European Journal of Orthodontics 23 (2001) 703–714

Maxillary morphology in obstructive sleep apnoea syndrome

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SUMMARY The aim of this case-control study was to test the hypothesis that maxillary morphology differs between obstructive sleep apnoea (OSA) patients and non-snoring, non-apnoeic subjects. Forty randomly selected patients [36 M, 4 F; mean age 49 ± 2 (SEM) years] with varying degrees of OSA (mean Apnoea/Hypopnoea Index 32 ± 4/hour) were compared with 21 non-snoring, non-apnoeic control subjects (18 M, 3 F; mean age 40 ± 2 years). An intra-oral assessment of the occlusion was carried out, particularly for the presence or absence of posterior transverse discrepancies. Maxillary dental arch width was assessed by standardized lateral inter-tooth measurements (inter-canine, inter-premolar, and inter-molar) from dental models. Palatal height and maxillary depth were also measured. The maxillary dental arch was described by a 4th order polynomial equation. The ratios of maxillary to mandibular width (max/mand) and maxillary to facial width (max/facial) were determined from standardized postero-anterior cephalometric radiographs in a subgroup of patients ($n = 29$) and all controls.

Twenty patients (50 per cent) had evidence of posterior transverse discrepancies compared with one control subject (5 per cent; $P < 0.01$). All patients had significantly reduced inter-canine, inter-premolar, and inter-molar distances ($P < 0.05$). The maxillary depth was also shorter ($P < 0.05$), but palatal height was not different. The quadratic coefficient of the polynomial equation was greater in the patients than in the controls ($P < 0.05$), indicative of greater arch tapering. Patients had smaller maxillary to mandibular and maxillary to facial width ratios ($P < 0.01$). These results suggest that OSA patients have narrower, more tapered, and shorter maxillary arches than non-snoring, non-apnoeic controls. Further work is required to determine the relevance of these findings in the pathophysiology of OSA.

Introduction

Obstructive sleep apnoea (OSA) is a common disorder affecting 4 per cent of men and 2 per cent of women in middle age (Young *et al*., 1993). It is characterized by repetitive obstruction of the upper airway during sleep, resulting in oxygen desaturation and sleep fragmentation. OSA is now recognized as a major public health problem (Phillipson, 1993) as its diverse symptoms and other sequelae are a source of significant morbidity (McNamara *et al*., 1994).

It is thought that upper airway narrowing and the normal physiological loss of muscle tone during the onset of sleep are important factors in the development of OSA (Cistulli and Sullivan, 1994). Studies using cephalometry have identified structural craniofacial abnormalities in patients with OSA and it is thought that these abnormalities result in upper airway narrowing, thereby predisposing to airway closure during sleep (Cistulli, 1996). The more commonly reported features are a decreased mandibular body length, an increased mandibular plane to

hyoid bone distance, an increased mandibular plane angle, a narrowed posterior airway space (PAS), and elongation of the soft palate (Riley *et al*., 1983; Rivlin *et al*., 1984; Jamieson *et al*., 1986; Lowe *et al*., 1986; deBerry-Borowiecki *et al*., 1988; Lyberg *et al*., 1989; Tangugsorn *et al*., 1995; Battagel and L'Estrange, 1996; Pracharktam *et al*., 1996). Less commonly reported features are a shorter anterior cranial base with a more acute cranial base flexure, a shorter maxillary length, a reduced bony pharynx, maxillomandibular retrognathia, and increased upper and lower facial heights (Riley *et al*., 1983; Jamieson *et al*., 1986; Lowe *et al*., 1986; deBerry-Borowiecki *et al*., 1988; Lyberg *et al*., 1989; Bacon *et al*., 1990; Yildirim *et al*., 1991; Tangugsorn *et al*., 1995; Battagel and L'Estrange, 1996). The correlation between some of these craniofacial abnormalities and apnoea severity further suggests that structural abnormalities are likely to play a role in the pathophysiology of OSA (Tsuchiya *et al*., 1992; Ferguson *et al*., 1995).

Maxillary constriction may be another craniofacial abnormality involved in the pathophysiology of OSA. The airway is a three-dimensional conduit and, hence, both transverse and sagittal dimensions need to be assessed. According to Hershey *et al*. (1976) subjects with maxillary constriction have increased nasal airway resistance (NAR) and resultant mouth breathing, features typically seen in OSA patients. Maxillary constriction is also associated with low tongue posture (Subtelny, 1954), which may result in retroglossal narrowing, another feature of OSA (Riley *et al*., 1983). Furthermore, patients with Marfan's syndrome characteristically have a high-arched palate with maxillary constriction and are known to have a high prevalence of OSA (Cistulli and Sullivan, 1993). In these patients, highly significant correlates have been found between the degree of maxillary constriction, NAR, and OSA severity (Cistulli *et al*., 1996; Cistulli and Sullivan, 2000). In further support of the hypothesis, Guilleminault *et al*. (1995) reported a high odds ratio for the presence of a high, narrow hard palate in the immediate family relatives of index cases with OSA. The aim of this study was to test the hypothesis that maxillary morphology differs between typical OSA patients and non-snoring, non-apnoeic controls.

Subjects and methods

Subjects

The patient sample consisted of randomly selected adults presenting for overnight polysomnography at the Sleep Disorders Laboratory of St George Hospital, a tertiary teaching hospital. Patients were selected on the basis of OSA symptoms, that is, snoring, witnessed apnoeas, and daytime hypersomnolence, as well as polysomnographic evidence of OSA [defined as an Apnoea/Hypopnoea Index (AHI) ≥ 5/hour]. The controls were dental colleagues recruited from the United Dental Hospital in Sydney at the time the cases arose. All controls were screened with a validated questionnaire to exclude snoring and excessive daytime sleepiness, the latter defined as an Epworth Sleepiness Score (ESS) > 10 (Johns, 1991). Assistance from partners was sought where possible. Edentulous subjects were excluded because of the inability to assess their maxillary arch dimensions without dental landmarks. Those with a history of jaw surgery or orthodontic treatment were also excluded. All subjects were Caucasian. Weight, height, and neck and waist circumference were measured in all subjects, and the body mass index (BMI) was calculated. Ethics Committee approval was obtained and all subjects gave informed consent.

Sleep studies

Nocturnal polysomnography was performed by a trained sleep laboratory technician in a standard fashion, as previously described (Cistulli and Sullivan, 1993). Calculated respiratory variables were AHI (the number of apnoeas and hypopnoeas per hour of sleep), and minimum arterial oxygen saturation ($minSaO_2$) during apnoeas. Apnoea was defined as cessation of airflow for at least 10 seconds. Hypopnoea was defined as a reduction in amplitude of airflow or thoracoabdominal wall movement of greater than 50 per cent of the baseline measurement for more than

10 seconds (O_2 desaturation need not occur), or the same reduction with an accompanying O_2 desaturation of at least 3 per cent (no time limit), and associated with arousal. These respiratory events were defined as obstructive if they occurred in association with continued diaphragm electromyogram (EMG) activity and thoraco-abdominal wall movement. Central events were defined as those accompanied by absence of diaphragm EMG activity and thoraco-abdominal wall movement.

Clinical orthodontic examination

An intra-oral assessment of the occlusion was carried out in all subjects by a single investigator (BS), particularly for the presence or absence of posterior transverse discrepancies. The shape of the dental arches and depth of the palatal vault were noted. Dental arch constriction was defined by the presence of two or more maxillary posterior teeth in an edge-to-edge cuspal relationship with their antagonists, or in frank crossbite. Skeletal maxillary constriction was defined by the presence of all the following features: (a) a narrow and high palatal vault, (b) a corresponding narrow arch form, and (c) unilateral or bilateral buccal tilting of the maxillary alveolar arches with posterior teeth in crossbite or edge-to-edge relationship with the lower teeth (Proffit and Ackerman, 1994).

Dental measurements

Dental impressions were taken to fabricate a set of upper and lower models. Alginate impression material (Unijel-II, Unitek/3M, Type I fast setting) was mixed according to the manufacturer's instructions. Impressions were poured within half-an-hour of being taken using orthodontic stone (Whip mix, ADA type III). All procedures were carried out by a single operator (BS). The following linear measurements were recorded from the upper study model using Vernier callipers (Beerendonk, Germany):

1. inter-canine distance, defined as the distance between the centroids of the canines, as described by Moyers *et al*. (1976);
2. inter-premolar distance at the first and second premolar regions, defined as the linear distance between the centroids of the first and second premolars, respectively;
3. inter-molar distance, defined as the linear measurement between the centroids of the first permanent molars (Figure 1).

In subjects where teeth were absent, the linear measurement was made to the apex of the edentulous ridge bisecting a line drawn from the centroids of the abutment teeth. A profile gauge was used to record the cross-sectional shape of the palate at the canine, first and second premolar, and first molar regions by lining up the gauge over the centroids of the teeth at each of these levels. The corresponding palatal height was then measured off the profile gauge with a steel ruler. The palatal index was defined as the ratio of the palatal height to the lateral dimension at the level of the canines, first and second premolars, and first molars. The depth of the maxilla was measured with a pair of three-dimensional Bow dividers (Korkhaus, Germany) and was defined as the distance from the midpoint of the most labial point of the central incisors to the point bisecting the line joining the distal midpoints of the maxillary first molars (Figure 1).

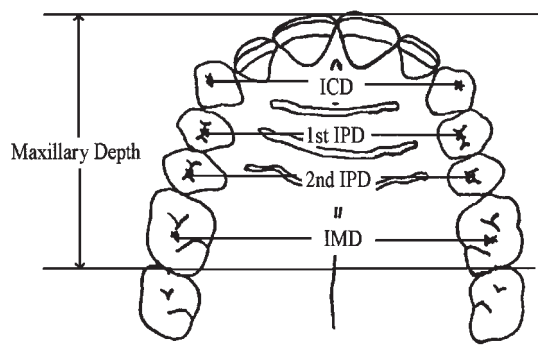

Figure 1 Schematic diagram of the maxillary dental arch illustrating the linear measurements taken from study models. ICD, inter-canine distance; 1st IPD, first inter-premolar distance; 2nd IPD, second inter-premolar distance; IMD, inter-molar distance.

Mathematical modelling of maxillary dental arch

The midpoints of the incisal edges and the buccal cusp tips of the maxillary teeth were marked on the casts, which were then photocopied at a 1:1 magnification. Overlay graph paper was then superimposed on the photocopied image. A line drawn through the distal contact points of the first molars was orientated parallel to the *X*-axis, the co-ordinates (0,0) coinciding with the contact point of the central incisors. Co-ordinates were then determined from the marked incisal edges and buccal cusp tips of the cast. A 4th order polynomial equation and curve describing the arch form was generated from these co-ordinates using Microsoft Excel software.

Cephalometric radiographs

Lateral and postero-anterior (PA) cephalograms were taken separately, with a uniform technique (Broadbent *et al.*, 1975). Each subject was instructed to close in centric occlusion, to take a breath and then slowly exhale. Radiographs were exposed during the expiratory phase. All radiographs were hand traced on acetate paper over a light-viewing box. Where bilateral landmarks presented two images, the average of the two was taken. The PA cephalograms were analysed according to Ricketts (1981). Maxillary width was defined as the distance between the points on the jugal process at the intersection of the outline of the tuberosity of the maxilla and the zygomatic buttress; mandibular width as the distance between the points at the lateral inferior margin of the antegonial protuberances, and facial width as the distance between the centre of the root of the zygomatic arch on each side (Figure 2). These measurements were used to calculate the maxillary to mandibular (max/mand) width ratio and maxillary to facial (max/facial) width ratio. The reference landmarks, lines, and angles used in the analysis of the lateral cephalograms in Figure 3 were:

Landmarks

Point A: the most posterior point on the curve of the maxilla between the anterior nasal spine and supradentale.

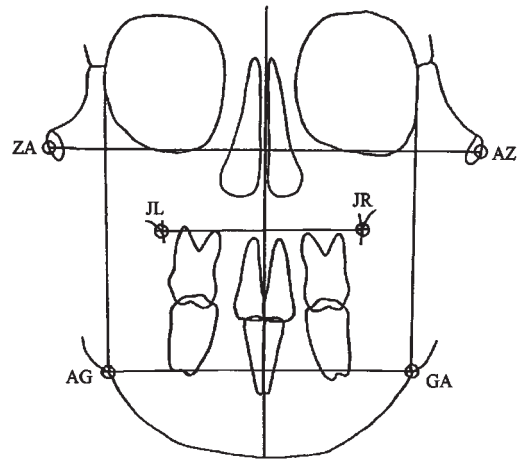

Figure 2 Schematic diagram of the postero-anterior cephalogram illustrating the linear measurements used in the analysis. ZA/AZ, centre of root of zygomatic arch; ZA, left; AZ, right. JL/JR, jugal processes at intersection of outline of maxillary tuberosity and zygomatic buttress; JL, left; JR, right. AG/GA, points at lateral inferior margin of antegonial protuberances; AG, left; GA, right.

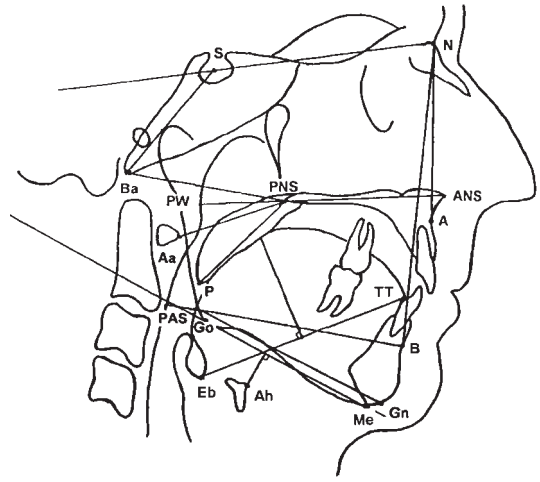

Figure 3 Schematic diagram of lateral cephalogram illustrating the linear and angular measurements used in the analysis.

Aa: the most anterior point on the atlas vertebrae.

Ah: the most anterior and superior point on the body of the hyoid bone.

Ar (articulare): the point of intersection of the inferior cranial base surface and the averaged posterior surfaces of the mandibular condyles.

ANS (anterior nasal spine): the tip of the median, sharp bony process of the maxilla at the lower margin of the anterior nasal opening.
Point B: the point most posterior to a line from infradentale to pogonion on the anterior surface of the symphyseal outline of the mandible.
Ba (basion): the most inferior, posterior point on the anterior margin of the foramen magnum in the median plane.
Co (condylion): the most posterior superior axial point on the curvature of the average of the right and left outline of the condylar head.
Eb: base of epiglottis.
Gn (gnathion): the most anterior-inferior point on the contour of the bony chin symphysis, determined by bisecting the angle formed by the mandibular plane and a line through pogonion and nasion.
Go (gonion): the midpoint of the angle of the mandible, found by bisecting the angle formed by the mandibular plane and a plane through articulare along the portion of the mandibular ramus inferior to it.
Me (menton): the most inferior point on the symphyseal outline.
MP (mandibular plane): a line joining Me to Go intersection.
Na (nasion): the junction of the frontonasal suture at the most posterior point on the curve at the bridge of the nose.
P: tip of soft palate.
Pg (pogonion): the most anterior point on the contour of the bony chin, determined by a tangent through nasion.
PNS: posterior nasal spine.
Pw (posterior pharyngeal wall): a point directly determined by the extension of the palatal plane to the pharyngeal wall.
S (sella): the centre of the pituitary fossa of the sphenoid bone determined by inspection.
SN: anterior cranial base.
TT: tongue tip.

Linear measurements

ANS–PNS: the length of the maxilla.
Aa–PNS: the distance between the anterior point of atlas to posterior nasal spine.
Ba–PNS: the distance between basion to posterior nasal spine.
PNS–Pw: distance from the posterior nasal spine to the posterior pharyngeal wall.
Go–Gn: length of the body of the mandible.
MP–H: distance from the mandibular plane to Ah, the most anterior and superior point on the body of the hyoid bone (H).
PAS: the distance between the posterior pharyngeal wall and the dorsal surface of the base of the tongue, measured on the line that intersects Go and B point (Riley *et al*., 1983).
PNS–P: length of the soft palate.
TGLt: length of the tongue, as measured from TT to Eb (Lowe *et al*., 1986).
TGHt: tongue height. The linear distance along the perpendicular bisector of the Eb–TT line to the tongue dorsum (Lowe *et al*., 1986).

Angular measurements

SNA: angle from sella to nasion to subspinale point.
SNB: angle from sella to nasion to supramentale point.
ANB: angle from Point A to nasion to Point B.
MP–SN: angle between line from mandibular plane and the line from sella to nasion.
Ba–SN: angle between basion to sella and sella to nasion.

Measurement error

Dental arch measurements and cephalometric analyses were carried out by a single investigator (BS). Intra-observer reliability was assessed by remeasurement of arch dimensions for 40 subjects, and duplicate tracing of 30 lateral and PA cephalograms by the same clinician one month later under the same conditions.

Statistical analyses

Data were stored and analysed using SPSS (Chicago, USA). The statistical significance of dichotomous outcomes in 2×2 contingency tables was established by means of a chi-squared test with Yates' correction or Fisher's exact test for small expected frequencies. For non-ordered, non-dichotomous factors, significance was

assessed using a chi-squared statistic. Unpaired *t*-tests were used to compare continuous outcomes between two independent groups where data were normally distributed. Wilcoxon's Ranked Sum Test was applied where the data were not normally distributed. Pearson correlation coefficients and linear regression evaluated the relationship between continuous outcome variables. The Spearman rank correlation was used if the distribution of numerical variables was skewed. An analysis of errors of arch and cephalometric measurements was carried out by calculating the mean differences between the initial and repeated set of measurements. The mean differences were recorded and the standard deviation of the differences calculated. The measurement error was derived according to Houston (1983). The index of reliability was the correlation between the repeat measurements, which evaluated the contribution of random errors. All values are presented as mean ± standard error of the mean (SEM). A P value of less than 0.05 was considered statistically significant.

Results

The patient sample consisted of 36 men and four women with a mean AHI of 32 ± 4/hour (range 6–101/hour) and mean $minSaO_2$ of 81 ± 2 per cent (range 53–93 per cent). Of the 35 subjects who completed the screening questionnaire, 18 men and three women were accepted as controls based on the absence of snoring or daytime sleepiness. Sex distribution did not differ significantly between patients and controls ($P > 0.05$; Fisher's exact test). Only a very small proportion of the sample comprised women and, hence, the results were pooled for males and females, as gender was unlikely to have a significant impact on outcome. The patients were significantly older and heavier than controls, with a greater neck and waist circumference, and showed a trend towards a shorter stature that was not statistically significant (Table 1). Skeletal relationship was examined using lateral cephalograms for 29 patients and all control subjects. Eleven patients did not present for radiographs to be taken. There was no significant difference in the distribution of skeletal classification between patients and controls ($\chi^2 = 1.69$; 2 df; $P > 0.05$; Table 2). Patients with radiographic data were significantly older than those without (51 ± 2 years versus 43 ± 3 years; $P < 0.05$), but did not differ in OSA severity or any other anthropomorphic characteristics.

Twenty patients (50 per cent) had posterior transverse discrepancies compared with one control subject (5 per cent) ($\chi^2 = 10.56$; 1 df; $P < 0.01$; 95 per cent CI for the difference: 27–63 per cent). Eight patients had bilateral crossbite, five a bilateral edge-to-edge cusp relationship and seven unilateral crossbite. In the subgroup of subjects for whom skeletal pattern was determined, 13 patients (45 per cent) had posterior transverse discrepancies compared with the one control subject (5 per cent; $\chi^2 = 7.81$; 1 df; $P < 0.01$; 95 per cent CI for the difference: 20–60 per cent).

The OSA patients had significantly narrower inter-tooth distances at all four levels, and a shorter maxillary depth (Table 3). There were no

Table 1 Anthropometric data for patients and controls.

	Patient group ($n = 40$)			Control group ($n = 21$)			
	Mean ± SEM	Min	Max	Mean ± SEM	Min	Max	P value
Age (years)	49 ± 2	25	74	40 ± 2	20	60	< 0.005
Weight (kg)	99 ± 3	62	134	81 ± 3	59	135	< 0.005
Height (cm)	173 ± 2	146	185	176 ± 2	163	190	0.06
BMI (kg/m^2)	33.3 ± 0.8	24.9	43.1	26.0 ± 1	20.2	38.6	< 0.005
Neck (cm)	44 ± 1	36	55	40 ± 1	33	48	< 0.005
Waist (cm)	111 ± 2	85	134	92 ± 3	72	121	< 0.0001

Table 2 Frequency distribution of patient and control groups by skeletal classification.

Group	Skeletal classification			
	I	II	III	Total
Patient	14	9	6	29
Control	14	4	3	21
Total	28	13	9	50

significant differences in palatal heights between patients and controls. The palatal index was significantly greater at the second inter-premolar and the first inter-molar regions in patients than in controls. Comparison of the polynomial equation coefficients indicated that the quadratic (x^2) coefficient was significantly greater in patients than in controls (0.027 ± 0.002 versus 0.020 ± 0.002; $P < 0.05$), indicative of greater tapering of the maxillary arch. There were no differences in any of the other polynomial coefficients between the two groups.

Analysis of the PA cephalograms showed a significant difference between patients and controls in the (max/mand) width ratio (0.68 ± 0.01 versus 0.73 ± 0.01; $P < 0.001$) and in the (max/facial) width ratio (0.44 ± 0.005 versus 0.47 ± 0.005; $P < 0.01$), both being reduced in the patient group. Analysis of the lateral cephalograms revealed that in the patient group, both the maxilla (ANS–PNS) and mandible (Go–Gn) were shorter when compared with controls. Patients had a greater mandibular plane to hyoid bone distance (MP–H), a longer soft palate (PNS–P), and a greater tongue length (TGLt) than controls. There was no significant difference between the two groups in any other cephalometric measurements (Table 4).

The measurement errors of arch dimensions, polynomial coefficients, and cephalometric variables were small and the reliability indices were high, indicating a high estimate of reproducibility (Table 5).

No significant correlations were found between any maxillary arch dimensions or cephalometric measurements and apnoea severity, defined by the AHI and the $minSaO_2$. Significant correlations, however, were found between the PAS and the lateral dimensions of the maxilla at the canine ($r = 0.37$; $P < 0.05$), first premolar ($r = 0.41$; $P < 0.05$), and molar ($r = 0.37$; $P < 0.05$) regions.

Discussion

This study supports the hypothesis that maxillary morphology differs between OSA patients and non-snoring, non-apnoeic controls. The prevalence of posterior transverse discrepancies in the current study was significantly higher in patients (50 per cent) than in controls (5 per cent). It is known that a Class III skeletal relationship is associated with crossbite and, therefore, it is

Table 3 Maxillary arch measurements in patients and controls.

Maxillary arch measurement	Patient group ($n = 40$)	Control group ($n = 21$)	P value
Inter-canine distance	32.00 ± 0.40	34.40 ± 0.40	<0.005
First inter-premolar distance	36.00 ± 0.50	38.50 ± 0.40	<0.005
Second inter-premolar distance	40.60 ± 0.50	43.10 ± 0.60	<0.005
Inter-molar distance	45.90 ± 0.60	48.50 ± 1.00	<0.05
Maxillary depth	33.60 ± 0.50	35.40 ± 0.60	<0.05
Palatal height at canine	8.20 ± 0.40	8.05 ± 0.50	NS
Palatal height at first premolar	15.50 ± 0.50	15.00 ± 0.50	NS
Palatal height at second premolar	20.00 ± 0.40	19.00 ± 0.50	NS
Palatal height at first molar	22.00 ± 0.40	21.20 ± 0.50	NS
Palatal index at canine	0.26 ± 0.01	0.23 ± 0.02	NS
Palatal index at first premolar	0.43 ± 0.02	0.39 ± 0.02	NS
Palatal index at second premolar	0.49 ± 0.01	0.45 ± 0.02	<0.05
Palatal index at first molar	0.47 ± 0.01	0.44 ± 0.02	<0.05

*All linear measurements are in millimetres; NS = not significant.

Table 4 Lateral cephalometric measurements in patients and controls.

Cephalometric variable	Patient group (n = 29)	Control group (n = 21)	P value
Linear measurements (mm)			
SN	76.97 ± 0.69	79.10 ± 0.93	NS
ANS–PNS	51.11 ± 0.59	54.85 ± 0.99	<0.01
Aa–PNS	32.84 ± 0.60	32.90 ± 0.74	NS
Ba–PNS	41.83 ± 0.56	43.05 ± 0.75	NS
PNS–Pw	23.44 ± 0.80	24.85 ± 0.59	NS
Go–Gn	72.36 ± 0.78	77.18 ± 1.15	<0.01
MP–H	18.45 ± 1.20	14.44 ± 1.23	<0.05
PAS	10.17 ± 0.46	10.45 ± 0.76	NS
PNS–P	40.64 ± 0.90	36.90 ± 0.81	<0.01
TGLt	81.35 ± 1.10	75.21 ± 1.60	<0.01
TGHt	37.20 ± 0.60	37.97 ± 0.62	NS
Angular measurements (°)			
SNA	79.55 ± 0.88	80.67 ± 0.92	NS
SNB	78.07 ± 0.98	79.00 ± 0.89	NS
ANB	1.48 ± 0.6	1.67 ± 0.60	NS
MP–SN	32.45 ± 1.36	28.98 ± 1.52	NS
Ba–SN	130.00 ± 0.76	130.14 ± 1.27	NS

possible that the higher prevalence of transverse discrepancies detected in patients was not entirely the result of true maxillary constriction. This, however, is unlikely, as skeletal pattern distribution did not differ between patients and controls. Although skeletal classification was assessed in a subgroup of subjects with the potential for selection bias, there was only a 5 per cent reduction in the prevalence of transverse discrepancies in patients within this subgroup compared with patients in the entire sample. In addition, the prevalence of transverse discrepancies in this subgroup still differed significantly, and in the same direction between patients and controls as in the original sample. Moreover, since it is possible to have skeletal maxillary constriction and simultaneous maxillary and mandibular dental arch constriction without the presence of a crossbite, the prevalence of posterior transverse discrepancies found in this study is considered to be conservative for the true prevalence of skeletal maxillary constriction. Analysis of the lateral cephalograms did not reveal a difference in the spatial arrangement of the maxilla that could explain the observed posterior transverse discrepancies between patients and controls.

The OSA patients in this study had significantly narrower dental arch forms when compared with normal controls. All four mean inter-tooth distances were significantly smaller by 2.4–2.6 mm in the patient group. In addition, the patients had a significantly shorter maxillary arch depth and maxillary length (ANS–PNS). These results are similar to those previously reported in OSA patients with Marfan's syndrome (Cistulli *et al*., 1996). Moreover, the max/mand and max/facial width ratios derived from the PA cephalometric radiographs were significantly smaller in the patients compared with the controls. These findings indicate that the observed maxillary constriction is skeletal in origin, rather than simply related to narrowing of the dental arch.

In order to describe the arch form in a quantitative fashion, 4th order polynomial equations were used because they give the most accurate and reproducible likeness of arch form (Biggerstaff, 1972). The equation describes a continuous curve, providing a more accurate and complete outline of the shape of the whole dental arch than can be achieved by inter-tooth distances alone. The patients were found to have a significantly larger quadratic (x^2) coefficient,

Table 5 Mean difference between repeated measurements, standard deviation of the difference, error, and reliability index for arch dimension variables, polynomial coefficients and cephalometric variables.

Variable	Mean difference	SD of difference	Error	Reliability Index
Arch dimension (mm)				
Inter-canine distance	0.05	0.110	0.077	0.998
First inter-premolar distance	0.06	0.112	0.079	0.998
Second inter-premolar distance	0.05	0.120	0.080	0.998
First inter-molar distance	0.03	0.116	0.080	0.998
Maximum palatal height	0.03	0.187	0.130	0.996
Maxillary depth	0.09	0.200	0.140	0.998
Polynomial coefficient				
x	0.0059	0.022	0.015	0.97
x^2 ($\times 10$)	0.2100	0.240	0.160	0.98
x^3 ($\times 10^3$)	0.0080	0.680	0.480	0.98
x^4 ($\times 10^4$)	0.0200	0.050	0.030	0.91
Cephalometric variable: linear measurements (mm)				
SN	0.05	0.35	0.25	0.990
ANS–PNS	0.41	0.50	0.35	0.985
Aa–PNS	0.18	0.35	0.24	0.994
Ba–PNS	0.53	0.47	0.33	0.983
PNS–Pw	0.30	0.45	0.32	0.990
Go–Gn	0.25	0.41	0.28	0.997
MP–H	0.20	0.38	0.26	0.996
PAS	0.10	0.24	0.17	0.997
PNS–P	0.42	0.53	0.37	0.990
GLt	0.47	0.56	0.40	0.990
GHt	0.22	0.36	0.25	0.990
Maxillary width	0.10	0.27	0.19	0.997
Mandibular width	0.29	0.44	0.31	0.996
Facial width	0.32	0.47	0.33	0.996
Cephalometric variable: angular measurements (°)				
SNA	0	0.46	0.32	0.980
SNB	0.02	0.43	0.30	0.990
ANB	0.05	0.54	0.38	0.970
MP–SN	0.03	0.60	0.42	0.990
Ba–SN	0.12	0.56	0.39	0.990

indicating a more tapered arch form than in control subjects.

In the present study there were no differences between the palatal heights at the canine, first and second premolar, and first molar regions between the patients and the controls. However, the palatal indices were significantly greater at the second inter-premolar and first inter-molar regions in the patient group, accounted for by the reduced inter-tooth distances. This finding indicates that the height of the palate, by itself, is not a reliable indicator of maxillary constriction and that the width must also be taken into account when assessing constriction.

In contrast to other studies using lateral cephalometry (Partinen *et al*., 1988; Davies and Stradling, 1990; Tsuchiya *et al*., 1992; Ferguson *et al*., 1995) no significant correlations were found between measurements from the lateral cephalograms and apnoea severity. Similarly, there were no significant correlations between any maxillary arch dimensions or measurements from the PA cephalograms and apnoea severity. This result contrasts with a report of OSA in patients with Marfan's syndrome where it was shown that there was a significant correlation between the palatal index and the AHI (Cistulli *et al*., 1996). One likely explanation relates to the

variable contribution of facial structure and obesity in the development of OSA. Subjects with Marfan's syndrome are characteristically tall and thin, in contrast to typical OSA patients who are centrally obese. Ferguson *et al*. (1995) and Tsuchiya *et al*. (1992) have proposed models of OSA in which the degree of craniofacial abnormality in a patient is related to the degree of obesity required to cause OSA. Hence, at one end of the spectrum are patients who are thin but have a significant degree of craniofacial abnormality (e.g. Marfan's syndrome) and at the other end are obese individuals without significant craniofacial abnormality. Therefore, one is unlikely to find a simple correlation between indices of craniofacial abnormality and OSA severity. Guilleminault *et al*. (1995) reported a high likelihood of a high, narrow hard palate in the immediate family relatives of index cases with OSA. They classified hard palates according to subjective descriptions, rather than objective measurements, such as those used in this study. Furthermore, Kushida *et al*. (1997) reported a clinical morphometric model for the prediction of OSA, which includes a number of anthropomorphic variables related to facial structure (including maxillary inter-tooth measurements) and obesity. Their model has a high sensitivity and excellent specificity for the presence of OSA, but not its severity. These studies support the hypothesis that maxillary morphology plays a role in the pathogenesis of OSA, and also highlight the complex interaction between skeletal and soft tissue factors in the development of OSA.

Significant correlations were found between the lateral dimensions of the maxilla at the canine, premolar and molar regions, and the PAS in the patient group. This suggests that a constricted oral cavity resulting from maxillary constriction may cause a low tongue posture and consequent retroglossal narrowing. A low tongue posture has been associated with a constricted maxilla and posterior crossbites in individuals with chronic upper airway obstruction (Subtelny, 1954). Furthermore, whilst tongue height did not differ between the patient and control groups in the present study, a low position of the hyoid bone was detected which also reflects a low tongue position (Solow *et al*., 1993).

The current investigation has some potential limitations. Firstly, referral bias could have resulted in a patient sample specifically with dental abnormalities. This, however, is an unlikely explanation for the results since the patients studied in the sleep laboratory were independently referred from a number of different respiratory physicians without knowledge of the study. Furthermore, the patients were selected at random. Thus, the patient sample in this study is likely to be representative of a typical clinical OSA population. It is possible, however, that with a larger sample size greater variability in structural abnormalities may have been detected in the patient group. Secondly, the controls were selected on the basis of a screening questionnaire. Ideally, the absence of OSA would have been confirmed by a sleep study, however, limited resources did not allow for this. Whilst screening by questionnaire cannot be completely accurate, the method used in this study has been validated and proven to be reliable (Johns, 1991, 1992; Kump *et al*., 1994). Finally, although an attempt was made to match patients and controls for age and BMI, the patient group was somewhat older and more obese. Whilst this may be an explanation for the presence of OSA in the patients, it does not explain the observed differences in maxillary morphology between the two groups. It is known that changes in craniofacial structure occur with age, however, these are minor and therefore unlikely to account for the differences between patients and control subjects found in the present research.

This study has potential therapeutic implications. With the growing recognition of the role of craniofacial abnormalities in the development of OSA, attention has focused on various strategies aimed at correcting or improving craniofacial structure. An orthodontic procedure for maxillary constriction and nasal obstruction is rapid maxillary expansion (RME). RME increases the width of the maxilla and has been shown in some studies to reduce nasal resistance (Hershey *et al*., 1976; Bishara and Staley, 1987). RME has the potential to improve or normalize upper airway dimensions and has been proposed

as a novel treatment modality for OSA. A pilot study has shown that RME can result in complete resolution of OSA in some patients with mild to moderate OSA and evidence of maxillary constriction (Cistulli *et al.*, 1998). This evidence, although preliminary, suggests a causative association between maxillary constriction and OSA. Further work is required to define the contribution of maxillary constriction in the pathophysiology of OSA.

Conclusions

Maxillary constriction may occur more commonly in patients with OSA, compared with non-snoring, non-apnoeic subjects. Maxillary constriction may play an important role in the development of OSA and, if so, this may have therapeutic implications.

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