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What changes in maxillary morphology from distraction osteogenesis maxillary expansion (DOME) correlate with subjective and objective OSA measures?

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Abstract

Objectives To correlate skeletal and airway measures on imaging with polysomnographic and self-reported measures after distraction osteogenesis maxillary expansion (DOME), in the effort to identify clinically relevant sites of expansion to guide treatment for adult patients with obstructive sleep apnea (OSA).

Materials and methods This is a retrospective study reviewing subjects who underwent DOME and had the complete set of the following data: peri-treatment cone-beam computed tomography (CBCT) scans, polysomnography (PSG), Epworth Sleepiness Scale (ESS), and nasal obstruction symptom (NOSE) scores.

Results Of 132 subjects who underwent DOME, 35 met inclusion criteria (71% men, mean age 27.7 ± 6.5 years, mean BMI 26.0 ± 6.4 kg/m^2) and were enrolled in the study. There was a significant reduction in the NOSE score from 11.4 ± 5.5 to 3.6 ± 3.1, in the ESS score from 12.0 ± 4.6 to 7.1 ± 4.7, and in the apnea–hypopnea index (AHI) from 17.1 ± 15.8 to 7.01 ± 6.2 ($p < 0.0001$), after DOME. Nasal floor width at the nasopalatine canal level showed a statistically significant correlation with AHI reduction ($p < .0001$).

Conclusions DOME is significantly associated with reduction of nasal obstruction, sleepiness, and severity of OSA. The findings suggest that expansion at the anterior third of the bony nasal passage, specifically where the nasopalatine canal is located predicts its clinical efficacy. This site may be a useful target anatomically via imaging.

Keywords Obstructive sleep apnea · Sleep medicine · Palatal expansion · Distraction osteogenesis maxillary expansion

Introduction

Functional maxillofacial and oropharyngeal anatomic interactions are critical to the pathophysiology of obstructive sleep apnea (OSA) [1]. Transverse maxillary hypoplasia with a high arch palate is associated with high nasal airway resistance and decreased volume for the tongue, leading to one of the phenotypes of patients with OSA [2].

Transverse maxillary expansion has been shown to reduce OSA severity. Rapid expansion increases nasal cavity volume, decreases nasal airflow resistance, and increases space for the tongue to protrude forward and upward. As a result, this stabilizes the posterior pharyngeal airway space against collapse during sleep [3].

With the advent of temporary anchorage devices (TAD), clinicians have been able to achieve more skeletal expansion and avoid dentoalveolar tipping, root resorption, periodontal defect, and relapse. There are reports of successful mini screw assisted rapid palatal expanders that overcome

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skeletal resistance without surgery, particularly in teenagers and young adults [4]. However, the results are not consistent in older adults with OSA.

Distraction osteogenesis maxillary expansion (DOME) was developed as a patient-specific surgical-orthodontic protocol for reliable, consistent, and effective maxillary expansion without the uncertainty and adverse effects of mini-implant–assisted rapid palatal expansion (MARPE) or surgically assisted rapid palatal expansion (SARPE) [5]. DOME combines a mini-implant–assisted palatal expander and minimally invasive maxillary osteotomy and has been shown to significantly reduce nasal obstruction and severity of OSA [6].

OSA is a complex condition with multifactorial etiology including alterations in critical pharyngeal closing pressure (Pcrit), loop gain, muscle responsiveness, and arousal index [7, 8]. Interventions that involve physical change such as DOME affect the Pcrit, and hence it is unlikely that anatomy alone is adequate to explain changes in the apnea–hypopnea index (AHI). Nevertheless, our treating clinicians do need general guidelines as to the range and location of maxillary expansion that correlate with treatment success. As actual anatomic studies have not been performed to provide useful clinical measures for maxillary expansion, this study aims to examine correlations between location and amount of expansion, with objective and subjective improvement in nasal breathing and OSA. We aimed to find clinical useful guidelines to help direct treating clinicians.

Materials and methods

Subjects

This is a retrospective study of subjects enrolled from September 2014 through October 2019, who underwent DOME. Subjects who met the following inclusion criteria were enrolled: (1) complete records including 3-D cone-beam computed tomography (CBCT), polysomnography, Epworth Sleepiness Scale (ESS), and nasal obstruction symptom (NOSE) score for both timing pre- and post-DOME, (2) greater than 6 mm of activation at the jackscrew level, and (3) no nasal surgery or other OSA treatments that might influence DOME outcome during the expansion period.

DOME protocol (Fig. 1)

Using CBCT, hybrid (bone-borne and tooth-borne)-type mini-implants assisted rapid maxillary expander was individually fabricated per subject. The thickness of palatal bone was used to identify optimal implant position and length. Mini-implants were placed with bi-cortical engagement of the palatal roof as close to the midpalate suture as possible, given that sufficient bone thickness was present [9] (Fig. 1A).

Conventional DOME surgical technique includes limited LeFort I osteotomy that may or may not require fracture of the pterygoid plates. A full description of the surgical and expansion procedures is well documented elsewhere [5, 6, 12]. The expander screw is turned up to 2 mm of maxillary separation to validate the successful MARPE placement and DOME procedure at the end of the procedure (Fig. 1B). After 7–10 days of the latency period, the expander device is activated 0.25 mm daily up to 6–10 mm of maxillary expansion, on average (Fig. 1C). The spaces created by DOME are closed by orthodontic treatment.

Sleep data collection

Subjects underwent attended polysomnography (PSG) 3 to 8 months after DOME according to the standards of the American Academy of Sleep Medicine [6]. Epworth Sleepiness Scale (ESS) and nasal obstruction and septoplasty effectiveness (NOSE) scale questionnaires were completed before the DOME procedure and 3 months after expansion.

CBCT protocol

CBCT scans were taken with a 23 cm × 17-cm field of view (FOV) and 0.3-mm voxel size level before and after DOME (average 6 weeks after surgery) (i-CAT; Hatfield, PA, USA). During the scan, subjects were instructed to maintain head position, avoid swallowing, and remain in centric occlusion with relaxed tongue and lip positions at end-expiration.

Data were imported to InViVoDental 6 (Anatomage, San Jose, CA, USA) for deidentified analysis. CBCT scans were oriented as follows: (1) nasion was chosen as the origin defined as the (0,0,0) coordinate and (2) the axial plane was defined with right orbitale, left orbitale, and right porion and (3) the midsagittal reference plane was derived by nasion and basion (Fig. 2A).

Skeletal and dental landmarks were marked using the 3D Analysis function of Nvivo software. 3D volume images were created, and widths were measured at multiple anterior–posterior sections to evaluate structural changes after DOME. All measurements were independently performed by 2 raters, where the mean measurements were used. It was impossible to blind pre and post DOME as the appliance and LeFort osteotomies could not be concealed on images (Fig. 2B).

For airway analysis, the skulls on the scans were oriented to a modified Frankfort horizontal (FH) by defining the axial plane at three points: right porion, right orbitale, and left orbitale. The skulls were oriented on the right sagittal view, the frontal view, and the transverse. The airway was divided into three regions: nasopharynx (superior to the palatal plane), upper oropharynx (between the palatal plane and the base of the soft palate at the most anterior point), and lower

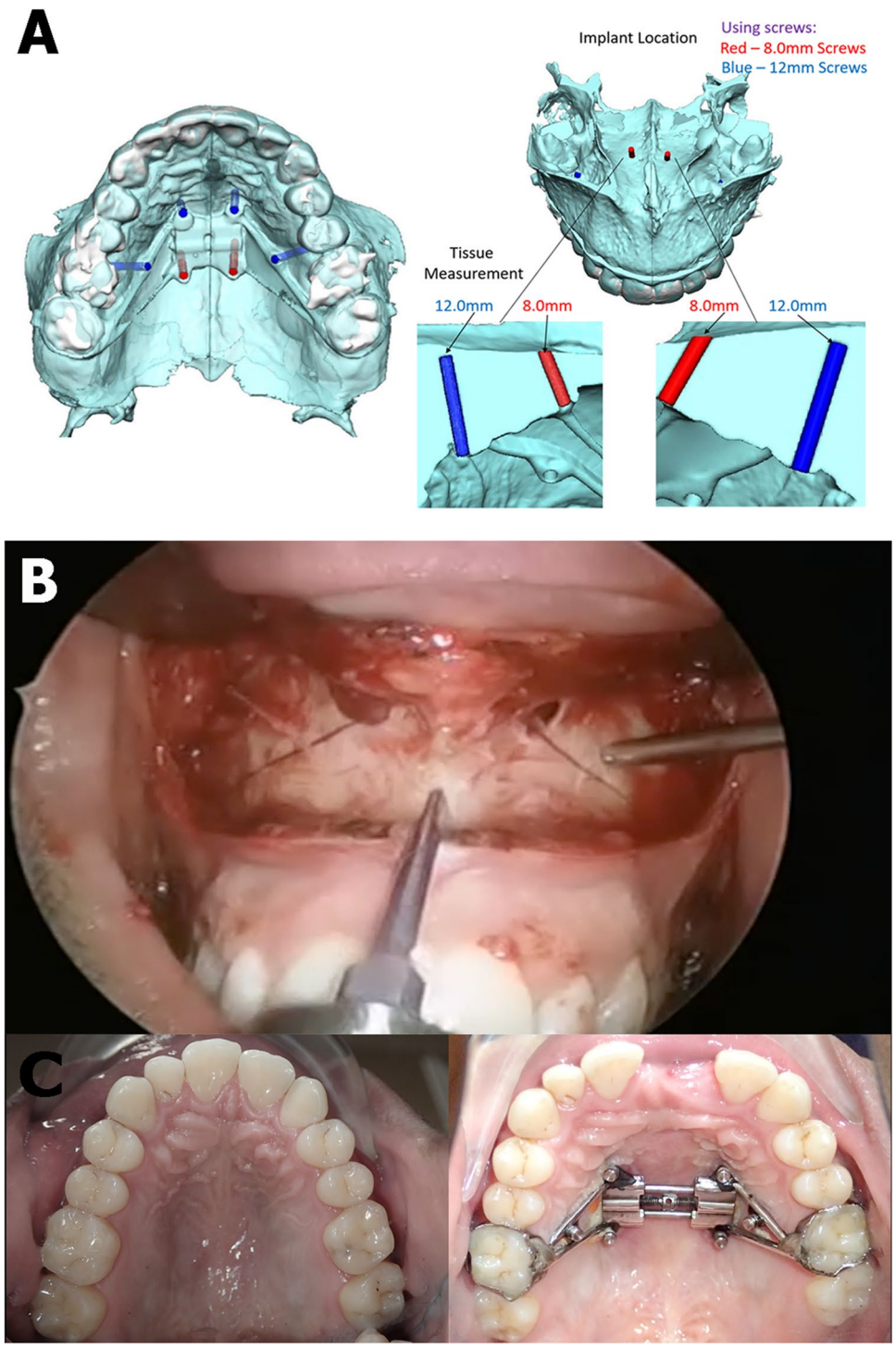

Fig. 1 **A** Custom-fabricated hybrid type mini-implant assisted maxillary expander is designed. Ideal location of palate and mini-implant length to get bi-cortical engagement of palate are estimated. **B** Limited Le-Fort I and mid-sagittal split without downfracture. **C** Pre-DOME occlusal view (left) and post-DOME occlusal view (right)

oropharynx (between the base of the soft palate and the base of the epiglottis). Airway segmentation threshold values were adjusted to eliminate imaging artifacts and were held constantly around 400 relative Hounsfield units. The minimal cross-sectional area (MCA) of the airway was calculated in square millimeters for each of the three regions using the airway function of Nvivo software as published previously. Minimal cross-sectional area (MCA) was measured for the nasopharynx, the upper oropharynx, and the lower oropharynx) to evaluate airway changes after DOME (Fig. 2C). Table 1 summarizes the variables defined for this study.

Statistical analysis

Inter-rater reliability was measured by intraclass correlation coefficient (ICC), Cronbach's alpha. Continuous

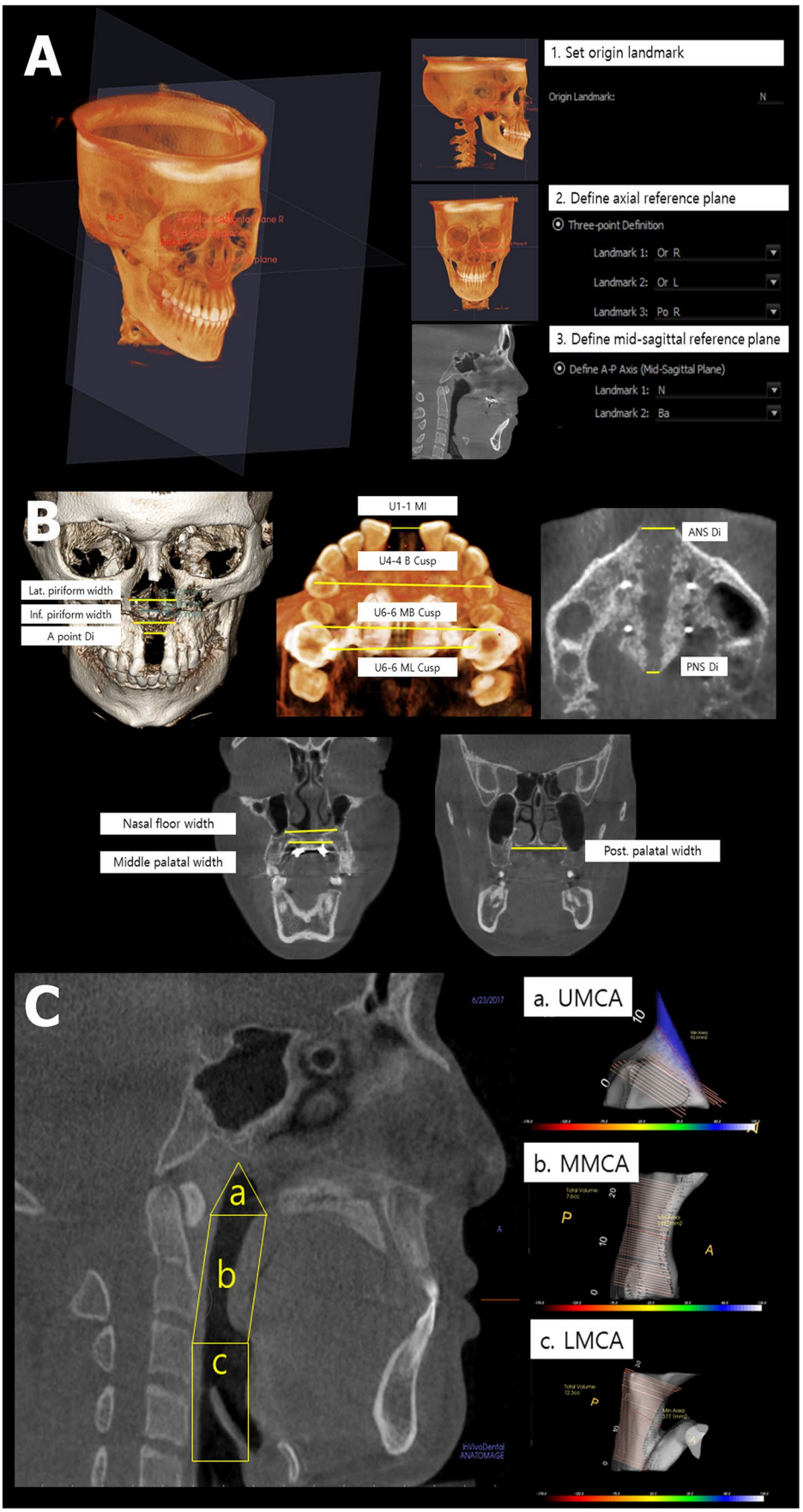

Fig. 2 **A** Reorientation of CBCT scans: All CBCT scans were oriented as follows: (1) nasion was chosen as the origin landmark which is described as the (0,0,0) coordinate; (2) the axial plane was defined with three different landmarks. In this study, right orbitale, left orbitale, and right porion were used. (3) Finally, the midsagittal reference plane was derived by two landmarks: nasion and basion. **B** Three-dimensional dental and skeletal measurements and analysis. **C** Regional airway measurement: measurements of the minimal cross-sectional areas (MCAs) of the pharynx. a MCA of the nasopharynx (UMCA), b MCA of the upper oropharynx (MMCA), c MCA of the lower oropharynx (LMCA)

Table 1 Airway, skeletal, and dental measurements

Measurements	Definition
Airway analysis	
UMCA, cm^3	Minimal cross-sectional area of the nasopharynx
MMCA, cm3	Minimal cross-sectional area of the upper oropharynx
LMCA, cm3	Minimal cross-sectional area of the lower oropharynx
Skeletal analysis	
ANS Di, mm	Distance between right and left ANS
PNS Di, mm	Distance between right and left PNS
A point Di, mm	Distance between right and left A point
Lat. piriform width, mm	Nasal cavity width at the external surface of the lateral piriform aperture level
Inf. piriform width, mm	Nasal cavity width at the external surface of the inferior piriform aperture level
Nasal floor width, mm	Nasal floor width at the nasopalatine canal closure
Post. nasal floor width, mm	Nasal floor width at the descending palatine opening level
Middle palatal width, mm	Palatal width at the nasopalatine canal closure
Post. Palatal Width, mm	Palatal width at the descending palatine opening level
Dental analysis	
U6-6 MB Cusp, mm	Distance between right and left mesial buccal cusp of U6
U6-6 ML Cusp, mm	Distance between right and left mesial lingual cusp of U6
U6-6 APEX, mm	Distance between right and left mesial lingual root apex of U6
U1-1 MI, mm	Distance between right and left mesioincisal angle of U1
U4-4 B Cusp, mm	Distance between right and left buccal cusp of U4

variables were expressed as the mean with standard deviation (SD). Categorical variables were expressed as numbers with percent. Normal distribution of variables assessed by the Shapiro Wilks test. Paired sample *t* test and Wilcoxon signed rank test were used to evaluate parameters before and after expansion. Correlations were assessed by Spearman correlation analysis. Logistic regression was used to examine the association of structural change and outcome,

For all statistical analysis, the results were considered significant at $p < 0.05$. Data analysis was performed using SPSS for Windows (version 24) and the language R (version 3.6.1).

Results

Of 132 subjects who underwent DOME, a total of 35 subjects met inclusion criteria and were selected for analysis, with 25 males (71%) and 10 females (29%). Baseline preoperative age was 27.7 ± 6.5 years, height 177.7 ± 9.1 cm, weight 81.7 ± 20.5 kg, and BMI 26.0 ± 6.4 kg/m^2 (mean ± SD).

Skeletal, dental airway changes (Table 2)

Maxillary expansion measured at anterior nasal spine (ANS) and posterior nasal spine (PNS) was 8.8 ± 3.3 mm and 3.1 ± 1.3 mm, respectively ($p < 0.0001$). The mean ANS expansion was 3 times greater than mean PNS expansion. Palatal width at the CT slice showing nasopalatine canal closure increased 5.0 ± 2.6 mm. Palatal width at the CT slice showing opening to descending palatine arteries increased by 2.0 ± 1.1 mm. Intermolar width increased 8.1 ± 1.7 mm at the intermesiobuccal cusp and 7.5 ± 2.3 mm at mesiolingual cusp ($p < 0.0001$). Inter-premolar width increased by 7.2 ± 2.3 mm ($p < 0.0001$).

Nasal floor width increased by 5.5 ± 2.4 mm at the piriform increased, 4.4 ± 2.1 mm at the nasopalatine canal closure, and 1.3 ± 1.8 mm at the descending palatine opening.

Airway analysis showed increase in the minimum cross-sectional areas (MCA) of the airway after DOME. Upper oropharynx MCA (MMCA) increased significantly after expansion ($p < 0.001$).

Questionnaires and sleep study outcomes

Perioperative apnea–hypopnea index (AHI), Epworth Sleepiness Scale (ESS), and nasal obstruction symptom evaluation (NOSE) showed significant differences after DOME ($p < 0.0001$). (Table 3).

Correlation between sleep parameters and anatomy

Nasal floor width changes showed statistically significant correlations with reduction in AHI. This is especially pronounced at the level of nasopalatine canal closure ($p < 0.0001$) (Table 4, Fig. 3).

Table 2 Airway, skeletal, and dental dimension and difference after DOME; pre-DOME (T1), post-DOME (T2), and changes (T2-T1)

Measurements	T1	T2	T2-T1	*p* value
Airway analysis				
UMCA, cm^3	279.9 ± 110.8	300.2 ± 122.8	20.3 ± 76.8	NS
MMCA, cm3	167.7 ± 99.8	191.6 ± 101.0	23.9 ± 35.6	< 0.001
LMCA, cm3	152.2 ± 89.8	181.6 ± 113.8	29.4 ± 60.2	< 0.01
Skeletal analysis				
ANS Di, mm	0	8.8 ± 3.3	8.8 ± 3.3	< 0.0001
PNS Di, mm	0	3.1 ± 1.26	3.1 ± 1.26	< 0.0001
PNS Di/ANS Di, ratio	-	-	0.4 ± 0.2	-
A point Di, mm	0.2 ± 0.2	9.4 ± 3.1	9.2 ± 3.1	< 0.0001
Lat. piriform width, mm	20.7 ± 2.6	20.9 ± 2.8	0.2 ± 1.6	NS
Inf. piriform width, mm	15.8 ± 2.6	21.4 ± 3.1	5.5 ± 2.4	< .0001
Nasal floor width, mm	18.2 ± 3.6	22.6 ± 4.3	4.4 ± 2.1	< .0001
Post. nasal floor width, mm	25.4 ± 2.6	26.6 ± 2.7	1.3 ± 1.8	< .0001
Middle palatal width, mm	18.5 ± 3.1	23.5 ± 3.2	5.0 ± 2.6	< .0001
Post. palatal width, mm	33.0 ± 2.7	35.0 ± 2.7	2.0 ± 1.	< .0001
Dental analysis				
U6-6 MB Cusp, mm	50.5 ± 5.0	58.6 ± 4.5	8.1 ± 1.7	< 0.0001
U6-6 ML Cusp, mm	38.9 ± 4.8	46.4 ± 3.8	7.5 ± 2.3	< 0.0001
U6-6 Apex, mm	49.6 ± 5.7	54.8 ± 5.6	5.3 ± 1.7	< 0.0001
U1-1 MI, mm	1.5 ± 0.7	8.8 ± 2.4	7.3 ± 2.7	< 0.0001
U4-4 B Cusp, mm	40.5 ± 4.2	47.7 ± 4.9	7.2 ± 2.3	< 0.0001

Data presented as mean ± standard deviation

p value, paired *t* test or Wilcoxon signed rank test between pretreatment (T1) and posttreatment (T2)

Table 3 Questionnaires and sleep study outcomes

	T1	T2	T2-T1	*p* value
ESS	12.0 ± 4.6	7.1 ± 4.7	−4.9 ± 3.3	< 0.0001
NOSE	11.4 ± 5.5	3.6 ± 3.1	−7.9 ± 4.8	< 0.0001
AHI	17.0 ± 15.8	7.0 ± 6.2	−10.0 ± 11.4	< 0.0001
% improvement ESS	-	-	43.3 ± 26.9	
% improvement NOSE	-	-	65.5 ± 34.0	
% improvement AHI	-	-	54.4 ± 25.6	

Data presented as mean ± standard deviation; *p* value, paired *t* test or Wilcoxon signed rank test between pretreatment (T1) and posttreatment (T2)

Discussion

Transverse maxillary hypoplasia has been recognized as one of the key anatomic contributors to OSA [2]. Many studies have shown maxillary expansion to be effective for pediatric OSA [10, 11] as related to its effect on the internal nasal valve [12]. However, there is limited published data for treatment of adults with OSA and transverse maxillary hypoplasia [13]. Furthermore, there is a lack of understanding as to where along the nasal passage does expansion make the most impact. There is also no clinically driven data suggesting how much expansion is generally needed for treatment success.

Although maxillary expansion started over a century ago, its application in adults with the aid of osteotomies was not reported till 1938 [14, 15]. The classic descriptions of surgically assisted palatal expansion (SARPE) involve aggressive LeFort I level osteotomies, lateral nasal wall osteotomies, and separation of the pterygomaxillary separation [16]. This can result in complications including but not limited to significant epistaxis, nonunion, and relapse [17–19]. DOME is less invasive as it mainly targets nasal floor expansion, particularly at the level of the internal nasal valve [20]. The use of mini-implant expanders further promote skeletal expansion. With virtual surgical planning and endoscopic guidance with use of osteotomy

Table 4 Correlation between AHI change and airway, skeletal, and dental changes after DOME

Measurements	AHI change	
	r_s	*p* value
Airway analysis		
UMCA, cm^3	0.19	NS
MMCA, cm3	−0.20	NS
LMCA, cm3	0.02	NS
Skeletal analysis		
ANS Di, mm	−0.38	0.03
PNS Di, mm	−0.29	NS
PNS Di/ANS Di, mm	0.16	NS
A point Di, mm	−0.37	0.03
Lat. piriform width, mm	0.19	NS
Inf. piriform width, mm	−0.38	0.03
Nasal floor width, mm	−0.69	< .0001
Post. nasal floor width, mm	−0.42	0.01
Middle palatal width, mm	-0.41	0.01
Post. palatal width, mm	0.18	NS
Dental analysis		
U6-6 MB Cusp, mm	−0.17	NS
U6-6 ML Cusp, mm	−0.18	NS
U6-6 Apex, mm	0.01	NS
U1-1 MI, mm	−0.36	0.05
U4-4 B Cusp, mm	−0.35	0.05

r_s, spearman correlation coefficient; *p* value, spearman correlation between AHI change and structural changes

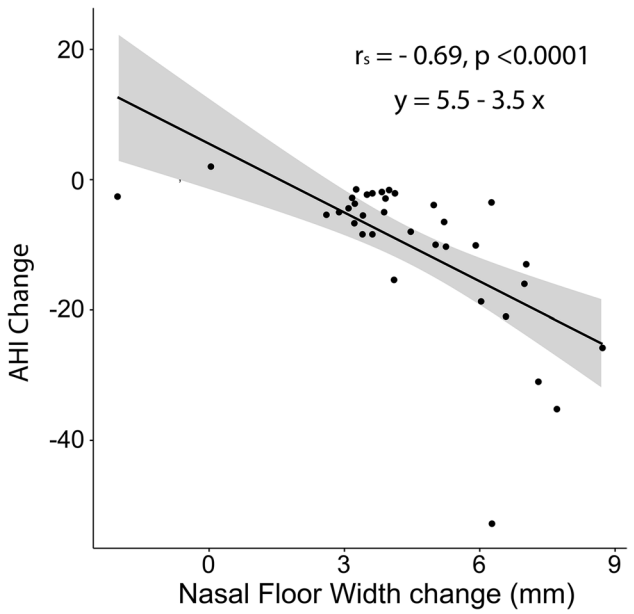

Fig. 3 Nasal floor width change and AHI change showed statistically significant correlation (R = −0.69, $p < .0001$)

guides, surgical treatment has become even less invasive and more patient-specific [21].

In this study we aimed to find the area of expansion that correlates with clinical outcomes. The hypothesized and measured targets included the ANS, the PNS, interpremolar and molar widths, and the nasopalatine canal. With regard to the nasopalatine canal, this had been previously used as a close surrogate to the location of the internal nasal valve [22]. These points were chosen for both consistency of measurement via imaging, as well as their potential in clinical utility. Specifically, these measurements were used to assess the efficacy of DOME on nasal breathing and OSA via patient self-reported measures and attended polysomnography. We found that the nasal floor width at the point of nasopalatine canal closure correlated significantly with AHI reduction, as well as subjective decrease in nasal obstruction symptoms and daytime sleepiness.

There have been contradictory results on nasal surgery and its effect on OSA. For patients with nasal obstruction and a high-arched palate and maxillary transverse hypoplasia, intranasal surgery as septoplasty, inferior turbinate reduction, and nasal vale stabilization tend to fail in improving obstructive symptoms [22]. DOME has shown to be an effective procedure for patients with this particular anatomic phenotype [5]. We previously demonstrated the effect of DOME on nasal breathing using validated internal nasal valve measures suggesting proof of its efficacy [20].

Here, we summarize some of the ways in which nasal obstruction affects the pathophysiology of OSA. Using the Starling resistor model, the upper airway can be viewed as a tube with a narrow proximal inlet corresponding to the internal nasal valve, and a more distal collapsible segment, which corresponds to the oro- and hypopharynx. A proximal obstruction at the level of the nose will generate more negative pressure distally at the level of the pharynx, resulting in airway collapse in OSA [23].

Nitric oxide (NO) is a potent pulmonary vasodilator that increases ventilation-perfusion ratio in the lung. It is produced in significant quantities in the nose and the paranasal sinuses [24]. Additionally, NO maintains upper airway dilator muscles tone, regulation of spontaneous respiration, and neuromuscular control during sleep [25]. Mouth breathing due to the narrow palatal vault as well as narrow nasal passages further reduces nasal breathing, and NO production, during sleep.

Inhibition of the nasal-ventilator reflex by bypassing the nose can also increase central and obstructive apneic episodes and decrease the minute and spontaneous ventilation [26]. Mouth breathing during sleep increases upper airway resistance up to 2.5 times when compared to nasal breathing [27]. This exacerbates an anatomic reduction of volume in retroglossal and retropalatal regions in OSA [28].

This study correlates skeletal and airway measurements with patient self-reported nasal obstructive symptoms, sleepiness, and objective polysomnography parameters including the AHI. Our findings suggest that in the entirety of maxillary expansion that is achieved with DOME, the amount of expansion at the anterior third of the bony nasal passage where the nasopalatine canal is located has clinical significance. This has two points of clinical significance. For one, it suggests to our surgeons and orthodontists that positive treatment results from maxillary expansion is associated with the narrow floor at the internal nasal valve. Furthermore, as both orthodontists and surgeons seek less invasive ways to promote maxillary expansion, the focus should be directed to the anterior third of the bony nasal passage in patients with OSA who have a narrow, high-arched palate.

Key limitations of this study are as follows: (1) CBCT is taken in an upright position, and in the awake state. However, taking the scans in an upright position mirrors how CBCT scans are acquired in a dental office making translation of our results practical for screening of dental patients, and (2) CBCT scans were taken before removal of the expander which may cause partial reduction in oral cavity volume. This may have masked the effect on pharyngeal measures.

Conclusions

DOME may be an effective treatment to widen the nasal floor and palatal vault in selected adult patients with OSA presenting with transverse maxillary hypoplasia. Specifically, the findings of this study suggest that expansion of the anterior one-third of the bony nasal passage is associated with success in treating nasal obstruction (subjective) and reduction of AHI (objective). For clinicians, this region of impact can be seen near the nasopalatine canal via CBCT, and physiologically, it is near the internal nasal valve.

Data availability The datasets generated during and/or analyzed during the current study are available from the corresponding author on reasonable request.

Declarations

Ethical approval This study was approved by the Institutional Review Board of Stanford University (Protocol 36385, IRB-58401) and of the University of Pacific (IRB2020-74).

Informed consent For this type of retrospective studies, formal consent is not required. All authors have seen and approved the manuscript.

Conflict of interest The authors declare no competing interests.

References

1. Cistulli PA (1996) Craniofacial abnormalities in obstructive sleep apnoea: implications for treatment. Respirology 1(3):167–174
2. Cistulli PA, Richards GN, Palmisano RG, Unger G, Berthon-Jones M, Sullivan CE (1996) Influence of maxillary constriction on nasal resistance and sleep apnea severity in patients with Marfan's syndrome. Chest 110(5):1184–1188
3. Iwasaki T, Saitoh I, Takemoto Y et al (2013) Tongue posture improvement and pharyngeal airway enlargement as secondary effects of rapid maxillary expansion: a cone-beam computed tomography study. Am J Orthod Dentofacial Orthop 143(2):235–245
4. Carlson C, Sung J, McComb RW, Machado AW, Moon W (2016) Microimplant-assisted rapid palatal expansion appliance to orthopedically correct transverse maxillary deficiency in an adult. Am J Orthod Dentofacial Orthop 149(5):716–728
5. Liu SY, Guilleminault C, Huon LK, Yoon A (2017) Distraction osteogenesis maxillary expansion (DOME) for adult obstructive sleep apnea patients with high arched palate. Otolaryngol Head Neck Surg 157(2):345–348
6. Yoon A, Guilleminault C, Zaghi S, Liu SY (2020) Distraction osteogenesis maxillary expansion (DOME) for adult obstructive sleep apnea patients with narrow maxilla and nasal floor. Sleep Med 65:172–176
7. Eckert DJ (2018) Phenotypic approaches to obstructive sleep apnoea - New pathways for targeted therapy. Sleep Med Rev 37:45–59
8. Eckert DJ, White DP, Jordan AS, Malhotra A, Wellman A (2013) Defining phenotypic causes of obstructive sleep apnea. Identification of novel therapeutic targets. Am J Respir Crit Care Med 188(8):996–1004
9. Lee RJ, Moon W, Hong C (2017) Effects of monocortical and bicortical mini-implant anchorage on bone-borne palatal expansion using finite element analysis. Am J Orthod Dentofacial Orthop 151(5):887–897
10. Camacho M, Chang ET, Song SA et al (2017) Rapid maxillary expansion for pediatric obstructive sleep apnea: a systematic review and meta-analysis. Laryngoscope 127(7):1712–1719
11. Vale F, Albergaria M, Carrilho E et al (2017) Efficacy of rapid maxillary expansion in the treatment of obstructive sleep apnea syndrome: a systematic review with meta-analysis. J Evid Based Dent Pract 17(3):159–168
12. Yoon A, Abdelwahab M, Liu S, Oh J, Suh H, Trieu M, Kang K, Silva D (2021) Impact of rapid palatal expansion on the internal nasal valve and obstructive nasal symptoms in children. Sleep Breath 25(2):1019–1027
13. Awad M, Gouveia C, Zaghi S, Camacho M, Liu SY (2019) Changing practice: Trends in skeletal surgery for obstructive sleep apnea. J Craniomaxillofac Surg 47(8):1185–1189
14. Timms DJ (1999) The dawn of rapid maxillary expansion. Angle Orthod 69(3):247–250
15. Koudstaal MJ, Poort LJ, van der Wal KG, Wolvius EB, Prahl-Andersen B, Schulten AJ (2005) Surgically assisted rapid maxillary expansion (SARME): a review of the literature. Int J Oral Maxillofac Surg 34(7):709–714
16. Machado Junior AJ, Crespo AN (2020) Expansion of the maxilla in adults with OSAS: myth or reality? Sleep Med 65:170–171
17. Dergin G, Aktop S, Varol A, Ugurlu F, Garip H (2015) Complications related to surgically assisted rapid palatal expansion. Oral Surg Oral Med Oral Pathol Oral Radiol 119(6):601–607
18. Williams BJ, Currimbhoy S, Silva A, O'Ryan FS (2012) Complications following surgically assisted rapid palatal expansion: a retrospective cohort study. J Oral Maxillofac Surg 70(10):2394–2402
19. Cakarer S, Keskin B, Isler SC, Cansiz E, Uzun A, Keskin C (2017) Complications associated with surgically assisted rapid palatal

expansion without pterygomaxillary separation. J Stomatol Oral Maxillofac Surg 118(5):279–282
20. Abdelwahab M, Yoon A, Okland T, Poomkonsarn S, Gouveia C, Liu SY (2019) Impact of distraction osteogenesis maxillary expansion on the internal nasal valve in obstructive sleep apnea. Otolaryngol Head Neck Surg 161(2):362–367
21. Liu SY, Ibrahim B, Abdelwahab M, Chou C, Capasso R, Yoon A (2022) A minimally invasive nasal endoscopic approach to distraction osteogenesis maxillary expansion to restore nasal breathing for adults with narrow maxilla. Facial Plast Surg Aesthet Med 24(6):417–421
22. Williams R, Patel V, Chen YF et al (2019) The upper airway nasal complex: structural contribution to persistent nasal obstruction. Otolaryngol Head Neck Surg 161(1):171–177
23. Georgalas C (2011) The role of the nose in snoring and obstructive sleep apnoea: an update. Eur Arch Otorhinolaryngol 268(9):1365–1373
24. Haight JS, Djupesland PG (2003) Nitric oxide (NO) and obstructive sleep apnea (OSA). Sleep Breath 7(2):53–62
25. Lundberg J (1996) Airborne nitric oxide: inflammatory marker and aerocrine messenger in man. Acta Physiol Scand 157(S633):4–27
26. McNicholas WT, Coffey M, Boyle T (1993) Effects of nasal airflow on breathing during sleep in normal humans. Am Rev Respir Dis 147(3):620–623
27. Fitzpatrick MF, McLean H, Urton AM, Tan A, O'Donnell D, Driver HS (2003) Effect of nasal or oral breathing route on upper airway resistance during sleep. Eur Respir J 22(5):827–832
28. Alves M Jr, Baratieri C, Nojima LI, Nojima MC, Ruellas AC (2011) Three-dimensional assessment of pharyngeal airway in nasal- and mouth-breathing children. Int J Pediatr Otorhinolaryngol 75(9):1195–1199

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