comparison between the study groups at T1 and T2. Treatment Sample (Red) and Control Sample (Green).

3.2.2.4 Posterior Palatal Halves Treatment Sample vs. Control Sample:

For the posterior halves, the mean surface areas on the crossbite side and non crossbite side at T1 in the treatment group were smaller than the corresponding sides in the control group but differences were not statistically significant (P= 0.749 and P= 0.224, respectively). After expansion (T2) the mean surface areas on the crossbite side and non crossbite side in the posterior halves was significantly greater in the treatment group than the corresponding sides in the control group; (P= 0.001and P= 0.003, respectively). The mean increase from T1-T2 in the treatment group on the crossbite side was 27.17mm^2 (Range= 15.83 - 38.5) and on the non crossbite side it was 25.26mm^2 (Range= 15.00 - 35.53) and both were statistically significant (P< 0.001). The mean increase in the control group on the side corresponding to the crossbite side was 6.55mm^2 (Range= (-3.27) - 16.37), on the other half

it was 2.68mm^2 (Range= (-7.47) - 12.83) with neither side statistically significant (P= 0.187 and P= 0.598, respectively).

Table 3.5 Palatal Surface Area Posterior Halves

Surface area Posterior halves (mm^2)	**Treatment Sample**		**Control Sample**		**P Value#**
Crossbite side	N	Mean ± SD	N	Mean ± SD	
T1	30	183.14 ± 21.22	30	184.71 ± 16.30	P= 0.749
T2	30	210.31 ± 22.62	27	191.26 ± 20.17	P= 0.001
P Value^		P< 0.001		P= 0.187	
Non-Crossbite side	N	Mean ± SD	N	Mean ± SD	
T1	30	180.67 ± 21.23	30	186.78 ± 17.00	P= 0.224
T2	30	205.94 ± 18.38	27	189.46 ± 20.74	P= 0.003
P Value^		P< 0.001		P= 0.598	

^ Paired sample t test; # Independent t-test

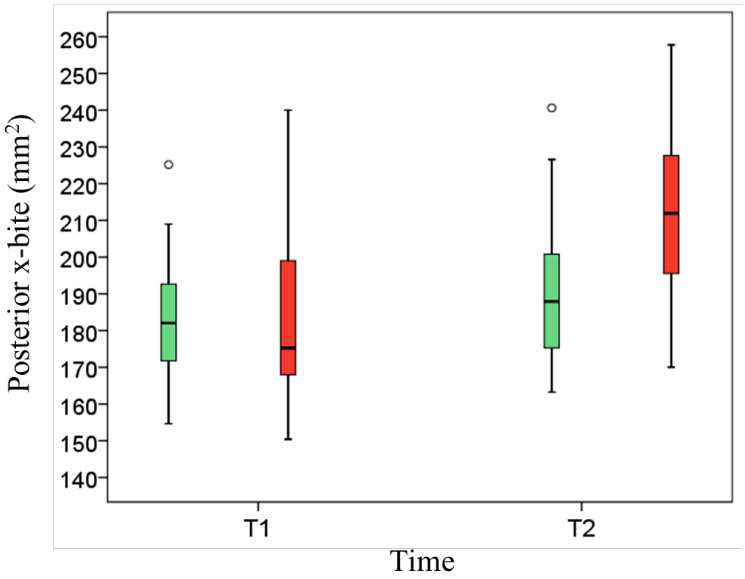

Figure 3.8 Palatal Surface Area Posterior Halves (X-Bite Side) - Comparison between the study groups at T1 and T2. Treatment Sample (Red) and Control Sample (Green).

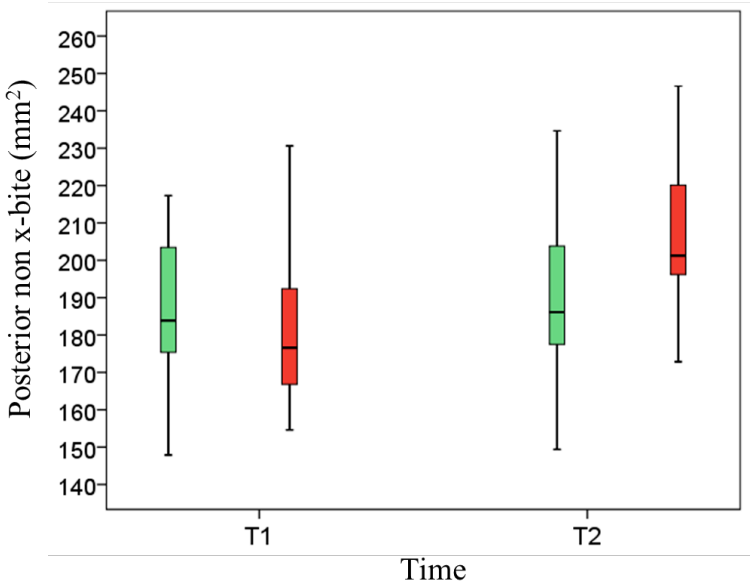

Figure 3.9 Palatal Surface Area Posterior Halves (Non X-Bite Side) - Comparison between the study groups at T1 and T2. Treatment Sample (Red) and Control Sample (Green).

3.2.2.5 Crossbite vs. Non Crossbite Sides

When comparing palatal surface area between the crossbite side and non crossbite side in the treatment sample and between the corresponding sides in the control sample at both time intervals (T1 & T2) there were no significant differences except for the middle halves in the treatment group at T2 (P= 0.047) and the anterior halves in the control group at T1 (P= 0.23).

Table 3.6 Palatal Surface Area Symmetry (Treatment Sample)

Surface area (mm^2) **Treatment Sample**	**Time**	**Crossbite side** Mean ± SD	**Non-Crossbite side** Mean ± SD	**P Value^**
Anterior	**T1**	103.3 ± 15.74	103.83 ± 15.85	P= 0.725
	T2	126.86 ± 21.88	125.99 ± 20.42	P= 0.560
Middle	**T1**	131.85 ± 18.43	129.09 ± 17.22	P= 0.110
	T2	146.98 ± 17.81	143.46 ± 17.66	P= 0.047
Posterior	**T1**	183.14 ± 21.22	180.67 ± 21.23	P= 0.169
	T2	210.31 ± 22.62	205.94 ± 18.38	P= 0.082

^ Paired sample t test

Table 3.7 Palatal Surface Area Symmetry (Control Sample)

Surface area (mm^2) Control Sample		Crossbite side Mean ± SD	Non-Crossbite side Mean ± SD	P Value^
Anterior	**T1**	107.10 ± 18.77	110.35 ± 18.80	P= 0.023
	T2	107.50 ± 20.05	109.17 ± 20.42	P= 0.233
Middle	**T1**	134.83 ± 16.84	135.40 ± 17.80	P= 0.760
	T2	135.35 ± 16.59	136.23 ± 16.52	P= 0.562
Posterior	**T1**	184.71 ± 16.30	186.78 ± 17.00	P= 0.229
	T2	191.26 ± 20.17	189.46 ± 20.74	P= 0.489

^ Paired sample t test

3.2.3 Palatal Volume (mm^3)

Palatal Volumes are shown in Tables 3.9-3.14 and Figures 3.10-3.16.

Figures show range, median and outliers. The treatment group is represented in red and control group is represented in green at both time intervals (T1 & T2).

3.2.3.1 Overall Comparison Treatment Sample vs. Control Sample at T1/T2:

The control sample had 27 cases at T2 similar to the surface area measurements. The mean volume in the treatment group at T1 was 3654.25mm^3 ± 726.32 while for the control group it was 3922.2mm^3 ± 605.20; this difference between them was not statistically significant (P= 0.126). After expansion the mean volume in the treatment group was 4581.80mm^3 ± 838.76 which was significantly larger than the control group it was 4082.76mm^3 ± 726.42 (P= 0.019). The mean increase from T1-T2 in the treatment group was

927.55mm^3 (Range= 521.89-1333.22) which was statistically significant (P< 0.001). The mean increase in the control group was 160.55mm^3 (Range= (-197.21) - 518.32) which was not significant (P= 0.372).

Table 3.8 Total Palatal Volume

Volume (mm^3)	Treatment Sample		Control Sample		P Value#
	N	Mean ± SD	N	Mean ± SD	
T1	30	3654.25 ± 726.32	30	3922.20 ± 605.20	P= 0.126
T2	30	4581.80 ± 838.76	27	4082.76 ± 726.42	P= 0.019
P Value^		P< 0.001		P= 0.372	

^ Paired sample t test; # Independent t-test

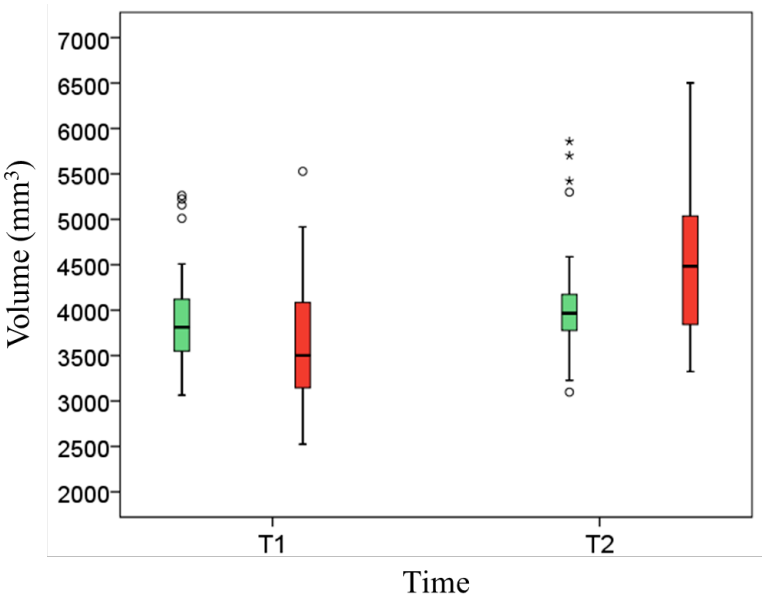

Figure 3.10 Total Palatal Volume - Comparison between the study groups at T1 and T2. Treatment Sample (Red) and Control Sample (Green).

3.2.3.2 Anterior Palatal Halves Treatment Sample vs. Control Sample:

When measuring the anterior halves, the mean volume on both the crossbite side and non crossbite side at T1 was less in the treatment group but not statistically significant than the corresponding sides in the control group (P= 0.300 and P= 0.139, respectively). After expansion the mean volumes on the crossbite side and the non crossbite side in the anterior halves became greater in the treatment group than the corresponding sides in the control group with a significant difference between them (P= 0.023 and P= 0.030, respectively). The mean increase from T1-T2 in the treatment group on the crossbite side was 115.59mm^3 (Range= 60.29 - 170.90) and on the non crossbite side it was 116.11mm^3 (Range= 60.83 - 171.39), both were statistically significant (P< 0.001). The mean change in the control group from T1-T2 on the side corresponding to the crossbite side in the treatment group was 16.94mm^3 (Range= (-41.82) - 75.71), on the other side it was 6.91mm^3 (Range= (-53.44) - 67.25) with neither statistically significant (P= 0.566 and P= 0.819, respectively).

Table 3.9 Palatal Volume Anterior Halves

Volume Anterior Halves (mm^3)	Treatment Sample		Control Sample		P Value#
Crossbite side	N	Mean ± SD	N	Mean ± SD	
T1	30	299.00 ± 95.00	30	326.50 ± 108.27	P= 0.300
T2	30	414.60 ± 117.63	27	343.44 ± 112.44	P= 0.023
P Value^		P< 0.001		P= 0.566	
Non-Crossbite side	N	Mean ± SD	N	Mean ± SD	
T1	30	298.88 ± 85.55	30	337.15 ± 110.41	P= 0.139
T2	30	414.99 ± 124.25	27	344.05 ± 116.11	P= 0.030
P Value^		P< 0.001		P= 0.819	

^ Paired sample t test; # Independent t-test

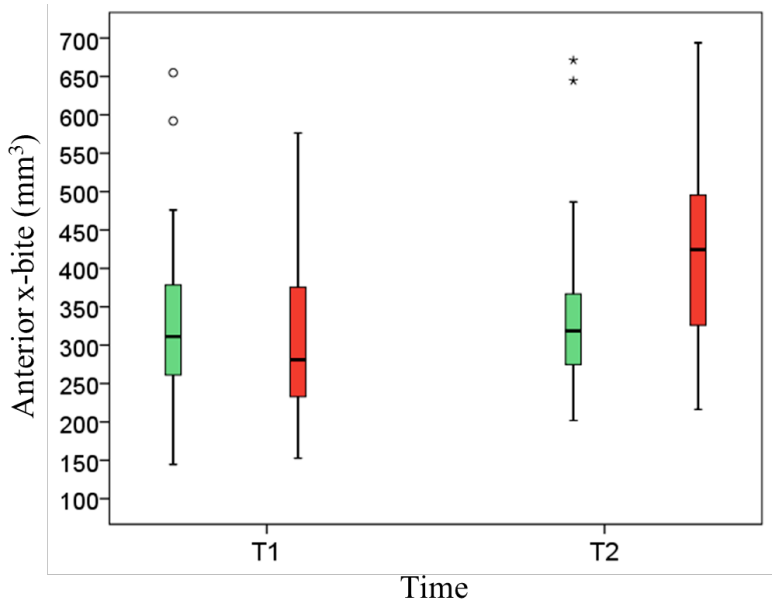

Figure 3.11 Palatal Volume Anterior Halves (X-Bite Side) - Comparison between the study groups at T1 and T2. Treatment Sample (Red) and Control Sample (Green).

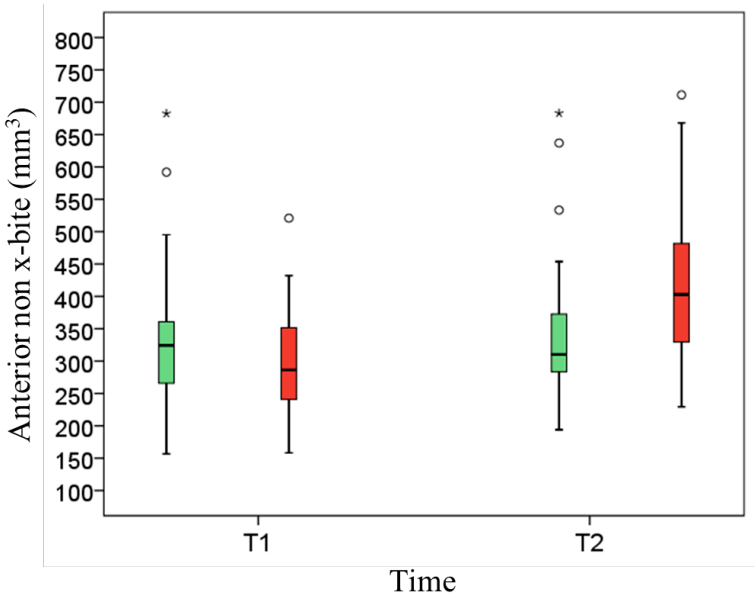

Figure 3.12 Palatal Volume Anterior Halves (Non X-Bite Side) - Comparison between the study groups at T1 and T2. Treatment Sample (Red) and Control Sample (Green).

3.2.3.3 Middle Palatal Halves Treatment Sample vs. Control Sample:

For the middle halves, the mean volumes on the crossbite side and non crossbite side at T1 in the treatment group was less than the corresponding sides in the control group; this showed no significant difference (P= 0.163 and P= 0.053, respectively). After expansion the mean volumes on the crossbite side and non crossbite side in the middle halves in the treatment group were greater than the corresponding sides in the control group which showed no statistically significant difference (P= 0.102 and P= 0.188, respectively). The mean increase from T1-T2 in the treatment group on the crossbite side was 133.41mm^3 (Range= 49.67 - 217.16) and on the non crossbite side it was 125.48mm^3 (Range= 51.83 - 199.14) and both were statistically significant (P= 0.002 and P= 0.001, respectively). The mean increase in the control group on the side corresponding to the crossbite side was 16.16mm^3 (Range= (-

51.54) - 83.85), on the other side it was 9.28mm^3 (Range= (-60.02) - 78.57) and neither were statistically significant (P= 0.634 and P= 0.789, respectively).

Table 3.10 Palatal Volume Middle Halves

Volume Middle halves (mm^3)	**Treatment Sample**		**Control Sample**		**P Value#**
Crossbite side	N	Mean ± SD	N	Mean ± SD	
T1	30	580.82 ± 154.83	30	632.44 ± 126.48	P= 0.163
T2	30	714.23 ± 168.88	27	648.60 ± 128.03	P= 0.102
P Value^		P= 0.002		P= 0.634	
Non-Crossbite side	N	Mean ± SD	N	Mean ± SD	
T1	30	576.36 ± 130.13	30	642.06 ± 127.85	P= 0.053
T2	30	701.84 ± 153.74	27	651.34 ± 132.44	P= 0.188
P Value^		P= 0.001		P= 0.789	

^ Paired sample t test; # Independent t-test

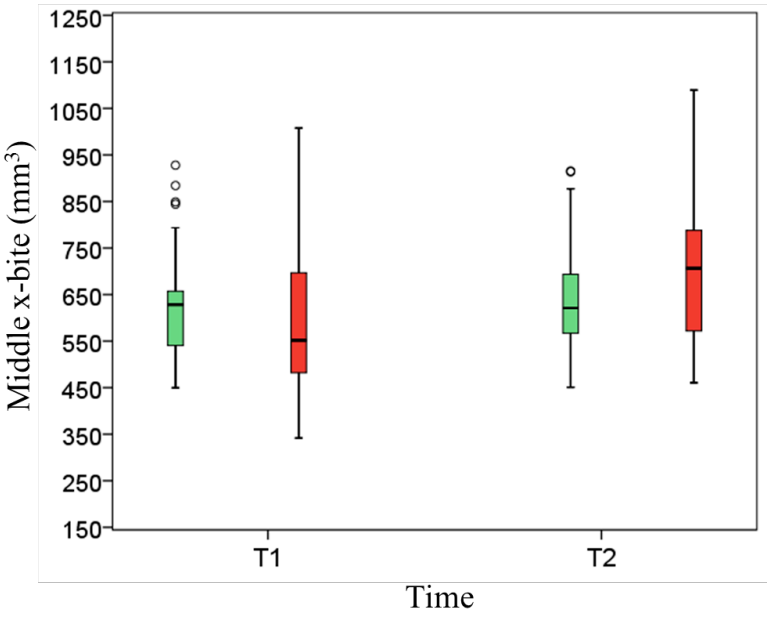

Figure 3.13 Palatal Volume Middle Halves (X-Bite Side) - Comparison between the study groups at T1 and T2. Treatment Sample (Red) and Control Sample (Green).

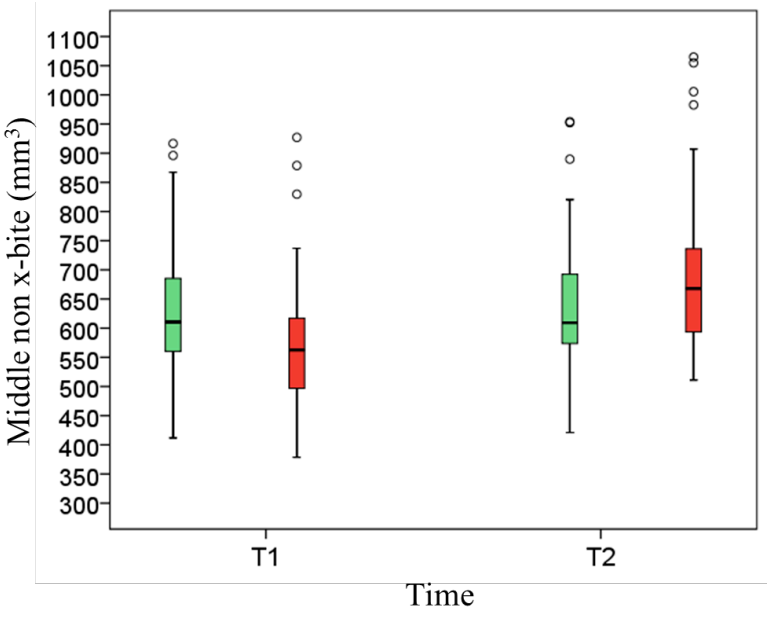

Figure 3.14 Palatal Volume Middle Halves (Non X-Bite Side) - Comparison between the study groups at T1 and T2. Treatment Sample (Red) and Control Sample (Green).

3.2.3.4 Posterior Palatal Halves Treatment Sample vs. Control Sample:

For the posterior halves, the mean volumes on the crossbite side and non crossbite side at T1 in the treatment group was smaller than the corresponding sides in the control group; both showed no significant difference (P= 0.583 and P= 0.165, respectively). After expansion the mean volumes on the crossbite side and non crossbite side in the posterior halves in the treatment group became significantly greater than the corresponding halves in the control group (P= 0.013 and P= 0.044, respectively). The mean increase from T1-T2 in the treatment group on the crossbite side was 233.10mm^3 (Range= 117.01 - 349.19) and on the non crossbite side it was 213.45mm^3 (Range= 118.97 - 307.92) and both were statistically significant (P< 0.001). The mean increase in the control group on the side corresponding to the crossbite side was 63.76mm^3 (Range= (-24.69) - 152.22), on the other side it was 50.11mm^3 (Range= (-41.70) - 141.93) and neither was statistically significant (P= 0.154 and P= 0.278, respectively).

Table 3.11 Palatal Volume Posterior Halves

Volume Posterior halves (mm^3)	Treatment Sample		Control Sample		P Value#
Crossbite side	N	Mean ± SD	N	Mean ± SD	
T1	30	958.18 ± 210.44	30	983.99 ± 145.01	P= 0.583
T2	30	1191.28 ± 237.81	27	1047.76 ± 182.81	P= 0.013
P Value^		P< 0.001		P= 0.154	
Non-Crossbite side	N	Mean ± SD	N	Mean ± SD	
T1	30	940.59 ± 179.54	30	1000.01 ± 146.13	P= 0.165
T2	30	1154.04 ± 185.99	27	1050.12 ± 192.62	P= 0.044
P Value^		P< 0.001		P= 0.278	

^ Paired sample t test; # Independent t-test

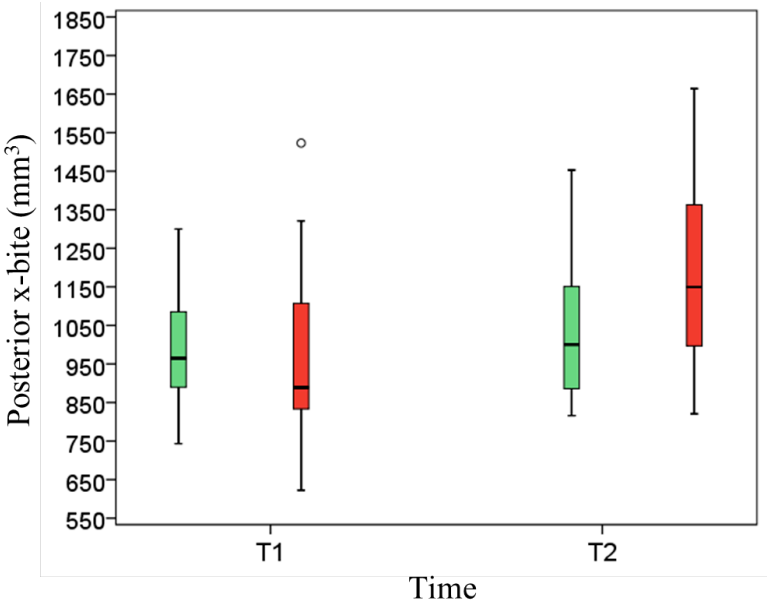

Figure 3.15 Palatal Volume Posterior Halves (X-Bite Side) - Comparison between the study groups at T1 and T2. Treatment Sample (Red) and Control Sample (Green).

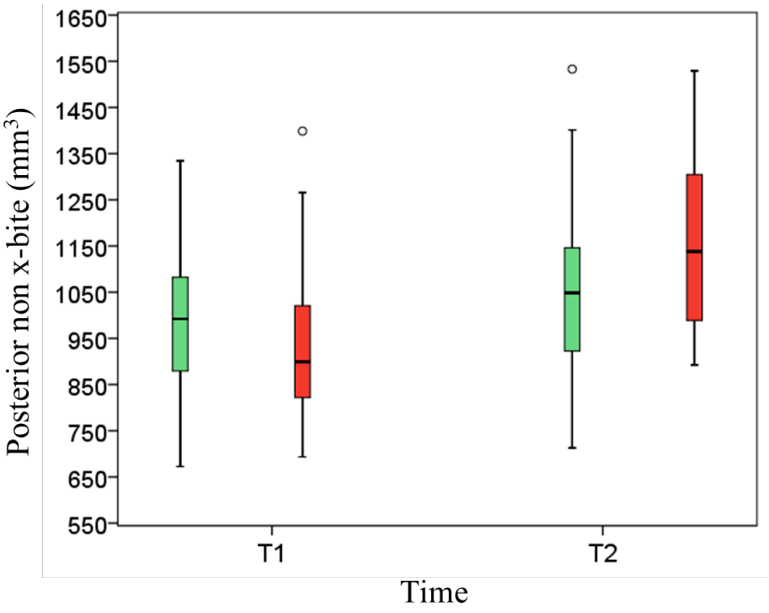

Figure 3.16 Palatal Volume Posterior Halves (Non X-Bite Side) - Comparison between the study groups at T1 and T2. Treatment Sample (Red) and Control Sample (Green).

3.2.3.5 Crossbite vs. Non Crossbite Sides

When comparing the volume between the crossbite side and non crossbite side in the treatment sample and between the corresponding sides in the control sample at both time intervals (T1 & T2) there were no significant differences except for the anterior halves in the control group at T1 (P= 0.026).

Table 3.12 Palatal Volume Symmetry (Treatment Sample)

Volume (mm^3)		Crossbite side	Non-Crossbite side	P Value^
Treatment Sample		Mean ± SD	Mean ± SD	
Anterior	**T1**	299.00 ± 95.00	298.88 ± 85.55	P= 0.984
	T2	414.60 ± 117.63	414.99 ± 124.25	P= 0.968
Middle	**T1**	580.82 ± 154.83	576.36 ± 130.13	P= 0.681
	T2	714.23 ± 168.88	701.84 ± 153.74	P= 0.307
Posterior	**T1**	958.18 ± 210.44	940.59 ± 179.54	P= 0.319
	T2	1191.28 ± 237.81	1154.04 ± 185.99	P= 0.137

^Paired t-test

Table 3.13 Palatal Volume Symmetry (Control Sample)

Volume (mm^3)		Crossbite side	Non-Crossbite side	P Value^
Control Sample		Mean ± SD	Mean ± SD	
Anterior	**T1**	326.50 ± 108.27	337.15 ± 110.41	P= 0.026
	T2	343.44 ± 112.44	344.05 ± 116.11	P= 0.885
Middle	**T1**	632.44 ± 126.48	642.06 ± 127.85	P= 0.316
	T2	648.60 ± 128.03	651.34 ± 132.44	P= 0.790
Posterior	**T1**	983.99 ± 145.01	1000.01 ± 146.13	P= 0.341
	T2	1047.76 ± 182.81	1050.12 ± 192.62	P= 0.917

^Paired t-test

3.2.4 Molar Angulation (°)

One model was excluded in the control sample because the first permanent molars were still erupting at T1.

Molar angulations are shown in Tables 3.15-3.17 and Figures 3.17-3.20.

Figures show range, median and outliers. The treatment group is represented in red and control group is represented in green at both time intervals (T1 & T2).

3.2.4.1 Bucco-Lingual Angle

The mean buccolingual (BL) angle in the treatment group on the crossbite side at T1 was 12.66° ± 5.77 while for the control group it was 12.76° ± 3.77; the difference was not statistically significant (P= 0.942). On the non crossbite in the treatment group the mean BL angle was 11.16° ± 4.13 and for the control group it was 12.93° ± 4.06; the difference was not statistically significant (P= 0.101). After expansion the mean BL angle in the treatment group on the crossbite side was 17.08° ± 6.79 while for the control group it was 12.04° ± 4.13 and this difference was statistically significant (P= 0.001). On the non crossbite in the treatment group the mean BL angle was 14.01° ± 5.72 and for the control group it was 11.76° ± 5.11; this difference was not statistically significant (P= 0.117). The mean increase in the BL angle on the crossbite side from T1-T2 in the treatment group was 4.41° (Range= 1.16 - 7.67) which was statistically significant (P= 0.009). The molar BL angle on the non crossbite side increased 2.86° from T1-T2 (Range= 0.27 - 5.44) which was also significant (P= 0.031). In the control group from T1-T2 there was a mean decrease in the BL angle of -0.71°on the side corresponding to the crossbite side (Range= (-2.76) - 1.33) which was not statistically significant (P=0. 488). On the control non crossbite side it was -1.17° (Range= (-3.6) - 1.26) which was also not significant (P= 0.338). When comparing the BL angle between the crossbite side and non crossbite side in the treatment sample and between the corresponding

sides in the control sample at both time intervals (T1 & T2) there was only a significant difference in the treatment group at T2 (P= 0.016).

Table 3.14 First Permanent Molars Bucco-Lingual Angle

Bucco-Lingual Angle (°)	**Treatment Sample**		**Control Sample**		**P Value#**
Crossbite side	N	Mean ± SD	N	Mean ± SD	
T1	30	12.66 ± 5.77	29	12.76 ± 3.77	P= 0.942
T2	30	17.08 ± 6.79	29	12.04 ± 4.13	P= 0.001
P Value^		P= 0.009		P= 0.488	
Non-Crossbite side	N	Mean ± SD	N	Mean ± SD	
T1	30	11.16 ± 4.13	29	12.93 ± 4.06	P= 0.101
T2	30	14.01 ± 5.72	29	11.76 ± 5.11	P= 0.117
P Value^		P= 0.031		P= 0.338	

^ Paired sample t test; # Independent t-test

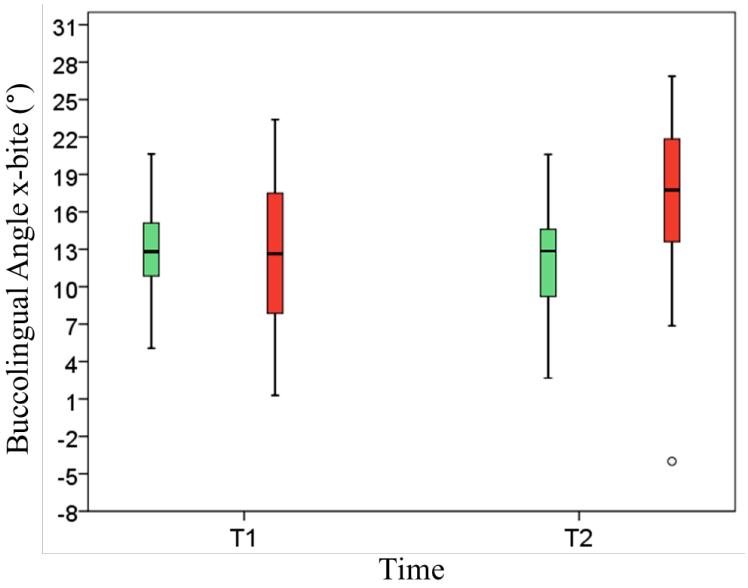

Figure 3.17 First Permanent Molar Bucco-Lingual Angle (X-Bite Side) - Comparison between the study groups at T1 and T2. Treatment Sample (Red) and Control Sample (Green).

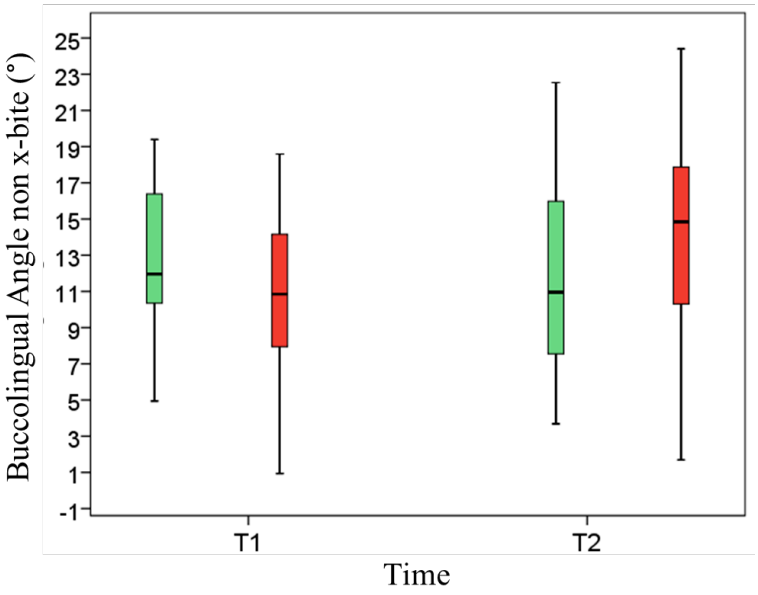

Figure 3.18 First Permanent Molar Bucco-Lingual Angle (Non X-Bite Side) - Comparison between the study groups at T1 and T2. Treatment Sample (Red) and Control Sample (Green).

3.2.4.2 Mesio-Distal Angle

The mean mesiodistal (MD) angle in the treatment group on the crossbite side at T1 was 4.03° ± 4.05 while for the control group it was 4.31° ± 3.31 with no statistically significant difference (P= 0.766). On the non crossbite in the treatment group the mean MD angle was 4.43° ± 4.25 and for the control group it was 4.24° ± 4.06; the difference was not statistically significant (P= 0.85). After expansion the MD mean angle in the treatment group on the crossbite side was 8.40° ± 5.51 while for the control group it was 2.90° ± 3.13; this difference was statistically significant (P< 0.001). On the non crossbite in the treatment group the mean MD angle was 8.93° ± 5.46 and for the control group it was 2.58° ± 3.36, a statistically significant difference (P< 0.001). The mean increase in the MD angle on the crossbite side from T1-T2 in the treatment group was 4.38° (Range= 1.88 - 6.89) which was statistically significant (P= 0.001), while on the non crossbite side it was 4.51° (Range= 1.97 - 7.04) which also was significant (P= 0.001). In the control group there was a mean decrease in the MD angle on the side corresponding to the crossbite side from T1-T2 of -1.41° (Range= (-3.07) - 0.26), while on the non crossbite side it was -1.66° (Range= (-3.43) - 0.12); both were not significant (P= 0.096 and P= 0.066, respectively). When comparing the MD angle between the crossbite side and non crossbite side in the treatment sample and between the corresponding sides in the control sample at both time intervals (T1 & T2) there were no significant differences in in both groups (P > 0.05).

Table 3.15 First Permanent Molars Mesio-Distal Angle

Mesio-Distal Angle (°)	Treatment Sample		Control Sample		P Value#
Crossbite side	N	Mean ± SD	N	Mean ± SD	
T1	30	4.03 ± 4.05	29	4.31 ± 3.31	P= 0.766
T2	30	8.40 ± 5.51	29	2.90 ± 3.13	P< 0.001
P Value^		P= 0.001		P= 0.096	
Non-Crossbite side	N	Mean ± SD	N	Mean ± SD	
T1	30	4.43 ± 4.25	29	4.24 ± 4.06	P= 0.85
T2	30	8.93 ± 5.46	29	2.58 ± 3.36	P< 0.001
P Value^		P= 0.001		P= 0.066	

^ Paired sample t test; # Independent t-test

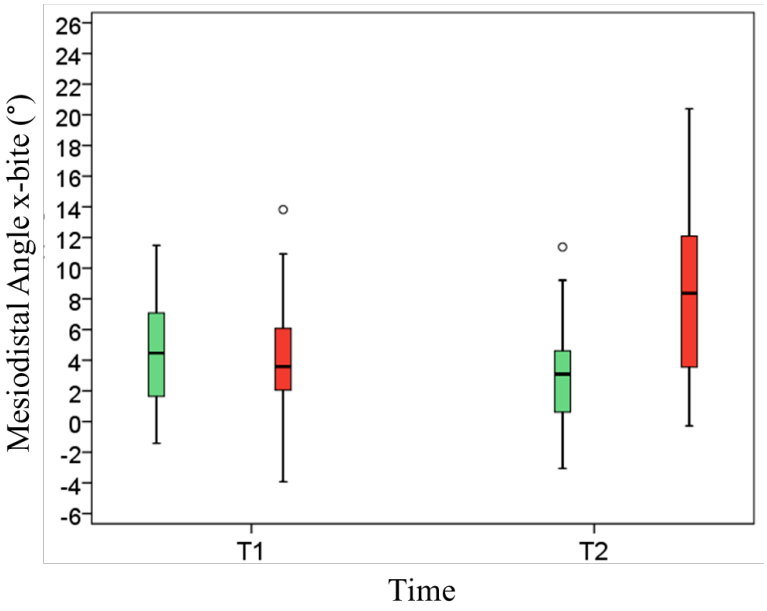

Figure 3.19 First Permanent Molar Mesio-Distal Angle (X-Bite Side) - Comparison between the study groups at T1 and T2. Treatment Sample (Red) and Control Sample (Green).

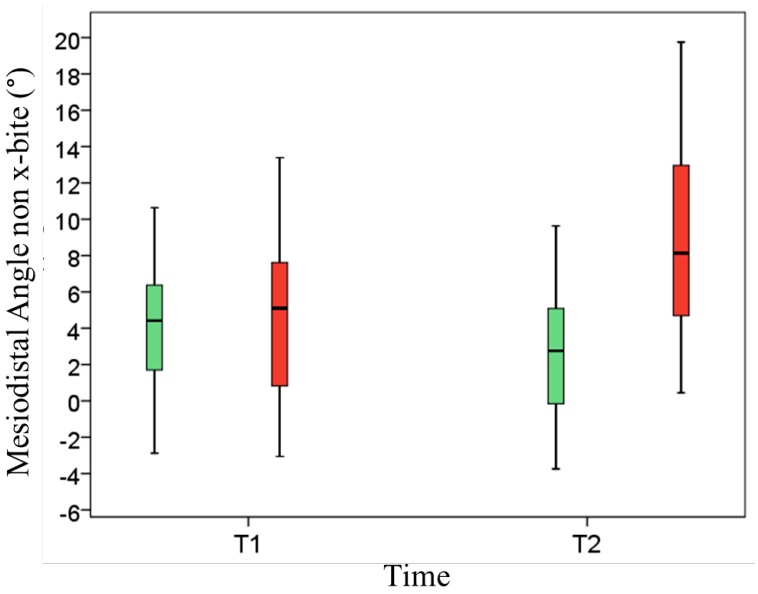

Figure 3.20 First Permanent Molar Mesio-Distal Angle (Non X-Bite Side) - Comparison between the study groups at T1 and T2. Treatment Sample (Red) and Control Sample (Green).

Table 3.16 First Permanent Molars Angle Crossbite vs. Non crossbite

Molar angle (°)	Time	Crossbite side	Non-Crossbite side	P Value^
Treatment Sample		Mean ± SD	Mean ± SD	
Buccolingual angle	**T1**	12.66 ± 5.77	11.16 ± 4.13	P= 0.243
	T2	17.08 ± 6.79	14.01 ± 5.72	P= 0.016
Mesiodistal angle	**T1**	4.03 ± 4.05	4.43 ± 4.25	P= 0.554
	T2	8.40 ± 5.51	8.93 ± 5.46	P= 0.415
Control Sample		Mean ± SD	Mean ± SD	
Buccolingual angle	**T1**	12.76 ± 3.77	12.93 ± 4.06	P= 0.659
	T2	12.04 ± 4.13	11.76 ± 5.11	P= 0.903
Mesiodistal angle	**T1**	4.31 ± 3.31	4.24 ± 4.06	P= 0.628
	T2	2.90 ± 3.13	2.58 ± 3.36	P= 0.414

^Paired t-test

Chapter 4: Discussion

This study was performed to show changes in the palatal dimensions and symmetry following slow maxillary expansion using a Haas-type appliance in the early mixed dentition compared to untreated controls; this has not previously been reported. The variables measured were the palatal width expressed as the intercanine and intermolar widths, the palatal surface area, the palatal volume and the molar angulations. The palate was divided into three parts: anterior, middle and posterior, which were further split into halves using the anatomical mid palatal plane to determine palate symmetry by measuring the surface area and volume of the resultant six parts.

All previous studies on first permanent molar angulation measured on models used either the intermolar angle or the inclination of each molar using two cusp tips. The intermolar angle does not show whether each individual molar angulation is different. Measurement using two cusp tips only gives the buccolingual representation of the molar angle. By contrast, our study measured the angulation of each first permanent molar in 2 planes (bucco-lingually and mesio-distally).

Despite using retrospective data, the sample size was determined by a power calculation. Our study and control groups were matched by gender, dental classification and dental age using the system for dental maturity based on four teeth to increase its validity (Demirjian & Goldstein, 1976).

4.1 Intercanine Width

The pretreatment ICW was significantly less than the control group, similar to Huynh et al. (Huynh, Kennedy, Joondeph, Bollen, 2009). After expansion the ICW increased by 4.65mm and became significantly greater than the control. Moriyasu et al. had slightly less expansion when treating during the late primary or early mixed dentition using a SME removable appliance (Moriyasu, Kuriyama, Kurihara, Fujihashi, Ohno, Asada, 2010). Their control group had similar findings to our study when compared to treatment group (Moriyasu, Kuriyama, Kurihara, Fujihashi, Ohno, Asada, 2010). In our study, the mean ICW increase was greater than mixed dentition unilateral posterior crossbite SME patients treated with either expansion plates or a quadhelix (Bjerklin, 2000). Petrén and Bondemark, using either an expansion plate or quad helix in the treatment of late mixed dentition unilateral posterior crossbite also showed less expansion across the canines than our study (Petrén & Bondemark, 2008). Other studies have used the canine tips to measure the intercanine width. Godoy et al. treated mixed dentition unilateral posterior crossbite with the same appliances as Bjerklin and also showed less canine expansion than our study; this may be attributed due to use of different appliances or an older age than our sample (Godoy, Godoy-Bezerra, Rosenblatt, 2011; Bjerklin, 2000; Petrén & Bondemark, 2008).

Wong et al. compared SME with 3 different appliances (Haas type, Hyrax and quad helix appliances) taken from the same practice as this study; his treatment ICW increase was similar to our study as was his control group mean increase in intercanine width from T1-T2 (Wong, Sinclair, Keim, Kennedy, 2011). Bell and LeCompte used an early mixed dentition quadhelix for functional posterior crossbite treatment with similar findings (Bell & LeCompte, 1981). Vargo et al. compared a quadhelix to bonded expanders SME in mixed

dentition again with similar findings to ours, without specifying the type of posterior crossbite (Vargo, Buschang, Boley, English, Behrents, Owen, 2007). Both studies used the cusp tips for the ICW measurement (Wong, Sinclair, Keim, Kennedy, 2011; Vargo, Buschang, Boley, English, Behrents, Owen, 2007).

Spillane and McNamara used mixed dentition acrylic splint RME with similar results to ours; however, the majority of their patients had no posterior crossbite (Spillane & McNamara, 1995). Our results were slightly greater than the Handelman's mixed dentition RME Haas appliances where the majority had a unilateral posterior crossbite (Handelman, Wang, BeGole, Haas, 2000). Similar methodology to our study was used for the ICW measurement (Spillane & McNamara, 1995; Handelman, Wang, BeGole, Haas, 2000). Using the cusp tips for this measurement, Sandikçioğlu and Hazar compared a quadhelix SME group to a hyrax RME group in the mixed dentition and found ~1.5mm less expansion with RME (Sandikçioğlu & Hazar, 1997). They had both unilateral or bilateral posterior crossbites (Sandikçioğlu & Hazar, 1997). Cozzani et al. had a greater mean ICW increase than our results after treating early mixed dentition unilateral or bilateral posterior crossbites with using a Haas type RME appliance (Cozzani, Guiducci, Mirenghi, Mutinelli, Siciliani, 2007). The addition of bilateral crossbites and different measurement method may explain their increased ICW (Cozzani, Guiducci, Mirenghi, Mutinelli, Siciliani, 2007). Lippold et al. reported reduced ICW increase compared to our findings following late primary or early mixed dentition RME with a bonded Hyrax appliance (Lippold, Stamm, Meyer, Végh, Moiseenko, Danesh, 2013). This could be due to different appliance, activation rate and methodology to measure the distance (cusp tips) (Lippold, Stamm, Meyer, Végh, Moiseenko, Danesh, 2013). Sari et al. reported similar results to ours using acrylic bonded RME

expanders in the mixed dentition; however, no mention was made of crossbite type and they used the cusp tips to measure ICW (Sari, Uysal, Usumez, Basciftci, 2003).

Lamparski et al. had less expansion comparing two types of RME in the mixed and permanent dentitions; they did not mention if the posterior crossbite was unilateral or bilateral (Lamparski, Rinchuse, Close, Sciote, 2003). Our results showed increased ICW compared to Adkins et al. with a Hyrax RME appliance in the late mixed or early permanent dentition (Adkins, Nanda, Currier, 1990). They had both unilateral and bilateral posterior crossbites in the treatment group but mostly unilateral (Adkins, Nanda, Currier, 1990). Both studies used the same method of measurement used in our study (Lamparski, Rinchuse, Close, Sciote, 2003; Adkins, Nanda, Currier, 1990).

Finally, ICW can be measured from CBCT scans. Gohl et al. found the mean ICW increase was less than our study results following RME in mixed or permanent dentitions. This may be due to different age range and RME with a different appliance (Hyrax) (Gohl, Nguyen, Enciso, 2010). Both bilateral and unilateral posterior crossbites were included (Gohl, Nguyen, Enciso, 2010).

In general, ICW with SME matches previous studies with RME. ICW increase appears to be improved with SME in younger ages compared to RME at older ages.

4.2 Intermolar Width

We used the greatest convexity of the lingual gingival margin of the first molar to measure IMW and noted a mean expansion of 4.76mm. The pre-expansion IMW was slightly but not statistically significantly less than the controls; after expansion, the IMW became statistically greater than the controls. Moriyasu et al. had slightly less IMW increase

during late primary or early mixed dentition SME using a removable appliance. (Moriyasu, Kuriyama, Kurihara, Fujihashi, Ohno, Asada, 2010). Petrén and Bondemark showed slightly less mean molar expansion using a quadhelix in the late mixed dentition treatment of unilateral posterior crossbite, and less IMW expansion for the expansion plate group (Petrén & Bondemark, 2008). Bjerklin found a reduced IMW increase compared to our results which may be due to the use of different appliances (Quadhelix and expansion plates) and older age groups (Bjerklin, 2000). The previous studies used the same method for IMW measurement (Moriyasu, Kuriyama, Kurihara, Fujihashi, Ohno, Asada, 2010; Petrén & Bondemark, 2008; Bjerklin, 2000).

Cusp tips were used in the IMW measurements in the Bell and LeCompte study where they treated early mixed dentition functional posterior crossbite with a quadhelix; the findings were similar to this study with a mean expansion amount of 4.9mm (Bell & LeCompte, 1981). Wong et al. used the molar centroid and showed slightly less mean increase in IMW (Wong, Sinclair, Keim, Kennedy, 2011). Huynh et al. found a greater amount of IMW expansion with SME in both Haas-type appliance group and hyrax group than our study; the quadhelix group showed similar expansion to our study (Huynh, Kennedy, Joondeph, Bollen, 2009). This was due to the inclusion of both bilateral and unilateral crossbite patients, and/or to the different method used to measure IMW (molar centroid) (Huynh, Kennedy, Joondeph, Bollen, 2009). Vargo et al. found a greater IMW increase with mixed dentition quadhelix and bonded expanders than our study (Vargo, Buschang, Boley, English, Behrents, Owen, 2007). This can be attributed to the use of the molar centroid to measure the IMW rather the lingual gingival margin (Vargo, Buschang, Boley, English, Behrents, Owen, 2007). The center of the fossa was also used to measure

IMW by Godoy et al. who treated unilateral posterior crossbite patients by quadhelix or expansion plates SME; they showed a greater mean increase in IMW than our study (Godoy, Godoy-Bezerra, Rosenblatt, 2011). This could be due to the different methodology used for the measurement (Godoy, Godoy-Bezerra, Rosenblatt, 2011).

CBCT scans were used by Corbridge et al. who measured IMW at the grooves of the cemento-enamel junction on CBCT with SME mixed dentition cases; they showed greater IMW expansion which could be attributed to the different method used for measuring the intermolar distance (Corbridge, Campbell, Taylor, Ceen, Buschang, 2011). Spillane and McNamara reported a greater IMW increase than our study with RME mixed dentition treatment; most of their sample had no posterior crossbite (Spillane & McNamara, 1995). Muchtisch et al. also found a greater increase in IMW using early mixed dentition acrylic splint RME treatment (Muchitsch, Winsauer, Wendl, Pichelmayer, Kuljuh, Szalay, Muchitsch 2012). Handelman et al. found a greater increase in the IMW with cemented RME than our study (Handelman, Wang, BeGole, Haas, 2000). This was likely due to RME treatment, different age range in the children group and involvement of different patterns of bites (Unilateral, bilateral posterior crossbite, no crossbite $\pm$ constriction) (Handelman, Wang, BeGole, Haas, 2000). Using the central fossa Cozzani et al. had a mean increase in IMW which was slightly less than our results after treating unilateral or bilateral posterior crossbite in similar age group with a RME Haas type (Cozzani, Guiducci, Mirenghi, Mutinelli, Siciliani, 2007). This could be due to including different types of crossbites with different activation rates of the appliance, and might also be because the appliance was not banded on the first permanent molar (Cozzani, Guiducci, Mirenghi, Mutinelli, Siciliani, 2007). Lippold et al. found the IMW increase with Hyrax RME was slightly greater than our

study (Lippold, Stamm, Meyer, Végh, Moiseenko, Danesh, 2013). This also could be due to different appliance, RME treatment and methodology to measure the distance (Lippold, Stamm, Meyer, Végh, Moiseenko, Danesh, 2013). Sandikçioğlu and Hazar used either the central fossa or centroid, and found the IMW expansion increase was greater in the SME group using quadhelix appliance and the RME group with Hyrax than in our study. However, the IMW expansion was less for the removable SME plate than our results (Sandikçioğlu & Hazar, 1997). This can be due to different age range included in each group, the use of different appliances with variable activation rates and different method used to measure the IMW (Sandikçioğlu & Hazar, 1997).

Adkins et al. reported a greater mean increase in IMW with Hyrax RME in late mixed or early permanent dentition than the findings of this study; about 38% had no posterior crossbite, 42 % had unilateral posterior crossbite and the rest had bilateral crossbite, which might have influenced the results (Adkins, Nanda, Currier, 1990). Lamparski et al. reported the mean pretreatment IMW was slightly less than our sample, but post expansion it was similar; this pretreatment difference might be due to the different age group (Range= 6-14 years old) (Lamparski, Rinchuse, Close, Sciote, 2003). Oliveira et al. used mixed and permanent dentitions Haas or Hyrax RME and reported that the greatest mean increase in IMW was with the Haas appliance, while for the Hyrax it was less than our results (Oliveira, Da Silveira, Kusnoto, Viana, 2004). This might be due to different treatment method or different age groups (Oliveira, Da Silveira, Kusnoto, Viana, 2004). Oliveira De Felippe et al. reported IMW increase following RME treatment of posterior crossbite less than our study with the IMW standard deviation at T2 of 12.20; this could be due to the combination of the results of 3 appliances used (Haas, Hyrax and bonded), different age groups or due to the

variation of the rates of activation (half of the sample had 2 turns/day, 42% had 1 turn/day and the rest 1 turn/every other day) (Oliveira De Felippe, Da Silveira, Viana, Kusnoto, Smith, Evans, 2008). Patients treated with acrylic bonded expander RME in the mixed and early permanent dentitions had a greater IMW mean increase measuring across the central fossa (Sari, Uysal, Usumez, Basciftci, 2003). This can be due to different age groups, different appliance used with different activation rates and different methodology of measuring IMW (Sari, Uysal, Usumez, Basciftci, 2003).

Using CBCT Gohl et al. found the mean increase intermolar distance was less than our results (Gohl, Nguyen, Enciso, 2010). This can be due to different age ranges, different RME appliance (Hyrax), the inclusion of both bilateral and unilateral posterior crossbites and different method of measuring IMW using CBCT scans (Gohl, Nguyen, Enciso, 2010). Weissheimer et al. measured the intermolar width on CBCT scans at the occlusal surface following RME treatment in the mixed or permanent dentition using Haas-type and Hyrax-type appliances. Their mean IMW increase was greater than this study, and can be due to the use of different appliances with RME treatment, different age group and the different measurement method (Weissheimer, de Menezes, Mezomo, Dias, de Lima, Rizzatto, 2011).

With permanent dentition RME treatment Kılıç et al. reported an increased amount of expansion from bilateral posterior crossbite using the same measurement method as Oliveira et al. (Kılıç, Kiki, Oktay, 2008)

4.3 Surface Area

When comparing treatment to the controls at T1, we found that there was no statistically significant difference. By T2, the surface area was statistically greater than the

control group. The mean total surface area showed a mean post expansion increase of 127.05mm^2. Our results for the treatment group showed smaller surface areas at both time intervals than Oliveira et al. and Oliveira De Felippe; this is due to difference in the method used to measure the surface area, as well as different appliances used with RME and different age groups (Oliveira, Da Silveira, Kusnoto, Viana, 2004 ;Oliveira De Felippe, Da Silveira, Viana, Kusnoto, Smith, Evans, 2008). Muchitsch et al. measured each cross sectional surface area between the molars, primary molars and canines; as such their results cannot be compared to ours (Muchitsch, Winsauer, Wendl, Pichelmayer, Kuljuh, Szalay, Muchitsch 2012). The surface area found by Primožič et al. was less than our results at both T1 and T2. They also had untreated controls, but at T2 the control group had slightly greater surface area than the treatment group, which is opposite to our study results (Primožič, Baccetti, Franchi, Richmond, Farčnik, Ovsenik, 2011). Their patients were all in the primary dentition at both T1 and T2, T2 records were taken 1 year follow up after T1 which was (6 months post retention); the appliance used and the methodology were different (Primožič, Baccetti, Franchi, Richmond, Farčnik, Ovsenik, 2011). In another study by Primožič et al. the surface area was slightly greater in the treatment group than the control group at T2; this was immediately following treatment and retention at 6months follow up (Primožič, Richmond, Kau, Zhurov, Ovsenik, 2011).

Asymmetry was an important part of this study. There were significant differences only in the middle halves of the treatment group at T2 and the anterior halves of the control group at T1. Although these differences were statistically significant, clinically they might not be significant. Primožič et al. assessed symmetry in patients with unilateral crossbite with functional shift by measuring the percentage of overlapping between the original surface area

and a horizontally flipped surface area (mirror image of the original surface area); their palates had symmetry (Primožič, Baccetti, Franchi, Richmond, Farčnik, Ovsenik, 2011).

4.4 Volume

Palate volume pre expansion was smaller but not statistically significant than the control. Post expansion the volume was statistically significant greater than the control group. The treatment group volumes at both T1 and T2 were smaller than reported by Oliveira De Felippe; this is due to difference in volume measurement which involved all the teeth up to the distal of the second permanent molars, different appliances used with RME and different age groups (Oliveira De Felippe, Da Silveira, Viana, Kusnoto, Smith, Evans, 2008). The volume found by Primožič et al. at both pre and post expansion was less than what we found (Primožič, Baccetti, Franchi, Richmond, Farčnik, Ovsenik, 2011). In their study at T2, the controls had greater palatal volume than the treated patients; which is opposite to our study (Primožič, Baccetti, Franchi, Richmond, Farčnik, Ovsenik, 2011). The cause of the differences between this study and our study were explained before in the surface area section (Primožič, Baccetti, Franchi, Richmond, Farčnik, Ovsenik, 2011). In another study by Primožič et al. the post treatment volume was slightly greater than the control group, which was immediately following treatment and retention at 6months follow up (Primožič, Richmond, Kau, Zhurov, Ovsenik, 2011).

Using CBCT scans Gohl et al. reported palatal volume almost 4 times larger than our study (Gohl, Nguyen, Enciso, 2010). This is because they measured the volume from the CEJ of the central incisor anteriorly to the PNS posteriorly and the age range was from ~9 to 16 (Gohl, Nguyen, Enciso, 2010).

Our study determined asymmetry by measuring the volumes of 6 halves; there were no significant differences in the treatment group, while the control group showed significant differences in the anterior halves at T1 only. Although these differences were statistically significant, clinically they might not be significant. Our study is the first known study to look at the symmetry of the palate by the volume.

4.5 Molar Angulation

Our study is the first study to measure both the individual buccolingual and mesiodistal molar angulations on both the crossbite and non crossbite side. Our methodology allows this evaluation without the use of CBCT radiology. Previous studies measured the buccolingual angulation with different methods.

In our study, the mean buccolingual angle in the crossbite side treatment group at T1 was 12.66° $\pm$ 5.77 while the non crossbite the mean angle was 11.16° $\pm$ 4.13. The post expansion mean angle on the crossbite side was 17.08° $\pm$ 6.79 while the non crossbite side post treatment mean angle was 14.01° $\pm$ 5.72. Therefore, the crossbite side increased its buccal lingual angle more than the non crossbite side. Oliveira et al. measured each molar inclination by using the angle at the intersection of a line through two cusp tips and a horizontal line passing through the right and left lingual cusps (Oliveira, Da Silveira, Kusnoto, Viana, 2004). Their Haas group increased molar inclination from T1-T2 ~7°, which was greater than our results; for their Hyrax group the change was very minimal on the right molar and ~1° on the left molar which was less than our study (Oliveira, Da Silveira, Kusnoto, Viana, 2004). This method was also used by Kılıç et al. only mesial cusps were used to construct the line for each molar and the mesiolingual cusps to make the transverse

line (Kılıç, Kiki, Oktay, 2008). The Hyrax group molar inclination increased ~9°, while for the acrylic bonded group the change was ~7°; both were greater than our results (Kılıç, Kiki, Oktay, 2008). The difference between these and our results is attributed to the different appliance used, the different activation rate and different method of measuring the molar angulation (Kılıç, Kiki, Oktay, 2008). The difference in the results between these two studies could be due to different age groups as well as different methodology (Oliveira, Da Silveira, Kusnoto, Viana, 2004; Kılıç, Kiki, Oktay, 2008).

CBCT scans were used by Weissheimer et al. to measure the molar angle using the intersection of a line passing along the long axis of the palatal root canal and a horizontal line (Weissheimer, de Menezes, Mezomo, Dias, de Lima, Rizzatto, 2011). They found the mean increase on the right permanent molar was ~7° while the left molar was ~6°; both results were greater than our results (Weissheimer, de Menezes, Mezomo, Dias, de Lima, Rizzatto, 2011). This could be due to different appliances used with RME treatment, different methodology and the combination of the Haas and Hyrax results for the molar angle (Weissheimer, de Menezes, Mezomo, Dias, de Lima, Rizzatto, 2011). They reported that the Hyrax group had more orthopedic effect than the Haas group, results that were opposite to Oliveira et al (Weissheimer, de Menezes, Mezomo, Dias, de Lima, Rizzatto, 2011; Oliveira, Da Silveira, Kusnoto, Viana, 2004).

Sari et al. used PA cephalometric x-rays and connected two lines passing through the long axis of the molars which pass between the buccal and lingual cusps to measure the intermolar angle (Sari, Uysal, Usumez, Basciftci, 2003). The mixed dentition increase in the intermolar angle was ~11°; for an average molar increase of 5.5°, which is greater than this study (Sari, Uysal, Usumez, Basciftci, 2003). For the permanent dentition group it was 5°

(2.5° for each molar) which is almost similar to our finding on the non-crossbite side, but less than what we found on the crossbite side (Sari, Uysal, Usumez, Basciftci, 2003). Handelman et al. also measured the angle formed by the intersection of two lines passing through buccal and lingual cusps of the first molars; he showed a decrease in IMA (intermolar angle) in adults of -6.2° while children showed negligible change (Handelman, Wang, BeGole, Haas, 2000). For the children group, the increase was smaller than our results possibly due to the older age group, different activation rates and different method of measurement (Handelman, Wang, BeGole, Haas, 2000). Wong et al. also used the same method in early mixed dentition unilateral crossbite cases. They found a mean increase in the buccal angle of each molar about 2° which was less than our study and might be due to the combination of the results of all the appliances used (Wong, Sinclair, Keim, Kennedy, 2011). For our controls there was a decrease in the buccal angulation from T1-T2, similar to Marshall et al. report on normal maxillary molars uprighting with aging (Marshall, Dawson, Southard, Lee, Casko, Southard, 2003).

The other angle measured was the mesiodistal angle, but there are no reports available in the literature for comparison. The mean mesiolingual angle in the treatment group on both the crossbite side and non crossbite sides were similar at T1. Likewise the treatment and control groups were similar at T1. The molar angles significantly increased on both the crossbite and non crossbite side post expansion. The amount of change in the treatment group was similar for crossbite and non crossbite sides. On the other hand, there was a decrease on both sides in the control group which was not statistically significant.

Finally, based on our results the study hypotheses are accepted except the second hypothesis which states that the palatal halves in unilateral posterior crossbite are asymmetric pretreatment.

Chapter 5: Conclusions

- Evaluation of early mixed dentition slow maxillary expansion with a Haas type appliance compared to matched untreated controls resulted in:

1. ~1:1 ratio of expansion across the canines and molars.
2. Pretreatment surface areas and volumes were smaller than the untreated controls.
3. Larger post expansion palatal surface areas and volumes than untreated controls.
4. The surface areas in the palatal halves were similar except for the middle halves post expansion.
5. The treated first permanent molar increased its buccal and distal inclination after expansion, while the controls showed a decrease in both inclinations.
6. The mean increase in the buccal inclination of the treated first permanent molar was significantly greater on the crossbite side, compared to the non crossbite side.

Chapter 6: Future Directions

Suggestions for further research include the following:

- Prospective randomized clinical trials
- Long term follow up following SME without additional orthodontic treatment
- The effects of different appliances with SME on palatal dimensions
- Comparing the dimensions and molar angulations in successful and relapse cases
- The impact of banding primary molars vs. permanent molars
- A growth study like Marshall et al. measuring the mesiodistal angulation of the permanent molars.
- Corresponding changes in lower molar angulation

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