

Cephalometric characteristics of nonobese patients with severe OSA

Eung-Kwon Pae, DDS, MSc, PhD; Kathleen A. Ferguson, MD, FRCPC, FCCP

Abstract: The purpose of this study was to determine the facial characteristics of nonobese patients with obstructive sleep apnea (OSA). Observational data on a cohort of patients was analyzed retrospectively. The subjects were classified into four groups: nonobese mild, obese mild, nonobese severe, and obese severe. The nonobese mild group included patients with a body mass index (BMI = kilogram/meter²) <25 and an apnea-hypopnea index (AHI) >5 and <15; the obese mild patients had a BMI >35 and an AHI >5 and <15; the nonobese severe patients had a BMI <25 and an AHI >40; the obese severe group had a BMI >35 and AHI >40. Thirty-three male patients referred for overnight polysomnography and lateral cephalometry who met the selection criteria were included. Between-group differences were examined pairwise by analysis of variance (ANOVA) with Bonferroni correction. Only two variables—lower facial height and overbite—were significantly different at $p < 0.05$ between the nonobese severe group and the obese mild group. A discriminant analysis on the cephalometric measurements revealed that patients in the nonobese severe group could be distinguished from patients in other groups by their facial characteristics. OSA patients do not have a homogenous bony structure of the face. In particular, OSA severity in nonobese severe patients may be associated with a vertical skeletal disharmony.

Key Words: Cephalometry, Lower facial height, OSA, Overbite, Upper airway

Although obstructive sleep apnea (OSA) patients tend to be obese, a significant number of them are not. A recent study suggested that fat deposition around the neck may be a factor associated with OSA in nonobese patients.¹ However, previous studies suggest that the orofacial skeletal structure of the nonobese OSA patient may differ from that of the obese OSA patient, and that the distinctive bony structure may lead to the development of OSA in nonobese individuals.^{2,3}

Lateral cephalometry is one of many imaging techniques that has been used to investigate the facial characteristics of OSA patients. Cephalometric differences between OSA patients and snoring and nonsnoring subjects have been documented.⁴⁻⁸ Lateral cephalometric characteristics of the bony structure in OSA patients include a retruded mandible, a low hyoid bone, and large overbite and overjet. Soft tissue characteristics include a long soft palate, a long and

large tongue, and a long pharynx. In fact, these structural characteristics are quite distinctive, suggesting that simply increasing body weight in nonapneic subjects would not necessarily cause them to develop OSA. This is supported by the fact that not all obese men and women have OSA, while some thin or normal weight individuals do.¹ Although weight control is a routine prescription for overweight OSA patients, weight loss does not always alleviate OSA.^{9,10}

We hypothesized that the orofacial structure of nonobese patients (BMI <25) with severe OSA (AHI >40) may differ from that of their obese counterparts (BMI >35). This implies that differences in the anatomical structure of the upper airway may be related to the development and progression of OSA. This observational study of orofacial bony structure was designed to investigate the existence of anatomical differences between nonobese and obese patients with OSA.

Author Address

Dr. Eung-Kwon Pae
Department of Orthodontics
University of Connecticut, School of Dental Medicine
Farmington, CT 06030-1725
E-mail: pae@up.uchc.edu

Eung-Kwon Pae, assistant professor, Department of Orthodontics, School of Dental Medicine, University of Connecticut Health Center, Farmington, Conn.

Kathleen A. Ferguson, assistant professor, Division of Respiratory, Department of Medicine, London Health Sciences Centre, The University of Western Ontario, London, Ontario, Canada.

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Materials and methods

Subjects

Male patients were selected from a cohort of individuals who had undergone overnight polysomnography, lateral cephalometry, complete medical and sleep history, and physical examination. The study population was divided, based on body mass index (BMI, kg/m^2), into a nonobese ($\text{BMI} < 25$) and an obese ($\text{BMI} > 35$) group. These groups were subdivided based on apnea/hypopnea index (AHI; number of apneas and hypopneas per hour total sleep time) into a mild ($5 < \text{AHI} < 15$) and a severe ($\text{AHI} > 40$) group. This process retained 33 OSA patients in the study sample out of a total of approximately 340 subjects.

Cephalometric variables

Cephalograms were taken at natural head posture in a standing body position. A detailed description of the cephalometric technique has been published.¹¹ Six linear and seven angular measurements were measured from the tracings by a ruler and a protractor up to a half-millimeter and a half-degree (Figure 1). Linear measurements included lower facial height (LFH, the linear distance between ANS and the bottom of the mandibular symphysis), total facial height (TFH, the linear distance between N and the bottom of the mandibular symphysis), overbite (OB, the vertical overlap of the maxillary and mandibular anterior teeth), overjet (OJ, the horizontal overlap of the maxillary and mandibular anterior teeth), mandibular plane to hyoid bone (MPH, the linear distance along a perpendicular line from the hyoid bone to the mandibular plane), and vertical airway length (VAL, the linear distance between the PNS and the base of the epiglottis). Angular measurements included SNA (the inner angle formed by the connection of

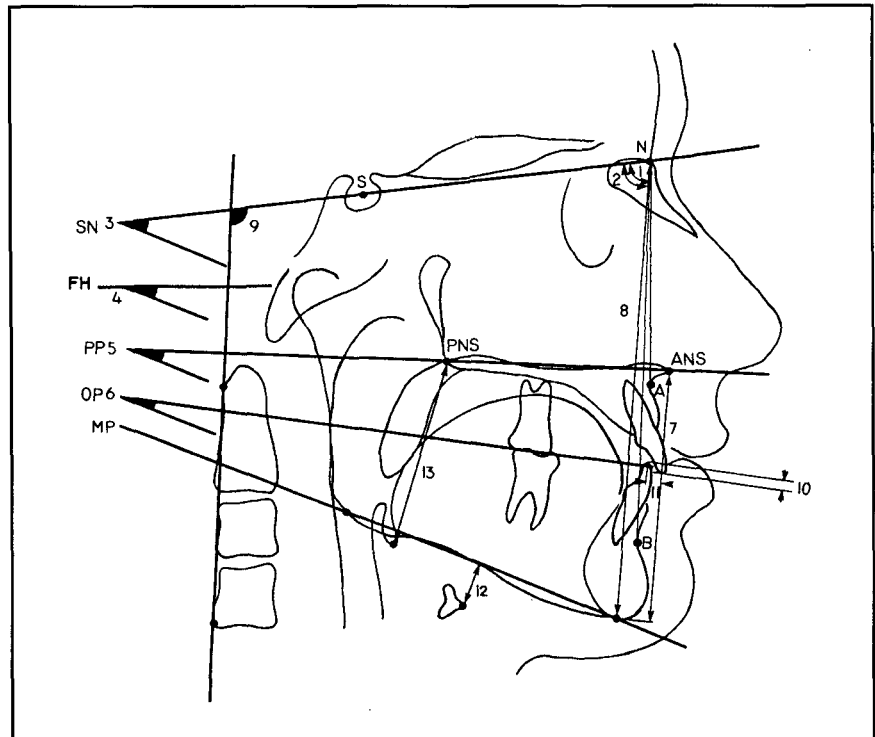


Figure 1

Diagrammatic representation of the anatomic points and planes used to identify craniofacial parameters on lateral cephalometric radiographs. S = center of sella turcica; N = nasion; ANS = anterior nasal spine; A = subspinale; B = supramentale; PNS = posterior nasal spine; SN = SN plane; FH = Frankfort horizontal plane; PP = palatal plane; MP = mandibular plane. Measurements: 1. SNA; 2. SNB; 3. SNMP; 4. FHMP; 5. PPMP; 6. OPMP; 7. LFH; 8. TFH; 9. HP; 10. OB; 11. OJ; 12. MPH; 13. VAL.

S [sella], N [nasion] and A [subspinale—the deepest point on the anterior surface of the maxillary alveolar bone] in that order), SNB (the inner angle between S, N, and B [supramentale—the deepest point on the anterior surface of the mandibular alveolar bone]); SNMP (the angle created by extension of the line from S to N and the mandibular plane); FHMP (the angle created by extension of the Frankfort horizontal plane and the mandibular plane); PPMP (the angle created by extension of the palatal plane and the mandibular plane); OPMP (the angle created by extension of the occlusal plane and the mandibular plane); and the head posture angle (HP, the angle created by the line from the second and fourth vertebral points and the line from S to N).

Overnight polysomnography

Polysomnography was performed on one night on all patients. The sleep study included electroencephalogram (EEG, C3/A2, C4/A1, O2/A1), electro-oculogram (EOG), submental electromyogram (EMG), left and right anterior tibialis EMG, electrocardiogram (ECG), thoraco-abdominal motion, oronasal airflow (expired CO_2), and arterial oxygen saturation with pulse oximetry using an ear probe sensor. The studies were scored manually, and the total apnea and hypopnea index (AHI) was calculated for the night. Obstructive apneas were defined as the cessation of airflow for at least 10 seconds accompanied by ongoing respiratory effort. Hypopneas were defined as a reduction in airflow of at least 50% for at least 10 seconds

accompanied by a reduction in respiratory effort and by an arousal or an arterial oxygen desaturation of at least 3%.

Statistical analysis

An ANOVA with Bonferroni correction for multiple comparison was performed for the demographic data, apnea indices, and cephalometric variables in order to examine differences between groups. This ANOVA test indicates which combinations of comparisons are statistically significant. A discriminant analysis was performed to check if the subgrouping based on BMI and AHI was equated with the statistical classification based on cephalometric measurements. All statistical analyses were performed with SPSS version 6.1.4. The statistical significance, $p < 0.05$, was considered to be clinically significant.

Results

Study patients

The 33 male patients were divided into four groups based on apnea severity and obesity: 9 subjects in the nonobese mild group, 9 in the nonobese severe group, 7 in the obese mild group, and 8 in the obese severe group. Demographic and polysomnographic data are summarized in Table 1. There was no statistical difference in age between the groups. Neither weight nor BMI differed significantly between the nonobese mild and the nonobese severe groups or between the mild obese and the severe obese groups. AHI was greater in the obese severe group than that in the nonobese severe group ($p < 0.005$), but it was the same in both mild groups.

Cephalometric variables

The linear and angular measurements from lateral cephalometry are presented in Table 2. The table shows that two variables, LFH and OB, differed significantly between

Table 1
Anthropometric measurements

	Nonobese mild (BMI<25, AHI<15)	Nonobese severe (BMI<25, AHI>40)	Obese mild (BMI>35, AHI<15)	Obese severe (BMI>35, AHI>40)
N = 33	9	9	7	8
Age (yrs)	49.11 ± 9.47	54.44 ± 10.47	45.86 ± 8.13	40.63 ± 12.61
AHI	7.65 ± 5.23	48.65 ± 8.48	10.77 ± 4.84	84.84 ± 31.44
BMI	24.46 ± 1.42	24.69 ± 1.86	37.03 ± 3.15	39.34 ± 5.55
Weight (Kg)	76.06 ± 9.14	76.40 ± 12.55	115.29 ± 12.62	118.50 ± 19.15

Mean ± SD (standard deviation)
BMI (body mass index) = weight in Kg/height² in meters; AHI (apnea hypopnea index)

Table 2
Comparisons of cephalometric measurements between groups

	Nonobese mild (9)	Nonobese severe (9)	Obese mild (7)	Obese severe (8)
SNA (degrees)	80.39 ± 3.77	80.67 ± 4.12	82.79 ± 1.93	81.75 ± 3.87
SNB (degrees)	76.67 ± 2.93	76.72 ± 3.29	78.50 ± 6.65	77.00 ± 4.22
SNMP (degrees)	33.50 ± 6.76	28.33 ± 6.37	32.50 ± 9.80	32.00 ± 5.26
FHMP (degrees)	31.56 ± 5.46	23.83 ± 6.90	29.14 ± 9.76	26.75 ± 5.82
PPMP (degrees)	24.17 ± 7.69	17.83 ± 4.73	24.86 ± 7.81	21.81 ± 6.04
OPMP (degrees)	17.67 ± 5.64	13.11 ± 4.53	14.71 ± 5.26	16.75 ± 3.28
LFH (mm)	75.61 ± 5.19	69.50 ± 5.70 --*	78.36 ± 5.63	76.25 ± 5.55
TFH (mm)	133.11 ± 6.47	127.39 ± 8.16	135.07 ± 7.71	132.13 ± 5.69
HP (degree)	107.22 ± 5.96	108.94 ± 9.50	114.14 ± 6.65	115.63 ± 7.47
OB (mm)	3.00 ± 2.82	5.17 ± 1.62 --*	1.71 ± 2.38	4.13 ± 2.40
OJ (mm)	5.06 ± 2.19	5.28 ± 3.28	3.57 ± 2.51	4.81 ± 3.63
H (mm)	25.11 ± 10.04	24.11 ± 9.98	22.71 ± 4.91	25.56 ± 5.40
VAL (mm)	82.50 ± 7.77	81.22 ± 7.51	83.14 ± 3.57	85.19 ± 6.23

* $p < 0.05$

Table 3
Canonical discriminant functions and significance

Functions	Eigenvalue	% of variance	Cumulative %	Canonical correlation
1	3.379	71.1	71.1	0.878
2	0.957	20.1	91.2	0.699
3	0.416	8.8	100.0	0.542

Functions	Wilks' Lambda	Chi-square	df	p
1 - 3	0.082	58.66	39	0.022 *

the nonobese severe group and obese mild groups ($p < 0.05$). However, when obese and nonobese patients were pooled and all mild and severe patients compared, OB was the only cephalometric variable that differed between groups ($p < 0.05$, 2.53 ± 2.70 mm for the mild group and 4.58 ± 2.24 mm for the

severe group). The results of a discriminant analysis are presented in Tables 3, 4, and 5. Three significant ($p = 0.022$) discriminant functions were obtained (Table 3). Coefficients for the functions are provided in Table 4. Table 5 reveals how the original subgroupings based on BMI and AHI differ from

Table 4
Canonical discriminant function coefficients

	Function		
	1	2	3
FHMP	-0.548	-2.252	0.292
H	0.256	-0.753	0.206
HP	1.357	0.520	-0.149
LFH	0.071	0.126	1.839
OB	-1.171	-0.046	0.455
OJ	-0.035	-0.193	-0.351
OPMP	-0.457	-0.070	1.282
PPMP	2.486	-0.435	-1.457
SNA	-0.164	0.404	-0.370
SNB	0.897	0.074	0.309
SNMP	-2.047	2.726	0.268
TFH	1.250	-1.096	-1.224
VAL	-1.060	1.242	-0.020

Variables are ordered by absolute size of correlation with function.

the classifications yielded by the discriminant functions based on cephalometric variables. The results show that all subjects in the obese groups were classified correctly. However, two subjects in the nonobese mild group and one subject in the nonobese severe group appeared to be misgrouped.

Discussion

Nonobese patients with severe OSA may have a short lower facial height and a deep overbite. Excessive overbite usually results from a mandible that is small and/or retruded relative to the maxilla. However, cephalometric variables assessed in this study suggest that the anteroposterior relationship of the mandible to the maxilla may not be the major reason for the large overbite in the group of nonobese severe patients since the mean ANB angle for the nonobese severe group was not significantly smaller than that of the other groups (Table 2). The results suggest that this particular group of patients had a short lower facial height, which results in excessive overbite.

A large overbite in combination

Table 5
Classification of subjects by canonical discriminant functions

	Predicted group			
Original group	1	2	3	4
Nonobese mild (n=9)	7 (77.8%)			2
Nonobese severe (n=9)		8 (88.9%)		1
Obese mild (n=7)			7 (100%)	
Obese severe (n=8)				8 (100%)

Percentages in parentheses indicate the proportion of subjects classified correctly by three canonical functions.

with short anterior facial height may indicate a small oral cavity. Orthodontists have considered bite depth to be a barometer of genioglossus (GG) muscle activity, because a hyperbasal tone of the GG muscle has been known to provoke a shallow bite or an anterior openbite.¹² Comparing characteristics of patients in other groups, a large overbite in combination with short anterior facial height could be translated as poor compensatory action of the genioglossus muscle against the crowded space. Insufficient activity of the tongue protruder, GG, against airflow limitation during sleep¹³ in nonobese OSA patients may maintain a deep overbite and a vertically short face. Conversely, due to strong masticatory muscles, the bony and dental cage for the tongue is so strong and tight¹⁴ that gaining a small amount of weight could increase apnea severity, even in the absence of morbid obesity. However, both explanations deserve further study.

Three discriminant functions were constituted to cluster the subjects into four subgroups. The functions show that most subjects in each group appeared to have cephalometric characteristics of their own group. However, two subjects in the nonobese mild group and one subject in the nonobese severe group would have been placed in the obese severe group (Table 5). In other words, they appeared to have cephalomet-

ric characteristics of an obese severe patient. This could mean that, as they gain weight, they might develop severe OSA, as measured by the AHI.

We excluded subjects with a BMI between 25 and 35 and an AHI between 15 and 40 from the study sample. The patients for this study were selected at the extremes of the spectrum of OSA to determine which skeletal characteristics would differ between nonobese severe patients and others. The differences between groups had to be large to be clinically meaningful and statistically significant. A recent study reported that fat deposition in the neck might contribute to OSA in nonobese patients.¹ However, those researchers did not quantify any skeletal variability in the subjects, whereas the limitation in our study is the lack of fat deposition data.

Many previous morphometric studies have focused on a comparison between OSA patients and asymptomatic normal subjects.^{4,5} When all OSA patients are pooled, weight becomes a strong predictor of OSA severity.¹⁵ The current study could not escape this limitation. The mean value of AHI for the obese severe group was significantly different from that of the nonobese severe group. Due to the subject selection criteria, we included any obese OSA patient with an AHI greater than 40. This may be why the variation in AHI was so large. The current study attempted

to view OSA patients from a different perspective, by subgrouping them based on the assumption that all OSA patients do not have similar skeletal structures and that obese and nonobese patients may be dissimilar.

Conclusions

The present study showed that nonobese patients with severe OSA tend to have a short lower facial height and deep overbite. We speculate that OSA problems in nonobese patients may be associated with a vertical skeletal disharmony of the oral cavity.

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References

1. Mortimore IL, Marshall I, Wraith PK, Sellar RJ, Douglas NJ. Neck and total body fat deposition in nonobese and obese patients with sleep apnea compared with that in control subjects. *Am J Respir Crit Care Med* 1998; 157:280-283.
2. Ferguson KA, Ono T, Lowe AA, Ryan F, Fleetham J. The relationship between obesity and craniofacial structure in obstructive sleep apnea. *Chest* 1995; 108:375-381.
3. Tsuchiya M, Lowe AA, Pae EK, et al. Obstructive sleep apnea subtypes by cluster analysis. *Am J Orthod Dentofac Orthop* 1992; 101:533-542.
4. Pépin JL, Lévy P, Veale D, et al. Evaluation of the upper airway in sleep apnea syndrome. *Sleep* 1992; 15:S50-S55.
5. Frohberg U, Naples RJ, Jones DL. Cephalometric comparison of characteristics in chronically snoring patients with and without sleep apnea syndrome. *Oral Surg Oral Pathol Oral Radiol Endod* 1995; 80:28-33.
6. Lowe AA, Özbek M, Miyamoto K, et al. Cephalometric and demographic characteristics of obstructive sleep apnea: An evaluation with partial least squares analysis. *Angle Orthod* 1997; 67:143-154.
7. Nelson S, Hans M. Contribution of craniofacial risk factors in increasing apneic activity among obese and nonobese habitual snorers. *Chest* 1997; 111:154-62.
8. Pae E-K, Lowe A, Fleetham JA. A role of pharyngeal length in obstructive sleep apnea patients. *Am J Orthod Dentofac Orthop* 1997; 111:12-17.
9. Pillar G, Peled R, Lavie P. Recurrence of sleep apnea without concomitant weight increase 7.5 years after weight reduction surgery. *Chest* 1994; 106:1702-1704.
10. Suratt P, McTier RF, Findly LJ, et al. Changes in breathing and the pharynx after weight loss in obstructive sleep apnea. *Chest* 1987; 92:631-637.
11. Pae E-K, Lowe A, Sasaki K, et al. A cephalometric and electromyographic study of upper airway structures in the upright and supine positions. *Am J Orthod Dentofac Orthop* 1994; 106:52-59.
12. Lowe AA. Correlation between orofacial muscle activity and craniofacial morphology in a sample of control and anterior openbite subjects. *Am J Orthod* 1980; 80:89-98.
13. White DP. Pathophysiology of obstructive sleep apnea. *Thorax* 1995; 50:797-804.
14. Shelton KE, Gay SB, Hollowell DE, et al. Mandible enclosure of upper airway and weight in obstructive sleep apnea. *Am Rev Respir Dis* 1993; 148:195-200.
15. Flemons WW, Whitelaw WA, Brant R, et al. Likelihood ratios for a sleep apnea clinical prediction rule. *Am J Respir Crit Care Med* 1994; 150:1279-1285.