

Dental arch morphology in south-east Asian adults with obstructive sleep apnoea: geometric morphometrics

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SUMMARY The association between dental arch morphology and the aetiology of obstructive sleep apnoea (OSA) is not clear. To compare dental arch morphology in 108 Asian adults with and without OSA. Overnight hospital polysomnography was performed, and sleep reports were obtained for all subjects. Standardized digital photographs were also taken of the subjects' upper and lower study models. Using 25 homologous landmarks, mean OSA and control dental arch configurations were computed, and subjected to finite-element morphometry (FEM), *t*-tests and principal components analysis (PCA). Mean upper and lower OSA dental arch morphologies were statistically different from respective Control upper and lower arch morphologies ($P < 0.05$). FEM of the upper arch indicated that the mean OSA configuration was 7–11% narrower in the transverse plane in the incisor

and canine regions when compared with the control configuration, and inter-landmark analysis (ILA) confirmed this finding. FEM for the lower arch indicated that the mean OSA configuration was 10–11% narrower in the antero-posterior plane in the pre-molar and molar regions, and confirmed by ILA. Using PCA, significant differences were also found between the two groups in the lower arch using the first two eigenvalues, which accounted for 90% of the total shape change ($P < 0.001$). Supporting their role as aetiological factors, size and shape differences in dental arch morphology are found in patients with OSA.

KEYWORDS: dental arch, adult, sleep apnoea, obesity, morphometric, Asian

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Introduction

Obstructive sleep apnoea (OSA) is recognized as a significant and serious public health issue. Researchers have estimated that approximately 2–4% of middle-aged women and men, respectively, have this condition, and the majority of cases remain undiagnosed (1). Undiagnosed and untreated, OSA is associated with significant morbidity and mortality, including excessive daytime sleepiness, increased risk of automobile accidents, hypertension, cardiovascular disease, stroke and metabolic abnormalities (2). Many factors contribute to the pathophysiology of OSA, and have been reported in a previous study (1). Dental arch constriction, partic-

ularly in the maxilla, was hypothesized as one of those factors (3). Evidently, patients with OSA appear to have narrower, more tapered and shorter maxillary arches when compared with non-snoring, non-apnoeic groups (4).

In addition, subjects with maxillary constriction were reported to have an increased nasal airway resistance and mouth-breathing features that are typically seen in patients with OSA (5). The influence of maxillary morphology was examined in patients with Marfan's syndrome. It was reported that maxillary constriction, with a high-vaulted hard palate, appears to be an important factor, by increasing nasal airway resistance (6). The familial tendency of narrow, high-vaulted

palates in the relatives of patients with OSA was also found to support a relationship between maxillary constriction and the aetiology of OSA (7). However, while palatal height is included as one predictive parameter for pre-disposition to OSA (8), palatal height measurements alone were reported to be unreliable as indicators of maxillary constriction (5). Nevertheless, differences in palatal heights were found between patients with OSA and control subjects at the level of the first pre-molar, second pre-molar and first molar (3).

Most previous studies have used conventional techniques to assess arch dimensions in subjects with OSA, and few, if any, have used robust geometric morphometric methods. In contrast, finite-element morphometry (FEM) has been used to assess airway and cranial base morphology in patients with OSA, and it has been found that an acute cranial base flexure angle is one important cranio-facial factor in Malay, which may be associated with OSA (9). In addition, FEM has been employed in studies of cranio-facial growth (10), facial soft tissue changes (11–13) and for the assessment of dental arch features of normal subjects (14) as also for patients with cleft lip and palate (10, 15). Size variation can mask subtle changes in shape, but by using FEM it is possible to decompose form into size and shape, enabling the separate analysis of these two components of morphology, as also an analysis of their relationship. The change in morphology is viewed as a deformation of an initial geometric configuration whose boundaries are formed by edges that connect anatomical landmarks into a final form (16). Thus, it is thought that FEM allows a better understanding and visualization of the magnitude and direction of morphological change (16). Nevertheless, published literature regarding dental arch morphology and its association with OSA is minimal, especially in comparison with the numerous articles that can be found concerning skeletal changes that are found in patients with OSA. Therefore, the aim of this study is to test the null hypothesis that no morphological differences in terms of arch size and shape are present in adult Malays with and without OSA. Rejection of the null hypothesis might indicate how dental arch morphology might better identify OSA in patients of diverse ethnicity.

Subjects and methods

This multi-disciplinary study took place in the Clinic of Otorhinolaryngology, Hospital Universiti Sains

Malaysia (HUSM). After obtaining informed consent, which was reviewed and approved at the institutional level, power and sample size software (17) was used to calculate the sample size based on comparing two means (dental arch of OSA and control subjects). To detect a difference of 1 mm (which is considered to be clinically significant) with 80% power, $\alpha = 0.05$ and a ratio between two groups of 1:1, 108 adult Malays aged 18–60 years were recruited for this study. A comparative cross-sectional study design was employed. The inclusion criteria were: age over 18 years; a diagnosis of OSA confirmed by overnight polysomnography (PSG) [apnoea–hypopnoea index (AHI) > 5 per hour of sleep], and the presence of at least six teeth each in the maxillary arch and mandibular arch respectively. Exclusion criteria for sample selection were any subjects with: psychiatric illness; sedative and/or alcohol intake; patient-specific disorders (such as neuromuscular disorders); edentulous patients; a history of orthodontic treatment; a history of reconstructive/orthogenetic or pharyngeal surgery; and any cranio-facial deformity, such as cleft lip and/or palate; and missing dental landmarks (see below). The study sample was divided into two groups. The first group consisted of 54 patients with OSA (defined as an AHI > 5 per hour of sleep) diagnosed with limited overnight PSG. The second group consisted of 54 healthy, non-OSA control subjects who did not have any apnoeic symptoms as evaluated by the attending physician and limited channel PSG. Each patient's age, gender, height and weight were recorded. Neck girth was measured at the level of the thyroid cartilage, and the body mass index (BMI) was calculated. Mean and s.d. were used to describe continuous variables and independent *t*-test were used to test the differences between the numerical variables using Statistical Package for Social Sciences (SPSS) for Windows version 11*. A *P*-value of 0.05 or less was considered statistically significant with 95% confidence intervals.

Sleep studies

An overnight hospital type III Sleep study was performed on each subject between 22:00 PM till 06:00 AM. All variables were recorded simultaneously

*SPSS Inc., Chicago, IL, USA.

and continuously with a portable diagnostic device[†] at HUSM Sleep Science Laboratory. This device has been reported to be suitable for use both in hospital and at home, and it has been validated against full PSG (18). The sleep parameters measured were as follows: nasal airflow, using two appropriately placed thermistors; thoraco-abdominal movements, via two piezoelectric bands; pulse oximetry, using a finger probe; snoring episodes, detected via a vibration sensor placed anterior to the sternomastoid muscle; and continuous actigraphy, to monitor and record body position.

Outputs from the portable diagnostic device were scored automatically and manually (if there was any uncertainty) by two sleep laboratory technicians without knowledge of the clinical characteristics of the patient. Using the automatically scored data, the AHI was computed using a computer program[†] Somnologica; Flaga Medical Devices. Occurrence of OSA was scored when there was cessation of breathing for >10 s associated with evidence of persistent respiratory effort. Hypopnoea was scored when there was >50% decrease in the airflow signal with >3% decrease in arterial oxygen saturation. Therefore, the severity of OSA was evaluated by the AHI, defined as the total number of apnoeas and hypopnoea divided by the total sleep time in minutes. Central and obstructive apnoeas were distinguished by the presence or absence of thoraco-abdominal movements during an apnoea. The AHI was calculated as the number of respiratory events per hour of recording time in bed, with the start of recording being the point at which respiration settled to a rhythmic, stable pattern. The end of the recording time was either the waking time recorded by the subject or the point at which the thoraco-abdominal tracings became disturbed, which was consistent with wakefulness. At the end of the study, individual results were communicated to each patient and, where appropriate, treatment was offered to patients diagnosed with OSA.

Geometric morphometrics

Upper and lower study models of all study subjects were obtained after taking alginate impressions. The study models were photographed using a digital camera in a standardized fashion. Next, x , y coordinates of 25 homologous landmarks (Fig. 1a and b) on the upper and lower study models were obtained using appropri-

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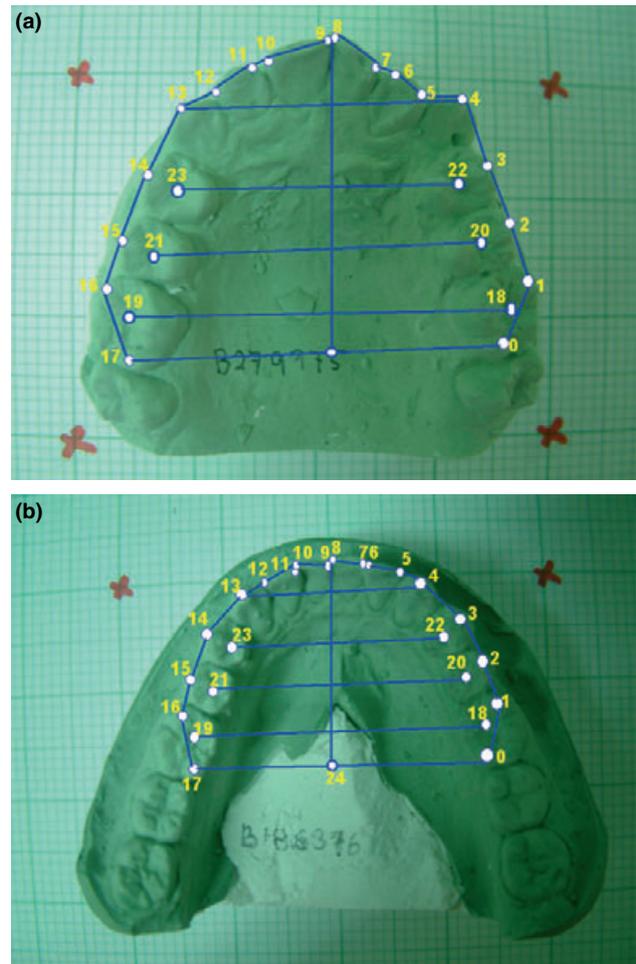


Fig. 1. (a) Definitions of homologous landmarks and inter-landmark distances employed for upper dental arch analyses. 0 = left disto-palatal cusp of first molar, 1 = left mesio-buccal cusp of first molar, 2 = left cusp tip of second pre-molar, 3 = left cusp tip of first pre-molar, 4 = left cusp tip of canine, 5 = left mesio-incisal edge of lateral incisor, 6 = left disto-incisal edge of lateral incisor, 7 = left mesio-incisal edge of central incisor, 8 = left disto-incisal edge of central incisor, 9 = right mesio-incisal edge of central incisor, 10 = right disto-incisal edge of central incisor, 11 = right mesio-incisal edge of lateral incisor, 12 = right disto-incisal edge of lateral incisor, 13 = right cusp tip of canine, 14 = right cusp tip of first pre-molar, 15 = right cusp tip of second pre-molar, 16 = right mesio-buccal cusp of first molar, 17 = right disto-palatal cusps of first molar, 18 = centroid of left first molar, 19 = centroid of right first molar, 20 = centroid of left second pre-molar, 21 = centroid of right second pre-molar, 22 = centroid of left first pre-molar, 23 = centroid of right first pre-molar, 24 = points bisecting the lines joining the distal midpoint of maxillary/mandibular first molars. (b) Homologous landmarks and inter-landmarks employed for lower dental arch analyses. The definitions are shown in (a).

ate software. All landmarks were digitized twice by the same investigator (SMB) on two different occasions. Procrustes superimposition was used to obtain a

generalized rotational fit i.e. all configurations were scaled to an equivalent size and registered with respect to one another (19). In addition, the software was used to perform an inter-landmark analysis (ILA) to detect changes in length and the statistical behaviour of the linear inter-tooth distances between landmarks in the Procrustes means. Thus, linear inter-landmark distances (inter-canine, inter-pre-molar, inter-molar and arch length) were computed (Fig. 1a and b) similar to linear measurements of previous studies (3, 4).

The mean dental arch morphology was determined for each group and a perturbation model was used to compare the mean OSA dental arch morphology with the mean Control dental arch morphology. The Procrustes means were also subjected to *t*-tests to identify elements showing significant changes. To demonstrate sources of heterogenic morphology, a FEM analysis incorporating a spline interpolation function was undertaken on a personal computer. FEM can be used to depict developmental transformations in terms of allometry (size-related shape-change) and anisotropy (directionality of shape change). Based on this approach differences can be described graphically as a size-change, shape-change or a combination of both (16). Change in form between the reference configuration and the final configuration is viewed as a continuous deformation, which can be quantified based on major and minor strains (principal strains). If the two strains are equal, the form change is characterized by a simple increase or decrease in size (20). However, if one of the principal strains changes to a greater extent than the other the transformation occurs in both size and shape. The product of the strains indicate a change in size if the result is not equal to one. A product >1 represents an increase in size equal to the remainder e.g. 1.30 indicate a 30% increase. On the other hand, a result of 0.80 indicates a 20% decrease in size. Changes in shape are determined by the ratio of the principal extensions, where a value not equal to one represents an observable change in shape. The products and ratios can be resolved for individual landmarks within the configuration and these can be linearized using a log-linear scale and pseudocolour-coded to provide a graphic display of size- and shape-change (20).

In addition, principal components analysis (PCA) can be used to compare different groups of patients with specific characteristics (21). Normally a few modes (the principal components) are sufficient to describe all of the shapes approximately. Importantly, the points

representing the shapes in the mode space are grouped according to their main characteristics (21). Thus, PCA is capable of determining axes that account for the maximal variance. If PCA is applied, the two most significant modes can be used for classification/diagnostic purposes (22).

Results

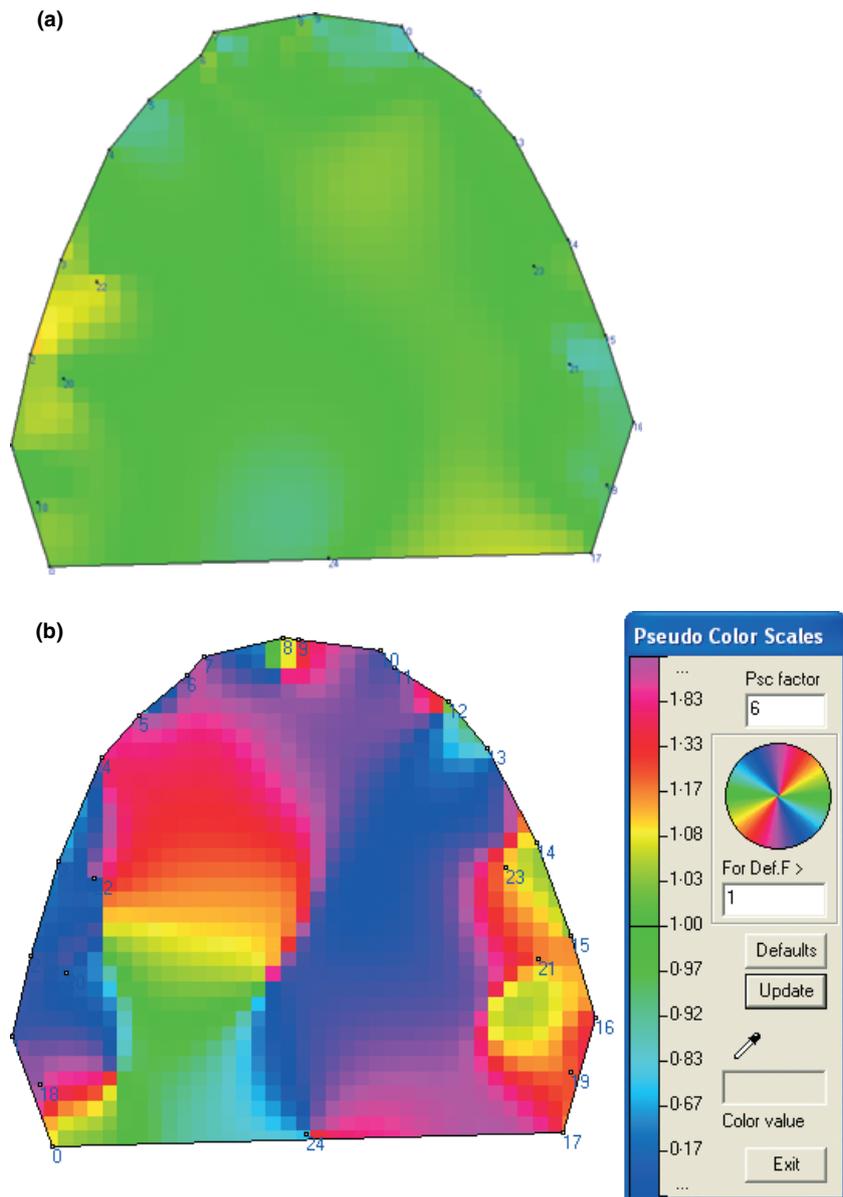
On duplicate digitization of the landmarks no significant differences were found ($P > 0.05$) using a method equivalent to Dahlberg's formula, and therefore the study digitization error was assumed to have no effect on the findings. In the current study, the BMI was significantly greater for the OSA group ($33.2 \text{ kg/m}^2 \pm 6.5$) when compared with the Control group ($22.7 \text{ kg/m}^2 \pm 3.5$, $P < 0.001$). In addition, the neck girth was greater for the OSA group ($43.4 \text{ cm} \pm 5.6$) when compared with the Control group ($35.8 \text{ cm} \pm 3.5$, $P < 0.001$). The AHI was also significantly greater for the OSA group ($42.9 \text{ per hour} \pm 29.6$) when compared with the Control group ($3.2 \text{ per hour} \pm 3.9$, $P < 0.001$). As well, the average and lowest oxygen saturations were significantly lower for the OSA group ($94.1\% \pm 4.4$, $77.1\% \pm 10.1$) when compared with the Control group ($97.8\% \pm 1.1$; $87.9\% \pm 7.1$, $P < 0.001$). These results are summarized in Table 1.

For OSA and Control upper dental arch configurations, the FEM results indicated that the normalized, mean OSA upper arch was significantly different when compared with the Control ($P < 0.05$). The pseudocolour-coded graphics produced by FEM indicated the regions in which size- or shape-changes were evident. Comparison of the upper OSA and Control configurations for size-change indicated asymmetrical changes with the mean OSA upper arch being 11% narrower in the incisor region ($P < 0.05$) and 7% narrower in canine region (Fig. 2a; $P < 0.01$). The direction of narrowing was in the oblique plane at about 45° (Fig. 2b; circular colour-scale, red and blue colouration). In support of these findings, ILA at landmarks 4–13 indicated that the mean OSA upper arch was 2% narrower in the transverse plane ($P = 0.038$) and 2.7% longer at landmarks 9–24 in the antero-posterior plane (Table 2; $P = 0.006$). Despite these findings, PCA failed to detect statistically significant changes when the upper arches of OSA and Controls were compared.

Table 1. Polysomnography data and physical examination findings subjected to independent *t*-tests with significance at $P < 0.05$

| Variables | OSA (s.d.) (<i>n</i> = 54) | Control (s.d.) (<i>n</i> = 54) | Mean difference (95% CI) | <i>P</i> -value |
|---|--------------------------------|------------------------------------|-----------------------------|-----------------|
| Apnoea-hypopnoea index (events per hour) | 42.9 (29.65) | 3.2 (3.95) | -39.6 (-47.76, -31.61) | 0.001 |
| Average oxygen saturation (%) | 94.1(4.44) | 97.8 (1.07) | 3.7 (2.50, 4.96) | 0.001 |
| Lowest oxygen saturation (%) | 77.1 (10.15) | 87.9(7.02) | 10.7 (7.46, 14.12) | 0.001 |
| Neck girth (cm) | 43.4 (5.64) | 35.8 (3.58) | -7.8 (-9.58, -5.97) | 0.001 |
| Body mass Index (kg/m ²) | 33.2 (6.59) | 22.7 (3.52) | -10.5 (-12.44, -8.62) | 0.001 |

OSA, obstructive sleep apnoea; s.d., standard deviation; CI, confidence interval.



| Landmarks | Definition | Change in distance | P-value |
|-----------|--|--------------------|---------|
| 4–13 | Maxillary inter-canine distance | 2–3% decrease | 0.038 |
| | Mandibular inter-canine distance | 3–4% decrease | 0.001 |
| 22–23 | Maxillary inter-first-pre-molar distance | NS | NS |
| | Mandibular inter-first-pre-molar distance | 3–5% decrease | 0.001 |
| 20–21 | Maxillary inter-second-pre-molar distance | NS | NS |
| | Mandibular inter-second-pre-molar distance | 3–5% decrease | 0.009 |
| 18–19 | Maxillary inter-molar distance | NS | NS |
| | Mandibular inter-molar distance | NS | NS |
| 9–24 | Maxillary length | 2–3% increase | 0.006 |
| | Mandibular length | 3–4% increase | 0.008 |

NS, no significant difference.

For the lower arch, the normalized mean OSA and Control dental arches were significantly different ($P < 0.05$). FEM of the lower OSA and Control configurations for size-change also indicated asymmetrical changes with the mean OSA lower arch being 10% narrower in the pre-molar region ($P < 0.05$) and 11% narrower in the molar region (Fig. 3a; $P < 0.05$). The direction of change was in the antero-posterior plane (Fig. 3b; purple colouration). In support of these findings, ILA at landmarks 4–13 indicated that the mean OSA lower arch was: 4% narrower in the transverse plane ($P = 0.001$); 3% longer at landmarks 9–24 in the antero-posterior plane ($P = 0.008$); 3% narrower in the transverse plane at landmarks 20–21 ($P = 0.009$), and 4% narrower in the transverse plane at landmarks 22–23 ($P = 0.001$). In further support of these findings, PCA indicated statistically significant changes when the lower arches of OSA and Controls were compared ($P < 0.001$) with the first two eigenvalues accounting for about 90% of the total shape-change.

Discussion

The strengths of the present study include relatively large samples of patients with OSA and Control subjects that underwent a limited hospital-based sleep study, and the deployment of robust geometric morphometric techniques for modelling and analysis. Nevertheless, the findings of the present study must be interpreted in the light of the following study limitations. First, the digital photographs of the study models cannot escape the limitations of 2D imaging. Second, generalization of the results to a larger population is limited because the sample subjects were care-seeking volunteers at a hospital clinic. This source of study subjects could introduce a potential selection bias into the investiga-

tion, and make our results more applicable to hospital populations rather than the general public. Third, while every attempt was made to match patients with OSA and to Control subjects for age and BMI, the OSA group was somewhat older and more obese. We believe that these differences may explain the presence of OSA in this group of patients but will not affect our observations in terms of any differences in dental arch morphology, similar to other study (4). Finally, generalization of the present results to different ethnicities, including other Asian populations is limited, because ethnic Malays may have unique cranio-facial features.

To date, six studies (3, 4, 6, 8, 23, 24) have used conventional techniques on study models to assess dental arch dimensions in Western subjects with OSA. Four of these investigations studied adults (3, 4, 6, 8) and two studied children (23, 24). In these studies, the control subjects' sleep states were confirmed by either questionnaire, clinical examination or by using the Epworth sleep scale. Out of these six studies, only two used PSG to confirm the Control group's sleep condition (6, 8). The results of this current study in Malaysian patients indicate that BMI and neck girth are significantly greater for the OSA group when compared with the Control group (Table 1). These findings support the view that obesity is one significant risk factor for OSA in this group of Malaysian patients, which is in good agreement with the findings obtained in Chinese (25) and Japanese populations (26). Contrary to the commonly held belief that OSA in Asians is found more frequently in non-obese subjects (27, 28), we found that severe OSA was found in obese Malaysian patients. This difference in findings could be because of environmental variation, as the majority of previously cited studies were undertaken on Chinese subjects living in Western societies. Thus, by utilizing

Table 2. Inter-landmark analysis for the upper and lower arch showing statistical differences by region

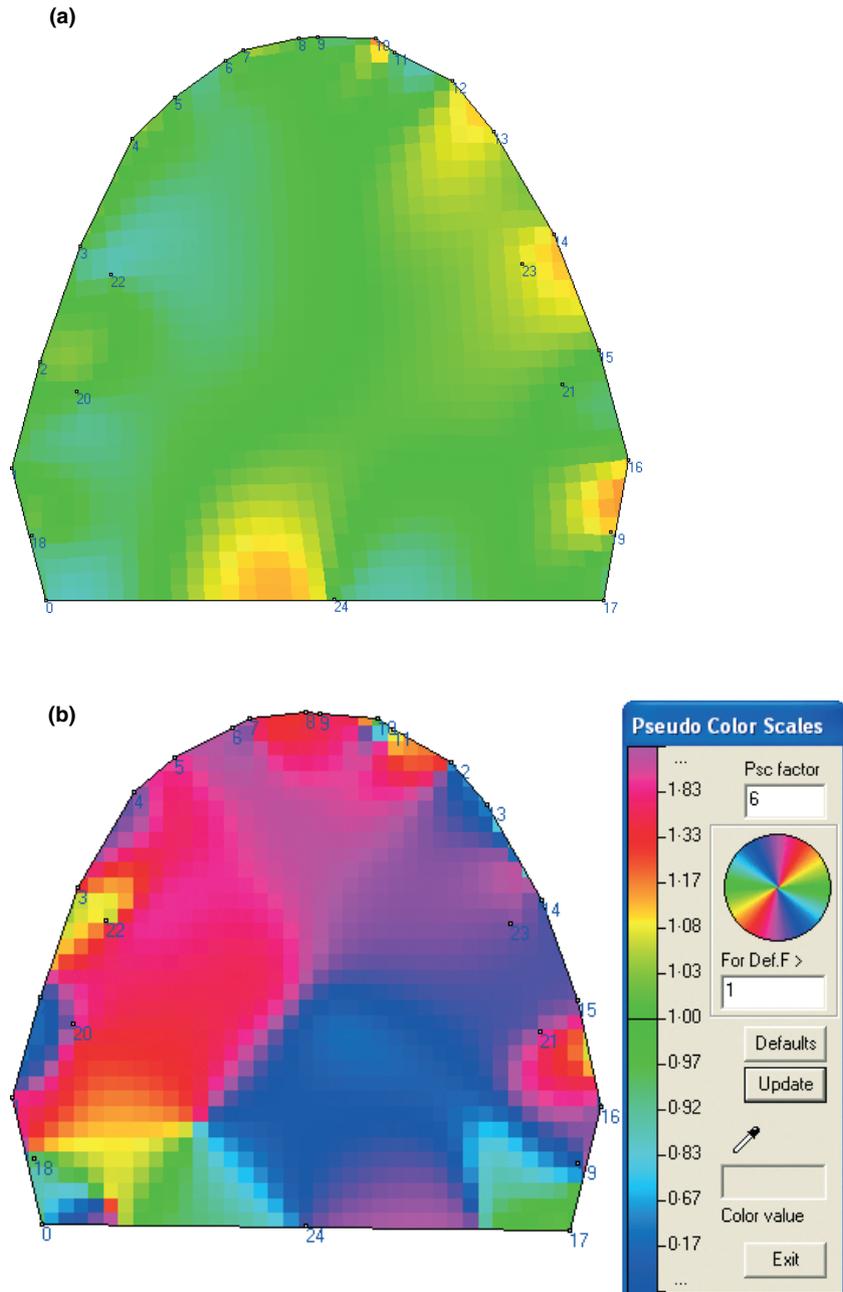


Fig. 3. (a) Finite-element morphometry (FEM) analysis of obstructive sleep apnoea (OSA) and Control lower arch configurations for size-change. Asymmetrical changes are noticeable with the mean OSA lower arch being 10% relatively narrower in the pre-molar region and 11% relatively narrower in the molar region. (b) FEM analysis for the direction of change in OSA and Control lower arch configurations. The circular colour scale indicates the direction of change. The direction of change is in the antero-posterior plane (purple colouration), consistent with arch shortening, as well as in oblique planes at about 45° (red and blue colourations), consistent with arch constriction.

the inter-ethnic variation hypothesis (29), which takes the influence of differences in dietary patterns, physical activity, level of education and public health awareness into account, the finding of OSA in obese Malaysian patients may be justified.

While most previous studies compared OSA dental arch morphology using conventional techniques (3, 4, 23, 24), a few studies (14, 15) have used FEM. To the best of our knowledge, there have been no previous studies that assess OSA dental arch morphology using

FEM analysis in Asian subjects. In this study, FEM indicated that Malaysian patients with OSA had significantly narrower maxillary and mandibular arch widths with an increase in maxillary and mandibular dental arch length when compared with Control subjects, even after correcting for size-variation. These findings support the notion that a narrow maxilla or mandible may lead to airway obstruction because of inadequate space for the tongue (30). Thus, when the space in the oral cavity is inadequate, it is possible that a

normal-sized tongue can gravitate to the back of the oro-pharynx when the palato-pharyngeal muscles become hypotonic during sleep, thus pre-disposing to OSA. Non-Asian patients with OSA have been reported to have narrower, more-tapered maxillary arches when compared with non-snoring, non-apnoeic Control subjects, which is in good agreement with our findings. In contrast, limited differences were found in maxillary depth and inter-tooth distances when patients with OSA and Control subjects were compared (3). While the exact causes of maxillary narrowing are not fully understood, a reduced maxillary inter-canine width in children was reported to be correlated with sleeping predominantly in a supine position (24). In addition, nasal airway features are also reported to be influenced by maxillary dimensions (6), and a familial tendency for maxillary narrowing has also been reported (7). Furthermore, narrow maxillary and mandibular inter-molar distances were also used in predictive models of OSA (8). These models illustrate the potential value of dental intra-oral examination in the screening and diagnosis of patients with OSA (31).

In this study, we also found that Malaysian patients with OSA tend to have increased maxillary and mandibular dental arch lengths compared with Control subjects. Increased maxillary dental arch length has also been reported in children aged 3–10 years suspected of having OSA (24). The exact cause of increased dental arch length in patients with OSA is not clearly understood. However, it has been hypothesized that severe obstruction of the posterior nasopharynx might increase the activity of the genioglossus muscle (which is important for tongue protrusion) to establish a sufficient oro-pharyngeal opening. Putatively, tongue protrusion may lead to increased lingual pressure on the anterior teeth, and simultaneous elongation of the dental arches with proclination of the incisors (24). In contrast, our data do not support the reports that adults with oro-pharyngeal obstruction have shorter lower dental arches (4). Similarly, shorter maxillary depths were reported also in children (23), which contrasts with our findings, but this difference may simply be attributable to inter-ethnic and chronologic variation. Moreover, none of the previous studies used PSG for control subjects, and this difference in methodology could contribute to the different results of the present study. In summary, Malaysian patients with OSA tend to have narrower but longer upper and lower dental arches. We suspect that these dental arch dimensions

may be associated with a shorter anterior cranial base (9), reflected by smaller antero-posterior facial dimensions. However, taking the present results into consideration, obese patients may show less obvious differences in facial profile because of the increased thickness of the soft-tissue drape (32). Nevertheless, we hypothesize that because of the cranio-caudal gradient of cranio-facial development, changes in maxillary dental arch size and shape lead to alterations in mandibular ontogeny. Thus, the mandibular arch plays a key role in the pathogenesis of OSA in Malaysian patients; a narrower mandible may represent a smaller airway because of concomitant effects of the tongue. But, further studies are needed to determine how these factors interact with pharyngeal parameters that contribute to the pathophysiology of OSA in obese patients.

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