Original Article

Maxillary Morphology in Obstructive Sleep Apnea: A Cephalometric and Model Study

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Abstract: The relationship between maxillary constriction and the etiology of obstructive sleep apnea (OSA) is not clear. This prospective case-control study compared maxillary morphology in 94 dentate subjects (47 OSA and 47 control subjects), using upright lateral cephalograms and study models. Each subject had height, weight, and neck circumference measurements recorded and underwent an orthodontic examination. An upright lateral cephalogram and dental impressions were obtained. All data were analyzed using the SPSS statistical package applying nonparametric tests at the 5% level of significance. Male and female subjects were examined separately, and statistically significant differences were found between the cephalometric measurements for OSA and the control subjects. The palatal angle was more obtuse in male OSA subjects ($P < .05$). The PNS-posterior pharyngeal wall was shorter ($P < .05$) and the soft palate longer in female OSA subjects ($P < .05$). Minimum palatal airway widths were significantly reduced in both male ($P < .01$) and female ($P < .001$) subjects. In the comparison of study model measurements, palatal heights in OSA subjects were greater ($P < .05$). Thus, maxillary morphological differences do exist between OSA and control subjects, supporting their role as a etiological factor. (*Angle Orthod* 2004; 74:648–656.)

Key Words: Obstructive sleep apnea; Etiology; Maxilla

INTRODUCTION

Obstructive sleep apnea (OSA) is a potentially life-threatening disorder, estimated to affect 3.9% of men and 1.2% of women.[1] OSA is diagnosed using a combination of a sleep history, supporting questionnaires, a clinical examination of the upper airway, and overnight sleep monitoring. Snoring and excessive daytime sleepiness are the most common presenting complaints. The Epworth sleepiness scale (ESS) questionnaire elicits the likelihood of falling asleep in eight different situations.[2] Overnight polysomnography is considered the ''gold standard'' for the diagnosis of OSA.

Guilleminault et al[3] studied the relationship between maxillary constriction and the etiology of OSA and reported a familial tendency of narrow, high palates in the relatives of OSA patients. Cistulli and Sullivan[4] have shown a high prevalence of OSA among patients with Marfan's syndrome.

Continuous positive nasal airway pressure has traditionally been the choice of treatment for moderate to severe OSA.[5] Mandibular advancement splints are used in the management of subjects with mild to moderate OSA.[6] Recently, the use of rapid maxillary expansion (RME) as a treatment modality for OSA has been put forward.[6,7]

RME increases the width of the maxilla, reduces nasal resistance,[7] and increases the intranasal capacity. Hershey et al[8] reported a 45–55% reduction in nasal airway resistance, which was maintained after removal of the appliance. Wertz[9] concluded that RME cannot be justified for the purposes of increased nasal permeability unless there is a relative maxillary arch width deficiency and the obstruction lies in the lower portion of the nasal cavity. Cistulli et al[10] investigated the effect of RME as a treatment in six young adults who underwent RME and elective surgical assistance and in four who were treated with RME alone. In seven cases, the apnea-hypopnea index (AHI) was reduced to normal values, and the authors attributed this to an improvement in nasal airflow, tongue posture, and soft palate function.

The role of the maxilla in the etiology of OSA is not well described, and this deficiency needs to be addressed to develop evidence-based practice. The aims of this study were to evaluate the role of the maxilla in the etiology of OSA and to determine any differences between sexes.

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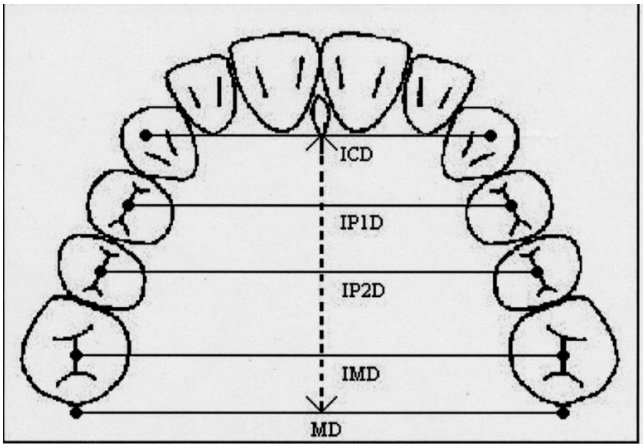

FIGURE 1. Diagram of the maxillary dental arch illustrating the linear measurements taken from study models. ICD, intercanine distance; IP1D, first interpremolar distance; IP2D, second interpremolar distance; IMD, intermolar distance; MD, molar depth.

MATERIALS AND METHODS

The test sample consisted of randomly selected Caucasian adults, referred from the Royal National Throat, Nose and Ear Hospital to the Royal London Dental Hospital (RLH). The control group consisted of Caucasian adults recruited from the Restorative Department waiting room of the RLH, accompanying parents or grandparents of patients attending the Orthodontic Department (RLH) and partners of the test group.

The inclusion criteria for the test group were over 18 years of age with a diagnosis of OSA confirmed by overnight polysomnography (AHI $>$ 5 per hour of sleep) and the presence of at least six teeth in the maxillary arch. The same criteria were applied to the control group except they had to have an ESS score $<$9 (exclude daytime sleepiness) and no reported snoring. The exclusion criteria for both groups were edentulous or partially dentate in the buccal segments, a history of orthodontic treatment, or a history of reconstructive/orthognathic surgery to the head and neck or pharyngeal surgery.

Ethical approval was obtained from the East London and City Health Authority research Ethics Committee. When all subjects were fully informed of the purpose and nature of the study, written consent was obtained.

Method

Data collection was divided into four sections:

1. Medical and sleep history including an ESS (Appendices 1 and 2).
2. Clinical examination with recordings of the subject's height, weight, and neck circumference. For each subject, their body mass index (BMI) was calculated (kg/m^2).[11]
3. Study cast analysis: measurements were taken from the maxillary cast using a pair of digital calipers (Mitutoyo Corporation, Kawasaki, Japan code number 500-321), with a resolution of 0.01 mm (Figures 1 and 2). The presence of a posterior transverse discrepancy was recorded as a buccal crossbite.[12]
4. Lateral cephalogram analysis: before the radiograph was taken, a thin layer of barium sulfate paste was applied to the dorsum of the tongue to enhance soft tissue identification. To standardize hyoid position, radiographs were exposed at the end of expiration, in the natural head position with the teeth in light occlusion.

A single operator traced the radiographs in a random order. The radiographs were orientated with the maxillary plane horizontal, and the landmarks used are shown in Figures 3 through 5. Each tracing was randomly digitized sequentially twice to a tolerance of 0.2 mm, using a GTCO Accutab digitizer (GTCO Corporation, Columbia, Md) and a customized software program. All data were converted to life-size using the known magnification for each radiograph. Linear, angular, and area measurements of the oropharynx, soft palate, and tongue were calculated.

Error study

Method error was calculated by repeat measurement of the 20 randomly selected models and cephalograms after

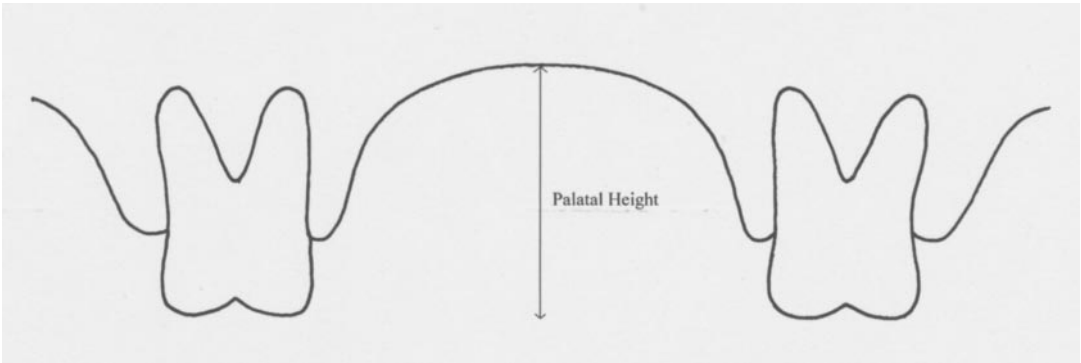

FIGURE 2. Diagram to show palatal height. Palatal heights are measured at the level of the canine, first premolar, second premolar, and first molar.

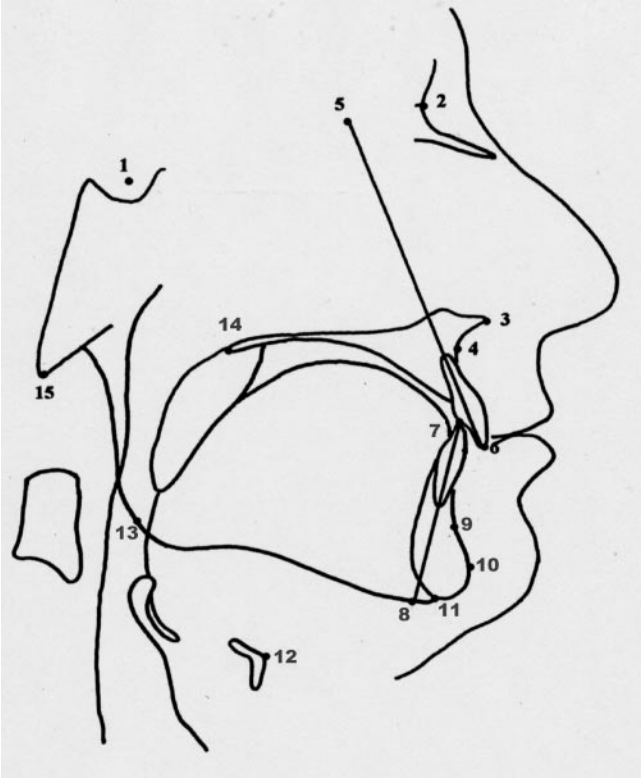

FIGURE 3. Standard cephalometric landmarks and measurements recorded. Except where listed below, points, lines, and planes conformed to British Standard definitions.[12] Points: 1, sella; 2, nasion; 3, ANS; 4, point A; 5, upper incisor apex projection to Frankfort horizontal; 6, upper incisor tip; 7, lower incisor tip; 8, lower incisor apex projection to the mandibular plane; 9, point B; 10, pogonion; 11, menton (point of intersection of lower mandibular border and symphyseal outline); 12, most anterior point on hyoid bone; 13, gonion; 14, PNS; 15, basion. Measurements: LAFH (menton to ANS); Sella-Nasion; UAFH (nasion to ANS); SNA; LPFH (gonion to PNS); Basion-PNS; UPFH (sella to PNS); ANS-PNS.

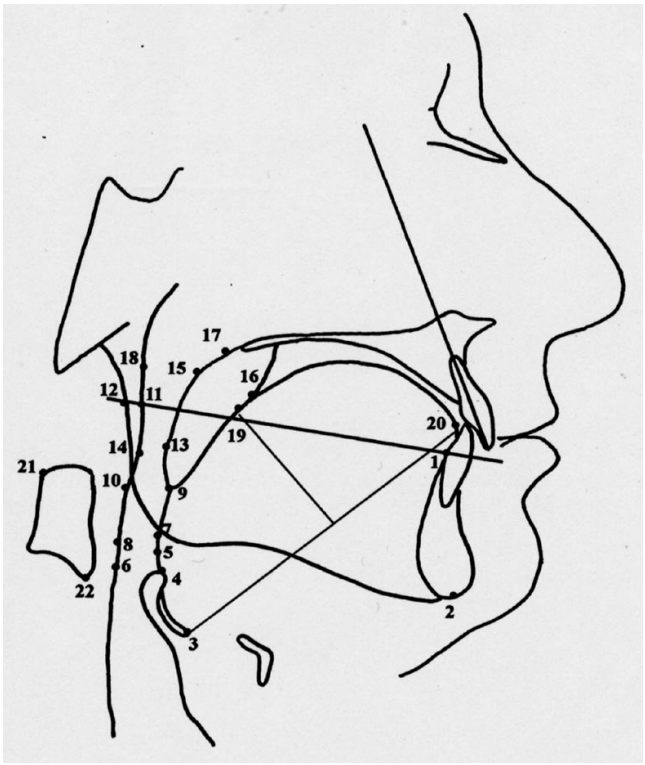

FIGURE 4. Oropharyngeal points and measurements. Points: 1, point of intersection of occlusal plane with lower incisor; 2, most inferior point on bony chin; 3, deepest point on vallecula; 4, tip of epiglottis; 5, most posterior point on tongue; 6, point on posterior pharyngeal wall (ppw) horizontally opposite 5; 7, point on tongue where postlingual airway is narrowest; 8, point on ppw where postlingual airway is narrowest; 9, tip of uvula; 10, point on ppw horizontally opposite 9; 11, point of intersection of occlusal plane with ppw; 12, point of intersection of occlusal plane with mandibular ramus; 13, point on soft palate where postpalatal airway is narrowest; 14, point on ppw horizontally opposite 13; 15, point on nasal surface where soft palate is at its thickest; 16, point on oral surface where soft palate is at its thickest; 17, most superior posterior point on soft palate; 18, point on ppw horizontally opposite 17; 19, point indicating tongue thickness (perpendicular to line from vallecula to tongue tip); 20, tip of tongue; 21, most superior posterior point of C2; 22, most inferior anterior point on C3. Measurements: soft palate thickness (distance between points 15 and 16); minimum palatal airway width (distance between points 13 and 14); palatal angle (ANS, PNS, uvula); PNS-posterior pharyngeal wall (distance between PNS and point 18); soft palate length (distance between PNS and point 9).

an interval of two weeks.[13] Random error was calculated using Dahlberg's equation[14] and by determining a reliability index.[13] Systematic error (bias) was assessed using the method of Houston.[13]

Statistical analysis

All data were analyzed using the SPSS statistical package (version 7.51 SPSS Inc., Chicago, Ill). Nonparametric tests were applied. For cephalometric and study model linear and angular measurements, the Mann-Whitney *U*-test was used, with statistical significance stated at the 5% level or less. For skeletal pattern and buccal crossbite parameters, the Fisher's exact test was used.

RESULTS

Error study

The model measurement error study produced an index of reliability generally greater than 93%. The error study revealed that 12 lateral cephalometric measurements had a Dahlberg value >1. Seven of these related to gonion, a point with a large envelope of error.[15] No systematic error (bias) was detected.

Demographic data

The male OSA and control subjects were well matched for age (Table 1), but the female OSA subjects were approximately seven years older than the control group (Table 2). All OSA subjects exhibited a higher BMI score, which was statistically significant ($P < .05$) in female subjects.

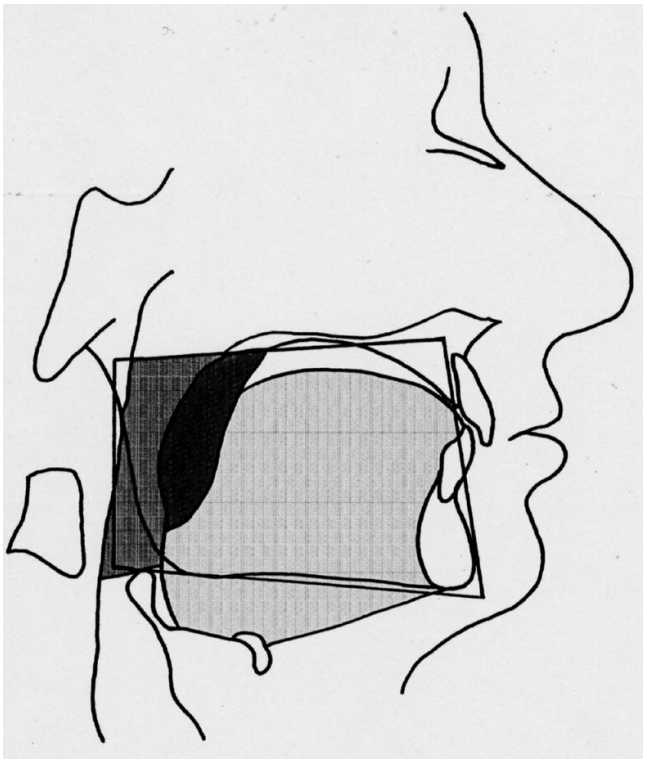

FIGURE 5. Oropharyngeal area and measurements recorded. Area measurements are displayed in three varying shades; tongue area in light gray, oropharyngeal area in mid-gray, and soft palate area in dark gray. Intermaxillary space area: delineated by a trapezium drawn through maxillary and mandibular planes, point 11, and point 1 (Figure 4). Soft palate area: outlined by the posterior pharyngeal wall (ppw), dorsal surface of the tongue, and soft palate; the superior boundary is a line parallel to the maxillary plane from PNS to ppw. A line parallel to this and tangential to the epiglottic tip forms the inferior boundary. Tongue area: delineated by the outline of the tongue within the oral cavity extending down to the vallecula, across to the anterior aspect of the hyoid bone and continuing to the most inferior aspect of the bony chin, then along the symphyseal outline to the tongue tip. Tongue proportion: tongue area as a percentage of the intermaxillary space area.

TABLE 1. Demographic Data for Male OSA and Control Subjects[a]

	OSA ($N = 34$)		Control ($N = 29$)		
	Median	Range	Median	Range	P Value[b]
Age (y)	47.2	23.8–59.6	48.7	27.8–61.5	NS
BMI (kg/m^2)	30	22.5–56.1	27.1	22–55.8	NS
Neck (cm)	42	38–45	38	32–41	***
ESS	12	3–18	5	2–9	***

[a] OSA indicates obstructive sleep apnea; NS, no statistically significant difference; ESS, Epworth sleepiness scale.
[b] Mann-Whitney statistical test.
*** $P < .001$.

Neck circumference, which reflects obesity, was statistically greater in both male ($P < .001$) and female ($P < .05$) subjects. The median ESS score for both male and female OSA subjects was statistically significant ($P < .001$), confirming that OSA subjects alone demonstrated excessive daytime sleepiness.

TABLE 2. Demographic Data for Female OSA and Control Subjects[a]

	OSA ($N = 13$)		Control ($N = 18$)		
	Median	Range	Median	Range	P Value[b]
Age (y)	53	27.8–65.7	46	29–58.7	NS
BMI (kg/m^2)	38	22–65	26.5	20–46.8	*
Neck (cm)	39	28–44	32	26–42	*
ESS	12	5–20	4.5	2–9	***

[a] OSA indicates obstructive sleep apnea; NS, no statistically significant difference; ESS, Epworth sleepiness scale.
[b] Mann-Whitney statistical test.
* $P < .05$; *** $P < .001$.

Cephalometric findings

The cephalometric findings are given in Tables 3 through 7.

Hard tissues. Measurements reflecting the anteroposterior (AP) and vertical skeletal dimensions of the maxilla showed the palatal angle to be more obtuse in the OSA group ($P < .05$), and the PNS-posterior pharyngeal wall distance significantly reduced in female OSA subjects (Figure 4, Tables 3–4).

Although not statistically significant, Sella-Nasion, SNA, and upper posterior facial height were reduced, whereas measurements for basion-PNS, upper anterior facial height, and palatal angle were all increased in the OSA subjects. No significant differences were found in the distribution of skeletal classification for OSA and control subjects (Table 5).

Soft tissues. In the OSA groups, the minimum palatal width was smaller both in men (five mm; $P < .01$) and in women (3.38 mm; $P < .001$). In female OSA subjects, the soft palate was longer (4.02 mm; $P < .05$), whereas in male subjects, the difference was not statistically significant. The thickness of the soft palate was marginally greater in OSA subjects than in controls; however, this did not reach statistical significance (Tables 6 and 7).

Study model measurements. In the OSA subjects, the palatal height was larger ($P < .05$) at the level of the first premolar, second premolar, and first molar (Figure 2). There were no significant differences in maxillary depth or intertooth distances (Tables 8 and 9).

Posterior transverse discrepancy. No statistically or clinically significant transverse dental differences were present between the groups (Table 10).

DISCUSSION

RME has been advocated recently as a treatment modality for OSA,[6,7] but few studies have been published investigating the relationship of the maxilla in OSA subjects. Therefore, this prospective case-control study was designed

TABLE 3. Skeletal Measurements in Male OSA and Control Subjects[a]

Measurement (mm or degree)	OSA ($N = 34$)		Control ($N = 29$)		P Value[b]
	Median	Range	Median	Range	
Sella-Nasion	67.8	58.95–75.11	68.3	61.86–75.03	NS
SNA	82.7	73.84–91.34	83.66	76.7–91.09	NS
Basion-PNS	44.98	36.1–59.21	43.04	34.81–52.97	NS
PNS-posterior pharyngeal wall	18.61	10.53–26.97	20.88	13.98–25.21	NS
ANS-PNS	52.26	43.4–60.11	52.4	46.76–57.03	NS
Upper posterior face height	46.74	40.4–52.47	47.21	42.25–54.17	NS
Upper anterior face height	52.08	41.78–58.5	51.33	40.2–56.12	NS
Palatal angle (ANS, PNS, uvula)	131.6	121.25–146.56	129.8	111.9–136.18	*

[a] OSA indicates obstructive sleep apnea; NS, no statistically significant difference.
[b] Mann-Whitney statistical test.
* $P < .05$.

TABLE 4. Skeletal Measurements in Female Subjects and Control[a]

Measurement (mm or degree)	OSA ($N = 13$)		Control ($N = 18$)		P Value[b]
	Median	Range	Median	Range	
Sella-Nasion	66.16	60.47–70.45	66.6	60.7–74.02	NS
SNA	80.25	75.35–85.75	81.92	72.94–88.12	NS
Basion-PNS	44.6	35.81–56.67	42.87	33.25–48.58	NS
PNS-posterior pharyngeal wall	17.34	14.26–24.8	20.02	12.81–26.06	*
ANS-PNS	47.76	44.86–58.33	43.62	38.21–52.3	NS
Upper posterior face height	43.66	36.44–49.21	44.31	35.21–48.19	NS
Upper anterior face height	48.12	38.82–51.48	47.54	43.72–51.14	NS
Palatal angle (ANS, PNS, uvula)	135.78	126.18–141.36	123.6	133.11–150.56	NS

[a] OSA indicates obstructive sleep apnea; NS, no statistically significant difference.
[b] Mann-Whitney statistical test.
* $P < .05$.

TABLE 5. Frequency Distribution of Skeletal Classifications II and III in OSA and Control Groups[a]

	OSA Group	Control Group	P Value[b]
Skeletal II	13	8	0.246
Percentage within group	56.5	38.1	NS
Skeletal III	10	13	
Percentage within group	43.5	61.9	

[a] OSA indicates obstructive sleep apnea; NS, no statistically significant difference.
[b] Fisher's exact test.

to assess the role of maxillary morphology in the etiology of OSA.

Age and sex distribution

The prevalence of OSA within the sample size compared favorably with published studies.[1] OSA and control subjects were well matched for age and sex. Male and female subjects were reported separately, unlike previous studies that have either combined the sexes or described results from male subjects only. Female OSA subjects were approximately seven years older than their control group. This sup-

TABLE 6. Cephalometric Soft Tissue Measurements in Male OSA and Control Subjects[a]

Measurement (mm or mm^2)	OSA ($N = 34$)		Control ($N = 29$)		P Value[b]
	Median	Range	Median	Range	
Soft palate length	39.25	30.62–51.64	38.59	29.21–47.83	NS
Soft palate area	3.97	1.89–5.52	3.87	2.25–5.18	NS
Soft palate thickness	10.78	5.4–13.12	10.28	7.92–16.02	NS
Minimum palatal airway width	3.2	1.09–11.9	7.7	1.87–15.17	**

[a] OSA indicates obstructive sleep apnea; NS, no statistically significant difference.
[b] Mann-Whitney statistical test.
** $P < .01$.

TABLE 7. Cephalometric Soft Tissue Measurements in Female OSA and Control Groups[a]

	OSA ($N = 13$)		Control ($N = 18$)		
Measurement (mm)	Median	Range	Median	Range	*P* Value[b]
Soft palate length	40.52	33.65–47.82	36.5	30.25–43.69	*
Soft palate area	3.37	1.96–4.47	3.28	1.8–7.21	NS
Soft palate thickness	9.51	6.71–11.93	8.38	6.04–11.84	NS
Minimum palatal airway width	5.4	1.09–7.63	8.78	5.09–13.44	***

[a] OSA indicates obstructive sleep apnea; NS, no statistically significant difference.
[b] Mann-Whitney statistical test.
* $P < .05$; *** $P < .001$.

TABLE 8. Maxillary Arch Measurements in Male OSA and Control Groups[a]

	OSA ($N = 34$)		Control ($N = 29$)		
Measurement (mm)	Median	Range	Median	Range	*P* Value[b]
Intercanine	33.6	21.75–39.91	33.86	25.67–38.89	NS
Inter–first premolar	35.34	26.14–42.84	35.8	29.9–39.34	NS
Inter–second premolar	40.19	33.42–48.7	40.1	33.8–44.35	NS
Intermolar	46.31	38–54.25	46.3	38.1–52.19	NS
Maxillary depth	39.58	32.33–45.38	38.4	33.75–46.78	NS
Palatal height at canine	11.21	7.1–16.1	11.2	7.02–20.12	NS
Palatal height at first premolar	16.36	8.67–28.41	15.2	8.2–24.12	*
Palatal height at second premolar	20	10.34–30.11	17.1	9.11–26.11	*
Palatal height at molar	22.43	11.41–32.11	18.1	11.7–27.44	*

[a] OSA indicates obstructive sleep apnea; NS, no statistically significant difference.
[b] Mann-Whitney statistical test.
* $P < .05$.

TABLE 9. Maxillary Arch Measurements in Female OSA and Control Groups[a]

	OSA ($N = 34$)		Control ($N = 29$)		
Measurement (mm)	Median	Range	Median	Range	*P* Value[b]
Intercanine	32.4	29.39–34.37	32.02	24.9–35.77	NS
Inter–first premolar	33.12	28.94–37.23	34.13	30.44–37.5	NS
Inter–second premolar	37.96	34.43–45.4	38.76	34.66–41.59	NS
Intermolar	45.08	37.03–49.5	42.7	36.54–48.3	NS
Maxillary depth	38.21	34.1–41.97	38.11	31.63–41.11	NS
Palatal height at canine	11.55	6.2–20.12	9.1	8.1–14.12	NS
Palatal height at first premolar	15.4	11.11–22.4	14.11	9.01–19.11	*
Palatal height at second premolar	22.1	14.4–28.33	16.2	11.1–22.12	*
Palatal height at molar	26.8	15.5–31.33	18.75	12.88–24.12	*

[a] OSA indicates obstructive sleep apnea; NS, no statistically significant difference.
[b] Mann-Whitney statistical test.
* $P < .05$.

TABLE 10. Frequency Distribution of Buccal Crossbite in OSA and Control Groups[a]

	OSA Group	Control Group	*P* Value[b]
Buccal crossbite present	14	11	0.641
Percentage within group	29.8	23.4	
Buccal crossbite absent	33	36	NS
Percentage within group	70.2	76.6	

[a] OSA indicates obstructive sleep apnea; NS, no statistically significant difference.
[b] Fisher's exact test.

ported the finding that the prevalence of OSA increases with age to approximately 50–55 years in men and 60–65 years in women, after which it appears to decline.[1,16,17]

Obesity

The male subjects were classified as overweight and the female subjects were classified as obese based on the BMI and neck circumference. These findings are consistent with other studies.[18–21] In the present study, however, a greater frequency of obesity and lower prevalence of OSA were

present in women, which is in agreement with the findings of Mohsenin.[22] Walker et al[23] concluded that OSA severity was less weight dependent in women than in men.

The neck circumference recorded was statistically greater in both male ($P < .001$) and female ($P < .05$) OSA subjects, and other studies have reported similar findings.[18,24–26] This is consistent with the findings of Whittle et al,[26] who demonstrated that normal men have a greater proportion of neck fat than normal women. These highlight the need to separate the sexes when evaluating the oropharyngeal dimensions in OSA subjects.

In the current study, the OSA group was obese and the control group classified as overweight. Predictive models of OSA have been proposed in which the degree of skeletal abnormality in a patient is inversely related to the degree of obesity required to cause OSA.[27–30]

Lateral cephalometric measurements

Traditionally, OSA has been associated with mandibular retrusion and hence a skeletal Cl II pattern.[31] However, in the current study, there was no significant differences in the distribution of skeletal classification (SNA, SNB) in OSA or control subjects (Table 5) in agreement with several previous studies.[18,20,32] Analyses did not reveal a significant difference in the AP or vertical planes (or both) for the maxilla between OSA and control subjects, nor between the sexes, confirming the findings of Seto et al.[18]

In the AP plane, the PNS-posterior pharyngeal wall measurements were reduced in all OSA subjects as previously reported by Battagel and Johal,[27] Battagel et al,[33] and Seto et al.[18] The anterior cranial base was shorter, resulting in a more retrusive facial complex and reduced pharyngeal airway.[28,32,34,35] Seto et al[18] reported a statistically significant shorter ANS-PNS in OSA subjects. However, in their study, over 25% of the OSA subjects failed to have radiographs taken.

The palatal angle (ANS-PNS-uvula) was significantly more obtuse in male OSA subjects ($P < .05$). Because all other maxillary skeletal measurements detected no significant differences, the discrepancy appeared to be with the soft palate and its orientation. Tsuchiya et al[28] proposed OSA subtypes based on the degree of obesity, the severity of OSA, and craniofacial data. In the current study, both the OSA and control subjects had BMI values classifying them as overweight, and significant differences were detected in the soft, but not hard tissue, measurements. Seto et al[18] found significant skeletal discrepancies, which may be explained by the difference in BMI values between their OSA and control groups, a smaller sample size, or incomplete data analyses (or all).

Soft tissues

In the present study, the soft palate of all OSA subjects was larger and occupied significantly more of the upper pharyngeal space than in the control subjects. Soft palate length was increased in OSA subjects, which is in agreement with other studies.[18,27,36] Furthermore, soft palate length increases with age,[37] and thus studies must match control subjects for age. Soft palate area was increased in all OSA subjects, but the values were less than reported by Battagel and L'Estrange.[20] The latter study only investigated men, with a significantly younger control group and consequent lower BMI values. An increase in soft palate thickness in all OSA subjects was not statistically significant but was in agreement with the studies of Battagel and Johal[27] and Battagel et al.[33] This increase in thickness may be attributed to an extension of adipose tissue from accumulations in the neck, although this is debatable.[38] Palatal airway width was significantly reduced in OSA subjects, confirming the findings of previous studies.[20,33,35]

Study model measurements

The relationship between maxillary constriction and the etiology of OSA is not clear. To date, only three studies have used models to assess the transverse arch dimensions of the maxilla in OSA subjects: Cistulli et al,[39] Kushida et al,[30] and Seto et al.[18] Cistulli et al[39] examined the influence of maxillary morphology in a sample of 13 patients with Marfan's syndrome. However, the suitability of Marfan's patients as OSA subjects is debatable.

The present study found a statistically significant difference in palatal heights between OSA and control subjects at the level of the first premolar, second premolar, and molar. However, these results were not in agreement with those of Cistulli et al[39] and Seto et al,[18] who did not find differences in palatal heights between OSA and control subjects. This was surprising in the study of Cistulli et al[39] because a high vaulted palate is very characteristic of Marfan's syndrome. This was confirmed by Seto et al,[18] who reported that palatal height measurement alone was not a reliable indicator of maxillary constriction. Kushida et al[30] included palatal height in their predictive morphometric model for OSA, but they defined palatal height as the distance from the dorsum of the tongue to the highest point of the palate.

The present study could find no significant differences in maxillary depth or intertooth distances. Again, this was not in agreement with previous studies.[18,39] The predictive morphometric model for OSA uses maxillary and mandibular intermolar distances measured from the second molar tooth,[30] with no allowances made for this tooth being absent. Seto et al[18] also found that the maxillary depth was significantly reduced in OSA subjects.

Posterior transverse discrepancy

In this study, no statistical significance was detected between OSA and control subjects in transverse dental dimensions. However, Seto et al[18] in their study, reported statistically significant posterior transverse discrepancies but

did not clarify whether they were skeletal or dental. Previous studies have shown that OSA is not exclusive to one skeletal classification.[20,32] Thus, it remains very questionable as to whether maxillary constriction can be a primary etiological factor in OSA and more importantly, whether treatment directed at this is evidence based. The present study, therefore, cannot support the use of RME as a treatment modality for subjects with OSA in the absence of any obvious maxillary constriction. Furthermore, such practices must engage in objective outcome measures, such as follow-up sleep studies to determine their therapeutic value.

CONCLUSIONS

1. Maxillary morphological differences exist between OSA and control subjects, identifying a potential etiological role in OSA.
2. Statistically significant differences exist between OSA and control subjects, in both maxillary skeletal morphology and oropharyngeal dimensions.
3. Study model analyses demonstrated that OSA subjects differ significantly from control subjects in palatal height measurements.

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APPENDIX 1

Questionnaire for OSA and control subjects

Personal details

Name:	Height (m):
Sex:	Weight (kg):
Group (OSA or control):	BMI:
Date of Birth:	Neck circumference (cm):

Medical history

Upper airway obstruction:
Cardiac anomalies (angina, myocardial infarction, cardiac failure)
Preexisting breathing disorders:
Hypertension:
Medication:
Thyroid disease:

Orthodontic examination

Teeth present:	Molar relationship:
Overjet:	Posterior buccal crossbite:
Overbite:	Scissor bite:

APPENDIX 2

The Epworth sleepiness scale

How likely are you to doze off or fall asleep in the following situations (in contrast to feeling just tired)?

Even if you have not been in some of these situations recently, try to work out how they would have affected you.

Use the scale to choose the most appropriate number for each situation:

0 = would *never* doze
1 = *slight* chance of dozing
2 = *moderate* chance of dozing
3 = *high* chance of dozing

Situation	Chance of dozing
Sitting and reading	. . . . .
Watching TV	. . . . .
Sitting, inactive in a public place (eg, a theatre or a meeting)	. . . . .
As a passenger in a car for an hour without a break	. . . . .
Lying down to rest in the afternoon when circumstances permit	. . . . .
Sitting and talking to someone	. . . . .
Sitting quietly after a lunch without alcohol	. . . . .
In a car, while stopped a few minutes in traffic	. . . . .
Total score	. . . . .