Original Article

The role of the external nasal measures of patients with sleep disordered breathing in determining disease severity

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ABSTRACT

Objectives: In this study, we aimed to investigate the influence of nasal anthropometric measurements on severity of obstructive sleep apnea syndrome (OSAS) and to assess the correlation between nasal anthropometric measurements and severity of OSAS after excluding internal factors causing nasal obstruction.

Patients and Methods: A total of 241 consecutive patients (181 males, 60 females; mean age 50.3±11.3 years; range, 18 to 65 years) with daily sleepiness and/or snoring complaints between February 1st 2018 and December 15th 2018 were included in the study. All patients were divided into eight groups as obese and non-obese according to the disease severity and BMI values.

Results: According to the obese and non-obese groups, none of the anthropometric measurements in the obese group were correlated with OSAS, while only nasal width (r: 0.282, p=0.001), nasal tip height (r: 0.235, p=0.008), and alar-pronasal distance (r: 0.156, p=0.031) were found to be correlated in the non-obese patient groups. Linear regression analysis of variables which appeared to correlate with the OSAS severity revealed that no variables except for age and BMI significantly contributed to the OSAS severity in the obese group. For the non-obese group, in addition to age and BMI values, nasal width significantly contributed to the disease severity.

Conclusion: Although there are many factors in the etiology of OSAS, no external nasal anatomic measurement, except for the nasal width, seems to be correlated with the disease severity.

Keywords: External nasal anatomy, nasal anthropometry, obstructive sleep apnea.

First described by Christian Guilleminault in 1973, obstructive sleep apnea syndrome (OSAS) occurs as a varying spectrum clinically from primary snoring during sleep to severe obstructive apnea.[1] When the disease is investigated etiologically, it appears to be linked to full or partial obstruction of the airway due to a combination of a variety of anatomic and/or neuromuscular disorders.[2] The disease is associated with functional disorders such as excessive daytime sleepiness, daytime headaches, disrupted concentration, traffic and work accidents, in addition to somatic disorders including systemic and pulmonary hypertension, ischemic heart disease, and cerebrovascular disease.[3]

Many authors have proposed a correlation of the disease or disease severity with a variety

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of inflammatory mediators,[4-7] biochemical markers,[8-10] and hormonal parameters.[11-13] Some studies have emphasized a correlation of the disease with respiratory tract anatomy and loss of genioglossus and pharyngeal muscle function causing pharyngeal collapse in individuals, although it is not possible to explain the problem with a single mechanism.[3,14]

Previous studies investigating the correlation between ethnicity and OSAS have reported craniofacial anatomic features are associated with OSAS development as much as general body and soft tissue features; however, this correlation is clear in the Caucasian race, while there is less correlation among African-Americans.[15,16] Huang et al.[2] assessed 358 patients with computed tomography (CT) in large-scale studies and emphasized that OSAS severity might be associated with factors such as obesity, minimal cross-section of the nasopharynx, and upper airway length. Additionally, although limited in numbers, there are studies reporting a correlation between the degree of congestion and OSAS severity in individuals with nasal congestion.[17,18] However, after excluding internal nasal pathologies such as septal deviation, internal nasal valve, and turbinate hypertrophy, there is no study available assessing the effect of nasal anthropometric measurements on OSAS severity.

The primary aim of this study was to assess the correlation of nasal anthropometric measurements after excluding internal nasal factors causing nasal obstruction with severity of OSAS in patients classified according to body mass index (BMI) values. The secondary aim was to assess the effect of obesity on nasal proportions and to evaluate the correlation between total nasal flow values as evidenced by anterior rhinomanometric measurements in patients without nasal pathologies with OSAS severity and BMI values.

PATIENTS AND METHODS

A total of 241 consecutive patients (181 males, 60 females; mean age 50.3±11.3 years; range, 18 to 65 years) with daily sleepiness and/or snoring complaints between February 1st 2018 and December 15th 2018 were included in the study. *Exclusion criteria were as follows: (i)* other sleep disorders such as central sleep apnea syndrome or narcolepsy, upper airway resistance syndrome, or restless legs syndrome; *(ii)* medical treatment history due to hypertension, thyroid replacement treatment, diabetes mellitus, hyperlipidemia or any active infection or any inflammatory disease; *(iii)* continuous positive airway pressure administration or any previous surgical intervention due to OSAS; *(iv)* any hepatic, pulmonary, renal or cardiac failure; *(v)* any surgical upper respiratory tract pathology leading to sleep apnea syndrome; and *(vi)* patients who were unwilling to participate in the study. Additionally, as we evaluated correlation between BMI and anthropometric variables with the OSAS severity, smokers were excluded to prevent any bias for the study reliability. Anterior rhinoscopic examination was performed and patients with any internal nasal pathologies including severe septal deviation, internal nasal valve, and turbinate hypertrophy which may limit nasal respiration were also excluded from the study. A written informed consent was obtained from each patient. The study protocol was approved by the Ahi Evran University Training and Research Hospital, Ethics Committee (No. 2018-19/106). The study was conducted in accordance with the principles of the Declaration of Helsinki. Anthropometric and blood pressure measurements.

The BMI was calculated by dividing the body weight by the square of height (kg/m^2). Daytime systolic and diastolic blood pressures were measured for all patients with a mercury pressure device at 08.00 AM, while sitting after five-min rest. The mean of three measurements was recorded. A detailed physical examination was performed with anatomic variations assessed that may cause sleep apnea syndrome and no surgical upper respiratory pathology found that may cause sleep apnea syndrome. Mallampati scores were recorded and Müller maneuver was performed to assess upper respiratory tract collapse during sleep.

Nasal anthropometric measurements

Patients attending the ear, nose and throat clinic with snoring complaints were examined. During examination, the following points were defined: nasion point where the nasal bone joins

the forehead bone on the median sagittal line; subnasal point at the central division between the nostrils where they meet the upper jaw; alar point - most protruding section of the nose laterally and pronasal - most protruding point of the nose externally on the median sagittal line. The patients were requested to sit comfortably during conventional measurements using a standard protractor and distance measures (Dasco Pro Inc., Rockford, IL, USA) as described by Farkas et al.[19] Figure 1 shows the lateral appearance of the nasal region (A), the anteroposterior-frontal appearance (B) and the inferior-basal appearance (C) with reference points used during nasal anthropometric measurements marked.

Epworth Sleepiness Scale (ESS) and PSG monitoring

The Turkish version of the ESS was used with the aim of evaluating the patient's tendency to sleep. The ESS has total points varying from 0 to 24 obtained from eight questions (each scored from 0 to 3).

To objectively evaluate the night sleep status of each participant, a PSG (Philips Respironics, Murrysville, PA, USA) device was used in the laboratory. This device makes assessments according to the American Academy of Sleep Medicine (AASM) 2007 criteria. Briefly, the evaluation parameters are nasal and oral air flow (using both nasal oral thermocouple and nasal pressure cannula), snoring sounds, thoracic/abdominal movements, oxygen saturation, leg movements and body position. The apnea/hypopnea index (AHI) and oxygen desaturation index (ODI) were automatically scored with computer software and later manually checked by a technician.

Apnea was defined as the cessation of at least 90% of air flow for at least 10 sec; hypopnea was defined as ≥3% oxygen desaturation or ≥50% reduction in air flow lasting ≥10 sec related with a stimulus; and a stimulus was defined as sudden shifts in electroencephalographic frequency lasting at least three sec. The AHI was defined according to the number of apnea and hypopnea events during sleep and OSAS severity was assessed according to AHI. It was categorized as normal (<5), mild (5-14.9), moderate (15-29.9) or severe (≥30).

Anterior rhinomanometric investigation

The anesthesia mask linked to the device was closed on the skin to ensure no air leaks from the mouth and nose, with patients requested to tightly close their lips and inspire through their nose. The value equivalent to the point where the mobile markers in the device stopped with the effect of the negative pressure formed

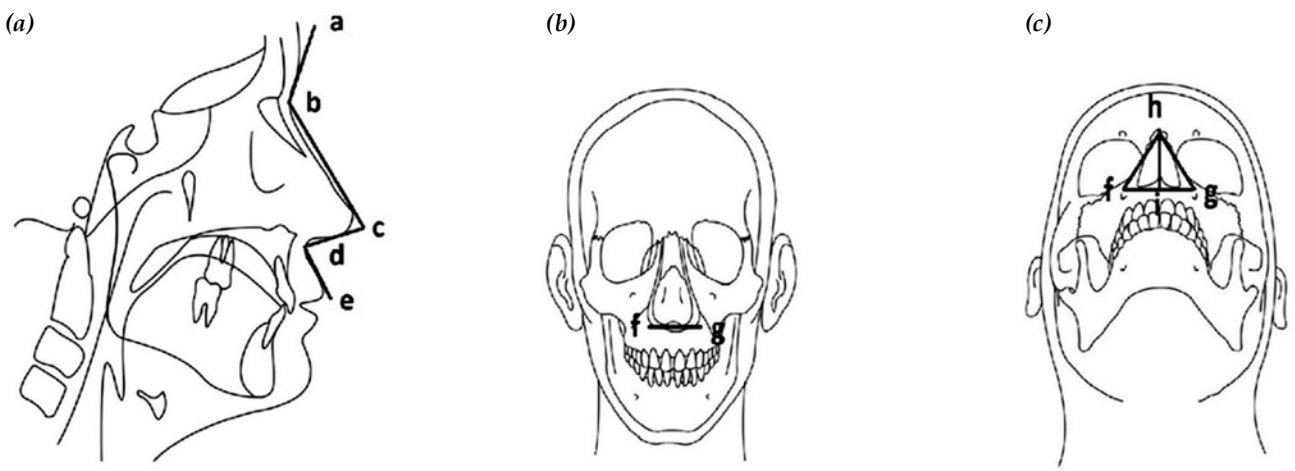

Figure 1. *Nasal region: lateral appearance* ***(a)****, anteroposterior-frontal appearance; distance from b.c.: nasal length (cm); distance from c.d.: nasal height (cm); angle abc: nasofrontal angle; angle cde: nasolabial angle.* ***(b)*** *Inferior-basal appearance; distance from f.g.: nasal width.* ***(c)*** *Nasal anthropometric measurements; distance from f.g: nasal width; distance from f.h.: alar-pronasal distance; distance from g.h.: alar-pronasal distance; distance from f.i.: alar-subnasal distance; distance from i.h.: alar-subnasal distance; distance from i.h.: nasal tip height.*

was recorded as nasal inspiratory peak flow (In-Check portable inspiratory flow meter, Part no: 1902164, Issue no:5, 09/2014 Clement Clarke International Limited, UK).

The patients were divided into a total of eight groups according to disease severity and BMI values as obese and non-obese patients. These were the non-obese control group [AHI <5, BMI ≤29.9 kg/m^2 (n=34)], obese control group [AHI <5, BMI >30 kg/m^2 (n=23)], non-obese mild OSAS group [AHI 5-15, BMI ≤29.9 kg/m^2 (n=36)], obese mild OSAS group [AHI 5-15, BMI >30 kg/m^2 (n=20)], non-obese moderate OSAS group [AHI 15-30, BMI ≤29.9 kg/m^2 (n=37)], obese moderate OSAS group [AHI 15-30, BMI >30 kg/m^2 (n=25)], non-obese severe OSAS group [AHI >30, BMI ≤29.9 kg/m^2 (n=42)] and obese severe OSAS group [AHI >30, BMI >30 kg/m^2 (n=24)].

Data including age, gender, BMI values, ESS, Müller, and Mallampati scores, nasal width (cm), nasal length (cm), nasal height (cm), alar-pronasal distance (cm), alar-subnasal distance (cm), nasolabial angle, nasofrontal angle, systolic blood pressure (mmHg), diastolic blood pressure (mmHg), mean O_2 saturation, minimal O_2 saturation and ODI values were recorded prospectively.

Statistical analysis

Statistical analysis was performed using the PASW for Windows version 17.0 software (SPSS Inc., Chicago, IL, USA). Data were expressed in mean ± standard deviation (SD), median (min-max), or number and frequency. For the comparison of multiple groups, analysis of variance (ANOVA) with the Tukey honestly significant difference test was used. Factors related to OSAS severity were assessed using the Pearson correlation analysis. Variables correlated with the OSAS severity were used to assess the contribution of multiple common variables to OSAS severity with the linear regression analysis. A p value of <0.05 was considered statistically significant.

RESULTS

Demographic and clinical features of the patients included in the study are summarized in Table 1. There was a statistically significant difference in the age of (p=0.040). The difference was observed to be due to patients included in Groups 2 and 6. In addition, there was a significant difference in the gender of the patients (p=0.027). The control group and mild OSAS group were not significantly different in terms of gender, while the male gender was dominant in the severe and moderate OSAS patient groups (Table 1).

In terms of the Müller and Mallampati scores, there was an increase in both parameters with the increase in OSAS severity, although the difference was not statistically significant (p=0.617 and p=0.654, respectively). On anterior rhinoscopy investigation, although there was no pathology increasing the internal nasal resistance, the total nasal flow level in the control group was significantly higher compared to the severe OSAS group based on the anterior rhinomanometric results. In terms of the nasal anthropometric values, the nasal length, nasal width, and nasal tip height values statistically significantly increased with the increase in OSAS severity, compared to the control group (p=0.001 for all) (Table 2).

For the factors associated with the OSAS severity, except for the nasolabial angle and nasofrontal angle values, anthropometric values were found to be correlated with the OSAS severity (Table 3). However, to assess whether these correlations were due to the effect of obesity, obese and non-obese patients were evaluated and none of the anthropometric measurements in the obese groups were found to be associated with OSAS. For non-obese patients, only nasal width (r: 0.282, p=0.001), nasal tip height (r: 0.235, p=0.008), and alar-pronasal distance (r: 0.156, p=0.031) were correlated, while there was no significant correlation for other nasal anthropometric measurements.

Variables which were apparently correlated with the OSAS severity were included in the linear regression analysis. As in the correlation analysis, when all groups were considered together, age, BMI, nasal width, nasal tip height, and alar-pronasal distance were significantly contributed to the OSAS severity. However, when the groups were separately evaluated as obese and non-obese patients and in the obese group, no variable except for age and BMI

Table 1. Demographic, hemodynamic, and polysomnographic data of the study groups

	Control (AHI <5)				Mild (AHI 5-15)				Moderate (AHI 15-30)				Severe (AHI >30)				
	Group 1 BMI ≤29.9 kg/m² (n=34)		Group 2 BMI >30 kg/m² (n=23)		Group 3 BMI ≤29.9 kg/m² (n=36)		Group 4 BMI >30 kg/m² (n=20)		Group 5 BMI ≤29.9 kg/m² (n=37)		Group 6 BMI >30 kg/m² (n=25)		Group 7 BMI ≤29.9 kg/m² (n=42)		Group 8 BMI >30 kg/m² (n=24)		
	n	Mean±SD	n	Mean±SD	n	Mean±SD	n	Mean±SD	n	Mean±SD	n	Mean±SD	n	Mean±SD	n	Mean±SD	*p*
Age (year)		49.6±15.4		47.8±8.3		43.6±10.6		49.9±14.2		49.9±10.0		56.8±5.6		53.8±10.0		53.1±9.9	0.040
Gender																	0.027
Female	10		12		10		9		11		5		10		3		
Male	24		11		26		11		26		20		32		21		
Epworth sleepiness scale		17.2±2.6		18.3±3.6		18.5±3.8		18.6±3.1		18.4±2.9		19.5±2.7		19.3±3.8		20.5±3.3	0.004
Mallampati score		2.4±1.2		2.4±0.8		2.4±1.2		2.6±1.0		2.6±1.2		2.8±0.5		2.7±1.2		2.8±0.7	0.617
Müller score		2.2±1.0		2.3±0.9		2.4±1.1		2.5±1.2		2.6±1.0		2.7±0.9		2.7±1.1		2.8±0.7	0.654
Hemodynamic parameters																	
Systolic BP (mmHg)		134.0±22.4		139.6±33.0		138.4±25.3		141.9±24.3		139.3±24.0		143.1±25.9		140.7±26.0		145.7±22.2	0.843
Diastolic BP (mmHg)		89.2±22.0		94.3±17.5		91.5±19.1		95.0±18.6		92.9±17.4		95.4±17.4		92.4±18.6		99.3±16.0	0.763
Heart rate (beat/min)		75.3±8.3		74.2±6.8		73.9±10.4		75.9±13.7		76.2±8.9		75.0±9.6		74.7±5.8		73.4±7.0	0.648
Polysomnographic study results																	
AHI		2.3±1.6		4.8±0.2		9.7±3.7		11.1±3.3		19.0±3.2		24.7±15.1		57.4±20.57		78.6±27.3	0.001
Mean SaO_2		95.1±5.6		96.4±6.6		93.2±5.1		93.3±5.6		91.8±5.9		91.7±6.0		90.9±5.8		88.7±5.3	0.001
Minimal SaO_2		87.7±7.3		85.9±7.3		80.0±7.2		81.7±7.8		77.5±8.0		75.0±8.2		69.3±8.2		68.6±6.7	0.001
ODI		2.7±1.5		9.9±4.9		10.7±6.5		15.8±9.2		27.0±20.8		24.8±10.0		63.1±28.5		89.0±38.6	0.001
Inspiratory nasal flow (L/dk)		133.3±15.6		128.2±18.2		126.7±13.7		120.5±22.4		100.4±23.9		93.9±20.5		102.1±18.4		85.9±12.8	0.001

AHI: Apnea-hypopnea index; BMI: Body mass index; SD: Standard deviation; BP: Blood pressure; SaO_2; Oxygen saturation; ODI: O_2 desaturation index; One-Way ANOVA (with Tukey HSD) was used.

Table 2. Anthropometric measurement results according to the study groups

	Control (AHI <5)		Mild (AHI 5-15)		Moderate (AHI 15-30)		Severe (AHI >30)		
	Group 1 BMI ≤29.9 kg/m² (n=34)	Group 2 BMI >30 kg/m² (n=23)	Group 3 BMI ≤29.9 kg/m² (n=36)	Group 4 BMI >30 kg/m² (n=20)	Group 5 BMI ≤29.9 kg/m² (n=37)	Group 6 BMI >30 kg/m² (n=25)	Group 7 BMI ≤29.9 kg/m² (n=42)	Group 8 BMI >30 kg/m² (n=24)	
	Mean±SD	Mean±SD	Mean±SD	Mean±SD	Mean±SD	Mean±SD	Mean±SD	Mean±SD	*p*
Nasal width (cm)	3.3±0.1	3.7±0.2	3.4±0.2	3.7±0.2	3.4±0.2	3.7±0.1	3.4±0.2	3.7±0.1	0.001
Nasal length (cm)	5.3±0.1	5.7±0.2	5.4±0.2	5.7±0.2	5.4±0.2	5.7±0.1	5.4±0.2	5.7±0.1	0.001
Nasal height (cm)	1.8±0.1	2.2±0.2	1.9±0.2	2.2±0.2	1.9±0.2	2.2±0.1	1.9±0.2	2.2±0.1	0.001
Alar pronasal distance (cm)	3.2±0.1	3.6±0.2	3.3±0.2	3.6±0.2	3.3±0.2	3.6±0.1	3.3±0.2	3.6±0.1	0.001
Alar subnasal distance (cm)	2.0±0.1	2.4±0.2	2.1±0.2	2.4±0.2	2.1±0.2	2.4±0.1	2.1±0.2	2.4±0.1	0.001
Nasolabial angle	100.7±1.8	97.2±5.1	97.0±5.7	96.2±6.2	97.9±5.9	97.7±6.3	96.5±4.7	94.5±7.4	0.011
Nasofrontal angle	127.5±7.4	133.7±1.8	130.0±5.7	129.2±6.2	130.9±5.9	130.7±6.7	129.5±4.7	130.2±5.1	0.011

AHI: Apnea-hypopnea index; BMI: Body mass index; SD: Standard deviation; One-Way ANOVA (with Tukey HSD) was used.

significantly contributed to the OSAS severity. In the non-obese group, in addition to age and BMI values, only nasal width significantly contributed to the OSAS severity (Table 4).

DISCUSSION

Obstructive sleep apnea syndrome is a very common disease affecting 2 to 17% of adult individuals and causing serious morbidity and mortality.[20] In addition to general health problems such as obesity and smoking in the etiology, although upper respiratory tract anatomic structure and pharyngeal soft tissue characteristics are blamed, the anatomic role of the nose which forms half of the upper respiratory system's resistance has not been fully clarified in the OSAS etiology.[18] In the literature, there is a limited number of studies investigating the correlation between nasal congestion and polysomnography,[2,3] and the current study is the first to evaluate the correlation of external nasal anatomy with severity of OSAS after excluding internal factors rhinoscopically.

In studies analyzing factors associated with OSAS severity in recent years, the common and strongest parameter obtained is obesity.[7-13] Tufik et al.[21] reported that obesity was one of the independent risk factors for patients with AHI ≥15, regardless of gender.[21] Based on these data, we grouped our patients as obese (BMI >30 kg/m²) and non-obese (BMI ≤29.9 kg/m²) patients. In this way, obesity was prevented from disrupting the homogeneity of results in the groups and from negatively affecting the results. Our aim was also to more clearly reveal the effect of obesity on nasal proportions and the correlation of obesity with OSAS. Taken together, the nasal measurements of obese individuals were observed to be significantly higher, compared to non-obese individuals. When nasal anthropometric measurements were assessed in all groups, they were found to be correlate with OSAS. However, with the aim of excluding the effect of the obesity factor, when obese and non-obese patients were assessed, only nasal width, nasal tip height, and alar-pronasal distance values were found to be correlated in the non-obese patient groups. According to linear regression analysis, only nasal width from among these factors significantly contributed

Table 3. Correlation of OSAS severity with nasal anthropometric measurement values

	All patients		Non-obese patients		Obese patients	
	r	*p*	r	*p*	r	*p*
Mallampati score	0.036	0.576	0.063	0.428	0.020	0.860
Müller score	0.044	0.542	0.055	0.487	0.032	0.842
Nasal width	0.244**	0.001	0.282**	0.001	0.046	0.754
Nasal length	0.223**	0.003	0.081	0.214	0.008	0.947
Nasal height	0.212**	0.006	0.095	0.157	0.014	0.934
Nasal tip height	0.248**	0.001	0.235**	0.008	0.044	0.761
Alar pronasal distance	0.239**	0.001	0.156*	0.031	0.008	0.947
Alar subnasal distance	0.217**	0.006	0.102	0.155	0.010	0.936
Nasolabial angle	0.030	0.654	0.117	0.137	0.204	0.069
Nasofrontal angle	0.036	0.675	0.094	0.166	0.186	0.078

OSAS: Obstructive sleep apnea syndrome; * Correlation is significant at the 0.05 level (2-tailed); ** Correlation is significant at the 0.01 level (2-tailed).

Table 4. Factors related to severity of OSAS based on logistic regression model

	All patients			Non-obese patients			Obese patients		
Independent variables	OR	*p*	95% CI	OR	*p*	95% CI	OR	*p*	95% CI
Age	0.189	0.006	0.07-0.310	0.173	0.022	0.005-0.186	0.244	0.029	0.006-0.103
Body mass index	0.249	0.001	0.024-0.780	0.126	0.042	0.005-0.186	0.152	0.031	0.033-0.248
Nasal tip height	-0.408	0.042	-3.696-0.070						
Nasal width	0.640	0.003	0.987-4.851	0.096	0.320	0.795-4.280			
Alar pronasal distance	0.166	0.017	0.018-0.992						

OSAS: Obstructive sleep apnea syndrome; OR: Odds ratio; CI: Confidence interval.

to the OSAS severity. The underlying reason for this is probably that, although we excluded internal factors causing nasal congestion, the etiology of OSAS is multifactorial and the fact is that many factors may cause it such as velopharyngeal soft tissue characteristics. Additionally, De Vito et al.[22] found 20 of 36 patients with OSAS assessed with rhinometric investigation had normal airway resistance, while only nine patients had increased nasal resistance in the supine position. Leitzen et al.[18] in their study analyzing the correlation between internal nasal anatomic features and OSAS severity found no correlation between the anatomic features and OSAS severity. Sakat et al.[23] in a study of 30 patients assessed the anatomic features of the nasopharyngeal region with multi-slice CT and reported that particularly inferior placement of the hyoid bone increased the severity of OSAS. Lyberg et al.[24] also showed that inferior placement of the hyoid bone increased OSAS severity and reported that this anatomic variation might cause less maxillomandibular development. However, consistent with previous studies, attempting to limit OSAS to only nasal anatomic features or nasal resistance and ignoring the multifactorial etiology underlying the disease would lead to misconceptions about treatment strategies and failure in fighting the disease.

Another parameter that our study examined was anterior rhinomanometric total nasal flow levels. In our study, total nasal flow levels in both severe OSAS patients and in the obese group were significantly lower, compared to

the relevant control groups. It has been well documented that OSAS is a multifactorial disease combining many variables. One of these variables is upper airway resistance which may be elevated due to reasons such as anatomic abnormalities including septal deviation and nasal turbinate hypertrophy, in addition to causes such as low upper respiratory tract muscle tone, increased fat content in parapharyngeal and upper airway tissues, and varied chest wall mechanics. However, previous studies evaluating the correlation between OSAS severity and increased nasal resistance have reported contradictory results to date. Tagaya et al.[25] found a positive correlation between the increased nasal resistance and reduced nasal flow in adult obese patients with the severity of OSAS. Rizzi et al.[26] obtained similar results to Tagaya et al.[25] in a study of pediatric age group patients (4-7 years). However, other studies fail to show any correlation between the increased severity of OSAS and increased nasal resistance and reduced nasal flow.[22,27] Our results are similar to the studies by Tagaya et al.[25] and Rizzi et al.[26] in terms of nasal flow values, and both increased OSAS severity and obesity separately reduced the nasal flow.

Despite applying very strict inclusion criteria for patient selection to assess nasal anatomic features, the main limitation of this study is that the velopharyngeal region was unable to be assessed. Another limitation is the lack of patients with different racial characteristics and that the correlation of nasal anthropometric measurements with OSAS severity was not evaluated according to race. However, Turkey generally contains the Caucasian phenotype and has a very homogeneous structure apart from the regional differences; therefore, our chance to include patients of different races is already very low. We believe that this topic should be studied in countries where individuals with different ethnic roots live together.

In conclusion, OSAS is a multifactorial disease with many factors playing roles in the etiology. Our study results show that, among nasal anthropometric values, external nasal anatomic measurements are not significantly correlated with the OSAS severity, except for the nasal width. However, further large-scale, prospective studies are needed to establish a definite conclusion.

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