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Original Article

Comparison between the Risk of Developing Sleep Disorders with Lung Mechanics and Thoracic Ultrasound Signals in Adults with Obesity

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Sleep Sci

Abstract

Objective The present study aimed to compare the risk of developing sleep disorders with abnormalities in lung mechanics, abnormal ultrasound signals, and anthropometric parameters in adults with obesity.

Materials and Methods A total of 50 individuals were assessed for the risk of obstructive sleep apnea (OSA) using the Mallampati classification, the Epworth Sleepiness Scale (ESS), the Snoring, Tiredness, Observed Apnea, High Blood Pressure, Body Mass Index, Age, Neck Circumference, and Gender (STOP-Bang) questionnaire, and the Sleep Apnea Clinical Score (SACS). Patients also underwent respiratory oscillometry, spirometry, and thoracic ultrasound.

Results The subgroup with abnormal respiratory oscillometry was more likely to have an ESS score indicating a high risk of developing OSA (87.5%) than the subgroup with normal respiratory oscillometry (42.9%) ($p = 0.024$). On thoracic ultrasound, the frequency of patients with a Mallampati classification of high risk of developing OSA was greater in the subgroup with > 2 B-lines (80%) than in the subgroup with ≤ 2 B-lines (25.7%) ($p = 0.0003$). The subgroup with subpleural consolidations was more likely to have an OSA-indicative ESS score (100%) than the subgroup without subpleural consolidations (41.9%) ($p = 0.004$). According to the multivariate analysis, > 2 B lines and body mass index were found to be independent variables for predicting the Mallampati classification, while subpleural consolidation was the only independent variable for predicting the ESS score.

Conclusion In adults with obesity, the greater the risk of developing OSA was, the worse the resistive and reactive parameters measured by respiratory oscillometry. Abnormal respiratory oscillometry and abnormal thoracic ultrasound are factors associated with a high risk of developing OSA.

Keywords
- obesity
- sleep
- respiratory oscillometry
- thoracic ultrasound
- spirometry

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Introduction

Obesity is a complex, multifactorial disease that has become more common worldwide in the past 50 years, with significant health and socioeconomic implications.[1] The two major contributors to the pandemic-level prevalence of obesity are believed to be changes in the food system and increased sedentary behavior.[2,3] Obesity negatively affects almost all physiological functions of the body and represents a significant threat to public health.[4] In fact, it is one of the most important risk factors for noncommunicable diseases, substantially increasing the incidence of diabetes mellitus, cardiovascular disease, stroke, dementia, osteoarthritis, various types of cancer, and obstructive sleep apnea (OSA), which is the most common form of sleep-disordered breathing.[4,5] Obesity is also associated with reduced quality of life and life expectancy, unemployment, lower socioeconomic productivity, and social disadvantage, increasingly creating an economic burden.[3]

As obesity affects the lungs and airways, lung volume shrinks, especially expiratory reserve volume. Although total lung capacity is preserved in many individuals with obesity, in cases of severe obesity, expiratory reserve volume stabilizes at a minimal operational value as the smaller airways close, and both total lung capacity and inspiratory capacity start to decrease. This combination of reduced lung volume and small airway closure increases the amount of work required to maintain ventilation.[6] Although neck circumference is closely associated with OSA, a reduction in lung volume also reduces the longitudinal traction of the upper airways, contributing to their collapse.[7] Overall, OSA and obesity hypoventilation syndrome have become more common and are associated with increased pulmonary morbidity and decreased quality of life.[1] Obstructive sleep apnea is characterized by repetitive upper-airway collapse, while obesity hypoventilation syndrome is characterized by sustained nocturnal hypoventilation causing diurnal hypercapnia.[1]

Respiratory mechanics can be assessed noninvasively and effortlessly by respiratory oscillometry, which consists of applying single-frequency or multifrequency pressure pulses to the airways.[8] Respiratory oscillometry has been increasingly used in individuals with obesity, in whom the effects on the reactance of the respiratory system (Xrs) are more apparent, which suggests that there is an increase in the heterogeneous narrowing of the airways and in the closing of the airways in the pulmonary periphery, although increased chest wall stiffness may also contribute to these changes.[9] In addition to impairing lung function, obesity is a predisposing condition for the formation of atelectasis, for which lesions are easily diagnosed by thoracic ultrasound.[10] An increase in adipose tissue in the abdomen, diaphragm, and intercostal muscles alters the pressure and volume properties of the chest, with a reduction in the chest wall and lung compliance favoring the formation of atelectasis.[11]

Although upper-airway collapsibility plays an important role in obesity in sleep disorders, the cycle of obstruction and restoration of upper-airway patency is expected to be accompanied by wide fluctuations in intrathoracic pressure and changes in the mechanical properties of the respiratory system.[12] In this sense, respiratory oscillometry is a sensitive technique for identifying abnormalities of the respiratory system in people with obesity and is recommended for evaluating sleep disorders,[13,14] while thoracic ultrasound can be used to diagnose pulmonary structural changes early that may even reflect the effects of tracheal traction.[15,16] Since excessive fat tissue can decrease chest wall retraction properties, leading to distal airway closure due to a lack of supporting structures and reduced lung volume, we hypothesized that there are interrelationships between lung structure/function, as evaluated by respiratory oscillometry and thoracic ultrasound, and the risk of developing sleep disorders, as evaluated by simple tools applied in clinical practice. Thus, in this study, we aimed to compare the risk of developing sleep disorders with abnormalities in lung mechanics, abnormal ultrasound signals, and anthropometric parameters in adults with obesity.

Materials and Methods

Study Design and Participants

A cross-sectional study was conducted between April and August 2023 with adults (aged $\geq$ 18 years) with obesity (body mass index [BMI] $\geq$ 30 kg/m^2) of both sexes of the Piquet Carneiro Policlinic at the State University of Rio de Janeiro, Rio de Janeiro, Brazil. The following exclusion criteria were adopted: history of smoking ($>$ 10 pack-years), upper respiratory tract infection in the 4 weeks prior to recruitment, history of previous or current cardiopulmonary disease, history of previous or current neuromuscular disease, disorders of the upper respiratory tract, and inability to perform the functional tests.

The project was approved by the Research Ethics Committee of the Bonsucesso Federal Hospital under the number CAAE-65762122.3.0000.5253, and all participants provided informed consent before the evaluations.

Measurements

The study participants completed an anamnesis questionnaire, and data such as age, sex, preexisting disease status, weight, height, BMI, and neck, waist, and hip circumferences were recorded. Neck circumference was measured from the midpoint of the cervical spine to the middle of the anterior neck. Waist circumference was measured based on the lower portion of the left costal margin and the left anterior superior iliac crest. Hip circumference was measured by taking the largest diameter of the gluteal region passing over the greater trochanters of the femur.[17]

All participants underwent upper-airway examination and evaluation by the Mallampati classification. The participants were instructed to breathe through the nose after a single swallow and to open their mouth wide with voluntary tongue protrusion without phonation. All evaluations were performed by the same investigator. The extent of oropharyngeal obstruction was determined by the Mallampati classification. Classes I and II were defined as having a low risk of developing OSA, while classes III and IV were defined as having a high risk of developing OSA.[18]

We used the Epworth Sleepiness Scale (ESS), which evaluates the probability that a person will fall asleep during ADLs. It consists of eight questions, each with a severity score of 0 to 3, resulting in a total score between 0 and 24 points. An ESS score ≥ 11 indicates excessive daytime sleepiness and a high risk of developing OSA.[19]

We used the Snoring, Tiredness, Observed Apnea, High Blood Pressure, Body Mass Index, Age, Neck Circumference, and Gender (STOP-Bang) questionnaire, which is easy to apply. There are eight yes or no questions. It was developed from variables already recognized as being associated with OSA. The presence of three or more affirmative answers indicates a high risk of developing OAS, while two or fewer affirmative answers indicates a low risk.[20]

The Sleep Apnea Clinical Score (SACS) was also used to assess the risk of developing OSA. It consists of three questions, in addition to the measurement of neck circumference and the assessment of the presence or absence of hypertension. The score ranges from 0 to 110 points, and scores < 15 points indicate that the patient is at high risk of developing OSA, while scores ≥ 15 points indicate low risk.[21]

Respiratory oscillometry was performed with Quark i2m equipment (Cosmed, Rome, Italy) according to the European Respiratory Society recommendations.[22] The participants were instructed to keep their lips around the mouthpiece and breathe normally for 40 seconds while keeping their cheeks pressed with their hands to reduce the need for an upper-airway shunt. Before each test, the system was calibrated as recommended by the manufacturer. The following resistive and reactive parameters were evaluated: respiratory system resistance at 5 Hz (R5) and 20 Hz (R20); heterogeneity of respiratory system resistance between 5 and 20 Hz (R5–R20); resonance frequency (Fres); Xrs at 5 Hz (X5) and 20 Hz (X20); and area under the reactance curve (AX, reactance between 5 Hz and Fres). Fres $>$ 12 Hz and AX $\geq$ 8.66 kPa/L/s were considered abnormal results.[23,24]

After a 5-minute rest period after respiratory oscillometry was performed, spirometry was performed with Spiromax equipment (Codax Ltda., Rio de Janeiro, RJ, Brazil), which was integrated into the Spiromatic 2.0 program (Engelógica, Rio de Janeiro, RJ, Brazil). The standardization used was based on the American Thoracic Society/European Respiratory Society guidelines.[25] The highest values of forced expiratory volume in 1 second (FEV_1) and forced vital capacity (FVC) of the 3 technically acceptable maneuvers were used for analysis. The values obtained from the participants were compared with the national reference values.[26] Restrictive ventilatory impairment was defined as a FVC $< 80\%$ of the predicted value, whereas obstructive ventilatory impairment was defined as a FEV_1/FVC ratio $< 70\%$ of the predicted value.[27] Spirometry was performed after respiratory oscillometry to avoid the influence of the forced expiratory maneuver and respiratory muscle fatigue on the respiratory oscillometry measurements.[13]

Finally, the participants underwent thoracic ultrasound with a Mindray equipment Z.One PRO model (Mindray Biomedical Electronics Co., Ltd., China) coupled to a linear multifrequency transducer of 7.5 to 10 MHz or to a convex transducer of 3.5 to 5 MHz in B mode. The evaluations were performed by sonographers with at least 10 years of experience. All thoracic ultrasounds were evaluated by two examiners, and when there was disagreement between them, it was resolved by consensus. With the participants in a sitting position, thoracic ultrasound signals were captured in six areas of each hemithorax as follows:[28] two anterior, two lateral, and two posterior areas. In the evaluation of abnormal signs on thoracic ultrasound, we sought to find $>$ 2 B-lines, coalescing B-lines, subpleural consolidations, and pleural thickening/irregularity.[29]

Statistical Analyses

Statistical analysis was performed using the IBM SPSS Statistics for Windows, version 26.0 (IBM Corp, Armonk NY, USA) software. The normality of the data distribution was assessed using the Shapiro-Wilk test. The data are expressed as measures of central tendency and dispersion suitable for numerical data and as n (%) for categorical data. Comparisons between categorical variables between two independent groups that were not normally distributed were analyzed using the Chi-squared test or Fisher exact test: if $\leq 20\%$ of the expected cell counts were < 5, then the Chi-squared test was used; if $> 20\%$ of the expected cell counts were < 5, then the Fisher exact test was used.[30] Comparisons of numerical variables between two independent groups that were not normally distributed were analyzed using the Mann-Whitney U test.[31] A multivariate analysis was performed using multiple linear regression to identify the independent variables that explained the variability in sleep disorders. Variable selection was performed with the stepwise forward method at the 5% level, which selects the smallest subgroup of independent variables that best explains the dependent variable.[30] The adopted significance level was 5%.

To contextualize the interpretation of null findings, a post-hoc power analysis was performed using G*Power 3.1.1 software (free) based on comparisons of the scales used to assess sleep disorders according to lung mechanics, ultrasound signs, and anthropometric parameters.

Results

Among the 57 adults with obesity who were eligible to participate in the study, 7 were excluded for the following reasons: upper respiratory tract infection in the 4 weeks prior to study recruitment ($n = 3$), history of cardiopulmonary disease ($n = 2$), or history of neuromuscular disease ($n = 2$). Thus, the sample consisted of 50 patients: 31 women and 19 men. The median (interquartile range) age and BMI were 42 (34–58) years and 37 (33–44) kg/m^2, respectively. The characteristics of the participants who were included in this study are shown in ►**Table 1**.

Regarding pulmonary function test results, 12 (24%) participants had abnormal spirometry, 10 (20%) had restrictive ventilatory impairment, 1 (2%) had obstructive ventilatory impairment, and 1 (2%) had mixed ventilatory impairment. On respiratory oscillometry, 42 (84%) participants had abnormal results; Fres was $>$ 12 Hz in 10 (20%) patients, while AX was $>$ 8.66 kPa/L/s in 37 (74%) participants. The most common abnormal sign on thoracic ultrasound was pleural

Table 1 Medians and interquartile ranges (25–75%) or numbers (percentages) for the characteristics of the participants included in the study.

Variables	Values
Demographics	
Age (years)	42 (34–58)
Female (%)	31 (62%)
Anthropometry	
Weight (kg)	109 (88–126)
Height (m)	1.67 (1.59–1.74)
Body mass index (kg/m^2)	37 (33–44)
Waist circumference (cm)	115 (104–125)
Hip circumference (cm)	117 (111–135)
Waist-to-hip ratio (cm)	0.94 (0.89–1.03)
Neck circumference (cm)	39 (36–43)
Clinical data	
Hypertension (%)	29 (58%)
Diabetes (%)	9 (18%)
History of smoking (%)	5 (10%)

Table 2 Medians and interquartile ranges (25–75%) or numbers (percentages) of the parameters obtained through pulmonary function tests and thoracic ultrasound.

Variables	Values
Spirometry	
FVC (% predicted)	89 (81–97)
FEV_1 (% predicted)	89 (82–98)
FEV_1/FVC (%)	85 (80–89)
$FEF_{25-75\%}$ (% predicted)	92 (73–111)
Respiratory oscillometry	
R5 (kPa/L/s)	6 (5–7.6)
R20 (kPa/L/s)	5.2 (4.3–6.5)
R5–R20 (kPa/L/s)	0.69 (0.05–1.53)
Fres (Hz)	5.2 (4.2–7.9)
X5 (kPa/L/s)	-2.2 (–3.3 to -1.5)
X20 (kPa/L/s)	-0.63 (–1.45 to -0.09)
AX (kPa/L)	17.2 (8.3–30.7)
Thoracic ultrasound	
> 2 B-lines (%)	15 (30%)
Coalescent B-lines (%)	2 (4%)
Subpleural consolidations (%)	7 (14%)
Pleural thickening/ irregularity (%)	36 (72%)

Abbreviations: AX, area under the reactance curve; $FEF_{25-75\%}$, forced expiratory flow during the middle half of the FVC maneuver; FEV_1, forced expiratory volume in 1 second; Fres, resonance frequency; FVC, forced vital capacity; R20, respiratory system resistance at 20 Hz; R5, respiratory system resistance at 5 Hz; R5–R20, heterogeneity of respiratory system resistance between 5–20 Hz; X20, respiratory system reactance at 20 Hz; X5, respiratory system reactance at 5 Hz.

thickening/irregularity, which was present in 36 (72%) participants. The pulmonary function test and thoracic ultrasound data are shown in ►**Table 2**.

Regarding the scales used to assess sleep disorders, 21 (42%) participants were at high risk of developing OSA according to the Mallampati classification, while 25 (50%) participants were at high risk of developing OSA according to the ESS score. According to the STOP-Bang questionnaire, 28 participants (56%) were at high risk of developing OAS, and only 12 (24%) participants were at high risk of developing OSA according to the SACS. The distribution of participants according to the scales used to assess sleep disorders is shown in ►**Table 3**.

When comparing the scores used to assess sleep disorders with the characteristics of the participants, hypertension was more prevalent among patients with high-risk STOP-Bang status (82.1%) than among those with low-risk STOP-Bang status (27.3%) ($p < 0.0001$), and hypertension was more prevalent among patients with a high-risk SACS (91.7%) than among those with a low-risk SACS (47.4%) ($p = 0.006$). Male sex was more prevalent among patients with a high-risk SACS (75%) than among those with a low-risk SACS (26.3%) ($p = 0.003$); in contrast, female sex was more prevalent among individuals with a low-risk SACS (73.7%) than among those with a high-risk SACS (25%) ($p = 0.003$).

According to the Mallampati classification, AX (14.8 [7–26] versus 19 [13–53] kPa/L, $p = 0.017$) was lower in participants at low risk of developing OSA than in those at high risk. According to the ESS results, FVC (93 [86–98] versus 86 [72–91]% predicted, $p = 0.018$), FEV_1 (97 [88–100] versus 87 [77–93]% predicted, $p = 0.010$), and forced expiratory flow during the middle half of the FVC maneuver ($FEF_{25-75\%}$) (103 [84–120] versus 83 [70–98], $p = 0.035$) were greater in participants at low risk of developing OSA than in those at high risk, while Fres (4.8 [4–6.4] versus 5.9 [5–14] Hz, $p = 0.039$) was lower in participants at low risk of developing OSA than in those at high risk. According to the STOP-Bang results, the FEV_1/FVC (87 [81–91] versus 82 [77–86]%, $p = 0.011$) was greater in participants at low risk of developing OSA than in those at high risk, while the R5–R20 (0.25 [0.05–0.71] versus 1.17 [0.49–2.03] kPa/L/s, $p = 0.001$) was lower in participants at low risk of developing OSA than in those at high risk. None of the scales used to assess sleep disorders showed significant differences between the subgroups with normal and abnormal spirometry. The subgroup with abnormal respiratory oscillometry was more likely to have an ESS score indicating a high risk of developing OSA (87.5%) than the subgroup with normal respiratory oscillometry (42.9%) ($p = 0.024$). Comparisons of the scales used to assess sleep disorders according to the participants' characteristics and pulmonary function test parameters are shown in ►**Table 4**.

When comparing the scores used to assess sleep disorders with abnormal thoracic ultrasound parameters, the frequency of patients with a Mallampati classification of high OSA

Table 3 Numbers (percentages) of the results of the scales used to assess sleep disorders.

Variables	Values
Mallampati classification	
Class I and II	29 (58%)
Class III and IV	21 (42%)
ESS	
Score < 11	25 (50%)
Score ≥ 11	25 (50%)
STOP-Bang	
Score ≤ 2	22 (44%)
Score > 3	28 (56%)
SACS	
Score < 15	38 (76%)
Score ≥ 15	12 (24%)

Abbreviations: ESS, Epworth sleepiness scale; SACS, Sleep Apnea Clinical Score; STOP-Bang, Snoring, Tiredness, Observed Apnea, High Blood Pressure, Body Mass Index, Age, Neck Circumference, and Gender.

risk was greater in the subgroup with > 2 B-lines (80%) than in the subgroup with ≤ 2 B-lines (25.7%) ($p = 0.0003$). The prevalence of an ESS score indicating a high risk of developing OSA was greater among patients with subpleural consolidations (100%) than among those without subpleural consolidations (41.9%) ($p = 0.004$).

Using a regression model, we observed that > 2 B-line status and BMI were significant independent variables for predicting the Mallampati classification. Subpleural consolidations were the only significant independent variable for predicting the ESS score. Hypertension, R5–R20, and the waist-to-hip ratio were significant independent variables for predicting STOP-Bang status. Neck circumference was the only significant independent variable for predicting the SACS. Stepwise forward regression analysis results for sleep disorders using anthropometry, clinical data, lung function, and thoracic ultrasound parameters are shown in ►**Table 5**.

Based on an a priori type I error of $\alpha = 0.05$ (two-tailed), the power analysis showed that significant effects were detected in the comparisons of the scales used to assess sleep disorders according to lung mechanics, ultrasound signs, and anthropometric parameters. The effect size varied between 0.81 and 1.37, and the power varied between 92 and 97%; therefore, the sample size was adequate to obtain significant results.[32]

Discussion

The main findings of the present study were that, in people with obesity, the risk of developing OSA was associated with lung mechanics assessed through respiratory oscillometry, considering both resistive and reactive parameters. Although the risk of developing OSA was not different in adults with obesity according to the spirometry, those with abnormal respiratory oscillometry were at high risk of developing OSA. Abnormal thoracic ultrasound findings were more likely to be found in people at high risk of developing OSA. Almost all of the adults with obesity had an abnormality in the respiratory oscillometry and, to a lesser extent, in the thoracic ultrasound, although they rarely had an abnormality in spirometry.

Sleep health has been increasingly recognized because it encompasses multiple dimensions, where sleep duration, sleep quality, and daytime sleepiness may be associated with obesity.[33] In many individuals with obesity, sleep disorders or abnormal respiratory oscillometry, there are minimal changes in spirometry, except very severe obesity, which can affect traditional pulmonary function test results.[34] Using simple and easy-to-administer questionnaires, we demonstrated that adults with obesity and abnormal respiratory oscillometry were at high risk of developing OSA, although the risk of developing OSA did not differ according to whether they had abnormal spirometry. Notably, patients with a high risk of developing OSA according to the ESS score were much more prevalent in the subgroup with abnormal respiratory oscillometry than in the subgroup with normal respiratory oscillometry. It is hypothesized that the relationship between the risk of developing sleep disorders and obesity represents the effects of high levels of proinflammatory cytokines or dysfunction of the hypothalamic—pituitary—adrenal axis and that the low-grade chronic inflammation that occurs in OSA may contribute to a greater incidence of pulmonary morbidity and mortality.[35,36] Notably, we used four criteria (the ESS score, STOP-Bang status, the SACS, and Mallampati classification) to assess the risk of developing OSA. As the diagnostic performance of clinical questionnaires and exam findings differ in their sensitivity, specificity, positive predictive value, and negative predictive value when used in various populations, the combination of these tools can improve the accuracy of assessing the risk of developing OSA.[37–39] Furthermore, we aimed to evaluate the associations of each of these four criteria with lung mechanics, ultrasound signs, and anthropometric parameters.

In people with obesity, increases in elastic recoil forces are associated with a reduction in functional residual capacity and with marked increases in both upper- and peripheral-airway resistance.[16] Given that high upper-peripheral airway resistance is a hallmark of OSA, respiratory oscillometry is particularly suitable for detecting airway obstruction and, therefore, for application in this population[34]. Using the STOP-Bang questionnaire, we observed that R5–R20 (which is a marker of abnormalities in the small airways) was greater in individuals at high risk of developing OSA (this finding was confirmed in the multivariate analysis). In line with our findings, Dattani et al. (2016)[40] reported that visceral adipose tissue mass was associated with R5–20 in individuals with obesity, indicating that the effect of abdominal mass load on peripheral-airway resistance may be even greater in the small airways. In individuals with obesity, respiratory system resistance measured by respiratory oscillometry increases due to a reduction in operating lung volume, but this change is not the entire reason for this increase, as the structure of the airways can be remodelled by exposure to

Table 4 Comparisons of the scales used to assess sleep disorders according to participant characteristics and pulmonary function tests.

	Mallampati classification: median (IQR)			ESS: median (IQR)			STOP-Bang: median (IQR)			SACS: median (IQR)		
	Low risk	High risk	*P*	Low risk	High risk	*p*	Low risk	High risk	*p*	Low risk	High risk	*p*
Age (years)	40 (36–57)	43 (31–62)	0.96	38 (29–58)	43 (36–58)	0.35	36 (30–42)	53 (37–62)	**0.005**	40 (33–58)	43 (36–60)	0.77
BMI (kg/m^2)	34.8 (33–42)	39.3 (36–49)	**0.010**	38.1 (34–48)	36.7 (33–42)	0.42	37 (33–43)	37 (34–45)	0.35	36.3 (33–45)	40 (36–43)	0.25
Waist circumference (cm)	113 (101–123)	120 (107–136)	0.10	114 (103–129)	115 (108–123)	0.82	110 (101–120)	121 (108–132)	**0.045**	111 (102–123)	120 (114–125)	**0.041**
Hip circumference (cm)	116 (110–130)	120 (113–139)	0.18	120 (113–129)	117 (110–124)	0.13	118 (114–135)	117 (109–136)	0.58	118 (111–137)	117 (110–132)	0.58
Waist-to-hip ratio (cm)	0.92 (0.88–1.01)	0.98 (0.91–1.05)	0.22	0.92 (0.88–0.99)	0.99 (0.90–1.05)	**0.034**	0.91 (0.87–0.97)	1.01 (0.89–1.07)	**0.005**	0.92 (0.88–0.99)	1.04 (1.02–1.08)	**0.001**
Neck circumference (cm)	39 (37–42)	39 (36–44)	0.57	39 (36–42)	41 (37–44)	0.18	39 (36–42)	41 (36–43)	0.29	38 (36–42)	44 (42–46)	**0.0001**
FVC (% predicted)	87 (80–100)	89 (82–94)	0.83	**86 (72–91)**	**93 (86–98)**	**0.018**	89 (82–95)	89 (80–98)	0.86	93 (82–102)	88 (80–95)	0.27
FEV_1 (% predicted)	94 (84–98)	88 (81–97)	0.50	**87 (77–93)**	**97 (88–100)**	**0.010**	90 (85–98)	88 (76–98)	0.61	93 (77–100)	88 (82–97)	0.63
FEV_1/FVC (%)	86 (80–89)	82 (78–89)	0.33	86 (79–89)	85 (81–89)	0.86	**87 (81–91)**	**82 (77–86)**	**0.011**	86 (81–89)	84 (76–86)	0.090
$FEF_{25-75\%}$ (% predicted)	95 (80–116)	85 (71–107)	0.29	**103 (84–120)**	**83 (70–98)**	**0.035**	92 (78–123)	90 (65–107)	0.42	97 (59–118)	92 (76–105)	0.89
R5 (kPa/L/s)	5.4 (4.8–6.8)	6.7 (5.3–8.7)	0.07	6.4 (5–7.3)	5.7 (5–8.9)	0.92	5.5 (4.9–6.6)	6.7 (5.1–9)	0.059	5.3 (5–9)	6.1 (5–7.1)	0.91
R20 (kPa/L/s)	4.8 (4.3–6.2)	5.6 (4.4–7)	0.23	5.3 (4.5–6.6)	4.8 (4.3–6.5)	0.53	5.2 (4.5–6)	5 (4.3–7)	0.97	5 (4.3–8.2)	5.2 (4.4–6.2)	0.73
R5–R20 (kPa/L/s)	0.69 (-0.06–1.48)	0.68 (0.17–1.78)	0.38	0.63 (0.04–1.81)	0.71 (0.18–1.51)	0.65	**0.25 (0.05–0.71)**	**1.17 (0.49–2.03)**	**0.001**	0.57 (0.03–1.42)	0.69 (0.07–1.64)	0.47
Fres (Hz)	5.1 (4–7)	5.6 (4.8–8.6)	0.25	**4.8 (4–6.4)**	**5.9 (5–14)**	**0.039**	4.9 (4.2–8.1)	5.5 (4.3–7)	0.82	4.9 (4.2–8)	5.3 (4.2–8)	0.74
X5 (kPa/L/s)	-2.2 (-3.3 to -1.4)	-2.2 (-3.9 to -1.6)	0.69	-2.2 (-3.3 to -1.9)	-2.2 (-3.3 to -1.3)	0.55	-2.2 (-3.2 to -1.4)	-2.2 (-3.3 to -1.5)	0.79	-1.9 (-3.3 to -1.3)	-2.2 (-3.3 to -1.9)	0.64
X20 (kPa/L/s)	-0.51 (-1.3 to -0.04)	-0.67 (-2 to -0.13)	0.31	-0.66 (-1.4 to -0.13)	-0.52 (-1.8 to -0.04)	0.66	-0.57 (-1.3 to -0.01)	-0.70 (-1.9 to -0.11)	0.39	-0.57 (-1.4 to -0.04)	-0.78 (-2.4 to -0.13)	0.31
AX (kPa/L)	**14.8 (7–26)**	**19 (13–53)**	**0.017**	14 (7–36)	19 (12–29)	0.55	16 (12–36)	19 (6–29)	0.77	15 (9–36)	18 (8–31)	0.78

Abbreviations: AX, area under the reactance curve; BMI, body mass index; ESS, Epworth Sleepiness Scale; $FEF_{25-75\%}$, forced expiratory flow during the middle half of the FVC maneuver; FEV_1, forced expiratory volume in 1 second; Fres, resonance frequency; FVC, forced vital capacity; R20, respiratory system resistance at 20 Hz; R5, respiratory system resistance at 5 Hz; R5–R20, heterogeneity of respiratory system resistance between 5 and 20 Hz; IQR, interquartile range; SACS, Sleep Apnea Clinical Score; STOP-Bang, Snoring, Tiredness, Observed Apnea, High Blood Pressure, Body Mass Index, Age, Neck Circumference, and Gender X20, respiratory system reactance at 20 Hz; X5, respiratory system reactance at 5 Hz.

Note: The comparisons were analyzed using the Mann-Whitney U test.

Table 5 Stepwise forward regression analysis for sleep disorders using anthropometry, clinical data, lung function, and thoracic ultrasound parameters.

Independent variables	B	SEB	*p*
Mallampati classification			
> 2 B-lines	2.388	0.775	0.002
BMI	0.094	0.050	0.049
ESS			
Subpleural consolidations	2.120	1.123	0.005
STOP-Bang			
Hypertension	3.191	1.029	0.001
R5–R20	1.310	0.539	0.015
Waist-to-hip ratio	13.01	5.791	0.025
SACS			
Neck circumference	0.418	1.136	0.002

Abbreviations: B, regression coefficient; BMI, body mass index; ESS, Epworth Sleepiness Scale; R5–R20, heterogeneity of respiratory system resistance between 5 and 20 Hz; SACS, Sleep Apnea Clinical Score; SEB, standard error of the regression coefficient.

proinflammatory adipokines or by lipid deposition.[34] In fact, Mahadev et al. (2013)[41] observed that, in addition to the reduction in FRC, peripheral-airway resistance in adults with obesity may also be increased by remodelling, which is characterized by fat deposits inside the body and injury to the bronchial mucosa due to the stress of opening and closing the small airways.

Another aspect that has been extensively studied in recent years in individuals with obesity is the change in the reactive properties of the respiratory system, since the increase in fat mass in the chest and abdomen shifts the elastic balance point between the chest and lungs, favoring the displacement of the pressure-volume curve.[14,34,42] In this sense, X5 and Fres may reflect the shift in the frequency-reactance curve, which is usually associated with increased elastance of the respiratory system.[42] Using the Mallampati classification, we observed that AX was greater in individuals at high risk of developing OSA. In fact, obesity can lead to fat deposition on the tongue and soft palate, affecting the size and collapsibility of the upper airways; this decreases the size of the retroglossal airways and increases the risk of developing OSA.[14] Using the ESS score, we observed that Fres was greater in individuals at high risk of developing OSA. Fres is the point at which the elastance and inertia of the respiratory system make equal and opposite contributions to the impedance, that is, at which $Xrs = 0$.[34] In line with our findings, Abdeyrim et al. (2015)[43] identified significant correlations between Xrs and OSA severity defined by the apnea-hypopnea index. These associations between Xrs and OSA severity may indicate that upper-airway stenosis or an abnormal increase in lung elastic recoil is a contributor to OSA.

We sought to evaluate the associations between the risk of developing sleep disorders and thoracic ultrasound parameters, as the latter are derived from a noninvasive, reproducible, fast, inexpensive, and radiation-free diagnostic method.[15] Interestingly, we observed that the frequency of patients with a Mallampati classification indicating a high risk of developing OSA was greater in individuals with > 2 B-lines, and the frequency of patients with an ESS score indicating a high risk of developing OSA was greater in individuals with subpleural consolidations (both findings were confirmed in the multivariate analysis). The clinical significance of B-lines depends mainly on their quality and quantity; B-lines are usually associated with interstitial changes, while subpleural consolidations may represent a fluid bronchogram or vascular image.[44] By dislocating the diaphragm upwards, abdominal adiposity can cause several abnormal signs on thoracic ultrasound, which can reduce lung volume and increase downwards tracheal traction, contributing to the pathophysiology of OSA.[45] In the present study, we observed a high frequency of abnormal thoracic ultrasound signals. In line with our study, Erol et al. (2022)[10] reported an 81% frequency of abnormal thoracic ultrasound signs in the postoperative period in individuals with obesity who underwent laparoscopic bariatric surgery. The authors attributed these abnormalities, at least in part, to the presence of atelectasis.

The strength of this study is that it revealed associations between the scores of the scales used to assess sleep disorders in adults with obesity—which are widely validated and easy to apply—and changes in respiratory mechanics and lung structure. Some limitations should be highlighted. First, our sample was small, and we did not use a control group. Secondly, we did not use nocturnal polysomnography, which is the gold standard for evaluating sleep disorders, although this diagnostic procedure is expensive, time-consuming, and laborious.[16] Third, our respiratory oscillometry and thoracic ultrasound measurements were not performed in the supine position because, in individuals with obesity, lung mechanics change even more in the supine position; the resulting decrease in lung volume may facilitate the collapse of the pharyngeal and intrathoracic airways due to the loss of caudal traction tension in both structures and contribute to the increase in peripheral-airway resistance.[14] Finally, we did not use body plethysmography, which is the gold standard for assessing lung volume when the subjects are seated in a sealed box; however, many of our patients were morbidly obese and were unable to access the box.

In conclusion, most adults with obesity have respiratory oscillometry abnormalities and abnormal signs on thoracic ultrasound, although only rarely do they have spirometric abnormalities. In these individuals, the greater the risk of developing OSA was, the worse the resistive and reactive parameters measured by respiratory oscillometry. In addition, abnormal respiratory oscillometry and abnormal thoracic ultrasound signals are factors associated with a high risk of developing OSA, even in patients with normal spirometry. To strengthen and validate our findings, longitudinal studies should employ nocturnal polysomnography to diagnose sleep disorders and body plethysmography to measure static lung volume in a larger sample of patients with obesity.

Ethical Approval of Studies/Informed Consent

The current study was approved by the Research Ethics Committee of Hospital Federal de Bonsucesso under the number CAAE-65762122.3.0000.5253, and it was conducted in accordance with the ethical principles consistent with the Declaration of Helsinki and all participants signed an informed consent form.

Credit Authorship Contribution Statement

SFS: conceptualization, formal analysis, methodology, and writing – original draft. CES: conceptualization, methodology, validation, and writing – review & editing. IMPPF: methodology, validation, and writing – review & editing. WOP: methodology, validation, and writing – review & editing. HPSA: methodology, validation, and writing – review & editing. AJL: conceptualization, formal analysis, funding acquisition, methodology, supervision, and writing – original draft.

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Conflict of Interests

The authors have no conflict of interests to declare.

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