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OBSTRUCTIVE SLEEP APNEA: PATHOPHYSIOLOGY, CLINICAL CONSEQUENCES, AND CONTEMPORARY MANAGEMENT

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ABSTRACT:
Obstructive sleep apnea is a common sleep-related breathing disorder characterised by recurrent episodes of partial or complete upper airway obstruction during sleep, resulting in intermittent hypoxaemia, hypercapnia, and sleep fragmentation. These disturbances lead to excessive daytime sleepiness, impaired neurocognitive function, reduced quality of life, and increased risk of cardiovascular, metabolic, and cerebrovascular disease. Despite its substantial global burden, OSA remains markedly underdiagnosed. This Review summarises current evidence on the epidemiology, aetiology, pathophysiology, diagnosis, and management of OSA in adult and paediatric populations. OSA arises from a complex interaction between anatomical airway vulnerability, impaired neuromuscular control, ventilatory instability, obesity, ageing, and genetic susceptibility. Recurrent upper airway collapses trigger sympathetic activation, endothelial dysfunction, oxidative stress, and systemic inflammation, providing mechanistic links to cardiometabolic morbidity. Diagnosis relies on clinical assessment supported by objective sleep testing, with in-laboratory polysomnography as the diagnostic gold standard. Management is individualised and includes lifestyle modification, positive airway pressure therapy, oral appliances, surgical interventions, and emerging neurostimulation approaches. Continuous positive airway pressure remains the first-line treatment for moderate to severe disease, offering substantial symptomatic benefit despite ongoing challenges with adherence.

KEYWORDS: Obstructive sleep apnea; Sleep-disordered breathing; Intermittent hypoxia; Apnea–hypopnea index; Polysomnography; Continuous positive airway pressure

INTRODUCTION:

Obstructive sleep apnea (OSA) is a prevalent sleep-related breathing disorder characterized by recurrent episodes of partial or complete upper airway obstruction during sleep, resulting in intermittent hypoxia, hypercapnia, and repeated sleep fragmentation. These events disrupt normal sleep architecture and lead to excessive daytime sleepiness, impaired cognitive function, reduced quality of life, and increased risk of cardiovascular and metabolic morbidity. OSA represents the most common form of sleep-disordered breathing and constitutes a significant public health concern due to its high prevalence, underdiagnosis, and association with adverse clinical outcomes. [1]

According to the third edition of the *International Classification of Sleep Disorders* (ICSD-3), OSA is classified under sleep-related breathing disorders and is subdivided into two major categories: adult obstructive sleep apnea and paediatric obstructive sleep apnea. This distinction reflects important differences in epidemiology, pathophysiology, clinical presentation, and management between adults and children. In adults, OSA is commonly associated with obesity, craniofacial abnormalities, and age-related reductions in upper airway muscle tone. In contrast, paediatric OSA is frequently linked to Adenotonsillar hypertrophy, craniofacial developmental factors, and, increasingly, childhood obesity. [2]

SIGNS AND SYMPTOMS:

OSA presents with a broad spectrum of nocturnal and daytime symptoms, reflecting recurrent upper airway obstruction, intermittent hypoxia, and sleep fragmentation. The most characteristic nocturnal manifestation is loud, habitual snoring, often punctuated by witnessed apneas, choking, gasping, or snorting sounds during sleep. These episodes are frequently followed by brief arousals, resulting in fragmented and non-restorative sleep. Other common nighttime symptoms include restless sleep, frequent awakenings, nocturia, excessive sweating, and gastroesophageal reflux.

Daytime symptoms are largely consequences of disrupted sleep architecture and chronic hypoxemia. Excessive daytime sleepiness is the hallmark feature in adults, ranging from mild fatigue to involuntary sleep episodes during routine activities such as reading, watching television, or even conversing. Cognitive impairment is common, including poor concentration, reduced vigilance, memory difficulties, and executive dysfunction. Affected individuals may also report morning headaches, dry mouth or throat on awakening, mood disturbances such as irritability, anxiety, or depression, and reduced quality of life. Less frequent symptoms include bruxism, erectile dysfunction, unexplained weight gain, and elevated blood pressure or heart rate.

OSA symptoms may be transient or situational in some individuals. Acute upper respiratory infections, tonsillitis, nasal congestion, or conditions such as infectious mononucleosis can temporarily exacerbate upper airway obstruction. Alcohol and sedative medications may also precipitate or worsen OSA episodes by reducing upper airway muscle tone and impairing arousal responses.

In adults, long-standing untreated OSA is associated with neurocognitive consequences, including impaired working memory and executive function, likely due to recurrent hypoxia and sleep disruption. Neuroimaging studies have demonstrated structural and functional brain changes, particularly involving the hippocampus and frontal cortex, and OSA has been linked to an increased risk of neurodegenerative disorders. In contrast, children with OSA often present differently. Excessive daytime sleepiness is uncommon; instead, children may appear hyperactive, irritable, inattentive, or exhibit behavioural and learning difficulties. Severe paediatric OSA may result in failure to thrive, reflecting increased work of breathing and reduced caloric intake. Adenotonsillar hypertrophy is the most common cause in children, though obesity is an increasingly important contributor, particularly in adolescents. [3-4]

AETIOLOGY:

The aetiology of OSA is multifactorial and involves a complex interaction between anatomical, neuromuscular, physiological, genetic, and lifestyle-related factors. Pharyngeal narrowing and closure during sleep occur due to sleep-related reductions in ventilatory drive, diminished upper airway muscle tone, and predisposing anatomical characteristics, all of which contribute significantly to upper airway obstruction during sleep.[5-6]

- **Anatomical and Structural Factors:** Anatomical abnormalities that reduce the calibre of the upper airway are central to the development of OSA. These include increased neck circumference, excess soft tissue deposition, craniofacial skeletal variations, and surrounding vascular or soft tissue structures that increase extraluminal pressure on the pharynx. Such factors enhance pharyngeal collapsibility and reduce the space available for airflow, particularly during sleep. Common anatomical risk factors include micrognathia, retrognathia, mandibular hypoplasia, facial elongation, inferior displacement of the hyoid bone, and Adenotonsillar hypertrophy. A narrow maxilla may further compromise airway patency by reducing nasal airway dimensions, often manifesting as nasal congestion that worsens in the supine position. Enlarged tonsils, especially in children but also in adults, are well-recognized contributors to OSA, and

tonsillectomy may provide partial or complete symptom relief, supporting their role in disease pathogenesis.

- **Neuromuscular Control and Upper Airway Muscle Tone:** Maintenance of upper airway patency depends on the activity of pharyngeal dilator muscles, particularly the genioglossus. During sleep, physiological reductions in neuromuscular tone increase susceptibility to partial or complete airway collapse. This effect is accentuated in individuals with structural airway narrowing or impaired neuromuscular control. Loss of muscle tone is more pronounced with advancing age and may be temporarily exacerbated by alcohol consumption, sedative-hypnotic medications, and other central nervous system depressants. Permanent neuromuscular impairment may result from traumatic brain injury, neuromuscular disorders such as myasthenia gravis, or neurological conditions affecting respiratory control.
- **Obesity and Fat Distribution:** Obesity is one of the strongest and most consistent risk factors for OSA, with a clear linear relationship between body mass index (BMI) and disease severity. Increased fat deposition around the neck and upper airway elevates extrinsic pressure on the pharynx, promoting airway collapse during sleep. Central fat distribution appears particularly important in increasing OSA risk. However, OSA is not confined to obese individuals. Patients with normal BMI may also develop OSA, suggesting that factors such as craniofacial anatomy, muscle mass, or reduced neuromuscular tone can independently contribute to airway collapsibility. These observations indicate that while obesity is a major contributor, it is not an essential prerequisite for OSA.
- **Age, Sex, and Hormonal Influences:** Advancing age is associated with reduced upper airway muscle tone and diminished neurological control of respiration, increasing vulnerability to airway obstruction during sleep. Male gender is a well-established risk factor, likely related to differences in fat distribution and upper airway anatomy. Women experience OSA less frequently during reproductive years, possibly due to the protective effects of progesterone on respiratory drive and airway muscle tone. However, prevalence increases after menopause and approaches that of men. Pregnancy also increases OSA risk due to hormonal changes, weight gain, and fluid retention.
- **Sleep Position:** Supine sleeping is an important positional risk factor for OSA. Gravity-induced posterior displacement of the tongue and soft tissues during supine sleep, combined with reduced muscle tone, increases airway obstruction. This effect is often compounded in individuals with neck obesity.
- **Lifestyle and Medication-Related Factors:** Lifestyle factors significantly influence OSA risk. Alcohol, sedatives, hypnotics, and other medications that increase sleepiness exacerbate airway collapse by relaxing upper airway muscles and suppressing protective arousal responses. Smoking increases OSA risk through upper airway inflammation, oedema, and fluid retention, although this risk appears reversible with smoking cessation. Exposure to cigarette smoke in children may also contribute to OSA by promoting lymphoid tissue hypertrophy. Allergic rhinitis and asthma further increase risk by causing chronic nasal obstruction and Adenotonsillar enlargement.
- **Associated Medical Disorders:** OSA is frequently associated with several medical conditions, although much of the evidence is derived from observational studies. Endocrine disorders such as diabetes mellitus, metabolic syndrome, hypothyroidism, and acromegaly are commonly linked to OSA. Neurological disorders, including stroke and spinal cord injury, as well as conditions such as congestive heart failure, atrial fibrillation, and obesity hypoventilation syndrome, are also strongly associated.

- **Genetic and Craniofacial Syndromes:** A genetic predisposition to OSA is suggested by familial clustering and the identification of several candidate genes, including *FTO*, *DLEU1*, *DLEU7*, *CTSF*, *MSRB3*, and *TRIM66*. Genetic influences may act directly or indirectly through intermediate phenotypes such as obesity, craniofacial structure, and ventilatory control. Certain craniofacial syndromes markedly increase OSA risk. Down syndrome is strongly associated with OSA due to hypotonia, macroglossia, and a narrow nasopharynx, with more than half of affected individuals experiencing OSA. Other syndromes, including Pierre Robin sequence and Treacher Collins syndrome, predispose to airway obstruction due to mandibular hypoplasia and reduced airway dimensions.
- **Postoperative and Special Situations:** OSA may also occur as a postoperative complication, particularly following pharyngeal flap surgery for velopharyngeal insufficiency, where surgically induced airway obstruction may impair respiration during sleep. Additionally, patients with known OSA are at increased risk of perioperative complications, emphasizing the importance of preoperative screening and risk mitigation.

PATHOPHYSIOLOGY:

OSA is characterized by recurrent episodes of partial or complete upper airway obstruction during sleep, resulting in intermittent hypoxia, hypercapnia, sleep fragmentation, and wide-ranging systemic effects. The pathophysiology of OSA is complex and multifactorial, involving anatomical airway narrowing, impaired neuromuscular control, altered ventilatory mechanics, and downstream cardiovascular, metabolic, and inflammatory consequences. [7-9]

Upper Airway Collapse and Ventilatory Mechanics: The fundamental mechanism underlying OSA is the collapse of a normally patent upper airway during sleep, most commonly behind the tongue and epiglottis. During wakefulness, airway patency is maintained by tonic activity of the pharyngeal dilator muscles. However, sleep—particularly rapid eye movement (REM) sleep—is associated with a marked reduction in upper airway muscle tone. This loss of neuromuscular support predisposes the airway to collapse, especially in individuals with anatomically narrowed airways. Upper airway obstruction is often triggered by negative collapsing pressure generated during inspiration. Additionally, progressive expiratory narrowing, particularly in the retropalatal region, contributes significantly to airway instability. The pressure–flow relationship through the upper airway can be explained using the collapsible tube model, wherein airflow limitation increases as intraluminal pressure falls below surrounding tissue pressure. The severity of airway narrowing during sleep correlates with body mass index (BMI), indicating that both anatomical factors (such as fat deposition) and neuromuscular control influence airway collapsibility.

Sleep Stage–Dependent Airway Instability: As sleep deepens from light non–rapid eye movement (NREM) sleep to slow-wave sleep and REM sleep, upper airway muscle tone progressively declines. During REM sleep, muscle tone in the throat, neck, and most skeletal muscles is nearly abolished. This allows posterior displacement of the tongue and relaxation of the soft palate, leading to reduced airway patency and impaired ventilation. When airflow is restricted, blood oxygen saturation gradually decreases while carbon dioxide levels rise. If hypoxemia or increased respiratory effort reaches a critical threshold, protective neurological mechanisms trigger brief arousals from sleep. These arousals restore muscle tone, reopen the airway, and normalize breathing. However, repeated arousals fragment sleep architecture and impair restorative sleep. In severe OSA, this cycle of airway collapse, hypoxia, arousal, and recovery may occur dozens of times per hour.

Sympathetic Nervous System Activation: Sleep is normally characterized by parasympathetic dominance. In OSA, recurrent hypoxia and hypercapnia stimulate peripheral and central chemoreceptors, leading to sustained sympathetic nervous system activation. Importantly, this heightened sympathetic activity persists not only during sleep but also while awake. Studies have demonstrated that sympathetic overactivity is significantly increased in patients with OSA compared to obese individuals without OSA, suggesting that OSA itself is a primary driver of autonomic dysregulation. Chronic sympathetic activation stimulates the renin–angiotensin–aldosterone system, resulting in elevated levels of angiotensin II and aldosterone. These hormonal changes promote vasoconstriction and renal sodium and water retention, contributing to the high prevalence of systemic hypertension observed in OSA patients.

Endothelial Dysfunction: Endothelial dysfunction is a key pathophysiological consequence of OSA. Under normal conditions, endothelial cells regulate vascular tone through the release of vasodilatory substances such as nitric oxide (NO). In OSA, intermittent hypoxia and oxidative stress impair endothelial function, leading to reduced NO bioavailability and increased vasoconstrictor activity. Elevated circulating levels of endothelin-1, a potent vasoconstrictor, have been observed in patients with untreated OSA. Importantly, treatment with continuous positive airway pressure has been shown to reduce endothelin-1 levels, improve endothelial function, and lower blood pressure, highlighting the reversible nature of these vascular abnormalities.

Systemic Inflammation and Oxidative Stress: OSA is increasingly recognized as a state of chronic low-grade systemic inflammation. Repeated cycles of hypoxia and reoxygenation promote the generation of reactive oxygen species, leading to oxidative stress. Elevated levels of inflammatory biomarkers, including interleukin-6 (IL-6) and tumour necrosis factor-alpha (TNF-α), are consistently observed in OSA patients. The combination of oxidative stress and inflammation contributes to endothelial injury, atherosclerosis, and increased cardiovascular risk. These mechanisms help explain the strong association between OSA and adverse cardiovascular outcomes, including coronary artery disease, stroke, and increased all-cause mortality.

Metabolic Dysfunction: OSA is closely associated with metabolic abnormalities, particularly insulin resistance and type 2 diabetes mellitus (T2DM). Large population studies have demonstrated a higher prevalence of impaired glucose tolerance, elevated fasting glucose levels, and diabetes in individuals with OSA. Dyslipidaemia, characterized by increased low-density lipoprotein cholesterol, triglycerides, and total cholesterol, is also commonly observed. Sleep fragmentation and intermittent hypoxia disrupt glucose metabolism and lipid regulation, further increasing cardiovascular risk. The coexistence of OSA with metabolic syndrome significantly amplifies morbidity and mortality.

Integrated Pathophysiological Models: The mechanisms underlying OSA have been debated across specialties. Pulmonologists and neurologists emphasize neuromuscular dysfunction, age-related muscle tone loss, and ventilatory control instability. Otorhinolaryngologists focus on structural airway narrowing due to enlarged tonsils, soft palate abnormalities, nasal obstruction, or epiglottic collapse. Oral and maxillofacial surgeons highlight mandibular hypoplasia and glossoptosis as primary anatomical drivers of airway obstruction.

DIAGNOSIS AND EVALUATION:

Clinical assessment of suspected OSA begins with a detailed evaluation of sleep quality, nocturnal symptoms, and daytime functional impairment. Self-reported history should be complemented by information from bed partners or witnesses, as patients are often unaware of abnormal nocturnal breathing. Witnesses should be questioned regarding habitual loud snoring, observed apneas, snorting or gasping episodes, choking, and motor restlessness during sleep. Other common nocturnal features include

fragmented sleep, frequent awakenings, nocturia, and unrefreshing sleep. Daytime manifestations typically include excessive sleepiness, fatigue, poor concentration, impaired vigilance, dry mouth or throat on awakening, and reduced cognitive performance. Subjective sleepiness may be quantified using the Epworth Sleepiness Scale (ESS); however, it is important to recognize that many patients with OSA do not report excessive sleepiness, and similar symptoms may arise from mood disorders or other sleep conditions. [10]

Assessment of safety risks is a critical component of the clinical evaluation. Excessive sleepiness significantly increases the risk of motor vehicle and occupational accidents. A history of prior accidents or near-miss events strongly predicts future risk. Individuals holding commercial or heavy vehicle licenses require stricter evaluation, and specialist input may be necessary. Patients bear responsibility for notifying relevant authorities if diagnosed with OSA, in accordance with local regulations.

Physical examination focuses on identifying predisposing anatomical and anthropometric factors. Measurement of body mass index, waist circumference, and neck circumference provides an estimate of obesity-related risk. Upper airway examination assesses for craniofacial abnormalities such as retrognathia, pharyngeal crowding, tonsillar hypertrophy, and nasal obstruction. The Friedman Tongue Position (FTP) is commonly used to grade airway narrowing with the tongue in a neutral position and provides useful information beyond the Mallampati score.

The gold standard for diagnosing OSA is overnight, in-laboratory Level 1 polysomnography (PSG). PSG comprehensively evaluates sleep architecture and respiratory physiology using electroencephalography (EEG), electrooculography, electromyography, electrocardiography, pulse oximetry, oronasal airflow sensors, and respiratory inductance plethysmography. Respiratory events are scored using four primary signals: airflow (oronasal thermal sensor and nasal pressure transducer), respiratory effort, and oxygen saturation. Apnea is defined by a ≥90% reduction in airflow lasting ≥10 seconds. Hypopnea is defined as a ≥30% reduction in airflow for ≥10 seconds accompanied by either ≥3–4% oxygen desaturation or an EEG-defined arousal, according to American Academy of Sleep Medicine (AASM) criteria. Based on respiratory effort, apneas are classified as obstructive, central, or mixed.

The apnea–hypopnea index (AHI), representing the number of apneas and hypopneas per hour of sleep, is used to quantify disease severity. In adults, OSA is classified as mild (5–15 events/hour), moderate (15–30 events/hour), or severe (>30 events/hour). The diagnosis of OSA syndrome is established when AHI is ≥5 events/hour in the presence of symptoms or when AHI (or respiratory disturbance index, RDI) is ≥15 events/hour irrespective of symptoms.

Home sleep apnea testing (HSAT) or portable monitoring has emerged as an accessible and cost-effective alternative for selected patients with a high pretest probability of moderate-to-severe OSA and without significant cardiopulmonary or neurological comorbidities. These devices must record, at a minimum, airflow, respiratory effort, and oxygen saturation and should be administered under the supervision of an AASM-accredited sleep program. Because HSAT lacks EEG monitoring, total recording time is used instead of total sleep time, potentially underestimating disease severity. Consequently, results are reported as the respiratory event index (REI) rather than AHI. Negative or inconclusive HSAT results in high-risk patients warrant confirmatory in-laboratory PSG.

MANAGEMENT AND TREATMENT:

The management of OSA is multifaceted and aims to reduce upper airway obstruction during sleep, alleviate symptoms such as excessive daytime sleepiness, and prevent long-term cardiovascular, metabolic, and neurocognitive complications. Treatment strategies include behavioural and lifestyle interventions, positive airway pressure therapies, oral appliances, surgical approaches, neurostimulation

techniques, and adjunct pharmacological therapy. Choice of treatment depends on disease severity, anatomical factors, patient preference, comorbidities, and treatment tolerance. [11-13]

Behavioural and Lifestyle Modifications: Lifestyle modification is the cornerstone of OSA management, particularly in overweight and obese individuals. Obesity is a major risk factor for OSA due to fat deposition around the upper airway, reduced lung volumes, and increased pharyngeal collapsibility. Weight loss has consistently been shown to reduce apnea–hypopnea index (AHI) and improve symptoms. Even a 5–10% reduction in body weight can significantly decrease OSA severity. Intensive lifestyle interventions have demonstrated reductions in AHI, inflammatory markers, blood glucose, and lipid levels. Consequently, weight loss is recommended as first-line therapy in patients with mild asymptomatic OSA and as adjunctive therapy across all severities. Avoidance of alcohol, smoking, and sedative medications is strongly advised, as these agents reduce upper airway muscle tone and impair arousal mechanisms, thereby worsening airway collapse. Positional therapy is beneficial in patients with positional OSA, as airway collapsibility is greater in the supine position. Techniques such as positional pillows, backpacks, or vibration-based positional trainers that discourage supine sleep can reduce AHI, although their efficacy is inferior to CPAP. Physical exercise, even in the absence of significant weight loss, has been shown to improve OSA severity by enhancing ventilatory control and upper airway muscle function. Emerging evidence also supports myofunctional therapy and pharyngeal muscle training, particularly in patients with mild OSA, through strengthening of the tongue and oropharyngeal muscles.

Positive Airway Pressure Therapy: Continuous positive airway pressure (CPAP) is the first-line treatment for moderate to severe obstructive sleep apnea and works by maintaining upper airway patency through constant positive pressure. It effectively reduces the apnea–hypopnea index, improves daytime sleepiness, quality of life, blood pressure, and neurocognitive function. However, long-term adherence is a major challenge, with high discontinuation rates. Strategies such as patient education, behavioural support, and optimized mask fitting can improve compliance. Alternative modalities, including bilevel and auto-titrating CPAP, may benefit patient's intolerant to standard CPAP. Although CPAP has not consistently reduced major cardiovascular events, it remains essential for symptom relief and functional improvement.

Oral Appliance Therapy: Oral appliances, particularly mandibular advancement devices (MADs), represent an effective alternative for patients with mild to moderate OSA or those intolerant of CPAP. MADs advance the mandible and tongue forward, increasing upper airway space and reducing collapsibility. Although less effective than CPAP in reducing AHI, higher adherence rates often result in comparable improvements in daytime sleepiness and blood pressure. MADs are most suitable for patients with lower BMI, mild to moderate disease, and adequate dentition. They are non-invasive, reversible, and generally well tolerated. Tongue retaining devices and hybrid appliances combining mandibular advancement with tongue stabilization are also available, though acceptance varies. In children, evidence supporting routine use of oral appliances is limited, and their role is mainly adjunctive in selected craniofacial abnormalities.

Surgical Management: Surgical intervention is not considered first-line therapy for OSA in adults but may be appropriate for selected patients who fail or cannot tolerate conservative treatments. Surgery aims to modify upper airway anatomy to reduce obstruction and is tailored to the site of collapse. Uvulopalatopharyngoplasty (UPPP), with or without tonsillectomy, is the most commonly performed procedure and can reduce AHI and improve daytime sleepiness in selected patients. However, outcomes vary, and long-term efficacy may be reduced by weight gain. Maxillomandibular advancement (MMA) is among the most effective surgical options, particularly in patients with craniofacial abnormalities,

producing substantial and sustained reductions in AHI. Other procedures include septoplasty, turbinate reduction, tongue base reduction, hyoid suspension, genioglossus advancement, and bariatric surgery in morbidly obese patients. In children, adenotonsillectomy remains the treatment of choice, particularly in those with Adenotonsillar hypertrophy, often resulting in resolution of OSA.

Hypoglossal Nerve Stimulation: Hypoglossal nerve stimulation (HNS) is an emerging therapy for patients with moderate to severe OSA who are intolerant of CPAP. This implantable device stimulates the hypoglossal nerve during inspiration, resulting in tongue protrusion and airway stabilization. Clinical trials have demonstrated sustained reductions in AHI, improved sleep quality, and reduced snoring over long-term follow-up. Ideal candidates include patients with moderate to severe OSA, BMI $\leq$32 kg/m², and absence of complete concentric palatal collapse, confirmed by drug-induced sleep endoscopy.

Pharmacological Therapy: Currently, no pharmacological agent is approved to treat the underlying airway obstruction in OSA. Drug therapy is primarily limited to symptom management or adjunctive treatment in selected populations. Central nervous system stimulants such as modafinil, armodafinil, and solriamfetol are FDA-approved for excessive daytime sleepiness associated with OSA, particularly in patients with residual sleepiness despite adequate CPAP use. These agents improve wakefulness by modulating dopamine and norepinephrine pathways but do not treat the primary pathophysiology of OSA. Careful monitoring is required due to potential abuse risk and cardiovascular effects. Anti-inflammatory therapies such as nasal corticosteroids and leukotriene antagonists have shown benefit in children with mild OSA related to Adenotonsillar hypertrophy and in adults with concurrent allergic rhinitis. Carbonic anhydrase inhibitors like acetazolamide may reduce AHI in selected patients with high ventilatory loop gain, though side effects limit routine use

EPIDEMIOLOGY OF OBSTRUCTIVE SLEEP APNEA

OSA is a highly prevalent yet markedly underdiagnosed sleep disorder, particularly affecting middle-aged and elderly populations worldwide. A large literature-based analysis using the American Academy of Sleep Medicine diagnostic criteria estimated that approximately 936 million adults aged 30–69 years have mild-to-severe OSA globally, with 425 million experiencing moderate-to-severe disease. Population-based studies indicate that OSA prevalence ranges from 9% to 38% in adults, increasing dramatically with age and reaching up to 80% in individuals older than 65 years. [14-15]

Men are affected more frequently than women; however, the prevalence in women rises after menopause, and mortality may be higher among women. OSA prevalence is strongly associated with obesity, with even modest weight gain significantly increasing disease risk. Ethnic differences have been reported, with higher prevalence observed in Hispanic, Black, and Asian populations. Despite its high burden, OSA remains substantially underdiagnosed, with estimates suggesting that over 80% of affected individuals are unaware of their condition, contributing to increased cardiovascular risk, metabolic disease, and healthcare costs.

CONCLUSION:

Obstructive sleep apnea is a common, multifactorial disorder with systemic pathophysiological effects that substantially increase cardiovascular, metabolic, and public health risk. Early recognition, improved screening, and individualized management—despite adherence challenges with CPAP—are essential to reduce long-term morbidity, mortality, and socioeconomic burden.

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