SRYAHWA PUBLICATIONS

Archives of Pulmonology and Respiratory Medicine
ISSN: 2639-362X | Volume 9 Issue 1, 2026
https://doi.org/10.22259/2639-362X.0901005

REVIEW ARTICLE

Obstructive Sleep Apnea: Anatomical, Neuromuscular and Metabolic Factors

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Received: 12 August 2026 Accepted: 27 August 2026 Published: 31 August 2026

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Abstract

Obstructive sleep apnea (OSA) is one of the most prevalent sleep-related breathing disorders, characterized by recurrent episodes of partial or complete upper airway obstruction during sleep. These events result in intermittent hypoxia, hypercapnia, and sleep fragmentation, contributing to significant cardiovascular and metabolic morbidity.

The pathophysiology of OSA is multifactorial and involves a complex interaction between anatomical susceptibility, impaired neuromuscular control of the upper airway, ventilatory control instability, and metabolic dysfunction. Obesity, insulin resistance, and hormonal alterations play a central role in disease development and progression.

This review aims to provide a comprehensive overview of the anatomical, neuromuscular, and metabolic mechanisms underlying OSA, with emphasis on their interaction and clinical implications.

Keywords: Obstructive Sleep Apnea, Upper Airway, Obesity, Insulin Resistance, Neuromuscular Control.

1. Introduction

Obstructive sleep apnea (OSA) represents a major public health concern with a steadily increasing prevalence worldwide, largely driven by the obesity epidemic[1,2]. It is estimated that moderate-to-severe OSA affects approximately 20–30% of the adult population[2].

OSA is characterized by recurrent episodes of apnea and hypopnea during sleep, leading to intermittent hypoxemia, hypercapnia, and sleep fragmentation. These disturbances result in activation of the sympathetic nervous system, oxidative stress, endothelial dysfunction, and systemic inflammation[3–5]. Consequently, OSA is strongly associated with cardiovascular diseases, metabolic disorders, and increased mortality[5].

Current understanding conceptualizes OSA as a disorder resulting from the interaction of four key pathophysiological traits: upper airway anatomical compromise, impaired neuromuscular responsiveness, ventilatory control instability (loop gain), and low arousal threshold[6].

2. Anatomical Factors

2.1 Craniofacial Abnormalities

Craniofacial structural abnormalities such as micrognathia and retrognathia reduce the posterior airway space and predispose to upper airway collapse[7]. Posterior displacement of the tongue narrows the oropharyngeal lumen, especially in the supine position.

Citation: Spiropoulou A, Spyropoulos V, Spyropoulou G, *et al.* Obstructive Sleep Apnea: Anatomical, Neuromuscular and Metabolic Factors. Archives of Pulmonology and Respiratory Medicine. 2026;9(1):16-18.

2.2 Soft Tissue Enlargement and Fat Deposition

Enlargement of the soft palate, uvula, and tonsils contributes to airway narrowing[8]. In obesity, fat deposition in the parapharyngeal region further increases airway collapsibility[9].

2.3 Upper Airway Mechanics

The pharynx is a compliant structure lacking rigid support. Airway patency depends on the balance between intraluminal and surrounding tissue pressures. During inspiration, negative pressure promotes collapse in susceptible individuals[6].

3. Neuromuscular Mechanisms

Upper airway patency is maintained by pharyngeal dilator muscles, particularly the genioglossus[10]. During sleep, especially REM sleep, neuromuscular tone decreases significantly[11].

In OSA patients, impaired reflex activation of these muscles in response to negative pressure contributes to airway instability[12]. Increased loop gain further promotes ventilatory instability and cyclical breathing[6].

4. Imaging and Diagnostic Approaches

Computed tomography (CT) and magnetic resonance imaging (MRI) are useful for evaluating upper airway anatomy[13].

Drug-induced sleep endoscopy (DISE) allows real-time assessment of airway collapse under sedation[14].

Ultrasound elastography is an emerging tool for evaluating tissue stiffness and may correlate with disease severity[15].

5. Metabolic and Endocrine Factors

5.1 Obesity

Obesity is the strongest risk factor for OSA, increasing prevalence up to tenfold[2],[16]. Fat accumulation in the neck narrows the airway, while central obesity reduces lung volumes[16].

5.2 Insulin Resistance and Diabetes

Intermittent hypoxia induces inflammatory pathways and insulin resistance[17]. The relationship between OSA and type 2 diabetes is bidirectional[18].

5.3 Hormonal Dysregulation

OSA is associated with altered appetite regulation, including increased ghrelin and reduced leptin sensitivity[19].

Hypothyroidism may further worsen OSA via reduced metabolic rate and neuromuscular tone[20].

6. Age and Sex Differences

OSA prevalence increases with age due to structural and neuromuscular changes[21].

It is more common in men, but the difference decreases after menopause, suggesting hormonal protection in females[22].

7. Systemic Consequences

OSA is strongly associated with hypertension, coronary artery disease, stroke, and heart failure[5],[23].

Chronic intermittent hypoxia and sympathetic activation are key mechanisms.

OSA is also linked to cognitive impairment, depression, and reduced quality of life[24].

8. Conclusion

Obstructive sleep apnea is a complex disorder involving anatomical, neuromuscular, and metabolic mechanisms. Understanding these interactions is essential for accurate diagnosis and individualized therapy.

Early intervention, particularly targeting obesity, may significantly improve outcomes.

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