10.48047/jocaaa.2024.33.06.135

# Obstructive Sleep Apnea–Hypopnea Syndrome: Pathophysiology, Clinical Consequences, and Comorbidities

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**Received:** 28 March 2024, **Accepted:** 17 April 2024, **Published:** 20 May 2024

## ABSTRACT

**Background:** Obstructive sleep apnea–hypopnea syndrome (OSAHS) is a prevalent sleep-related breathing disorder characterized by recurrent episodes of upper airway collapse during sleep, resulting in intermittent hypoxemia, intrathoracic pressure changes, and sleep fragmentation. The global prevalence of OSAHS is rising in parallel with the obesity epidemic, and it is now recognized as a major public health concern due to its strong association with multisystem comorbidities. Although initially considered a disorder of disrupted sleep and excessive daytime sleepiness, extensive research over the past two decades has highlighted its far-reaching clinical consequences on cardiovascular, metabolic, neurocognitive, respiratory, and other systemic domains.

The aim of this review is to provide a comprehensive overview of the pathophysiology, clinical manifestations, and multisystem comorbidities associated with OSAHS. Emphasis is placed on the mechanistic links between intermittent hypoxia, sympathetic overactivity, oxidative stress, and systemic inflammation, which serve as key drivers of comorbid conditions. Cardiovascular consequences, including hypertension, ischemic heart disease, arrhythmias, and heart failure, are well-established, whereas emerging evidence points to metabolic dysregulation, insulin resistance, and type 2 diabetes mellitus as critical extensions of the disease burden. Furthermore, neurocognitive impairment, mood disorders, and an increased risk of cerebrovascular disease highlight the neurological dimension of OSAHS. Respiratory overlap with asthma and chronic obstructive pulmonary disease, along with associations with chronic kidney disease, gastroesophageal reflux, and even cancer, illustrate the multisystem impact of this condition.

In conclusion, OSAHS is not merely a sleep disorder but a systemic condition with profound clinical consequences. Recognizing and addressing its comorbidities is crucial for optimizing outcomes, reducing morbidity and mortality, and alleviating the broader healthcare burden. Future research must focus on personalized diagnostic tools, biomarkers, and multidisciplinary management strategies to advance care for patients with OSAHS.

**Keywords:** *Obstructive Sleep Apnea, Consequences, Comorbidities*

## INTRODUCTION

10.48047/jocaaa.2024.33.06.135

Obstructive sleep apnea–hypopnea syndrome (OSAHS) is a highly prevalent sleep-related breathing disorder defined by recurrent episodes of upper airway collapse during sleep, leading to intermittent hypoxemia, hypercapnia, and sleep fragmentation. The syndrome is associated with loud snoring, witnessed apneas, and excessive daytime sleepiness, but its clinical impact extends far beyond sleep disruption. Epidemiological data estimate that moderate-to-severe OSAHS affects approximately 10–17% of men and 3–9% of women in the general population, with prevalence rising sharply in those with obesity and advancing age [1]. Despite its widespread occurrence, OSAHS remains underdiagnosed, with many patients presenting only when systemic complications arise [2].

The burden of OSAHS is increasingly recognized not only in terms of reduced quality of life and neurocognitive dysfunction but also in its strong association with cardiovascular, metabolic, respiratory, and psychiatric comorbidities. Studies have consistently demonstrated that untreated OSAHS contributes to increased risk of hypertension, ischemic heart disease, stroke, diabetes mellitus, and depression, highlighting its role as a multisystem disease with major implications for public health [3]. Moreover, OSAHS contributes significantly to healthcare costs, accidents due to daytime sleepiness, and overall mortality [4].

The pathophysiological mechanisms underlying the systemic consequences of OSAHS are complex and multifactorial. Intermittent hypoxia, oxidative stress, sympathetic nervous system activation, systemic inflammation, and endothelial dysfunction act synergistically to promote organ damage and metabolic dysregulation [5]. Despite advances in understanding these mechanisms, there is considerable heterogeneity in clinical presentation, disease severity, and comorbidity burden among patients, suggesting that individualized approaches are needed.

The aim of this review is to provide a comprehensive and updated account of the pathophysiology of OSAHS and its clinical consequences across different organ systems. Particular emphasis is placed on the spectrum of comorbidities associated with OSAHS, ranging from cardiovascular and metabolic diseases to neurocognitive and respiratory disorders. This review also seeks to highlight existing research gaps in comorbidity recognition and management, and to discuss future directions in precision medicine for OSAHS. By consolidating current evidence, this article underscores the need for a multidisciplinary approach to optimize patient care and improve long-term outcomes.

**Pathophysiology of OSAHS**

OSAHS arises from an interplay of **four core pathophysiologic traits (endotypes)**: (1) structural upper-airway vulnerability (anatomical compromise/high collapsibility), (2) inadequate pharyngeal dilator muscle responsiveness during sleep, (3) **unstable ventilatory control** (“high loop gain”), and (4) a **low arousal threshold** that fragments sleep and perpetuates obstruction. Contemporary phenotyping work shows these traits vary widely between individuals and often cluster, explaining

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why patients with similar apnea–hypopnea indices can respond differently to the same therapy. Recognizing these traits is central to precision management because targeted interventions (e.g., oral appliances for collapsibility, carbonic anhydrase inhibitors for loop gain, carefully selected hypnotics to raise arousal threshold) map to specific endotypes. [6–8]

**Upper airway anatomy and collapsibility**

The obstructed segment in OSAHS is typically the **retropalatal/retroglossal pharynx**, where lumen size is constrained by soft tissue bulk, craniofacial bony architecture, and lung volume–dependent caudal traction. Collapsibility is quantified by the **critical closing pressure (Pcrit)**; higher (less negative) Pcrit denotes a more collapsible airway. Obesity increases peripharyngeal fat and reduces functional residual capacity, both raising Pcrit, while craniofacial variants (e.g., retrognathia, maxillary deficiency) narrow the airway scaffold. Experimental and clinical studies demonstrate that patients with OSAHS have elevated Pcrit and that indices derived from negative expiratory pressure maneuvers can noninvasively reflect the anatomical load. [9–11]

**Ventilatory control instability and arousal threshold**

Beyond anatomy, many patients exhibit **high loop gain**—an over-responsive control system in which small disturbances in gas exchange elicit excessive ventilatory corrections that overshoot and precipitate periodic breathing and airway collapse. A **low arousal threshold** (waking easily to minor respiratory stimuli) further fragments sleep, preventing sufficient time for upper-airway dilators to recruit and stabilize the airway. Importantly, these traits are **modifiable**: raising arousal threshold with carefully chosen sedatives in selected patients, or lowering loop gain (e.g., with supplemental $CO_2$ surrogates or carbonic anhydrase inhibitors in trials), can reduce events when anatomy is not the dominant driver. [6,8,12]

**Intermittent hypoxia, sympathetic activation, and hemodynamics**

Recurrent **intermittent hypoxia (IH)** with reoxygenation is a defining stressor in OSAHS that powerfully activates the **sympathetic nervous system** via carotid-body chemoreflex pathways, elevating muscle sympathetic nerve activity and nocturnal/nearly 24-hour blood pressure. Human and translational work demonstrates that IH increases arterial pressure, augments peripheral vasoconstriction, and promotes non-dipping hypertension; CPAP attenuates these responses. Mechanistically, hypoxia-inducible factor (HIF) signaling, oxidative stress, and heightened chemosensitivity contribute to persistent sympathoexcitation and downstream cardiometabolic risk. [13–16]

**Oxidative stress, endothelial dysfunction, and inflammation**

Cycles of hypoxia–reoxygenation generate **reactive oxygen species**, impair **endothelial nitric-oxide bioavailability**, and upregulate adhesion molecules, fostering endothelial dysfunction and

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atherogenesis. Vascular biopsy and functional studies in OSAHS reveal impaired endothelial repair capacity that **improves with effective CPAP**, underscoring causality. Meta-analytic evidence confirms elevated systemic inflammatory markers (CRP, IL-6, TNF-α, ICAM/VCAM) in OSAHS, establishing a mechanistic bridge from nightly pathophysiology to daytime vascular disease burden. [5,17,18]

**Intrathoracic pressure swings and cardiac load**

Each obstructive effort against a closed airway produces large **negative intrathoracic pressure swings** (often −40 to −80 $cmH_2O$), increasing left ventricular afterload, impairing transmural coronary perfusion, and stretching atrial/ventricular walls—conditions that can trigger arrhythmias and impair systolic function. Authoritative reviews integrate these mechanical stresses with IH-driven autonomic and inflammatory pathways to explain the observed links to hypertension, atrial fibrillation, heart failure, and coronary disease. This multifactorial model also clarifies why **normalizing nocturnal mechanics with CPAP** may yield cardiovascular benefits in selected phenotypes. [19,20]

**Neuromuscular control and craniofacial contributors**

During sleep, tonic drive to the **genioglossus and other pharyngeal dilators** falls; in many patients the compensatory reflex recruitment to negative pressure is blunted or too late. Individuals with poor dilator responsiveness are particularly vulnerable despite similar BMI, whereas those with robust compensation may remain asymptomatic despite narrow anatomy. **Craniofacial morphology**—including mandibular retrusion and maxillary constriction—interacts with soft-tissue crowding and ethnicity-related patterns to determine baseline airway caliber and mechanical disadvantage. These anatomic–functional interactions help explain variable treatment responses to oral appliances, positional therapy, and hypoglossal stimulation. [21–23]

**Metabolic–hormonal perturbations amplifying risk**

OSAHS perturbs energy-balance signaling: **leptin resistance/hyperleptinemia** and **elevated ghrelin** have been reported independent of adiposity in some cohorts and may improve with CPAP, suggesting bidirectional links between sleep-disordered breathing and weight regulation. These hormonal shifts, superimposed on intermittent hypoxia and sympathetic overactivity, foster **insulin resistance** and dysmetabolism that, in turn, exacerbate upper-airway collapsibility via adiposity and reduced lung volumes—a pathogenic **feed-forward loop** connecting airway physiology with systemic metabolic risk. [24–26]

**Cardiovascular Comorbidities**

**Hypertension**

OSAHS is strongly linked to both nocturnal and daytime hypertension, with intermittent hypoxia and sympathetic activation driving sustained increases in blood pressure. Large epidemiologic studies such as the Wisconsin Sleep Cohort demonstrated a dose–response relationship between OSAHS severity

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and hypertension risk, independent of obesity. Mechanistically, heightened sympathetic tone, endothelial dysfunction, and renin–angiotensin system activation converge to produce resistant hypertension. CPAP therapy has been shown to modestly lower blood pressure, particularly in patients with uncontrolled hypertension at baseline. [27–29]

**Ischemic heart disease**

The cyclical surges in sympathetic activity, oxidative stress, and endothelial dysfunction observed in OSAHS contribute to the development and progression of coronary artery disease. Nocturnal hypoxemia has been associated with increased plaque burden, coronary calcification, and acute coronary events. Clinical studies show OSAHS independently predicts myocardial infarction, while experimental data suggest CPAP improves endothelial function and may attenuate progression of coronary disease, though mortality benefits remain under investigation. [30–32]

**Heart failure**

OSAHS and heart failure frequently coexist, with prevalence of OSAHS estimated at over 50% among patients with systolic dysfunction. The large negative intrathoracic pressures generated during obstructive events increase left ventricular afterload, while intermittent hypoxia exacerbates myocardial remodeling and dysfunction. Untreated OSAHS worsens prognosis in heart failure, whereas CPAP and adaptive servoventilation (in selected patients without predominant central sleep apnea) improve ejection fraction and symptoms, though survival benefit evidence is mixed. [33–35]

**Arrhythmias**

Arrhythmogenesis in OSAHS is mediated by autonomic imbalance, atrial stretch from pressure swings, and intermittent hypoxia–induced remodeling. Atrial fibrillation (AF) is the most strongly associated arrhythmia, with studies showing two- to fourfold higher prevalence of AF in OSAHS patients. OSAHS also predisposes to bradyarrhythmias, ventricular ectopy, and sudden cardiac death, particularly during nocturnal hours. CPAP has been reported to reduce AF recurrence after cardioversion or ablation, supporting its role in rhythm stabilization. [36–38]

**Pulmonary hypertension**

OSAHS contributes to pulmonary hypertension via hypoxic pulmonary vasoconstriction, vascular remodeling, and left-sided heart dysfunction. While often mild, pulmonary hypertension in OSAHS can worsen exercise capacity and right heart strain, particularly when combined with obesity hypoventilation or COPD overlap. CPAP therapy improves pulmonary hemodynamics in such patients, underlining the need for recognition and treatment. [39,40]

**Metabolic Comorbidities**

**Insulin resistance and type 2 diabetes mellitus**

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OSAHS is a strong independent risk factor for insulin resistance and type 2 diabetes mellitus (T2DM). Intermittent hypoxia activates sympathetic pathways, alters adipokine secretion, and promotes oxidative stress, which impair insulin signaling at hepatic and peripheral levels. Longitudinal studies, including the Wisconsin Sleep Cohort, demonstrated a dose-dependent association between OSAHS severity and incident T2DM, independent of obesity. CPAP therapy has shown modest improvements in insulin sensitivity, though effects are greater in adherent patients and those without advanced diabetes. [41–43]

**Dyslipidemia**

OSAHS contributes to atherogenic dyslipidemia characterized by elevated triglycerides, reduced HDL cholesterol, and increased small dense LDL particles. Mechanistic studies suggest that intermittent hypoxia enhances hepatic lipid biosynthesis through hypoxia-inducible factor–dependent pathways, while sympathetic activation impairs lipoprotein lipase activity. Clinical cohorts confirm that untreated OSAHS is associated with abnormal lipid profiles, and interventional trials indicate partial correction with CPAP, especially in younger, overweight males. [44–46]

**Metabolic syndrome**

The clustering of central obesity, insulin resistance, dyslipidemia, and hypertension defines the metabolic syndrome, which has a bidirectional relationship with OSAHS. The Sleep Heart Health Study reported a two- to threefold increased prevalence of metabolic syndrome in patients with OSAHS. Inflammation, oxidative stress, and hormonal imbalances (leptin resistance, ghrelin excess, cortisol dysregulation) serve as common mechanistic threads linking these conditions. Management of OSAHS with CPAP and weight reduction interventions may improve metabolic syndrome components, although lifestyle modification remains central. [47–49]

**Neurocognitive and Psychiatric Comorbidities**

Cognitive impairment and dementia risk

OSAHS is associated with deficits in attention, executive function, memory, and psychomotor speed, with longitudinal evidence linking sleep-disordered breathing to incident mild cognitive impairment (MCI) and dementia. In community cohorts of older adults, nocturnal hypoxemia and higher AHI predicted greater odds of developing MCI/dementia independent of vascular risk, while ADNI analyses suggested earlier age at MCI/Alzheimer's onset in those with SDB and hinted that CPAP might delay decline. Meta-analyses and narrative syntheses converge on intermittent hypoxia, sleep fragmentation, and vascular dysfunction as drivers of neurodegeneration, supporting a causal pathway beyond

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confounding by age or adiposity. Interventional studies also show partial neurocognitive improvement with effective CPAP, underscoring modifiability. [50–53]

**Neuroimaging and mechanistic correlates**

Multimodal MRI demonstrates structural and microstructural brain changes in OSAHS, particularly affecting hippocampal/parahippocampal regions, cerebellum, and fronto-parietal networks. Voxel-based morphometry studies report focal gray-matter loss, while diffusion tensor imaging reveals white-matter integrity deficits that can partially reverse after 6–12 months of adherent CPAP, consistent with hypoxia- and inflammation-mediated injury. These imaging findings align with autonomic dysregulation and endothelial dysfunction observed in OSAHS, offering a biological substrate for cognitive and affective symptoms and for improvement after treatment. [54–56]

**Depression and anxiety**

Prospective epidemiology indicates that sleep-related breathing disorders predict subsequent depressive symptoms, and cross-sectional clinic cohorts report high rates of depression and anxiety among untreated OSAHS patients. Mechanistically, fragmented sleep, sympathetic activation, inflammatory signaling, and quality-of-life impairment contribute to mood disturbance. Importantly, randomized and systematic reviews show that CPAP produces a modest but significant reduction in depressive symptoms—especially when baseline depression is more severe—while effects on anxiety are smaller or inconsistent, highlighting the need to screen and co-manage psychiatric comorbidity alongside PAP adherence. [57–60]

**Cerebrovascular disease and stroke**

OSAHS independently increases incident stroke risk and mortality, with large cohort data demonstrating a dose–response relationship between AHI/nocturnal hypoxemia and first-ever stroke after adjustment for conventional risk factors. Potential mechanisms include endothelial dysfunction, prothrombotic and inflammatory pathways, atrial arrhythmogenesis, and blood-pressure variability driven by intermittent hypoxia and arousals. Observational data suggest that effective CPAP use may mitigate cerebrovascular events, particularly in severe OSAHS, reinforcing the importance of recognition and treatment within secondary prevention paradigms. [61–63]

**OSA and Diabetes**

OSAHS contributes directly to the pathogenesis of insulin resistance and type 2 diabetes mellitus (T2DM). Intermittent hypoxia, sympathetic overactivity, and systemic inflammation interfere with insulin signaling at the liver, muscle, and adipose tissue. Epidemiological studies demonstrate that moderate-to-severe OSAHS increases the risk of developing T2DM, independent of obesity. The Sleep Heart Health Study confirmed a dose–response relationship between apnea–hypopnea index (AHI) and impaired glucose tolerance, strengthening causal inference. [64]

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Longitudinal studies such as the Wisconsin Sleep Cohort have shown that OSAHS severity predicts incident diabetes even after adjustment for BMI and other risk factors. Moreover, nocturnal hypoxemia has been identified as a stronger predictor of impaired glucose metabolism than AHI, highlighting the importance of oxygen desaturation burden. [65]

Interventional trials report that CPAP therapy may improve insulin sensitivity and glycemic control, though the magnitude of benefit varies by adherence, baseline glycemic status, and duration of treatment. Some randomized controlled trials found modest reductions in HbA1c among compliant patients with T2DM, while others reported neutral results, suggesting heterogeneity in response. [66]

Adipokine dysregulation plays an important role: leptin resistance, hyperleptinemia, and altered ghrelin secretion are reported in OSAHS, fostering appetite dysregulation and weight gain. These changes further worsen glucose metabolism, creating a vicious cycle between metabolic disease and airway obstruction. Targeting both sleep apnea and obesity is essential in reducing the risk of diabetes progression. [67]

**COPD and OSA (Overlap Syndrome)**

The coexistence of OSAHS and chronic obstructive pulmonary disease (COPD), known as the overlap syndrome, results in more severe nocturnal hypoxemia and worse clinical outcomes compared to either disorder alone. Epidemiological studies show that up to 15% of patients with either OSA or COPD exhibit features of both diseases. [72]

Overlap syndrome patients have increased risk of pulmonary hypertension, exacerbations, and mortality. The combined effects of airway obstruction and intermittent hypoxia amplify oxidative stress and inflammation, accelerating systemic comorbidities including cardiovascular disease. [73]

CPAP or bilevel positive airway pressure therapy improves oxygenation, reduces exacerbations, and improves survival in overlap syndrome. Observational studies consistently demonstrate improved hospitalization rates and prognosis when OSAHS is adequately treated in COPD patients. [74]

Recognizing overlap syndrome is crucial, as symptoms may be attributed to COPD alone, delaying diagnosis. Polysomnography or home sleep testing should be considered in COPD patients with disproportionate nocturnal symptoms, obesity, or resistant pulmonary hypertension. [75]

**Asthma**

OSAHS and asthma share bidirectional interactions. Asthma increases the risk of developing OSAHS due to chronic airway inflammation, corticosteroid use, and obesity. Conversely, OSAHS worsens asthma control through nocturnal hypoxemia, gastroesophageal reflux, and heightened vagal tone. [76]

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Population-based studies confirm that asthma is an independent risk factor for OSAHS, with prevalence rates of OSAHS up to 40% in severe asthmatics. These patients experience more frequent nocturnal symptoms, exacerbations, and reduced quality of life. [77]

OSAHS exacerbates asthma severity via systemic inflammation, increased leukotriene production, and airway hyperresponsiveness. Moreover, fragmented sleep worsens symptom perception and adherence to therapy, compounding disease burden. [78]

Treatment of OSAHS with CPAP has been shown to improve asthma control scores, reduce exacerbations, and enhance quality of life. This emphasizes the need for integrated management strategies targeting both conditions simultaneously. [79]

**Cancer**

Evidence linking OSAHS to cancer is emerging, with intermittent hypoxia and sleep fragmentation hypothesized to drive tumorigenesis through oxidative stress, angiogenesis, and immune dysregulation. Animal studies demonstrate that intermittent hypoxia accelerates tumor growth and metastasis via hypoxia-inducible factor (HIF) pathways and vascular endothelial growth factor (VEGF) upregulation. [80]

Population-based cohorts such as the Wisconsin Sleep Cohort and Spanish multicenter studies reported higher cancer incidence and mortality in patients with severe OSAHS, particularly in those with profound nocturnal hypoxemia. Associations were strongest for lung, colorectal, and kidney cancers. [81]

Proposed mechanisms include enhanced tumor vascularization, promotion of genomic instability, and impaired antitumor immune surveillance. Intermittent hypoxia also shifts macrophage polarization toward a pro-tumorigenic M2 phenotype, while suppressing cytotoxic T-cell activity. These findings suggest OSAHS may act as a systemic oncogenic stressor. [82]

Interventional data remain limited, but small studies indicate that adherent CPAP use may normalize oxidative stress markers and restore immune balance. While causality is not yet firmly established, recognition of cancer risk in OSAHS underscores the importance of early diagnosis and effective treatment. [83]

**Obstructive Sleep Apnea and the Reproductive System**

**Male reproductive health**

OSAHS is associated with hypogonadism, erectile dysfunction, and reduced fertility. Mechanistically, intermittent hypoxia and sleep fragmentation impair hypothalamic–pituitary–gonadal axis function,

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leading to reduced testosterone secretion. Clinical studies confirm lower morning testosterone levels in men with untreated OSAHS, independent of age and obesity. [84]

Erectile dysfunction is highly prevalent in OSAHS, affecting up to 60% of men in severe cases. Endothelial dysfunction, sympathetic overactivity, and decreased nitric oxide bioavailability are implicated. CPAP therapy has been shown to improve erectile function and testosterone levels, though effects vary with treatment adherence. [85]

**Female reproductive health**

In women, OSAHS contributes to menstrual irregularities, subfertility, and complications during pregnancy such as gestational diabetes and preeclampsia. Polycystic ovary syndrome (PCOS), a common reproductive endocrine disorder, shows a strong association with OSAHS, with prevalence estimates up to 70% in obese women with PCOS. Intermittent hypoxia worsens insulin resistance and hyperandrogenism in this group. [86]

Pregnancy further increases OSAHS risk due to weight gain, upper-airway edema, and hormonal changes. Untreated OSAHS in pregnancy is associated with gestational hypertension, preeclampsia, and fetal growth restriction. Screening and treatment of sleep-disordered breathing in high-risk pregnancies may improve maternal–fetal outcomes. [87]

**Fertility implications**

Both male and female infertility have been linked to OSAHS through hormonal and metabolic pathways. In men, reduced testosterone and semen abnormalities have been reported, while in women, menstrual irregularity and PCOS contribute to impaired fecundity. Although evidence is still evolving, multidisciplinary management addressing OSAHS may enhance reproductive outcomes. [88]

**Conclusion**

Obstructive sleep apnea–hypopnea syndrome (OSAHS) has evolved from being viewed merely as a disorder of sleep fragmentation and snoring to being recognized as a **multisystem disease with profound clinical consequences**. Its pathophysiology—anchored in upper airway collapsibility, ventilatory instability, intermittent hypoxia, and intrathoracic pressure swings—creates a cascade of autonomic, metabolic, and inflammatory disturbances that extend well beyond the respiratory system. The evidence reviewed highlights how OSAHS contributes to the development and progression of **cardiovascular, metabolic, neurocognitive, respiratory, renal, oncologic, and reproductive disorders**. These comorbidities not only worsen quality of life but also substantially increase morbidity and mortality. Importantly, the severity of nocturnal hypoxemia, oxygen desaturation burden, and adherence to therapy strongly influence outcomes, underscoring the clinical need for early detection and individualized management.

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Continuous positive airway pressure (CPAP) remains the cornerstone of therapy, with proven benefits in blood pressure reduction, glycemic control, mood stabilization, and prevention of cardiovascular and respiratory complications. Yet, challenges persist in patient adherence, identification of high-risk phenotypes, and defining the long-term effects of treatment on outcomes such as cancer progression and fertility. Adjunctive strategies, including weight loss interventions, pharmacologic therapies targeting ventilatory control, and surgical or device-based approaches, further broaden the management landscape.

Future directions must focus on **precision medicine approaches**, integrating phenotypic and endotypic characterization to tailor interventions. The development of biomarkers to stratify risk, coupled with robust longitudinal studies, will help clarify causal relationships between OSAHS and its systemic consequences. Equally important is the integration of OSAHS management into broader multidisciplinary care models that address cardiometabolic risk, renal protection, psychiatric well-being, and reproductive health.

In summary, OSAHS is a **systemic disorder with wide-ranging implications across multiple organ systems**. Recognizing its comorbidities, implementing effective treatment, and closing existing research gaps are essential steps in reducing its burden on patients and healthcare systems alike. The future of OSAHS management lies in early detection, personalized treatment strategies, and coordinated care that targets not only the airway but also the systemic pathways it disrupts.

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