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A RETROSPECTIVE EVALUATION OF **OROFACIAL AIRWAY DIMENSIONS AFTER RAPID MAXILLARY EXPANSION**

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ABSTRACT

Objective: The purpose of the study was to assess the sagittal oro-pharyngeal airway dimensions after rapid maxillary expansion in Indian populations.

Materials and methods: Pre and post treatment lateral cephalograms of 100 patients treated with rapid maxillary expander were obtained. Cephalometric values (pre and post expansion) of upper and lower airway spaces in sagittal dimension were recorded and compared.

Results: There was a significant increase in the sagittal dimension of the upper airway. However, no substantial difference was observed with respect to the sagittal dimension of lower airway.

Conclusion: Rapid maxillary expansion definitely improves the upper pharyngeal airway, but haslimited influence on the lower pharyngeal airway.

Key Words: Rapid Maxillary expansion, oropharyngeal airway dimensions, lateral cephalograms

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INTRODUCTION

Constricted maxillary arch is commonly associated with narrowing of the pharyngeal airway along with cross bite (unilateral/bilateral), occlusal disharmony and aesthetic problems^{1,2}. A glance in to the literature reveals the role maxillary constriction in the aetiology of obstructive sleep apnoea (OSA)³⁻⁵ OSA is characterized by the episodic cessation of breath during sleep. This results in oxygen desaturation and frequent arousal from sleep⁶.Several studies have reported that patients with OSA have abnormal cephalometric dentofacial morphologic patterns associated with reduced cranial base length and angle, increased ANB angle, hyperdivergent mandibular plane, elongated maxillary and mandibular teeth, narrowing of the upper airway, long and large soft palate, and large tongue ^{1,2,7–10}.

Haskell et al reported that OSA has a high prevalence rate of 4% in men and 2% in women¹¹. The most serious consequences of OSA include cardiovascular diseases such as hypertension, tachycardia, atherosclerosis, increased risk for cerebrovascular accidents, coronary artery disease and more¹². 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 time¹³. 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 lumen¹⁴.

Preventing the collapse of the lumen of the pharynx during sleep is the main objective in treatment of OSA. At present, several treatment options based on the severity of the apnoeic events are rendered, including continuous positive airway pressure (CPAP) therapy, surgical treatments and mandibular repositioning therapy. Oral appliances have been reported to improve breathing by decreasing nasal resistance and reducing the apnoea 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 effects¹¹.

Rapid maxillary expansion is the treatment of choice for the correction of maxillary transverse width deficiency since 1860¹⁵. A great deal of research has been carried in this field since then ^{16, 17,} ⁸. Several studies reported that RME produces craniofacial structural changes along with dentofacialchanges ^{17, 18, 19}. Maxillary expansion increases the nasal volume and reduces the nasal airflow resistance, hence, improves the nasal respiration^{19–24}. Hence, our research project aims to describe the upper and lower pharyngeal airway changes after rapid maxillary expansion in Indian population.

Materials and methods:

In this study, lateral cephalograms of 100 patients who had maxillary constriction requiring maxillary expansion were included.

Inclusion criteria

- a) 12–16 years of age.
- b) No history of previous orthodontic treatment.
- c) No history of any surgical procedure directed at their nasal cavities or pharyngeal airway prior to or during treatment.
- d) No craniofacial anomalies/ syndromes.

Lateral cephalograms were obtained prior to the treatment for evaluating the upper and lower pharyngeal airway. Expansion was done using hyrax-type maxillary expander banded on the maxillary first premolars and first molars. The patients were monitored weekly for appropriate activation of the appliance. Hyrax was turned1 or 2 times per day (0.25-0.5 mm) until the desired expansion was achieved. Slight overexpansion was obtained anticipating mild relapse. The appliance was kept in situ for a time period of 6 months for consolidation followed which the appliance was removed and Hawley's appliance was delivered for retention. Post treatment cephalograms (obtained the day of removal of the appliance)was taken to evaluate the upper and lower pharyngeal airway after expansion. The pre and post treatment values were compared to see the net change in the upper and lower pharyngeal airway following expansion.

The lateral cephalometric images for each subject were taken using the same imaging device. The dimensions of the upper and lower pharyngeal airways were measured directly from the cephalometric radiograph according to the McNamara Airway Analysis ^{25, 26}. Upper airway width was measured from point on posterior outline of soft palate to closest point on posterior pharyngeal wall, taken on anterior half of soft palate and lower airway width was measured from intersection of posterior border of tongue and inferior border of mandible to closest point on posterior pharyngeal wall (Figure 1). For inter examiner reliability, measurements for 15 randomly selected patients were repeated by an equally trained examiner, 15 days after the original measurements. For intra examiner reliability, the same examiner repeated the measurements for 15 randomly selected patients almost one month after the first measurements. The statistical analysis was done using SPSS software. An analysis was done using the Student's independent samples t-test for comparison of the variables (pre-treatment airway measurements and the post-treatment airway measurements).

Results:

	Pre Treatment Value	Post Treatment Value	Difference	P value
Upper pharyngeal airway	15.4 ± 1.7 mm	17.1 ± 1.1 mm	1.6 ± 1.5 mm	0.06 **
Lower pharyngeal airway	11.4 ± 1.9 mm	12.1 ± 1.2 mm	$0.3 \pm 1.4 \text{ mm}$	0.21

Table: 1 Comparison of pre and post treatment values of oropharyngeal airway. ** *P* < 0.05 denotes significant change.

A change in upper and lower pharyngeal airway was observed following RME expansion (Table:1). After treatment, the upper pharyngeal airway was significantly (p Value 0.06) increased by 1.6 ± 1.5 mm. However, there was no significant difference (p Value 0.21) in the lower pharyngeal airway measurement (mean = 0.3 ± 1.4 mm).

Orofacial airway dimensions after rapid maxillary expansion



Figure 1

Discussion:

Obstructive sleep apnoea is characterised by the collapse of the pharyngeal airway space resulting in air way obstruction, decreased oxygen saturation and disrupted sleep. Collapse may frequently occur at retroglossal and retropalatal regions ²⁷. Therefore any abnormality in tonsils, adenoids, soft palate, uvula, tongue and lateral pharyngeal walls can affect the airway space. Cephalometric studies have shown that abnormalities in the craniofacial region like narrowed posterior air space, elongation of the soft palate, mandibular deficiency, and inferiorly placed hyoid bone relative to the mandibular plane predispose to OSA by its adverse e ects on the oropharyngeal airway^{1,2,26,28}. Furthermore, maxillary constriction might play a major role in the pathophysiology of OSA since it is associated with low tongue posture that could result in oropharynx.

Although the pharynx is a 3 dimensional structure and patients are usually evaluated wake and upright position, lateral cephalometry is commonly used in clinical practice in our country because of its relative simplicity, accessibility, low cost and minimal radiation. So this technique reveals a variety of soft and hard tissue abnormalities that may indicate patients with narrow and collapsible upper airways. Cephalometry has provided substantial insight in to the pathophysiology of OSA, identifying craniofacial deformities. Moreover posterior airway space can be easily measured from the lateral cephalograms.

In the present study, it was observed that the rapid maxillary expansion showed a significant increase in the upper pharyngeal airway, while there was no significant di erence in the lower pharyngeal airway. An increase in nasal cavity volume and thereby significant reduction in the nasal airway resistance is the advantages of rapid maxillary expansion^{19, 31}. Therefore, maxillary expansion certainly have a positive role on decreasing nasal resistance and increasing the upper airway. This is in agreement with previous studies conducted so far on the effect of RME in the treatment of cases with inadequate nasal capacity and chronic respiratory problems ^{19,24}. Though it is impossible to quantify the linear changes in the size of nasal cavities, RME has been proven to lessen the nasal air resistance ^{32, 33}.

RME should be done in conjunction with transverse constriction of maxilla, not to be performed solely for improving the airway³³.

The average duration of treatment in the present study was 6 months. Age related growth of nasopharynx was an important factor taken in to consideration. Growth has been reported to influence the size and shape of the nasopharynx. It is believed that the total depth of the nasopharynx is established in the first or second year of life ³⁷, while its length continues to increase until maturity. This increase in length was attributed to the descent of the hard palate and cervical vertebrae away from the cranial base ³⁸. Bergland found a 38% increase in nasopharyngeal height from six years of age to maturity ³⁹.

It was found in the present study that after RME treatment, the upper and lower airways were increased. However, the increase was only significant in the upper airway. Literature is abundant with reports stating that the upper pharyngeal depth increases with age, whereas the lower pharyngeal depth was established early in life ³⁸. Johnston and Richardson studied the changes in the pharyngeal skeletal size, pharyngeal soft tissue thickness, pharyngeal airway depth, and soft palate dimensions in addition to standard craniofacial measurements. The results showed absolutely no change in the nasopharyngeal skeletal dimensions, while the antero-posterior depth of the nasopharyngeal lumen increased as a result of a reduction in thickness of the posterior nasopharyngeal wall. Their findings indicate that pharyngeal morphology is not immutably established during childhood and adolescence, but changes throughout adult life ³⁹. RME expansion would truly improve the pharyngeal airway dimensions in maxillary constriction cases which would have positive role on nasal airway resistance.

Conclusion:

There is a significant association between craniofacial morphology and pharyngeal airway sagittal dimensions. After RME, sagittal measurement of hypopharyngeal airway showed no significant change, but a statistically significant increase in nasopharyngeal level was clearly evident in the present study. The change in pharyngeal airway dimensions over time requires research. Advanced three dimensional imaging techniques could provide research a better understanding of the dynamic chang upper and lower airways throughou treatment.

References

- Cistulli PA, Richards GN, Palmisano H Unger G, Berthon-Jones M, Sullivan O Influence of maxillary constriction on resistance and sleep apnea severity in p with Marfan's syndrome. Chest 1996;110(5):1184-8.
- Cistulli PA, Sullivan CE. Influence of maxillary morphology on nasal airway resistance in Marfan's syndrome. Acta Laryngologica 2000;120(3):410-3.
- Johal A, Conaghan C. Maxillary morp in obstructive sleep apnea: a cephalom and model study. Angle Orthodontist 2004;74(5):648-56.
- Kushida CA, Efron B, Guilleminault C predictive morphometric model for the obstructive sleep apnea syndrome. Ann Internal Medicine 1997;127(8):581-7.
- Seto BH, Gotsopoulos H, Sims MR, C PA. Maxillary morphology in obstruct sleep apnoea syndrome. European Jou Orthodontics 2001;23(6):703-14.
- Guilleminault C, Quera-Salva MA, Ni Murcia G, Partinen M. Central sleep a and partial obstruction of the upper air Annals of Neurology 1987;21(5):465-
- Guilleminault C, Tilkian A, Dement W sleep apnea syndromes. Annual Review Medicine 1976;27:465-84..
- U. B. Baik, M. Suzuki, K. Ikeda, J. Su and H. Mitani. Relationship between cephalometric characteristics andobstr sites in obstructive sleep apnea syndro Angle Orthodontist, vol. 72, no. 2, pp. 124–134, 2002.
- 9. J. M. Battagel and P. R. L'Estrange, "T cephalometric morphology of patients

further digital hers with	obstructive sleep apnoea (OSA)," European Journal of Orthodontics, vol. 18, no. 6, pp. 557–569, 1996.
ges in the It RME RG, CE.	 A. A. Lowe, M. M. O[°] zbek, K. Miyamoto, E. K. Pae, and J. A. Fleetham. Cephalometric and demographic characteristics of obstructive sleep apnea: an evaluation with partial least squares analysis. Angle Orthodontist, vol. 67, no. 2, pp. 143–153, 1997.
nasal patients 7 Oto-	11. Jennifer A Haskell, John Mccrillis, Bruce S Haskell, James P Scheetz, William C Scarfe, Allan G Farman Effects of Mandibular Advancement Device (MAD) on airway dimensions assessed with cone-beam computed tomography Seminars in Orthodontics 2009;15:132-58.
hology netric	 Madani D, FaridenMadani D: Definitions, abbreviations, and acronyms of sleep apnea. Atlas Oral Maxillofacial Surg N Am 2007;15:69-80.
C. A e nals of	 Sharabi Y, Dagan Y, Grossman E. Sleep apnea as a risk factor for hypertension (Review). Curr Opin Nephrol Hypertens 2004;13:359- 64.
Cistulli ive rnal of	14. McCrillis J, Haskell J, Haskell BS, Brammer M, Chenin D, Scarfe W and Farman A. Obstructive sleep apnea and the use of cone beam computed tomography in airway imaging: a review 2009;15:63-9.
no- pnea 'way.	15. DJ Timms. The dawn of rapid maxillary expansion, Angle Orthodontist 1999;69(3);247-50.
9. /C. The w of	16. Haas AJ. Palatal expansion: just the beginning of dento facial orthopedics. American Journal of Orthodontics 1970;57(3):219-55.
gawara,	17. Haas AJ. Long-term post treatment evaluation of rapid palatal expansion. Angle Orthodontist 1980;50(3):189-217.
uctive me.	 Haas AJ. Rapid expansion of the maxillary dental arch and nasal cavity by opening the midpalatal suture. Angle Orthod 1961;31(2):73-90.
The with	19. Hershey HG, Stewart BL, Warren DW. Changes in nasal airway resistance associated with rapid maxillary expansion. American

Journal of Orthodontics 1976;69(3):274-84.

- 20. Timms DJ. The effect of rapid maxillary expansion on nasal airway resistance. British Journal of Orthodontics 1986;13(4):221-8.
- 21. Warren DW, Hairfield WM, Seaton D, Morr KE, and Smith LR. The relationship between nasal airway size and nasal-oral breathing.American Journal of Orthodontics and Dentofacial Orthopedics 1988;93(4):289-93.
- 22. Warren DW, Hershey GH, Turvey TA, Hinton VA, Hairfield WM. The nasal airway following maxillary expansion. American Journal of Orthodontics and Dentofacial Orthopedics 1987;91(2):111-6.
- 23. Wertz RA. Changes in nasal airflow incident to rapid maxillary expansion. Angle Orthodontist 1968;38(1):1-11.
- 24. Wertz RA. Skeletal and dental changes accompanying rapid midpalatal suture opening. American Journal of Orthodontics 1970;58(1):41-66.
- 25. McNamara JA. Orthodontics and Dentofacial Orthopedics, Needham Press, Ann Arbor, Mich, USA, 2001.
- 26. Jacobson A and Jacobson RL. Radiographic Cephalometry: From Basics to 3-d Imaging, Quintessence Publishing, Chicago, Ill, USA, 2006.
- 27. Schellenberg JB, Maislin G and Schwab RJ. Physical findings and the risk for obstructive sleep apnea. The importance of oropharyngeal structures Am J RespirCrit Care Med. 2000 Aug; 162(2 Pt 1):740-8.
- 28. Cistulli PA. Craniofacial abnormalities in obstructive sleep apnoea: implications for treatment, Respirology 1996;1(3):164-74.
- 29. Riley R, Guilleminault C, Herran J, and Powell N. Cephalometric analyses and flowvolume loops in obstructive sleep apnea patients, Sleep 1983;6(4):303-11.
- 30. Subtelny JD. Width of the nasopharynx and related anatomic structures in normal and unoperated cleft palate children, American

Journal of Orthodontics 1955;41(12):889-909.

- 31. Compadretti GC, Tasca I, and Bonetti. Nasal airway measurements in children treated by rapid maxillary expansion, American Journal of Rhinology 2006;20(4):385-93.
- 32. Doruk C, S"ok"uc"u O, Sezer H, and Canbay EI. Evaluation of nasal airway resistance during rapid maxillary expansion using acoustic rhinometry, European Journal of Orthodontics 2004;26(4):379-401.
- 33. Enoki C, Valera FCP, Lessa FCR, Elias AM, Matsumoto MAN and W T Anselmo-Lima. Effect of rapid maxillary expansion on the dimension of the nasal cavity and on nasal air resistance, International Journal of Pediatric Otorhinolaryngology 2006;70(7):1225-30.
- 34. Lee SH, Choi JH, Shin C, Lee HM, and Kwon SY. How does open-mouth breathing influence upper airway anatomy? Laryngoscope 2007;117(6):1102-6.
- 35. Paul JL and Nanda RS. Effect of mouth breathing on dental occlusion, Angle Orthodontist 1973;43(2):201-6.
- 36. Linder Aronson S. and Leighton BC. A longitudinal study of the development of the posterior nasopharyngeal wall between 3 and 16 years of age, European Journal of Orthodontics 1983;5(1):47-58.
- 37. Brodie AG. On the growth pattern of the human head. From the third month to the eighth year of life, American Journal of Anatomy 1941;68(2):209-62.
- 38. King EW. A roentgenographic study of pharyngeal growth, Angle Orthod 1952;22(1):23-37.
- Bergland O. The bony nasopharynx. A roentgencraniometric study, Acta Odontologica Scandinavica 1963;21(suppl 35):31-137.
- 40. Tsai HH. Developmental changes of pharyngeal airway structures from young to adult persons, Journal of Clinical Pediatric Dentistry 2007;31(3):219-21.

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