Comparison of Cephalometric Variables in Non-obese and Obese Patients with Obstructive Sleep Apnea

Önder Öztürk[1], Süleyman Hakan Tuna[2], Hüseyin Alkış[3], Mehmet Has[1], Hüseyin Avni Balcıoğlu[4], Hakan Türkkahraman[3], Ahmet Akkaya[1]

[1]Department of Chest Diseases, Sleep Disorders Center, Faculty of Medicine, Süleyman Demirel University, Isparta, Turkey
[2]Department of Prosthodontics, Faculty of Dentistry, Süleyman Demirel University, Isparta, Turkey
[3]Department of Orthodontics, Faculty of Dentistry, Süleyman Demirel University, Isparta, Isparta, Turkey
[4]Department of Anatomy, Faculty of Dentistry, İstanbul University, İstanbul, Turkey

ABSTRACT

Objective: To compare the cephalometric variables of obese (body mass index (BMI) ≥30) and non-obese (BMI<30) Turkish male patients with obstructive sleep apnea syndrome (OSAS).

Materials and Methods: OSAS diagnosed 85 patients who were obese [n=37; mean age (±SE), 49.41±1.54 year] and non-obese [n=48; mean age (±SE) 46.92±1.39 year] were included in the study. The cephalometric measurements and polysomnographic data of the patients were compared and a discriminatory analysis was performed.

Results: The apnea-hypopnea index (AHI) was significantly higher in obese patients (p<0.01). Bimaxillary protrusion was found in obese patients (p<0.05). The non-obese patients with AHI ≥ 30 had an increased mandibular plane angle In the stepwise discriminant analysis done separately in obese and non-obese patients according to AHI; only the hyoid bone position was included in the model in obese patients and the estimated success of discrimination of AHI's level (<30 and ≥30) was 70.3%. Age, anterior face and posterior face height were included to the model in non-obese patients and the estimated success of discrimination was found as 79.2%.

Conclusion: Craniofacial morphology has an effect on the severity of OSAS. If the craniofacial morphology tends toward a worsening of OSAS with obesity, the severity of the OSAS increases.

Key Words: Cephalometry, obesity, obstructive sleep apnea syndrome, polysomnography

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Introduction

Obstructive sleep apnea syndrome (OSAS) is characterized by loud snoring and periodic breathing with repetitive apneas, hypopneas, arousals leading to fragmented sleep, and excessive daytime sleepiness. It is a common condition, affecting at least 2% of adult females and 4% of adult males (1). Several causes for OSAS have been suggested. It appears to result from a variable combination of anatomical and pathophysiological factors, some of which may be due to genetics (2, 3).

Although there is no consistent evidence that the differences in the upper airway anatomy have a substantial impact on the occurence of OSAS, (4) the etiology of OSAS includes abnormalities in both the pathophysiology and anatomy of the airway and associated facial structures (5). The factors that cause obstruction of the upper airway include anatomical and functional factors, and anatomical factors such as obesity and craniofacial deformity are thought to play a major role (6, 7). However, the determinants of upper airway collapse are multiple, airway anatomy being only one such determinant. It has

been suggested that bony craniofacial abnormality is more important as a risk factor in non-obese than in obese OSAS patients (8, 9). This may be the reason why some OSAS subjects are of normal weight and physique. Furthermore, the etiology of OSAS in obese patients may be different from that in non-obese patients (9, 10).

Obesity, being a risk factor for OSAS, may affect upper airway morphology in the absence of bony craniofacial abnormality. In obese patients who have a distribution of body fat mainly over the upper part of their body, the resistance of the upper airway during sleep tends to be very high (11, 12). It was demonstrated that the closing pressures of the passive pharynx were distinctively higher in apneic patients than in normal subjects matched for age and body mass index (BMI) (13). However, the possible influence of age and BMI on cephalometric measurements has frequently been neglected (10). It has also been suggested that the discrepancy in the cephalometric measurements may depend on race (14). A previous study suggested that there may be differences between Asians and Caucasians in the degree to which obesity and craniofacial anatomy serve as risk factors, and that the etiology of OSAS in

Address for Correspondence: Dr. Önder Öztürk, Department of Chest Diseases, Sleep Disorders Center, Faculty of Medicine, Süleyman Demirel University, Isparta, Turkey Phone: +90 246 211 93 32 E-mail: dronderozturk@gmail.com

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obese patients may differ from that in non-obese patients (15). It is also thought that Turkish OSAS patients may present some different cephalometric features from those of other Caucasian patients due to racial physical differences.

The objectives of the present study were to compare the cephalometric variables of obese and non-obese Turkish male patients with OSAS. Another objective was to clarify the relationship between cephalometric variables and obesity to predict the severity of AHI.

Materials and Methods

Study population

Eighty-five Turkish adult male patients with OSAS were recruited from patients who complained of habitual snoring, apneas, and daytime sleepiness, and were referred to the Sleep Disorders Center at Suleyman Demirel University. All patients were examined by one of two pulmonologists experienced in sleep medicine, and they underwent an overnight polysomnography. Cephalometry was performed on all patients the morning after the polysomnography. The patients were recruited on the basis of the following inclusion criteria: dentate in both jaws, apnea-hypopnea index (AHI) ≥5 events per hour (events.h^{-1}) during an overnight polysomnographic study, and no apparent craniofacial deformities. The patients were excluded if there was any previous history of pharyngeal surgery, orthognathic surgery, or poor general dental health. This study was approved by the local research ethics committee, and informed consent was obtained from all patients before the polysomnographic study.

The OSA population was further classed into non-obese and obese OSA groups by BMI, using the cut-off point of <30 and ≥30 kg.m^{-2} (16). Each group was also classified into two subgroups according to the AHI, using the cut-off point of <30 and ≥30 events.h^{-1} (17). The characteristics and polysomnographic data for the patients are presented in Table 1.

Polysomnography

On the evening of the sleep study, a comprehensive physical examination which included anthropometric measurements-such as height and weight - for the calculation of body mass index (BMI) (18), neck circumference (at the level of the cricothyroid membrane), and waist circumference (at the level of the umbilicus)-were performed for all patients. The patients filled out a self-administered questionnaire that included questions about the symptoms normally associated with OSAS and also filled out the Epworth sleepiness scale (ESS), previously translated into Turkish and validated (19). All patients underwent a sleep study consisting of an overnight polysomnographic examination (Compumedics, 44 channels E series, Australia), which included an EEG (C3-A2, C4-A1, O1-A2, and F4-A1), bilateral electrooculogram, chin and lower leg electromyogram, oronasal flow as measured by a nasal cannula, thoracic and abdominal respiratory movements as recorded by piezoelectric belts, ECG, oxygen saturation as measured by finger oximetry, and body position. A desaturation event (dip) was considered when oxygen saturation (SaO$_2$) decreased by 3% or more in relation to the baseline. The sleep studies were staged according to the Rechtschaffen and Kales (20) criteria by two trained sleep clinicians who were blind to the clinical characteristics of each patient.

Apnea was defined as the cessation of airflow for 10 sec or longer. Hypopnea was defined as an airflow reduction of at least 50% over preceding epochs or an amplitude reduction of at least 50% of the thoracoabdominal belts (also compared to prior epochs), together with the presence of either arterial oxygen desaturation equal to or greater than 3%, or an arousal. AHI was determined by the frequency of these events per hour during sleep time based on the results of the overnight polysomnography. The patients with an AHI of 5 or higher were considered to have OSAS (17).

Cephalometric analysis

A standard lateral cephalogram was obtained during the end-expiration phase for each subject with the teeth in centric occlusion and without swallowing. A cephalostat was used to keep the subject's head in such a position that the Frankfort horizontal plane was parallel to the ground during exposure. Each radiograph was scanned into digital format at 300 dpi resolution, stored in TIFF format, and transferred to Dolphin imaging software (Dolphin, Chatsworth, California, USA) for digitization of landmarks. Each image was digitally traced twice by the same investigator (H.A.), 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. In each lateral cephalogram, 14 landmark points were identified (Figure 1). A total of 17 variables related to both craniofacial skeletal and soft tissue morphology were measured as angular (degrees) or linear (millimeters). The angles and linear measurements and their abbreviations are given in Figure 2.
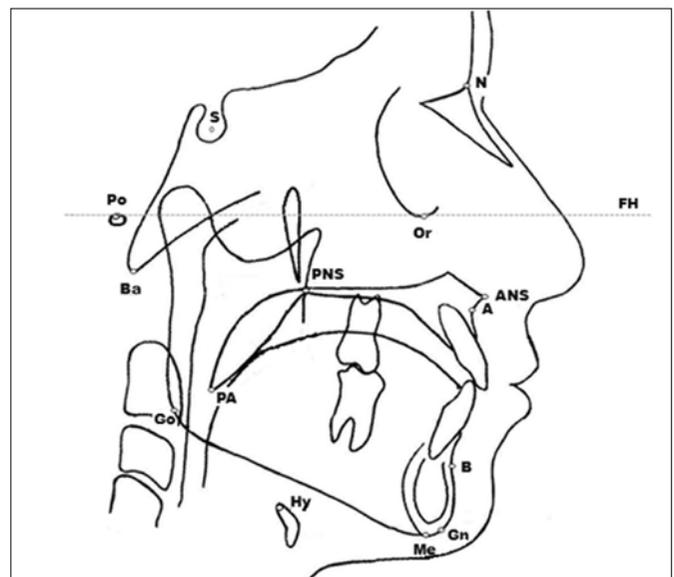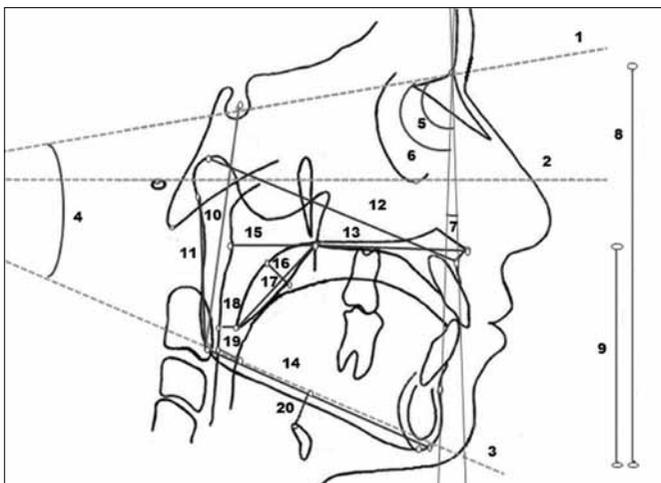

Figure 1. Landmarks on lateral cephalograms: S: sella; N: nasion; Or: orbitale; ANS: anterior nasal spine; PNS: posterior nasal spine; A: point A; B: point B; G:. gnathion; Me: menton; Go: gonion; Hy: hyoid bone; PA: tip of uvula; Po: porion; Ba: basion

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Figure 2. Linear and angular measurements on lateral cephalograms 1: S-N plane; 2: Frankfurt horizontal plane; 3: Mandibular plane; 4: Mandibular plane angle (SNGoGn°); 5: SNA°; 6: SNB°; 7: ANB°; 8: Anterior face height (Na-Me); 9: Lower face height (ANS-Me); 10: Posterior face height (S-Go); 11: Ramus height (Ar-Go); 12: Middle face length (Co-A); 13: Maxillar length (ANS-PNS); 14: Mandibular body length (Go-Gn); 15: Superior airway space; 16: Maximum soft palate thickness (Max. spt); 17: Uvula length (PNS-PA); 18: Middle airway space; 19: Inferior airway space; 20: Distance of hyoid bone to mandibular plane (Hy-MPPerp)

Statistical analysis

In this study, two factors were evaluated: one was the AHI factor, which had two levels (<30 events.h^{-1}and ≥30 events.h^{-1}), and the other one was the BMI factor, which had two levels (<30 kg.m^{-2} and ≥30 kg.m^{-2}). The Mann-Whitney U test was used for the parameters (mean SaO$_2$%, weight, neck circumferences, superior airway space, posterior face height), which did not provide a precondition of parametric tests to assess the differences between the levels of the AHI and BMI factors. The factorial analysis of variance (ANOVA) was applied for the parameters that provided the preconditions. Bivariate correlation analysis using Pearson's correlation coefficient was used to examine the positive relation between the cephalometric measurements and BMI with AHI. The stepwise discriminant analysis was performed using AHI as a dependent variable in obese and non-obese patients respectively. The results are expressed as mean±SE in this study. All of the statistical analyses were performed using the SPSS statistical software package (version 16.0, SPSS, Chicago, Ill.), with the level of significance set at $p<0.05$ and $p<0.01$.

Results

The patients had a mean age of 48.0±1.04, with a mean BMI of 30.53±0.61 kg.m^{-2}, had a mean AHI of 33.82±2.92 events.h^{-1} (n=85) ranging from 5 to 109.3, which was significantly higher in obese patients (Mann Whitney U test, $p<0.01$). Thirty-three of the 85 patients had an AHI≥30 events.h^{-1}. Overall, 48 of the 85 patients were non-obese and 37 were obese. The mean neck circumference was 40.47±0.30 cm. We found that the largest neck circumferences were more prevalent in the obese patients. The anthropometric, cephalometric and polysomnographic variables of the study group are shown in Table 1.

The ANOVA results of the demographic and polysomnographic parameters and cephalometric measurements: The AHI-BMI interaction and the differences between BMI factor means and AHI factor means were not statistically significant for age, inferior airway space, middle airway space, maximum soft palate thickness (Max.spt), mandibular plane angle (SNGoGn°), SNA°, SNB°, ANB°, anterior face height, ramus height, uvula length and lower face height. Although the differences between the mean waist circumference ($p<0.01$), height ($p<0.05$), maxillary length ($p<0.01$), mandibular body length ($p<0.05$), posterior face height ($p<0.01$), and middle face length ($p<0.05$) of the BMI factor were statistically significant, the AHI-BMI interaction and the differences between the mean levels of the AHI factor were not statistically significant. There were statistically significant differences between the mean levels of the AHI factor for the arousal index ($p<0.01$), awake mean SaO$_2$% ($p<0.05$), lowest SaO$_2$% ($p<0.01$), and Hy-MPPerp ($p<0.05$). However, the AHI-BMI interaction and the differences between the mean levels of the BMI factor were not statistically significant (Table 2).

In non-parametric analysis: the mean SaO$_2$ Des. (%) was significantly different between the mean ranks of the AHI<30 events.h^{-1} and AHI≥30 events.h^{-1} in obese and non-obese patients (Mann-Whitney U test, $p<0.01$); but weight, neck circumference, ESS, superior airway space, and posterior face height parameters were not statistically significant.

Relationship between AHI, BMI, neck and waist circuferences: AHI was found to be significantly correlated with BMI ($r=0.49$, $p<0.01$), neck circumferences ($r=0,39$, $p<0.01$) and waist circumferences ($r=0.40$, $p<0.01$) in the study. Also a positive correlation was found between BMI and neck circumferences ($r=0,62$, $p<0.01$) and waist circumferences ($r=0.76$, $p<0.01$) in the study.

Relationship between AHI and the cephalometric measurements: While there was a positive relationship between AHI and mandibular body length ($r=0.33$, $p<0.05$) in non-obese patients with AHI≥30 events.h^{-1}, AHI was correlated negatively with SPAS ($r=-0.34$, $p<0.05$) and posterior-anterior face height ($r=-0.73$, $p<0.05$) in non-obese patients with AHI<30 events.h^{-1}. AHI was significantly correlated with IAS ($r=0.78$, $p<0.01$) in obese patients with AHI<30 events.h^{-1}, HyMPPerp ($r=0.45$, $p<0.05$) and ANS-Me ($r=0.44$, $p<0.05$) in obese patients with AHI≥30 events.h^{-1}.

Relationship between BMI and the cephalometric measurements: BMI showed a positive significant correlation with IAS ($r=0.39$, $p<0.05$), SPAS ($r=0.36$, $p<0.05$) and MAS ($r=0.45$, $p<0.01$) in non-obese patients with AHI<30 events.h^{-1}. However, cephalometric measurements did not significantly correlate with BMI in the non-obese patients with AHI≥30 events.h^{-1}. In obese patients with AHI<30 events.h^{-1}, there was a positive correlation with maxillary length ($r=0.69$, $p<0.01$), posterior-anterior face height ($r=0.57$, $p<0.05$) and a negative correlation with SNGoGn ($r=-0.62$, $p<0.05$). A positive correlation was found between BMI and SNGoGn ($r=0.57$, $p<0.05$) and posterior-anterior face height ($r=0.47$, $p<0.05$) in obese patients with AHI≥30 events.h^{-1}.

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Table 1. Anthropometric, clinical, and cephalometric data of analyzed obese and non-obese patients

Demographic features	Whole group n=85	Non-obese (BMI<30 kg.m⁻²) n=48		Obese (BMI ≥30 kg.m⁻²) n=37	
		AHI<30 events.h⁻¹ n=38	AHI ≥30 events.h⁻¹ n=10	AHI<30 events.h⁻¹ n=14	AHI ≥30 events.h⁻¹ n=23
		Mean±SE			
Age (yr)	48.0±1.04	48.39±1.61	41.30±2.02	48.21±2.21	50.13±2.03
Height (cm)	170.16±0.70	171.05±0.83	173.30±1.43	170.21±1.25	167.30±1.87
Weight (cm)	88.18±1.67	78.55±1.25	83.80±2.44	96.29±3.00	101.04±3.75
BMI (kg.m⁻²)	30.53±0.61	26.85±0.38	27.87±0.49	33.14±0.69	36.19±1.38
Neck circumference (cm)	40.47±0.30	39.05±0.33	38.90±0.46	41.43±0.36	42.91±0.62
Waist Circumference (cm)	100.75±1.52	92.39±1.94	98.60±2.60	109.00±2.24	110.48±2.73
Polysomnographic data					
AHI (events.h⁻¹)	33.82±2.92	15.79±1.24	55.56±6.67	18.18±2.15	63.68±5.07
Arousal Index (arousals.h⁻¹)	23.73±1.71	19.88±2.08	36.90±3.28	13.35±2.51	30.67±3.94
Awake mean SaO_2%	93.69±0.19	94.05±0.29	93.90±0.41	94.29±0.41	92.65±0.36
Lowest SaO_2%	79.19±0.95	83.08±0.94	76.50±3.14	81.50±1.77	72.52±1.99
Mean SaO_2 Des.%	5.99±0.34	4.61±0.26	7.80±1.23	4.50±0.33	8.39±0.79
ESS	6.28±0.49	5.37±0.74	8.40±1.48	5.14±0.88	7.57±1.01
Cephalometric data					
Inferior airway space (mm)	12.59±.41	11.51±0.61	13.23±1.20	12.86±1.06	13.93±0.63
Superior airway space (mm)	19.53±0.42	20.25±0.53	19.79±1.15	20.40±0.66	17.68±1.08
Middle airway space (mm)	8.48±0.30	7.83±0.42	9.33±0.74	8.19±0.64	9.36±0.69
Maximum soft palate (mm)	12.87±0.24	12.47±0.29	12.81±0.81	12.60±0.65	13.74±0.55
SNGoGn°	31.55±0.69	31.82±1.05	32.01±2.95	29.77±1.69	31.99±0.96
Maxillary length(mm)	56.55±0.43	55.22±0.62	53.87±0.54	58.32±1.04	58.84±0.73
Mandibular body length(mm)	90.89±0.63	90.40±0.99	87.82±1.52	93.80±1.72	91.30±0.98
SNA°	82.12±0.48	82.80±0.72	81.57±1.92	82.71±1.18	80.87±0.72
SNB°	78.67±0.43	78.77±0.71	78.07±1.75	80.06±0.85	77.91±0.60
ANB°	3.46±0.27	4.04±0.36	3.62±0.81	2.82±0.94	2.83±0.46
Anterior face height (mm)	139.58±0.83	137.39±1.31	141.50±2.16	140.27±1.67	141.92±1.59
Posterior face height (mm)	92.26±0.77	90.52±1.04	89.62±3.33	94.90±1.84	94.67±1.14
Ramus height (mm)	52.66±0.62	51.15±0.82	52.12±2.49	54.01±1.23	54.58±1.21
Middle face height (mm)	96.96±0.51	96.83±0.85	94.07±1.55	98.06±1.14	97.77±0.74
Hy-MPPerp (mm)	22.38±0.68	22.19±0.91	23.09±2.06	19.10±1.71	24.39±1.37
Uvula length (mm)	43.77±0.58	43.73±0.84	43.10±1.66	44.32±1.39	43.80±1.26
Lower face height (mm)	78.23±0.72	76.88±1.15	78.10±1.98	80.39±1.40	79.20±1.27

AHI: apnea-hyponea index. BMI: body mass index. Sa: saturation. ESS: Epworth Sleepiness Scale. Hy-MPPerp: distance of hyoid bone to mandibular plane. SNGoGn0: mandibular plane angle. SNA°: angle between lines from S to N and from subspinale (A) to N; SNB°: angle between lines from S to N and from supramentale (B) to N; ANB°: angle between lines from A and B to N

In the stepwise discriminant analysis done separately in obese and non-obese patients according to the AHI: The age, anterior face height, and middle face length were included in Fisher's linear discriminant function in non-obese patients. The results obtained from the discriminant analysis are shown in Table 3a. The total result for this discrimination was 79.2%. If the diagnosis was made using parameters other than AHI, the estimated success of discrimination was 79.2%. The estimated successes were distributed as follows: AHI<30 events.h⁻¹ at 76.3%, AHI≥30 events.h⁻¹ at 90% (Table 3b).

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Table 2. AHI-BMI interaction table showing the statistically significant differences

Parameters	Diagnosis	BMI		Total
		BMI<30 kg.m^{-2}	BMI≥30 kg.m^{-2}	
Height (cm) (p<0.05)	AHI<30 events.h^{-1}	171.05±5.09	170.21±4.66	170.64±0.98
	AHI≥30 events.h^{-1}	173.30±4.52	167.30±8.96	170.30±1.19
	Total	172.18 A ±1.12	168.76 B±1.06	
Waist Circumference (cm) (p<0.01)	AHI<30 events.h^{-1}	92.39±11.97	109.0±8.37	100.69±1.79
	AHI≥30 events.h^{-1}	98.60±8.22	110.48±13.08	104.54±2.16
	Total	95.49 A±2.03	109.74 B±1.94	
Arousal Index (arousals.h^{-1}) (p<0.01)	AHI<30 events.h^{-1}	19.89±12.84	13.35±9.89	18.12 a ±12.27
	AHI≥30 events.h^{-1}	36.90±10.38	30.67±18.87	32.56 b ±16.84
	Total	23.42±14.11	24.12±17.95	
Awake mean SaO$_2$% (p<0.05)	AHI<30 events.h^{-1}	94.05±1.83	94.29±1.54	94.17 a ±0.27
	AHI≥30 events.h^{-1}	93.90±1.29	92.65±1.72	93.28 b ±0.32
	Total	93.98±0.30	93.47±0.29	
Lowest SaO2% (p<0.01)	AHI<30 events.h^{-1}	83.08±5.77	81.50±6.61	82.29 a ±1.19
	AHI≥30 events.h^{-1}	76.50±9.94	72.52±9.53	74.51 b ±1.44
	Total	79.79±1.35	77.01±1.29	
Maxillary length (mm) (p<0.01)	AHI<30 events.h^{-1}	55.22±3.83	58.32±3.88	56.77±0.56
	AHI≥30 events.h^{-1}	53.87±1.72	58.84±3.52	56.36±0.68
	Total	54.54 A±0.64	58.58 B±0.61	
Mandibular body length (mm) (p<0.05)	AHI<30 events.h^{-1}	90.40±092	93.80±1.51	92.10±0.89
	AHI≥30 events.h^{-1}	87.82±1.79	91.29±1.18	89.56±1.07
	Total	89.11 A±1.01	92.55 B±0.96	
Hy-MPPerp (mm) (p<0.05)	AHI<30 events.h^{-1}	22.19±0.99	19.10±1.64	20.65 a ±0.96
	AHI≥30 events.h^{-1}	23.09±1.94	24.39±1.28	23.74 b ±1.16
	Total	22.64±1.09	21.75±1.04	
Posterior face height (mm) (p<0.01)	AHI<30 events.h^{-1}	90.52±1.11	94.90±1.84	92.71±1.07
	AHI≥30 events.h^{-1}	64.18±1.50	67.84±1.27	92.15±1.30
	Total	90.07 A±1.22	94.79 B±1.16	
Middle face height (mm) (p<0.05)	AHI<30 events.h^{-1}	96.83±0.75	98.06±1.24	
	AHI≥30 events.h^{-1}	94.07±1.47	97.74±0.97	97.44±0.73
	Total	95.45 A±0.83	97.92 B±0.79	95.92±0.88

*a: the differences between diagnosis. A: the differences between BMI. Hy-MPPerp: distance of hyoid bone to mandibular plane

Hy-MPPerp was included in Fisher's linear discriminant function in obese patients. The results obtained from the discriminant analysis are shown in Table 4a. The total result for this discrimination was 70.3%. If the diagnosis was made using parameters other than AHI, the estimated success of discrimination was 70.3%. The estimated successes were distributed as follows: AHI<30 events.h^{-1} at 71.4%, the AHI≥30 events.h^{-1} at 69.6% (Table 4b).

Discussion

The main finding of this study is that the craniofacial morphology and BMI have an effect on the severity of OSAS. There are obvious sex differences in the craniofacial skeletal characteristics that contribute to OSAS severity and, to evaluate OSAS severity, different anthropometric and cephalometric measurements should be used for men and women (12). Furthermore, it has been reported that there are racial differences between the prevalence and characteristics of OSAS (21). Thus, the male patients were included in the study and the cephalometric features of Turkish male patients with OSAS were evaluated.

Although OSAS, being a progressive disease, increases with age (22) the possible influence of age on cephalometric measurements has been frequently neglected (10). However, Sakakibara et al. (10) could not determine any correlation be-

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Table 3a. The result of discriminant analysis of non-obese patients

	OSAS severity	
	AHI<30 events hour^{-1}	AHI≥30 events hour^{-1}
Age	-0.39	-0.57
Anterior face height	2.18	2.67
Middle face height	2.44	2.59
(Constant)	-167.29	-184.64
Fisher's linear discriminant functions		

Table 3b. The distribution of estimated success according to the severity of OSAS

Diagnosis		Predicted Group Membership		Total
		AHI<30 events. hour^{-1}	AHI≥30 events. hour^{-1}	
n	AHI≥30 events.hour^{1}	29	9	38
	AHI≥30 events.hour^{1}	1	9	10
%	AHI≥30 events.hour^{1}	76.3	23.7	100
	AHI≥30 events.hour^{1}	10	90	100
a. 79.2% of original grouped cases correctly classified				

Table 4a. The result of discriminant analysis of obese patients

	OSAS severity	
	AHI<30 events hour^{-1}	AHI≥30 events hour^{-1}
Hy-MPPerp	0.45	0.58
(Constant)	-4.99	-7.72
Fisher's linear discriminant functions		
Hy-MPPerp: Distance of hyoid bone to mandibular plane		

Table 4b. The distribution of estimated success according to the severity of OSAS

Diagnosis		Predicted Group Membership		Total
		AHI<30 events. hour^{-1}	AHI≥30 events. hour^{-1}	
n	AHI≥30 events.hour^{-1}	10	4	38
	AHI≥30 events.hour^{-1}	7	16	10
%	AHI≥30 events.hour^{-1}	71.4	28.6	100
	AHI≥30 events.hour^{-1}	30.4	69.6	100
a. 79.2% of original grouped cases correctly classified				

tween age and cephalometric variables. In the present study, the means of age were not statistically different in the AHI-BMI interaction and between the mean levels of the AHI factor and the BMI factor, so a possible relationship between cephalometric measurements and age could not be found in our study.

Obesity, which tends toward excessive soft tissue in the upper airway, might be related to OSAS. It has been demonstrated that BMI and neck size statistically significantly increased with the severity of OSAS (12) which also seems to be in agreement with our data (p<0.01). Although the previous studies showed a correlation between the neck circumference and OSAS (23, 24). Dahlqvist et al. (25) reported that the neck circumference was not independently related to the AHI. In the present study, the neck circumference and also waist circumference were correlated with the AHI and increased with the BMI.

Obesity, because of neck and soft tissue fat deposition and increased pressure on the neck of submental adipose tissue, may predispose to upper airway obstruction (8). Watanabe et al. (26) also showed that patients with closure of the velopharynx-only type were more obese and had less severe craniofacial abnormalities. In contrast, patients with closure of the combined velopharynx and oropharynx type were less obese and had more severe craniofacial abnormalities. It has been reported that obesity and craniofacial abnormalities contribute synergistically to an increase in the collapsibility of the passive pharyngeal airway (26). However, the upper airway shows anatomic differences during awake and sleep periods, and an occlusion occurs at different levels along the upper airway for patients with OSA. The obstruction begins with fat deposits on the lateral pharyngeal wall, where it cannot be seen in cephalometric graphs (23). The upper airway and soft tissue measurements did not show any statistically significant differences between the mean levels of the BMI and the AHI in the present study.

Studies on sleep-disordered breathing in Europe and America have shown relationships with obesity and craniofacial morphology (26, 27). However, the data in the literature are inconsistent, such that studies evaluating cephalometric anomalies in patients with OSAS have found no clear-cut morphological characteristics (28). Moreover, one study showed that gender and racial variations are present in cephalometric parameters (29). Furthermore, Ishiguro et al. (30) reported that skeletal conditions were probably the factors that contributed most to a further increase in the severity of Japanese male OSAS.

Sadeghianrizi et al. (31) showed that craniofacial morphology differs between obese and normal adolescents. In general, obesity was associated with bimaxillary prognathism and relatively greater facial measurements. The measurements of maxillary length, mandibular body length, posterior face height, middle face length, and lower face height were found increased in obese patients over non-obese patients. The findings in adult men were in accordance with the study of Sadeghianrizi et al. (31). The craniofacial bones are structured during adolescence and do not change in adults (32). It is considered that these measurements cannot be increased by gaining weight in adulthood. Therefore, obese adults might be obese during adolescence and might have a genetic tendency. However, no significant relationship between these measurements and the AHI was found in this study.

Although obesity is a factor very much related to the presence of OSAS, (22) some non-obese patients have severe OSAS (5). The reason for this might be explained by

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the comparison of obese patients with AHI<30 events.h^{-1} and non-obese patients with AHI≥30 events.h^{-1}, as shown in Table 1. Non-obese patients with severe OSAS were characterized by a greater tongue thickness, an inferiorly positioned hyoid bone, a greater mandibular body length, and a longer soft palate (2). In the present study, although the hyoid bone position was not statistically associated with the BMI, there was a strong association with the AHI. As the position of the hyoid bone becomes more inferior, the condition becomes more severe, as mentioned in the literature (12). In the stepwise discriminant analysis, hyoid bone position was included and the estimated success of discrimination of AHI's level was 70.3% in the obese patients. The mandibular body length, maxillary length, and middle face length (sagittal measurements) in non-obese patients with AHI≥30 events.h^{-1} were found to be less than the ones measured in obese patients with AHI<30 events.h^{-1}. These patients had a divergent face type with increased SNGoGn°. The reason the AHI was less than 30 depended on obesity with normal craniofacial morphology in obese patients with AHI<30 events.h^{-1}. The craniofacial morphology affected the severity of OSAS with non-obese patients, and if the craniofacial morphology changed with a tendency toward OSAS with obesity, the severity of OSAS increased more, as shown in Table 1.

One of the major limitations of this study was the lack of cephalograms taken in the supine and sleep positions. The upper airway obstruction usually occurs during sleep, so cephalograms taken in the supine position have the advantage of closely resembling those of the sleep position (33). However, the reproducibility of the head position in the supine position is difficult. In a study that compared supine and upright cephalograms, no significant difference in the skeletal pattern was found (33, 34). The clinical meaningfulness of group comparisons of cephalometric measurements may be questionable, because the traditionally used lateral cephalograms do not provide three-dimensional information (13). However, it has been reported by several investigators that cephalometric measurements had clinical significance. Therefore they can be used for the evaluation of the pathogenesis and pathophysiology of OSAS in a patient, and for the selection of the appropriate treatment modality (34).

Conclusion

The craniofacial morphology and BMI have an influence on AHI. The craniofacial morphology is effective in the severity of OSAS in non-obese patients. If the craniofacial morphology changes favored OSAS in obese patients, the severity of the OSAS increased more.

Conflict of Interest
No conflict of interest was declared by the authors.

References

1. Young T, Palta M, Dempsey J, Skatrud J, Weber S, Badr S. The occurrence of sleep-disordered breathing among middle-aged adults. N Engl J Med 1993;328:1230-5. **[CrossRef]**
2. Tsai HH, Ho CY, Lee PL, Tan CT. Cephalometric analysis of non-obese snorers either with or without obstructive sleep apnea syndrome. Angle Orthodon 2007;6:1054-61. **[CrossRef]**
3. Redline S, Tishler PV, Tosteson TD, Williamson J, Kump K, Browner I, et al. The familial aggregation of obstructive sleep apnea. Am J Respir Crit Care Med 1995;151:682-7.
4. Sforza E, Bacon W, Weiss T, Thibault A, Petiau C, Krieger J. Upper airway collapsibility and cephalometric variables in patients with obstructive sleep apnea. Am J Respir Crit Care Med 2000;161:347-52.
5. Battagel JM, Johal A. A cephalometric comparison of normal weight and obese subjects with obstructive sleep apnoea. Radiography 2000;6:283-92. **[CrossRef]**
6. Pracharktam N, Nelson S, Hans MG, Broadbent BH, Redline S, Rosenberg C, et al. Cephalometric assessment in obstructive sleep apnea. Am J Orthod Dentofacial Orthop 1996;109:410-9. **[CrossRef]**
7. Baik UB, Suzuki M, Ikeda K, Sugawara J, Mitani H. Relationship between cephalometric characteristics and obstructive sites in obstructive sleep apnea syndrome. Angle Orthod 2002;72:124-34.
8. Ferguson KA, Ono T, Lowe AA, Ryan CF, Fleetham JA. The relationship between obesity and craniofacial structure in obstructive sleep apnea. Chest 1995;108:375-81. **[CrossRef]**
9. Nelson S, Hans M. Contribution of craniofacial risk factors in increasing apneic activity among obese and nonobese habitual snorers. Chest 1997;111:154-62. **[CrossRef]**
10. Sakakibara H, Tong M, Matsushita K, Hirata M, Konishi Y, Suetsugu S. Cephalometric abnormalities in non-obese and obese patients with obstructive sleep apnoea. Eur Respir J 1999;13:403-10. **[CrossRef]**
11. Partinen M, Guilleminault C, Quera-Salva MA, Jamieson A. Obstructive sleep apnea and cephalometric roentgenograms. The role of anatomic upper airway abnormalities in the definition of abnormal breathing during sleep. Chest 1988;93:1199-205. **[CrossRef]**
12. Tsai HH, Ho CY, Lee PL, Tan CT. Sex differences in anthropometric and cephalometric characteristics in the severity of obstructive sleep apnea syndrome. Am J Orthod Dentofacial Orthop 2009;135:155-64. **[CrossRef]**
13. Isono S, Remmers JE, Tanaka A, Sho Y, Sato J, Nishino T. Anatomy of pharynx in patients with obstructive sleep apnea and in normal subjects. J Appl Physiol 1997;82:1319-26.
14. Yu X, Fujimoto K, Urushibata K, Matsuzawa Y, Kubo K. Cephalometric analysis in obese and nonobese patients with obstructive sleep apnea syndrome. Chest 2003;124:212-8. **[CrossRef]**
15. Li KK, Powell NB, Kushida C, Riley RW, Adornato B, Guilleminault C. A comparison of Asian and white patients with obstructive sleep apnea syndrome. Laryngoscope 1999;109:1937-40. **[CrossRef]**
16. World Health Organization. Obesity: preventing and managing the global epidemic. World Health Organization, Geneva. 1997.
17. Sleep-related breathing disorders in adults: recommendations forsyndrome definition and measurement techniques in clinical-research. The Report of an American Academy of Sleep Medicine Task Force. Sleep 1999;22:667-89.
18. Revicki DA, Israel RG. Relationship between body mass indices and measures of body adiposity. Am J Public Health 1986;76:992-4. **[CrossRef]**
19. Ağargün MY, Çilli AS, Kara H, Bilici M, Telcioğlu M, Semiz ÜB ve ark. Epwort uykululuk ölçeği 'nin geçerliği ve güvenirliği. Türk Psikiyatri Dergisi 1999;10:261-7.
20. Rechtschaffen A, Kales A. A manual of standardized terminology, techniques and scoring system for sleep stages of human

Balkan Med J
2011; 28: 244-51

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Cephalometric Variables, Obesity and OSAS **251**

subjects. In: Rechtschaffen A, Kales A, eds. National Institute of Health Publication, Washington DC: US Government Printing Office;1968.p.1-12.

21. Redline S, Tishler PV, Hans MG, Tosteson TD, Strohl KP, Spry K. Racial differences in sleep-disordered breathing in African Americans and Caucasians. Am J Respir Crit Care Med 1997;155:186-92.

22. Berger G, Berger R, Oksenberg A. Progression of snoring and obstructive sleep apnoea: the role of increasing weight and time. Eur Respir J 2009;33:338-45. **[CrossRef]**

23. Yagi H, Nakata S, Tsuge H, Yasuma F, Noda A, Morinaga M, et al. Morphological examination of upper airway in obstructive sleep apnea. Auris Nasus Larynx 2009;36:444-9. **[CrossRef]**

24. Tsai WH, Remmers JE, Brant R, Flemons WW, Davies J, Macarthur C. A decision rule for diagnostic testing in obstructive sleep apnea. Am J Respir Crit Care Med 2003;167:1427-32. **[CrossRef]**

25. Dahlqvist J, Dahlqvist A, Marklund M, Berggren D, Stenlund H, Franklin KA. Physical findings in the upper airways related to obstructive sleep apnea in men and women. Acta Otolaryngol 2007;127:623-30. **[CrossRef]**

26. Watanabe T, Isono S, Tanaka A, Tanzawa H, Nishino T. Contribution of body habitus and craniofacial characteristics to segmental closing pressures of the passive pharynx in patients with sleep-disordered breathing. Am J Respir Crit Care Med 2002;165:260-5. **[CrossRef]**

27. Verin E, Tardif C, Buffet X, Marie JP, Lacoume Y, Andrieu-Guitrancourt J, et al. Comparison between anatomy and resistance of upper airway in normal subjects, snorers and OSAS patients. Respir Physiol 2002;129:335-43. **[CrossRef]**

28. Miles PG, Vig PS, Weyant RJ, Forrest TD, Rockette HE Jr. Craniofacial structure and obstructive sleep apnea syndrome-a qualitative analysis and meta-analysis of the literature. Am J Orthod Dentofacial Orthop 1996;109:163-72. **[CrossRef]**

29. Lee JJ, Ramirez SG, Will MJ. Gender and racial variations in cephalometric analysis. Otolaryngol Head Neck Surg 1997;117:326-9. **[CrossRef]**

30. Ishiguro K, Kobayashi T, Kitamura N, Saito C. Relationship between severity of sleep-disordered breathing and craniofacial morphology in Japanese male patients. Oral Surg Oral Med Oral Pathol Oral Radiol Endod 2009;107:343-9. **[CrossRef]**

31. Sadeghianrizi A, Forsberg CM, Marcus C, Dahllöf G. Craniofacial development in obese adolescents. Eur J Orthod 2005;27:550-5.

32. Thilander B, Persson M, Adolfsson U. Roentgen-cephalometric standards for a Swedish population. A longitudinal study between the ages of 5 and 31 years. Eur J Orthod 2005;27:370-89. **[CrossRef]**

33. Brander PE, Mortimore IL, Douglas NJ. Effect of obesity and erect/supine posture on lateral cephalometry: relationship to sleep-disordered breathing. Eur Respir J 1999;13:398-402. **[CrossRef]**

34. Cistulli PA. Craniofacial abnormalities in obstructive sleep apnoea: implications for treatment. Respirology 1996;1:167-74. **[CrossRef]**