

Hüseyin Özdemir
Remzi Altın
Ayhan Söğüt
Fikret Çınar
Kamran Mahmutyazıcıoğlu
Levent Kart
Lokman Uzun
Halit Davşancı
Sadi Gündoğdu
Nazan Tomaç

Craniofacial differences according to AHI scores of children with obstructive sleep apnoea syndrome: cephalometric study in 39 patients

Received: 12 December 2003
Revised: 10 February 2004
Accepted: 16 February 2004
Published online: 16 March 2004
© Springer-Verlag 2004

H. Özdemir (✉) · K. Mahmutyazıcıoğlu
H. Davşancı · S. Gündoğdu
Department of Radiology,
Medical School, Zonguldak Karaelmas
University, Kozlu, 67600
Zonguldak, Turkey
E-mail: hozdemir37@yahoo.com
Tel.: +90-372-2610169
Fax: +90-372-2610155

R. Altın · L. Kart
Department of Pulmonology,
Medical School, Zonguldak Karaelmas
University, Zonguldak,
Turkey

A. Söğüt · N. Tomaç
Department of Paediatrics,
Medical School,
Zonguldak Karaelmas University,
Zonguldak, Turkey

F. Çınar · L. Uzun
Department of Otolaryngology,
Medical School, Zonguldak Karaelmas
University, Zonguldak, Turkey

Abstract *Background:* Cephalometry is useful as a screening test for anatomical abnormalities in patients with obstructive sleep apnoea syndrome (OSAS). *Objective:* To evaluate comprehensively the cephalometric features of children with OSAS, with or without adenotonsillar hypertrophy, and to elucidate the relationship between cephalometric variables and apnoea-hypopnoea index (AHI) severity. *Materials and methods:* The study population consisted of 39 children, aged 4–12 years, with OSAS. Cephalometry was analysed using 11 measurements of the bony structures, their relationships and the size of the airways. Additionally, adenoid and tonsillar hypertrophy were graded. *Results:* Cranial base angles (BaSN and BaSPNS) were found to correlate with increasing levels of AHI scores ($P < 0.001$). Protrusion of the maxilla (SNA) and mandible (SNB) did not correlate with AHI scores ($P > 0.05$). The length of the mandibular plane (GnGo) and the minimal posterior

airway space (MPAS) were inversely correlated with AHI scores ($P < 0.001$). There was positive correlation between MPAS and GnGo ($r = 0.740$, $P < 0.001$), and negative correlation between MPAS and gonial angle (ArGoGn) ($r = -0.541$, $P < 0.001$). There was significant correlation between cephalometric data and adenotonsillar hypertrophy concerning BaSN, BaSPNS, ArGoGn, GnGoH, BaN-GnGo, MPAS, GnGO and MPH ($P < 0.001$). *Conclusions:* There is significant correlation between cephalometric data and AHI score severity in children with OSAS. Adenotonsillar hypertrophy affects the cephalometric measurements adversely. The study clearly mandates the institution of early and effective therapy of adenotonsillar hypertrophy in children with OSAS.

Keywords Skull · Radiography · Cephalometry · Obstructive sleep apnoea syndrome · Apnoea-hypopnoea index · Adenotonsillar hypertrophy · Child

Introduction

Obstructive sleep apnoea syndrome (OSAS) is a frequent, albeit underdiagnosed problem in children. It is a serious and potentially life-threatening disorder that is far more common than generally believed. If left un-

treated, OSAS may lead to substantial morbidity affecting multiple target organs and systems. The immediate consequences of OSAS in children include behavioural disturbance and learning deficits, pulmonary hypertension and compromised somatic growth [1]. However, if not treated promptly and early in the course

of the disease, OSAS may also impose long-term adverse effects on neurocognitive and cardiovascular function [2, 3]. Craniofacial anatomic risk factors are said to play a role in OSAS, together with the mechanism of upper airway compliance and muscle function [4, 5, 6]. Cephalometry is useful as a screening test for anatomical abnormalities in patients having OSAS [5, 7]. Many studies have used cephalometric analysis to assess the craniofacial morphological features of children and adults with OSAS [8, 9, 10, 11, 12], but the apnoea-hypopnoea index (AHI) severity and cephalometric findings in children have not been previously compared. In this study the aim was to evaluate comprehensively the cephalometric features of children with suspected OSAS, with or without adenotonsillar hypertrophy, and to elucidate the relationship between cephalometric variables and AHI severity.

Materials and methods

Study population

This study was carried out on 39 children with suspected OSAS, ranging in age from 4 to 12 years (mean \pm SD, 7.5 ± 1.7 years). There were 18 boys and 21 girls. Patients with a history of previous treatment of OSAS (including tonsillectomy and adenoidectomy), personal or family history of neuromuscular disorder or craniofacial syndrome, chronic lung disease or those who were referred for titration of continuous positive airway pressure therapy attributable to sleep-disordered breathing therapy were excluded. All patients underwent cephalometric and polysomnographic (PSG) examinations. Informed written consent was obtained from all patients.

Cephalometric analysis

A lateral projection of the skull was obtained using a tele Diagnost radiographic machine (Philips Medical Systems, The Netherlands). A cephalostat was used to keep the subject's head in a position such that the Frankfort horizontal line was parallel to the floor during exposure. A total of 11 angular (degrees) or linear (millimetres) variables related to both craniofacial skeletal and soft-tissue morphology were measured by a single observer in a single-blind manner. Every measurement was performed three times by the same observer, who did not know the clinical status of the patient, and the mean value of the two most proximate measurements was used for the statistical analyses to ensure reliability. The method consisted of recognising the landmarks by visual analysis and digitising the measurements of angles and distances using computer software. To allow correction for projection enlargement of the linear measurements, the radiographs were taken with a 21-mm, round, steel median calibration marker in place. The cephalometric landmarks, angles and linear measurements are defined in Table 1. Adenoid and tonsillar hypertrophy were graded according to Michal et al. [13]. Adenoid hypertrophy was graded using nasendoscopy as follows: grade 1, adenoid obstructing 1–25% of the choana; grade 2, adenoid obstructing 25–50% of the choana; grade 3, adenoid obstructing 50–75% of the choana; grade 4, adenoid obstructing 75–100% of the choana. Tonsillar hypertrophy was graded as follows: grade 1, tonsils are in the tonsillar pillar; grade 2, tonsils are protruding out of the tonsillar pillar; grade 3, tonsils

Table 1 Definitions of cephalometric landmarks, angles and linear measurements

	Definitions of cephalometric landmarks
S	Sella: midpoint of the fossa hypophysealis
N	Nasion: anterior point at the frontonasal suture
ANS	Anterior nasal spine: most anterior point of the nasal spine
PNS	Posterior nasal spine: most posterior point of the nasal spine
A	Deepest anterior point in the concavity of the anterior maxilla
Ar	Posterior ramus plane: the intersection of a line along the posterior border of the mandible and the inferior border of the basilar occipital bone
B	Deepest anterior point in the concavity of the anterior mandible
Go	Gonion: a mid-plane point at the gonial angle located by bisecting the posterior and inferior borders of the mandible
Gn	Gnathion: the most inferior point in the contour of the chin
Ba	Basion: most posteroinferior point on the clivus
H	Most anterosuperior point of the hyoid bone
	Definitions of cephalometric angles
ArGoGn	Angle formed by line connecting the articulate, gonion and gnathion
BaSN	Cranial base flexure: angle formed by intersection of lines drawn from the nasion to the sella and from the sella to the basion
BaSPNS	Angle formed by the line connecting the basion, sella, and posterior nasal spine
GnGoH	Hyoid angle: angle formed by line connecting the gnathion, gonion, and hyoid
SNA	Angle from sella to the nasion to the subspinal point
SNB	Angle from sella to the nasion to the supramental point
ANB	Angle from the subspinal point to the nasion to the supramental point
BaN-GnGo	Angle formed by the basal plane and the line connecting the gnathion and gonion
	Definitions of cephalometric linear measurement
MPAS	Retrolingual posterior air space: the minimal distance (in millimetres) between the base of the tongue and nearest point on the posterior pharyngeal wall
MPH	The distance between the mandibular plane and hyoid
GnGO	The length of the mandibular plane

reaching midpoint between anterior tonsillar pillar and uvula; grade 4, tonsils reach the uvula. Adenoid and tonsillar hypertrophy of grades 3 and 4 were considered pathological.

Sleep study

Full PSG can be successfully performed in infants and children of any age provided appropriate equipment and well-trained staff are available. Paediatric studies must be scored and interpreted using age-appropriate criteria. The American Thoracic Society has published a con-

sensus statement outlining the requirements for paediatric PSG, some of which are described below [14]. All patients underwent full overnight PSG 1 h before their usual bedtime. Sixteen parameters were recorded. Six EEG leads (bilateral frontal, central, and occipital), bilateral electro-oculographic leads, and submental electromyographic leads were applied. These surface leads were used to measure the stages of sleep, including time spent in rapid eye movement (REM) sleep. Motion of the chest wall and abdomen were measured by respiratory inductance plethysmography. Airflow from the nose and mouth was measured with thermocouples. Oxyhaemoglobin saturation was measured by pulse oximetry. Body motion was recorded with pretibial electromyographic leads. Cardiac rhythm was monitored with standard ECG leads.

In the present study, obstructive apnoea was defined as cessation of airflow for at least 10 s with paradoxical respiratory effort. Hypopnoea was defined as a reduction in airflow with synchronous chest wall and abdominal motion, resulting in either arousal or oxyhaemoglobin desaturation of at least 4%. Central apnoea was defined as a 10-s period without airflow or respiratory effort. The type, number and duration of respiratory events were noted, as well as the median and minimum oxyhaemoglobin saturation. Apnoea index (AI) was calculated as the average number of apnoeic episodes per hour of sleep. The AHI is defined as the average number of apnoeic plus hypopnoeic episodes per hour of sleep. In this study, OSAS was defined as the presence of clinical symptoms (habitual snoring, apnoea, etc.) and AI or AHI ≥ 1 .

Statistical analysis

Results were evaluated using SPSS 11.0 statistical program (SPSS Inc., Chicago, Ill., USA). Correlation coefficients were determined by the Pearson method for the associations between the cephalometric variables and degree of AHI score severity. The Pearson correlation method was also used to test the relationships of the craniofacial variables with age, BMI, gender and the adenotonsillar hypertrophy. The values are given as mean \pm standard deviation (SD), unless otherwise stated.

Results

Demographic characteristics of patients with OSAS are given in Table 2. Table 3 shows mean values of cephalometric measurements of the patients. The cephalometric landmarks, angles and linear measurements are given in Figs. 1, 2.

Table 2 Demographic characteristics of the patients

Demographic features	Descriptive values Mean \pm SD
Age range	7.5 \pm 1.7 years 4–12
BMI	18.4 \pm 3.5
Range	13–30
Gender	
Boys	21
Girls	18
AHI	5 \pm 5.1
Range	1–27
Mean saturation	96.4 \pm 1.5
Range	93–98
Lowest saturation	88.4 \pm 3.1
Range	77–93

Table 3 Cephalometric data of the all patients

Cephalometric data	Descriptive values Mean \pm SD
BaSN	126.5 \pm 2.6
Range	122–132
BaSPNS	56.3 \pm 2.0
Range	53–60
ArGoGn	131 \pm 3.4
Range	125–138
GnGoH	25.5 \pm 2.7
Range	22–33
SNA	79.7 \pm 1.2
Range	78–82
SNB	74.7 \pm 1.4
Range	73–78
ANB	5.2 \pm 0.7
Range	3.7–7.0
BaN-GnGo	57.6 \pm 2.1
Range	54–62.4
MPAS	11.9 \pm 1.8
Range	8.9–14.3
GnGO	64.4 \pm 2.9
Range	57–69
MPH	13.6 \pm 1.1
Range	11.7–16.7

Correlations between AHI and cephalometric measurements

Correlations between AHI scores and individual cephalometric measurements are given in Table 4. Decreasing cranial base angles (BaSN and BaSPNS) were found to correlate with increasing levels of AHI scores ($P < 0.001$). Increased gonial angle (ArGoGn) and hyoid angle (GnGoH) were associated with higher AHI scores ($P < 0.001$). Protrusion of the maxilla (SNA) and mandible (SNB) did not correlate with AHI scores ($P > 0.05$). There was also no correlation between SNB angle and length of the mandibular plane (GnGo) ($P > 0.05$). GnGo and the minimal posterior airway space (MPAS) were inversely correlated with AHI scores

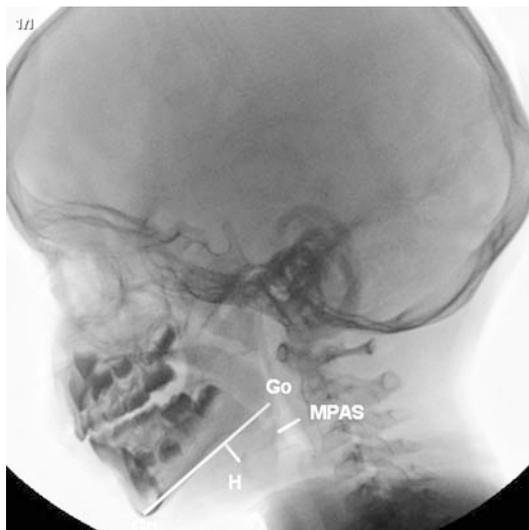


Fig. 1 The demonstration of cephalometric landmarks and linear measurements

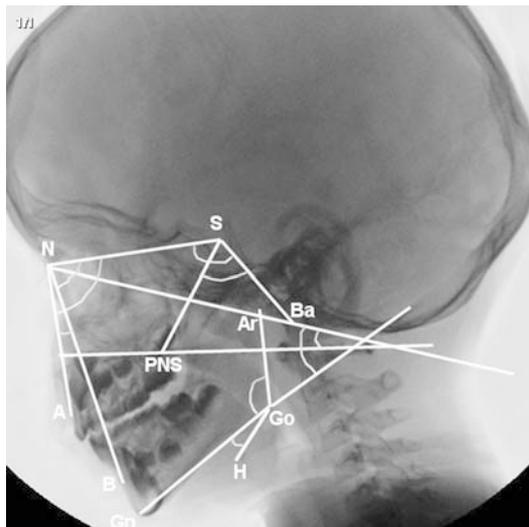


Fig. 2 The demonstration of cephalometric landmarks and angles

Table 4 Correlation between cephalometric data and AHI scores

Cephalometric measurements	AHI (<i>r</i>)	<i>P</i> value
BaSN	-0.671	<i>P</i> < 0.001
BaSPNS	-0.619	<i>P</i> < 0.001
ArGoGn	-0.587	<i>P</i> < 0.001
GnGoH	0.837	<i>P</i> < 0.001
SNA	0.144	<i>P</i> > 0.05
SNB	0.015	<i>P</i> > 0.05
ANB	0.110	<i>P</i> > 0.05
BaN-GnGo	0.730	<i>P</i> < 0.001
MPAS	-0.772	<i>P</i> < 0.001
GnGO	-0.813	<i>P</i> < 0.001
MPH	0.820	<i>P</i> < 0.001

(*P* < 0.001). There was positive correlation between MPAS and GnGo (*P* < 0.001), and negative correlation between MPAS and ArGoGn (*P* < 0.001).

The effects of confounding factors such as age, BMI and gender were investigated on cephalometric measurements. There were no correlations between these factors and cephalometric measurements (*P* > 0.05).

Correlation between AHI scores and adenoid hypertrophy

There was a significant correlation between AHI scores and adenoid hypertrophy (*r* = 0.780, *P* < 0.001).

Correlation between cephalometric data and adenoid hypertrophy

Correlation of cephalometric data and adenoid hypertrophy severity is given in Table 5. There were significant correlations between cephalometric data and adenoid hypertrophy concerning BaSN, BaSPNS, ArGoGn, GnGoH, BaN-GnGo, MPAS, GnGO and MPH (*P* < 0.001). It was also found that ANB angle was significantly correlated with adenoid hypertrophy (*P* < 0.05). When the effect of tonsillar hypertrophy severity was investigated on cephalometric measurements, there were statistically important correlations for BaSN (*P* < 0.001), BaSPNS (*P* < 0.001), ArGoGn (*P* < 0.05), GnGoH (*P* < 0.001), BaN-GnGo (*P* < 0.001), MPAS (*P* < 0.001), GnGO (*P* < 0.001) and MPH (*P* < 0.001). No correlation was found between SNA, SNB, ANB angles and tonsillar hypertrophy severity (*P* > 0.05).

Interrelationships among the craniofacial variables are given in Table 6. There were strong interrelations between cranial base parameters, the length of the mandibular plane, the minimal posterior airway space and gonial angle and maxillary and mandibular pro-

Table 5 Correlation of cephalometric data and adenoid hypertrophy (AH) severity

Cephalometric Measurement	AH severity (<i>r</i>)	<i>P</i> value
BaSN	-0.801	<i>P</i> < 0.001
BaSPNS	-0.700	<i>P</i> < 0.001
ArGoGn	0.625	<i>P</i> < 0.001
GnGoH	0.798	<i>P</i> < 0.001
SNA	0.119	<i>P</i> > 0.05
SNB	-0.104	<i>P</i> > 0.05
ANB	0.314	<i>P</i> > 0.05
BaN-GnGo	0.765	<i>P</i> < 0.001
MPAS	-0.816	<i>P</i> < 0.001
GnGO	-0.764	<i>P</i> < 0.001
MPH	0.784	<i>P</i> < 0.001

Table 6 Interrelationships among the craniofacial variables by Pearson correlation

	Age	BaSN	BaSPNS	ArGoGn	GnGoH	SNA	SNB	ANB	MPAS	GnGo
BaSN	NS									
BaSPNS	NS	<0.001								
ArGoGn	NS	<0.001	<0.001							
GnGoH	NS	<0.001	<0.001	<0.001						
SNA	NS	NS	NS	NS	NS					
SNB	NS	NS	NS	NS	NS	<0.001				
ANB	NS	NS	NS	NS	NS	NS	NS			
MPAS	NS	<0.001	<0.001	<0.001	<0.001	NS	NS	NS		
GnGo	NS	<0.001	<0.001	<0.001	<0.001	NS	NS	NS	<0.001	
MPH	NS	<0.001	<0.001	<0.001	<0.001	NS	NS	NS	<0.001	<0.001

trusion parameters. It was found that MPAS had a statistically significant correlation with ArGoGn and GnGo ($P < 0.001$).

Discussion

OSAS is a breathing disorder during sleep characterized by prolonged partial upper airway obstruction and/or intermittent complete obstruction that disrupts normal ventilation during sleep and normal patterns. The prevalence of childhood OSAS is difficult to estimate, largely because published studies use different PSG criteria for its ascertainment. Reports range from 0.7 to 10.3% [14]. The mechanisms responsible for upper-airway obstruction in children with OSAS are highly complicated and as yet not fully understood. OSAS in children is a dynamic process resulting from a combination of structural and neuromotor abnormalities, rather than from structural abnormalities alone. Certain forms of craniofacial anatomical defects, including mandibular deficiency, tongue and soft palate enlargement, and inferior displacement of the hyoid bone, have been suggested as predisposing factors for upper-airway obstruction during sleep [2, 15, 16, 17, 18, 19, 20]. Cephalometric analysis has been used to characterize skeletal and soft tissue relationships in children [8, 9, 10, 11, 12]. Cephalometry can provide extensive data on the landmarks pertinent to the upper airways. In the literature, many cephalometric studies have been done to study the craniofacial architecture of children with OSAS [4, 7]. However, correlative data between cephalometric parameters and AHI scores are sparse. In this study of children with OSAS, increased AHI scores were found to correlate with the following craniofacial differences demonstrated by cephalometry: skull-base angles (BaSN and BaSPNS), GnGo and ArGoGn, hyoid position as given by MPH and GnGoH, and MPAS at the level of the tongue base.

Cranial base components (BaSN and BaSPNS) are considered to be of primary importance in facial equilibrium [11] and do not change greatly from childhood to adulthood [21]. Excessive flexure or relatively acute angulation of the skull base refers to a decreased BaSN

measurement. We found that nearly all the reductions of the BaSN in patients with severe obstructions were caused by reduction of the BaSPNS, which defines the bony limits of the nasopharyngeal space. Bacon et al. [11] noted a similar reduction of BaSPNS in adults with obstructive sleep apnoea. They found that the reduction in BaSPNS was associated with decreased sagittal facial length, which they interpreted as facial and pharyngeal compression. Thus it appears that acute angulation of the skull base is associated with posterior displacement of the facial skeleton and shortening of the AP dimensions of the pharynx [11, 22]. In addition, excessive cranial-base flexure may play a role in the development of OSAS.

The position in the sagittal direction of the maxilla and mandible is established by using the SNA and SNB angles [7]. Decreased SNA is associated with retroposition of the maxilla. Our SNA measurement was comparable with the published literature [4, 23]. However, we did not find any significant correlation between SNA angle and AHI scores in the present study.

Retroposition of the mandible, indicating future vertical growth, is also important and is usually assessed by the SNB angle. Hochban and Brandenburg [24] studied 400 patients with OSAS and found that retroposition of mandible was associated with reduced retrobasilingual space. Retroposition of the mandible was also associated with a shorter corpus (GnGo length). Only this measurement was considered potentially abnormal in the patients with OSAS in the meta-analysis by Miles et al. [25] Although SNB angle in our study was comparable with the literature [4], we found no significant correlation between AHI scores and SNB measurements. This might be due to interdependence between BaSN and these measures of maxillary and mandibular protrusion [4].

MPAS is another important measurement in the evaluation of patients with OSAS. We found that MPAS had a statistically significant correlation with ArGoGn and GnGo (Table 6). In children with a more obtuse ArGoGn and shorter GnGo, AHI scores tended to be more pronounced. ArGoGn and GnGo measurements were compatible with published data [5]. Since the genioglossus muscle has its origin at the internal surface of

the anterior mandible, both shortening of the GnGo and an increase in the ArGoGn result in relative retrodisplacement of the tongue (as in our study). Furthermore, these mandibular changes result in reduced protrusor mechanical efficiency of the genioglossus muscle, which could contribute to posterior airway obstruction, as the muscle tone is diminished during sleep [26, 27]. In the present study, we found inverse correlation between MPAS and ArGoGn. On the other hand, there was positive correlation between MPAS and GnGo. These correlations were also in agreement with previous studies [4, 27].

Caudal placement of the hyoid bone has repeatedly been shown to correlate with OSAS in children [8], and this was confirmed in our study; MPH distance and GnGoH angle were positively correlated with increased AHI scores. The overall effects of caudal hyoid placement include narrowing of the MPAS and decreased mechanical efficiency of the genioglossus muscle protrusor action causing OSAS.

Many causes have been found to lead to OSAS, but adenotonsillar hypertrophy remains the most common [13, 28]. Correlation of OSAS severity with adenotonsillar size remains to be established. A lack of correlation between adenoid size and severity of apnoea has also been documented [29]. In contrast, Jain and Sahni [28] reported a correlation between increased adenoid size and AHI scores, whereas a correlation between tonsillar hypertrophy and AHI scores was not observed. They calculated the adenotonsillar size in millimeters and graded tonsillar size according to a four-level scale. We graded adenoid hypertrophy with regard to choanal obstruction using nasendoscopy. We found significant correlation between adenoid and tonsillar severity grades and AHI scores. Shintani et al. [30] compared cephalometric results in children with OSAS and in age-matched controls. They found decreased angles of SNA and SNB in children with adenotonsillar hypertrophy. We did not find a correlation between SNA and SNB angles and adenotonsillar hypertrophy.

Children with adenoidal hypertrophy have nasal obstruction with consequent chronic mouth breathing [4]. Adverse developmental effects of chronic mouth breathing include increased height of the nasomaxillary complex, obtuse ArGoGn, and secondary caudal shifting of the hyoid, as has been shown in adults [31]. In addition, we have shown that both adenoid and tonsillar hypertrophy severity were positively correlated with ArGoGn, GnGoH, BaN-GnGo, and MPH. Adenoid and tonsillar hypertrophy severity was negatively correlated with BaSN, BaSPNS, MPAS and GnGo. These results were in agreement with previous studies [31, 32]. Additionally, these results suggest that children who are chronic mouth breathers owing to adenoid hypertrophy develop secondary changes in craniofacial morphologic characteristics that predispose them to adulthood OSAS. Therefore, it is well documented that adenoidectomy is effective in relieving snoring in children as it relieves nasopharyngeal obstruction. However, excessive flexure of the cranial base is a risk factor for incomplete clinical response to adenoidectomy [33]. Because excessive cranial base flexure may play a role in the development of OSAS the decrease in operating length of the pharyngeal dilator muscles compromises their efficiency [12, 33]. Guilleminault et al. [33] suggested that maintaining nasal breathing during childhood is important for preventing alterations of the facial skeleton. Therefore, we suggest that nasal obstruction of long duration must be avoided throughout the growing process. Persistent nasal obstruction should be corrected surgically early in life, even if, in most cases, the original cause will resolve spontaneously.

In conclusion, the present study shows that there is considerable correlation between cephalometric data and AHI score severity in children with OSAS. Adenotonsillar hypertrophy affects the cephalometric measurements adversely. The currently available information clearly mandates the institution of early and effective therapy for adenotonsillar hypertrophy in children with OSAS.

References

1. Brouillette RT, Fernbach SK, Hunt CE (1982) Obstructive sleep apnea in infants and children. *J Pediatr* 100:31–40
2. Lipton AJ, Gozal D (2003). Treatment of obstructive sleep apnea in children: do we really know how? *Sleep Med Rev* 7:61–80
3. Marcus CL (2001) Sleep-disordered breathing in children. *Am J Respir Crit Care Med* 164:16–30
4. Finkelstein Y, Wexler D, Berger G, et al (2000) Anatomical basis of sleep-related breathing abnormalities in children with nasal obstruction. *Arch Otolaryngol Head Neck Surg* 126:593–600
5. Miyao E, Miyao M, Ohta T, et al (2000) Differential diagnosis of obstructive sleep apnea syndrome patients and snorers using cephalograms. *Psychiatry Clin Neurosci* 54:659–664
6. Marcus CL, McColley SA, Carroll JL, et al (1994) Upper airway collapsibility in children with obstructive sleep apnea syndrome. *J Appl Physiol* 77:918–924
7. Paoli JR, Lauwers F, Lacassagne L, et al (2001) craniofacial differences according to the body mass index of patients with obstructive sleep apnoea syndrome: cephalometric study in 85 patients. *Br J Oral Maxillofac Surg* 39: 40–45

8. Jamieson A, Guilleminault C, Partinen M, et al (1986) Obstructive sleep apneic patients have craniomandibular abnormalities. *Sleep* 9:469–477
9. Strelzow VV, Blank RH, Basile A, et al (1988) Cephalometric airway analysis in sleep related breathing disorders. *Laryngoscope* 98:1149–1158
10. DeBerry-Borowiecki B, Kukwa AA, Blanks RH (1988) Cephalometric analysis for diagnosis and treatment of obstructive sleep apnea. *Laryngoscope* 98:226–234
11. Bacon WH, Krieger J, Turlot JC, et al (1988) Craniofacial characteristics in patients with sleep apneas syndrome. *Cleft Palate J* 25:374–378
12. Maltais F, Series F, Carrier G, et al (1991) Cephalometric measurements in snorers, non-snorers and sleep-apnea patients. *Thorax* 46:419–423
13. Greenfeld M, Tauman R, DeRowe A, et al (2003) Sleep related breathing disorders due to adenotonsillar hypertrophy in infants. *Int J Pediatr Otorhinolaryngol* 67:1055–1060
14. Schechter MS; Section on Pediatric Pulmonology, Subcommittee on Obstructive Sleep Apnea Syndrome (2002) Technical report: diagnosis and management of childhood obstructive sleep apnea syndrome. *Pediatrics*. 109:e69
15. Bacon WH, Turlot JC, Krieger J, et al (1990) Cephalometric evaluation of pharyngeal obstructive factors in patients with sleep apneas syndrome. *Angle Orthod* 60:115–122
16. Isono S, Remmers JE, Tanaka A, et al (1996) Static properties of the passive pharynx in sleep apnea. *Sleep* 19:175–177
17. Ellis PD, Williams JE, Shneerson JM (1993) Surgical relief of snoring due to palatal flutter: a preliminary report. *Ann R Coll Surg Engl* 75:286–290
18. Croft CB, Pringle M (1991) Sleep nasendoscopy: a technique of assessment in snoring and obstructive sleep apnea. *Clin Otolaryngol* 16:504–509
19. Lugaresi E, Cirignotta F, Montagna P (1989) Snoring: pathogenesis, clinical and therapeutic aspects. In: Kryger MH, Roth T, Dement WC (eds) *Principles and practice of sleep medicine*. Saunders, Philadelphia, pp 494–500
20. Nelson S, Hans M (1997) Contribution of craniofacial risk factors in increasing apneic activity among obese and non-obese habitual snorers. *Chest* 111:154–162
21. Engman LT, Spriestersbach DC, Moll KL (1965) Cranial base angle and nasopharyngeal depth. *Cleft Palate J* 45:32–39
22. Harris EF (1993) Size and form of the cranial base in isolated cleft lip and palate. *Cleft Palate Craniofac J* 30:170–174
23. Zucconi M, Strambi L, Ferini SP, et al (1993) Craniofacial cephalometric evaluation in habitual snorers with and without obstructive sleep apnoea. *Otolaryngol Head Neck Surg* 109:1007–1013
24. Hochban W, Brandenburg U (1994) Morphology of the viscerocranium in obstructive sleep apnoea syndrome: cephalometric evaluation of 400 patients. *J Craniomaxillofac Surg* 22:205–213
25. Miles PG, Vig PS, Weyant RJ, et al (1996) Craniofacial structure and obstructive sleep apnoea syndrome: a qualitative analysis and meta-analysis of the literature. *Am J Orthod Dentofacial Orthop* 109:163–172
26. Kuna S, Remmers JE (1985) Neural and anatomic factors related to upper airway occlusion during sleep. *Med Clin North Am* 69:1221–1242
27. Brown IG, Bradley TD, Phillipson EA, et al (1985) Pharyngeal compliance in snoring subjects with and without obstructive sleep apnea. *Am Rev Respir Dis* 132:211–215
28. Jain A, Sahni JK (2002) Polysomnographic studies in children undergoing adenoidectomy and/or tonsillectomy. *J Laryngol Otol* 116:711–715
29. Grundfast KM, Wittich DJ Jr (1982) Adenotonsillar hypertrophy and upper airway obstruction in evolutionary perspective. *Laryngoscope* 92:650–656
30. Shintani T, Asakura K, Kataura A (1998) The effect of adenotonsillectomy in children with OSAS. *Int J Pediatr Otorhinolaryngol* 44:51–58
31. Woodside DG, Linder-Aronson S, Lundstrom A, et al (1991) Mandibular and maxillary growth after change mode of breathing. *Am J Orthod Dentofacial Orthop* 100:1–18
32. Tomer BS, Harvold EP (1982) Primate experiments on mandibular growth direction. *Am J Orthod* 82:114–119
33. Guilleminault C, Partinen M, Praud JP, et al (1989) Morphometric facial changes and obstructive sleep apnea in adolescents. *J Pediatr* 114:997–999

Copyright of Pediatric Radiology is the property of Kluwer Academic Publishing / Academic and its content may not be copied or emailed to multiple sites or posted to a listserv without the copyright holder's express written permission. However, users may print, download, or email articles for individual use.