*Original Article*

# Decreased maxillary sinus volume is a potential predictor of obstructive sleep apnea

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**ABSTRACT**

**Objectives:** To investigate the associations between nasal airway volume and the presence and severity of obstructive sleep apnea (OSA) in adults.

**Materials and Methods:** The medical records of adult patients who visited the sleep clinic at University Hospital between June 2013 and April 2017 and underwent overnight polysomnography for the diagnosis of obstructive sleep apnea were reviewed retrospectively. Using computed tomography, the volumes of the nasal airways and maxillary sinuses were measured, and associations with the presence and severity of OSA were analyzed while controlling for the effects of possible confounders such as lateral cephalometric variables, maxillary widths, tongue/hyoid position, and soft palate dimensions.

**Results:** Comparison between normal subjects and patients with OSA revealed that the latter had decreased ratios of maxillary sinus volume to whole nasal airway volume ($P = .029$) than normal subjects. OSA severity was greater in those with inferior positions of the hyoid ($P = .010$), in older patients ($P = .011$), and in those with high body mass index ($P = .001$). The volume of the total nasal airway or maxillary sinuses were not associated with OSA severity.

**Conclusions:** A decreased ratio of maxillary sinus volume to whole nasal airway volume is associated with adult OSA. However, OSA severity is not associated with either maxillary sinus volume or whole nasal airway volume. (*Angle Orthod.* 2020;90:556–563.)

**KEY WORDS:** Obstructive sleep apnea; Airway volume; Nasal cavity; Maxillary sinus; Apnea/hypopnea index; Adults

## INTRODUCTION

Obstructive sleep apnea (OSA) is the most common type of sleep apnea and is caused by partial or complete obstruction of the upper pharyngeal airway. The soft tissues of the pharyngeal wall collapse due to inactivity of the compensatory dilator muscles during sleep. The anatomic predispositions for OSA include jaw relations such as mandibular retrognathism,[1,2] obesity,[3] and nasal obstruction;[4,5] however, the condition is usually characterized by the presence of a narrow pharyngeal airway lumen as the site of the obstruction.[6,7] There are several controversies regarding the association of pharyngeal dimensions with the pathogenesis of OSA. Although some studies reported that patients with a narrow pharyngeal airway had an increased risk of OSA,[7–10] other studies concluded that there was no significant association between pharyngeal airway dimensions and the presence of OSA.[11,12]

A possible reason for this disagreement may be that the pharyngeal airway is nonrigid, and its dimensions continually change throughout respiration, which may lead to inaccuracies in the dimensional analysis of radiographs.[13,14] Additionally, because radiographs are taken when patients are in an alert state, they do not represent the state of the airway during sleep when the activity of the compensatory dilator muscles changes.[15] Other factors such as soft tissue tonicity, tongue

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muscle activation, and head posture are related to the patency of the upper airway during respiration.[16–19]

Unlike the pharyngeal airway, the nasal airway, which consists of the nasal cavity and paranasal sinuses, is dimensionally stable and not affected during sleep because it is surrounded by bony structures. Nasal obstruction has been designated as a risk factor for OSA,[1,5] and a computational fluid dynamics (CFD) study suggested that the turbulence of airflow created in the nasal cavity had a significant effect on the development of pharyngeal obstructions.[20] However, no clinical research has focused on the nasal airway including the paranasal sinuses and its association with the pathogenesis of OSA. Previous studies have assessed the whole upper pharyngeal airway, or the retropalatal and retroglossal airway regions alone.[9,10,12] The purpose of this study was to investigate the associations between nasal airway volume and the presence and severity of OSA in adults.

## MATERIALS AND METHODS

The study protocol was approved by the Institutional Review Board of Korea University Hospital (IRB AN17114-001), and informed consent from the patients was waived. The medical records of patients who visited the sleep clinic at Korea University Hospital between June 2013 and April 2017 with the chief complaint of snoring and sleep disturbance were reviewed retrospectively. The inclusion criteria were: adult patients aged 18 years and older who underwent overnight polysomnography (PSG) for the diagnosis of OSA and computed tomography (CT) of the ostiomeatal unit (OMU) to investigate anatomic deformities in the nasal region. The exclusion criteria were: (1) adenotonsillar hypertrophy, (2) chronic or acute rhinosinusitis and allergic rhinitis, (3) craniofacial abnormalities, (4) septal deviation angle (angle of the maximal septal deflection off the midsagittal plane) of $>10°$, (5) history of craniofacial surgery, and (6) morbid obesity (body mass index $>$ 40 kg/m$^2$). As a result, a total of 109 patients (89 males and 20 females, mean age: 42.2 $\pm$ 14.6 years) were included in the study.

PSG was performed at the sleep center of Korea University Hospital and recorded using the RemLogic recorder (Embla, Broomfield, CO, USA). PSG results were summarized, and the following variables were recorded to ascertain the severity of OSA: (1) apnea-hypopnea index (AHI): the number of apneas plus hypopneas per hour of sleep, (2) mean $O_2$ saturation, (3) lowest $O_2$ saturation, (4) respiratory disturbance index (RDI), and (5) oxygen desaturation index (ODI). Patients were divided into four groups according to the 2007 criteria of the American Academy of Sleep Medicine.[21] Group 0 consisted of normal subjects with an AHI $<$ 5 (n $=$ 18), Group 1: patients with an AHI $>$ 5 but $<$ 15 (mild OSA, n $=$ 28), Group 2: patients with an AHI $>$ 15 but $<$ 30 (moderate OSA, n $=$ 25), and Group 3: patients with an AHI $\geq$ 30 (severe OSA, n $=$ 38).

### CT Acquisition and Airway Volume/Maxillary Width Measurement

Patients underwent OMU CT either the same day or the day before PSG. CT images of all subjects were obtained using the Somatom Definition Flash (Siemens Medical Solutions, Erlangen, Germany) with the following parameters: 120 Kv, 280 mA, 512 $\times$ 512 pixels, and a 12.6 $\times$ 12.6-cm field of view. The OMU CT scans were taken with the patient in the supine position and had a slice thickness of 1 mm and an increment of 1 mm.

The CT images were imported to Mimics software (version 10.11, Materialise Medical Co, Leuven, Belgium) for segmentation of the nasal airway. For reorientation, a plane passing through nasion (N), anterior nasal spine (ANS), and the posterior nasal spine (PNS) was defined as the midsagittal reference plane (MSRP). Additionally, 3D segmentation of the nasopharyngeal airway was performed with a threshold setting of -1024 to -500 Hounsfield units to isolate the airway space according to the criteria outlined in previous studies.[22] The anterior border was defined as the external nares, while the superior border was the plane parallel to the palatal plane, a plane perpendicular to the MSRP and passing through ANS and PNS that contains N. Additionally, the posterior border was defined as the plane perpendicular to the MSRP and passing through PNS and basion (Figure 1); thus, the nasal cavity and paranasal sinuses were included. The segmented airway models were exported as stereolithography files for volumetric assessment. The right and left maxillary sinuses were also isolated from the whole nasal airway for a separate volumetric assessment. The volumes of the whole nasal airway and maxillary sinuses were measured in cubic millimeters, using Geomagic software (Geomagic Studio 13.0, Geomagic, Inc, Research Triangle Park, NC).

### Cephalometric Evaluation

Transverse maxillary dimensions were measured in the axial CT images using the Mimics software. The anterior and posterior maxillary widths were measured at the level of the root apices of the maxillary canines and mesiobuccal roots of the maxillary first molars, respectively (Figure 2).

Conventional cephalometric landmarks composed of the anteroposterior and vertical skeletal measurements, as well as the hyoid and tongue positions, and soft palate angulations were assessed using the

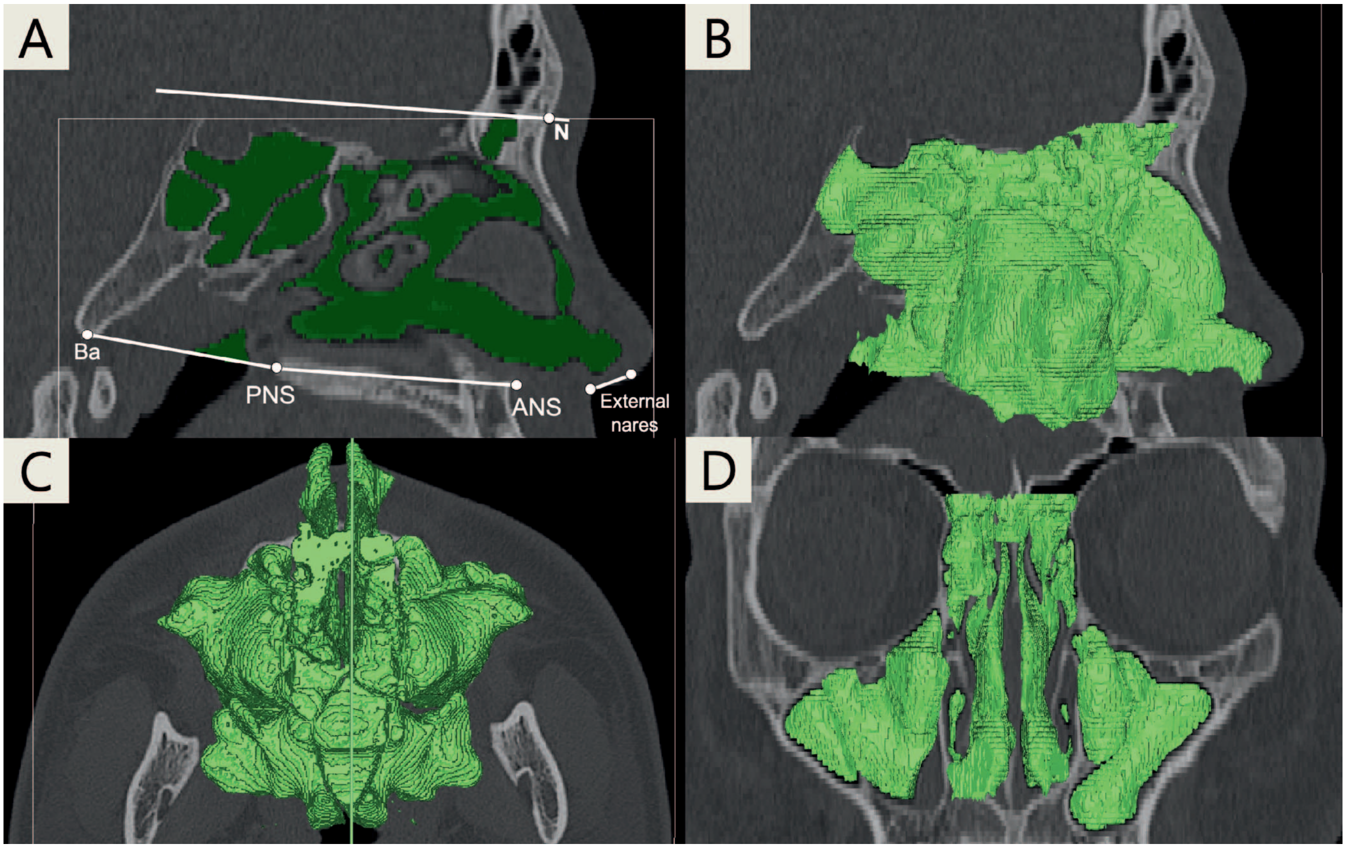


**Figure 1.** Segmentation of the nasal airway. The anterior border was defined as the external nares; the superior border was the plane parallel to the palatal plane, which was the plane that contained nasion (N) and connected the anterior nasal spine (ANS) and the posterior nasal spine (PNS). The posterior border was the plane that passed through PNS and basion (Ba) (A). Sagittal (B), axial (C), and frontal (D) views of the segmented airway models.

V-Ceph software (version 7.0, Osstem, Seoul, Korea; Figure 3). Positional changes in the angulation of the soft palate according to the position from the supine position to the upright position were measured by using two-dimensional lateral cephalometric radiographs taken in the upright position, and from lateral cephalometric images taken in the supine position, derived from OMU CT using the Invivo software (version 5.0, Anatomage, CA).

One researcher (HKS) performed all the measurements and was blinded to the individual PSG results. To examine intra-investigator reliability, the images of 10 randomly selected patients were remeasured by the same investigator 4 weeks after the first measurement. Intraclass correlation coefficients (ICCs) ranged from 0.97 to 0.99 for all measurements.

### Statistical Analysis

After performing descriptive statistics, the results were compared among the groups using the Kruskal-Wallis test. Multinomial logistic regression analysis was used to compare the nasal airway volumes for the four groups. Binary logistic regression analysis was used to investigate factors for normal subjects (AHI $< 5$) and OSA patients (AHI $\geq 5$). In the patients with OSA, linear regression analysis was performed to investigate the factors associated with OSA severity using AHI. All statistical analyses were performed using SPSS software for Windows (version 20.0, SPSS, Armonk, NY). $P$ values of $<.05$ were considered statistically significant.

## RESULTS

The descriptive statistics of the normal and OSA patient groups according to severity based on the AHI scores are displayed in Table 1. The mean AHI of groups 0, 1, 2, and 3 were 2.9 $\pm$ 0.9, 8.1 $\pm$ 3.0, 23.0 $\pm$ 3.8, and 54.4 $\pm$ 17.3, respectively. There was a significant difference in age, the BMI, and waist circumference (Table 1). There were no significant differences among the groups in the sagittal and vertical skeletal patterns represented by the ANB, SN-MP, and AB-MP measures (Table 1). However, there was a significant difference in the transverse dimension of the maxilla. The anterior and posterior maxillary widths significantly decreased from Group 0

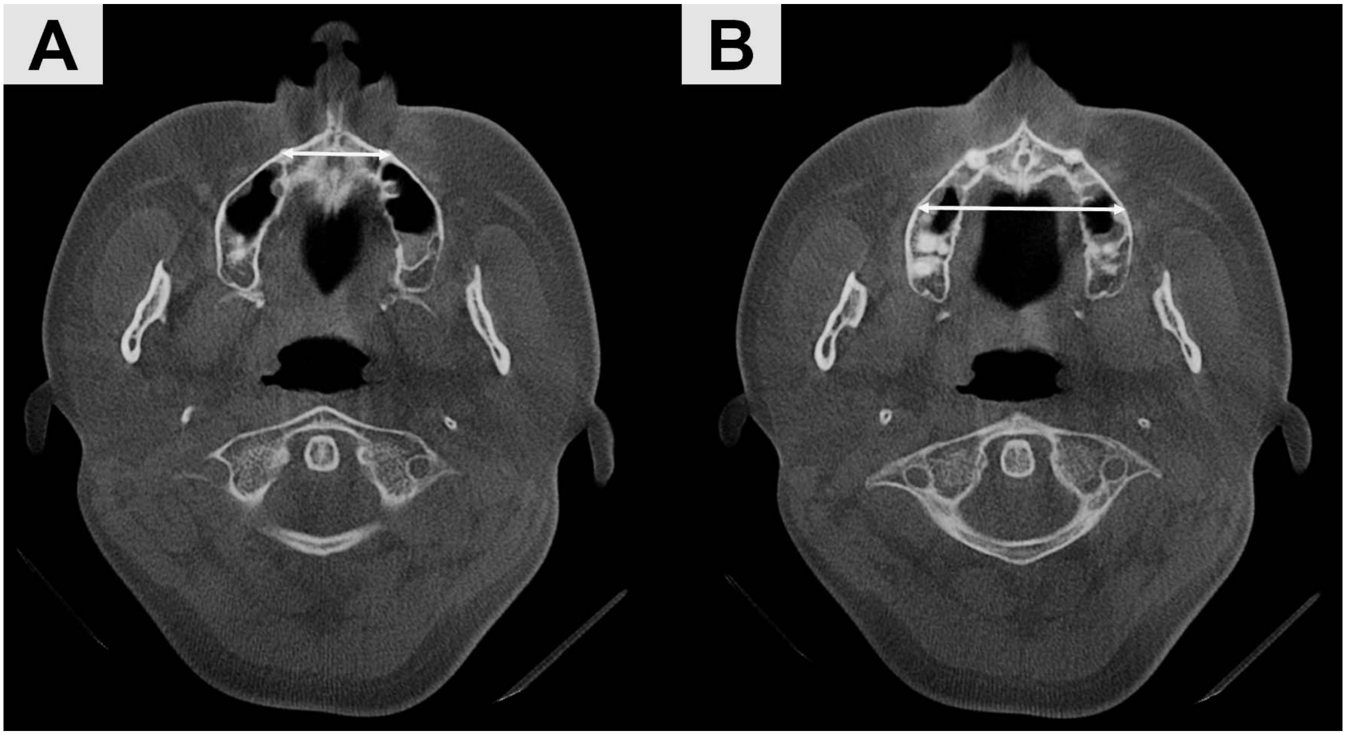


**Figure 2.** Linear measurement of maxillary widths. After reorientation, the anterior (A) and posterior (B) maxillary widths were measured at the level of the root apices of the maxillary canines and first molars, respectively.

to Group 3 (trend $P = .001$ and $P = .012$ for anterior and posterior maxillary widths, respectively, Table 1). Among the groups, the severity of OSA increased with more inferior positioning of the hyoid bone (hyoid-PP, trend $P < .001$, Table 1) and the anterior dorsum of the tongue (tongue ant, trend $P = .022$, Table 1). The total nasal airway volume showed no significant difference between the groups; the maxillary sinus volume (trend $P = .034$) and its ratio to the total nasal airway (trend $P = .006$) significantly decreased from group 0 to group 3 (Table 1).

Comparison between the normal subjects and patients with OSA revealed that the decrease in sinus/nasal airway ratio ($P = .029$) was associated with OSA after adjusting for age, sex, and SN-MP (Table 2). In patients with OSA, the severity indicated by AHI increased with greater age ($P = .011$), BMI ($P = .001$), and vertical position of the hyoid ($P = .010$, Table 3). The nasal airway volume and maxillary sinus volume were not associated with the severity of OSA and, therefore, they were excluded from the regression model.

## DISCUSSION

Decreased maxillary sinus volume was associated with the development of OSA in adults. Although maxillary sinus volume was significantly smaller in patients with OSA than normal subjects, it was not associated with the severity of OSA as indicated by the AHI. Therefore, it may be speculated that maxillary sinus volume might be a predisposing factor for the development of OSA but not an aggravating factor. Rodriguez et al. analyzed the volume of the nasal cavity alone and reported no association between nasal cavity volume and the severity of OSA.[23] According to a CFD study that investigated the influence of the nasal airway on the airflow patterns in the pharynx, both maxillary sinuses and other paranasal sinuses had negative effects on the airway flow into the pharynx.[20] That study used two anatomic models taken from two actual patients; the pharyngeal airflow was affected by the maxillary sinuses in one patient and by the paranasal sinuses excluding the maxillary sinuses in the other patient. However, the results of the present clinical study showed that only maxillary sinuses were associated with the presence of OSA.

Another factor that distinguished OSA patients from the normal subjects was the vertical angulation of the mandible, indicated by the SN-MP angle. Patients with downward and backward growth of the mandible had an increased risk of OSA. This type of vertical mandibular growth pattern was related to a lower position of the hyoid,[24] and hyoid position was associated with OSA severity. This result was in line with those of previous studies that reported possible

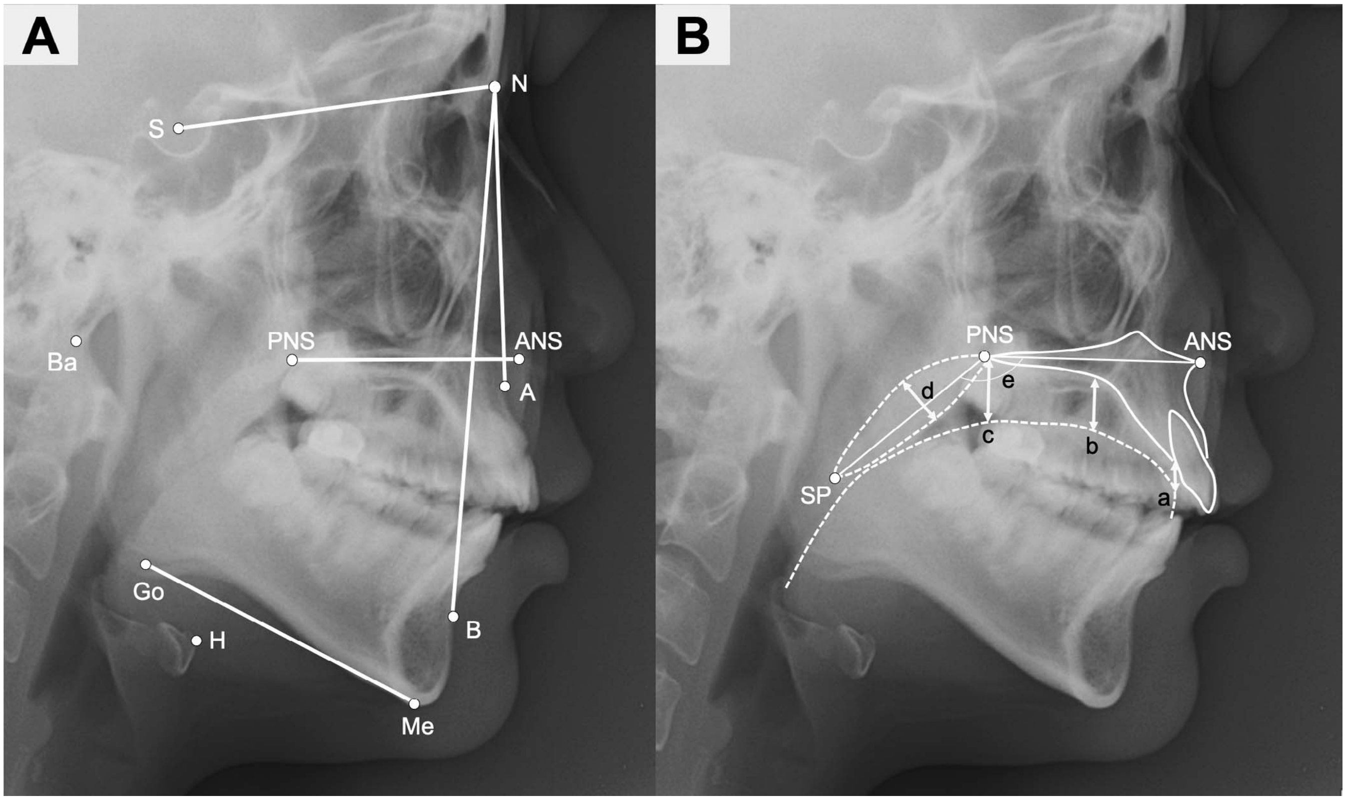


**Figure 3.** Lateral cephalometric analysis of the skeletal components and soft tissues such as tongue position and soft palate angulations. The following anatomic landmarks were identified for cephalometric analysis: S, sella; N, nasion; PNS, posterior nasal spine; ANS, anterior nasal spine; A, point A; B, point B; Me, menton; Go, gonion; H, hyoid; SP, soft palate point; a, anterior tongue position; b, middle tongue position; c, posterior tongue position; d, soft tissue thickness; e, soft palate angulation.

associations between a retruded mandible and the development of OSA.[2,25,26]

Having a small and narrow maxilla was also reported as being a predisposing factor for the development of OSA.[25,27–29] A similar relationship was observed in the OSA patient groups in the present study, in which patients with decreased anterior and posterior maxillary widths had higher AHI in the analysis of intergroup comparisons. The anterior maxillary width showed greater associations with OSA severity than did the posterior maxillary width. Additionally, a significant correlation was observed between the maxillary sinus volume and posterior maxillary width (correlation coefficient $= 0.394$, $P < .001$).

Since decreased maxillary sinus volume might be an etiologic factor for the development of OSA, clinicians should rule out the possible associations of a contracted maxillary sinus with breathing disturbances during sleep. In addition, possible causes of decreased sinus volume should be identified and treatment to enhance the patency of the nasal airway should be considered. Maxillary expansion may be performed for those who have maxillary transverse deficiencies. A CFD study of a patient who underwent miniscrew-assisted rapid palatal expansion showed a significant increase in the cross-sectional area of the nasal airway and an improvement in pharyngeal airflow.[30] In addition, a systematic review and meta-analysis of maxillary expansion in adult OSA confirmed that maxillary expansion improved the AHI and lowest oxygen saturation.[31]

The ideal way to measure nasal airflow is still controversial, but volumetric analysis of nasal airway based on segmentation of CT images may have limitations in evaluating nasal airway function. As the nasal airway consists of dynamic structures such as the turbinates and cartilaginous structures, alar cartilages and lateral cartilages, functional tests such as nasal resistance would provide additional information on nasal function, warranting further prospective study.

Another limitation of this study was that, due to the retrospective nature of this study, there were more patients with severe OSA than normal subjects and patients with mild to moderate OSA. Therefore, results of PSG, such as AHI, RDI, flow limitation index, and oxygen desaturation index did not exhibit a normal distribution, and logarithmic transformation was done for linear regression analysis.

**Table 1.** Descriptive Statistics of the Normal and OSA Patient Groups According to Severity by the Apnea-Hypopnea Index (AHI)[a]

| | Group 0 (n = 18) AHI < 5 | Group 1 (n = 28) 5 ≤ AHI < 15 | Group 2 (n = 25) 15 ≤ AHI < 30 | Group 3 (n = 38) 30 ≥ AHI | Total (n = 109) | *P** | Trend *P*** |
|---|---|---|---|---|---|---|---|
| Demographics | | | | | | | |
| Age (n) | 32.4 ± 13.6 | 40.4 ± 14.3 | 46.0 ± 14.2 | 45.5 ± 13.7 | 42.1 ± 14.6 | .005 | .001 |
| BMI (n) | 25.3 ± 2.9 | 25.8 ± 3.4 | 26.8 ± 3.8 | 27.6 ± 3.2 | 26.6 ± 3.4 | .047 | .005 |
| Waist circumference (cm) | 88.6 ± 9.8 | 90.7 ± 8.3 | 93.8 ± 8.1 | 95.6 ± 9.0 | 92.8 ± 9.0 | .023 | .002 |
| Neck circumference supine (cm) | 37.9 ± 3.3 | 38.9 ± 3.7 | 39.1 ± 3.1 | 40.2 ± 3.2 | 39.2 ± 3.4 | .092 | .014 |
| Polysomnography results | | | | | | | |
| AHI (n) | 2.5 ± 1.4 | 8.6 ± 3.0 | 23.0 ± 3.8 | 54.0 ± 16.8 | 26.7 ± 23.5 | <.001 | <.001 |
| RDI (n) | 5.1 ± 2.0 | 11.4 ± 3.5 | 25.1 ± 4.2 | 55.6 ± 16.0 | 28.9 ± 22.9 | <.001 | <.001 |
| Flow limitation index (total, %) | 37.4 ± 13.9 | 37.4 ± 17.0 | 35.0 ± 14.7 | 24.6 ± 13.3 | 32.4 ± 15.7 | .001 | <.001 |
| Sleep efficiency (n) | 93.1 ± 5.0 | 87.6 ± 9.6 | 87.2 ± 10.8 | 86.4 ± 8.4 | 88.0 ± 9.1 | .064 | .024 |
| Spontaneous arousal events (n) | 60.7 ± 29.9 | 65.6 ± 53.9 | 41.8 ± 27.0 | 24.5 ± 21.9 | 45.0 ± 38.7 | <.001 | <.001 |
| Lowest oxygen saturation (%) | 92.0 ± 2.5 | 87.6 ± 3.8 | 82.5 ± 6.8 | 76.1 ± 14.6 | 83.1 ± 11.1 | <.001 | <.001 |
| Cephalometric analysis | | | | | | | |
| ANB (°) | 2.4 ± 3.3 | 1.9 ± 2.7 | 1.9 ± 1.5 | 2.8 ± 2.1 | 2.3 ± 2.4 | .352 | .339 |
| SN-MP (°) | 34.1 ± 6.2 | 33.9 ± 7.1 | 35.4 ± 7.1 | 35.9 ± 5.5 | 35.0 ± 6.4 | .577 | .197 |
| AB-MP (°) | 70.2 ± 4.5 | 70.7 ± 6.5 | 68.4 ± 3.7 | 68.8 ± 5.0 | 69.4 ± 5.1 | .291 | .133 |
| Ant mx width (mm) | 34.7 ± 2.9 | 34.5 ± 2.7 | 33.4 ± 3.0 | 31.8 ± 3.5 | 33.2 ± 3.3 | .019 | .001 |
| Post mx width (mm) | 62.8 ± 5.2 | 61.3 ± 5.3 | 60.4 ± 4.6 | 58.9 ± 5.0 | 60.4 ± 5.2 | .039 | .012 |
| Hyoid-PP (mm) | 73.4 ± 7.8 | 76.5 ± 11.4 | 78.7 ± 7.2 | 82.2 ± 7.9 | 78.5 ± 9.2 | .004 | <.001 |
| Tongue_ant (mm) | 1.2 ± 1.6 | 0.4 ± 1.2 | 1.2 ± 1.9 | 2.0 ± 2.8 | 1.3 ± 2.1 | .027 | .022 |
| Tongue_mid (mm) | 3.7 ± 3.8 | 3.3 ± 4.1 | 2.2 ± 2.5 | 4.1 ± 5.8 | 3.4 ± 4.5 | .422 | .740 |
| Tongue_post (mm) | 7.8 ± 3.7 | 10.6 ± 5.2 | 7.9 ± 2.8 | 10.2 ± 5.3 | 9.4 ± 4.7 | .056 | .318 |
| Soft palate angulation change (°) | 0.7 ± 3.2 | 1.0 ± 2.3 | 1.2 ± 1.6 | 2.1 ± 2.5 | 1.4 ± 2.4 | .141 | .027 |
| Soft palate thickness (mm) | 11.2 ± 1.6 | 11.9 ± 2.3 | 11.2 ± 2.0 | 10.8 ± 1.5 | 11.2 ± 1.9 | .135 | .138 |
| Airway analysis | | | | | | | |
| Total nasal airway volume ($cm^3$) | 78.6 ± 27.3 | 72.9 ± 19.7 | 67.8 ± 16.6 | 72.1 ± 19.8 | 72.4 ± 20.5 | .414 | .295 |
| Maxillary sinus volume ($cm^3$) | 38.8 ± 18.6 | 31.8 ± 13.3 | 24.9 ± 12.4 | 29.8 ± 14.4 | 30.7 ± 15.0 | .022 | .034 |
| Sinus/nasal airway ratio (%) | 47.8 ± 8.0 | 42.2 ± 10.4 | 35.0 ± 13.1 | 39.7 ± 9.8 | 40.6 ± 11.2 | .002 | .006 |

[a] BMI indicates body mass index; OSA, obstructive sleep apnea; RDI, respiratory disturbance index.
* *P* = difference among the groups using Kruskal-Wallis test.
** *P* = trend from group 0 to group 3 using the trend test.

A decreased ratio of maxillary sinus volume to the whole nasal airway showed a significant association with the presence of OSA. Interventions that decrease the maxillary sinus volume, such as maxillary dental implants with sinus lift procedures and molar intrusion during orthodontic treatment, might contribute to the development of OSA; these possibilities warrant further investigation. In addition, nasal inflammatory conditions such as allergic rhinitis increase the risk of developing OSA.[32] Volumetric analysis of nasal airway in patients with nasal congestion or other pathologic conditions may provide additional information on the effect of nasal airway dimensions on the development of OSA.

**Table 2.** Factors Discriminating Normal Subjects (AHI < 5, n = 18) and OSA Patients (AHI ≥ 5, n = 91)[a]

| Independent Variable | B | SE | *P* |
|---|---|---|---|
| Sinus/nasal airway ratio[b] | −8.661 | 3.965 | .029 |
| Age[b] | 0.064 | 0.025 | .011 |
| Sex[c] | 2.479 | 0.859 | .004 |
| SN-MP[b] | 0.090 | 0.053 | .092 |

[a] AHI indicates Apnea-Hypopnea Index; B, regression coefficient; OSA, obstructive sleep apnea; SE, standard error; sinus/nasal airway ratio, the ratio of maxillary sinus volume to total nasal airway volume.
[b] Positive regression coefficient value indicates an increased chance of having OSA with one unit increase of the independent variable; whereas, a negative value indicates decreased chance of having OSA with one unit increase of the independent variable.
[c] Reference group is female, thus indicating that males have a greater chance of having OSA than females.

## CONCLUSIONS

- Adult patients with OSA had a decreased ratio of maxillary sinus volume to whole nasal airway volume

**Table 3.** Factors Associated With the Severity of OSA Indicated by AHI[a]

| Independent Variable | B | SE | *P* |
|---|---|---|---|
| Age | 0.015 | 0.006 | .011 |
| BMI | 0.075 | 0.023 | .001 |
| Hyoid-PP | 0.023 | 0.009 | .010 |

[a] AHI indicates Apnea-Hypopnea Index; B, regression coefficients; BMI, body mass index; Hyoid-PP, vertical position of the hyoid measured from the palatal plane; OSA, obstructive sleep apnea; SE, standard error.

compared to normal subjects. However, OSA severity was not associated with either maxillary sinus volume or whole nasal airway volume.

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