

Sleep-Disordered Breathing and Its Impact on Respiratory Health: Mechanisms, Clinical Significance, and Treatment Strategies

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Abstract

Background:

Sleep-disordered breathing is a clinically and physiologically heterogeneous disorder of sleep respiration. The disorders include obstructive sleep apnea, central sleep apnea, and obesity hypoventilation syndrome. Besides disturbed sleep quality, SDB has far-reaching impacts on respiratory physiology, such as impairment of lung function, abnormalities of gas exchange, and acceleration of chronic respiratory disease.

Objectives:

This study evaluates the impact of SDB on respiratory health as regards prevalence, physiological mechanisms, clinical effects, and management.

Methods:

A cross-sectional observation study was performed in 300 suspected SDB adults. Diagnosis was confirmed by polysomnography, while spirometry and arterial blood gas analysis assessed respiratory health. Demographics, BMI, comorbid illness, and smoking were also obtained.

Results:

Prevalence of SDB was 85% in the study group, primarily OSA. Moderate-to-severe SDB was associated with reduced FEV1, hypoxemia, and hypercapnia. Male gender and obesity were independent risk factors.

Conclusions:

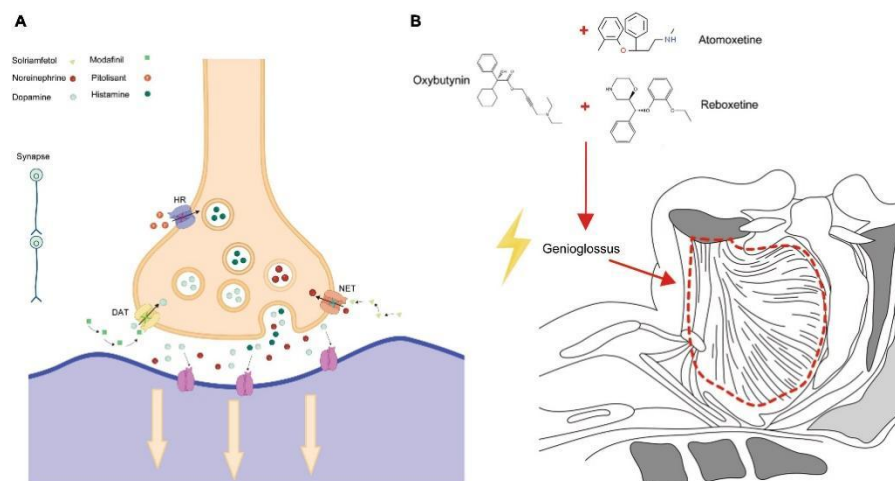
SDB has a deleterious effect on respiratory health by altering pulmonary mechanics, causing nocturnal hypoxemia, and exacerbating chronic lung disease. Early identification and treatment with CPAP, weight loss, and risk factor modification are crucial in avoiding complications.

Keywords: Polysomnography, CPAP, Respiratory physiology, SDB

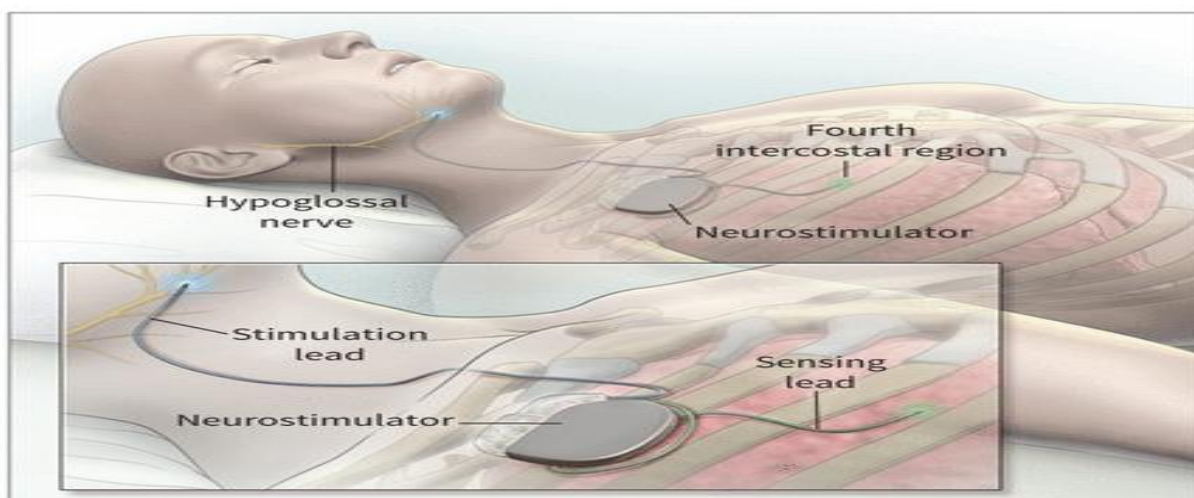
Introduction

Sleep-disordered breathing refers to a spectrum of sleep respiratory disturbances that impair ventilation and oxygenation [1]. The most common subtype, obstructive sleep apnea, is characterized by repeated cycles of upper airway partial or complete obstruction during sleep, causing intermittent hypoxemia, sleep

disturbance, and sympathetic activation [2]. Central sleep apnea results from abnormal central regulation of ventilation, and obesity hypoventilation syndrome is obesity-induced chronic alveolar hypoventilation [3].



Together, these conditions are a major worldwide health condition with increasing prevalence [4]. The morbidity of SDB on respiratory function is considerable. Chronic intermittent hypoxemia and hypercapnia are the cause of oxidative stress, systemic inflammation, and remodeling of pulmonary vessels [5]. These pathophysiological alterations compromise gas exchange, decrease ventilator capacity, and hasten the progression of chronic respiratory disease. Chronic obstructive pulmonary disease (COPD), asthma, and interstitial lung disease patients frequently deteriorate with SDB if it is not treated [6]. In addition, SDB correlates with pulmonary hypertension, blunted ventilator drive, and risk of acute respiratory failure. Epidemiologic studies recognize OSA in 9–38% of the adult population with a higher prevalence among obesity, men, and older adults [7]. Importantly, the majority of patients remain undiagnosed due to nonspecific findings such as nocturnal snoring, witnessed apnea, and excessive daytime sleepiness [8].



The diagnostic gold standard, polysomnography, provides accurate measurement by the apnea–hypopnea index (AHI) but is unavailable in resource-poor settings. SDB affects respiratory health and vice versa [9]. On the one hand, chronic respiratory disease worsens SDB by reducing upper airway stability and lung

volume; on the other, undertreated SDB accelerates lung function loss and worsening prognosis [10]. This bidirectional impact requires early diagnosis and treatment in order to prevent long-term sequelae. This study investigates the prevalence, clinical characteristics, and respiratory consequences of SDB in adults presenting with sleep-related complaints, while also emphasizing mechanisms, clinical implications, and management strategies to mitigate disease burden.

Methodology

A cross-sectional study was conducted from January 2020 to December 2023 at a tertiary care hospital. A total of 300 adults (18–70 years) with suspected SDB were enrolled. Inclusion criteria included habitual snoring, daytime sleepiness, or witnessed apneas. Exclusion factors were prior CPAP therapy, neuromuscular disease, or recent acute respiratory infection. Overnight polysomnography was utilized to confirm diagnosis and classify severity (mild, moderate, severe) in all subjects using the apnea–hypopnea index (AHI). FEV1, FVC, and FEV1/FVC ratio were evaluated using spirometry. Arterial blood gases were measured for PaO₂, PaCO₂, and oxygen saturation. BMI, smoking status, and comorbidities (COPD, asthma, hypertension) were recorded. Analysis was conducted on data using SPSS version 26, with ANOVA and t-tests. Statistical significance was set as p-values <0.05.

Results

Of 300 participants, 255 (85%) had SDB diagnosed. OSA was detected in 65%, CSA in 12%, and OHS in 8%, whereas 15% of them did not have a sleep disorder. The majority of OSA subjects were male obese subjects and were more prevalent in smokers. The moderate-to-severe OSA subjects had significant reductions in FEV1 and PaO₂ with elevated PaCO₂ than non-SDB subjects.

Table 1. Demographic and Clinical Characteristics

Variable	SDB Group (n=255)	Non-SDB Group (n=45)	p-value
Age (years)	49.6 ± 10.3	46.2 ± 9.8	0.04
Male (%)	62%	48%	0.03
BMI (kg/m ²)	32.1 ± 5.8	27.4 ± 4.6	<0.01
Smokers (%)	38%	24%	0.05

Table 2. Respiratory Function Parameters

Parameter	Mild OSA	Moderate OSA	Severe OSA	Non-SDB	p-value
FEV1 (% predicted)	82.4 ± 6.7	76.2 ± 7.5	69.8 ± 8.4	88.9 ± 5.9	<0.01
PaO ₂ (mmHg)	82.6 ± 6.3	77.2 ± 7.1	70.5 ± 8.9	89.1 ± 5.7	<0.01
PaCO ₂ (mmHg)	38.4 ± 3.2	41.6 ± 4.1	46.7 ± 5.0	36.9 ± 2.8	<0.01

Discussion

This study demonstrates that sleep-disordered breathing greatly incapacitates respiratory health, and the magnitude of impairment is proportionate to reduced lung function and deranged gas exchange [11]. There were significant decreases in FEV1 and oxygen saturation and increases in PaCO₂ among

moderate-to-severe OSA patients [12]. The findings are in agreement with the hypothesized hypothesis that SDB is causally responsible for ventilator inefficiency and aggravation of chronic respiratory disease. Pathophysiology includes recurring intermittent hypoxemia, oxidative stress, and activation of the sympathetic nervous system [13]. These pathophysiological changes lead to pulmonary vasoconstriction, vascular remodeling, and decreased compliance, finally compromising respiratory reserve. The resulting hypercapnia in obese and severe OSA subjects also reflects poor ventilator compensation, exposing them to chronic respiratory failure and pulmonary hypertension [14]. The increased prevalence of SDB among obese individuals also points towards a synergistic role of excess body mass, reduced lung volumes, and increased collapsibility of the upper airway [15]. Male dominance of OSA prevalence is consistent with previous reports attributing variability on the basis of hormonal and anatomical factors. Current smoking was also present as a contributing factor, very likely through its influence upon chronic airway inflammation and neuromuscular control of the upper airway [16]. Clinically, this outcome stresses the need for SDB screening among high-risk patients, such as obesity, chronic respiratory disease, or inexplicable respiratory distress. Polysomnography remains the gold standard for diagnosis but less sophisticated portable equipment can be employed to provide access in low-resource settings. Management strategies need to adopt a multimodal strategy [17]. Continuous positive airway pressure (CPAP) remains the gold standard therapy for OSA, with respiratory and sleep parameters improved. Comorbid therapy including weight loss, smoking cessation, and comorbidity therapy further optimizes outcomes. Non-invasive ventilation may be required to reverse hypoventilation in patients with OHS [18]. Strong as this study is, it has limitations. Its cross-sectional design precludes causal inference, and data from a hospital-based sample may not reflect community prevalence. Longitudinal studies in the future are warranted to determine if treatment of SDB changes the natural course of chronic respiratory disease and improves long-term outcomes. In conclusion, the study further reinforces the bidirectional interaction of SDB with respiratory health and emphasizes the importance of early identification and directed interventions to mitigate disease burden.

Conclusion

Sleep-disordered breathing imposes a severe impact on respiratory health in the terms of impaired lung function, chronic hypoxemia, and hypercapnia. It is prevalent among males and obese people and frequently complicates chronic respiratory diseases. Early identification and treatment, particularly with CPAP therapy, weight management, and lifestyle modification, are critical in optimizing outcomes. Treatment of SDB within composite respiratory care can reduce morbidity, improve the quality of life, and prevent advancement of chronic respiratory disease.

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