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THE EFFECTS OF RAPID MAXILLARY EXPANSION ON ARCH ASYMMETRY IN CHILDREN

by

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Submitted for partial fulfilment of the requirements for the degree of Master of Science in Dentistry

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Abstract

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The objectives of the present study were to evaluate the effects of rapid maxillary expansion (RME) in children with unilateral posterior crossbite (UPXB) on molar subdivision malocclusions. The extent and location of the asymmetry in the maxilla and in the mandible were measured. Secondly, the nature of the molar subdivision malocclusions was assessed. In particular, the specific variables contributing to molar subdivision malocclusions were identified.

The sample consisted of thirty-six consecutively treated children with UPXB and molar subdivision. The treatment modality was rapid maxillary expansion only. Dental casts were taken before treatment (T1) and post retention (T2). The mean age was 9.25 yrs (S.D. = 1 yr.) at T1 and 12.7 yrs (S.D. = 1 yr.) at T2. The dental casts were digitized by using the digital imaging system

were digitized by using the digital imaging system (Bioscan OPTIMAS® Imaging System, Seattle, Washington). The arch asymmetries were measured in both anteroposterior (AP) and transverse planes of space. Unilateral posterior crossbite were corrected in 83% of the cases. Fifty percent (3/6) of the unsuccessful cases exhibited one or two teeth in edge-to-edge occlusion. The greatest amount (3.7 mm) of arch asymmetry was found in the mandibular transverse dimension, which was significantly reduced at T2 by 1.7 mm. It is believed that this reduction in mandibular transverse asymmetry was due to the maxillary expansion. This resulted in a reduction of mandibular transverse displacement.

Only 27% (10/36) of molar subdivision malocclusions were completely corrected (0 mm difference between right and left sides). The mandibular asymmetry was found to be the primary contributor to molar subdivision. The combined effects of the mandibular shift, and skeletal and dentoalveolar asymmetries, produced the mandibular asymmetry. Once the maxilla was adequately expanded and the mandibular shift was reduced, the mandibular AP asymmetry became the predominant factor in the remaining

subdivision malocclusions. In addition, the remaining mandibular transverse asymmetry contributed to the molar subdivision malocclusions at T2. Adjunctive asymmetric orthopedic therapy, in addition to maxillary expansion, may be necessary for children with UPXB and molar subdivision malocclusions, when no dental asymmetries are present.

Dedicated to

My parents Teng-Huei Wu and Jing-Lang Roan
for their endless love and support.

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INTRODUCTION

Unilateral posterior crossbite (UPXB) is believed to be caused by a bilaterally constricted maxilla and to result in a mandibular shift in order to facilitate a better occlusion (Moyers 1966, Schroder and Schroder 1984, Kutin and Hawes 1969, Zhu 1996). It has been observed that the mandible could shift laterally and/or anteroposteriorly (Bishara 1994, Lewis 1976, O'Byrn et al. 1995). As a result, two types of asymmetric malocclusions, a lower dental midline deviation and a molar subdivision relationship commonly accompany UPXB (Ben-Bassat 1993, O'Byrn et al. 1995, Zhu et al. 1996, Brin et al. 1996).

The prevalence of UPXB was 8-23% in the primary and mixed dentitions (Kutin and Hawes 1969, Kisling 1981, Thilander et al. 1984, Kurol and Berglund 1992). Only 8% of reported UPXB cases self-corrected (Kutin and Hawes 1969, Kurol and Berglund 1992). Consequently, early treatment of unilateral posterior crossbite is strongly recommended in order to establish a stable occlusion and avoid possible deleterious effects on the developing

temporomandibular joint (Myers et al. 1980, Vadiakas 1991, Schroder and Schroder 1984, Pirttiniemi et al. 1990, Zhu et al. 1996, Hesse et al 1997). Maxillary expansion is the most recommend treatment modality. Numerous studies have examined the effects and stability of maxillary expansion with a variety of appliances. However, so far there is no any study available regarding the extent and location of arch asymmetry in UPXB and the effects of maxillary expansion on arch asymmetry in UPXB.

The high prevalence of molar subdivision was reported in both mixed and permanent dentition (US 1974, Kelly and Harvey 1977). In a large scale of oral health survey conducted by US government, class II or class III molar subdivision were present in 20.6% of 23,492 children and in 25.7% of 22,284 adolescent dentition (US 1974, Kelly and Harvey 1977). Molar subdivision malocclusions and midline deviation present difficulties in orthodontic treatment because of the asymmetric occlusal relationship and the obscurity of the underlying factors responsible for this asymmetric malocclusions. In spite of high incidence of molar subdivision in children and adolescent, Only four studies have evaluated the arch asymmetry in adults with untreated molar subdivision malocclusions (Alavi et al. 1988, Rose et al.

1994, de Araujo et al. 1994, O'Byrn et al. 1995). They found that the mandibular asymmetry is the primary contributor to molar subdivision. Their results shaded some light in this area. However, these cross-sectional studies only recruited non-growing adults. In treating children with UPXB and molar subdivision malocclusions, the following questions arise: From where are these asymmetric malocclusions originating? Is there any change of arch asymmetry in response to maxillary expansion? What is the possibility of correcting subdivision by maxillary expansion alone, given the facts that the maxillary expansion can eliminate the mandibular shift and possibly the dentoalveolar compensatory asymmetry in growing children. In the present study, children with combined UPXB and molar subdivision were recruited. Arch asymmetries measured before maxillary expansion (T1) and after retention (T2) were compared. The average duration between T1 and T2 was forty-one months. The relationships between molar subdivision and arch asymmetries before and after maxillary expansion were assessed as well in order to investigate the questions mentioned above.

LITERATURE REVIEW

Craniofacial and Dentoalveolar Asymmetry

Asymmetry of the craniofacial structures can have secondary effects on the dentition and the occlusion. Extreme examples of facial asymmetry may present major clinical orthodontic problems. The most common condition of this type is cleft of the lip and palate but there are many other developmental, pathological, traumatic and iatrogenic causes of asymmetry of the head and face with dental consequences. At the other end of the scale, mild asymmetry can occur in an otherwise normal face. One of the most comprehensive surveys was carried out by Woo (1931) who made a total of 50,400 direct measurements on eight hundred Egyptian skulls. He showed that the right side of the cranium, including the frontal, parietal, temporal, and in some respects the sphenoid bones, is markedly larger while the left side of the facial complex, including the malar and maxillary bones, is larger. He concluded that human skulls are definitely and markedly asymmetrical and this asymmetry is not due to bones of the individual differing from a symmetrical

type but rather from asymmetry within the cranium itself. Later, it was shown that the degree of facial asymmetry was less than the degree of cranial asymmetry (Pearson and Woo 1935). Woo hypothesized that the mandibular asymmetry may be correlated to that of the maxilla.

In the study of craniofacial asymmetry, Cook (1980) described three categories:

1. Minor, subclinical asymmetry. This exists in most, if not all, otherwise normal individuals and has no effect on normal occlusion.

2. Gross asymmetry associated with trauma, pathology, or genetic anomaly. Compensatory dentoalveolar asymmetry may or may not reduce the effect on the occlusion.

3. Clinically apparent craniofacial asymmetry with dental malocclusions, but not related to category 2 above.

Cook studied this third category using lateral, posteroanterior (PA) and submentovertex cephalometric radiography. He found that it is impossible to identify all the important landmarks on PA cephalometric radiography due to multiple superimposition. In addition, he found that even a 5° horizontal rotation of the headholder could reverse the apparent dominant side of asymmetry as determined by PA cephalometric radiography. Similar investigation of vertical rotation in submentovertex cephalometric radiography showed no

change in asymmetrical dominance but a relative shift of points. A 5° rotation is equivalent to less than 5 mm of movement of the earposts, well within the range of patient-positioning error and human anatomical variability.

White (1982) has questioned the value of using porion as the true transverse axis for the skull. He measured seventy-three human crania and found that the porion showed a sagittal variability in relation to the subspinale-basion midline. Nahoun et al. (1964) had similar finding while used oriented lateral, posteroanterior, and verticosubmental cephalometric radiographs to analyze skull in three dimensions.

Mulick (1965) studied craniofacial asymmetry with the "serial twin-study method". The sample consisted of eighteen sets of twins: six sets of identical twins and twelve sets of fraternal twins. Measurements were obtained from lateral and PA cephaloradiographs. He concluded that if there is any effect of heredity in producing symmetry, it could not be measured in his sample. Hence, environment plays a more dominant role in producing symmetry.

From the standpoint of orthodontics, Lundstrom (1961) classified asymmetry into two categories: 1)

qualitative asymmetry, which results from missing teeth or cleft palate; and 2) quantitative asymmetry, which was related to size of teeth, location of the teeth in the dental arches and location of the arches in the head. Lunstrom also included a number of factors that might be responsible for arch asymmetry. These were:

1. finger-sucking and similar habits
2. asymmetric chewing habits
3. loss of contact points through dental caries
4. extraction of deciduous teeth
5. extraction of permanent teeth
6. trauma

The raphe-median line has long been used in the orthodontic practice. It had been questioned whether the center of the palate, represented by the raphe-median line, is a reliable axis of symmetry. Lundstrom evaluated a sample of twenty-eight subjects with nearly ideal or anatomically correct occlusion. He found that differences between the palatine raphe and the center of the dental arch were small. However, in a larger study of 139 boys of the same age (including the above 28), there was a tendency for the first molar on the left side to be farther from the raphe and more anterior than its antimere (Lundstrom, 1961). The average difference was

0.45 ± 0.14 mm. Lundstrom concluded that recorded asymmetries in the AP or lateral direction must be assumed to be due, in part, to asymmetry of the raphe line. The limited value of the raphe line as an axis of symmetry is particularly obvious when it is not straight but S-shaped.

By merging dental cast and PA cephalometric data of the same individuals with a symmetroscope, Truque (1956) investigated the relationship of the maxillary dental arch to the palatine raphe and to the X-plane, which was a midsagittal plane of the upper facial skeleton. The result showed that the palatine raphe tended to deviate to the right of X-plane anteriorly after the two planes crossed in the first molar area.

The Optocom was used in the study of Mayers (1977). This system was designed to transform landmarks from dental casts into an X, Y, Z co-ordinate system. He used a sample of 208 non-orthodontically treated subjects to investigate the asymmetry of the maxillary and mandibular dentition with respect to the palatine raphe. The palatine raphe was defined by two points on the palatine raphe: One point was the most posterior point on the palatine raphe, and the other point was the point located between the most medial extension of the most anterior

pair of rugae. He found that the maxillary molars tended to deviate to the left with respect to the palatine raphe. He also found that the maxillary incisors tended to deviate to the right with respect to the palatine raphe.

Posterior Crossbite

Definition and Prevalence

Crossbite, according to Wood (1962), is an abnormal buccal, labial, or lingual relationship of a tooth or teeth of the maxilla, the mandible, or both when the teeth of the two arches are in occlusion. It may include one or more teeth and it may be bilateral or unilateral. Posterior crossbite is a common malocclusions of the primary and the mixed dentition in children. The reported prevalence of this malocclusions ranged from 7.1% to 23.3% (Kisling 1981, Thilander et al. et al. 1984, Heikinheimo et al. 1987, Kerosuo 1990, Kurol and Berglund 1992). Kutin and Hawes (1969) examined 515 children and found that 7.7% of the children had posterior crossbite. In Scandinavian populations, the prevalence of posterior crossbite was reportedly higher (9.5%-23.3%).

Classification and Etiology

Moyers (1966) classified crossbite as dental, muscular or osseous. According to his definitions, dental crossbite involve only tipping of teeth. The condition is localized in the alveolar process and does not affect the size or shape of basal bone. Tipping can result from many factors, including traumatic injuries, supernumerary teeth, eruption aberrations and inadequate arch lengths. In this dental crossbite group, the upper and lower midlines will coincide when the jaws are apart and diverge as teeth come into occlusion. On the other hand, the muscular type of crossbite involves muscular adjustment to tooth interference. Moyers (1966) included the cross disharmonies of the craniofacial skeleton in the osseous category. There may be asymmetrical growth of the maxilla or mandible or a lack of agreement of their widths. Moyers (1966) concluded that lack of harmony between maxillary and mandibular widths usually is due to a bilaterally constricted maxilla, in which case the muscles shift the mandible to one side to acquire sufficient occlusal contact for mastication and comfort. This theory has been supported by the studies of Kutin and Hawes (1969) and Schroder and Schroder

(1984) who observed that bilateral crossbite presenting as unilateral crossbite due to a functional shift of the mandible account for 85-97.5 % of the crossbites cases.

Other evidence suggests that the etiology is typically multifactorial and can relate to genetic, congenital, environmental, functional or habitual origin.

Linder-Aronson (1970) showed that children with nasopharyngeal obstruction showed a lower tongue position measured at rest and a short hypotonic upper lip. Linder-Aronson (1970) concluded that an altering of the skeletal and muscular environment was responsible for the increased incidence of buccal posterior crossbite, open-bite. More recently, a study of 120 children who displayed hypertrophied adenoids showed that 47% of the children examined had developed a posterior crossbite (Oulis et al. 1994).

An increased prevalence of posterior crossbite in children who use pacifiers has been reported by Øgaard et al. (1994), Linder-Aronson (1970) and Bresolin et al. (1983) as well. In Øgaard's et al. (1994) study of 445 children, it was showed that posterior crossbite could be predicted with upper intercanine arch width alone. The longer the child had the dummy-sucking habit, the

stronger the effect on the intercanine arch width. Their findings support a hypothesis by Larsson (1986):

> "The high prevalence of posterior crossbite in young dummy-suckers probably is due to an increased activity of the cheeks combined with a reduced lingual support for the deciduous upper molars and canines as the tongue is forced backward and downward. Perhaps also the low position of the tongue can widen the lower arch, and in this way contribute to a posterior crossbite in the deciduous dentition."

Molar Subdivision Malocclusions

Unilateral posterior crossbite is believed to be caused by a bilaterally constricted maxilla and to result in a mandibular shift in order to facilitate a better occlusion (Moyers 1966, Schroder and Schroder 1984, Kutın and Hawes 1969, Zhu et al. 1996). It has been observed that the mandible could shift laterally and/or anteroposteriorly (Bishara et al. 1994, Lewis 1976, O'Byrn et al. 1995). As a result, two types of asymmetrıc malocclusions, a lower dental midline deviation and a molar subdivision relationship commonly accompany UPXB (Ben-Bassat et al. 1993, O'Byrn et al. 1995, Zhu et al. 1996, Brin et al. 1996).

Angle (1889) defined molar subdivision as the asymmetric molar relationship on bilateral sides. For example, one side presents class I and the other side presents class II or class III molar relationship.

The high prevalence of molar subdivision was reported in both mixed and permanent dentition (U.S. 1974, Kelly and Harvey 1977). In a large scale of oral health survey conducted by U.S. government, class II or class III molar subdivision were present in 20.6% of 23,492 children and in 25.7% of 22,284 adolescent (Kelly and Harvey 1977).

Molar subdivision malocclusions present difficulties in orthodontic treatment because of the asymmetric occlusal relationship. Attention has mainly focused on various asymmetric mechanics. Wertz (1975) and Cheney (1952, 1961) described asymmetric extraction strategies affecting the mandibular arch in the treatment of Class II subdivision malocclusions. Haack, Weinstein (1958) and Garland et al. (1981) have advocated the use of asymmetric headgear (unilateral face bow). Tweed (1966) used Sliding jigs associated with unilateral class II elastic to correct dental asymmetries. Unilateral class II elastic wear with a coil spring to tip a maxillary molar distally was used by many clinicians. Unilateral tip-back moment with segmental arch wire mechanics was proposed by Shroff et al. (1997). Most of the mechanics concentrate at maxillary molars, which was not proved to be the cause of molar subdivision by any studies.

In spite of high incidence of molar subdivision, Only four studies have evaluated the arch asymmetry in adults with untreated molar subdivision malocclusions (Alavi et al. 1988, Rose et al. 1994, de Araujo et al. 1994, O'Byrn et al. 1995).

There is evidence that the mandible may account for some of the asymmetry in subdivision malocclusions. In a study relating mandibular asymmetry to pain dysfunction, Williamson and Simmons (1979) measured submentovertex and posteroanterior cephalometric radiographs to assess mandibular asymmetry. They defined horizontal asymmetry as a difference in length between right and left halves of the mandible measured from the midpoint of the posterior of the condyle head to the most anterior part on the mandible. For the fifty-three orthodontic patients in the study, a mean horizontal asymmetry of 2.67 mm was calculated. One-third showed 3 mm or more horizontal asymmetry. Williamson and Simmons also noted that not all patients with significant (3 mm) asymmetry showed occlusal asymmetry. Therefore they hypothesized that dental or skeletal (glenoid fossa) compensation may count for the camouflage of these asymmetries.

Alavi et al. (1988) compared a group of twenty-eight subjects having normal occlusion with a same-size

group having molar subdivision malocclusions. Mean ages were 17.1 years for both groups. Additional criteria for the malocclusions group included (1) the absence of any severely maligned or blocked out teeth, and (2) no apparent functional mandibular shifts as reported in the clinical history. The sources of measurements included PA and lateral cephalometric radiographs as well as dental models. The median palatal raphe was used as maxillary dental midline. Buccal cusp tips of the posterior teeth and the midincisal edges of the anterior teeth on the casts were the landmarks. Dental asymmetries were calculated by determining the absolute difference between homologous measurements as related to both reference lines. For the lateral cephalometric radiographs, right and left side landmarks were distinguished with the aid of the Broadbent-Golden orientator. Linear measurements were made in a horizontal direction from all the landmarks. A combination of variables with regard to dental arch and facial asymmetries was found to best discriminate between the two groups. Among these variables, the two variables describing the AP asymmetry of the mandibular and maxillary first molar appeared to be the primary contributors to the differences obtained. However, due

to the limitation of assessment of PA and lateral cephalometric radiography, whether the asymmetric mandibular molar position was due to a skeletal asymmetry or a dentoalveolar asymmetry could not be determined precisely. Regarding the maxillary asymmetry, the variables describing AP maxillary dentoalveolar asymmetry and maxillary asymmetry were not among the discriminating list except the AP maxillary molar asymmetry, assessed by lateral cephalometric radiography.

Rose et al. (1994) used submentovertex radiographs to compare mandibular symmetry between a group of twenty-eight adult subjects exhibiting class II subdivision and thirty adult subjects with class I malocclusions as the control group. All of the subjects had full permanent dentition in both maxillary and mandibular arches through the first permanent molars, none of them had any previous orthodontic treatment, nor any lateral functional mandibular shifts on closure. Mandibular asymmetry was assessed in both transverse and AP dimensions at three co-ordinate systems, which represented the cranial floor, the mandible, and the mandibular dentition (Figure 1, 2). The cranial floor co-ordinate system consisted of the line connecting the foramina spinosum landmarks and its perpendicular bisector (Figure 2). The line connecting

right and left condylar midpoints and its perpendicular bisector generated the mandibular skeletal coordinate system (Figure 1). The dental coordinate system derived from the line connecting right and left distal molar points (DMP) and its perpendicular bisector (Figure 1). Rose et al. (1994) found that whether the position of the mandibular molars was measured relative to the cranial floor or within the mandible itself, the mandibular first molar was located more posteriorly, by a mean of 1.79 mm and 1.37 mm, respectively, in the study group, on the class II side of the subdivision malocclusions. In addition, he found that the mandible that exhibited no other significant asymmetry. With three different co-ordinate systems, dentoalveolar and skeletal asymmetry, which contributing mandibular asymmetry can be differentiated. Because of the difficulties of locating maxillary submentovertex landmarks, this study was limited to evaluate the mandible only. Therefore, he suggested that any difference found in mandibular asymmetry certainly would not exclude the possibility that maxillary dentoalveolar or skeletal asymmetry may also contribute to molar subdivision malocclusions.

In the following year, O'Byrn et al. (1995), using the same methodology as Rose et al. (Figure 1, 2),

studied mandibular asymmetry in thirty adults with an untreated unilateral posterior crossbite which concurred with either class II subdivision malocclusions on crossbite side or with class III subdivision malocclusions on non-crossbite side. In addition to submentovertex (SMV) radiographs, horizontally corrected tomograms were taken to analyze the condyle position within the glenoid fossa. The results showed that relative to a mandibular coordinate system, the mandibular first molar on the crossbite side was found to be relatively more lateral and relatively distal in comparison to the contralateral side. Skeletally, the mandible showed no significant asymmetry. With respect to the cranial floor, the mandible was "rotated" so that the condyle on the crossbite side was positioned relatively posteriorly in comparison to the contralateral side. A relative posterior positioning of the glenoid fossa was inferred in his study, since there was no demonstrable mandibular skeletal asymmetry or condyle displacement within the fossa as shown on corrected tomograms in the crossbite group as compared with the class 1 group. His findings support the hypothesis of TMJ remodeling as a consequence of unilateral posterior crossbite.

Department of Orthodontics

Brin et al. (1996) studied the skeletal and functional effects of treatment for unilateral posterior crossbite in children. The experimental group consisted of twenty-four children in the mixed dentition stage with UPXB who were treated with removable expansion plates and a control group of ten age-matched children with normal occlusion. The pre-treatment records included PA cephalograms, dental models and the electrognathography, used for "jaw tracking". Only one set of records was taken for the control group. The mean duration for active treatment was ten months. The six months post-active treatment dental models were taken for twenty-two patients. Electrognathographs were taken for eighteen patients and PA cephalograms were taken for only seven patients. They found that a stable dental maxillary arch expansion of at least 1.5 mm but a complete elimination of UPXB in only 50% of the cases. The pattern of the mandibular movement during chewing revealed by the electrognathograph showed a high prevalence of reverse sequencing (RS) in UPXB children. A reduction in RS pattern was noted after treatment, but this reduction was not statistically significant. The frequent persistence of a class II subdivision relationship and lower midline deviation after maxillary expansion were found. In his

samples, fifteen out of twenty-four children presented class II subdivision, and only 40% (6/15) of the subdivision malocclusions were corrected after maxillary expansion. In the attempt to evaluate the arch asymmetry, the distances from the mesiopalatal cusps of maxillary permanent first molars and mesiobuccal cusps of mandibular permanent first molars to the midlines were measured with dividers and a ruler. When unequal distances (more than 1 mm of right-left difference) were measured, the arch was considered transversely asymmetric. For the maxillary midline definition, they extend the median raphe anteriorly to the point which posterior to the incisive papilla. The lower midline was defined as continuity in relation to the palatal raphe. They reported that in about 50% of the cases, the maxillary arch was symmetric before and after treatment. In the control group, the maxillary arch symmetry was demonstrated in 57% of the cases. In the mandibular arch, they found 50% of the cases (11/22) had a "spontaneous" arch width increase, 27% (6/22) had no change, and 23% (5/22) had a decrease in arch width.

Early Treatment for Unilateral Posterior Crossbite Malocclusions

Eight percent of the cases diagnosed as posterior crossbite ın primary dentition self-corrected during the mixed dentition (Kutin and Hawes 1969, Heikinheimo et al. 1987, Kurol and Berglund 1992). However, early treatment of posterior crossbite has been recommended due to the facts that crossbite in the primary dentition is transferred to the permanent dentition in a majority of the cases, and due to the risk of posterior crossbite leading to progressive mandibular dysfunction and may result ın skeletal asymmetry (Thilander et al. 1984, Pirttiniemi et al. 1990).

It has also been suggested that the later we treat crossbite malocclusions, the greater is the risk of damaging the temporomandibular joint (Pirttınıemı et al. 1990, 1991, Hesse et al. 1997). Pirttiniemi et al. (1991) found the condyles were more asymmetrically positioned in relation to posterior walls of glenoid fossa in subjects with crossbite than ın the subjects with normal occlusion. The correlation between functional posterior crossbite correction and condylar position changes was confirmed by Hesse et al. (1997). By using the corrected tomographs, they found that

compared to crossbite side, the condyle was positioned more anteriorly on the non-crossbite side than the crossbite side. After maxillary expansion, the condyle moved posteriorly and superiorly on the non-crossbite side.

Another reason for early treatment of crossbite is that in the transverse dimension, growth slows down earlier than in the sagittal or vertical dimension (Zhu et al. 1996).

In unilateral crossbite malocclusions, chewing may be affected, as often most of the chewing is believed to take place on the crossbite side. Since no difference was found in the EMG amplitude of the activity of the masseter muscles, it has been questioned whether unilateral chewing is present (Ingerall and Thilander, 1975). However, since the temporalis muscles showed an asymmetric EMG activity both during chewing and in the postural position, it was believed that a negative influence on the growth pattern may still exist as the muscles adapt to avoid cuspal interference.

Treatment of Posterior Crossbite in Children

The treatment modalities of Posterior Crossbite in Children have been suggested as followed :

1) Equilibration to eliminate mandibular shift

2) Bonding composite to the crown of a deciduous tooth

3) Active expansion of a constricted maxillary arch

In the present study, for all the subjects, active expansion of a constricted maxillary arch is the goal of treatment, therefore the following discussion only concentrates on maxillary expansion.

Appliance Design

Varieties of appliances have been used for the expansion of the maxillary arch. Four of the more frequently used appliances are the Haas, Hyrax®, bonded acrylic splint, and the quad-helix. The first two appliances are banded expanders. The Hyrax® expander consists of bands placed on the maxillary first permanent molars and first premolars (Figure 3). If first premolars are not available during early mixed dentition, then second primary molars are good substitute teeth for banding. Supporting wires extend anteriorly from the molars along the buccal and lingual surfaces of the posterior teeth, to add rigidity to the appliance. A midline jack screw is incorporated into the wire framework and is in close proximity to the palatal contour. The Haas expander has these figures as the Hyrax does, plus two additional acrylic pads attached to the

wire framework and closely contacting the palatal mucosa. There are no bands needed in the bonded expander; instead, the posterior occlusal acrylic coverage closely adapts to the buccal and palatal surfaces of the first permanent molars and premolars or primary molars. Many clinicians (McNamara and Baudon 1993, Sarver and Johnston 1989) believed that with the posterior occlusal acrylic coverage acting as a posterior bite block, bonded palatal expander produces less vertical dimensional changes than banded palatal expander does.

The force levels created during RME have been shown to vary, depending upon the age of the individual and the design of the appliance. An expansion screw activated once or twice daily will produce approximately 0.25-0.5 mm of expansion of the maxilla each day (hence the term "rapid maxillary expansion"). The Haas and Hyrax® expanders have been shown to produce orthopedic forces between 3-10 pounds by Isaacson and co-workers (1964), Zimring and Isaacson (1965), and Chaconas and Caputo (1982).

Chaconas and Caputo (1982) designed a three-dimensional anatomic model duplicated from a human skull and used different birefringent materials to simulate the various craniofacial structures. They compared five

appliances: Haas expander, Minne expander, Hyrax® expander, quad helix, and a removable expander. They found that each appliance produced different load-activation characteristics. Stresses produced by fixed appliances were concentrated in the anterior region of the palate, progressing posteriorly toward the palatine bones. These stresses radiated superiorly along the perpendicular plates of the palatine, the lacrimal, nasal, and zygomatic bones, and the pterygoid plates of the sphenoid. The authors believed that the quad helix, although it caused palatal separation, was the least effective orthopedic device. They also observed that the removable expanders were displaced before producing sufficient pressure to cause midpalatal splitting.

Effects of RME on the Sutures

Rapid maxillary expansion occurs when the force applied to the teeth and the maxillary alveolar processes exceeds the limits needed for orthodontic tooth movement. The applied pressure acts as an orthopedic force that separates the midpalatal suture. The appliance compresses the periodontal ligament, bends the alveolar processes, tips the anchor teeth, and gradually opens the midpalatal suture (Haas 1961). Ekstrom, Hennrickson, and Jensen (1977) found that the mineral content within the

suture rose rapidly during the first month after the completion of suture opening. In the bone beside the suture, the mineral content decreased sharply during the first month, but returned to its initial level within three months. Ten Gate et al. (1977) found that opening of the suture involves tissue injury followed by a proliferation repair phenomenon that ultimately leads to regeneration of the suture.

Wertz's study (1970) of three dry skulls, one adult and two children in the mixed dentition, indicated that the shape of the anteroposterior palatal separation was nonparallel in all three skulls. Viewed frontally, the maxillary suture was found to separate superoinferiorly in a nonparallel manner (Wertz 1970, Hass 1961,1970). The separation was pyramidal in shape with the base of the pyramid located at the oral side of the bone.

Relation between Amount of Sutural Separation and Extent of Molar Expansion

Krebs (1958, 1959, 1964) studied maxillary expansion with metallic implants. He placed implants in the alveolar process lingual to the upper canines, along the infrazygomatic ridge and buccal to the upper first molars. He found that the mean increase in intermolar distance measured on the casts was 6 mm, while the mean

increase in infrazygomatic ridge distance was 3.7 mm. In twenty of twenty three patients examined, the amount of sutural opening was either equal to or less than one half the amount of dental arch expansion. He also found that the average sutural opening was twice as great between the incisors than it was between the molars.

Effects of RME on the Maxillary Arch Width

A mean overall expansion of 6.0 mm at the molars and 6.2 mm in alveolar width was observed by Spillane (1990) in mixed dentition patients.

Effects of RME on the Maxillary Posterior Dentition

With the initial bending and compression of the periodontal ligament, there is a definite change in the long axis of the posterior teeth. Hicks (1978), using slow maxillary expansion, found that the angulation between right and left molars increased from 1-24° during expansion. Not all of the change, however, is caused by alveolar bending, but is partly due to tipping of the teeth in the alveolar bone. This tipping is usually accompanied by some extrusion (Byrum 1971, Hicks 1978).

Timms (1968) and Herberger (1987) observed that maxillary molars tipped buccally during expansion, then uprighted during the retention period. The uprighting of

the molars to nearly their pre-treatment angulation during the retention period resulted in the expanded arch width decreasing slightly.

Effects of RME on the Mandible

It is generally agreed that with RME there is a concomitant tendency for the mandible to swing downward and backward. There is some disagreement regarding the magnitude and the permanency of the change (Haas 1965,1970, Wertz 1970). The consistent opening of the mandibular plane during RME is probably explained by the disruption of occlusion caused by extrusion and tipping of maxillary posterior teeth along with alveolar bending.

Effects of RME on the Mandibular Dentition

Following RME, the mandibular teeth have been observed to upright (Haas 1965,1970) or to remain relatively stable over the short period of treatment (Wertz 1970). Gryson (1977) recorded changes in maxillary and mandibular intercanine and intermolar widths before and after expansion in thirty-eight patients. The ages of the groups ranged between six and thirteen years. The mean increase in the mandibular intermolar width was 0.4 mm; most patients either had no change or showed an increase of up to 1 mm.

In Brin et al. study (1996), the mandibular intermolar widths before and after maxillary expansion of twenty-two children in mixed dentition were measured. They found 50% of the cases (11/22) had "spontaneous" arch width increases, 27% (6/22) had no change, and 23% (5/22) had arch width decreases.

Wertz (1970) studied mandibular intermolar width in forty-eight patients who had received RME. No gain in intermolar width was seen in thirty-five patients; a gain of 0.5-2.0 mm was found in twelve patients, and a loss of 1.0 mm occurred in one patient.

Factors Influencing Treatment Response

A number of factors may influence the type of tooth movement and stability of the expansion achieved during rapid maxillary expansion. These factors may include appliance design, the age at which the expansion is initiated and the method of retention.

Appliance Design

Rapid versus slow maxillary expansion

There are two schools of thought concerning the speed of palatal splitting (Bell 1982, Firacelli 1978, Haas 1961, Kerbs 1958, Wertz 1970). Advocates of "rapid" expansion (1-4 weeks) believe that it results in minimum tooth movement (tipping), and maximum skeletal

displacement. Advocates of "slower" expansion (2-6 months) believe that it produces less tissue resistance in the circummaxillary structures and better bone formation in the intermaxillary suture, two factors that help minimize post-expansion relapse.

The rate of rapid maxillary expansion is 0.2-0.5 mm per day and can result in an increase in intermolar width of up to 10 mm. Skeletal changes are approximately 50% of the total change (Wertz 1970, Bell 1982).

Slow expanders like the quad helix and W-spring can transmit forces ranging between several ounces and two pounds (Firacelli 1978, Hicks 1978). They can separate the maxillae, particularly in the deciduous and mixed dentitions. The rate of separation varies between 0.4-1.1 mm per week. Slow expansion can result in an increase in intermolar width of up to 8 mm. Skeletal changes are estimated to be 16-30% of the total change and vary with age (Bell 1982).

Several investigators have examined the effect on arch dimensions of appliances that have been used for palatal expansion. A comparison of treatment effects produced by a banded rapid palatal expander and bonded expander was conducted by Sarver and Johnston (1989). They examined patients who had received a banded expander

(thirty seven females, aged seven to twenty nine years and twenty three males, aged eight to fourteen years) and observed a downward and forward movement of maxilla. Patients treated with a bonded expander (fourteen female and six males, mean age of 10.8 years) experienced a decreased anteroinferior maxillary displacement as compared to the patients treated with banded expanders. Sarver and Johnston (1989) surmised that, due to decreased anterior movement and clockwise rotation of the maxilla, Class II individuals would be better treated with a bonded, rather than banded, expander.

Herold (1989) examined the effect of Hyrax®, quad-helix, and removable expansion appliances upon tooth movement, arch dimensional changes and long-term stability. Fifty patients were split into three groups: a Hyrax® group (nineteen females and three males, average age 13.0 years), a quad-helix group (sixteen females and four males, average age 12.3 years) and a removable appliance group (five females and six males, average age 11.2 years). The evaluation of the quad-helix and removable appliance groups revealed that the molar teeth tilted buccally upon expansion. In contrast, the Hyrax® expansion appliance produces bodily movement of the

molars. The differences between the end of treatment and post-expansion (average 4.9 years out of retention) measurements indicated that the stability of expansion by different appliances varied. The quadhelix group (where 90% of the patients had no crossbite at the end of the treatment) was the least successful, with only 40% maintaining a crossbite correction at long-term recall. In contrast, the Hyrax® group, the successful maintenance of crossbite correction changed from 68.4-63.1%, with only one patient having crossbite relapse. For the removable appliance group, 81.8% achieved crossbite correction, with 72.7% maintaining this correction. However, as Herold mentioned, because this group was much small than the other two, less value can be placed upon the results.

Age or Maturation

Growth at the midpalatal suture was thought to cease at the age of three years (Latham 1971). By means of implants, Bjork and Skielle (1974) found that growth at the suture might be occurring as late as thirteen years of age. Persson and Thilander (1977) in a study on cadavers found that 5% of the sutures were obliterated by age twenty five years, yet the variation was such that a

fifteen-year-old cadaver had an ossified suture, while a twenty seven-year-old cadaver had an unossified suture.

Younger individuals were observed to have increased cellular activity in the midpalatal suture Ten Gate and co-workers (1977). Melsen (1972), in a biopsy study of eight individuals aged eight to nineteen years old, reported an increase in the degree of cellular activity in treated patients. In the individuals eight to twelve years old, osteoblastic activity was observed primarily near the bone surfaces, but was also found in bundles of fibers that formed bone bridges connecting the halves of the maxilla. In older individuals, RME resulted in numerous microfractures adjacent to the sutural area. Only in younger patients who had not attained maximum pubertal growth did true stimulation of sutural growth occur.

Brin and associates (1981) evaluated the relationship between RME and cyclic nucleotides in the suture. They concluded that older animals are less responsive to the applied forces than younger animals, hence the decreased ability of the older group to adapt to the forces of RME.

Krebs (1964) was one of the first to report a relationship between age at time of expansion and the

degree of midpalatal suture opening. Orthopedic change accounted for nearly 50% of the increase in arch width in patients aged 8-12 years old. The 13-19 year old group experienced approximately 30% of the increase in arch width via skeletal change. The younger individuals also experienced less relapse following expansion.

Wertz (1970) reported an interesting age difference in intermolar width changes following RME. He divided his sample into three age groups: Under 12, 12-18, and over 18 years. He found that after expansion and during fixed retention there was little relapse in any of the three groups (-0.5, -0.6, and -0.5 mm, respectively). On the other hand, each age group behaved differently from the time of appliance removal to the end of retention. The group under 12 years of age had a further increase in intermolar width of approximately 16%, the 12-18 years group had a relapse of approximately 10%, and the over 18 years group had a relapse of approximately 63%.

Method of Retention and Relapse Tendency

Hicks (1978) observed that the amount of relapse is related to the method of retention after expansion. With no retention, a 45% relapse was found to occur as compared with only a 10-23% relapse with fixed retention

and a 22-25% relapse with removable retention. Krebs (1959, 1964) found that after fixed retention was discontinued, there was a substantial reduction in dental arch width. This tendency continued for up to five years.

After a review of the literature, Bell (1982) concluded that slow expansion is less disruptive to the sutural systems. Slow expansion that maintains tissue integrity apparently needs 1-3 months of retention, which is significantly shorter than the 3-6 months recommended by Ekstrom et al. (1977) for rapid expansion. Mew (1983) advocated a total retention period of 1.5-4 years, depending on the extent of expansion.

Department of Orthodontics

THE STATEMENT OF THESIS

The objectives of the present study were to investigate the presence of arch asymmetry in children with UPXB and molar subdivision malocclusions and to evaluate the effects of rapid maxillary expansion on this type of malocclusions. The extent and location of the asymmetry in the maxilla and the mandible were measured. Secondly, the nature of the molar subdivision malocclusions was assessed. The specific asymmetric variables contributing in each arch to malocclusions were identified. The specific goals of the present study are as follow:

1. To determine the extent and location of arch asymmetries present in unilateral posterior crossbite (UPXB) and to evaluate the effectiveness of using rapid palatal expanders in correcting UPXB and molar subdivision malocclusions.

2. To determine the extent and location of arch asymmetries related to molar subdivision malocclusions at two periods of time.

3. To identify, if possible, some specific arch asymmetric variable contributing to molar subdivision, which can be effectively corrected or reduced by rapid maxillary expansion.

MATERIALS AND METHODS

Sample Selection

The sample consisted of thirty-six consecutively treated patients with combined unilateral posterior crossbite and molar subdivision malocclusions treated with bonded or banded palatal expansion appliances. These cases were selected from the office files of an orthodontist certified by the American Board of Orthodontics. Each case had complete records including dental casts and cephalometric radiographs at two time periods: Pre-treatment (T1) and before comprehensive orthodontic treatment with fixed appliances (T2). The mean age was 9 years 3 months at T1 and 12 years 8 months at T2. The average duration between T1 and T2 was forty-one months. The modality of the treatment consisted of expansion of the maxillary arch by means of bonded or banded palatal expansion appliances (Figures 3 and 4). The bonded palatal expander was fabricated to have acrylic coverage of the occlusal and palatal surfaces with a Hyrax® expansion mechanism embedded in the palatal acrylic. The occlusal table was wide enough to allow for

at least 5 mm expansion on each side for mandibular posterior tooth support. This appliance design was activated one turn every other day. The banded palatal expansion appliances were of the Hyrax® design (without acrylic), or Haas appliance (with palatal acrylic). This appliance was activated by one turn a day (0.25 mm). These appliances were activated until the palatal cusps of maxillary first molars were almost in crossbite position with the buccal cusps of lower first molars. A three to four month retention phase followed active expansion with the expander stabilized in place. Once the appliance was removed after the retention phase, a removable acrylic retainer was placed. The retainer was to be worn full time for 3-6 months and then night wear as long as possible.

Measurement of Dental Casts

The serial maxillary and mandibular dental casts were measured with a digital imaging system (Bioscan OPTIMAS® Imaging System, Seattle, Washington).

Cast Evaluation

All of the models were trimmed with a standard procedure in centric occlusion (Fig.10). A black HB automatic pencil with a 0.3 mm tip was used to mark the following anatomic landmarks on the maxillary and mandibular dental casts at the two time periods:

1. The posterior two thirds of the mid-palatal raphe was highlighted with the pencil. A piece of transparent Plexiglas™ with a straight green line was then placed on the occlusal surface of the cast. The straight green line was superimposed on the most posterior extension of the median raphe and best fit to the pencil line of the posterior 2/3 of the median raphe while looking directly from above. A black pencil mark was made on the maxillary central incisor at the intersection of the straight green line with the incisal edge. This was the anterior palatal midpoint.

2. Mesiobuccal and distobuccal cusp tips of the first permanent molars

3. The lowest points of mesiopalatal gingival margins of the maxillary first permanent molars

4. Buccal cusp tips of permanent premolars or mesiobuccal and distobuccal cusp tips of the first and second primary molars

5. Cusp tips of primary or permanent canines

6. Midpoint of two maxillary central incisors

7. Midpoint of two mandibular central incisor

8. Mandibular midline: The maxillary and mandibular casts were occluded together in centric occlusion. The palatal anterior midpoint was then transferred to mandibular incisal edge. This was the anterior mandibular

midpoint. Secondly, Looking at both casts from behind (lingual aspect), a piece of Plexiglas™ with a straight green line was held and superimposed on the maxillary posterior palatal midpoint, posterior two thirds of the median raphe and the mandibular anterior midpoint. A black pencil mark was then made on the mandibular base at the intersection of the green straight line with posterior base. This was the posterior mandibular midpoint. The line connecting anterior and posterior mandibular midpoints served as the mandibular midline.

The two sets of casts of each case were consecutively marked to ensure that the location of all the landmarks were identical at each time period. Once the landmarks were marked, the occlusal image of each dental cast was then captured into the computer.

Image Capturing

The image component of the system consisted of a digital camera mounted on a copy stand with attached lighting (Figure 5), a cast holder (Figure 6) and a Plexiglas™ stand (Figure 7). The dental cast was mounted on a specially designed cast holder that could be rotated freely and be elevated up and down (Figure 6). First, the screws for locking orientation and height of the table were loosened. The Plexiglas™ stand was used to orient the occlusal plane of the cast parallel to the camera lens. This was done by contacting the teeth of the model bilaterally with the transparent Plexiglas™

plate (Figure 7). A standardized distance from the occlusal plane to the camera lens was also obtained at the same time by adjusting the screw of the cast holder. Once the ideal position was reached, the screws of the cast holder were tightened. The dental cast holder could also be turned 90° in order to evaluate the maxillary first molars angulation from the distal aspect bilaterally (Figures 6, 11). The position of the maxillary first molar was aligned at the same level as the transparent Plexiglas™ plate was.

The lens was calibrated to the nearest 0.2 mm at a set distance from the lens to a point located directly below the bottom surface of the Plexiglas™. The Plexiglas™ stand was then removed and the lighting intensity was adjusted to reach the optimal contrast as viewed on an adjacent video monitor. The dental cast, as viewed on the video monitor, was magnified three times. The image was stored on a zip drive for later analysis. A schematic drawing of the entire system is shown in Figure 9. The cast was removed, landmark digitization and computation was completed. Each cast was digitized with a mouse from the image on the video monitor.

Data Collection

For the maxillary arch, the anterior and posterior palatal midpoints were first digitized. A palatal midline was then constructed by connecting these two points. Linear measurements were made from each of the eight bilateral dental landmarks perpendicular to the palatal midline (Figure 10). An anterior reference line was constructed perpendicular to the palatal midline through the midpoint between the central incisors. Anteroposterior measurements of the same dental landmarks were then made perpendicular to this anterior reference line (Figure 10).

For the mandibular arch, a similar procedure was followed. The anterior and posterior mandibular midpoints were connected with a straight line, creating a mandibular midline (Figure 10). An anterior reference line was constructed perpendicular to the mandibular midline. Linear measurements were taken in both transverse and anteroposterior directions (Figure 10). The measurements taken on the dental models are illustrated in Figure 10.

Asymmetries were calculated by determining the absolute difference between homologous measurements as related to both reference lines. Dental midline

deviations were assessed by making linear measurements from the midpoint between the central incisors to the palatal midline and its analogue on the mandibular model. If the midline deviated to the right side of the palatal/mandibular midline, the value was positive. If the midline deviated to the left side of the palatal/mandibular midline, the value was negative.

Maxillary permanent first molar angulation was determined by calculating the angle of intersection of lines drawn through the mesiobuccal and mesiopalatal cusps of the maxillary first molars (Figure 11). An angulation of less than 180° indicated that the teeth were tipped buccally, while values over 180° indicated that the teeth were tipped lingually.

The molar relationship was quantified by measuring the distance between the mandibular mesiobuccal groove and the maxillary mesiobuccal cusp tip in centric occlusion on the model. A digital caliper (Fowler Sylvac Caliper Ultra-Cal III[1]) was used and measured to the hundredth of a millimeter. Class I molar relationship was defined as the middle of the mesiobuccal cusp of the maxillary first molar in the exact middle of the buccal

[1] This caliper is the product of Fred P. Fowler Co. Inc. 626 Rowe Street, P.O. Box 299, Newton, MA 02166

value was zero. If the maxillary mesiobuccal cusp tip was mesial to the mandibular mesiobuccal groove (class II molar relationship), then the value was positive. The amount of subdivision was then calculated by the molar value on the right side minus that on the left side. Therefore, a positive subdivision value would be a class II subdivision right side, while a negative value indicated a subdivision on the left side.

The success of UPXB correction was strictly defined as complete elimination of the UPXB, irrespective of the midline position and subdivision categories. Cases in which a single tooth remained in an edge-to-edge relationship was considered a failure. Arch width of the maxilla and mandible were calculated by adding bilateral homologous tooth landmarks to midline distance together.

Statistical Analysis

The percentiles of successful UPXB and subdivision correction were calculated. Twenty-seven variables of arch asymmetries and arch width were obtained from maxillary and mandibular casts. Means and standard deviations were calculated for each of the variables at T1 and T2. The paired Student t-test was used to compare all the variables at T1 with those at T2. A variable was

considered significant if its p-value was below 0.0018. This level was chosen in accordance with the Bonferroni correction as follows. The alpha for the entire set of comparisons was chosen to be 0.05 and was distributed equally across the entire set of twenty-seven comparisons. Therefore, after Bonferroni correction, the significant p-value was 0.0018 (0.05/27= 0.0018). This controls the type I error probabilities in the entire set of planned comparisons so that the maximum probability of type I error is 0.05.

The second objective of the present study was to determine the extent and location of the arch asymmetries contributing to the molar subdivision malocclusions at T1 and T2. The Pearson correlation coefficient tests were performed to relate the amount of molar subdivision to that of every arch asymmetry variable at T1 and T2. The amounts of molar subdivision and arch asymmetry were determined by the right-hand side value minus the left-hand side value. Significant level was set at $p < 0.05$.

An attempt to identify specific variables contributing to molar subdivision was made. All subjects were divided into two subgroups based on the amount of molar subdivision correction. The author arbitrarily chose to divide the sample into two subgroups with those

equal or greater than I mm of molar subdivision correction (subgroup A) and those lesser than I mm of molar subdivision correction (subgroup B). The author felt that 1 mm change would indicate clinic significance. Coincidentally, there were eighteen subjects in each subgroup. Paired Student t-tests were used to evaluate arch asymmetric changes between T1 and T2 for two subgroups: Subdivision correction $\geq$ 1mm correction (subgroup A) and subdivision correction < 1mm (subgroup B).

RESULTS

Reliability of the Measurements

In order to examine possible measurement error, 12.5% of the sample was randomly selected and re-digitized and re-measured on the captured images seven days following the initial digitization and measurement of the same sample. Intra-class correlation tests were used to test the original and re-assessed measurements. The result demonstrated a satisfactory reliability of measurements (Table 1). The r-value of the majority of measurements was between 0.99 and 0.98. The lowest r value was 0.96. ($r = MSB-MSW/MSB+MSW$, $p < 0.05$)

The Success Rate of Unilateral Posterior Crossbite and Molar Subdivision Correction

The success rate for correction of unilateral posterior crossbite was 83.34% (Table 2). For class II molar subdivision correction, however, only ten of thirty-six (27%) subjects had corrected completely (0 mm difference between right and left sides). There were eight subjects having molar subdivision malocclusions less than 1mm at T2. However, the average molar

subdivision amount was reduced from 2.32 (±1.33) mm at T1 to 1.46 (± 1.48) mm at T2.

Arch Dimensional Changes during Maxillary Expansion and Retention.

Arch width expansion was determined by the difference of corresponding buccal cusps and palatal gingival points on pre-expansion (T1) and post-expansion (T2) dental casts. Means and standard deviations for maxillary and mandibular arch width at canines, premolars, and first molars are listed in Table 3. A significant increase ($p < 0.0018$) in arch width occurred as a result of treatment at all measurement points of the maxilla (Table 3). There was a slight increase in mandibular arch width. However, these changes were not statistically significant.

Arch symmetry changes were measured in two dimensions; the transverse direction and the anteroposterior (AP) direction. Means and standard deviations for maxillary and mandibular arch asymmetry changes at canines, premolars, first molars, maxillary midline and mandibular midline are listed in Table 3. The maxillary arch presented mild transverse and AP arch asymmetries at T1 (Table 3). After expansion, there were no significant changes in both maxillary transverse and

AP arch symmetries (Table 3). The mandibular arch presented moderate transverse and mild AP arch asymmetries at T1 (Table 3). After maxillary expansion, there was a significant decrease ($p < 0.0018$) in mandibular arch transverse asymmetry. In addition, mandibular midline also showed a significant decrease ($p < 0.0018$) from 1.7 mm at T1 to 1.1 mm at T2. There were no significant changes in mandibular AP arch asymmetries at T2.

Bodily Tipping or Bodily Movement of Anchor Teeth

Transpalatal width increases measured at the buccal and palatal gingival points were compared to determine if rapid maxillary expansion caused a buccal tipping of the anchor teeth. The average increases in transpalatal width, measured at the buccal and palatal gingival points of the maxillary first molars, were 3.94 ($\pm$1.92) mm and 3.69 ($\pm$2.17) mm respectively. There was no significant difference observed from T1 to T2 ($p = 0.153$). In addition, inter-first molar angles recorded at T1 and T2 were compared. The average inter-first molar angles were 165.47° ($\pm$4.78) at T1, and 165.67° ($\pm$5.28) at T2. As in the inter-first molar distances, no significant difference were observed.

Correlation between the Class II Molar Subdivision and Arch Asymmetries from T1 to T2

The Pearson product-moment correlation test was used to determine if correlation existed between molar subdivision and arch asymmetry. Significant correlations ($p < 0.05$) between molar subdivision malocclusions and arch asymmetry at T1 were found in the mandibular transverse measurements. In order of significance were the primary first molar, canine, second molar, permanent first molar and mandibular midline (Table 4). Significant correlations also were found in all mandibular AP asymmetry variables (Table 4) at T1.

There was no significant correlation between molar subdivision and maxillary arch asymmetry except maxillary first permanent molars in the transverse dimension: ($r = -0.355$, $p < 0.03$). The negative correlation indicated that the narrower the maxillary side was, the more the molar subdivision malocclusions presented on the same side, irrespective of crossbite side.

At T2, the sequence of the correlation between molar subdivision and arch asymmetry changed. The most significant correlations ($p < 0.05$) were found in mandibular AP asymmetry variables. This was followed by the mandibular transverse asymmetry variables (Table 4).

The strength of relationship between mandibular dental midline and subdivision was reduced at T2 ($r = 0.382$, $p < 0.022$). There were also significant correlations ($p < 0.05$) between molar subdivision and maxillary AP asymmetries at T2 (Table 4).

Arch Dimensional Changes in Two Subgroups: Molar Subdivision Correction $\geq$ 1mm and Molar Subdivision Correction < 1mm Subgroup

Paired Student t-test was used to compare arch width and asymmetry at T1 and T2 for each subgroup. Mean and standard deviations of each variable in each subgroup were listed in Table 5 and 6. The results showed that the maxillary arch width significantly increased in both subgroups at T2. Although the average increase in subgroup A was greater than that in subgroup B, there were no statistically significant difference between the increases of the two subgroups. Neither subgroup at T2 had a significant increase in mandibular arch width and there were no significant differences between the two subgroups.

In subgroup A, there were significant decreases ($p < 0.0018$) in mandibular transverse arch asymmetry in all measurements at T2 (Table 5). There was 0.9 mm reduction of the mandibular midline deviation in subgroup A ($p = 0.006$) and 0.4 mm reduction in subgroup B ($p = 0.044$).

However, both reduction were not statistically significant due to an extremely small p-value (P = 0.0018). In subgroup B, there were no significant decreases in mandibular transverse arch asymmetry in all measurements at T2 (Table 6). For all other changes in mandibular and maxillary arch symmetry, both subgroups presented small and non-significant changes at T2 (range from 0.02 mm to 0.5 mm).

Paired Student t-test was also used to compare the numbers of turns for active expansion and the patients' ages at T1 between two subgroups (Table 8). Mean and standard deviations of these two variables in each subgroup were listed in Table 8. No significant differences were found between these two subgroups regarding the amount of active expansion at T1 and the age of initiating treatment (p = 0.5, p = 0.12 respectively).

DISCUSSION

Limitations of Present Study

Some limitations exist in the present study. The sample size is not large due to the very specific sample criteria. The subjects of the present study were obtained from one orthodontic clinic. Patients were specifically selected that had combined UPXB and molar subdivision malocclusions, and were later treated by the same orthodontist with the same treatment modality (rapid maxillary expansion appliances). In addition, this is a longitudinal study, in which the average participation time for every subject was forty-one months. In comparison to previous publications, these limitations are not unique. Only five studies had been done regarding the skeletal and dentoalveolar asymmetries in molar subdivision malocclusions (Alavi et al. 1988, de Araujo et al. 1990, Rose et al. 1994, O'Byrn et al. 1995, Ben-Bassat et al. 1996). Two of them also included UPXB (O'Byrn et al. 1995, Ben-Bassat et al. 1996). Four of the five studies were cross-sectional studies (Alavi et al. 1988, de Araujo 1990, Rose et al. 1994, O'Byrn et al.

1995). Their sample sizes of experiment groups were also small, ranging between twenty-eight to thirty. The only longitudinal study by Brin et al. (1996) consisted of only fifteen subjects with combined UPXB and molar subdivision malocclusions.

The other limitation is the lack of a control group. The one control group could be two sets of records from untreated, age-matched children with combined unilateral posterior crossbite and molar subdivision malocclusions. Another control group could probably be non-orthodontically treated, age-matched children with normal occlusions. These were not done since the similarity of growth pattern between these two groups is questioned.

Several studies have shown that untreated UPXB may lead to craniofacial skeletal asymmetry (Thilander et al. 1984, Pirttiniemi et al. 1990), which indicated that a different growth pattern may develop in this type of malocclusions. If this is the case, then the comparison between two groups at two periods can not provide accurate information in respect to the effects of treatment. Therefore, in the present study, there was only one group of subjects, and records were taken before treatment and after retention. The pre-treatment record served as the control of the same subject.

Reliability of Measurement

In the present study, the reliability of measurements was satisfactory (r = 0.998-0.96). This was attributable to several factors. First, the specially designed adjustable cast holder allowed re-orientation of the occlusal plane of the model parallel to the camera lens at a standardized distance. In the author's opinion, the precise positioning of the model was probably overlooked by many studies. For example, in Alavi et al. (1988), the models were photographed with a 35 mm telephoto set-up at a standard object film distance. The distance was assumed to be from the base of the model to the camera, because no special paralleling aids were mentioned. The method of divider and ruler were used in Brin's study. Cook (1980) found that while using the PA cephalometric radiograph for assessment of craniofacial asymmetry, that less than 5 mm of head rotation could reverse the apparently dominant side of the asymmetry. This explained the importance of the object's orientation, while assessing symmetry. Secondly, the digital image system can provide accurate measurements through a software program that has the ability to adjust the magnification of the captured image. Thirdly, when

magnified three times, the images on the screen make precise digitization easier.

Correction of Unilateral Posterior Crossbite

In the present study, there were strict criteria for the success of the crossbite correction. The presence of a single tooth in edge-to-edge occlusion was considered a failure. In the failed group, three out of six (50%) subjects had only a single tooth in edge-to-edge occlusion.

The success rate for correction of unilateral posterior crossbite was 83% (30/36). This is higher than that of most of the other studies (Table 7). There were several studies conducted for treatment effects of Hyrax®, Haas, and bonded RME. The success rates of crossbite correction were not reported in some articles (Spillane 1990, Brust 1992, Herberger 1987). Instead, the authors reported arch width change. In addition, their sample included patients with crossbite and non-crossbite malocclusions.

The mean maxillary arch width increase in the present study was 3.9 mm at first permanent molar with a S.D. of 1.9 mm. This result is similar to the one found by Spillane (1990), who observed 4.8 mm of retained expansion, and Brust (1992) who observed 5.1 mm of

retained expansion before phase II treatment, but less than the 6.3 mm of retained expansion, which was found by Herberger (1987).

The high success rate of correcting unilateral posterior crossbite may be attributed to the adequate orthopedic expansion of the maxilla obtained from the rapid maxillary expansion. This result is in agreement with Hass (1970). In addition, a sufficient retention period, including at least three months fixed retention and three months full-time removable retainer wear, followed by night time wear in most cases, ensured the stability of expansion.

Maxillary Molar Angulation

There were no significant maxillary molar angular changes from T1 to T2. This indicated that the net increase in maxillary width was gained from bodily tooth movement. Tipping of the maxillary molars may have occurred during expansion, but relapsed from T1 to T2. Spillane (1990) and Brust (1992) also showed similar results.

Mandibular Arch Width Changes

A small, insignificant increase in arch width (0.2-0.5 mm) was found in the mandibular arch after maxillary expansion and retention. This arch width increase may be

due to the normal growth during the transition from the primary to the early permanent dentition observed by Sinclair and Little (1983). They observed a small, insignificant increase (0.6 mm) in intermolar width in males and a statistically significant inter-molar width loss (0.9 mm) in females from the mixed dentition to early adulthood, while evaluating sixty-five untreated subjects. Gryson (1977) found no correlation between the change in mandibular intercanine and intermolar distances with respect to the increase in maxillary intercanine and intermolar distances. Therefore, it can be concluded that, in general, RME could influence the mandibular dentition, but the accompanying changes are neither pronounced, predictable, nor significant.

Arch Asymmetries and Their Changes after RME

Maxillary Asymmetry

A small amount of AP and transverse asymmetries were exhibited in the maxillary dentition (mean $\pm$ sd. = 0.8-1.1 mm $\pm$ 0.7-0.9mm) at T1 and T2. However, there was minimal arch asymmetry changes during expansion and retention (mean of T1-T2 = 0.05 mm). These findings supported the theory proposed by Kutin and Hawes (1969), Moyers (1966), Wertz (1970) that unilateral posterior

crossbite usually is caused by bilateral maxillary constriction.

In the current study, it appeared that the maxillary arches had been symmetrically expanded. This disagrees with the findings of Hermanson et al. (1985). They used a symmetrograph to measure the casts and found that the expansion effect was not symmetric on the two sides of maxilla. Before the maxillary expansion, the side with normal transverse occlusion was wider than the crossbite side. After expansion, the crossbite side was found to be wider than the normal side. This disagreement probably can be explained by the fact that he used the median line as the maxillary midline, while this study used a different definition. In the present study, only the posterior two thirds of median raphe was used as palatal midline, because the reliability of the anterior portion of the palatine raphe line has been questioned. Truque (1956) found that palatine raphe tended to deviate to the right of the mid-sagittal plane anteriorly after the first molar area. Lundstrom (1961) found that when raphe line was not straight but s-shaped, its value as an axis of symmetry was limited. He concluded that the recorded asymmetries in the AP or lateral direction must be assumed to be due, in part, to asymmetry of the raphe

line. In order to eliminate the variation of the median raphe, especially at the anterior portion, only the most posterior two-thirds was used as the palatal midline in the present study.

Mandibular Arch Asymmetries

In the present study, a greater amount of asymmetries was found in the transverse measurements (from 3.7 mm to 3.2 mm), than the AP measurements (from 1.5 mm to 1.1 mm) at T1. There are three factors which have been suggested to contribute to the asymmetries observed (Bishara 1994). These are mandibular functional displacement, and mandibular skeletal and dentoalveolar asymmetry. Mandibular displacement is a common finding in unilateral crossbite malocclusions. Mandibular lateral displacement was observed in 45 of the 57 unilateral crossbite cases (78.9%) of Thilander et al. (1984) study. Many others (Helm 1968, Ingervall 1974, Ingervall et al. 1978, and Kurol and Berglund et al. 1992) have observed the same. In the present study, the incidences of the functional displacement were not recorded at T1, but the significant reduction of mandibular transverse asymmetry at T2 supports the observations mentioned above.

Several researchers (Myers et al. 1980, Mongini 1984, Mongini and Schmid 1987, Pirttiniemi et al. 1990, 1991, Hesse et al. 1997) have reported the asymmetric morphology of temporomandibular joint in the subjects with unilateral posterior crossbite. Pirttiniemi et al. (1991) found the condyles were more asymmetrically positioned in relation to posterior walls of glenoid fossa in subjects with crossbite than in the subjects with normal occlusion. The correlation between functional posterior crossbite correction and condylar position changes was confirmed by Hesse et al.(1997). They found that compared to crossbite side, the condyle was positioned more anteriorly on the non-crossbite side than the crossbite side. After maxillary expansion, the condyle moved posteriorly and superiorly on the non-crossbite side.

The adaptive nature of the glenoid fossa has been shown in many primate studies (Adams et al. 1972, McNamara 1975, 1979, Hinton 1984, Woodside et al. 1987) after alteration of mandibular function and position. In human study, O'Byrn et al. (1995) found that the glenoid fossa was relocated more distally on the crossbite side in young adult with an untreated unilateral crossbite, while comparing to a class I malocclusions group.

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Therefore, the mandibular skeletal asymmetry, either in the condyle or the glenoid fossa, due to crossbite, may occur in the present study also.

The possible presence of tooth size discrepancy and incisors crowding may contribute to dentoalveolar asymmetry and midline deviation (Lundstrom 1969, kutin and Hawes 1969). This was not investigated in the present study.

Significant reductions($p < 0.0018$) were found in the transverse mandibular asymmetry and in the mandibular midline deviation forty-one months after maxillary expansion. There were no significant reductions in AP asymmetries (Table 3). The most likely factor contributing to asymmetric reduction is the reduction of mandibular displacement due to the unilateral posterior crossbite correction. In the study of Brin et al., the incidence of mandibular shift was reduced from 83.3% (20/24) before maxillary expansion to 4.2% (1/24) after maxillary expansion. In the Ben-Bassat et al. study, the incidence of mandibular shift was reduced from 85.3% (29/34) before maxillary expansion to 8.8% (3/34) after maxillary expansion.

There is a possibility of early remodeling of the glenoid fossa and the condyle in children. Mongini and

glenoid fossa and the condyle in children. Mongini and Schmid (1987) used repositioning splint, followed by orthodontic treatment to correct the mandibular asymmetry in eleven growing children. Remodeling of the glenoid fossa and condylar head were observed from serial transcranial radiographs over an average thirty-seven months of observation period (Mongini and Schmid 1987). In the present study, no additional treatment was provided to reposition the mandible in a more symmetric position. Therefore, the effect of maxillary expansion alone on the correction of mandibular skeletal asymmetry is questionable.

Dentoalveolar asymmetries, if any, would remain the same at T2, because there was no active forces applied to either the mandible or to the mandibular dentition from T1 to T2.

Subdivision Correction and its Relation to Arch Asymmetries

Only ten cases (27%) of molar subdivision malocclusions were successfully corrected in the current study. This success rate is lower than that of Brin's study (40%). But Brin's sample was much smaller (15). In the present study, the amount of molar subdivision was quantified. This measurement of molar subdivision may

help the clinicians relate the amount of molar subdivision to arch asymmetries and thereby authentic a specific treatment.

In reviewing the correlation between the subdivision and arch asymmetries at T1 and T2, the most significant and highly correlated variables were mandibular transverse asymmetry ($r = 0.85$, $p < 0.0001$) at T1 and mandibular AP asymmetry ($r = 0.65$, $p < 0.0001$) at t2. These findings indicate that mandibular transverse asymmetry has a stronger relationship with the subdivision malocclusions than mandibular AP asymmetry, while a subdivision coexists with a unilateral posterior crossbite malocclusions. Once the unilateral posterior crossbite was corrected and the mandibular shift was reduced, the relationship between mandibular AP asymmetry and molar subdivision malocclusions became more dominant. The mandibular transverse asymmetry, in the canines and premolars area, was second ($r = 0.49$, $p < 0.002$). The maxillary AP asymmetry was third ($r = -0.4$, $p < 0.018$). These findings were different from the mainstream of clinical orthodontists with the majority of mechanics treating molar subdivision malocclusions addressing the maxillary molars. (Tweed 1966, Haack and Weinstein 1958, Garland et al. 1981, Shroff et al. 1997).

There are several studies supporting our findings. de Araujo et al. (1994) evaluated the skeletal and dental arch asymmetry of thirty subjects with class II molar subdivision. The frequencies of molar asymmetry found on the casts were significantly higher on the mandible than on the maxilla ($p < 0.01$). The frequencies of mandibular midline deviation were also significantly higher than that of the maxillary midline deviation ($p < 0.01$).

Rose et al. (1994) studied the mandibular asymmetry in subjects with untreated molar subdivision malocclusions and compared these with a control group (class I normal occlusion). He used submentovertex radiographs to assess the mandibular asymmetry with three co-ordinate systems : The cranial floor, the mandible and the dentition. Rose et al. found that whether the position of the mandibular molars was measured relative to the cranial floor or within the mandible itself, the mandibular first molar was located more posteriorly (mean = 1.90 mm) on the class II side of the subdivision malocclusions than the control group (class I normal occlusion). O'Byrn et al. (1995) used the same system to assess adult subjects with combined unilateral posterior crossbite and molar subdivision malocclusions. They also took corrected tomograms for each subject in order to

assess condylar position within the glenoid fossa. They found that relative to a cranial floor co-ordinate system, the mandibular first molar on the crossbite side was relatively more lateral (mean = 5 mm) and more distal (mean = 2.17 mm) in comparison to the control group (class I occlusion). Similar findings were also found relative to the mandibular coordinate system.

Maxillary dentoalveolar AP asymmetry began to show a significant negative correlation ($r = -0.4$, $p < 0.018$) with subdivision at T2. It may be due to a small (about 0.3 mm), non-significant increase in maxillary AP dentoalveolar asymmetry at T2. The results of Leighton's (1988) longitudinal study of molar relationship support our findings. The asymmetric change of molar relationship was found between the mixed and permanent dentitions. The amount of this change was related to variations of deciduous molar size, especially that of the maxillary second primary molars.

The effect of mandibular shift reduction on subdivision correction was demonstrated in the current study. In subgroup "A" (subdivision correction > 1 mm), the mandibular transverse asymmetries had a statistically significant reduction at T2 (average 1.7 mm, $p < 0.001$), while subgroup "B" (subdivision correction < 1 mm) did

not. Although the number of turns of expansion was no significantly different in these two subgroups (average of 32.5 turns in subgroup A vs. 30 turns in subgroup B), the amount of retained maxillary expansion was greater, in subgroup "A" than that in subgroup "B" (4.5 mm vs. 3.2 mm). This was not statistically different. However, this may explain why subgroup "A" had more mandibular shift reduction than subgroup "B". In addition, the timing of treatment may not play an role in differentiate these two subgroups since there were no significant differences of patients' age at T1 between two subgroups (mean age at T1 of subgroup A and Subgroup B were 107 months and 114 months, respectively).

In summary, it has shown that there were remaining mandibular skeletal asymmetry and possible dentoalveolar asymmetry contributing to molar subdivision malocclusions after the unilateral crossbite was corrected. The average amount of molar subdivision was reduced after correction of the unilateral posterior crossbite in the present study. However, only 27% of the subjects had complete molar subdivision malocclusions correction. Therefore, adjunctive asymmetric orthopedic therapy, in addition to maxillary expansion, is suggested for children with unilateral posterior crossbite and molar

subdivision malocclusions, when no dental asymmetries exist.

For the future studies, using a combination of submentovertex radiography, corrected tomograms, and precise model analysis to evaluate the skeletal and dentoalveolar asymmetries, may further clarify the treatment effects at different regions of craniomandibular complex and define more specific treatment modalities.

CONCLUSIONS

The objectives of the present study were to investigate the presence of arch asymmetry in children with unilateral posterior crossbite (UPXB) and molar subdivision malocclusions and to evaluate the effects of rapid maxillary expansion (RME) on this type of malocclusions. Secondly, the nature of the molar subdivision were assessed. The subjects consisted of thirty-six consecutively treated children with unilateral posterior crossbite and molar subdivision malocclusions. Rapid maxillary expansion was the only treatment modality. Dental casts were taken pre-treatment (T1) and post retention (T2). The mean duration between T1 and T2 was forty-one months. The dental casts were digitized by using the Digital Imaging System (Bioscan OPTIMAS®) and measurements were computed. The arch asymmetries were measured in both AP and transverse dimensions. The findings list as follow:

1. Long-term stable results of rapid maxillary expansion for UPXB correction were achieved in a majority (83%) of the children.

2. Only 27% of the molar subdivision were corrected completely.

3. The greatest amount of arch asymmetry was found in the mandibular transverse measurements (3.7mm). This was statistically reduced at T2.

4. This asymmetry reduction was due to maxillary expansion that resulted in a reduction of mandibular displacement.

5. The mandibular transverse asymmetry is the primary contributor to molar subdivision. The molar subdivision malocclusions appears to coexist with UPXB. The combined effects of the mandibular shift, and the skeletal and dentoalveolar asymmetries result in the mandibular asymmetry.

6. The mandibular AP asymmetry became the most dominant factor (followed by mandibular transverse asymmetry and maxillary AP asymmetry) related to remaining molar subdivision malocclusions at T2.

7. It was found that the more the mandibular transverse asymmetry was reduced by RME, the more the molar subdivision was reduced. The mandibular AP asymmetry, however, was minimally affected by RME. This had a more significant relationship with remaining molar subdivision at T2.

8. Adjunctive asymmetric orthopedic therapy, in addition to maxillary expansion, may be necessary for children with UPXB and molar subdivision malocclusions, when no dental asymmetries are present.

9. little changes in maxillary permanent first molar angulation was noted forty-one months after RME.

10. RME did not result in significant increase in the mandibular arch width.

APPENDIX

Figures
Tables

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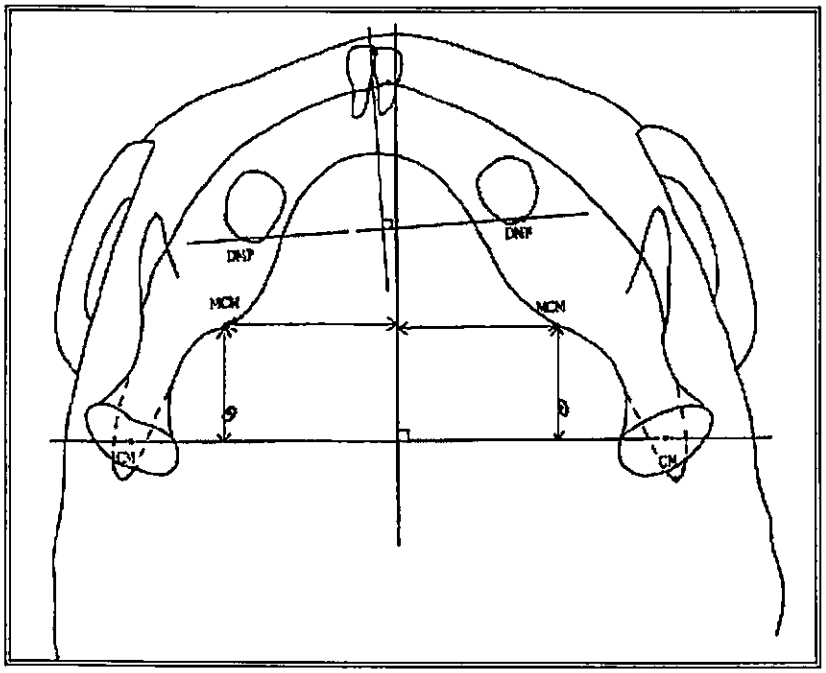


Figure 1. Mandibular skeletal and dental coordinate systems. Spatial position of medial contour of the mandible (MCM) to mandibular skeletal coordinate system is illustrated. DMP: distal molar point. (Rose et al. 1994)

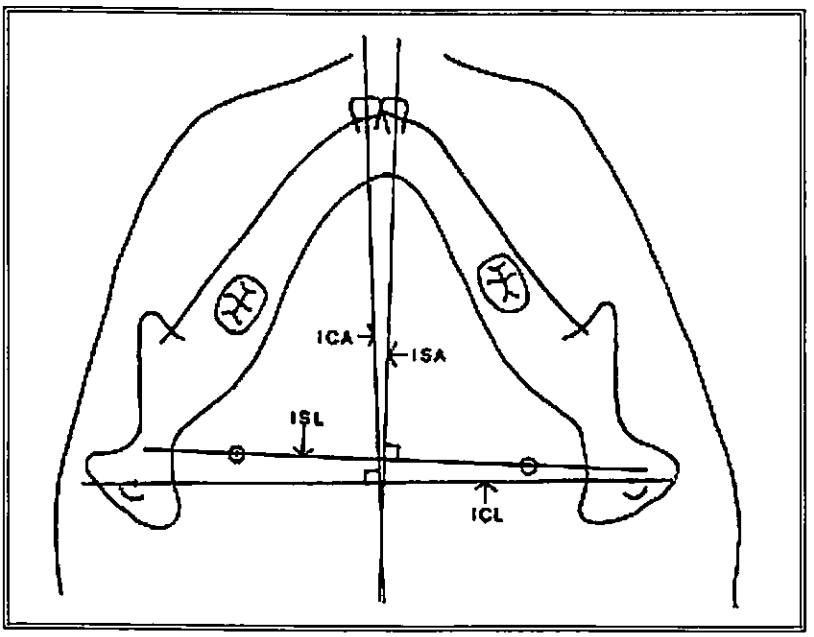


Figure 2. Submentovertex coordinate systems. Intercondylar line (lCL), intercondylar axis (ICA), interspinosum line (lSL), interspinosum axis (ISA). (O'Byrn et al. 1995)

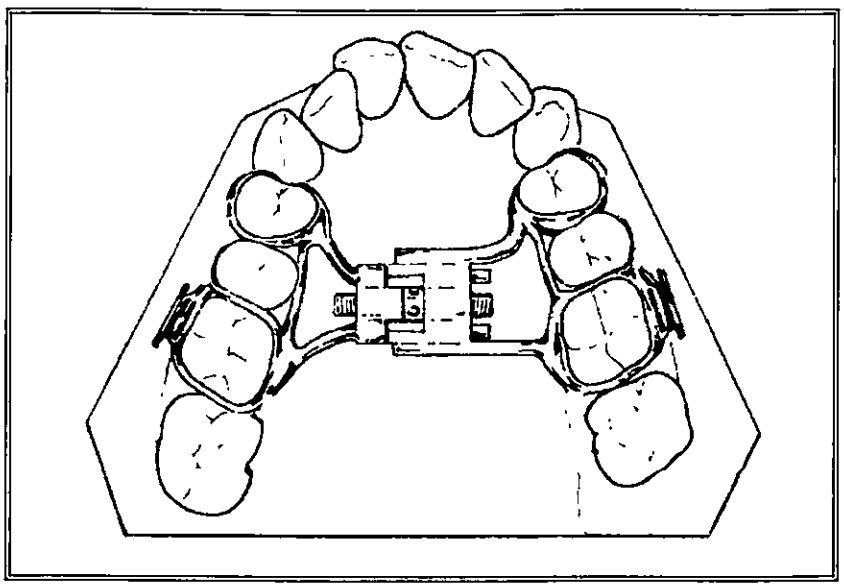


Figure 3. Hyrax®-type Maxillary Expander
(Modified graph from McNamara and Brudon 1993)

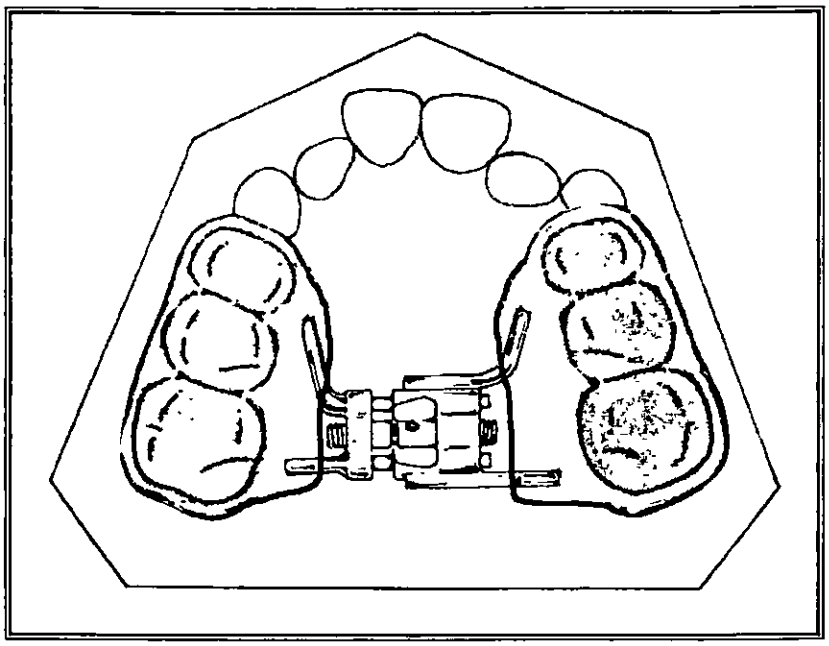


Figure 4. Bonded maxillary expander
(Modified graph from McNamara and Brudon 1993)

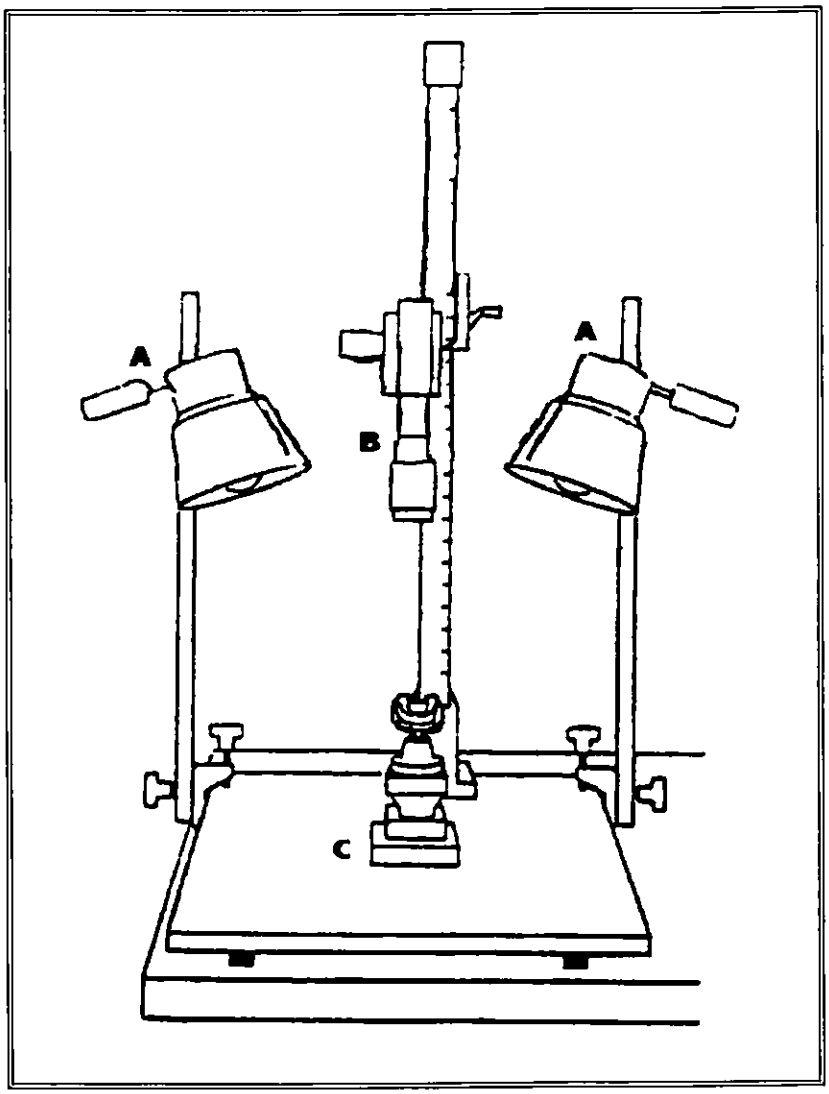


Figure 5. Photo stand with A) lighting, B) digital camera, and C) cast holder.

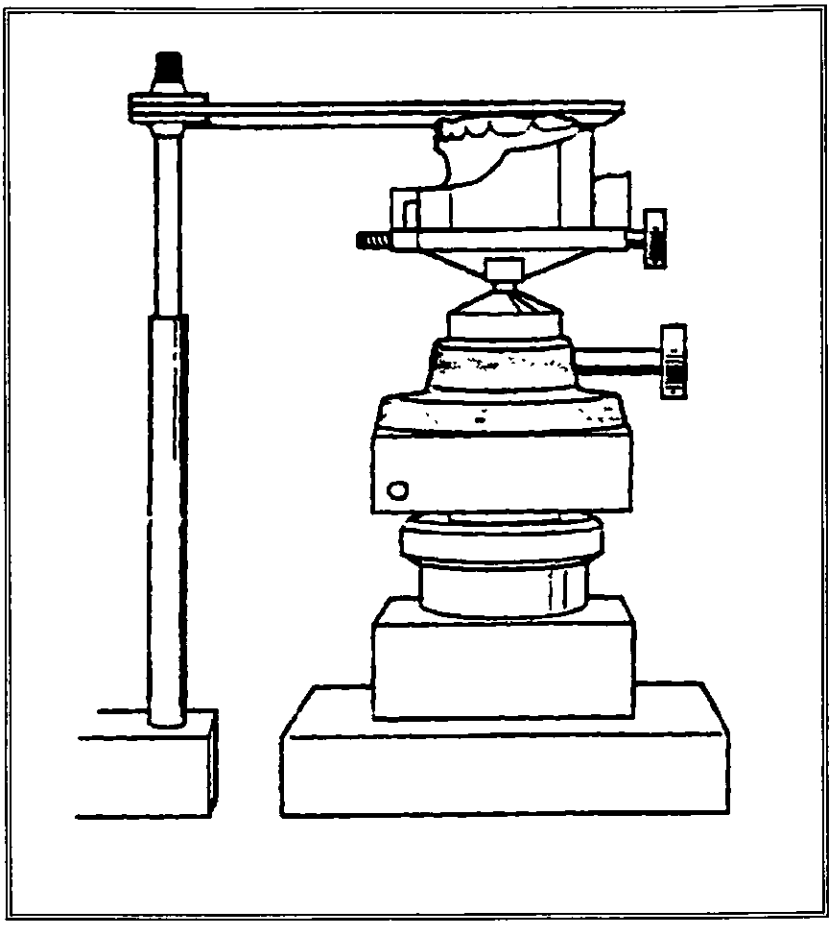


Figure 7. The stand holding the Plexiglas™ can be adjusted to the height of the cast holder, making the occlusal plane parallel to the lens.

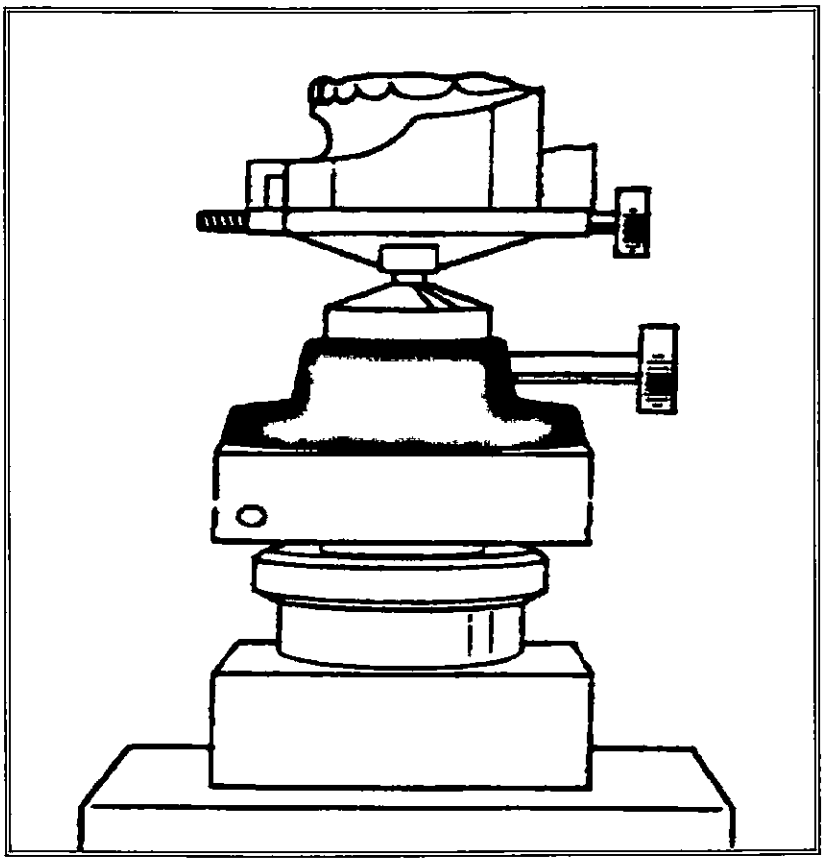


Figure 6. Cast holder with model

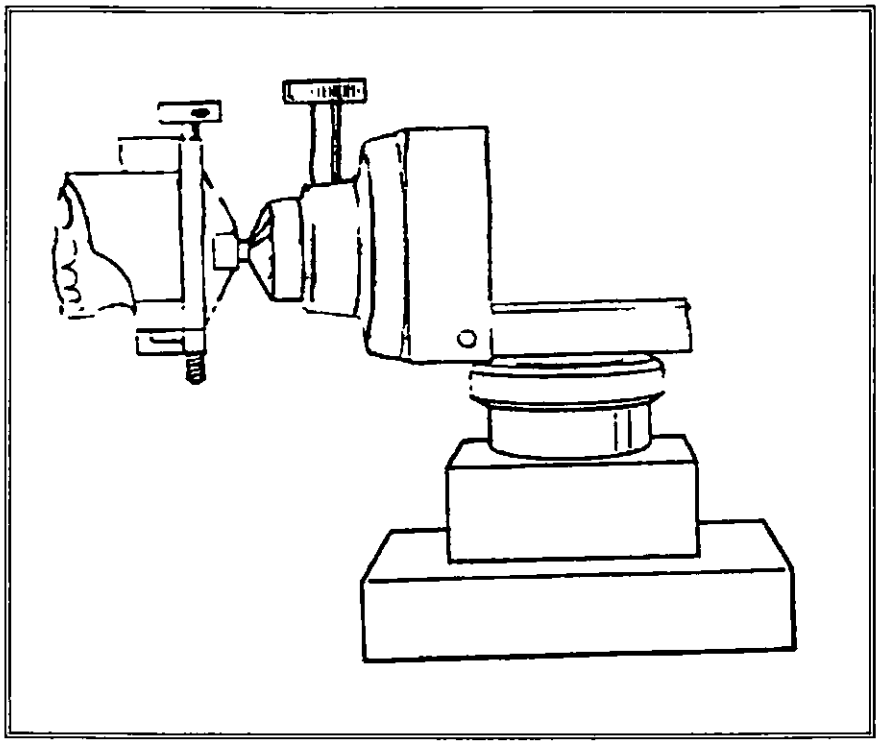


Figure 8. Cast holder can be turned 90° for measurement of bilateral maxillary first molar angulation.

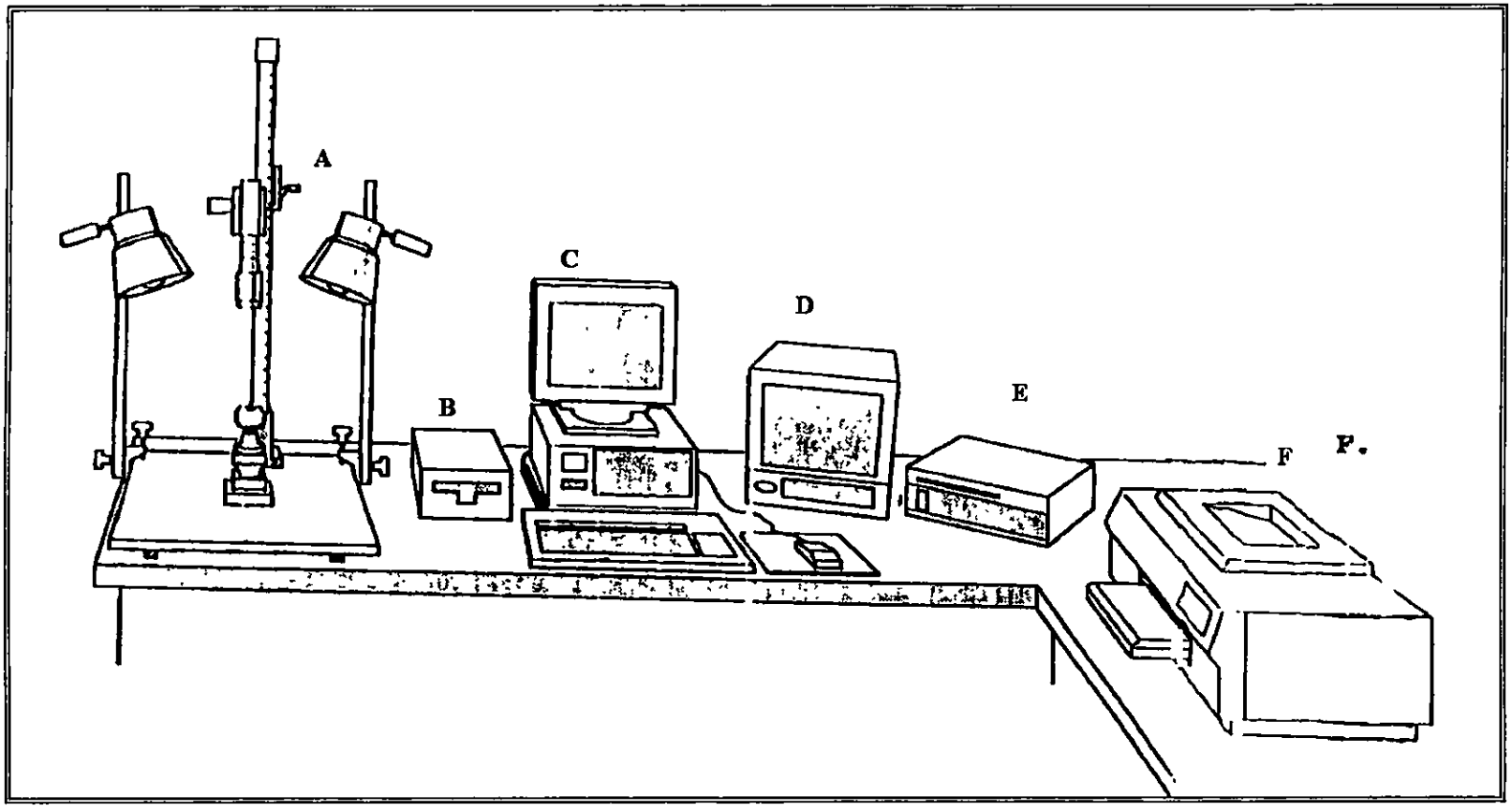


Figure 9. Schematic of Digital Imaging System:
A) Imaging component of Digital Imaging System containing of photo stand, digital camera, lighting and cast holder
B) Optical disc drive storage of images on optic discs
C) IBM 486 computer with color monitor
D) Video monitor
E) Video printer for hard copy of images
F) Laser printer

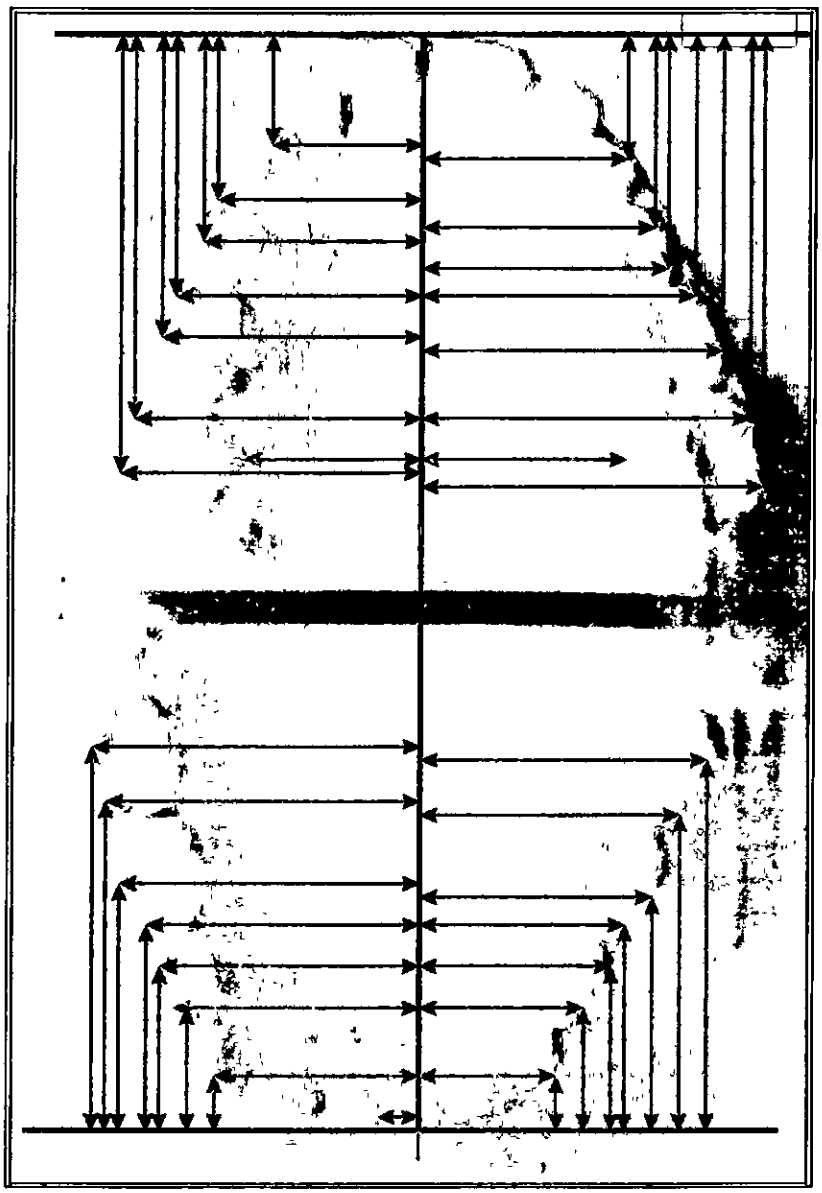


Figure 10. Diagram showing the linear measurements made from bilateral tooth landmarks to anterior and palatal/mandibular midlines on the maxillary and mandibular dental models.

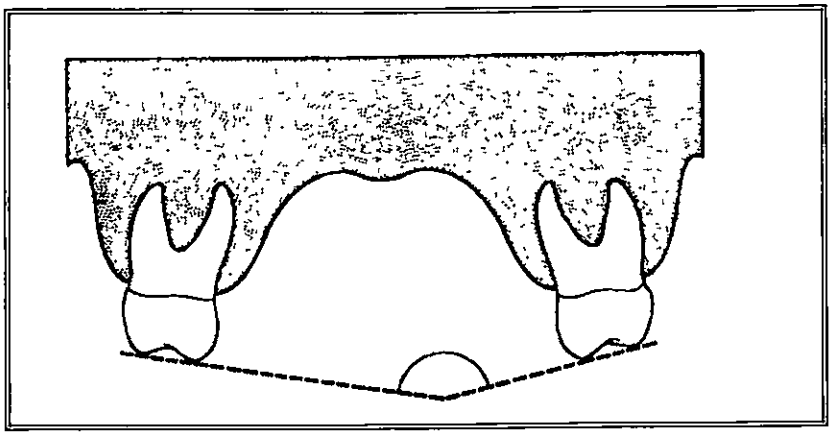


Figure 11. The molar angulation measurement was determined by the angle of intersecting lines drawn through the mesiobuccal and mesiopalatal cusp tips of the right and left first permanent molars.

Table 1.--Intraclass correlation coefficients for repeated measurements through digital imaging system

Measurement **Transverse :**	**r**	**Measurement**	**r**	**Measurement**	**r**
6 / distobuccal cusp - midline	0.981	6 / distobuccal cusp - midline	0.997	/ 6 distobuccal cusp - midline	0.998
6 / mesiobuccal cusp - midline	0.986	6 / mesiobuccal cusp - midline	0.997	/ 6 mesiobuccal cusp - midline	0.998
6 / mesiopalatal margin - midline	0.982	E / distobuccal cusp - midline	0.996	/ E distobuccal cusp - midline	0.998
E / distobuccal cusp - midline	0.982	E / mesiobuccal cusp - midline	0.997	/ E mesiobuccal cusp - midline	0.995
E / mesiobuccal cusp - midline	0.973	D / mesiobuccal cusp - midline	0.994	/ D mesiobuccal cusp - midline	0.992
D / mesiobuccal cusp - midline	0.987	C / cusp - midline	0.997	/ C cusp - midline	0.983
C / cusp - midline	0.983	/ 6 mesiopalatal margin - midline	0.978	/ 6 distobuccal cusp - midline	0.992
/ 6 mesiobuccal cusp - midline	0.992	/ E mesiobuccal cusp - midline	0.989	/ E distobuccal cusp - midline	0.991
/ D mesiobuccal cusp - midline	0.995	/ C cusp - midline	0.987		
A-P Measurement	**r**	**A-P Measurement**	**r**	**A-P Measurement**	**r**
6 / distobuccal cusp - reference line	0.996	/ 6 distobuccal cusp - reference line	0.994	/ 6 distobuccal cusp - reference line	0.997
6 / mesiobuccal cusp - reference line	0.961	/ 6 mesiobuccal cusp - reference line	0.994	/ 6 mesiobuccal cusp - reference line	0.997
E / distobuccal cusp - reference line	0.993	/ E distobuccal cusp - reference line	0.992	/ E distobuccal cusp - reference line	0.994
E / mesiobuccal cusp - reference line	0.995	/ E mesiobuccal cusp - reference line	0.989	/ E mesiobuccal cusp - reference line	0.993
D / mesiobuccal cusp - reference line	0.978	/ D mesiobuccal cusp - reference line	0.989	/ D mesiobuccal cusp - reference line	0.993
C / cusp - reference line	0.956	/ C cusp - reference line	0.960	/ C cusp - reference line	0.986
6/ distobuccal cusp - reference line	0.997	E/ distobuccal cusp - reference line	0.992	D/ mesiobuccal cusp - reference line	0.991
6/ mesiobuccal cusp - reference line	0.996	E/ mesiobuccal cusp - reference line	0.988	C/ cusp - reference line	0.986
Angular Measurement :		6/ - /6	0.960		

Table 2.--Description of subjects and the success rate of malocclusion correction

	T1 (before treatment)	T2 (After retention)
No. of subjects	36	36
Age / SD	9 yr 3 mo /12.7 mo	12 yr 8 mo /1 yr 4 mo
Gender	19 girls	19 girls
	17 boys	17 boys
malocclusion	17 class II subdivision on right side	16 class II subdivision on right side
	19 class II subdivision on left side	10 class II subdivision on left side
	(27% of cases achieved complete correction of molar subdivision)	
cross-bite side	22 crossbite on right side	3 cross-bite on right side
	14 crossbite on left side	1 edge-to-edge bite on right side
		2 edge-to-edge bite on left side
	(83% of cases achieved complete correction of unilateral posterior crossbite)	

Table 3.--Statistics of arch asymmetries and arch width at T1 and T2 (including paired t tests for measurements at T1 and at T1)

Variable	T1		T2		Paired Difference (T2 - T1)		P
Transverse difference	**Mean**	**SD**	**Mean**	**SD**	**Mean**	**SD**	
6	1.043	(0.898)	1 056	(0 915)	0.013	(1.139)	0 946
E or 5	1.049	(0.883)	1.097	(0.770)	0.048	(1 192)	0 817
D or 4	0.947	(0 735)	0.925	(0.768)	-0 020	(0 966)	0 903
C or 3	0.811	(0.834)	0.793	(0.793)	-0 018	(0 916)	0.933
6	3.678	(2.193)	2 014	(1 698)	**-1.664**	(2 163)	***0.000****
E or 5	3.197	(1.929)	1.850	(1.434)	**-1.347**	(1.872)	***0.000****
D or 4	3.156	(1.901)	1.829	(1.363)	**-1.327**	(1 752)	***0.000****
C or 3	3.408	(1.957)	1 799	(1 516)	**-1.608**	(1.723)	***0.000****
Maxillary dental midline	0.588	(0.675)	0 378	(0 693)	-0 210	(0.802)	0.125
Mandibular dental midline	1.729	(1.100)	1.053	(0.914)	**-0.676**	(1.083)	***0.001****
A-P difference							
6	1 088	(0 841)	1.442	(0.860)	0.353	(1.200)	0.086
E or 5	0.914	(0.962)	1.213	(0.30)	0.299	(1.205)	0 158
D or 4	0.859	(0 682)	1 246	(0.790)	0.387	(0.968)	0.022
C or 3	0.795	(0.702)	0.947	(0 711)	0 152	(0 833)	0.437
6	1.512	(1.079)	1.465	(1.124)	-0 046	(1.115)	0.805
E or 5	1.374	(0.912)	1 264	(1.051)	-0 110	(1 052)	0.553
D or 4	1.449	(1.136)	1.269	(1 149)	-0 181	(0.814)	0 198
C or 3	1.063	(0.742)	0.947	(0.711)	0.152	(0 833)	0.437
6-6 angle	166 8	(4 785)	166 1	(5.282)	0 7	(3 37)	0.906
Arch Width							
6-6	39.793	(2.055)	43 666	(2.549)	**3.874**	(1.963)	***0.000****
E-E or 5-5	40.104	(2.136)	43 681	(3 110)	**3.577**	(2.261)	***0.000****
D-D or 4-4	34.618	(2.124)	37 831	(3.188)	**3.213**	(2 039)	***0.000****
C-C or 3-3	29 006	(1 683)	32 410	(1 811)	**3.404**	(1.855)	***0.000****
6-6	45.094	(2.35)	45.288	(2.409)	0.194	(1.549)	0.457
E-E or 5-5	38.274	(2.248)	38 386	(2 484)	0.112	(1 929)	0.742
D-D or 4-4	32.336	(2.241)	32 838	(2.169)	0 502	(1.756)	0.100
C-C or 3-3	25.641	(1.659)	25 561	(1.569)	-0 080	(1.697)	0 802

*Statistically significant (Bonferroni correction was 0.05 alpha level divided by 27 variables=0.0018)

Table 4.--Pearson product-moment correlation between molar subdivision and arch sides differences at T1 and T2

Molar subdivision vs.	T1		T2	
Transverse difference	r	P	r	P
6	**-0.355***	0.033	**-0.397***	0.016
E or 5	-0.019	0.910	-0.208	0 239
D or 4	0.232	0 180	-0.005	0 975
C or 3	0 235	0 281	0.333	0.163
6	**0.725*****	0.0000	0.195	0.254
E or 5	**0.771*****	0 0000	**0.418***	0.014
D or 4	**0.852*****	0.0000	**0.487****	0.002
C or 3	**0.826*****	0 0000	**0.488****	0.006
Maxillary dental midline	-0.022	0 898	-0.100	0.561
Mandibular dental midline	**0.578*****	0.0002	**0.382***	0.022
A-P difference				
6	-0.064	0 711	**-0.382***	0 021
E or 5	-0.160	0.364	**-0.405***	0.018
D or 4	-0 127	0.466	**-0.365***	0.029
C or 3	-0.004	0 986	-0.038	0.878
6	**0.516*****	0 001	**0.652*****	0.0000
E or 5	**0.473****	0.004	**0.570*****	0.0004
D or 4	**0.386***	0.021	**0.593*****	0.0001
C or 3	**0.411***	0.026	**0.375***	0.045

The amount of subdivision and differences of sides were determined by right-hand side value minus left-hand side value.

Statistic significance: * P<0.05, ** P<0.01, *** P<0.001

Table 5.--Statistics of arch asymmetries and arch width at T1 and T2 (including paired t tests for measures at T1 and at T1)in Subgroup A (subdivision correction ≥1mm)

Variable	T1		T2		Paired Difference (T2 - T1)		P
Transverse difference	**Mean**	**SD**	**Mean**	**SD**	**Mean**	**SD**	
6	0 858	(0.582)	0.906	(0 632)	0 048	(0 929)	0.829
E or 5	0.814	(0.714)	1.013	(0 769)	0.199	(1.007)	0.414
D or 4	0.853	(0.744)	0 737	(0.732)	-0.116	(0.754)	0.535
C or 3	0.689	(0.723)	0.871	(0 886)	0.182	(0.862)	0 462
6	3.678	(2.095)	1 909	(1 953)	**-1.768**	(1.878)	***0.001****
E or 5	3 442	(1.894)	1 897	(1.668)	**-1.545**	(1.808)	***0.00****
D or 4	3.622	(1.845)	1.717	(1 514)	**-1.905**	(1 723)	***0.000****
C or 3	3.737	(2.038)	1.811	(1.806)	**-1.926**	(1.829)	***0.001****
Maxillary dental midline	0 653	(0.802)	0.209	(0 422)	-0 444	(0.718)	0.018
Mandibular dental midline	1.954	(1.188)	1 016	(1.148)	-0 938	(1 271)	0 006
A-P difference							
6	0.931	(0.583)	1.334	(0.880)	0.403	(1.166)	0.161
E or 5	0.744	(0.625)	1.123	(0 743)	0.379	(1.138)	0.176
D or 4	0.801	(0.509)	1 064	(0.692)	0.263	(1.024)	0.292
C or 3	0.903	(0.764)	0.852	(0.700)	-0.051	(0.914)	0.845
6	1.742	(1.154)	1.592	(1.339)	-0.150	(1.305)	0.633
E or 5	1.528	(1.037)	1.297	(1.112)	-0 231	(1 139)	0.415
D or 4	1.645	(1.102)	1.470	(1.344)	-0.175	(0.989)	0.463
C or 3	1.178	(0.853)	1.116	(1 001)	-0.061	(0 942)	0.798
6-6 angle	167.19	(3 520)	166 37	(4 19)	-0.818	(2 33)	0 155
Arch Width							
6-6	39 635	(1.799)	44 177	(2.199)	**4.542**	(1.909)	***0.000***
E-E or 5-5	40 257	(2.133)	44 191	(3.158)	**3.934**	(2.422)	***0.000***
D-D or 4-4	35 292	(1 699)	38.944	(2 668)	**3.652**	(2 351)	***0.000***
C-C or 3-3	28 822	(1 846)	32.932	(1.271)	**4.111**	(1.623)	***0.000***
6-6	45.262	(1.824)	45.980	(2.079)	0.718	(1.389)	0.43
E-E or 5-5	38.387	(2.511)	38.682	(2 095)	0 295	(2.032)	0.558
D-D or 4-4	32.745	(2.230)	33.177	(1 891)	0.432	(1.941)	0.359
C-C or 3-3	25.587	(1.752)	25.495	(1.405)	-0.092	(1.714)	0.833

*Statistically significant (Bonferroni correction was 0.05 alpha level divided by 27 variables=0.0018)

Table 6.--Statistics of arch asymmetries and arch width at T1 and T2 (including paired t tests for measures at T1 and at T1)in Subgroup B (subdivision correction <1mm).

Variable	T1		T2		Paired Difference (T2 - T1)		P
Transverse difference	**Mean**	**SD**	**Mean**	**SD**	**Mean**	**SD**	
6	1.227	(1 117)	1.205	(1 130)	0.022	(1.343)	0.945
E or 5	1.313	(0.999)	1 191	(0.785)	0.122	(1.385)	0.729
D or 4	1.054	(0.982)	0.996	(0.962)	0.059	(1.121)	0.797
C or 3	1.075	(1.062)	0.625	(0.575)	0 450	(0.954)	0.300
6	3.679	(2.348)	2.120	(1.448)	1.559	(2.466)	0.016
E or 5	2.937	(1.993)	1.801	(1.190)	1 136	(1.974)	0.036
D or 4	2.664	(1.887)	1.949	(1.218)	0 715	(1.611)	0.086
C or 3	3.002	(1.849)	1.785	(1.134)	1.218	(1.564)	0.016
Maxillary dental midline	0.522	(0.535)	0.546	(0.867)	-0.024	(0.831)	0.902
Mandibular dental midline	1.503	(0.986)	1.089	(0 634)	0 414	(0 808)	0.044
A-P difference							
6	1.246	(1.031)	1.549	(0.85)	-0 303	(1.264)	0.322
E or 5	1.105	(1.232)	1.314	(1.211)	-0 209	(1 308)	0.532
D or 4	0.916	(0.832)	0.427	(0.858)	-0.511	(0.922)	0.031
C or 3	0 56	(0 529)	1.152	(0.756)	-0.592	(0.395)	0.014
6	1.281	(0 976)	1.339	(0.880)	-0.058	(0.913)	0.793
E or 5	1.21	(0 756)	1.229	(1.017)	-0.019	(0.971)	0.937
D or 4	1.242	(1.166)	1.056	(0.891)	0.1865	(0.607)	0.223
C or 3	0.922	(0.58)	0.545	(0.458)	0.376	(0.604)	0.044
6-6 angle	163.74	(5.34)	164.96	(6.23)	1.22	(3.98)	0.211
Arch Width							
6-6	39.950	(2.325)	43.155	(2.827)	**3.205**	(1.829)	***0.000****
E-E or 5-5	39.932	(2.196)	43.107	(3.051)	**3.175**	(2.067)	***0.000****
D-D or 4-4	33.981	(2 329)	36.779	(3 349)	**2.798**	(1 656)	***0.000****
C-C or 3-3	29.405	(1.318)	31.278	(2.383)	1.873	(1.400)	0.022
6-6	44.926	(2.825)	44.596	(2 571)	-0.330	(1 558)	0.382
E-E or 5-5	38.153	(2.007)	38.070	(2.877)	-0.083	(1.860)	0.860
D-D or 4-4	31.904	(2.237)	32.479	(2.436)	0.576	(1.592)	0.155
C-C or 3-3	25.707	(1.604)	25.642	(1.808)	-0.065	(1.747)	0.895

*Statistically significant (Bonferroni correction was 0.05 alpha level divided by 27 variables=0.0018)

Table 7.--Unilateral crossbite treatment with fixed or removable appliance : Success rate

Author	Year	Dentition	Method	Success rate	Retention or Post-retention
Schroder	1984	primary	Coffin loop	27/32 (84%)	1 year
Purcell	1985	mixed		20/29 (70%)	
Hermanson*	1985	mixed	removable	16/17 (94%)	2.5 yrs post-R
			quad-helix	26/27 (96%)	11months post-R
Herold**	1989	mixed	Hyrax	12/19 (63%)	5.5 yrs post-R
			qual-helix	8/20 (40%)	4.5 yrs post-R
			removable	8/11 (73%)	4.7 yrs post-R
Kurol	1992	mixed	qual-helix	4/7 (57%)	2 yrs post-R
Brin	1996	mixed	removable	12/24 (50%)	6 month
Wu	1997	mixed	Hyrax®/ bonded RPE	30/36 (83%)	2.8 yrs post-R

* The criteria of success was not given in the article. All of the subjects were treated by general practitioners.

** In this study, children with both unilateral and bilateral crossbite were included.

Table 8.--Comparison of numbers of turns for active expansion and patients' age at T1 between subgroup A and subgroup B

Variables	Subgroup A Subdivision correction ≥ 1 mm		Subgroup B Subdivision correction < 1 mm		P
	Mean	SD	Mean	SD	
Numbers of turns for expansion	31.6	(6.2)	30.3	(4.3)	0.50^{NS}
Age at T1	107.8 (mo)	(13.4)	114.4 (mo)	(11.6)	0.12^{NS}

NS = non significant

REFERENCES

Adams CD, MC Meikle, KW Norwick, and DL Turpin. 1972. Dentofacial remodeling produced by intermaxillary forces in Macaca mulatta. Arch Oral Biol 17:1519-35.

Adkins MD, Nanda RS, and Currier GF. 1990. Arch perimeter changes on rapid palatal expansion. Am J Orthod Dentofacial Orthop 97:194-99.

Alavi, D, E BeGole and B Schneider. 1988. Facial and dental arch asymmetries in class II subdivision malocclusions. Am J Orthod Dentofacial Orthop 93:8-46.

Angle, EH. 1899. Classification of malocclusion. Dental Cosmos 41:248-64.

Bell, RA. 1982. A review of maxillary expansion in relation to rate of expansion and patient's age. Am J Orthod Dentofacial Orthop 81:2-7.

Ben-Bassat, Y, A Yaffe, I Brin, J Freeman, and Y Ehrlich. 1993. Functional and morphological-occlusal aspects in children treated for unilateral posterior crossbite. Eur J Orthod 15:57-63.

Bishara, SE, PS Burkey and JG Kharouf. 1994. Dental and facial asymmetries: A review. Angle Orthod 64:89-98.

Bjork, A and V Skieller. 1974. Growth in width of the maxilla by implant method. Scand J Plast Reconstr Surg 8:26-33.

Borell, G. 1982. Posterior crossbites– recognition, evaluation and treatment. N Y State Dent J 48:82-6.

Bresolin, D, PA Shapiro and GG Shapiro. 1983. Mouth breathing in allergic children: its relationship to dentofacial development. Am J Orthod Dentofacial Orthop 83:334-339.

Brin, I, Z Hirshfield, JL Shanfeld and Z Davidovheh. 1981. Rapid palatal expansion in cats: Effect of age on sutural cyclic nucleotides. Am J Orthod Dentofacial Orthop 79:162-75.

Brin I, Y Ben-Bassat, Y Blustein, J Ehrlich, N Hochman, Y Marmary. 1996. Skeletal and functional effects of treatment for unilateral posterior crossbite. Am J Orthod Dentofacial Orthop. 109:173-9.

Brust, EW. 1992. Arch dimensional changes concurrent with expansion in the mixed dentition. Masters Thesis, University of Michigan, Ann Arbor, MI.

Byrum, AG, Jr. 1971. Evaluation of anterior-posterior and vertical skeletal changes in rapid palatal expansion cases as studied by lateral cephalograms. Am J Orthod Dentofacial Orthop 60:419.

Chaconas, SJ, and AA Caputo. 1982. Observation of orthopedic force distribution produced by maxillary orthodontic appliance. Am J Orthod Dentofacial Orthop 82:492-501.

Cheney, EA. 1952. The influence of dentofacial asymmetries upon treatment procedures. Am J Orthod Dentofacial Orthop 38:934-45.

Cheney, EA. 1961. Dentofacial asymmetries and their clinical significance. Am J Orthod Dentofacial Orthop 47:814-29.

Cleall, JF, Bayne DI, Posen JM, and Subtelny,JD. 1965. Expansion of the midpalatal suture in the monkey. Angle Orthod 35:23-35.

Cook, JT. 1980. Asymmetry of the craniofacial skeleton. Br J Orthod 7:33-38.

de Araujo, TM, RS Wilhelm and MA Almeida. 1994. Skeletal and dental arch asymmetries in class II division I subdivision malocclusion. J Clin Pediatr Dent 18(3):181-185.

Ekstrom, C, CO Hennrickson and R Jensen. 1977. Mineralization in the midpalatal suture after orthodontic expansion. Am J Orthod Dentofacial Orthop 71:449-55.

Firacelli, JP. 1978. A brief review of maxillary expansion. J Pedod 3:29-35.

Fischer, B. 1954. Asymmetries of the dentofacial complex. Angle Orthod 24:179-92.

Garland, HH, CW Houghton and CJ Burstone. 1981. Unilateral race bows: A theoretical and laboratory analysis. Am J Orthod Dentofacial Orthop 79:229-249.

Graber, T and B Swain. 1975. Current Orthodontic Concepts and Techniques. Philadelphia. WB Saunders.

Gryson, IA. 1977. Changes in mandibular interdental distance concurrent with rapid maxillary expansion. Angle Orthod 47:186-92.

Haack, DC and S Weinstein. 1958. The mechanics of centric and eccentric cervical traction. Am J Odontia 44:346-357.

Haas, AJ. 1961. Rapid expansion of the maxillary dental arch and nasal cavity by opening the mid-palatal suture. Angle Orthod 31:73-90.

Haas, AJ. 1965. The treatment of maxillary deficiency by opening the mid-palatal suture. Angle Orthod 35:200-17.

Haas, AJ. 1970. Just the beginning of dentofacial orthopedics. Am J Orthod Dentofacial Orthop 57:219-55.

Haas, AJ. 1980. Long-term post-treatment evaluation of rapid palatal expansion. Angle Orthod 50:189-217.

Heikinheimo, K, K Salmi and S Myllarniemi. 1987. Long term evaluation of orthodontic diagnosis made at the ages of 7 and 10 years. Eur J Orthod 19:151-159.

Hellman, M. 1939. Some facial features and their orthodontic implications. Am J Orthod Oral Surg 25:927-51.

Herberger T. 1987. Rapid palatal expansion: Long term stability and periodontal implications. Masters Thesis, University of Pennsylvania, Philadelphia, PA.

Hermanson, H, J Kurol and A Ronnerman. 1985. Treatment of unilateral posterior crossbite with quad-helix and removable plates: A retrospective study. Eur J Orthod 7:97-102.

Herold,JS. 1989. Maxillary expansion: A retrospective study of three methods of expansion and their long-term sequelae. Br J Orthod 16:195-200.

Hesse, KL, J Artun, D Joondeph and DB Kennedy. 1997. Changes in condylar position and occlusion associated with maxillary expansion for correction of functional unilateral posterior crossbite. Am J Orthod Dentofacial Orthop 111:410-8.

Hicks, EP. 1978. Slow maxillary expansion: A clinical study of the skeletal vs. dental response to low magnitude farce. Am J Orthod Dentofacial Orthop 73:121-41.

Hinton RJ and JA McNamara. 1984. Temporal bone adaptations in response to protrusive function in juvenile and young adult rhesus monkeys (Macaca mulatta). Eur J Orthod 6:155-74.

Ingerval, B and B Thilander. 1975. Activity of temporal and masseter muscles in children with a lateral forced bite. Angle Orthod 45:249-58.

Jerrold, L and LJ Lowenstein. 1990. The midline : Diagnosis and treatment. Am J Orthod Dentofacial Orthop 97:453-62.

Kelly, J and Harvey, C. 1977. An Assessment of the Occlusion of the Teeth of Youths 12-17 Years - National Center for Health Statistics, U.S. Public Health Service, Washington DC. United States. DHEW No(HRA) 77-1644.

Kerosuo, H. 1990. Occlusion in the primary and early mixed dentition in a group of Tanzanian and Finnish children. ASDC J Dent Child 7:293-298.

Kisling, E. 1981. Occlusal interference in the primary dentition. ASDC J Dent Child 48:181-191.

Korkhaus, G. 1930. A new orthodontic synrmetrograph. Int J Orthod Oral Surg Rad 16:665-668.

Krebs AA. 1958. Expansion of the midpalatal suture studies by means of metallic implants. Trans Eur Orthod Soc 34:163-171.

Krebs AA. 1964. Midpalatal suture studies by implant method over a seven year period. Trans Eur Orthod Soc 40:131-142.

Kurol, J. and L Berglund. 1992. Longitudinal study and cost benefit analysis of the effect of early treatment of posterior cross-bites in the primary dentition. Eur J Orthod 14:173-179.

Kutin, G. and RR Hawes. 1969. Posterior cross-bites in the deciduous and mixed dentition. Am J Orthod Dentofacial Orthop 56:491-504.

Larsson, E. 1986. The effect of dummy-sucking on the occlusion: A review. Eur J Orthod 8:127-30.

Latham, RA. 1971. The development, structure and growth pattern of the human mid-palatal suture. J Anat 108:31-41.

Leighton BC and WH Feasby. 1988. Factors influencing the development of molar occlusion: A longitudinal study. Br J Orthod 15:99-103.

Letzer, G and J Kronman. 1967. A posterior-anterior cephalometric evaluation of craniofacial asymmetry. Angle Orthod 37:205-11.

Lewis, D. 1976. The deviated midline. Am J Orthod Dentofacial Orthop 70:601-16.

Linder-Aronson, S. 1970. Adenoids: their effect on mode of breathing and nasal airflow and their relationship to characteristics of the facial skeleton and the dentition. Acta Otolaryngol Suppl 265:1-132.

Lundstrom, A. 1961. Some asymmetries of the dental arches, jaws, and skull, and their etiological significance. Am J Orthod Dentofacial Orthop 7:81-106.

Mayers, CA. 1977. Asymmetry of the maxillary and mandibular dentition with respect to the palatine raphe. Masters Thesis. Case Western Reserve University, Cleveland, OH.

McNamara, JA. 1975. Functional adaptations in the temporomandibular joint. Dent Clin North Am 19:457-71.

McNamara, JA and DS Carlson. 1979. Quantitative analysis of temporomandibular joint adaptations to protrusive function. Am J Orthod Dentofacial Orthop 76:593-611.

McNamara, JA, Jr and WL Brudon. 1993. Orthodontic and Orthopedic Treatment in the Mixed Dentition. Ann Arbor, MI. Needham Press. 131-145.

Melsen, B. 1972. A histologic study of the influence of sutural morphology and skeletal maturation on rapid palatal expansion in children. Trans Eur Orthod Soc 499-507.

Mew, J. 1983. Relapse following maxillary expansion. A study of 25 consecutive cases. Am J Orthod Dentofacial Orthop 33:56-61.

Mongini, F. 1984. Abnormalities in condylar and occlusal positions. In: Abnormal Jaw Mechanics. Chicago, Quentessence. 23-43.

Mongini, F and W Schmid. 1987. Treatment of mandibular asymmetries during growth—A longitudinal study. Eur J Orthod 9:51-67.

Moyers, RE. 1966. Handbook of Orthodontics. 2nd ed. Chicago. Year Book Medical Publishers, Inc. 332-341.

Mulick, J. 1965. An investigation of craniofacial asymmetry using the serial twin-study method. Am J Orthod Dentofacial Orthop 52:112-129.

Myers, D, J Barenie, R Bell and E Williamson. 1980. Condyle position in children with functional posterior crossbite: Before and after crossbite correction. Pediatr Dent 2:190-4.

Nahoun, HI, J Fiasconaro and N Disalvo. 1964. The verticosubmental reongenogram in cephalometric. J Am Dent Assoc 69:13-150.

O'Byrn, BL, C Sadowsky, B Schneider and EA BeGole. 1995. An evaluation of mandibular asymmetry in adults with unilateral posterior crossbite. Am J Orthod Dentofacial Orthop 107:394-400.

Øgaard, B, E Larsson and R Lindsten. 1994. Effect of sucking habits, cohort, sex, intercanine arch widths, and breast or bottle feeding on posterior crossbite in Norwegian and Swedish 3-year-old children. Am J Orthod Dentofacial Orthop 106:161-166.

Oulis, CJ, GP Vadiakas, J Ekonomides and J Dratsa. 1994. The effect of hypertrophic adenoids and tonsils on the development of posterior crossbite and oral habits. J Clin Pediatr Dent 18:197-201.

Pearson, K and TL Woo. 1935. Further investigation of the morphmetric characters of the individual bones of the human skull. Biometrika 27:424-465.

Persson, M and B Thilander. 1977. Palatal suture closure in man from 15-35 years of age. Am J Orthod Dentofacial Orthop 72:42-52.

Pirttiniemi, P, T Kantomaa and P Lahtela. 1990. Relationship between craniofacial and condyle path asymmetry in unilateral posterior crossbite patients. Eur J Orthod 12:408-13.

Pirttiniemi, P, A Raustia, T Kantomaa and J Pyhtinen. 1991. Relationships of bicondylar position to occlusal asymmetry. Eur J Orthod 13:441-5.

Rose, JM, C Sadowsky and EA BeGole. 1994. Mandibular skeletal and dental asymmetry in Class II subdivision malocclusions. Am J Orthod Dentofacial Orthop 105:489-95.

Sarver, DM and MW Johnston. 1989. Skeletal changes in vertical and anterior displacement of the maxilla with bonded rapid expansion appliances. Am J Orthod Dentofacial Orthop 95:462-466.

Schroder, U and I Schroder. 1984. Early treatmwent of unilateral posterior crossbites in children with bilateral contracted maxilla. Eur J Orthod 6:65-69.

Shroff, B, SJ Lindauer, CI Burstone and JB Leiss. 1995. Segmented approach to simultaneous intrusion and space closure: Biomechanics of the three piece base arch appliance. Am J Orthod Dentofacial Orthop 107:136-143

Shroff, B, SJ Lindauer and CJ Burstone. 1997. Class II subdivision treatment with tip-back moment. Eur J Orthod 19:93-101.

Sinclair PM and Little RM. 1983. Maturation of untreated normal occlusions. Am J Orthod Dentofacial Orthop 83:114-23.

Spillane LM. 1990. Arch dimensional changes in patients treated with maxillary expansion during the mixed dentition. Masters Thesis, University of Michigan, Ann Arbor, MI.

Thilander, B, S Wahlund and B Lennartson. 1984. The effect of early interceptive treatment in children with posterior cross-bite. Eur J Orthod 6:25-34.

Timms, DJ. 1968. An occlusal analysis of lateral maxillary expansion with midpalatal suture opening. Dent Pract Trans 18:435-440.

Timms, DJ. 1980. A study of basal movement with rapid maxillary expansion. Am J Orthod Dentofacial Orthop 77:500-7.

Truque, MG. 1956. A study of the asymmetries of the maxillary dental arch in relation to raphe palatinus and the mediastinal plane of the upper facial skeleton. Masters Thesis. University of Michigan. Ann Arbor, MI. ii + 24 p.

Tweed, C. 1966. Clinical Orthodontics. St Louis. C.V. Mosby Company.

US Department of Health, Education, and Welfare. 1974. An Assessment of the Occlusion of the Teeth of Youths 6-11 Years - National Center for Health Statistics. Rockville, Maryland, United States. DHEW No(HRA) 74-1344.

Vadiakas GP and MW Roberts. 1991. Primary posterior crossbite: Diagnosis and treatment. J Clin Pediatr Dent 16:1-4.

Vazquez, F, J Grostic, A Fonder and K DeGoer. 1982. Eccentricity of the skull correlation with dental malocclusion. Angle Orthod 52:144-58.

Wertz, RA. 1970. Skeletal and dental changes accompanying rapid palatal suture opening. Am J Orthod Dentofacial Orthop 58:41-66.

Wertz, RA. 1975. Diagnosis and treatment planning of unilateral Class II malocclusions. Angle Orthod 45:85-94.

White, JC. 1982. A study of craniofacial asymmetry. Unpublished Masters Thesis. Case Western Reserve University, Cleveland, OH.

White, JC. 1982. A study of craniofacial asymmetry. Unpublished Masters Thesis. Case Western Reserve University, Cleveland, OH.

Williamson, EH and MD Simmons. 1979. Mandibular asymmetry and its relation to pain dysfunction. Am J Orthod Dentofacial Orthop 76:612-7.

Woo, TL. 1931. On the asymmetry of the human skull. Biometrika 22:324-352.

Woo, TL. 1937. A biometric study of the human molar bone. Biometrika 29:113-123.

Woodside, DG, A Metaxas, and G Altuna. 1987. The influence of functional appliance therapy on glenoid fossa remodeling. Am J Orthod Dentofacial Orthop 92:181-198.

Wood, A. 1962. Anterior and posterior cross-bites. J Dent Children 29:280-86.

Zhu, JF, R Crevoisier, DL King and CM Mills. 1996. Posterior crossbite in children. Compendium 17 (11): 1051-1067.

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