# Assessment of cephalometric parameters and correlation with the severity of the obstructive sleep apnea syndrome 

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#### Abstract

Background In individuals diagnosed with obstructive sleep apnea syndrome (OSAS), variations in craniofacial structure have been inconsistently documented, showing differing degrees of alteration between obese and nonobese patients. In addition, sleep disturbance has also been shown to induce disequilibrium in this population of patients. This pilot observational study aimed to assess craniofacial values in obese and nonobese subpopulations of patients with OSAS and their correlation and association with the severity of OSAS. We also assessed whether OSAS patients are characterized by an impaired equilibrium in relation to and associated with the severity of OSAS. Methods We included all consecutive adult patients with OSAS. Through cephalometry, we assessed the upper (UPaUPp) and lower (LPa-LPp) pharynx diameters, superior anterior facial height (Sor-ANS), anterior facial height (ANS-Me), anterior vertical dimension (Sor-Me), posterior facial height (S-Go) and craniovertebral angle (CVA). Furthermore, we analyzed postural equilibrium through a stabilometric examination. Results Forty consecutive OSAS patients ( $45 \%$ female with a mean age of $56 \pm 8.2$ years) were included. The subgroup of nonobese patients had a reduced UPa-UPp ( $p=0.02$ ). Cephalometric measurements were correlated with the severity of OSAS in nonobese patients, whereas only Sor-ANS was correlated with the severity of OSAS in the obese subpopulation. In the overall population, altered craniofacial values are associated with severe OSAS. Although there are differences in equilibrium between obese and nonobese OSAS patients, the stabilometric measurements were not correlated or associated with OSAS severity. Conclusion Altered craniofacial values and compromised equilibrium in OSAS patients are linked to OSAS severity. Therefore, the management of OSAS should be tailored not only to weight management but also to craniofacial and postural rehabilitation to enhance patient outcomes.


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## Introduction

Obstructive Sleep Apnea Syndrome (OSAS) is a sleep disorder characterized by recurrent episodes of partial or complete obstruction of the upper airway during sleep, leading to disrupted breathing patterns, known as apnea or hypopnea, resulting in oxygen desaturations [1]. Various pathophysiological mechanisms contribute to OSAS, including impaired muscle responsiveness, the inability of the upper airway to dilate or stiffen in response to an increase in ventilatory drive, and notably, anatomical narrowing of the upper airways [2, 3]. The diagnosis of OSAS is established through cardiorespiratory monitoring and polysomnography [4]. Although some concerns exist [5], OSAS is classified for severity on the apnea-hypopnea index (AHI) as mild (AHI: 5-15), moderate (AHI: 15-30), or severe ( $\mathrm{AHI}>30$ ) [6]. AHI is computed by summing the total number of apneas and hypopneas observed during sleep and dividing by the total sleep time in hours [6].
OSAS is associated with many diseases or disorders, such as cardiovascular diseases [7], metabolic disorders (e.g., diabetes) [8], gastrointestinal diseases (e.g., gastroesophageal reflux disease) [9], respiratory disorders (e.g., asthma) [10], emotional and psychological disorders [11] and increased mortality [12].
The gold standard for sleep apnea syndrome treatment is continuous positive airway pressure (CPAP), which consists of applying continuous pressure to the upper airways to keep them patent [13, 14]. In the case of moderate sleep apnea syndrome or failure of CPAP, a mandibular advancement device is a possible alternative therapy. It allows the jaw to advance, either fixed or progressive, increasing the diameter of the upper airways [15]. For obese patients, significant weight loss sometimes improves the symptoms associated with OSAS and reduces the correlated cardiovascular risk [16]. Maxillofacial surgery has proven to be quite effective [13]. In fact, OSAS patients are characterized by facial alterations that may benefit of surgical approaches, including improvements in associated symptoms like mandibular and cervical pain and bruxism $[17,18]$. Therefore, the treatment of OSAS requires a holistic and multidisciplinary approach.
The influence of craniofacial morphology on the pathogenesis of OSAS is controversial [19]. Cephalometric evaluation has been used to determine the typical facial shape of OSAS patients [20]. The cervical, hyoid, and mandibular positions may affect the severity of OSAS [17, 18, 21]. Little is known about the use of the stabilizing platform used in OSAS, and it has not been widely applied [22-24]. In particular, stabilometric analysis is a method to assess postural control and balance, through the assessment of the center of gravity and the patient's
path length, under various conditions (opened or closed eyes and mouth) [22-24].
Despite the current extensive investigation of correlation between craniofacial alterations and the severity of OSAS [25-29], we designed this pilot observational study to assess craniofacial values obtained through cephalometric analysis and their correlation and association with the severity of OSAS (as defined by an $\mathrm{AHI}>30$ ). We further conducted a stabilometric analysis to assess whether OSAS severity could compromise equilibrium.

## Methods

This pilot observational study was conducted at the University Hospital "R. Dulbecco" of Catanzaro (Italy) from January to June 2023, following the STrengthening the Reporting of OBservational studies in Epidemiology (STROBE) guidelines for observational studies [30] and adhering to the ethical principles outlined in the Declaration of Helsinki and its subsequent amendments. Approval for the study was granted by the local Ethics Committee "Comitato Etico Sezione Area CentroRegione Calabria" (Approval n. 372 on December 15th, 2022). Prior to participation, written informed consent was obtained from all enrolled patients. No modifications to the methods or study outcomes were introduced after the commencement of the study. Individual, deidentified datasets generated or analyzed during the study are available upon reasonable request from the corresponding author.

## Patients

We included all adult (i.e., $\geq 18$ years/old) outpatients who visited the pneumology clinic and received a diagnosis of OSAS, established through type II polysomnography, as defined by the American Academy of Sleep Medicine guidelines [4]. Patients with one or more of the following criteria were excluded: (1) body mass index (BMI) $>37 \mathrm{~kg} / \mathrm{m}^{2}$; (2) temporal-mandibular joint disorders [18], (3) fixed oral or mobile prosthesis; (4) previous radiant therapy in the head-district neck; (5) previous maxillofacial or upper airway surgery; (6) any smoking; (7) alcohol abuse; (8) use of sedative-hypnotic drugs (benzodiazepines, etc.), and/or comorbidities potentially affecting the equilibrium (for example vestibular disorders, cerebral stroke, neurodegenerative diseases, myopathies, arthritis, neuropathies). In addition, before study inclusion, all patients underwent dentistry and otorhinolaryngological examinations to exclude the presence of ankyloglossia and nasal obstruction and fibroscopy to assess the tonsillar and nasopharyngeal grading [31, 32].

The diagnosis of OSAS was obtained through cardiorespiratory monitoring and polysomnography (Somtè, Compumedics, Australia), as per guidelines [33]. The
equipment comprises five cables for recording the electrocardiogram and heart rate (HR) via two bipolar leads, a nasal cannula to detect the flow-meter trace, a microphone for snoring recording, two piezoelectric belts for measuring thoraco-abdominal movements, a digital pulse oximeter for peripheral arterial oxyhemoglobin saturation $\left(\mathrm{SpO}_{2}\right)$ measurement, and a gravity sensor for patient position tracking. The recordings were reviewed by the same operator and classified according to the American Academy of Sleep Medicine standards into obstructive, central, and/or mixed episodes, as well as apnoic and/or hypopnoic events [33]. Hypopnea was defined as oxygen desaturation by a $3 \%$ threshold. Sleep apnea was classified as central or obstructive based on the presence or absence of breathing effort. The oxygen desaturation index (ODI) assesses the frequency of desaturation episodes $>3 \%$ per hour of sleep, while time below $90 \%$ (TC90) calculates the fraction of saturation time $<90 \%$ [34].

## Data collection and analysis

For every patient anthropometric (BMI) and demographic (age and gender) and AHI data have been collected. Each patient underwent cephalometric analysis with OrisCeph software ${ }^{\circledR}$ (Henry Schein ONE Srl, Cernusco sul Naviglio, Italy) and a stabilometric assessment (Lizard ${ }^{\circledR}$, Lizardmed Srl, Monza, Italy).
Cephalometry data were obtained by lateral-lateral skull teleradiography and orthopantomography for the analysis of the upper airways, skeletal bases, and cervical spine (Fig. 1).
We assessed the following measurements, as depicted in Fig. 2:

- The antero-posterior diameter of the upper pharynx, measured between the posterior pharyngeal wall and the soft palatal tip, parallel to the palatal plane (UPaUPp) [35];
- The antero-posterior diameter of the lower pharynx, measured between the posterior and anterior pharyngeal wall (base of tongue), along the mandibular plane (LPa-LPp) [35];
- The superior anterior facial height (Sor-ANS) distance between the supraorbital point (Sor) and the anterior nasal spine (ANS);
- The linear distance between the ANS and the menton (Me) (ANS-Me), representing the anterior facial height [29];
- The distance between the Sor and Me (Sor-Me), representing the anterior vertical dimension;
- The total posterior facial height (S-Go) assessed through the distance between the Sella (S) and the Gonion (Go) [27];
- The craniovertebral angle (CVA) is delineated by the McGregor plane (which extends from the base of the occipital bone to the posterior nasal spine) and the odontoid process (which spans from the apex of the odontoid process to the most anterior and inferior points of the C 2 vertebral body) [36].

Stabilometric examination was performed using a static stabilometric platform (Lizard ${ }^{\circledR}$, Lizardmed Srl, Monza, Italy) under three conditions: (1) open eyes (OE); (2) closed eyes (CE); and (3) open eyes with the mouth opened (MO). In all three settings, we assessed the surface area (SA) the patient used to search for the center of gravity and the patient's path length (PL). Finally, we calculated, under all conditions, the variance in the

(a)

(b)

Fig. 1 X-ray in lateral position (a) and Orthopantomography (b)

a


C

b

| ANS | Anterior Nasal Spine |
| :---: | :---: |
| Go | Gonion |
| $\mathbf{M e}$ | Menton |
| $\mathbf{S}$ | Sella |
| $\mathbf{L P a}$ | Anterior lower pharyngeal wall |
| $\mathbf{L P p}$ | Posterior lower pharyngeal wall |
| $\mathbf{U P a}$ | Anterior upper pharyngeal wall |
| $\mathbf{U P p}$ | Posterior upper pharyngeal wall |
| Sor | Supraorbital Point |
| $\mathbf{C V A}$ | Craniovertebral angle |

Fig. 2 a measures of the distance between the differt facials points measured $\mathbf{b}$ diameter of upper and lower pharynx c.delimitation of craniovertebral angle
velocityas the ratio between the velocity of the center of gravity displacement and the number of measurements.

## Statistical analysis

Given the relatively small sample of patients, the data are expressed as the median (25th-75th interquartile range) and minimum to maximum (min-max) values. The data are presented for the overall population and for two subgroups of patients stratified according to BMI ( $<$ or $\geq 30 \mathrm{~kg} / \mathrm{m}^{2}$ ). Continuous data were compared with the Mann-Whitney $U$ test. The correlation between the severity of OSAS and the severity of OSAS according to the cephalometric and stabilometric analysis was determined by Spearman rank correlation ( $\rho$ ). Regression lines of rank-transformed data were fitted by BMI strata. Logistic regression analysis was performed to assess the association between cephalometric and stabilometric measurements and the presence of severe OSAS, as defined by an $\mathrm{AHI}>30$. According to the logistic regression model, the data are expressed as odds ratios (ORs) and $95 \%$ confidence intervals (CIs). P values $<0.05$ were
considered to indicate statistical significance in all tests. All analyses were performed using the $R$ package ( $R$ Development Core Team).

## Results

We included 40 consecutive patients ( $45 \%$ female and $55 \%$ male). The average (SD) age was $56 \pm 8.2$ years. The mean (SD) AHI was $31.44 \pm 13.8$, and the mean (standard deviation) ODI was $32.16 \pm 17.3$. Twenty-three patients had a $\mathrm{BMI}<30 \mathrm{~kg} / \mathrm{m}^{2}$, whereas 17 patients had a BMI $\geq 30 \mathrm{~kg} / \mathrm{m} 2$. None of the patients had ankyloglossia or nasal obstruction.

## Cephalometry

Cephalometric data from the overall population and from patients with a $\mathrm{BMI}<$ or $\geq 30 \mathrm{~kg} / \mathrm{m}^{2}$ are reported in Table 1.

All measurements were similar between the two subpopulations stratified according to BMI ( $\mathrm{p}>0.05$ ), except for the UPa-UPp $(p=0.02)$.

Table 1 Cephalometric measures in the overall population and stratified according to BMI

|  | Overall population $(n=40)$ | $\begin{aligned} & \mathrm{BMI}<30 \mathrm{~kg} / \mathrm{m}^{2} \\ & (\mathrm{n}=23) \end{aligned}$ | $\begin{aligned} & \mathrm{BMI} \geq 30 \mathrm{~kg} / \mathrm{m}^{2} \\ & (\mathrm{n}=17) \end{aligned}$ | P value |
| :---: | :---: | :---: | :---: | :---: |
| UPa-UPp-mm | $\begin{aligned} & 12.8[11-14.3] \\ & (6-17.6) \end{aligned}$ | $\begin{aligned} & 12.5[10.6-13.8] \\ & (6-17.6) \end{aligned}$ | $\begin{aligned} & 14.2[11.1-14.8] \\ & (10-17.4) \end{aligned}$ | 0.02 |
| LPa-LPp-mm | $\begin{aligned} & 11.15[9.55-12.6] \\ & (7.1-17,7) \end{aligned}$ | $\begin{aligned} & 12.1[8.9-12.6] \\ & (7.1-17.7) \end{aligned}$ | $\begin{aligned} & 11.1[10.5-12.4] \\ & (8.3-17.5) \end{aligned}$ | 0.77 |
| S-Go | $\begin{aligned} & 61.75[57.15-72.45] \\ & (26.7-80.07) \end{aligned}$ | $\begin{aligned} & 61.7 \text { [57.1-72.2] } \\ & (26.7-74.8) \end{aligned}$ | $\begin{aligned} & 61.9[61.5-74.6] \\ & (33.9-80.07) \end{aligned}$ | 0.24 |
| SOR-ANS-mm | $\begin{aligned} & 54.4[51.05-61.65] \\ & (24.3-65.4) \end{aligned}$ | $\begin{aligned} & 54.1[52.3-56.8] \\ & (24.3-62.9) \end{aligned}$ | $\begin{aligned} & 60.3 \text { [49.8-61.9] } \\ & (38.5-65.4) \end{aligned}$ | 0.37 |
| ANS-Me | $\begin{aligned} & 57.3 \text { [54.3-60.55] } \\ & (27-77.9) \end{aligned}$ | $\begin{aligned} & 57.2[54.2-60.1] \\ & (27-70.7) \end{aligned}$ | $\begin{aligned} & 60.4[54.63-72.4] \\ & (38.4-77.9) \end{aligned}$ | 0.19 |
| Grown SOR-ANS/ANS-Me | $\begin{aligned} & 6.65[1.8-8.7] \\ & (-12-18.7) \end{aligned}$ | $\begin{aligned} & 7.13 \text { [5.52] } \\ & (-4.6 \text { to } 14.1) \end{aligned}$ | $\begin{aligned} & 2.98[7.77] \\ & (-12 \text { to } 18.7) \end{aligned}$ | 0.06 |
| CVA | $\begin{aligned} & 91.95[83.4-101.7] \\ & (80.5-107.8) \end{aligned}$ | $\begin{aligned} & 94[91.3-101.7] \\ & (80.5-103.9) \end{aligned}$ | $\begin{aligned} & 87.7[83.3-103.7] \\ & (80.5-107.8) \end{aligned}$ | 0.66 |
| SOR-Me | $\begin{aligned} & 110.5 \text { [106.24-120.25] } \\ & (49.3-137.7) \end{aligned}$ | $\begin{aligned} & 110.3 \text { [109.2-119.1] } \\ & (49.3-125.5) \end{aligned}$ | $\begin{aligned} & 120.2[100.2-134.91] \\ & (75.16-137.7) \end{aligned}$ | 0.20 |

Data are expressed as median [25th-75th interquartile range] and minimum to maximum (min-max)
$P$ values are referred to comparisons between patients with $\mathrm{BMI}<30 \mathrm{~kg} / \mathrm{m}^{2}$ or $\geq 30 \mathrm{~kg} / \mathrm{m}^{2}$
UPa-UPp, anterior-posterior diameter of the upper pharynx; LPa-LPp, anterior-posterior diameter of the lower pharynx; S, sella; Go, Gonion; SOR, supraorbital point; ANS, anterior nasal spine; Me, menton; CVA, CranioVertebral Angle

Table 2 shows the correlations between the measurements obtained from the cephalometric analysis and the presence of severe OSAS in the overall population and in the two subpopulations.

In the whole study sample, significant correlations were found between the ANS-Me, Sor-ANS, S-Go, and SorMe scores and severe OSAS. These findings were further confirmed in patients with a $\mathrm{BMI}<30 \mathrm{~kg} / \mathrm{m}^{2}$. In contrast, in patients with a $B M I \geq 30 \mathrm{~kg} / \mathrm{m}^{2}$, we found a significant correlation between Sor-ANS and severe OSAS (Table 2).
The association between these variables and the AHI was stronger (i.e., greater slope) in patients with a
$\mathrm{BMI}<30 \mathrm{~kg} / \mathrm{m}^{2}$ than in those with a $\mathrm{BMI} \geq 30 \mathrm{~kg} / \mathrm{m}^{2}$, as depicted in Fig. 3.
By chance, 20 patients had an $\mathrm{AHI} \leq 30$ (range 5 to 30 ), and 20 patients had an $\mathrm{AHI}>30$ (range 30.6 to59.8). As shown in Table 3, univariate logistic regression analyses demonstrated that ANS-Me, Sor-ANS-mm, S-Go, and Sor-Me were associated with a greater odds ratio for severe OSAS in the overall population. We also performed an analysis of the subpopulation of patients stratified according to BMI.
In patients with a $\mathrm{BMI}<30 \mathrm{~kg} / \mathrm{m}^{2}$, Sor-ANS and SorMe were associated with increased ORs for severe OSAS,

Table 2 Correlations between cephalometric measures and presence of severe OSAS in the overall population and stratified according to BMI

| $\rho$ | Overall population$(n=40)$ |  | $\begin{aligned} & \mathrm{BMI}<30 \mathrm{~kg} / \mathrm{m}^{2} \\ & (\mathrm{n}=23) \end{aligned}$ |  | $\begin{aligned} & \mathrm{BMI} \geq 30 \mathrm{~kg} / \mathrm{m}^{2} \\ & (\mathrm{n}=17) \end{aligned}$ |  |
| :---: | :---: | :---: | :---: | :---: | :---: | :---: |
|  | $\rho$ | $P$ value | $\rho$ | $P$ value | $\rho$ | $P$ value |
| BMI | 0.23 | 0.15 | 0.28 | 0.19 | $-0.08$ | 0.77 |
| ANS-Me | 0.58 | $<0.01$ | 0.69 | $<0.01$ | 0.42 | 0.10 |
| SOR-ANS-mm | 0.64 | $<0.01$ | 0.79 | $<0.01$ | 0.58 | 0.02 |
| S-Go | 0.42 | 0.01 | 0.49 | 0.02 | 0.48 | 0.05 |
| SOR-Me | 0.61 | $<0.01$ | 0.81 | <0.01 | 0.40 | 0.11 |
| CVA | $-0.27$ | 0.09 | $-0.28$ | 0.20 | -0.24 | 0.36 |
| UPa-UPp-mm | 0.03 | 0.86 | 0.00 | 0.99 | 0.11 | 0.66 |

BMI, Body Mass Index; ANS, anterior nasal spine; Me, menton; SOR, supraorbital point; S, sella; Go, Gonion; CVA, CranioVertebral Angle; UPa-UPp, anterior-posterior diameter of the upper pharynx


Fig. 3 Regression lines between the AHI and the ANS-Me, Sor-ANS-mm, S-Go, and Sor-Me scores in patients with a BMI < $30 \mathrm{~kg} / \mathrm{m}^{2}$ (hollow circles) and a $\mathrm{BMI} \geq 30 \mathrm{~kg} / \mathrm{m}^{2}$ (full circles)

Table 3 Univariate logistic regressions of the presence of severe OSAS according to cephalometric data in the whole sample and in patients stratified by BMI

|  | Overall population$(n=40)$ |  | $\begin{aligned} & \mathrm{BMI}<30 \mathrm{~kg} / \mathrm{m}^{2} \\ & (\mathrm{n}=23) \end{aligned}$ |  | $\begin{aligned} & \mathrm{BMI} \geq 30 \mathrm{~kg} / \mathrm{m}^{2} \\ & (\mathrm{n}=17) \end{aligned}$ |  |
| :---: | :---: | :---: | :---: | :---: | :---: | :---: |
|  | OR (95\% CI) | $P$ value | OR (95\% CI) | $P$ value | OR (95\% CI) | $P$ value |
| ANS-Me | 1.10 (1.01-1.19) | 0.02 | 1.21 (0.94-1.56) | 0.14 | 1.07 (0.98-1.17) | 0.13 |
| SOR-ANS-mm | 1.30 (1.09-1.56) | $<0.01$ | 1.60 (1.02-2.49) | 0.04 | 1.20 (1.01-1.43) | 0.04 |
| S-Go | 1.10 (1.01-1.16) | 0.02 | 1.08 (0.97-1.21) | 0.15 | 1.09 (0.99-1.19) | 0.07 |
| SOR-Me | 1.10 (1.01-1.15) | 0.02 | 1.23 (1.02-1.5) | 0.03 | 1.05 (1.00-1.12) | 0.07 |

ANS, anterior nasal spine; Me, menton; SOR, supraorbital point; S, sella; Go, Gonion; OR, odds ratio; $95 \% \mathrm{Cl}, 95 \%$ Confidence Interval
whereas in patients with a $\mathrm{BMI} \geq 30 \mathrm{~kg} / \mathrm{m}^{2}$, only Sor-ANS was associated with severe OSAS.

## Stabilometric examination

Table 4 shows the stabilometric measurements assessed in the overall population and in the subgroup of patients stratified by BMI under different examination conditions (eyes open, eyes close and teeth contact).

Compared to patients with a $\mathrm{BMI}<30 \mathrm{~kg} / \mathrm{m}^{2}$, those with a $\mathrm{BMI} \geq 30 \mathrm{~kg} / \mathrm{m}^{2}$ were characterized by higher values of SA, PL and velocity variance in all the examined conditions.
Notably, we did not find any correlation between the stabilometric measurements and the AHI, either in the overall population or in the subgroup of patients stratified according to BMI. An additional file shows this in more detail (see Additional file 1).

Table 4 Stabilometric measurements in the overall population and stratified according to BMI

|  | Overall population $(n=40)$ | $\begin{aligned} & \mathrm{BMI}<30 \mathrm{~kg} / \mathrm{m}^{2} \\ & (\mathrm{n}=23) \end{aligned}$ | $\begin{aligned} & \begin{array}{l} B M I \geq 30 \mathrm{~kg} / \mathrm{m}^{2} \\ (\mathrm{n}=17) \end{array} \end{aligned}$ | $P$ value |
| :---: | :---: | :---: | :---: | :---: |
| SA-OE mm² | $\begin{aligned} & 69.88 \text { [57.08-94.45] } \\ & (25.94-315.57) \end{aligned}$ | $\begin{aligned} & 58.2[41.27-92.1] \\ & (25.94-118.6) \end{aligned}$ | $\begin{aligned} & 79.32[62.2-100] \\ & (55.13-315.57) \end{aligned}$ | 0.048 |
| PL-OE mm | $\begin{aligned} & 314.45[290-381] \\ & (216.73-606.5) \end{aligned}$ | $\begin{aligned} & 291[257.4-324.4] \\ & (216.73-424.62) \end{aligned}$ | $\begin{aligned} & 324.4[308.78-381.1] \\ & (275.05-606.5) \end{aligned}$ | 0.015 |
| SA-CE mm ${ }^{2}$ | $\begin{aligned} & 126.55 \text { [76.75 217.77] } \\ & (28.4-831.7) \end{aligned}$ | $\begin{aligned} & 77.2[53.24-196.82] \\ & (28.4-357.46) \end{aligned}$ | $\begin{aligned} & 199.76[126.5-278.7] \\ & (101.74-831.7) \end{aligned}$ | 0.001 |
| PL-CE mm | $\begin{aligned} & 495.05 \text { [354.7-639.35] } \\ & (229.6-979.63) \end{aligned}$ | $\begin{aligned} & 354.8 \text { [280.5-546.14] } \\ & (229.6-694.07) \end{aligned}$ | $\begin{aligned} & 546.12 \text { [495.04-643.53] } \\ & (409.1-979.63) \end{aligned}$ | 0.003 |
| SA-MO mm ${ }^{2}$ | $\begin{aligned} & 69.33 \text { [54.26-91.12] } \\ & (25.87-689.72) \end{aligned}$ | $\begin{aligned} & 54.27 \text { [35.2-87] } \\ & (25.87-186.66) \end{aligned}$ | $\begin{aligned} & 83.74 \text { [69.3-91.13] } \\ & (59.35-689.72) \end{aligned}$ | 0.012 |
| PL-MO mm | $\begin{aligned} & 331.54[286.5-377.28] \\ & (201.74-878) \end{aligned}$ | $\begin{aligned} & 287 \text { [233.7-352.75] } \\ & (201.74-493.76) \end{aligned}$ | $\begin{aligned} & 343.73 \text { [331.53-380.55] } \\ & (310.92-878) \end{aligned}$ | 0.004 |
| Velocity variance | $\begin{aligned} & 28 \text { [16-34] } \\ & (7-62) \end{aligned}$ | $\begin{aligned} & 19[10-31] \\ & (7-54) \end{aligned}$ | $\begin{aligned} & 30[26-41] \\ & (20-62) \end{aligned}$ | 0.008 |

Data are expressed as median [interquartile range] e (Min-Max). SA, surface area; PL, Path length; OE, opened eyes; CE, closed eyes; MO, mouth opened

## Discussion

In this pilot observational study, we found that obese and nonobese OSAS patients have different craniofacial morphologies that are correlated with the AHI and are associated with severe OSAS. In addition, although obese OSAS patients were characterized by a different equilibrium as assessed by a stabilometric assessment, we could not find any correlation or association with the severity of OSAS.
Research on cephalometric predictors in patients with OSAS is a cornerstone of sleep medicine [37, 38], as is the investigation of early predictive factors such as craniofacial morphology in children [39]. In OSAS, upper airway obstruction can originate from various sources, including soft tissue collapse, such as the soft palate, or alterations in the position or dimensions of structures such as the tongue, maxilla, or mandible [40-43]. Upper airway obstruction can lead also to intermittent hypoxia, that has systemic effects in OSAS individuals [44]. The evaluation of craniofacial soft tissue is also crucial. For instance, Lee et al. reported that OSAS patients often exhibit broader and flatter mid- and lower-thirds of the face, accompanied by reduced maxillary and mandibular lengths [45]. Similarly, Tyan et al. identified significant correlations between craniofacial measurements and the severity of OSAS [46].
Our findings are in line with previous data reported by other studies. The evidence that OSAS patients are characterized by a different craniofacial morphology has also been described by Akpinar et al.; these authors reported that nonobese OSAS patients have smaller posterior airway spaces and different craniofacial morphologies than do habitual snorers (non-OSAS) and controls [19]. In fact, OSAS patients are characterized by mandibular
micrognathias and retrognathia [47] and the retroposition of the maxilla and mandible [48-50]. OSAS are also characterized by a reduction in the posterior airway space or a multilevel obstruction of the airways [51, 52]. Indeed, systematic reviews have recently reported that a reduced pharyngeal airway space and inferiorly placed hyoid bone are the cephalometric parameters most strongly associated with the severity of OSAS $[53,54]$.
We herein reported that in nonobese OSAS patients, cephalometric measures were moderately correlated with the severity of OSAS; in addition, both the Sor-ANS and Sor-Me distances were associated with severe OSAS in nonobese patients, whereas only the Sor-ANS distance was associated with OSAS severity in obese patients. These findings may be explained by different underlying mechanisms in that subpopulation of patients. In fact, it may be postulated that in subjects with a $\mathrm{BMI}<30 \mathrm{~kg} / \mathrm{m}^{2}$, the severity of OSAS is mainly linked to alterations in skull facial dynamics. In these subjects, obesity likely plays a marginal role in the etiopathogenesis of the disease. Conversely, individuals with first-degree obesity develop OSAS for reasons binding mainly to obesity, which predisposes them to alveolar hypoventilation and airway collapse during sleep. In support of this hypothesis, Tangugsorn et al. also assessed differences in the cervicocraniofacial skeleton between obese and nonobese OSAS patients [55]. Researchers have found that among nonobese individuals with OSAS, anatomical irregularities are primarily limited to the skeletal structures of the cervical and craniofacial regions. In contrast, obese OSAS patients exhibit greater abnormalities in the soft tissue morphology of the upper airway, head posture, and position of the hyoid bone [55]. We also reported a certain correlation between the cephalometric assessment and the AHI (see Table 2).

Patients with OSAS exhibit sleep fragmentation and deprivation, which are suggested to be underlying factors contributing to impairments across various systems, including motor coordination [56-58]. The equilibrium is upheld through the ongoing and efficient integration of vestibular, visual, and proprioceptive inputs within the central nervous system [59]. These sensory data are consolidated and processed in the cerebellum, facilitating the stabilization and upkeep of the body's center of gravity through coordinated postural muscle contractions [60]. In this regard, OSAS patients were shown to be affected by a significant impairment in the vestibulo-ocular and sacculocollic reflexes and posturographic parameters [60-62]. It has also been suggested that these alterations may result from a chronic hypoxemic state that leads to a progressive reduction in vestibular function, generating disequilibrium [62, 63]. Consistent with a previous study [22], we observed an increase in the area and velocity variance with eyes closed and teeth in contact, whereas the area was reduced under conditions with eyes open and teeth in contact, indicating strong muscle rigidity and a continuous search for the center of gravity, supported by the abnormal increase in the length of the trace (the number of oscillations the subject makes to find the center of gravity). Interestingly, it has also been demonstrated that daytime postural stability is influenced by and associated with nocturnal breathing disorders [56]. In our investigation, however, we found neither a correlation between postural disorders and the AHI nor an association between measurements from stabilometric examination and the presence of severe OSAS.
Our study has several limitations. First, given the study aim, we were unable to compute a precise sample size. Although other studies have recruited larger numbers of patients than ours [48], here, we report the findings of a pilot observational study with a total sample ( 40 patients) similar to other studies [49, 50] or to their OSAS subpopulation [19]. In addition, we conducted nonparametric statistical analysis to be more conservative in the results [64]. Second, our findings may contrast with some literature data; this may also be due to an underpowered sample of patients. Third, given the observational nature of the study, the investigation may be affected by selection bias, and the findings may not be representative of a broader population; in addition, there could be several confounding variables [65]. This bias is dampened in our study by the enrollment of all consecutive OSAS patients, with no patients refusing to participate. Finally, while the measurements are standardized, it's important to note that these results are from a singlecenter study. Further investigations conducted at multiple centers are necessary to validate and generalize the findings.

## Conclusions

OSAS patients have altered craniofacial values, which are also related to the severity of OSAS according to the presence of obesity, although they are not the only determinants of severe OSAS. In addition, these patients have a compromised equilibrium unrelated to obesity and not associated with disease severity. Further and larger studies are required to confirm our findings.

## Supplementary Information

The online version contains supplementary material available at https://doi. org/10.1186/s12967-024-05194-8.

Additional file 1:Table S1. Correlations between stabilometric measures and presence of severe OSAS in the overall population and stratified according to BMI. Table S2. Univariate logistic regressions of the presence of severe OSAS according to stabilometric data in the whole sample and in patients stratified by BMI.

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## Author contributions

G.N., E.G. and L.P. designed the study and contributed equally to its realization; V.B. and A.B. collected the data; E.G. and F.L. wrote the article; C.P., and C.B. and all OSAS groups provided technical support; G.T. and A.P. performed the statistical analysis and helped in interpreting the results; A.G. and A.A. contributed to data conceptualization; N.L., G.P., and F.M. revised the final version of the article; A.G., A.B. and E.B. provided data support and reviewed the article for important intellectual content. All authors have read and agreed to the published version of the article.

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## Data availability

The data that support the findings of this study are available upon request from the corresponding author. The data are not publicly available because of privacy or ethical restrictions.

## Declarations

## Ethics approval and consent to participate

The study was conducted in accordance with the Declaration of Helsinki and approved by the Calabria Region Ethics Committee under protocol code 372 on December 15th, 2022.

Informed consent
Written informed consent was obtained from all subjects prior to study inclusion.

## Competing interest

Prof. Federico Longhini contributed to the development of a new helmet for mechanical ventilation, and he is designated an inventor (European Patent number 3320941) not related to the present manuscript. He also received speaking fees from Draeger, Intersurgical and Fisher \& Paykel. The remaining authors have no relevant financial or nonfinancial interests to disclose.

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## References

1. Jordan AS, McSharry DG, Malhotra A. Adult obstructive sleep apnoea. Lancet. 2014;383(9918):736-47. https://doi.org/10.1016/S0140-6736(13) 60734-5.
2. Eckert DJ, White DP, Jordan AS, Malhotra A, Wellman A. Defining phenotypic causes of obstructive sleep apnea. Identification of novel therapeutic targets. Am J Respir Crit Care Med. 2013;188(8):996-1004. https://doi. org/10.1164/rccm.201303-0448OC.
3. Wellman A, Eckert DJ, Jordan AS, Edwards BA, Passaglia CL, Jackson AC, Gautam S, Owens RL, Malhotra A, White DP. A method for measuring and modeling the physiological traits causing obstructive sleep apnea. J Appl Physiol (1985). 2011;110(6):1627-37. https://doi.org/10.1152/japplphysiol. 00972.2010.
4. Kapur VK, Auckley DH, Chowdhuri S, Kuhlmann DC, Mehra R, Ramar K Harrod CG. Clinical practice guideline for diagnostic testing for adult obstructive sleep apnea: an american academy of sleep medicine clinical practice guideline. J Clin Sleep Med. 2017;13(3):479-504. https://doi.org/ 10.5664/jcsm. 6506.
5. Pevernagie DA, Gnidovec-Strazisar B, Grote L, Heinzer R, McNicholas WT, Penzel T, Randerath W, Schiza S, Verbraecken J, Arnardottir ES. On the rise and fall of the apnea-hypopnea index: a historical review and critical appraisal. J Sleep Res. 2020;29(4): e13066. https://doi.org/10.1111/jsr. 13066.
6. Lin WC, Huang CC, Chen HL, Chou KH, Chen PC, Tsai NW, Chen MH, Friedman M, Lin HC, Lu CH. Longitudinal brain structural alterations and systemic inflammation in obstructive sleep apnea before and after surgical treatment. J Transl Med. 2016;14(1):139. https://doi.org/10.1186/ s12967-016-0887-8[pii].
7. Kendzerska T, Leung RS, Aaron SD, Ayas N, Sandoz JS, Gershon AS. Cardiovascular outcomes and all-cause mortality in patients with obstructive sleep apnea and chronic obstructive pulmonary disease (overlap syndrome). Ann Am Thorac Soc. 2019;16(1):71-81. https://doi.org/10.1513/ AnnalsATS.201802-1360C.
8. Ding H, Huang JF, Xie HS, Wang BY, Lin T, Zhao JM, Lin QC. The association between glycometabolism and nonalcoholic fatty liver disease in patients with obstructive sleep apnea. Sleep Breath. 2019;23(1):373-8. https://doi.org/10.1007/s11325-018-1744-1.
9. You CR, Oh JH, Seo M, Lee HY, Joo H, Jung SH, Lee SH, Choi MG. Association between non-erosive reflux disease and high risk of obstructive sleep apnea in Korean population. J Neurogastroenterol Motil. 2014;20(2):197-204.
10. Ayache M, Kellner P, Chiang A. Asthma exacerbation in the spouse of a patient with obstructive sleep apnea. J Clin Sleep Med. 2018;14(9):1631-2.
11. Kuptanon T, Chukumnerd J, Leejakpai A, Preutthipan A. Reliability and validity of Thai version quality of life questionnaire (OSA-18) for pediatric obstructive sleep apnea. J Med Assoc Thai. 2015;98(5):464-71.
12. Choi JW, Song JS, Lee YJ, Won TB, Jeong DU. Increased mortality in relation to insomnia and obstructive sleep Apnea in Korean patients studied with nocturnal polysomnography. J Clin Sleep Med. 2017;13(1):49-56. https://doi.org/10.5664/jcsm. 6386.
13. Mohammadieh A, Sutherland K, Cistulli PA. Sleep disordered breathing: management update. Intern Med J. 2017;47(11):1241-7. https://doi.org/ 10.1111/imj. 13606.
14. Farrell PC, Richards G. Recognition and treatment of sleep-disordered breathing: an important component of chronic disease management. J Transl Med. 2017;15(1):114. https://doi.org/10.1186/ s12967-017-1211-y[pii].
15. Kuna ST, Woodson LC, Solanki DR, Esch O, Frantz DE, Mathru M. Effect of progressive mandibular advancement on pharyngeal airway size in anesthetized adults. Anesthesiology. 2008;109(4):605-12. https://doi.org/ 10.1097/ALN.0b013e31818709fa.
16. Qian Y, Tan JB, Wang T, Bressington D, Zhou HJ, Li MY, Liu XL. Quality appraisal and descriptive analysis of clinical practice guidelines for selfmanaged non-pharmacological interventions of cardiovascular diseases: a systematic review. J Transl Med. 2024;22(1):215. https://doi.org/10.1186/ s12967-024-04959-5.
17. Langaliya A, Alam MK, Hegde U, Panakaje MS, Cervino G, Minervini G. Occurrence of temporomandibular disorders among patients undergoing treatment for obstructive sleep apnoea syndrome (OSAS) using mandibular advancement device (MAD): a systematic review conducted according to PRISMA guidelines and the Cochrane handbook for systematic reviews of interventions. J Oral Rehabil. 2023;50(12):1554-63. https:// doi.org/10.1111/joor. 13574.
18. Barkhordarian A, Demerjian G, Chiappelli F. Translational research of temporomandibular joint pathology: a preliminary biomarker and fMRI study. J Transl Med. 2020;18(1):22. https://doi.org/10.1186/s12967-019-02202-0.
19. Akpinar ME, Celikoyar MM, Altundag A, Kocak I. The comparison of cephalometric characteristics in nonobese obstructive sleep apnea subjects and primary snorers cephalometric measures in nonobese OSA and primary snorers. Eur Arch Otorhinolaryngol. 2011;268(7):1053-9. https:// doi.org/10.1007/s00405-010-1448-z.
20. Tangugsorn V, Krogstad O, Espeland L, Lyberg T. Obstructive sleep apnea (OSA): a cephalometric analysis of severe and non-severe OSA patients. Part II: A predictive discriminant function analysis. Int J Adult Orthodon Orthognath Surg. 2000;15(3):179-91.
21. Bharadwaj R, Ravikumar A, Krishnaswamy NR. Evaluation of craniofacial morphology in patients with obstructive sleep apnea using lateral cephalometry and dynamic MRI. Indian J Dent Res. 2011;22(6):739-48. https:// doi.org/10.4103/0970-9290.94566.
22. Clavel L, Remy-Neris S, Skalli W, Rouch P, Lespert Y, Similowski T, Sandoz B, Attali V. Cervical spine hyperextension and altered posturo-respiratory coupling in patients with obstructive sleep apnea syndrome. Front Med (Lausanne). 2020;7:30. https://doi.org/10.3389/fmed.2020.00030.
23. Stevens D, Jackson B, Carberry J, McLoughlin J, Barr C, Mukherjee S, Oh A, McEvoy RD, Crotty M, Vakulin A. The impact of obstructive sleep apnea on balance, gait, and falls risk: a narrative review of the literature. J Gerontol A Biol Sci Med Sci. 2020;75(12):2450-60. https://doi.org/10.1093/gerona/ glaa014.
24. Demir T, Aslan K, Demirkiran M. Evaluation of postural balance in patients with obstructive sleep apnoea syndrome. Neurol Neurochir Pol. 2020;54(1):83-9. https://doi.org/10.5603/PJNNS.a2019.0059.
25. Ciavarella D, Lorusso M, Campobasso A, Cazzolla AP, Montaruli G, Burlon G, Lo Muzio E, Laurenziello M, Tepedino M. Craniofacial morphology in obstructive sleep apnea patients. J Clin Exp Dent. 2023;15(12):e999-1006. https://doi.org/10.4317/jced.61104.
26. Tepedino M, Illuzzi G, Laurenziello M, Perillo L, Taurino AM, Cassano M, Guida L, Burlon G, Ciavarella D. Craniofacial morphology in patients with obstructive sleep apnea: cephalometric evaluation. Braz J Otorhinolaryngol. 2022;88(2):228-34. https://doi.org/10.1016/j.bjorl.2020.05.026.
27. Soares MM, Romano FL, Dias F, de Souza JF, de Almeida LA, Miura CS, Itikawa CE, Matsumoto MA, Anselmo-Lima WT, Valera FCP. Association between the intensity of obstructive sleep apnea and skeletal alterations in the face and hyoid bone. Braz J Otorhinolaryngol. 2022;88(3):331-6. https://doi.org/10.1016/j.bjorl.2020.06.008.
28. Stipa C, Cameli M, Sorrenti G, Ippolito DR, Pelligra I, Alessandri-Bonetti G. Relationship between cephalometric parameters and the apnoea-hypopnoea index in OSA patients: a retrospective cohort study. Eur J Orthod. 2020;42(1):101-6. https://doi.org/10.1093/ejo/cjz038.
29. Naughton MT, Monteith BD, Manton DJ, Dever P, Schachter LM, O'Brien PE, Dixon JB. Shorter mandibular length is associated with a greater fall in AHI with weight loss. J Clin Sleep Med. 2015;11(4):451-6. https://doi.org/ 10.5664/jcsm. 4604.
30. von Elm E, Altman DG, Egger M, Pocock SJ, Gotzsche PC, Vandenbroucke JP. The Strengthening the Reporting of Observational Studies in Epidemiology (STROBE) Statement: guidelines for reporting observational studies. Int J Surg. 2014;12(12):1495-9. https://doi.org/10.1016/j.ijsu.2014.07.013.
31. Hwang SH, Guilleminault C, Park CS, Kim TW, Hong SC. Usefulness of adenotonsillar size for prediction of severity of obstructive sleep apnea and flow limitation. Otolaryngol Head Neck Surg. 2013;149(2):326-34. https://doi.org/10.1177/0194599813490892.
32. Neri G, Cazzato F, Mastronardi V, Pugliese M, Centurione MA, Di Pietro R, Centurione L. Ultrastructural regenerating features of nasal mucosa following microdebrider-assisted turbinoplasty are related to clinical recovery. J Transl Med. 2016;14(1):164. https://doi.org/10.1186/ s12967-016-0931-8.
33. Berry RB, Budhiraja R, Gottlieb DJ, Gozal D, Iber C, Kapur VK, Marcus CL, Mehra R, Parthasarathy S, Quan SF, Redline S, Strohl KP, Davidson Ward SL, Tangredi MM. Rules for scoring respiratory events in sleep: update of the 2007 AASM manual for the scoring of sleep and associated events. Deliberations of the sleep apnea definitions task force of the American academy of sleep medicine. J Clin Sleep Med. 2012;8(5):597-619. https:// doi.org/10.5664/jcsm. 2172.
34. Tamisier R, Bocquillon V, Treptow E, Destors M, Salvat M, Borrel E, Pepin J. Prevalence and factors contributing to daytime and nocturnal hypoxemia in chronic heart failure patients. Respiration. 2019;97(3):213-22. https://doi.org/10.1159/000490734.
35. Laranjo F, Pinho T. Cephalometric study of the upper airways and dentoalveolar height in open bite patients. Int Orthod. 2014;12(4):467-82. https://doi.org/10.1016/j.ortho.2014.10.005.
36. Rocabado M. Biomechanical relationship of the cranial, cervical, and hyoid regions. J Craniomandibular Pract. 1983;1(3):61-6. https://doi.org/ 10.1080/07345410.1983.11677834.
37. Kushida CA, Littner MR, Morgenthaler T, Alessi CA, Bailey D, Coleman J Jr, Friedman L, Hirshkowitz M, Kapen S, Kramer M, Lee-Chiong T, Loube DL, Owens J, Pancer JP, Wise M. Practice parameters for the indications for polysomnography and related procedures: an update for 2005. Sleep. 2005;28(4):499-521. https://doi.org/10.1093/sleep/28.4.499.
38. Mehta A, Qian J, Petocz P, Darendeliler MA, Cistulli PA. A randomized, controlled study of a mandibular advancement splint for obstructive sleep apnea. Am J Respir Crit Care Med. 2001;163(6):1457-61. https://doi. org/10.1164/ajrccm.163.6.2004213.
39. Ciavarella D, Lo Russo L, Mastrovincenzo M, Padalino S, Montaruli G, Giannatempo G, Cassano M, Laino L, Lo Muzio L. Cephalometric evaluation of tongue position and airway remodelling in children treated with swallowing occlusal contact intercept appliance (S.O.C.I.A.). Int J Pediatr Otorhinolaryngol. 2014;78(11):1857-60. https://doi.org/10.1016/j.ijporl. 2014.08.008.
40. Hotwani K, Sharma K, Jaiswal A. Evaluation of tongue/mandible volume ratio in children with obstructive sleep apnea. Dental Press J Orthod. 2018;23(4):72-8. https://doi.org/10.1590/2177-6709.23.4.072-078.oar.
41. Tabatabaei Balaei A, Sutherland K, Cistulli P, de Chazal P. Prediction of obstructive sleep apnea using facial landmarks. Physiol Meas. 2018;39(9): 094004. https://doi.org/10.1088/1361-6579/aadb35.
42. Tepedino M, lancu-Potrubacz M, Ciavarella D, Masedu F, Marchione L, Chimenti C. Expansion of permanent first molars with rapid maxillary expansion appliance anchored on primary second molars. J Clin Exp Dent. 2018;10(3):e241-7. https://doi.org/10.4317/jced.54585.
43. Endo S, Mataki S, Kurosaki N. Cephalometric evaluation of craniofacial and upper airway structures in Japanese patients with obstructive sleep apnea. J Med Dent Sci. 2003;50(1):109-20.
44. Morin R, Mauger JF, Amaratunga R, Imbeault P. The effect of acute intermittent hypoxia on postprandial triglyceride levels in humans: a randomized crossover trial. J Transl Med. 2021;19(1):268. https://doi.org/ 10.1186/s12967-021-02933-z.
45. Lee RW, Petocz P, Prvan T, Chan AS, Grunstein RR, Cistulli PA. Prediction of obstructive sleep apnea with craniofacial photographic analysis. Sleep. 2009;32(1):46-52.
46. Tyan M, Espinoza-Cuadros F, Fernandez Pozo R, Toledano D, Lopez Gonzalo E, Alcazar Ramirez JD, Hernandez Gomez LA. Obstructive sleep apnea in women: study of speech and craniofacial characteristics. JMIR Mhealth Uhealth. 2017;5(11): e169. https://doi.org/10.2196/mhealth.8238.
47. Rintala A, Nordstrom R, Partinen M, Ranta R, Sjoblad A. Cephalometric analysis of the obstructive sleep apnea syndrome. Proc Finn Dent Soc. 1991;87(1):177-82.
48. Battagel JM, L'Estrange PR. The cephalometric morphology of patients with obstructive sleep apnoea (OSA). Eur J Orthod. 1996;18(6):557-69. https://doi.org/10.1093/ejo/18.6.557.
49. Zucconi M, Ferini-Strambi L, Palazzi S, Curci C, Cucchi E, Smirne S. Craniofacial cephalometric evaluation in habitual snorers with and without obstructive sleep apnea. Otolaryngol Head Neck Surg. 1993;109(6):100713. https://doi.org/10.1177/019459989310900606.
50. deBerry-Borowiecki B, Kukwa A, Blanks RH. Cephalometric analysis for diagnosis and treatment of obstructive sleep apnea. Laryngoscope. 1988;98(2):226-34. https://doi.org/10.1288/00005537-198802000-00021.
51. Riley RW, Powell NB, Guilleminault C. Obstructive sleep apnea and the hyoid: a revised surgical procedure. Otolaryngol Head Neck Surg. 1994;111(6):717-21. https://doi.org/10.1177/019459989411100604.
52. Djupesland G, Lyberg T, Krogstad O. Cephalometric analysis and surgical treatment of patients with obstructive sleep apnea syndrome A preliminary report. Acta Otolaryngol. 1987;103(5-6):551-7.
53. Neelapu BC, Kharbanda OP, Sardana HK, Balachandran R, Sardana V, Kapoor P, Gupta A, Vasamsetti S. Craniofacial and upper airway morphology in adult obstructive sleep apnea patients: a systematic review and meta-analysis of cephalometric studies. Sleep Med Rev. 2017;31:79-90. https://doi.org/10.1016/j.smrv.2016.01.007.
54. Armalaite J, Lopatiene K. Lateral teleradiography of the head as a diagnostic tool used to predict obstructive sleep apnea. Dentomaxillofac Radiol. 2016;45(1):20150085. https://doi.org/10.1259/dmfr. 20150085.
55. Tangugsorn V, Krogstad O, Espeland L, Lyberg T. Obstructive sleep apnoea: multiple comparisons of cephalometric variables of obese and non-obese patients. J Craniomaxillofac Surg. 2000;28(4):204-12. https:// doi.org/10.1054/jcms.2000.0147.
56. Degache F, Goy Y, Vat S, Haba Rubio J, Contal O, Heinzer R. Sleep-disordered breathing and daytime postural stability. Thorax. 2016;71(6):543-8. https://doi.org/10.1136/thoraxjnl-2015-207490.
57. Archbold KH, Borghesani PR, Mahurin RK, Kapur VK, Landis CA. Neural activation patterns during working memory tasks and OSA disease severity: preliminary findings. J Clin Sleep Med. 2009;5(1):21-7.
58. Liang YY, Chen J, Peng M, Zhou J, Chen X, Tan X, Wang N, Ma H, Guo L, Zhang J, Wing YK, Geng Q, Ai S. Association between sleep duration and metabolic syndrome: linear and nonlinear Mendelian randomization analyses. J Transl Med. 2023;21(1):90. https://doi.org/10.1186/ s12967-023-03920-2.
59. Redfern MS, Yardley L, Bronstein AM. Visual influences on balance. J Anxiety Disord. 2001;15(1-2):81-94. https://doi.org/10.1016/s0887-6185(00) 00043-8.
60. Micarelli A, Liguori C, Viziano A, Izzi F, Placidi F, Alessandrini M. Integrating postural and vestibular dimensions to depict impairment in moderate-to-severe obstructive sleep apnea syndrome patients. J Sleep Res. 2017;26(4):487-94. https://doi.org/10.1111/jsr. 12516.
61. Kayabasi S, Iriz A, Cayonu M, Cengiz B, Acar A, Boynuegri S, Mujdeci B, Eryilmaz A. Vestibular functions were found to be impaired in patients with moderate-to-severe obstructive sleep apnea. Laryngoscope. 2015;125(5):1244-8. https://doi.org/10.1002/lary.25021.
62. Gallina S, Dispenza F, Kulamarva G, Riggio F, Speciale R. Obstructive sleep apnoea syndrome (OSAS): effects on the vestibular system. Acta Otorhinolaryngol Ital. 2010;30(6):281-4.
63. Sowerby LJ, Rotenberg B, Brine M, George CF, Parnes LS. Sleep apnea, daytime somnolence, and idiopathic dizziness-a novel association. Laryngoscope. 2010;120(6):1274-8. https://doi.org/10.1002/lary.20899.
64. Nahm FS. Nonparametric statistical tests for the continuous data: the basic concept and the practical use. Korean J Anesthesiol. 2016;69(1):814. https://doi.org/10.4097/kjae.2016.69.1.8.
65. Hammer GP, du Prel JB, Blettner M. Avoiding bias in observational studies: part 8 in a series of articles on evaluation of scientific publications. Dtsch Arztebl Int. 2009;106(41):664-8. https://doi.org/10.3238/arztebl.2009. 0664.

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