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ПАТОФИЗИОЛОГИЯ ОБСТРУКТИВНОГО АПНОЭ СНА

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SUMMARY

Obstructive sleep apnea (OSA) is a common sleep-related breathing disorder characterized by recurrent episodes of upper airway obstruction leading to apnea and hypopnea. A combination of anatomical and functional factors contributes to the development of OSA: narrowing of the upper airway (e.g., due to obesity or craniofacial features), reduced tone of pharyngeal dilator muscles during sleep, increased ventilatory drive (ventilatory control instability, high loop gain), and a low arousal threshold. These factors promote pharyngeal collapse during sleep with episodes of hypoxia and arousals. Recurrent intermittent hypoxia and sleep fragmentation trigger a cascade of reactions – sympathetic activation, oxidative stress, systemic inflammation, endothelial dysfunction — which underlie cardiovascular complications (hypertension, atherosclerosis, arrhythmias), metabolic disturbances (insulin resistance, appetite-related hormonal imbalance), and neurocognitive consequences (daytime sleepiness, cognitive dysfunction).

KEYWORDS: *obstructive sleep apnea, pathophysiology, intermittent hypoxia, cardiovascular complications, metabolic disorders.*

CONFLICT OF INTEREST. The authors declare no conflict of interest.

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РЕЗЮМЕ

Обструктивное апноэ сна (ОАС) — распространенное расстройство дыхания во сне, характеризующееся повторяющимися эпизодами обструкции верхних дыхательных путей, приводящими к апноэ и гипопноэ. В развитии ОАС участвует сочетание анатомических и функциональных факторов: сужение верхних дыхательных путей (например, из-за ожирения, краниофациальных особенностей), снижение тонуса мышц-глоточных дилататоров во сне, повышенная возбудимость дыхательного центра (нестабильность вентиляционного контроля, высокий loop gain) и низкий порог пробуждения. Эти факторы способствуют коллапсу глотки во сне с эпизодами гипоксии и пробуждениями. Повторяющаяся интермиттирующая гипоксия и фрагментация сна запускают каскад реакций – симпатическую активацию, оксидативный стресс, системное воспаление, эндотелиальную дисфункцию – что лежит в основе сердечно-сосудистых осложнений (гипертония, атеросклероз, аритмии), метаболических нарушений (инсулинорезистентность, дисбаланс гормонов аппетита) и нейрокогнитивных последствий (дневная сонливость, когнитивные нарушения).

КЛЮЧЕВЫЕ СЛОВА: обструктивное апноэ сна; патофизиология; интермиттирующая гипоксия; сердечно-сосудистые осложнения; метаболические нарушения.

КОНФЛИКТ ИНТЕРЕСОВ. *Авторы заявляют об отсутствии конфликта интересов.*

Introduction

Obstructive sleep apnea (OSA) is a syndrome in which recurrent partial or complete closure of the upper airway occurs during sleep, leading to cessation or reduction of pulmonary ventilation (apneas and hypopneas). These episodes cause a drop in blood oxygen saturation and activate stress responses, often culminating in brief awakenings (arousals). Epidemiological studies indicate that OSA is extremely common: globally, approximately 1 billion people aged 30–69 years are affected, with around 425 million having moderate to severe disease. The prevalence of OSA increases with age and is more frequent in men than in women (ratio approximately 2:1). The main risk factor is obesity: excessive fat deposition in the neck and pharyngeal region leads to airway narrowing, so the rise in body mass index correlates strongly with OSA risk. Other predisposing factors include neck circumference (>43 cm in men and >38 cm in women), craniofacial structural anomalies (micrognathia, retrognathia, narrow maxilla), tonsillar hypertrophy (especially important in childhood), chronic nasal obstruction, smoking, and meno-

pause in women. Family history and certain neurological disorders (neuropathies) may contribute to OSA by impairing the function of pharyngeal dilator muscles. Thus, OSA is a multifactorial condition arising at the intersection of anatomical predisposition and functional disturbances of respiratory regulation during sleep [1, 2].

The clinical manifestations of OSA — snoring, choking episodes during sleep, daytime sleepiness, fatigue, impaired concentration — are nonspecific; therefore, diagnosis requires instrumental verification (overnight polysomnography or respiratory monitoring. The importance of timely detection of OSA is dictated not only by its impact on quality of life but also by its long-term consequences. Repeated episodes of hypoxemia and arousals initiate a cascade of pathophysiological reactions leading to target-organ damage. OSA is considered an independent risk factor for a wide range of conditions — arterial hypertension, coronary artery disease, stroke, cardiac arrhythmias, heart failure, type 2 diabetes, nonalcoholic fatty liver disease, cognitive and behavioral disorders. Although epidemiological data convincingly demonstrate an increased risk of these disorders in OSA, whether treatment of OSA (e.g., CPAP therapy) reduces the incidence of cardiovascular events remains a matter of debate. This review presents current concepts of OSA pathogenesis and the systemic alterations caused by this condition, taking into account the strength of the available evidence. Particular attention is given to factors determining airway collapse during sleep and to the pathophysiological mechanisms underlying cardiovascular and metabolic complications in OSA [3].

Pathophysiological Mechanisms of OSA Development

Current concepts of OSA pathogenesis identify four major interrelated mechanisms: anatomical susceptibility to upper airway collapse, insufficient activity of pharyngeal dilator muscles, instability of ventilatory control, and abnormally low arousal threshold. Each of these factors contributes to sleep-related apnea in different patients to varying degrees.

Anatomical factors

Anatomical narrowing and increased compliance of the pharyngeal walls form the substrate for airway obstruction during sleep. While awake, pharyngeal muscle tone maintains airway patency; however, during sleep muscle tone falls, and in individuals with a narrow airway this leads to pharyngeal collapse — especially during inspiration due to negative intraluminal pressure. Patients with OSA frequently exhibit excessive fat deposition in the neck and pharyngeal regions associated with obesity, which mechanically narrows the airway and increases its collapsibility. In addition to obesity, anatomical contributors include craniofacial features — low hyoid position, retrognathia and micrognathia, narrow maxilla — as well as hypertrophy of the palatine tonsils and adenoids (particularly in children). These factors reduce the upper airway lumen and increase the likelihood of its closure during sleep.

Pharyngeal collapsibility is quantitatively described by the critical closing pressure (Pcrit): in healthy individuals, Pcrit is usually negative (< –5 cm H_2O), whereas in OSA it is often elevated to around 0 cm H_2O or even positive. In one study, average Pcrit in patients with severe OSA was – 0.3 cm H_2O compared with – 6.2 cm H_2O in healthy subjects. Notably, about 20% of patients with severe OSA had relatively non-collapsible airways (Pcrit – 2 to – 5 cm H_2O, i.e., near normal), and other pathophysiological abnormalities compensated for their limited anatomical impairment. Thus, anatomical predisposition is important but not the sole component of OSA pathogenesis [5, 6].

Neuromuscular factors

Maintenance of pharyngeal patency relies heavily on reflex activation of its dilator muscles. Normally, a decrease in airway pressure during inspiration elicits rapid reflex contraction of pharyngeal muscles (e.g., the genioglossus), counteracting collapse. In OSA, this compensatory response is often insufficient: dilator muscle activity during sleep fails to increase adequately in response to dropping pressure. Studies reveal that approximately 30–40% of OSA patients demonstrate markedly weakened or delayed genioglossus responses to negative pressure. One possible cause is neuropathy of pharyngeal mechanoreceptors from repeated traumatic collapses, reducing sensitivity and impairing the reflex arc. Additionally, chronic neuromuscular alterations (e.g., myopathy in obesity or age-related sarcopenia of pharyngeal muscles) may reduce airway functional performance. Consequently, some patients fail to generate sufficient increases in pharyngeal muscle tone during inspiration, promoting recurrent obstruction [7].

Ventilatory control instability

In some patients, OSA is significantly influenced by dysregulated central respiratory control — enhanced chemosensitivity leading to ventilatory instability. Normally, CO_2 accumulation after a hypopnea/apnea stimulates the respiratory center, restoring ventilation. However, when loop gain is high — reflecting an amplified ventilatory response — recovery becomes excessive: hyperventilation ensues, lowering CO_2 below baseline, which reduces respiratory drive and precipitates another apnea. This vicious cycle of hyperventilation–hypoventilation underlies periodic breathing. Elevated loop gain is observed in roughly one-third of OSA patients. In the aforementioned group of individuals with relatively non-collapsible airways, severe apnea was attributable to high loop gain: their respiratory center sensitivity to CO_2 was nearly twice that of patients with more pronounced anatomical defects. Ventilatory instability prolongs and increases the frequency of apneas, especially when combined with a low arousal threshold (see below). Therapeutically reducing loop gain (e.g., via oxygen therapy or acetazolamide) has lowered apnea severity in experimental settings, confirming this mechanism's relevance [7].

Low arousal threshold

Abnormalities in arousal processes also contribute to OSA pathogenesis. Some patients exhibit an excessively low arousal threshold: even minimal increases in breathing effort or slight hypoxia trigger awakening. On one hand, early arousal may limit apnea duration and the depth of desatura-

tion. On the other hand, frequent sleep interruptions exacerbate sleep fragmentation and sympathetic activation, sustaining a vicious cycle. Physiological studies indicate that roughly 30–40% of OSA patients have a pathologically low arousal threshold, contributing to chronic instability of sleep and breathing. The opposite scenario — an excessively high arousal threshold — occurs less commonly but may cause prolonged apneas with severe hypoxemia because the patient fails to awaken in time. Achieving an optimal balance of arousal reactivity is important for stabilizing sleep-related breathing. Pharmacological modulation of the arousal threshold (e.g., with non-benzodiazepine hypnotics to moderately increase it) has shown initial promise in reducing the apnea–hypopnea index in pilot studies [8].

Systemic Consequences and Complications of OSA

Recurrent hypoxic episodes and arousals in OSA initiate a complex array of pathological changes across multiple organs and systems. Below are the most significant consequences from a pathophysiological perspective and based on the available evidence.

Cardiovascular abnormalities

OSA is recognized as an independent risk factor for cardiovascular disease. Intermittent nocturnal hypoxia, fluctuations in intrathoracic pressure, and arousals activate the sympathetic nervous system and systemic inflammation, resulting over time in endothelial dysfunction, hypercoagulability, and atherogenesis. Chronic OSA is associated with the development of arterial hypertension, coronary artery disease, heart failure, stroke, and cardiac arrhythmias (particularly atrial fibrillation). In a major 10-year prospective study, untreated severe OSA significantly increased the risk of stroke and heart failure, independent of other risk factors. Meta-analyses of population studies also confirm that OSA is associated with a 20–30% increase in relative risk of fatal and nonfatal cardiovascular events. Chronic nocturnal hypoxemia is believed to play a central role by inducing inflammatory cytokine release, endothelial dysfunction, and accelerated atherosclerosis. Moreover, abrupt sympathetic surges during arousals lead to transient blood pressure spikes, fostering myocardial hypertrophy and vascular remodeling. Thus, OSA-related pathophysiological effects create strong preconditions for cardiovascular damage [9].

A substantial body of clinical data supports the association of OSA with cardiovascular disease. Observational studies show that patients with severe OSA exhibit increased prevalence and incidence of hypertension and other cardiac disorders compared with individuals without sleep apnea. Furthermore, treatment of OSA can improve certain surrogate markers: for example, CPAP typically reduces elevated blood pressure by 2–3 mmHg on average. However, results of large randomized controlled trials with hard endpoints have been less definitive. The SAVE trial (nearly 2700 patients) found no reduction in cardiovascular events (myocardial infarction, stroke) when CPAP was added to standard therapy in patients with cardiovascular disease and comorbid OSA. Limited adherence to CPAP and preexisting subclinical yet irreversible cardiovascular injury may have reduced intervention efficacy. Nonetheless, a meta-analysis using individual patient data demonstrated that patients with good CPAP adherence experience reduced risk of stroke and other complications compared with untreated individuals. Mandibular advancement devices and hypoglossal nerve stimulation used to treat OSA have also been linked to improvements in certain cardiovascular risk markers (e.g., heart rate variability, nocturnal blood pressure). Thus, while OSA is clearly associated with cardiovascular abnormalities, whether active treatment prevents major cardiovascular events remains an active area of research and expert discussion [8].

Metabolic effects

Obstructive sleep apnea is closely linked to obesity and components of the metabolic syndrome. More than 70% of OSA patients are overweight or obese, and apnea episodes themselves contribute to further weight gain through hormonal and metabolic shifts. Nocturnal hypoxia and sleep fragmentation activate stress pathways, promote insulin resistance, and impair glucose tolerance. Population-based studies show that moderate to severe OSA is associated with a 30–40% increased risk of developing type 2 diabetes over approximately 10 years of follow-up. This relationship persists even after adjusting for BMI, indicating an independent contribution of OSA to glucose dysregulation. Pathophysiologically, intermittent hypoxemia in OSA induces pro-oxidant and pro-inflammatory pathways (e.g., increasing expression of HIF-1α and NF-κB), driving systemic inflammation and dysfunction of white adipose tissue. As a result, insulin resistance develops, fasting insulin and glucose levels rise, and metabolic homeostasis is disrupted.

Chronic sleep loss and sympathetic activation also disturb leptin and ghrelin signaling — key appetite-related hormones — promoting overeating and further weight gain. Thus, a vicious cycle forms: OSA worsens metabolic disturbances, which in turn exacerbate apnea severity. Treatment can partially improve metabolic markers. In several studies, CPAP use reduced HbA1c levels in patients with diabetes, although findings across trials are inconsistent. Weight reduction remains the most effective intervention for breaking the OSA–metabolic syndrome connection. Intensive weight-loss programs or bariatric surgery have been shown to substantially reduce OSA severity (≥50% reduction in apnea–hypopnea index) while simultaneously improving insulin sensitivity and lipid profiles. This underscores the pivotal role of obesity as the shared pathophysiological mechanism linking OSA and metabolic disorders [10–12].

Neurocognitive and other consequences

OSA adversely affects central nervous system function. Chronic sleep fragmentation reduces slow-wave and REM sleep, manifesting as daytime sleepiness, impaired attention, memory deficits, and increased fatigue. Intermittent hypoxia damages neurons via oxidative stress and activation of programmed cell death. Patients with severe OSA demonstrate reduced gray matter volume in the hippocampus and prefrontal cortex, correlating with cognitive impairment. Clinically, this appears as deficits in executive function and increased

depressive and anxiety symptoms. OSA also significantly increases the risk of motor vehicle accidents due to daytime sleepiness — by 2–2.5 times according to meta-analyses. Treatment that improves sleep quality typically enhances cognitive performance and reduces daytime sleepiness, although some individuals retain residual deficits despite adequate CPAP therapy.

Another critical aspect is the relationship between OSA and stroke. In addition to raising stroke risk, OSA also worsens post-stroke recovery. Hypoxemic episodes aggravate ischemic brain injury and slow neuroplasticity. Therefore, screening and treatment for OSA are considered essential elements of stroke rehabilitation [13–15].

OSA also affects other organs and systems. Chronic intermittent hypoxia may impair kidney function, contributing to the progression of chronic kidney disease and refractory hypertension. Patients with severe OSA more frequently exhibit erectile dysfunction and decreased testosterone levels, attributed to hypoxemia and sleep fragmentation. A possible association between OSA and cancer risk has been proposed: some observational studies report higher cancer-related mortality in individuals with pronounced nocturnal hypoxemia. Intermittent hypoxia may stimulate angiogenesis and tumor progression, but current evidence remains inconsistent and insufficient for definitive conclusions. Additionally, OSA worsens the course of chronic diseases — e.g., type 2 diabetes, chronic obstructive pulmonary disease (the "overlap syndrome"), and obesity hypoventilation syndrome (Pickwick syndrome). The coexistence of OSA and COPD leads to higher rates of pulmonary hypertension and respiratory failure, making recognition and treatment of OSA crucial in such patients [14].

Prospects for Personalized Therapy

Modern understanding of OSA pathophysiology creates opportunities for personalized treatment approaches. CPAP remains the standard therapy, effectively reducing apnea severity in most patients. However, tolerance to CPAP is often limited, and some patients have residual symptoms despite treatment. Since OSA is a heterogeneous disorder, identifying the predominant pathophysiological mechanism in each individual enables targeted alternative interventions. Distinguishing phenotypic "endotypes" of OSA (predominantly anatomical, neuromuscular, ventilatory control instability, or low arousal threshold) is important for therapy selection.

For patients in whom anatomical abnormalities predominate (e.g., craniofacial anomalies or tonsillar hypertrophy), surgical interventions can significantly improve outcomes. In adults, uvulopalatopharyngoplasty and its modern variants, as well as airway-expansion surgeries (e.g., mandibular expansion), are widely used. In children, adenotonsillectomy is often the first-line treatment, relieving nasopharyngeal obstruction. For positional OSA, positional therapy devices that prevent supine sleep are effective [16].

When low pharyngeal muscle activity is the primary issue, hypoglossal nerve stimulation emerges as a promising therapy. Implantable stimulators periodically activate the genioglossus during sleep to prevent airway collapse. Clinical trials (e.g., the STAR trial) demonstrated >50% reduction in apnea–hypopnea index in patients intolerant to CPAP. Myofunctional therapy — specialized exercises for the tongue and pharyngeal muscles — can also improve muscle tone and moderately reduce OSA severity [17].

For patients with high loop gain (ventilatory control instability), approaches targeting respiratory center sensitivity are being studied. Supplemental nocturnal oxygen can reduce ventilatory oscillations and decrease apnea frequency in such patients. Respiratory stimulants (e.g., acetazolamide), effective in high-altitude periodic breathing, are currently under investigation for OSA.

Finally, in patients with excessively low arousal thresholds, cautious pharmacological elevation of this threshold is of interest. Low doses of certain hypnotics (zopiclone, eszopiclone) have increased sleep stability without worsening breathing in clinical studies, allowing apnea to persist long enough for airway patency to be restored without arousal, thereby improving ventilation. In a pilot randomized trial, adding eszopiclone to CPAP in patients with low arousal threshold yielded an additional 30% reduction in apnea–hypopnea index compared with CPAP alone [18].

The approaches described above reflect a shift toward personalized OSA therapy based on pathophysiological profiling. Combinations of methods (e.g., mild sedative + oxygen to raise arousal threshold and reduce loop gain) have shown synergistic effects in small studies. Development of precision-medicine algorithms is expected to enhance treatment efficacy and overcome limitations of CPAP. Simplified diagnostic tests for OSA phenotyping (such as determining critical pressure via CPAP dial-down or testing oxygen sensitivity) are already being proposed for clinical practice. These tools may allow clinicians to prescribe targeted alternative or adjunctive therapies based on the dominant mechanism of obstruction [19, 20].

Conclusion

Obstructive sleep apnea is a complex, multifactorial condition affecting multiple levels of respiratory regulation — from upper airway anatomy to neuroreflex mechanisms. Understanding of OSA pathophysiology has advanced considerably in recent years, enabling identification of phenotypic variants and the development of personalized treatment strategies. OSA is associated with increased risk of cardiovascular, metabolic, and neurocognitive complications, although the extent of these effects and their reversibility vary according to patient characteristics and comorbidities. Modern management of OSA requires a multidisciplinary approach integrating lifestyle modification, device-based and surgical interventions, and when necessary, pharmacological agents targeting specific pathophysiological pathways. Large prospective studies remain essential to clarify long-term effects of OSA treatment on the prevention of myocardial infarction, stroke, and other outcomes, as well as to optimize therapeutic combinations. Nevertheless, existing evidence underscores that recognition and treatment of OSA are crucial for improving long-term prognosis and quality of life. Ongoing research in OSA pathophysiology promises further refinement of treatment strategies and improved patient outcomes.

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