

Effects of Rapid Maxillary Expansion on Upper Airway; A 3 Dimensional Cephalometric Analysis

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EFFECTS OF RAPID MAXILLARY EXPANSION ON UPPER AIRWAY;
A 3 DIMENSIONAL CEPHALOMETRIC ANALYSIS

by

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ABSTRACT

EFFECTS OF RAPID MAXILLARY EXPANSION ON UPPER AIRWAY;
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Yoon H. Chang D.D.S.

Marquette University, 2011

The purpose of this study was to use cone-beam computed tomography (CBCT) to assess changes in the volume and cross sectional areas of the upper airway in children with maxillary constriction treated by rapid maxillary expansion (RME).

The study group consisted of 5 males and 9 females with mean age of 12.93 years with posterior cross bite and constricted maxilla who were treated with hyrax expander. Pre and post RME CBCT scans were analyzed with 3D Dolphin 11.0 software to measure the retropalatal (RP) and retroglossal (RG) airway changes. The transverse width changes were evaluated from the maxillary inter 1st molar and inter 1st pre molar mid lingual alveolar plate points. Pre and post RME scans were compared with paired *t* test and Pearson correlation test was done on data reaching significance.

Only the cross sectional airway measured at posterior nasal spine (PNS) to Basion (Ba) level showed a statistically significant increase ($P=0.0004$). The inter-molar and inter-premolar mid lingual alveolar plate distances increased equally by 4.76 mm and were statistically significant ($P < 0.0001$). The percentage increase at the 1st premolar level was significantly larger than at the 1st molar level ($P= 0.035$). PNS-Ba cross sectional area increase was highly correlated with the maxillary 1st molar mid lingual inter alveolar plate width ($p=0.0013$).

In conclusion, RME produced a numerically equal amount of expansion between the mid inter-lingual plates of maxillary 1st molars and 1st premolars. However, when the percentage change was calculated, a greater opening was observed at the 1st premolar level suggesting a triangular shape of opening. In regard to the upper airway, a moderate increase of the cross sectional area adjacent to the hard palate was found and this increase was deemed to be highly dependent on the expansion between the maxillary 1st molars. Further studies with a larger sample size and incorporating breathing evaluations are needed to estimate the real impact of the RME on the airway.

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LIST OF ABBREVIATIONS

ANS: anterior nasal spine

Ba: basion

CBCT: cone beam computed tomography

CT: computed tomography

EP plane: plane parallel to the P plane contacting the most superior aspect of the epiglottis

MCA: minimal cross sectional airway

N: nasion

PNS: posterior nasal spine

P plane: plane formed by connecting PNS and Ba and oriented to be always parallel to the floor

RG: retroglossal

RP: retropalatal

RME: rapid maxillary expansion

SME: slow maxillary expansion

SP plane: plane parallel to the P plane contacting the most inferior aspect of the soft palate

CHAPTER I
INTRODUCTION

Rapid maxillary expansion (RME) is one of the most frequently used techniques for the correction of maxillary width deficiency or posterior cross bite and to expand the arch perimeter to alleviate dental crowding (Baccetti et al. 2001). The expansion is accomplished by a heavy force originating from a RME appliance resulting in maximum orthopedic expansion with minimum orthodontic tooth movement (Garrett et al.). Evaluation of the airway is considered an important diagnostic test in Orthodontics in part due to the potential impact of high resistance airways contributing to an abnormal growth of the naso-maxillary complex and the potential to increase the vertical facial dimension in young children (Subtelny, Tso et al.). The advent of cone beam computerized tomography (CBCT) has made 3- dimensional depiction of the craniofacial structures readily accessible using significantly lower radiation than conventional medical computed tomography (CT) images (Tso et al.).

Traditionally, respiratory air spaces have been evaluated by the use of cephalometric radiographs (Tso et al.). However, the complexity of the 3D anatomy and the superimposition of bilateral structures limits an accurate evaluation of important anatomical features. Among the existing 3D imaging techniques, CBCT has become the ideal method to evaluate airway patency due to its significantly lower radiation than medical CT, higher contrast between the hard and soft tissues, greater spatial resolution than medical CT, lower cost and easier access and availability to dentists (Tso et al. Lundlow et al.).

Previous airway study using CBCT images, taken from a supine position, demonstrated that the retropalatal (RP) airway was significantly smaller in

children with constricted maxilla compared to controls. Nevertheless, RME therapy in these children caused only molar width expansion with no significant differences in the absolute values and percentages of the oropharyngeal volumes (Zhao et al.). In a different study, RME has been found to increase the RP volume, nasal cavity dimension, improve breathing, and has been also theorized to produce a positive change in the upper airway dimensions (Enoki et al.). Whether or not the RME therapy produces changes in the upper airway dimension is still unclear.

Our research project aims to describe the upper airway changes before and after RME correlated to transverse width changes in children between 9 and 16 years of age undergoing comprehensive orthodontic treatment. . The null hypothesis is that RME produces no changes in the mean volume and minimal cross sectional airway of the upper airway within the same individual between pre- and post- RME.

CHAPTER II
REVIEW OF LITERATURE

History of RME

Rapid maxillary expansion (RME) is a commonly used non-surgical maxillary expansion technique (Ekstrom et al 1976) for the correction of maxillary width deficiency and posterior cross bite by increasing the width of the dental arch (Hass 1970) and of the nasal cavity (Enoki et al 2006). Emerson C. Angell described the first clinical use of RME in 1860 reporting a case of a fourteen year old girl in whom a jackscrew across the roof of the mouth with its ends bearing against the first and second bicuspids of one side to the other corrected the maxillary transverse deficiency (Angell 1860).

Despite initial arguments against this novel technique based on the possibility of inducing serious disturbance in the surrounding hard and soft tissue, RME was attempted with varying degree of success by several practitioners during the late 1890's through the late 1920's. The earliest report of RME to specifically enhance breathing dates back to 1903 when G. Brown observed that the nasal width increased after separating the maxilla in young individuals. A few years later, a RME study evaluating the intranasal changes revealed that the distance between the lateral walls of the nasal cavity below the inferior concha increased and the subjective intranasal respiration improved (Wright 1912). During the 1930's and 1940's the use of maxillary expansion was almost completely abandoned in the United States due to the widespread acceptance of the functional theory advocating bone growth in presence of vigorous function and proper dental relations.

Over a century after the first RME publication, Hass re-introduced the concept of RME based on a successful pilot animal study followed by a human case series consisting of 45 subjects with maxillary or nasal insufficiency. The expansion was accomplished by activating the Jackscrew 0.5mm per day (0.25 mm in the morning and 0.25 mm in the evening) for 21 consecutive days followed by a retention phase of 3 months. Pre, post and follow up records (frontal, lateral cephalometric X rays, dental casts and patient's subjective opinion) demonstrated the existence of a significant expansion between the mid palatal sutures, between lateral walls of the nasal cavity and the maxillary intermolar distance along with unanimous subjective improvement in nasal respiration. In addition, a triangular pattern of maxillary suture opening with the base towards the palate and the apex towards the nose, an initial forward and downward movement of the maxilla, mesial drift of the maxillary incisors after initial diastema formation, and uprighting of the mandibular teeth were also reported.

Hass postulated that the initial gross reaction of the maxillary expansion was a lateral bending of the alveolar processes followed by a gradual opening of the mid palatal suture and that the zygomatic buttresses caused the separation of the maxillary halves to be wedged shaped with the apex towards the nasal cavity (Hass 1961, 1970). Interestingly, fifty years after Hass documented his findings, very little additional information has been added to this topic other than confirming what has already been reported.

Skeletal Response and Stability

RME can be achieved through the use of tooth-tissue borne or tooth-borne appliances that are fixed to the teeth either by bands or chemical bonding which are capable of producing heavy forces in the range of 15 to 50 Newton (Lagravere et al. 2005). Originally, RME was thought to provide mostly orthopedic movement of the maxillary bones with minimal orthodontic tooth movement (OTM). However, OTM continues during the retention phase until bone stability is reached, by 4 months true orthopedic maxillary transverse width gain accounts for about half the gained expansion while the remaining comes from the lateral dental movements on their supporting bone (Proffit 2007).

In a cone beam computed tomography (CBCT) study evaluating 3 months post RME skeletal response in 30 consecutive orthodontic patients, the maxillary 1st inter-premolar (P1) and 1st inter-molar (M1) width measured from each buccal plates increased 6 mm and 6.6 mm respectively. However, when the expansion was further analyzed, the sutural orthopedic expansion accounted for only 55% and 38 % at P1 and M1 respectively of the total expansion. The remaining expansion was derived from a significant dental tipping accounting for 39% and 49% at P1 and M1 respectively and a minor contribution from the alveolar plate expansion added 6% and 13% at P1 and M1 respectively. The combined data clarified how the maxillary expansion actually occurs and also demonstrated that a decreasing orthopedic skeletal effect and increasing orthodontic tipping and alveolar bending effect exist from anterior to posterior (Garrett et al. 2008).

Slow maxillary expansion on the other hand, consists of expanding the palate at a much lower rate using smaller expanding forces (0.5 mm per week) equivalent to the maximum rate at which the tissues of the midpalatal suture can adapt (Proffit, 2007). A study analyzing the long term effects of maxillary expansion from initial, post treatment and post retention dental casts measuring the points intersecting the lingual groove and the gingival margin of the maxillary first molars revealed that both: slow maxillary expansion (SME) and RME techniques were efficient in correcting the transverse discrepancy. The arch width for the SME group increased by 3.4 mm with 0.29 mm relapse while the RME group increased by 5.95mm and relapsed 0.46 mm at 10 year post retention follow up. Unfortunately, a direct comparison of maxillary expansion efficiency could not be reached due to the decision of using SPE or RPE based on the severity of the transverse discrepancy preferring SPE when smaller transverse discrepancies were present (Filho et al. 2008).

Side Effects

Secondary effects of RME relate to the heavy forces produced by the RME appliance which could produce bite opening, microtrauma of the midpalatal suture and temporo-mandibular joint structure and root resorption among others (Lagravere et al. 2005). Periodontal involvement is the most commonly cited side effect of RME due to the possibility of damaging the buccal cortical plates and developing gingival recessions when high forces are directed towards the

banded teeth. A retrospective study analyzing the periodontal effect of RME in 17 growing patients demonstrated that immediately after the expansion the first maxillary molar buccal plate thickness is reduced by 0.5 mm. However, at 6 months post expansion, only the lingual bone plate thickness of both first molars was significantly increased with no differences in the ratio between intermolar widths at the apex and crown levels (Ballanti et al. 2009).

In a CBCT study evaluating the buccal alveolar bone changes 3 months after the end RME activation with Hyrax appliance, it was found that the buccal bone thickness decreased 1.1 mm and 1.2 mm for the 1st premolars and 1st molars respectively while the buccal marginal bone level decreased by 4.5 mm and 2.9 mm respectively. This study suggested that the buccal movement of teeth may potentiate the probability of buccal bone dehiscence at the maxillary 1st premolar due to the increased buccal marginal bone loss associated with apical narrowing at this level (Rungcharassaeng et al. 2007). Although periodontal consequences may be present after RME, available literature demonstrate that buccal bone thickness returns to normal level and no periodontal concern should be raised if the patient had an initially normal buccal bone thickness (Timms and Moss).

Treatment Timing

Like all craniofacial sutures, the mid palatal suture becomes more tortuous and interdigitated with increasing age. In children up to ten years of age, almost

any expansion device will tend to separate the mid palatal suture. However, by adolescence a relatively heavy force from a rigid jackscrew is needed to separate the interdigitated suture (Proffit 2007).

In this context, Baccetti et al. evaluated patients with different stages of cervical vertebrae maturation index and found that the early treated individuals who had not reached the pubertal growth spurt at the onset of RME showed on average 3mm of expansion of the mid-palatal suture while the late treated ones averaged only 0.9mm. His finding suggested that an effective long-term change at the skeletal level occurs when the patients were treated prior to pubertal peak growth and higher dental effect is present in individuals treated after pubertal growth spurt (Baccetti T. et al. 2001).

Distant Skeletal Effects

Finite element analysis (FEA) is defined as a computer simulation method performed by dividing the interested region into discrete elements interconnected at nodes with assigned material property that represents the physical property of the model. A FEA study evaluating the effects of the maxillary expansion on the neighboring bones demonstrated that in the closed suture model (adult type suture) significant stress areas were present at the buccal alveolar processes, distal aspect of the maxilla, inferior aspect of the zygomatic arches and pterygomaxillary fissure region (Lee et al. 2009). Thus, areas surrounding the zygomatic processes were suggested to provide a buttressing effect against the

forces of expansion. In the patent midpalatal suture model (growing child) however, the pterygomaxillary fissure demonstrated to be the highest stress point. This finding confirmed the impact of maxillary expansion in facilitating the treatment effects of a class III facemask therapy in growing individual (Lee et al. 2009). In the same patent suture model, tension stress was also present at the upper portion of the nasal cavity suggesting that the palatal expansion with heavy forces in young children may create undesirable changes in the nose (Lee et al. 2009, Proffit 2007). For both groups, the lateral displacement of the maxillary halves appeared nonparallel, with a slightly wider opening towards the anterior and the separation of the maxilla occurring as if a hinge was positioned superiorly at the base of the nose (Lee et al. 2009).

Clinical studies evaluating the effects of orthopedic expansion via RME postulated that not only bodily separation of the midpalatal suture exists, but also buccal rotational force on the maxillary alveolar shelves and changes to the surrounding frontomaxillary, zygomaticomaxillary, zygomaticotemporal and pterygopalatine sutures (Garrett et al. 2008).

Skeletal Dimension and Airway

The typical features that are characteristic of persons who have difficulty breathing through their nose is exemplified by the long face syndrome. The prototype of this condition includes an increase in lower facial height, lip apart posture, narrow alar base, and frequently self-reported "mouthbreathing".

Intraorally, a narrow maxillary arch with a high palatal vault and a posterior crossbite with a class II malocclusion are usually found (Vig 1998).

In a longitudinal study done by Subtelny it was found that a persisting hypertrophic adenoid tissue is seldom found in children with allergic reaction or reaction towards an infectious agent. This condition was capable of approximating the adenoid tissue with the superior surface of the soft palate to create a blockage of the nasopharyngeal cavity. Subtelny suggested that the obturation of the nasopharynx may induce a mouth breathing pattern that in the long term may increase the vertical facial dimension possibly leading towards a class II division 1 type of malocclusion (Subtelny 1954).

In a recent systematic review, the lack of nasal airway patency was found to be associated with oral breathing and considered to be a contributing factor towards an abnormal development of skeletal structures through the potential increase of vertical dimension of the face in a growing individual (Major 2006).

Maxillary Constriction and Apnea

Maxillary constriction has also been postulated to play a role in the pathophysiology of obstructive sleep apnea (OSA). Despite the reference of multiple contributing factors for the development of OSA including retrognathic mandible, shorter AP face length, reduced distance from the posterior nasal spine (PNS) to posterior pharyngeal wall, lower positioned hyoid bone, larger soft palate, smaller pharynx, larger tongue size, obesity and combination thereof

(Johal 2007), a constricted maxilla has also been associated with narrowing the upper airway dimension and increasing the risk for OSA by inducing a low tongue posture (Subtelny 1954). Sleep apnea is defined as a decrease in respiration yielding hypoxia and hypercapnia during sleep, caused by either neurologic origin or actual physical blockage of the airway also known as OSA. Subjects with centrally driven apneic event present no effort to overcome the apnea, whereas the opposite is true for the OSA sufferers.

The American Academy of Sleep Medicine defines OSA as episodes of breathing cessation or absence of respiratory airflow for over 10 seconds despite respiratory effort. Epidemiology reports indicate that this is a highly prevalent respiratory sleep disorder affecting 4% of men and 2% of women (Haskell 2009). The most serious consequences of OSA are the cardiovascular diseases such as hypertension, tachycardia, atherosclerosis, increased risk for cerebrovascular accidents, coronary artery disease and more (Madani 2007). The pathogenesis of these effects is still being studied but it is generally accepted that the intermittent hypoxia and hypercapnia episodes triggers homeostatic compensations in the body, leading to cardiovascular diseases over time (Sharabi 2004). It is believed that the sleep induced relaxation of the muscles attached to the soft tissues of the pharynx is aggravated by gravity and the retropositioning of the tongue mass during supine position narrowing the airway lumen (McCrillis 2009).

Treatment of OSA consists in preventing the collapse of the lumen of the pharynx during sleep. At present, several treatment options based on the

severity of the apneic events are rendered, including continuous positive airway pressure (CPAP) therapy, surgical treatments and mandibular repositioning devices therapy. Oral appliances have been reported to improve breathing by decreasing nasal resistance and reducing the apnea hypopnea index (AHI). For breathing to take place, patency of the pharynx or upper airway is vital. With the exception of the two ends of the airway, the nares and the small intrapulmonary airways, the pharynx is the only collapsible segment of the respiratory tract with the potential to be altered by diverse treatment effects (Haskell 2009).

Upper Airway

According to Lenza et al. the upper airway can be divided into smaller segments to better understand the physiologic changes as well as the treatment effects (Lenza et al. 2010). The various portions of the upper airway, superiorly to inferiorly include (Fig. 1):

Nasopharynx: The upper most portion of the airway, mainly the nose. It begins with the nares extending back to the hard palate at the superior portion of the soft palate. This includes the nasal septum and the nasal turbinates.

Retropalatal airway (velopharynx): This area extends from the hard palate to the inferior tip of the soft palate, including the uvula and the uppermost segment of the posterior pharyngeal wall. Major muscles include the tensor palatini and levator palatini, which elevate the soft palate, and the musculus uvulae providing elevation of the uvula.

Retroglossal airway (oropharynx): This area includes the oral cavity, beginning with the back portion of the mouth and extending rearward to the base of the tongue or tip of the epiglottis. Tonsils and tongue muscles are located in this segment.

Hypopharynx: The area extends from the tip of the epiglottis to the lowest portion of the airway at the larynx.

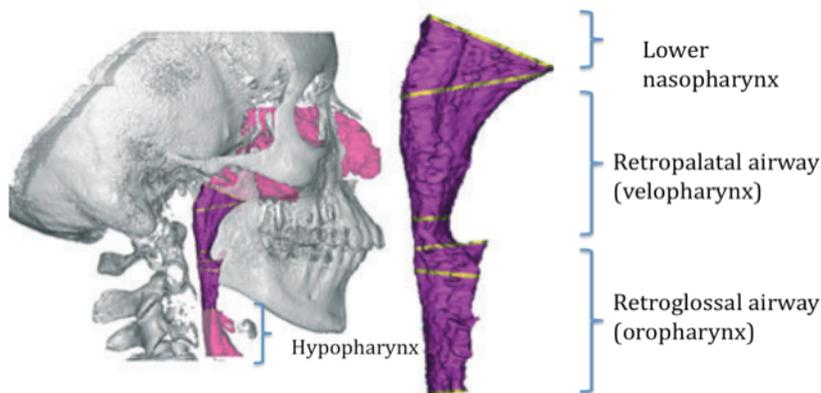


Fig. 1. Schematic diagram of upper airway

Airway Studies and CBCT

Evaluation of the upper airway has become an important diagnostic test in several subspecialties of dentistry (Tso, 2009), in part due to the controversial (Warren et al, 1991) but potential impact of high resistance airways contributing towards an abnormal growth of the naso-maxillary complex, increasing the

vertical facial dimension in young patients (Linder-Aronson, 1970) and the potential role of constricted airways in the pathophysiology of obstructive sleep apnea (OSA) (Haskell J. et al. 2009). Studies on the changes of upper airway dimensions have consisted of analyzing the post-treatment effects of RME with dental casts (Oliveira De Felipe et al. 2008), human skull models (Gautam et al. 2007), 2-dimensional cephalometric radiographs (Haas 1970), 3-dimensional (3D) imaging techniques including magnetic resonance images, CT, CBCT (Garrett 2007), acoustic rhinometry and computed rhinomanometry (Enoki et al. 2009).

However, certain limitations exist in each of these studies. Acoustic rhinometry was found to lack accuracy when it comes to discerning expansion or constrictions less than 3 to 4mm (Djupesland et al 2001). Lateral and posteroanterior cephalometric radiographs have been traditionally used to compare the dimensional changes in the maxilla and the upper airway. However, the complexity of the 3D airway anatomy added to the superimposition of the bilateral structures, magnification differences and difficulties in landmark identification may well have overlooked important anatomical features relevant to the airway analysis, questioning the accuracy of 2-dimensional (2D) representations (Chung et al. 2004). Major et al. found that there was at best, a moderate correlation ($r=0.68$) between linear measurements of the upper airway in a 2D cephalometric film and the diagnosis of the upper airway blockage, suggesting that 2D cephalograms should be used only as a screening tool for airway obstruction (Major 2006).

The available 3D techniques including MRI and computed tomography may depict the true morphology of the airway; however, their use is limited by high radiation, high cost and restricted accessibility. Among all the existing 3D imaging techniques, CBCT has become an alternative technique to CT scanning for a comprehensive head and neck evaluation due to its significantly lower overall effective radiation dose and greater spatial resolution than medical CT, high contrast between the hard and soft tissues, lower cost and easier access and availability to dentists (Mah 2004, Ogawa 2007, Tso 2009). Despite the fact that with CBCT, it is not possible to discriminate between the various soft tissue structures, it is possible to determine the boundaries between soft tissues and air spaces making CBCT a potential diagnostic method to analyze airway dimensions (Lenza et al 2010).

OBJECTIVE

The objective of this study was to evaluate the changes of the upper airway dimension and transverse width after RME therapy in growing children through a comparison between pre- (T1) and post- (T2) RME treatment CBCT scans. The null hypothesis is that RME produces no changes in the mean volume and MCA of upper airway within the same individual between pre- and post- RME. We also evaluated the differences in the minimum cross sectional area of the upper airway, retropalatal airway volume, retroglottal airway volume, cross sectional areas of the superior and inferior border of the retropalatal airway

as well as the inferior border of the retroglossal airway. In addition, the transverse widths were measured between the bilateral mid palatal alveolar plates between of the two maxillary first molars and of the two maxillary first premolars.

CHAPTER III
MATERIALS AND METHODS

A total of 14 children (5 boys, 9 girls) with mean age of 12.93 (1.91) years, ranging from 9.67 to 16.02 years participated in this study. The subjects were recruited from the Department of Developmental Sciences/ Orthodontics at Marquette University School of Dentistry. The institutional review board approved this research (#HR-1905) and informed consent from the parents as well as informed assent from the patient were obtained prior to participation.

The inclusion criteria comprised of children between the ages of 9 and 16 with unilateral or bilateral posterior cross bites scheduled to receive RME as an integrative part of their comprehensive orthodontic treatment. Patients were excluded if there was a history of craniofacial anomaly and systemic disease.

All patients were treated with a hyrax type maxillary expander banded on the maxillary first premolars and first molars. The planned activation protocol consisted of 1 turn (0.25mm) per day for 28 consecutive days or until the resolution of posterior cross bite. Clinical observation of 2 to 3 mm overexpansion marked the termination of expansion and the beginning of the retention phase consisted of tying off the jackscrew with a ligature wire and placing a smooth composite material over it. The initial CBCT scan (T1) was taken between 0 to 14 days prior to cementation of the maxillary expander and the progress CBCT scan (T2) was taken between 3 to 4 months after completion of active maxillary expansion to allow new bone to fill in the space at the suture, and the skeletal expansion to become stable (Proffit 2007).

CBCT System and Definition of Airway Spaces

All CBCT scans were taken by one certified radiologist (L.K) at the Radiology department at Marquette University School of Dentistry, using Scanora 3D from Sorodex (Made in Finland) under an extended field of view mode (14.5 x 13.0 cm). The overall effective radiation dose was 125 μ Sv, with 0.35mm voxel size, total scanning time of 20 seconds and effective radiation time of 4.5 seconds. Patients were seated upright with their chin supported on an adjustable platform and the Frankfort horizontal plane parallel to the floor while the rotating source detector captured a volumetric image of the patient's head. Just before scanning, all patients were instructed to keep their teeth in contact throughout the scanning process. The obtained images were reconstructed and imported as DICOM (digital imaging and communications in medicine) data files into Dolphin imaging software (version 11.0 Dolphin Imaging and Management Solutions, Chatsworth, California).

All CBCT images were first oriented so that at the frontal view the skeletal midline (Nasion to anterior nasal spine (ANS)) was perpendicular to the floor and at the axial view the mid sagittal line (mid point between the maxillary incisors to PNS) was perpendicular to the floor (Fig.2,3).

In cases of asymmetry, the orientation was made as close as possible to these guidelines. Once the image was properly oriented, the software was able to create a 2D simulated lateral cephalometric image at the mid sagittal plane. From this view the airway analysis tool was used to define the airway of interest. Because the nasal cavity contained multiple connecting air cavities, turbinates and rarefactions, a clear segmentation was not possible and it was excluded

from our measurements.

In our study, we determined the limits of the upper airway as follows (Fig. 4): Anteriorly by the posterior wall of the soft palate and base of the tongue. Posteriorly by the posterior pharyngeal wall. Superiorly by a plane traced on the midsagittal view connecting the posterior nasal spine (PNS) to basion (Ba) arbitrarily named as “P plane”. Inferiorly by a plane traced on the midsagittal view parallel to the “P plane” passing through the most superior point of the epiglottis arbitrarily named as “EP plane”. The upper airway was divided into 2 segments to further evaluate the specific effect of RME. The upper segment or retropalatal (RP) airway (Fig. 5) limited superiorly by the P plane and inferiorly by a horizontal plane crossing the most postero-inferior point of the soft palate arbitrarily named as “SP plane” (Lenza 2010).

To increase the accuracy of the airway measurements, once the PNS and Ba points were selected in the midsagittal view, the P plane was re-oriented so that it became parallel to the floor and subsequent planes (SP and EP planes) were traced parallel to P plane. The inferior segment or the retroglossal (RG) airway (Fig. 6) was limited superiorly by the SP plane and inferiorly by the Ep plane (Zhao et al. 2010). Once each airway has been demarcated, Dolphin 3-D software allowed the selection of the airway by defining a threshold range of CT units that characterized all empty spaces of the head and neck region.

In our study, we arbitrarily standardized the threshold range to 60 units after observing consecutively that this unit provided the most comprehensive

airway selection without adding or leaving out upper airway space with the exception of 2 patients whose threshold range was decreased to 50 units. Because the air space exhibits a lower CT value than the more dense surrounding soft tissue, it was possible to produce a clean segmentation of the airway. Using the sinus/airway analysis option, boundary position, seed point and update volume option, airway volumes for the oropharyngeal, RP, RG airways, minimal cross sectional area (MCA) (Fig. 7) and cross sectional area for P plane (Fig. 8), SP plane and EP plane (Fig. 9) were obtained.

To evaluate the effect of the RME appliance over the transverse dimension, mid lingual alveolar plate points were first located from the axial view for each of the maxillary first bicuspid and first molars and their transverse widths were measured from the coronal view to enhance visibility and accuracy (Fig. 10, 11). This step was performed using the digitize/measure option. All measurements were performed by one of the investigators (Y.H.C) who was trained and calibrated to identify 3D landmarks on axial, sagittal and coronal planes by a certified radiologist (L.K.).

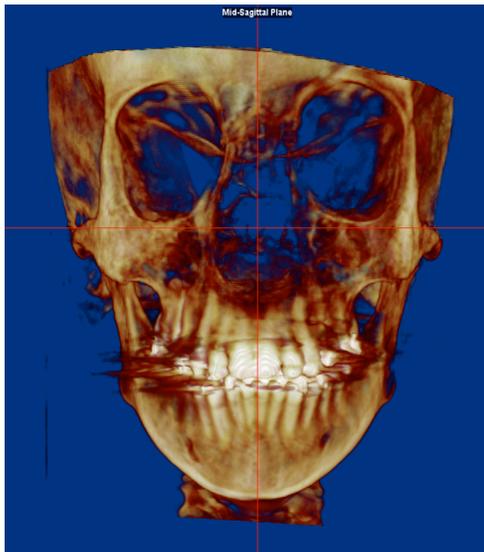


Fig. 2. Skeletal midline orientation from frontal view

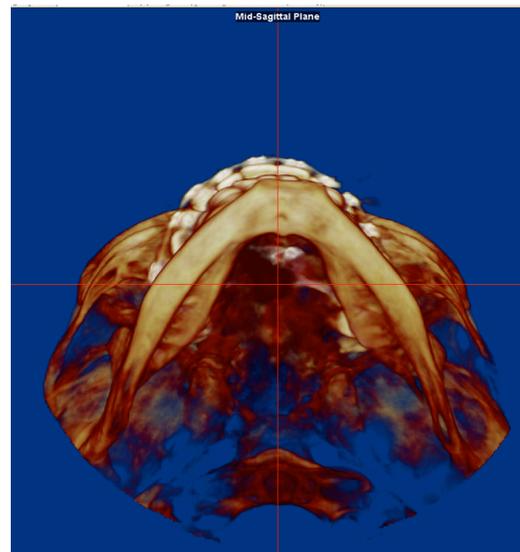


Fig. 3. Mid-sagittal line orientation from axial view

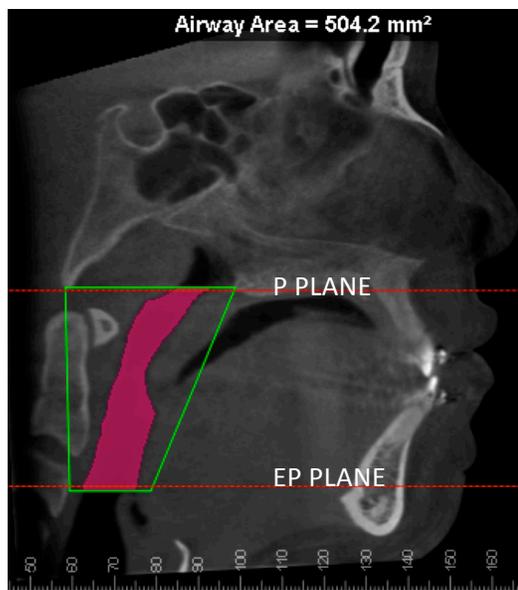


Fig. 4. Total upper airway



Fig. 5. Retropalatal airway

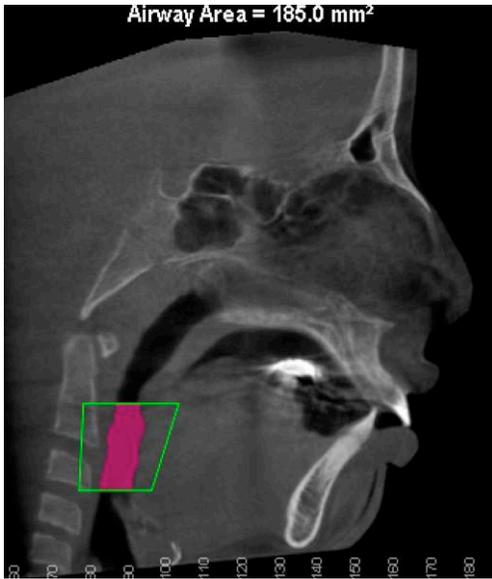


Fig. 6. Retroglottal airway (RG)

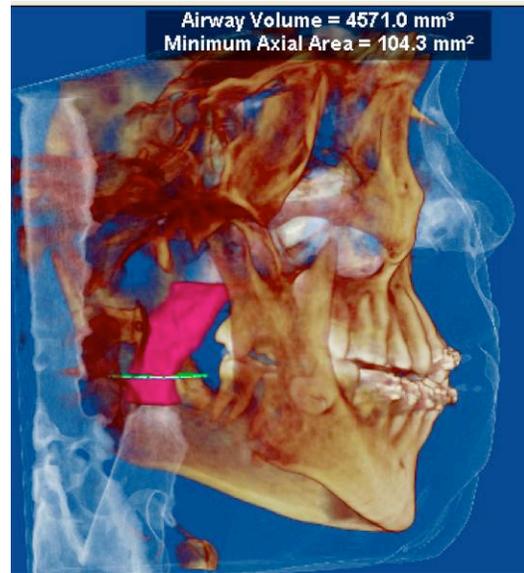


Fig. 7. Minimal cross sectional airway (MCA)



Fig. 8. "P" plane cross sectional airway

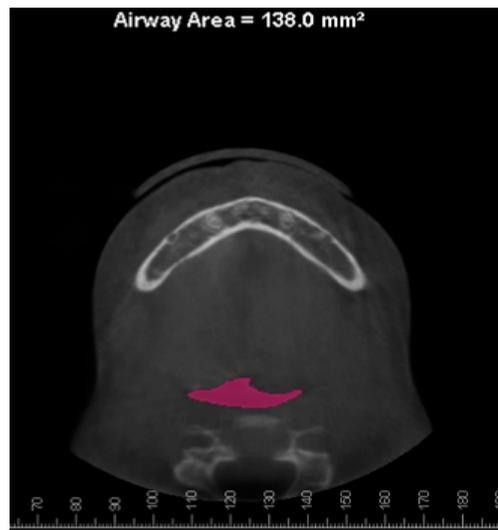


Fig. 9. "EP" plane cross sectional airway



Fig. 10. Location of the mid lingual alveolar plates at the maxillary 1st molars from axial view airway

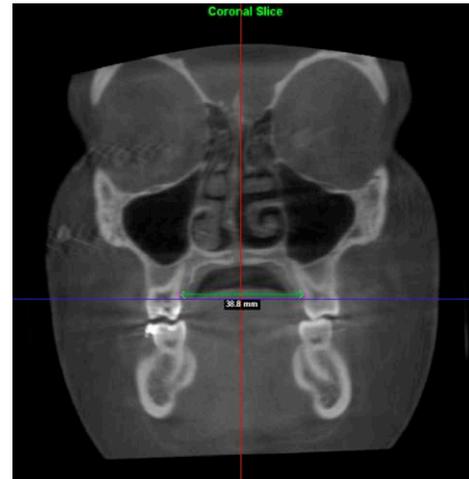


Fig. 11. Inter-mid lingual plates at the maxillary 1st molar level from coronal plane

Statistical Analysis

Pre and post RME dimensions were compared by using paired *t* test. To improve accuracy, all measurements were repeated 3 times with 1 week apart and the means were used for comparison. Intra-examiner reliability coefficient was calculated for 3 random parameters using the Shrout-Fleiss measure of reliability. Comparison analysis for each variable included only the changes “ $[(T2/T1)-1] \times 100\%$ ” due to the different dimensions of anatomical structures among individuals. To investigate possible correlation between the variables reaching significance, *Pearson* correlation analysis was performed. All analyses were based on significant level of 0.05.

CHAPTER IV

RESULTS

The intra examiner reliability coefficient for the randomly selected parameters were 0.995, 0.853 and 0.982 for the RP sagittal area, P plane cross sectional area and 1st inter molar linear measurement respectively. The T2 CBCT scan was taken 105.6 (14.52) days (ranging from 90 to 133 days) after the retention phase started. On average there was 158.4 (27.29) days (ranging from 119 to 211 days) interval between the T1 and T2 scans.

Descriptive statistics of T1 and T2 measurements are listed in Table 1 and 2 respectively. Percentage differences between T2 and T1 are listed in Table 3. No significant changes were found for the mid sagittal area and volumes for the upper airway and its segments between T1 and T2. The P plane cross sectional area (measured from PNS to Ba) increased by 58.5% and it was the only airway parameter that showed statistical significance ($p=0.0004$) Table 3. The MCA was mostly found within the RP airway and increased on average by 16.6%.

The transverse expansion measured between the mid-lingual aspects of the maxillary 1st molars and 1st bicuspid were equal at 4.76mm ($p=0.000$); however, the average percent increase $[(T2/T1)-1 \times 100\%]$ at the 1st premolar level (19.2%) was larger than that at 1st molar level (14.4%) ($p = 0.035$). Finally, the “P” plane cross sectional area increase was highly correlated with the inter mid lingual plates width measured at the level of the maxillary 1st molars ($p=0.0013$) (See table 4).

Table 1. Descriptive statistics of T1 airway parameters

	<i>Measurements</i>	<i>Mean</i>	<i>Median</i>	<i>Std Dev</i>	<i>Q1</i>	<i>Q3</i>
T1	Sag area1 (mm ²)	290.6	274.5	92.0	220.9	261.7
Retropalatal	Volume1 (mm ³)	6277.5	5084.2	2980.0	3777.0	9382.2
	MCA1 (mm ²)	163.2	127.5	104.0	103.0	224.7
	MCA- SP (mm)	4.86	3.25	6.01	2.00	6.00
	SP area1 (mm ²)	143.0	124.8	67.5	100.1	190.8
	P area1 (mm ²)	225.2	241.9	80.5	158.9	277.6
	Sag area2 (mm ²)	291.3	271.9	92.6	213.8	364
	Volume2 (mm ³)	6303.6	5229.3	3012.0	3589.6	9429
	MCA2 (mm ²)	163.3	127.5	104.4	102.9	224.7
	MCA-SP (mm)	4.89	3.50	6.02	2.0	6.0
	SP area2 (mm ²)	157.0	132.6	82.3	99.6	181.8
	P area2 (mm ²)	215.0	204.9	119.6	114.9	268.8
	Sag area3 (mm ²)	294.8	278.5	94.2	213.8	370.1
	Volume3 (mm ³)	6378.7	5300.2	3006	3855.1	9451.6
	MCA3 (mm ²)	164.1	127.6	104.0	102.9	224.7
	MCA-SP3 (mm)	5.11	3.5	5.82	2.0	5.0
	SP area3 (mm ²)	156.4	147.4	83.1	80.5	203.8
	P area3 (mm ²)	246.1	240.8	107.4	158.9	306.6
T1	Sag area (mm ²)	292.3	277.8	92.75	216.2	365.3
Retropalatal	Volume (mm ³)	6319.9	5204.5	2997.6	3756.6	9461.1
averages	MCA (mm ²)	163.5	128.0	104.1	102.9	224.7
	SP area (mm ²)	152.1	140.1	73.0	98.6	209.7
	P area (mm ²)	228.8	237.0	97.7	158.9	286.7
T1	Sag area (mm ²)	242.1	240.7	54.1	205.3	286.4
Retroglossal	Volume (mm ³)	4873.9	4309.5	1945.4	3525.0	6707.6
T1 Width	Intermolar (1 st)	32.0	32.0	2.88	29.9	33.6
(mm)	Interbis (1 st)	24.0	24.5	2.43	22.6	26.0
	Intermolar (2 ⁿ)	31.9	31.6	2.79	29.7	33.8
	Interbis (2 nd)	24.1	24.4	2.45	22.4	25.8
	Intermolar (3 rd)	31.8	31.7	2.98	30.3	33.5
	Interbis (3 rd)	24.0	24.3	2.31	22.5	25.6
T1 Width	Intermolar	31.9	31.7	2.85	30.6	33.8
average(mm)	Interbis	24.0	24.4	2.39	22.5	25.7

T1 Total airway	Area1 (mm ²)	533.6	536.4	122.0	453.2	621.3
	Volume1 (mm ³)	11204.0	12217.3	4103.0	7209.8	13921.8
	EP area1 (mm ²)	218.6	220.9	103.2	128.3	308.9
	Area2 (mm ²)	533.7	533.5	124.7	440.8	626.2
	Volume2 (mm ³)	11161.3	12217.3	4138.3	7104.6	13844.2
	EP area2 (mm ²)	218.8	213.3	105.6	128.3	308.9
	Area3 (mm ²)	535.9	536.5	124.5	444.9	626.2
	Volume3 (mm ³)	11216.0	12355.8	4146.0	7141.0	13844.2
	EP area3 (mm ²)	213.7	196.0	93.0	138.0	316.3
T1 Total airway averages	Area (mm ²)	534.4	535.5	123.7	446.3	620.7
	Volume (mm ³)	11193.8	12263.5	4128.8	7139.7	13870.1
	EP area (mm ²)	217.0	210.2	99.4	128.4	316.6

Table 2. Descriptive statistics of T2 airway parameters

	<i>Measurement</i>	<i>Mean</i>	<i>Median</i>	<i>Std Dev</i>	<i>Q1</i>	<i>Q3</i>
T2 Retropalatal Averages	Sag area1 (mm ²)	304.3	313.7	110.0	212.2	384.9
	Volume1 (mm ³)	7482.0	7391.8	3295.3	4366.5	9907.9
	MCA1 (mm ³)	167.8	119.7	93.7	100.6	255.8
	MCA-SP (mm)	1.96	2.0	5.37	1.0	5.0
	SP area1 (mm ²)	158.7	146.8	76.0	101.1	219.5
	P area1 (mm ²)	322.0	324.5	104.2	264.2	404.1
	Sag area2 (mm ²)	307.3	315.8	109.2	226.4	384.9
	Volume2 (mm ³)	7556.1	7312.8	3294.9	4514.7	10247.4
	MCA2 (mm ²)	163.0	119.7	99.2	100.6	255.8
	MCA-SP2 (mm)	1.96	2.0	5.11	1.0	5.0
	SP area2 (mm ²)	156.9	145.2	70.6	106.5	210.5
	P area2 (mm ²)	324.7	316.7	104.2	292.5	387.9
	Sag area3 (mm ²)	304.0	303.2	106.9	215.9	388.9
	Volume3 (mm ³)	7525.3	7207.7	3308.0	4479.0	9891.9
	MCA3 (mm ²)	172.2	119.7	104.7	98.4	255.8
	MCA-SP3 (mm)	1.93	2.0	5.36	1.0	5.0
	SP area3 (mm ²)	175.5	145.6	104.0	103.6	243.9
P area3 (mm ²)	337.9	320.7	132.3	258.5	405.5	
T2 Retropalatal Averages	Sag area (mm ²)	305.2	312.0	108.6	215.4	386.3
	Volume (mm ³)	7521.2	7304.1	3296.2	4453.4	10015.7
	MCA (mm ²)	167.7	119.7	98.6	101.9	255.8
	SP area (mm ²)	163.7	145.9	78.7	101.2	237.8
	P area (mm ²)	328.2	323.7	107.1	273.9	403.4
T2 Retroglossal	Sag area (mm ²)	234.5	207.8	82.4	192.1	244.5
	Volume (mm ³)	5407.7	4512.7	3105.2	3409.1	5567.6
T2 Width (mm)	Intermolar (1 st)	36.7	36.1	3.30	34.2	39.7
	Interbis (1 st)	28.9	29.0	3.24	26.8	30.3
	Intermolar (2 ⁿ)	36.7	36.1	3.00	34.1	38.8
	Interbis (2 nd)	28.8	29.0	3.11	26.6	30.3
	Intermolar (3 rd)	36.6	36.0	3.03	34.1	38.8
	Interbis (3 rd)	28.7	28.7	3.19	26.6	30.5
T2 average	Intermolar	36.7	36.0	3.09	34.3	39.4

width (mm)	Interbis	28.8	29.0	3.16	26.7	30.5
T2 Total airway	Area1 (mm ²)	539.6	513.1	152.3	481.2	659.7
	Volume1 (mm ³)	12926.4	11259.1	5602.0	8461.8	15881.2
	EP area1 (mm ²)	210.5	197.3	88.3	128.2	269.7
	Area2 (mm ²)	539.1	508.6	155.9	473.2	664.2
	Volume2 (mm ³)	12914.1	11291.1	5672.6	8375.1	15983.5
	EP area2 (mm ²)	225.0	213.3	87.1	167.3	259.2
	Area3 (mm ²)	540.4	508.4	153.0	477.5	659.7
	Volume3 (mm ³)	12946.1	11195.5	5634.9	8503.4	15881.2
	EP area3 (mm ²)	228.0	241.3	70.0	191.4	250.6
T2 Total airway averages	Area (mm ²)	539.7	509.4	153.7	477.3	661.2
	Volume (mm ³)	12928.9	11248.6	5635.9	8446.8	15915.3
	EP area (mm ²)	221.1	204.8	78.4	169.7	259.2

Table 3. Percentage change between T2 and T1 measurement

Type	Measurements	T1 (S.D.)	T2 (S.D.)	$[(T2/T1)-1]$ x100%	T2-T1
Retro-palatal Airway	P plane (mm ²)	228.77 (97.7)	328.2 (107.1)	43.46%	
	SP plane (mm ²)	152.1 (73)	163.7 (78.7)	7.6%	
	Volume (mm ³)	6319.9 (2997.6)	7521.2 (3296.2)	19%	
	Sagittal area (mm ²)	292.3 (92.75)	305.2 (108.6)	4.41%	
	MCA (mm ²)	163.5 (104.1)	167.7 (98.6)	2.55%	
Retro-glossal airway	EP plane (mm ²)	217 (99.4)	221.1 (78.4)	1.9%	
	Volume (mm ²)	4873.9 (1945.4)	5407.7 (3105.2)	10.95%	
	Sagittal area (mm ²)	242.1 (54.1)	234.5 (82.4)	-3.13%	
Total airway	Volume (mm ²)	11193.8 (4128.8)	12928.9 (5635.9)	15.5%	
	Sagittal area (mm ²)	534.4 (123.7)	539.7 (153.7)	0.99%	
Transverse width	6-6 (mm)	31.9(2.85)	36.7(3.09)	14.9%	4.76
	4-4 (mm)	24 (2.39)	28.8(3.16)	19.8%	4.76

Table 4. Descriptive statistics of age at T1 (years)

<i>Mean</i>	<i>Median</i>	<i>Std Dev</i>	<i>Q1</i>	<i>Q3</i>
12.94	13.08	1.91	11.43	14.21

Table 5. Descriptive statistics of age at T2 (years)

<i>Mean</i>	<i>Median</i>	<i>Std Dev</i>	<i>Q1</i>	<i>Q3</i>
13.37	13.50	1.91	11.83	14.66

Table 6. Descriptive statistics of days between T2 and T1

<i>Mean</i>	<i>Median</i>	<i>Mode</i>	<i>Std Dev</i>	<i>Q1</i>	<i>Q3</i>
158.4	154	147	27.29	142	168

Table 7. Descriptive statistics of days between T2 and retention phase start

<i>Mean</i>	<i>Median</i>	<i>Mode</i>	<i>Std Dev</i>	<i>Q1</i>	<i>Q3</i>
105.6	105	105	14.52	92	111

Table 8. Retropalatal measurements Paired *t* testsSagittal area (mm²)

<i>Mean Diff</i>	<i>95% CL Mean</i>	<i>t Value</i>	<i>P-value</i>
-12.90	(-59.79, 33.99)	-0.59	0.5625

Volume(mm³)

<i>Mean Diff</i>	<i>95% CL Mean</i>	<i>t Value</i>	<i>P-value</i>
-1201.2	(-2944.3, 541.8)	-1.49	0.1604

MCA (mm²)

<i>Mean Diff</i>	<i>95% CL Mean</i>	<i>t Value</i>	<i>P-value</i>
-4.167	(-68.08, 59.75)	-0.14	0.8902

“SP” plane axial area (mm²)

<i>Mean Diff</i>	<i>95% CL Mean</i>	<i>t Value</i>	<i>P-value</i>
-11.56	(-64.84, 41.71)	-0.47	0.6469

“P” plane axial area (mm²)

<i>Mean Diff</i>	<i>95% CL Mean</i>	<i>t Value</i>	<i>P-value</i>
-99.44	(-144.9, -54.00)	-4.73	0.0004

Table 9. Retroglossal measurements Paired *t* testsSagittal area (mm²)

<i>Mean Diff</i>	<i>95% CL Mean</i>	<i>t Value</i>	<i>P-value</i>
7.59	(-49.01, 64.18)	0.29	0.7766

Volume (mm³)

<i>Mean Diff</i>	<i>95% CL Mean</i>	<i>t Value</i>	<i>P-value</i>
-533.8	(-2547.1, 1479.4)	-0.57	0.5765

“EP” plane axial area (mm²)

<i>Mean</i>	<i>95% CL Mean</i>	<i>t Value</i>	<i>P-value</i>
-4.12	(-70.44, 62.20)	-0.13	0.8952

Table 10. Paired *t* Tests of total airway measurementsSagittal area (mm²)

<i>Mean Diff</i>	<i>95% CL Mean</i>	<i>t Value</i>	<i>P-value</i>
-5.31	(-90.61, 79.99)	-0.13	0.8951

Volume (mm³)

<i>Mean Diff</i>	<i>95% CL Mean</i>	<i>t Value</i>	<i>P-value</i>
-1735.1	(-5182.6, 1712.5)	-1.09	0.2967

Table 11. Transverse width Paired *t* Tests

Inter mid lingual plates at maxillary 1st molars level (measurement comparison)

<i>Mean Diff</i>	<i>95% CL Mean</i>	<i>t Value</i>	<i>P-value</i>
-4.55	(-5.56, -3.54)	-9.71	<.0001

Inter mid lingual plates at maxillary 1st premolars (measurement comparison)

<i>Mean Diff</i>	<i>95% CL Mean</i>	<i>t Value</i>	<i>P-value</i>
-4.59	(-5.44, -3.72)	-11.52	<.0001

Inter mid lingual plates at maxillary 1st molars level (percentage comparison)

<i>Mean Diff</i>	<i>95% CL Mean</i>	<i>t Value</i>	<i>P-value</i>
0.877	(0.850, 0.904)	-9.91	<.0001

Inter mid lingual plates at maxillary 1st premolars level (percentage comparison)

<i>Mean Diff</i>	<i>95% CL Mean</i>	<i>t Value</i>	<i>P-value</i>
-0.842	(0.814, 0.869)	-12.52	<.0001

Table 12. Correlation analysis

“P” plane axial cross sectional area v/s maxillary inter- 1st molars width

<i>U axial mean(std dev)</i>	<i>6-6 mean(std dev)</i>	<i>Correlation Coefficient</i>	<i>P-value</i>
-99.44 (78.70)	-4.55 (1.75)	-0.77	0.0013

CHAPTER V

DISCUSSION

Several craniofacial abnormalities including retrognathic mandible, shorter AP face length, reduced distance from the posterior nasal spine (PNS) to posterior pharyngeal wall, lower positioned hyoid bone, larger soft palate, smaller pharynx, larger tongue size, obesity and combination thereof have been named as being part of the pathophysiology of OSA (Johal 2007). It is hypothesized that these abnormalities predispose to OSA through the constricting effect on the upper airway dimensions. Maxillary constriction in particular has been postulated to play a role in the pathophysiology of OSA because of its association with low tongue posture that may contribute to the oropharynx airway narrowing (Subtelny 1954). Pirelli et al. grouped 31 children with OSA and followed them up to 4 months after RME treatment. All of these children had their apnea-hypopnea index decreased while their mean maxillary cross sectional width expanded to about 4.5mm. In our study, the MCA was maintained pre and post RME possibly suggesting that a breathing improvement may have developed from soft tissue changes other than MCA. Further studies using rhinomanometric assessment to measure air pressure and rate of airflow during breathing as well as sleep studies measuring all phases of breathing in a comprehensive manner need to be done to validate a possible association or correlation between RME and breathing improvement.

Enoki et al. evaluated the effect of RME on the nasal cavity in 29 children comparing acoustic rhinometric and computed rhinomanometric values before (T1), immediately after (T2) and 90 days post RME (T3). Their results stated no significant difference for the MCA at the level of the nasal valve and of the inferior

turbinate through the acoustic rhinometric evaluation. Nevertheless, despite the absence of MCA change, the computed rhinomanometry detected a progressive decrease in the inspiration and expiration resistance reaching statistical difference between T1 and T3 indicating that the benefits of RME may be a modest functional improvement based on bony expansion rather than mucosal change (Enoki et al. 2006). Our findings indicate that not only bony expansion is found after RME, but also a significant cross sectional area increase immediately posterior to the hard palate. Interestingly, the P plane cross sectional area increase was correlated to the expansion at the 1st molar level suggesting that the airway change is dependent on the transverse width gain. We believe that the effect of RME on the upper airway is local in nature and diminishes further down possibly as a result of soft tissue adaptation. In other words, the further away from the maxillary suture, the less effect on the upper airway.

Studies in the field of imaging the airway have emphasized that the airway dimensions can change with the phase of respiration (Bhattacharyya N. et al. 2000). Studies using functional 3-D CT techniques have shown the variability of the airway dimension behind the tongue at 10 seconds scan interval and also have demonstrated the changes seen after a mandibular advancement device (MAD) is placed in the mouth. Interestingly, the effect of MAD on the airway occurred more laterally than anteroposteriorly increasing the cross sectional area (Kyung SH et al. 2005). This lateral effect on the airway is also perceived in our study by the lack of change in the sagittal area measurements suggesting that the antero-posterior effect of the RME on the upper airway is not significant.

One of the limitations of our study is that the subjects were not given a special instruction for breathing other than to keep the teeth in contact during the 20 second scan. During this time both inspiration and expiration would have taken place and may have contributed differently to the airway size and shape. However, a special breathing instruction might have introduced an artificial mechanism differing from the airway observed during quiet breathing with the possibility of producing an erroneous depiction of the three dimensional structure. To test the effects of the different phases of respiration and swallowing, one of our investigator (D.L.) volunteered to have 2 CBCT scans in a row while performing 3 to 4 swallowings during the CBCT scan. These images demonstrated that both volumetric and cross sectional area measurements were considerably different due to the blurred tongue and unequal soft palate position. None of our CBCT images presented any blurriness at the tongue neither at the soft palate level.

The amount of transverse width gained at the mid lingual alveolar plate of maxillary 1st molars and 1st premolars was identical at 4.76 mm. However, the percentile increase $[(T2/T2)-1 \times 100\%]$ at the 1st premolar level (19.2% avg.) was larger than that at 1st molar level (14.4% avg.), revealing a triangular expansion with the base located anteriorly. This is in agreement with previous studies in which the maxillary expansion was evaluated using axial CT (Lione et al), multislice CT (Ballanti et al. 2010) and CBCT (Garrett et al).

A retrospective analysis of 10 adult human airways using CBCT images scanned while sitting upright demonstrated that the position of the MCA varied

but was more often located in the oropharyngeal region (Tso et al). In a MRI study where subjects were evaluated both during waking and sleep states it was concluded that the smallest cross sectional area was located in the RP area in 13 of 15 subjects (Trudo et al 1998). In our study, the MCA was mostly found within the RP airway with the exception of 3 patients whose MCA was located in the RG airway. According to Tso et al, the range of the MCA in healthy adults varied from 90 to 360 mm² (Tso et al 2009).

In an airway study evaluating subjects with OSA using spiral CT, it was found that the average MCA for the OSA patients was 67.1 mm² while the control subjects had a mean value of 177.8 mm² (Galvin JR, 1989). In our study, the mean MCA at T1 was 163.5 mm² and it ranged from 71.5 to 461.5 mm² (Table 1). These numbers compares favorably with the healthy population previously mentioned. Whether or not airway dimensions scanned during quiet breathing correlate with apneic events during sleep is still a controversial matter. However, there is evidence that OSA subjects demonstrate smaller cross sectional areas of the airway, implying that there is a range in size for the airway in normal subjects, and that subjects with OSA can be below this range (Tso et al.)

It is important to recognize that due to the 3D nature of the scans, small tracing variations could potentially bring significant differences in the airway measurements. The P plane orientation parallel to the floor was aimed to minimize the inherent tracing variations by ensuring that subsequent PNS and Ba points selection produce a line parallel to the floor and its cross sectional area could be reliably measured from the axial view. Secondly, the PNS and Ba

points were always visible at the mid sagittal view and it became evident after a few trial tracings that they provided the most reliable and easily detectable points to define the superior boundary of the upper airway (Lenza et al 2010). The RP airway inferior limit (RG airway superior limit) was defined as a line parallel to P plane contacting the most inferior aspect of the uvula or soft palate (SP plane) in reference to a previous study (Zhao et al. 2010, Lenza et al. 2010).

One of the benefits of a prospective study is the collection of data after the establishment of a study protocol. This enables to define a desired sample group as well as to create a standardized intervention that may demonstrate a stronger relationship between the two than that of a retrospective study (Manolio et al). In our study, we proposed to take a T1 CBCT scan between 0 and 14 days prior to the expander cementation, to turn the Hyrax expander once a day for 28 consecutive days (or until the resolution of the cross bite) and to take the T2 CBCT scan between 3 to 4 months after completion of the maxillary expansion. Despite our efforts to strictly follow our initial proposal, there was on average 158.4 days (ranging from 119 to 211 days) between T1 and T2 scans. This wide range of days between the two scans may be explained by the presence of 2 patients whose parents misunderstood how to turn the expander, missed appointments or a lacked of compliance with the expansion protocol.

There has been an increased public awareness of radiation dose of CBCT scans due to the publication of a CBCT related article in a national news paper (Bodganich W, Craven J. 2010). In this regards, there is no doubt that the “as low as reasonably achievable” (ALARA) principle should be enforced to avoid

unnecessary exposure to all of our patients (Farman 2005). However, the reader and the general public should acknowledge that the radiation dose of a CBCT scan is low. The extended field of view CBCT scan used in our study expose the patients to radiation equivalent of 6.4 times the conventional panoramic x-rays or 15 days of background radiation (Ludlow 2006). The medical CT scans expose the patient to 1,500 to 20,000 μSv depending on scanning location and the mass of the individual (Danforth et al. 2000).

According to the U.S Nuclear Regulatory Commission most people receive 3mSv per year of radiation from background sources and 0.6mSv per year from artificial sources totaling an average annual dose of 3.6mSv (USNRC Biological Effects of Radiation 2004). Although radiation may cause cancers at high doses and high dose rates, currently there are no data to unequivocally establish the occurrence of cancer following exposure to low doses (dose rating below 100mSv). Interestingly, people living in areas having high levels of background radiation (above 10mSv per year) such as Denver Colorado, have shown no adverse biological effects.

At present, The United States Nuclear Regulatory Commission (USNRC) has limited the radiation exposure to the public to 1mSv (1000 μSv) per year and our CBCT scans represents 25% of that allowed dosage well below their safety limit (USNRC Biological Effects of Radiation 2004). However it is thought that no amount of radiation can regarded as "safe". It is an ethical obligation for all health care providers to limit unnecessary radiation exposure to our patients.

CHAPTER VI
CONCLUSIONS

Based on the results of our study, RME produced a numerically parallel expansion of the mid palatal suture and a triangular shape of expansion with the base facing anteriorly when percentage change was calculated. In regard to the airway, a moderate increase of the cross sectional area adjacent to the hard palate was observed. This cross sectional area increase was highly dependent on the expansion between the 1st molars. The RME effect on the airway diminished as it moved further away from the mid palatal suture possibly due to the compensation generated by the surrounding soft tissues in a 3D frame.

Further studies with a larger sample size and incorporating breathing evaluation would be necessary to estimate the real impact of the RME on the airway.

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