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Obstructive Sleep Apnea and Orofacial Myofunctional Profile in Adults: A Cross-Sectional Study

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Purpose: To examine the orofacial myofunctional profile and its determinants in adults with obstructive sleep apnea (OSA).

Patients and Methods: A sample of 229 patients with OSA who underwent polysomnographic monitoring from June 1 to July 31, 2023, were enrolled in the study. All participants completed questionnaires, scales, and physical measurements to obtain information on general demographics, orofacial myofunction, degree of snoring, neck circumference, waist circumference, BMI, and OSA characteristics.

Results: The proportions of participants with normal weight, overweight, and obesity were 24.5%, 44.1%, and 31.4%, respectively. Mild, moderate, and severe OSA patients accounted for 16.6%, 21.4%, and 62%, respectively. The median total orofacial myofunction score was 90. Larger neck circumference (r=−0.18, P=0.007), higher BMI (r=−0.216, P=0.001), and more severe OSA (r=−0.191, P=0.004) were associated with poorer orofacial myofunction. Age, obesity, diabetes, and RDI were significant predictors of total orofacial myofunction.

Conclusion: All OSA patients have varying degrees of orofacial myofunction insufficiency in this study, which is more pronounced in overweight and obese individuals. The evaluation and intervention of orofacial myofunction should be emphasized in patients with OSA who are older, overweight or obese, who have comorbidities of other metabolic disorders, and who have poor PSG indicators.

Keywords: OSA, sleep disorder, muscle functions, obesity, predictors

Introduction

Obstructive sleep apnea (OSA) is a common chronic clinical condition characterized by repeated collapse and obstruction of the upper airway during sleep,[1] leading to intermittent hypoxia and hypercapnia, which in turn causes a variety of cardiac, cerebral, pulmonary, and vascular complications.[2] The most commonly identified daytime consequence of OSA is sleepiness, which can lead to impaired quality of life, increased road traffic accidents, and a significant burden of healthcare and financial.[3,4] Fundamentally, OSA is characterized by the interaction between impaired pharyngeal anatomy and insufficient upper airway dilator muscle function during sleep.[5] Beyond this, the low arousal threshold and high loop gain of anatomically impaired structures also play a crucial role in determining whether OSA occurs and its severity.[5–8] However, the expression of these pathophysiological features exhibits considerable variability among individuals.[9] In contrast, all patients with OSA present with varying degrees of compromised upper airway anatomy, with nearly 70% of these individuals displaying a combination of the remaining three features.[5] Damage to one or more of these non-anatomical characteristics can contribute to the development of OSA and influence its severity. Conversely, a favorable non-anatomical phenotype may confer protection against OSA in individuals with fragile upper airway anatomy.[10] Research indicates that pharyngeal dilator responses are significantly impaired during sleep in approximately 30% of patients with OSA,[6] with the primary muscles closely associated with the respiratory process being the chin, tongue, palate, and suspensory muscles. Previous studies[11–13] have summarized the alterations in upper airway muscle function that contribute to OSA. These changes include an increased proportion of glycolytic fast muscle type II fibers,

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which generate greater muscle force compared to oxidative slow muscle type I fibers, albeit with a higher susceptibility to fatigue. Additionally, there is dysfunction in the mechanical coupling of upper airway muscles, which diminishes the impact of tissue displacement during dilator muscle contraction on upper airway patency. Furthermore, impairment of muscle contractile function arises from sensory and motor denervation of the upper airway dilator muscles.

Obesity is the commonest and most recognized risk factor for OSA, and certain patients with impaired pharyngeal anatomy due to obesity may be protected from OSA by enhancing upper airway dilator muscle responsiveness during sleep,[14] and yet the mechanisms behind this relationship are not fully understood. Specific patterns of fat distribution and types of fat may be the pathophysiologic basis for this relationship.[15–17] Increased localized fat deposition around the pharynx and surrounding soft tissues (reflected in a larger neck circumference) compromises airway space, resulting in a narrower and more collapsible airway. Fat deposition around the waist (reflected in a larger waist circumference) leads to a decrease in lung capacity, which reduces caudal traction on the pharynx, thus increasing pharyngeal collapsibility. A 10% weight gain confers a sixfold increased risk of developing OSA and is associated with an approximately 32% elevation in the apnea hypopnea index (AHI).[18] Similarly, a higher BMI is associated with increased neck and waist circumference, a greater prevalence of OSA, poorer sleep quality, and impaired orofacial myofunctional status.[19] Moderate weight reduction effectively mitigates existing OSA and reduces incident OSA risk.

Other OSA risk factors also decrease upper airway neuromuscular function. The repeated vibration of the pharyngeal and palatal regions caused by prolonged snoring in patients with OSA can result in damage to the upper airway muscles and nerves.[20] The prevalence of OSA in adults increases with age, with primary causes including[21,22] increased parapharyngeal fat deposition, alterations in muscle activity that diminish the pharyngeal dilator muscle's responsiveness to chemical and mechanical stimuli during sleep, and anatomical changes such as elongation of the soft palate, narrowing of the pharyngeal lumen due to thickening of the pharyngeal fat pads, and modifications in the shape of the bony structures surrounding the pharyngeal airway. The risk of OSA in patients with diabetes is three to four times higher than in the non-diabetic population.[23] Contributing factors include[24] the frequent association with obesity, which leads to excessive fat distribution that narrows the upper airway and thickens the neck. Long-term blood glucose abnormalities result in microvascular, peripheral, and central neuropathy, causing decreased muscle tone and dysfunction of the upper airway, thereby impairing the respiratory center. Hypertension over-activates the sympathetic nervous system, resulting in abnormal neuromodulation of the respiratory muscles, which weakens their responsiveness and coordination, increasing the risk of airway collapse.[25] Dyslipidemia may induce a systemic low-grade inflammatory state characterized by fat deposits in and around upper respiratory tract tissues, adversely affecting muscle function.[26] Additionally, elevated levels of cholesterol and other lipids can increase oxidative stress, impairing cell membrane and mitochondrial function, which in turn weakens muscle strength.[27] Orofacial myofunctional therapy (OMT) has emerged as a treatment approach for upper airway collapse. Its method consists of repeated isometric and isotonic workouts that improve upper airway muscular tone, endurance, and coordination, hence lowering sleep-related airway collapse and clinical symptoms.[28] Clinical trials comparing OMT therapies across various OSA severity levels and treatment regimens show reductions in snoring, subjective/objective tiredness, and AHI, as well as increased lowest arterial oxygen saturation and improved sleep-related quality of life.[29–31]

The existing theoretical basics consist of the following components: (1) Insufficient function of upper airway dilator muscles is a notable pathophysiological characteristic of OSA, and OMT has been shown to effectively manage this condition. (2) Patients with both OSA and overweight or obesity exhibit a more severe disease profile compared to those with OSA alone. This severity is linked to orofacial neuromuscular dysfunction and fat tissue infiltration.[32] Identified research gaps include: (1) The anticipated rise in the prevalence of overweight and obese populations will likely lead to an increased incidence of OSA. Adequate pharyngeal muscle function is crucial for mitigating or eliminating OSA; however, the orofacial myofunctional characteristics and influencing factors in OSA patients remain inadequately understood. (2) Prior investigations have indicated that age, body mass index (BMI), and comorbidities serve as risk factors for OSA, potentially resulting in damage to upper airway muscles, particularly among individuals with severe OSA. Nevertheless, a standardized method for assessing such muscular damage is lacking. Thus, the study aims to employ a standardized evaluation tool to investigate the orofacial myofunctional characteristics and influencing factors in OSA patients, providing valuable insights for disease prevention and targeted intervention. We hypothesize that orofacial

myofunction in patients with OSA is associated with disease severity and obesity, with both inter- and intra-group differences. Furthermore, these differences may be moderated by factors such as age, gender, and comorbidities.

Methods

Study Population

From June 1 to July 31, 2023, the study enrolled OSA patients from the Department of Otorhinolaryngology and Respiratory at the First Affiliated Hospital of Chongqing Medical University using a convenience sampling method. Written informed consent was obtained from all participants. All methods were performed in accordance with the Declaration of Helsinki.

Inclusion criteria: (a) meet the diagnostic criteria of the Multidisciplinary Diagnostic and Treatment Guidelines for Obstructive Sleep Apnea in Adults (2018 version) developed by the Sleep Medicine Specialty Committee of the Chinese Physicians' Association;[33] (b) age ≥18 years old; (c) did not receive any therapeutic measures related to OSA; (d) have a good ability to communicate and understand, and voluntarily participate in this study. Exclusion criteria: (a) patients with severe anatomical structure damage, such as craniofacial deformity; (b) patients with combined severe cardiopulmonary diseases, malignant tumors, neuromuscular diseases, hypothyroidism, gastroesophageal reflux or suffering from other serious organ diseases; (c) patients with long-term heavy alcohol consumption, taking sedative-hypnotic or muscle relaxant drugs; (d) patients with psychiatric diseases or cognitive dysfunction. The primary outcome measure is orofacial myofunction, with secondary outcome measures including disease severity and obesity levels. This study has been approved by the Ethics Committee of Chongqing Medical University (review approval number: 2022107) and registered on https://www.chictr.org.cn/ (registration number: ChiCTR2200066741).

Polysomnographic Examination

All participants completed a full night's sleep using the SOMNO JFD5001 bedside PSG monitoring system (SOMNO medics GmbH, Germany), from 10 PM to 7 AM the following day, for a total of 9 hours. The monitoring Included electrocardiogram, electroencephalogram, electrooculogram, airflow, oxygen saturation (measured by a fingertip pulse oximeter), chest and abdominal respiratory movements, snoring, and body posture during sleep. All digitized signals were saved on computers and analyzed by a specialized sleep respiratory physician. Indicators included apnea hypopnea index (AHI), lowest arterial oxygen saturation ($LSaO_2$), mean arterial oxygen saturation ($MSaO_2$), respiratory disturbance index (RDI), oxygen desaturation index (ODI), the percentage of total recorded time with oxygen saturation level <90% (TS90%) and longest apnea time (LAT). The definition of indicators follows the guidelines for the diagnosis and treatment of OSA.[34–36] AHI was defined as the sum of the average number of apneas and hypoventilation per hour. Apnea was defined as the loss or marked reduction (≥90% decrease from baseline amplitude) of oronasal respiratory airflow for ≥10s. Hypopnea was defined as a decrease in oronasal airflow of ≥30% from the baseline level accompanied by a decrease in arterial oxygen saturation (SaO_2) of ≥4%for a duration of ≥10s. AHI was used as the main judgment criterion for disease grading: mild OSA, AHI of 5 or ≤15; moderate OSA, AHI >15 and ≤30; severe OSA, AHI >30. The RDI was the sum of the average number of apnea, hypopnea, and respiratory effort-related microarousal events per hour. The ODI was defined as the number of SaO_2 decreases of ≥3% per hour during sleep, reflecting the number of desaturation-related respiratory events per hour.

Orofacial Myofunctional Evaluation

The Orofacial Myofunctional Evaluation Scale (OMES), which has demonstrated good reliability and validity, was used.[37,38] The assessment includes the evaluation of orofacial soft and hard structures, mobility, and functions, showing a Cronbach's α coefficient of 0.852 in a previous study.[39] It was conducted by an investigator trained in systematic anatomy with five years of outpatient otolaryngology experience, alongside an otolaryngologist (MD). The methodology adhered to the examination criteria of the OMES protocol[38] and defined normal and dysfunctional assessments of posture, mobility, and function for each area. Disagreements were resolved by consensus, without knowledge of the participants. The scale comprises three main dimensions: appearance and posture, mobility, and functions, with a total of

37 entries, including lip appearance and posture, vertical mandibular posture, checks appearance and posture, face appearance, resting tongue position condition, palatal appearance and posture, movement of lips, tongue, jaws, and buccal region, as well as respiratory function, swallowing function (including lip movements during swallowing, tongue movements, other behavioral changes, and swallowing efficiency), and masticatory function (focusing on chewing habits and other behavioral changes during chewing). Chewing and swallowing assessments were conducted using small biscuits and water, uniformly provided by the researcher. All entries were scored in three general categories: 3 = normal, 2 = inadequate ability, and 1 = lack of ability or inability to perform. The total score for the OMES was 100, with smaller scores indicating a greater degree of abnormalities in orofacial myofunction. The Cronbach's α coefficient in this study was 0.854.

Other Covariates

Participants' gender, age, comorbidities based on the physician's diagnosis, smoking history, alcohol consumption history, educational levels, marital status, family history, and degree of snoring were collected through a self-administered questionnaire. Height, weight, neck circumference (NC), and waist circumference (WC) were measured twice by the same person using the same calibrated hospital equipment, and the average of the two measurements was taken. Body mass index (BMI) is calculated as weight divided by the square of height $\left(kg/m^2\right)$. Normal is defined as a BMI of 18.5–23.9 kg/m^2, obesity is defined as a BMI ≥28 kg/m^2, and overweight is defined as a BMI ≥24 kg/m^2.[40] Snoring data were collected through self-reports from participants and/or reports from family members. According to the guideline,[34] snoring is categorized into three levels: mild snoring (snoring sounds thicker than normal breathing sounds), moderate snoring (snoring that is louder than normal human speech), and severe snoring (snoring so loud that people in the same room cannot sleep).

Statistical Analysis

All data were analyzed with Statistical Product and Service Solutions (SPSS) software (version 25.0, IBM Corporation, Armonk, NY, USA). The Shapiro–Wilk test was employed to estimate the normality of data distribution. Participants were categorized into three groups: mild, moderate, and severe OSA. Their characteristics were provided by subgroup, using the median with interquartile range (IQR) or as a percentage of individuals for description. Kruskal–Wallis *H*-tests or Mann–Whitney test for continuous variables and Chi-Square test for categorical variables were used to make compare differences between groups. When comparing more than two groups, pairwise comparisons were conducted, and the significance level was adjusted using the Bonferroni correction method to control for Type I errors. Spearman correlation analysis was used to assess the correlation between variables, and multiple regression was performed for linear correlation analysis among multiple independent variables. All tests were two-sided, with a significance level of 0.05.

Results

Total Analysis of Participants

A total of 259 individuals were surveyed, from which 28 were excluded due to non-cooperation that led to incomplete data collection, 1 was excluded for having hypothyroidism, and another 1 for experiencing facial muscle spasm. Ultimately, 229 participants completed all survey procedures, yielding a response rate of 88.4%. Among the participants, 75 (32.7%) had attained a high school education or lower, while 154 (67.3%) held a college degree or higher. Furthermore, 176 (76.9%) were married, whereas 53 (23.1%) were unmarried, divorced, or widowed. As shown in Table 1, 183 (79.9%) were male and 46 (20.1%) were female, with a median (IQR) age of 41.0 years (34.0–52.0 years). By disease severity, there were 38 participants with mild OSA, 49 with moderate OSA, and 142 with severe OSA, with more than half (53.9%) of the participants having a family history of OSA. Compared with mild and moderate patients, those with severe OSA had a higher proportion of males (85.9%), comorbid hypertension (28.2%), as well as rates of overweight and obesity (83.8%). More than half were current smokers (52.8%) and demonstrated greater NC (39.0 cm [37.0–40.0 cm]) and WC (96.0 cm [89.6–101.0 cm]). The median scores (IQR) for AHI, $MSaO_2$, $LSaO_2$, RDI, ODI, TS90%, and LAT were 38.9 (21.0–60.8), 95.0 (94.0–96.0), 81.0 (71.0–87.0), 38.9 (21.0–60.8), 30.6 (12.2–58.1),1.60

Table 1 Participants' Characteristics, Stratified by Severity of OSA

Characteristics	Overall	Mild OSA	Moderate OSA	Severe OSA	H value [e]	P value [a]
No. of participants	229	38	49	142		
Age, (median [IQR]), y	41.0 [34.0, 52.0]	35.0 [29.0, 49.3]	41.0 [35.0, 48.0]	41.5 [35.3, 52.0] [A]	7.97	0.019[d]
Gender, No. (%)						
Male	183 (79.9)	24 (63.2)	37 (75.5)	122 (85.9)	10.42	0.005[c]
Female	46 (20.1)	14 (36.8)	12 (24.5)	20 (14.1)		
Family history of OSA, No. (%)	123 (53.9)	25 (65.8)	25 (51.0)	73 (51.8)	2.58	0.275
NC, (median [IQR]), cm	38.0 [36.0, 40.0]	35.5 [33.0, 37.8]	38.0 [36.0, 39.0]	39.0 [37.0, 40.0] [A]	21.85	<0.001[b]
WC, (median [IQR]), cm	93.5 [87.0, 99.0]	87.0 [83.6, 93.8]	92.0 [85.0, 97.0]	96.0 [89.6, 101.0] [AB]	21.86	<0.001[b]
BMI, No. (%), kg/m^2						
Normal (18.5≤BMI<24)	56 (24.5)	18 (47.4)	15 (30.6)	23 (16.2)	19.10	0.001[c]
Overweight (24≤BMI<28)	101 (44.1)	14 (36.8)	22 (44.9)	65 (45.8)		
Obese (BMI≥28)	72 (31.4)	6 (15.8)	12 (24.5)	54 (38.0)		
Current smoker, No. (%)	101 (44.1)	12 (31.6)	14 (28.6)	75 (52.8)	11.59	0.003[c]
Current alcohol use, No. (%)	95 (41.5)	12 (31.6)	20 (40.8)	63 (44.4)	2.03	0.362
Comorbidities, No. (%)						
Hypertension	52 (22.7)	6 (15.8)	6 (12.2)	40 (28.2)	0.31	0.039[d]
Diabetes	19 (8.3)	2 (5.3)	3 (6.1)	14 (9.9)	–	0.543
CHD	10 (4.4)	2 (5.3)	1 (2.0)	7 (4.9)	–	0.665
Hyperlipidemia	43 (18.8)	6 (15.8)	10 (20.4)	27 (19.0)	6.51	0.855
PSG characteristics, (median [IQR])						
AHI, events/h	38.9 [21.0, 60.8]	9.3 [7.0, 11.4] [B]	23.0 [18.8, 26.2]	54.7 [41.1, 75.4] [AB]	167.74	<0.001[b]
$MSaO_2$, %	95.0 [94.0, 96.0]	96.0 [96.0, 97.0]	96.0 [95.0, 97.0]	94.0 [92.0, 95.0] [AB]	68.85	<0.001[b]
$LSaO_2$, %	81.0 [71.0, 87.0]	90.0 [84.3, 91.8]	85.0 [82.0, 90.0]	76.0 [62.0, 82.0] [AB]	85.03	<0.001[b]
RDI, events/h	38.9 [21.0, 60.8]	9.2 [7.0, 11.1] [B]	23.0 [18.8, 26.2]	54.4 [41.1, 75.4] [AB]	167.74	<0.001[b]
ODI, events/h	30.6 [12.2, 58.1]	6.0 [3.1, 10.3] [B]	13.9 [8.8, 21.8]	50.6 [32.0, 69.2] [AB]	144.06	<0.001[b]
TS90%, %	1.6 [0.1, 11.6]	0.0 [0.0, 0.2]	0.2 [0.0, 1.0]	8.2 [1.3, 22.0] [AB]	97.97	<0.001[b]
LAT, s	50.3 [28.5, 77.0]	23.5 [19.1, 45.9]	36.0 [22.0, 54.0]	60.8 [43.3, 81.0] [AB]	45.90	<0.001[b]
OMES, (median [IQR]), score						
Total	90.0[87.0, 93.0]	92.0 [90.0, 95.0]	91.0 [88.0, 94.0]	90.0 [86.0, 92.0] [A]	12.85	0.002[c]
Appearance and posture	15.0 [14.0, 16.0]	16.0 [15.0, 17.0]	16.0 [15.0, 16.0]	15.0 [14.0, 16.0] [A]	7.94	0.019[d]
Mobility	54.0 [53.0, 56.0]	55.0 [54.0, 56.0]	55.0 [53.0, 56.0]	54.0 [52.0, 56.0] [A]	7.93	0.019[d]
Functions	21.0 [20.0, 22.0]	21.0 [20.0, 22.0]	21.0 [19.0, 22.0]	21.0 [19.3, 22.0]	1.86	0.395

(*Continued*)

Table 1 (Continued).

Characteristics	Overall	Mild OSA	Moderate OSA	Severe OSA	H value [e]	P value [a]
Degree of snoring, No. (%)						
No snoring	4 (1.7)	2 (5.3)	2 (4.1)	0 (0.0)	–	<0.001[b]
Mild snoring	26 (11.4)	12 (31.6)	5 (10.2)	9 (6.3)		
Moderate snoring	75 (32.8)	12 (31.6)	21 (42.9)	42 (29.6)		
Severe snoring	124 (54.1)	12 (31.6)	21 (42.9)	91 (64.1)		

Notes: A is a statistically significant difference between this group and group 1 (Mild OSA), and B is a statistically significant difference between this group and group 2 (Moderate OSA). In intergroup comparisons, the Bonferroni correction was applied to adjust the significance level, setting the corrected threshold for each comparison at α/m, where m denotes the number of comparisons and α=0.05. [a] P value calculated using Kruskal–Wallis *H*-tests or Chi-Square test. [e] H value calculated using Kruskal–Wallis *H*-tests. [b] P<0.001; [c] P<0.01; [d] P<0.05.

Abbreviations: OSA, obstructive sleep apnea; NC, Neck circumference; WC, Waist circumference; BMI, Body mass index; CHD, Coronary heart disease; PSG, Polysomnography; AHI, apnea hypopnea index; $MSaO_2$, mean arterial oxygen saturation; $LSaO_2$, lowest arterial oxygen saturation; RDI, respiratory disturbance index; ODI, oxygen desaturation index; TS90%, the percentage of total recorded time with oxygen saturation level <90%; LAT, longest apnea time; OMES, orofacial myofunctional evaluation scale; IQR, interquartile range.

(0.1–11.6), and 50.3 (28.5–77.0), respectively, demonstrating significant variability among patients with OSA of different severity (P<0.001). The evaluation of orofacial myofunction consisted of three dimensions, demonstrating a very high internal consistency, with an Intraclass Correlation Coefficient (ICC) of 0.990 (95% CI: [0.987, 0.992]). The median scores (IQR) for "appearance and posture" were 15.0 (14.0–16.0), for "mobility" were 54.0 (53.0–56.0), for "functions" were 21.0 (20.0–22.0), and the overall median score (IQR) was 90.0 (87.0–93.0). The differences in "appearance and posture" score (P=0.019), "mobility" score (P=0.019), and total score (P=0.002) in the evaluation of orofacial myofunction were statistically significant among patients with differing severity of OSA. Patients with severe OSA exhibited more severe snoring (P<0.001).

Correlations between Participants' Orofacial Myofunction and Demographic, Anthropometric, and PSG Characteristics

As shown in Table 2, the appearance and posture category in orofacial myofunction exhibited significant negative correlations with WC, BMI, AHI, RDI, ODI, TS90%, and OSA severity variables, as well as significant positive

Table 2 The Spearman Correlation Between Orofacial Myofunction and Collection Variables

	OMES (Appearance and Posture)	OMES (Mobility)	OMES (Functions)	OMES (Total)
Age		r = −0.349(0.000[a])		r = −0.281(0.000[a])
WC	r = −0.207(0.002[b])			r = −0.18(0.007[b])
BMI	r = −0.219(0.001[b])			r = −0.216(0.001[b])
Hypertension		r = −0.174(0.008[b])		r = −0.171(0.010[c])
Diabetes		r = −0.179(0.007[b])	r = −0.133(0.045[b])	r = −0.175(0.008[b])
CHD		r = −0.139(0.035[c])		
AHI	r = −0.196(0.003[b])			r = −0.185(0.005[b])
$LSaO_2$	r = 0.204(0.002[b])	r = 0.164(0.013[c])		r = 0.21(0.001[b])
$MSaO_2$	r = 0.174(0.008[b])			r = 0.157(0.018[c])

(*Continued*)

Table 2 (Continued).

	OMES (Appearance and Posture)	OMES (Mobility)	OMES (Functions)	OMES (Total)
RDI	r = −0.198(0.003[b])			r = −0.187(0.005[b])
ODI	r = −0.218(0.001[b])	r = −0.148(0.025[c])		r = −0.202(0.002[b])
TS90%	r = −0.196(0.003[b])	r = −0.18(0.006[b])		r = −0.228(0.001[b])
OSA severity	r = −0.15(0.023[c])	r = −0.157(0.017[c])		r = −0.191(0.004[b])

Notes: P value calculated using Spearman correlation analysis. [a] $p < 0.001$; [b] $p < 0.01$; [c] $p < 0.05$.
Abbreviations: OMES, orofacial myofunctional evaluation scale; WC, Waist circumference; BMI, Body mass index; CHD, Coronary heart disease; AHI, apnea hypopnea index; $LSaO_2$, lowest arterial oxygen saturation; $MSaO_2$, mean arterial oxygen saturation; RDI, respiratory disturbance index; ODI, oxygen desaturation index; TS90%, the percentage of total recorded time with oxygen saturation level <90%; OSA, obstructive sleep apnea.

correlations with the $LSaO_2$ and $MSaO_2$ variables. The "mobility" category showed significant negative correlations with age, hypertension, diabetes, CHD, ODI, TS90%, and OSA severity variables, and significant positive correlations with $LSaO_2$. The "functions" category was only significantly negatively correlated with diabetes; that is, the worse the patient's function of breathing, chewing and swallowing, the higher the risk of comorbid diabetes (r=−0.133, P=0.045). Total orofacial myofunction presented significant negative correlations with age, WC, BMI, hypertension, diabetes, AHI, RDI, ODI, TS90%, and OSA severity, along with significant positive correlations with the $LSaO_2$ and $MSaO_2$ variables. These variables were recognized as potential independent predictors of orofacial muscle function.

Predictors of Orofacial Myofunction

Indicators with significance in correlation analysis, as shown in Table 2, were integrated into the multiple stepwise linear regression analysis. The results were shown in Table 3. To ensure the robustness of the regression analysis,

Table 3 Predicting Orofacial Myofunction in Patients with OSA, Multiple Stepwise Regression Analysis

Model	R^2	Predictors	B	Beta	[95% CI]	P value [a]	VIF
OMES (total)	0.187	Constant	96.573		[94.127, 99.018]	0.000[b]	
		Age	−0.115	−0.264	[−0.167, −0.063]	0.000[b]	1.021
		Obese	−1.918	−0.173	[−3.310, −0.525]	0.007[c]	1.115
		Diabetes	−3.103	−0.166	[−5.365, −0.841]	0.007[c]	1.038
		RDI	−0.024	−0.131	[−0.047, −0.001]	0.041[d]	1.119
OMES (appearance and posture)	0.066	Constant	13.526		[12.571, 14.482]	0.000[b]	
		$LSaO_2$	0.025	0.257	[0.013, 0.037]	0.000[b]	1.000
OMES (mobility)	0.149	Constant	58.484		[56.632, 60.337]	0.000[b]	
		Age	−0.093	−0.289	[−0.133, −0.054]	0.000[b]	1.021
		Diabetes	−2.195	−0.158	[−3.907, −0.483]	0.012[d]	1.033
		RDI	−0.019	−0.138	[−0.035, −0.002]	0.027[d]	1.014
OMES (functions)	0.071	Constant	21.187		[20.884, 21.490]	0.000[b]	
		Obese	−0.877	−0.209	[−1.410, −0.344]	0.001[c]	1.011
		Diabetes	−1.035	−0.147	[−1.931, −0.138]	0.024[d]	1.011

Notes: [a] P value calculated using multiple stepwise regression analysis. [b] $p < 0.001$; [c] $p < 0.01$; [d] $p < 0.05$.
Abbreviations: RDI, respiratory disturbance index; $LSaO_2$lowest arterial oxygen saturation; CI, confidence interval; OMES, orofacial myofunctional evaluation scale; VIF, Variance Inflation Factor.

multicollinearity among independent variables was assessed across all models. The results indicated that all variance inflation factors (VIFs) were below 5, suggesting no significant multicollinearity. Age, obesity, diabetes, and RDI were identified as independent predictors in the OMES (total) prediction model (R^2= 18.7%, P<0.05). $LSaO_2$ was an independent predictor in the OMES (appearance and posture) prediction model (R^2=6.6%, P<0.05). Age, diabetes, and RDI were independent predictors in the OMES (mobility) prediction model (R^2=14.9%, P<0.05). Obesity and diabetes were independent predictors in the OMES (functions) prediction model (R^2=7.1%, P<0.05). In Models 1 and 3, the effect of age is more pronounced, with OMES (total) and OMES (mobility) expected to decrease by 0.264 and 0.289 standard deviations, respectively, for each standard deviation increase in age. In Model 4, the effect of BMI is even greater, with OMES (functions) anticipated to decrease by 0.209 standard deviations for each standard deviation increase.

Effect of BMI on PSG Characteristics and Orofacial Myofunction

Regarding the PSG characteristics, AHI, RDI, ODI, and TS90% were significantly higher in the obese group compared with the overweight and normal weight groups (P<0.001), while $MSaO_2$ and $LSaO_2$ were lower (P<0.001), as shown in Table 4. In terms of orofacial myofunction, a statistically significant difference was observed between the obese and

Table 4 Comparison of PSG Characteristics, Orofacial Myofunction, Stratified by BMI

Characteristics	Overall	Normal	Overweight	Obese	H[e]	P[a]
Age, (median [IQR]), y	41.0 [34.0, 52.0]	38.5 [30.8, 50.5]	43.0 [35.0, 51.0]	40.5 [34.0, 53.3]	2.30	0.317
Gender, No. (%)					7.53	0.023[d]
Male	183 (79.9)	38 (67.9)	87 (86.1)	58 (80.6)		
Female	46 (20.1)	18 (32.1)	14 (13.9)	14 (19.4)		
Comorbidities, No. (%)						
Hypertension	52 (22.7)	5 (8.9)	19 (18.8)	28 (38.9)	2.55	<0.001[b]
Diabetes	19 (8.3)	4 (7.1)	6 (5.9)	9 (12.5)	2.51	0.286
CHD	10 (4.4)	0 (0.0)	3 (3.0)	7 (9.7)	–	0.019[d]
Hyperlipidemia	43 (18.8)	7 (12.5)	19 (18.8)	17 (23.6)	17.67	0.279
NC, (median [IQR]), cm	38.0 [36.0, 40.0]	35.8 [32.8, 37.0] [B]	38.0 [36.0, 39.0]	39.5 [38.0, 42.0] [AB]	49.37	<0.001[b]
WC, (median [IQR]), cm	93.5 [87.0, 99.0]	84.0 [78.0, 88.0] [B]	93.5 [89.0, 97.0]	100.0 [96.0, 106.0] [AB]	100.87	<0.001[b]
PSG characteristics, (median [IQR])						
AHI, events/h	38.9 [21.0, 60.8]	27.1 [12.7, 45.6] [B]	38.7 [21.8, 56.6]	53.8 [30.1, 79.0] [AB]	21.71	<0.001[b]
$MSaO_2$, %	95.0 [94.0, 96.0]	96.0 [95.0, 97.0] [B]	95.0 [94.0, 96.0]	94.0 [92.0, 96.0] [AB]	26.45	<0.001[b]
$LSaO_2$, %	81.0 [71.0, 87.0]	86.5 [79.0, 90.3] [B]	82.0 [74.0, 86.0]	76.0 [61.5, 82.0] [AB]	30.69	<0.001[b]
RDI, events/h	38.9 [21.0, 60.8]	27.1 [12.6, 45.6]	38.7 [21.8, 56.6]	53.3 [30.1, 79.0] [AB]	21.59	<0.001[b]
ODI, events/h	30.6 [12.2, 58.1]	12.9 [7.0, 33.8] [B]	31.7 [13.9, 55.6]	42.9 [20.1, 71.5] [AB]	28.93	<0.001[b]
TS90%, %	1.6 [0.1, 11.6]	0.1 [0.0, 4.3] [B]	1.4 [0.2, 10.8]	5.4 [0.8, 28.7] [AB]	31.56	<0.001[b]
LAT, s	50.3 [28.5, 77.0]	46.5 [25.6, 81.1]	52.0 [36.0, 77.0]	49.5 [28.0, 74.1]	0.53	0.767
OMES, (median [IQR]), score						
Total	90.0 [87.0, 93.0]	91.0 [88.8, 94.0]	91.0 [88.0, 93.0]	89.0 [85.0, 91.3] [AB]	14.51	0.001[c]
Appearance and Posture	15.0 [14.0, 16.0]	16.0 [15.0, 17.0]	15.0 [14.0, 16.0]	15.0 [14.0, 16.0] [A]	9.59	0.008[c]

(*Continued*)

Table 4 (Continued).

Characteristics	Overall	Normal	Overweight	Obese	H[e]	P[a]
Mobility	54.0 [53.0, 56.0]	54.0 [53.0, 56.0]	55.0 [53.0, 56.0]	54.0 [51.0, 55.3] [B]	6.28	0.043[d]
Functions	21.0 [20.0, 22.0]	21.0 [20.0, 22.0]	21.0 [20.0, 22.0]	20.0 [19.0, 22.0] [B]	9.92	0.007[c]

Notes: A is a statistically significant difference between this group and group 1 (Normal), and B is a statistically significant difference between this group and group 2 (Overweight). In intergroup comparisons, the Bonferroni correction was applied to adjust the significance level, setting the corrected threshold for each comparison at α/m, where m denotes the number of comparisons and α=0.05. [a] P value calculated using Kruskal–Wallis *H*-tests or Chi-Square test. [e] H value calculated using Kruskal–Wallis *H*-tests. [b] P<0.001; [c] P<0.01; [d] P<0.05.

Abbreviations: BMI, Body mass index; CHD, Coronary heart disease; NC, Neck circumference; WC, Waist circumference; PSG, Polysomnography; AHI, apnea hypopnea index; $MSaO_2$, mean arterial oxygen saturation; $LSaO_2$, lowest arterial oxygen saturation; RDI, respiratory disturbance index; ODI, oxygen desaturation index; TS90%, the percentage of total recorded time with oxygen saturation level <90%; LAT, longest apnea time; OMES, orofacial myofunctional evaluation scale; IQR, interquartile range.

overweight groups in the total score, including the categories of "mobility" and "functions", with the obese group scoring lower. Additionally, the difference between the obese group and the normal BMI group was statistically significant for the total score and the "appearance and posture" category, with the normal BMI group achieving higher scores. The obese group was more prone to suffer from OSA comorbidities such as hypertension and CHD (P<0.05). NC and WC of OSA patients showed significant variability among different BMI groups (P<0.001).

Effect of Gender on Orofacial Myofunction in OSA Patients

As presented in Table 5, female patients exhibited notably diminished scores (P<0.05) in the "appearance and posture" category for mandible entries (2 [2,3]), and in the "functions" category for mastication entries (5 [4,5]) when compared to

Table 5 Comparison of Orofacial Myofunction in OSA Patients, Stratified by Gender

OMES	Maximum Scores	Female (n=46)	Male (n=183)	Z	P[a]
		Median [IQR]	Median [IQR]		
Appearance and posture	18	16 [14.75,16.25]	15 [14,16]	−0.496	0.620
Lips	3	3 [3,3]	3 [3,3]	−0.487	0.626
Mandible	3	2 [2,3]	3 [2,3]	−2.031	0.042[b]
Cheek	3	3 [2,3]	3 [2,3]	−0.506	0.613
Face	3	3 [3,3]	3 [2,3]	−1.231	0.218
Tongue	3	2 [2,3]	2 [2,3]	−0.758	0.448
Palate	3	2 [2,3]	2 [2,3]	−1.457	0.145
Mobility	57	55 [52.75,56]	54 [53,56]	−0.942	0.346
Lips	12	12 [10,12]	12 [10,12]	−0.906	0.365
Tongue	18	17 [16,18]	17 [16,18]	−0.235	0.814
Mandible	15	15 [14,15]	15 [14,15]	−0.006	0.995
Cheek	12	12 [11,12]	12 [11,12]	−0.231	0.817
Functions	25	20.5 [19,22]	21 [20,22]	−1.107	0.268
Breathing	3	2 [2,2]	2 [2,2]	−0.522	0.601

(*Continued*)

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Table 5 (Continued).

OMES	Maximum Scores	Female (n=46)	Male (n=183)	Z	P[a]
		Median [IQR]	Median [IQR]		
Deglutition	15	8 [7,9]	8 [7,9]	−0.212	0.832
Mastication	7	5 [4,5]	5 [4,7]	−1.971	0.049[b]
Total score	100	90 [87,94]	90 [87,93]	−0.090	0.928

Notes: [a] P value calculated using Mann–Whitney Test. [b] P<0.05.
Abbreviations: OSA, obstructive sleep apnea; OMES, orofacial myofunctional evaluation scale; IQR, interquartile range.

male OSA patients in terms of orofacial myofunction. At the gender level, no statistically significant differences were observed in the remaining dimensions and entries of orofacial myofunction. To further elucidate the causes of gender-based differences in orofacial myofunction, the effects of various confounding demographic, health-related, and disease-related variables were progressively controlled through multiple linear regression models, with results presented in Table 6. Multicollinearity among independent variables was assessed across all models, with all VIFs below 5, indicating no significant multicollinearity. In all examined models, there was an absence of a significant relationship between gender and orofacial myofunction, suggesting that gender is not a significant determinant of orofacial myofunctional impairments.

Table 6 Association Between Orofacial Myofunction and Gender in Multiple Linear Regression

Model	Variables	β	S. E	t	P[a]	β (95% CI)
Model 1	Intercept	89.93	0.38	235.22	<0.001	89.93 (89.18 ~ 90.68)
	Male					0.00 (Ref)
	Female	−0.71	0.85	−0.83	0.405	−0.71 (−2.38 ~ 0.96)
Model 2	Intercept	90.22	5.22	17.27	<0.001	90.22 (79.98 ~ 100.46)
	Male					0.00 (Ref)
	Female	−0.22	1.03	−0.21	0.831	−0.22 (−2.24 ~ 1.80)
Model 3	Intercept	94.86	9.11	10.42	<0.001	94.86 (77.01 ~ 112.71)
	Male					0.00 (Ref)
	Female	−0.29	1.53	−0.19	0.848	−0.29 (−3.29 ~ 2.70)
Model 4	Intercept	95.14	9.17	10.38	<0.001	95.14 (77.17 ~ 113.10)
	Male					0.00 (Ref)
	Female	−0.43	1.65	−0.26	0.793	−0.43 (−3.66 ~ 2.79)

Notes: [a] P values were calculated based on one-way linear regression analyses of sex-disaggregated OMES (total) by taking variables with P values <0.2 and incorporating them into a multifactorial linear regression model.Model 1: Crude model.Model 2: Control for demographic factors, including educational levels, marital status and age.Model 3: Increased control of health-related factors, including NC, MC, BMI and history of smoking, alcohol use and family history.Model 4: Increased control of disease-related factors, including hypertension, diabetes, CHD and hyperlipidemia.
Abbreviations: OMES, orofacial myofunctional evaluation scale; NC, Neck circumference; WC, Waist circumference; BMI, Body mass index; CHD, Coronary heart disease; β, Regression Coefficient; S.E, Standard Error; CI, Confidence Interval; Ref, Reference.

Discussion

To the best of our knowledge, with the rise of precision medicine, the focus of OSA treatment has gradually shifted from a one-size-fits-all approach, represented by continuous positive airway pressure ventilation, to individualized treatment based on pathophysiological characteristics.[41] As far as the pathophysiological phenotype of upper airway muscle insufficiency is concerned, more studies have examined the role of various therapies—such as nerve stimulation, orofacial myofunctional therapy,[42] pharmacological treatments, weight loss, and oral appliance[43]—in the improvement of health outcomes for patients and the objective indexes of PSG, rather than conducting cross-sectional investigations on the characterization of the phenotype and its relationship with OSA. Studies are rare in reporting the level of orofacial myofunction and its relationship to disease severity, obesity, and possible predictors in patients with OSA.

In this cross-sectional survey study, 229 participants had a median total orofacial myofunction score of 90, indicating the presence of orofacial myofunctional impairments. To elucidate the determinants of impact, an initial correlation analysis was performed, which showed that older age, larger NC, higher BMI, comorbid hypertension and diabetes, worse PSG characteristics (AHI, $LSaO_2$, $MSaO_2$, RDI, ODI, and TS90%) and more severe OSA were associated with worse orofacial myofunction.

The relationship between orofacial myofunction and the severity of OSA in patients was explored. In different severity groups of OSA, significant differences were observed in the scores of OMES across three categories: "appearance and posture" "mobility" and "functions" as well as in the total score. Specifically, compared to patients with mild OSA, those with severe OSA exhibited poorer orofacial myofunction and PSG indicators. A study by Attali et al suggested that the functional activity of the lingual muscles was impaired in non-obese or non-severely obese patients with moderate to severe OSA compared to those with non- or mild OSA.[44] This impairment may be attributed to the fact that upper airway obstruction during sleep in patients with moderate-to-severe OSA is primarily driven by anatomical structures.[5] Previous study has also confirmed that patients with severe OSA exhibit different muscle patterns compared to normal individuals.[45] Nighttime snoring is a common symptom among patients with OSA. In this study, only a small minority of OSA patients (1.7%) did not snore, while 64.1% of those with severe OSA experienced significant snoring. This phenomenon is partially associated with the occurrence of multisystem complications resulting from long-term severe snoring and the impairment of upper airway neuromuscular function.[46]

The relationship between orofacial myofunction and obesity in patients with OSA was explored. Higher BMI was associated with poorer "appearance and posture" category and total score, with no significant correlation with "mobility" and "functions" categories. Cheek appearance may serve as an immediately visible substitute for tongue and pharyngeal fat deposition, as well as muscle function in OSA screening.[37] The $LSaO_2$ has a significant influence on the "appearance and posture" category. Regression analyses showed that obesity is one of the significant influences in the "functions" category and total score, in line with Silva et al's study.[19] Moderately to severely obese patients with OSA exhibit reduced upper airway volume,[47] tongue enlargement,[48] and larger NC and WC. A prior study on the relationship between simple obesity and orofacial myofunction has also shown that moderate changes in orofacial mobility have been observed in obese individuals with a BMI $\geq 40\ kg/m^2$.[49] Investigating the non-OSA population also supported the differences in breathing, swallowing, and chewing functions between obese and non-obese individuals, as evidenced by lower scores on the functional dimensions.[49]

Older age and the presence of comorbidities are associated with poorer orofacial myofunction. Regression analyses showed that age was one of the most important factors influencing the total orofacial myofunction score and the "mobility" category. A survey of functionally independent older adults showed that 40% exhibited hypomobility of the oral maxillary system structure, and 23% to 34% of them were found to be deficient or lacking in function when deflecting air from the cheeks and jaws to the right or left.[50] The combination of hypertension, CHD, and diabetes was associated with worse scores in the "lip, tongue, mandible, and cheek mobility" category.

In previous studies, it has been consistently demonstrated that the prevalence of OSA is higher in men (24%) compared to women (9%).[51] It is essential to explore gender-based differences in orofacial myofunction. Numerous researchers have investigated this disparity, exploring factors such as variances in upper airway anatomy between males and females,[52,53] as well as differences in the respiratory neural control of upper airway dilator muscles.[54] In

general, females tend to require more time for chewing until the food bolus is swallowed and exhibit smaller bite sizes during mastication.[55] Conversely, males display larger facial dimensions, higher bite force, increased chewing frequency, and superior masticatory performance compared to females.[56] Our results demonstrated that females exhibited inferior performance in mandible entries within the "appearance and posture" category, as well as in mastication entries within the "functions" category. Notably, there are no significant gender differences in the breathing entries. The stability of breathing control, along with airway collapse, plays a critical role in maintaining upper airway patency during sleep.[57] On one hand, the respiratory centers in both sexes exhibit remarkably similar activation patterns. The respiratory activity of the genioglossus muscle is affected by reflex responses to negative airway pressure during inspiration, as well as by central activation independent of reflex mechanisms. Any gender-related differences in these mechanisms could potentially contribute to the higher prevalence of OSA observed in men.[58] Previous investigations have indicated no significant differences in diaphragm and genioglossus muscle activity between young healthy men and women during rest or in response to brief hypoxic stimuli while awake. Furthermore, the relationship between pharyngeal pressure and genioglossus activity appears consistent across genders, irrespective of hypoxic status.[59] This finding does not support the hypothesis that the elevated prevalence of OSA in men is attributable to potential gender differences in the respiratory neural control of upper airway dilator muscles.[54] Conversely, some studies suggest that significant gender differences in pharyngeal collapse are not driven by variations in muscle activation but rather by fundamental differences in upper airway anatomical structure and/or tissue characteristics between the sexes.[60] After adjusting for remaining confounders, including demographic, health-related, and disease-related factors, gender was found not to significantly predict the level of orofacial myofunction. This finding contrasts with the study by Kim et al,[61] potentially due to the significant disparity in sample sizes between males and females in our research, with only 46 females, which may have resulted in insufficient statistical power. Furthermore, gender may not be a key determinant of orofacial myofunction levels in this study.

Our study is one of the few to explore the relationship between phenotypic alterations and OSA based on pathophysiologic features, providing information for basic research related to OSA. In OSA screening, the OMES may be useful for prioritizing PSG diagnostics to facilitate risk stratification. Furthermore, the potential utility of OMES in OSA treatment should be expanded upon, particularly regarding its ability to predict outcomes of orofacial myofunctional therapy.

However, the study has several limitations, primarily due to the convenience sampling conducted at a single large tertiary hospital. Additionally, the significant disparity in the male-to-female ratio within the sample may reduce the external validity of the findings. Future research should employ a more rigorous sampling strategy to conduct multi-center, large-sample surveys. Moreover, the extent of snoring and daytime sleepiness was assessed by self-report from the OSA patients and their family members, meaning that the authenticity of the data relies heavily on the candor of patients and their trust in researchers. Furthermore, obesity is a significant contributor to OSA, which can, in turn, exacerbate obesity. Although our within-group two-by-two comparison based on BMI subgroups indicated that the normal BMI group outperformed the obese group in total OMES scores and "appearance and posture" scores, this analysis was limited to the OSA population. Future studies could strengthen these findings by including a non-OSA group as a control.

Conclusion

Orofacial myofunction insufficiency is present in all OSA patients in our study, albeit to varying degrees, with the condition being more severe in overweight and obese individuals. Higher BMI is associated with larger NC, WC, and more severe OSA, particularly in the presence of comorbidities such as hypertension and diabetes. In this case, the assessment and intervention of orofacial myofunction should be emphasized in patients with OSA who are older, overweight or obese, comorbid with other metabolic disorders, and have poor PSG indicators. The conclusions of this study were derived from the analysis of data obtained through a single-center cross-sectional survey. Future research could benefit from employing multi-center, large-sample panel data analyses to further investigate the characteristics of orofacial myofunction, influencing factors, and change trajectories in patients with OSA. Such approaches may elucidate

potential causal relationships and offer more robust theoretical guidance for precision medicine practices tailored to the pathophysiological characteristics of the disease.

Data Sharing Statement

The data that support the findings of this study are available from the corresponding author, S.X., upon reasonable request.

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Author Contributions

X.Y. and Y.W. contributed to data curation, formal analysis, and writing - original draft. J.C. and X.Y. conducted the investigation, validation, and writing—review & editing. S.X. was responsible for conceptualization, methodology, and writing—review & editing. All authors gave final approval of the version to be published; have agreed on the journal to which the article has been submitted; and agree to be accountable for all aspects of the work.

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Disclosure

The authors report no conflicts of interest in this work.

References

1. Pham LV, Schwartz AR. The pathogenesis of obstructive sleep apnea. *J Thorac Dis*. 2015;7(8):1358–1372. doi:10.3978/j.issn.2072-1439.2015.07.28
2. Gleeson M, McNicholas WT. Bidirectional relationships of comorbidity with obstructive sleep apnoea. *Eur Respir Rev*. 2022;31(164):210256. doi:10.1183/16000617.0256-2021
3. Lee SA, Paek JH, Han SH. Sleep hygiene and its association with daytime sleepiness, depressive symptoms, and quality of life in patients with mild obstructive sleep apnea. *J Neurol Sci*. 2015;359(1–2):445–449. doi:10.1016/j.jns.2015.10.017
4. Waldman LT, Parthasarathy S, Villa KF, et al. Understanding the burden of illness of excessive daytime sleepiness associated with obstructive sleep apnea: a qualitative study. *Health Qual Life Outcomes*. 2020;18(1):128. doi:10.1186/s12955-020-01382-4
5. Eckert DJ, White DP, Jordan AS, et al. defining phenotypic causes of obstructive sleep apnea. identification of novel therapeutic targets. *Am J Respir Crit Care Med*. 2013;188(8):996–1004. doi:10.1164/rccm.201303-0448OC
6. Osman AM, Altree TJ, Eckert DJ. The scent of love is in the air(way): a potential drug target for sleep apnea? *Sleep*. 2023;46(4):zsad019. doi:10.1093/sleep/zsad019
7. Edwards BA, Andara C, Landry S, et al. Upper-airway collapsibility and loop gain predict the response to oral appliance therapy in patients with obstructive sleep apnea. *Am J Respir Crit Care Med*. 2016;194(11):1413–1422. doi:10.1164/rccm.201601-0099OC
8. Bosi M, De Vito A, Kotecha B, et al. Phenotyping the pathophysiology of obstructive sleep apnea using polygraphy/polysomnography: a review of the literature. *Sleep Breath*. 2018;22(3):579–592. doi:10.1007/s11325-017-1613-3
9. Wellman A, Eckert DJ, Jordan AS, et al. A method for measuring and modeling the physiological traits causing obstructive sleep apnea. *J Appl Physiol*. 2011;110(6):1627–1637. doi:10.1152/japplphysiol.00972.2010
10. Eckert DJ. Phenotypic approaches to obstructive sleep apnoea – new pathways for targeted therapy. *Sleep Med Rev*. 2018;37:45–59. doi:10.1016/j.smrv.2016.12.003
11. Kimoff RJ. Upper airway myopathy is important in the pathophysiology of obstructive sleep apnea. *J Clin Sleep Med*. 2007;3(6):567–669. doi:10.5664/jcsm.26964
12. Mezzanotte WS, Tangel DJ, White DP. Influence of sleep onset on upper-airway muscle activity in apnea patients versus normal controls. *Am J Respir Crit Care Med*. 1996;153(6):1880–1887. doi:10.1164/ajrccm.153.6.8665050
13. Cori JM, O'Donoghue FJ, Jordan AS. Sleeping tongue: current perspectives of genioglossus control in healthy individuals and patients with obstructive sleep apnea. *Nat Sci Sleep*. 2018;10:169–179. doi:10.2147/NSS.S143296
14. Sands SA, Eckert DJ, Jordan AS, et al. Enhanced upper-airway muscle responsiveness is a distinct feature of overweight/obese individuals without sleep apnea. *Am J Respir Crit Care Med*. 2014;190(8):930–937. doi:10.1164/rccm.201404-0783OC
15. Sutherland K, w LRW, Cistulli PA. Obesity and craniofacial structure as risk factors for obstructive sleep apnoea: impact of ethnicity. *Respirology*. 2012;17(2):213–222. doi:10.1111/j.1440-1843.2011.02082.x

16. Schwartz AR, Patil SP, Laffan AM, et al. Obesity and obstructive sleep apnea: pathogenic mechanisms and therapeutic approaches. *Proc Am Thorac Soc*. 2008;5(2):185–192. doi:10.1513/pats.200708-137MG
17. Schwartz AR, Patil SP, Squier S, et al. Obesity and upper airway control during sleep. *J Appl Physiol*. 2010;108(2):430–435. doi:10.1152/japplphysiol.00919.2009
18. Peppard PE, Young T, Palta M, et al. Longitudinal study of moderate weight change and sleep-disordered breathing. *JAMA*. 2000;284(23):3015–3021. doi:10.1001/jama.284.23.3015
19. da Silva NC, da Silva GPJT, Onofri SMM, et al. Obstructive sleep apnea and orofacial myofunctional aspects in obesity. *Sleep Breath*. 2023;27:1351–1358. doi:10.1007/s11325-022-02738-4
20. Sowho M, Sgambati F, Guzman M, et al. Snoring: a source of noise pollution and sleep apnea predictor. *Sleep*. 2020;43(6):zsz305. doi:10.1093/sleep/zsz305
21. Zhang QJ, Zhang Y. Advances in the study of risk factors and different treatment modalities for OSAHS in adults. *Adv Clin Med*. 2023;13(7):10893–10903. doi:10.12677/ACM.2023.1371521
22. Redline S, Kirchner HL, Quan SF, et al. The effects of age, sex, ethnicity, and sleep-disordered breathing on sleep architecture. *Arch Int Med*. 2004;164(4):406–418. doi:10.1001/archinte.164.4.406
23. Hicks D. Obstructive sleep apnoea: its link with diabetes. *Nurs Times*. 2011;107(40):31–32.
24. Liu LY, Liu SF. Progress in the etiologic study of obstructive sleep apnea hypopnea syndrome. *J Mudanjiang Med College*. 2019;40(4):95–97.
25. Mochol J, Gawrys J, Gajecki D, et al. Cardiovascular disorders triggered by obstructive sleep apnea—a focus on endothelium and blood components. *Int J Mol Sci*. 2021;22(10):5139. doi:10.3390/ijms22105139
26. Mitra AK, Bhuiyan AR, Jones EA. Association and risk factors for obstructive sleep apnea and cardiovascular diseases: a systematic review. *Diseases*. 2021;9(4):88. doi:10.3390/diseases9040088
27. May AM, Mehra R. Obstructive sleep apnea: role of intermittent hypoxia and inflammation. *Semin Respir Crit Care Med*. 2014;35(5):531–544. doi:10.1055/s-0034-1390023
28. Guimarães KC, Drager LF, Genta PR, Marcondes BF, Lorenzi-Filho G. Effects of oropharyngeal exercises on patients with moderate obstructive sleep apnea syndrome. *Am J Respir Crit Care Med*. 2009;179(10):962–966. doi:10.1164/rccm.200806-981OC
29. Ieto V, Kayamori F, Montes MI, et al. Effects of oropharyngeal exercises on snoring: a randomized trial. *Chest*. 2015;148(3):683–691. doi:10.1378/chest.14-2953
30. Atilgan E, Kunter M, Algun ZC. Are oropharyngeal exercises effective in obstructive sleep apnea syndrome? *J Back Musculoskelet Rehabil*. 2020;33(2):209–216. doi:10.3233/BMR-171101
31. Tang SX, Qing J, Wang YW, et al. Clinical analysis of pharyngeal musculature and genioglossus exercising to treat obstructive sleep apnea and hypopnea syndrome. *J Zhejiang Univ Sci B*. 2015;16(11):931–939. doi:10.1631/jzus.B1500100
32. Wen Y, Xie SQ, Zhou JR, et al. Clinical features of obstructive sleep apnea-hypopnea syndrome with overweight or obesity and related influencing factors. *J Chongqing Med Univ*. 2023;48(11):1387–1392.
33. Chinese Medical Doctors Association, Sleep Medicine Committee. Multidisciplinary guidelines for the diagnosis and treatment of obstructive sleep apnea in adults. *Chin Med J*. 2018;98(24):1902–1914.
34. Zhang XL, Latscha U, Janecek P, Campana A. Guidelines for the diagnosis and treatment of obstructive sleep apnea hypopnea syndrome (2011 Revision). *Chinese Journal of Tuberculosis and Respiratory Medicine*. 1976;2012;(01):9-12:193–195. doi:10.1016/s0002-9378(16)33297-5
35. Berry RB, Budhiraja R, Gottlieb DJ, et al. Rules for scoring respiratory events in sleep: update of the 2007 AASM manual for the scoring of sleep and associated events. *J Clin Sleep Med*. 2012;8(5):597–619. doi:10.5664/jcsm.2172
36. Iber C. The AASM manual for the scoring of sleep and associated events: rules, terminology, and technical specification.
37. Prikladnicki A, Martinez D, Brunetto MG, et al. Diagnostic performance of cheeks appearance in sleep apnea. *Cranio*. 2018;36(4):214–221. doi:10.1080/08869634.2017.1376426
38. de FCM, Ferreira CLP. Protocol of orofacial myofunctional evaluation with scores. *Int J Pediatric Otorhinolaryngol*. 2008;72(3):367–375. doi:10.1016/j.ijporl.2007.11.012
39. Li J, Yang TT. An investigation of malocclusion and orofacial myofunction therapy. *Electronic J General Dentistry*. 2017;4(7):9–11+14.
40. Obesity Group of the Endocrinology Branch of the Chinese Medical Association. Expert consensus on prevention and treatment of adult obesity in China. *Chin J Endocrinol Metab*. 2011;27(09):711–717.
41. Continuous positive airway pressure for the treatment of obstructive sleep apnoea/hypopnoea syndrome. Available from: https://www.nice.org.uk/guidance/ta139. Accessed September 01, 2025.
42. Suzuki M, Okamoto T, Akagi Y, et al. Efficacy of oral myofunctional therapy in middle-aged to elderly patients with obstructive sleep apnoea treated with continuous positive airway pressure. *J Oral Rehabil*. 2021;48(2):176–182. doi:10.1111/joor.13119
43. Lorenzi-Filho G, Almeida FR, Strollo PJ. Treating OSA: current and emerging therapies beyond CPAP. *Respirology*. 2017;22(8):1500–1507. doi:10.1111/resp.13144
44. Attali V, Weber M, Rivals I, et al. Moderate-to-severe obstructive sleep apnea syndrome is associated with altered tongue motion during wakefulness. *Eur Arch Otorhinolaryngol*. 2023;280(5):2551–2560. doi:10.1007/s00405-023-07854-9
45. O'Connor-Reina C, Rodriguez-Alcala L, Ignacio JM, et al. Assessment of muscular weakness in severe sleep apnea patients: a prospective study. *Otolaryngol Head Neck Surg*. 2023;169(3):725–733. doi:10.1002/ohn.283
46. Luyster FS. Impact of obstructive sleep apnea and its treatments on partners: a literature review. *J Clin Sleep Med*. 2017;13(3):467–477. doi:10.5664/jcsm.6504
47. Schwab RJ, Pasirstein M, Pierson R, et al. Identification of upper airway anatomic risk factors for obstructive sleep apnea with volumetric magnetic resonance imaging. *Am J Respir Crit Care Med*. 2003;168(5):522–530. doi:10.1164/rccm.200208-866OC
48. Kim AM, Keenan BT, Jackson N, et al. Tongue fat and its relationship to obstructive sleep apnea. *Sleep*. 2014;37(10):1639–1648. doi:10.5665/sleep.4072
49. Castro MCZ, Santos CMD, Lucas RE, et al. Oral motor function in obesity. *J Oral Rehabil*. 2022;49(5):529–534. doi:10.1111/joor.13313
50. Silva DNM, Couto E de AB, Becker HMG, et al. Orofacial characteristics of functionally independent elders. *Codas*. 2017;29(4):e20160240. doi:10.1590/2317-1782/20172016240

51. Senaratna CV, Perret JL, Lodge CJ, et al. Prevalence of obstructive sleep apnea in the general population: a systematic review. *Sleep Med Rev*. 2017;34:70–81. doi:10.1016/j.smrv.2016.07.002
52. Brown IG, Zamel N, Hoffstein V. Pharyngeal cross-sectional area in normal men and women. *J Appl Physiol*. 1986;61(3):890–895. doi:10.1152/jappl.1986.61.3.890
53. Martin SE, Mathur R, Marshall I, et al. The effect of age, sex, obesity and posture on upper airway size. *Eur Respir J*. 1997;10(9):2087–2090. doi:10.1183/09031936.97.10092087
54. Popovic RM, White DP. Influence of gender on waking genioglossal electromyogram and upper airway resistance. *Am J Respir Crit Care Med*. 1995;152(2):725–731. doi:10.1164/ajrccm.152.2.7633734
55. Park S, Shin WS. Differences in eating behaviors and masticatory performances by gender and obesity status. *Physiol Behav*. 2015;138:69–74. doi:10.1016/j.physbeh.2014.10.001
56. Scudine KG de O, Pedroni-Pereira A, Araujo DS, et al. Assessment of the differences in masticatory behavior between male and female adolescents. *Physiol Behav*. 2016;163:115–122. doi:10.1016/j.physbeh.2016.04.053
57. Jordan AS, Wellman A, Edwards JK, et al. Respiratory control stability and upper airway collapsibility in men and women with obstructive sleep apnea. *J Appl Physiol*. 2005;99(5):2020–2027. doi:10.1152/japplphysiol.00410.2004
58. Innes JA, Morrell MJ, Kobayashi I, et al. Central and reflex neural control of genioglossus in subjects who underwent laryngectomy. *J Appl Physiol*. 1995;78(6):2180–2186. doi:10.1152/jappl.1995.78.6.2180
59. Jordan AS, Catcheside PG, O'Donoghue FJ, et al. Selected Contribution: genioglossus muscle activity at rest and in response to brief hypoxia in healthy men and women. *J Appl Physiol*. 2002;92(1):410–417. doi:10.1152/japplphysiol.00461.2001
60. Pillar G, Malhotra A, Fogel R, et al. Airway mechanics and ventilation in response to resistive loading during sleep. *Am J Respir Crit Care Med*. 2000;162(5):1627–1632. doi:10.1164/ajrccm.162.5.2003131
61. Kim S, Lee KY, Siddiquee AT, et al. Gender differences in association between expiratory dynamic airway collapse and severity of obstructive sleep apnea. *Eur Radiol*. 2024;34(6):3730–3741. doi:10.1007/s00330-023-10322-x