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Review

Obstructive sleep apnea and comorbidities: a current perspective from diagnosis to treatment

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ABSTRACT

Obstructive sleep apnea syndrome (OSAS) is a significant health condition characterized by recurrent upper airway obstructions during sleep, with widespread systemic consequences. This review aims to present a comprehensive overview of OSAS, encompassing its pathophysiological foundations, risk factors, clinical manifestations, associated comorbidities, and contemporary diagnostic and therapeutic strategies. OSAS not only impairs sleep quality but also adversely affects cardiovascular, metabolic, neurocognitive, and psychological health, thereby reducing both lifespan and quality of life. While polysomnography remains the gold standard for diagnosis, home sleep tests and subjective assessment tools are increasingly utilized in clinical practice. Treatment approaches vary depending on disease severity and individual characteristics, ranging from lifestyle modifications and positive airway pressure devices to oral appliances and surgical interventions. In recent years, promising innovations such as neurostimulation and pharmacotherapy have emerged in OSAS management. This review highlights the multidimensional impact of OSAS at both individual and societal levels, emphasizing the need for early diagnosis and integrated treatment strategies.

Keywords: Obstructive sleep apnea, OSAS, comorbidities, diagnosis and treatment

INTRODUCTION

Obstructive sleep apnea syndrome (OSAS) is a prevalent sleep disorder characterized by recurrent complete (apnea) or partial (hypopnea) obstruction of the upper airway during sleep. Despite ongoing respiratory effort during these episodes, airflow is reduced or completely interrupted, leading to oxygen desaturation, sleep fragmentation, and non-restorative sleep.[1] OSAS is not limited to snoring and excessive daytime sleepiness; it exerts broad adverse effects on cardiovascular, metabolic, and neurocognitive systems, significantly reducing both quality of life and survival.[2] In pediatric populations, the condition is particularly important due to its association with growth retardation and behavioral problems.[3]

Epidemiological data indicate that the prevalence of OSAS is increasing globally. A comprehensive meta-analysis in the elderly population reported a global prevalence exceeding 35% in this age group.[4] The rates are similarly high among adults. A recent study conducted in the United States revealed that approximately one in four adults meets diagnostic criteria for OSAS, yet 80% of these individuals remain undiagnosed.[5] In a large-scale cohort study among middle-aged individuals, the prevalence was found to be 57.7% in men and 41.7% in women, with a significant association with cardiometabolic comorbidities.[6] Importantly, not only prevalence but also access to and adherence with treatment constitute critical aspects of OSAS epidemiology. A recent meta-analysis in elderly individuals demonstrated that continuous positive airway pressure (CPAP) therapy significantly reduces both mortality and morbidity.[7]

Moreover, OSAS is not merely an individual concern but a major public health issue directly impacting population health. When left undiagnosed and untreated, it contributes to an increased burden of cardiovascular diseases, diabetes, obesity, depression, and cognitive impairments.[10] Current literature underscores the importance of prioritizing OSAS in public health policies and enhancing access to diagnostic and therapeutic services.[8] Additionally, global warming due to climate change has been proposed to exacerbate OSAS prevalence and severity by impairing sleep quality, potentially intensifying its health burden in the coming years.[9,10] Collectively, these findings highlight that OSAS should be addressed as a critical public health concern, with attention to its clinical, societal, environmental, and economic dimensions.

PATHOPHYSIOLOGY

The pathophysiology of OSAS arises from the interplay between anatomical and neurophysiological factors. Structural characteristics of the upper airway are among the most critical determinants of the disorder. A narrow pharyngeal airway, hypertrophy of the soft palate and uvula, macroglossia (enlarged tongue), or retrognathia (posteriorly

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positioned mandible), are craniofacial anomalies that increase the risk of upper airway collapse during sleep.[11-13] In children, adenotonsillar hypertrophy is one of the most common anatomical substrates for OSAS.[3] These structural narrowings, when combined with the reduction of muscle tone during sleep, lead to repeated airway obstructions.[14]

The decrease in muscle tone is particularly pronounced during REM sleep due to pharyngeal muscle relaxation. Under normal circumstances, these muscles maintain airway patency through tonic activity; however, reduced activation during sleep, together with anatomical narrowing, facilitates apneic and hypopneic episodes. Therefore, OSAS is not only an anatomical problem but also a disorder associated with impaired neuromuscular control.[11-13]

This process not only diminishes sleep quality but also initiates systemic biological effects. Recurrent hypoxic episodes lead to oxygen desaturation and carbon dioxide retention, triggering oxidative stress and inflammatory responses. Endothelial dysfunction, heightened sympathetic nervous system activity, and hormonal imbalances result in the development of multifactorial outcomes such as hypertension, atherosclerosis, and insulin resistance.[11,13] Thus, OSAS transcends being merely a sleep disorder characterized by snoring, evolving into a disease with multisystemic consequences.

One of the most powerful determinants in the pathophysiology of OSAS is obesity and increased neck circumference. Fat accumulation in the neck exerts external pressure, narrowing the upper airway, while visceral adiposity adversely affects respiratory mechanics. This mechanical narrowing, when combined with metabolic disturbances such as leptin resistance, insulin resistance, and chronic inflammation, exacerbates the progression of OSAS.[1,11,16,23] Indeed, clinical studies have demonstrated a strong correlation between weight gain and OSAS severity in obese individuals, with neck circumference serving as an important prognostic indicator.[6,23]

Recent studies have shown that OSAS cannot be explained by anatomical factors alone; non-anatomical mechanisms, such as impaired central respiratory control, alterations in sleep architecture, and differences in arousal threshold, also play a critical role in disease phenotypes.[14] Therefore, understanding and managing OSAS requires a comprehensive approach that considers not only anatomical features but also neurophysiological and metabolic mechanisms.

In conclusion, the pathophysiology of OSAS is the result of a complex interaction involving anatomical airway narrowing, reduced muscle tone, obesity-related compression, recurrent hypoxia, and inflammatory-oxidative processes. Accordingly, the disorder should be regarded as a comprehensive clinical entity that significantly affects not only sleep but also long-term health.[11-14,16,23]

RISK FACTORS

OSAS is a complex disorder that does not arise from a single cause, but rather results from the convergence of multiple interrelated risk factors (**Figure 1**). While each of these factors is significant on its own, they often act synergistically, collectively increasing the overall burden of disease.

One of the strongest determinants of OSAS is obesity. Particularly, the accumulation of adipose tissue in the neck region leads to narrowing of the upper airway and significantly increases the risk of apnea development.[23] Notably, weight gain not only creates mechanical pressure but also facilitates the development of OSAS through metabolic and hormonal pathways. Therefore, obesity is considered a multifaceted factor that aggravates the risk profile via both direct and indirect mechanisms.[11,13]

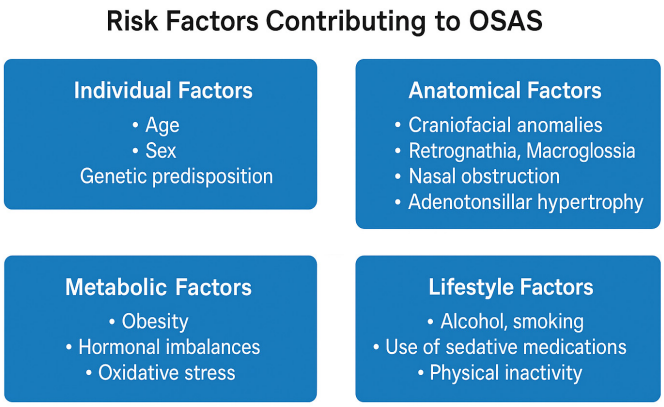

Figure 1. Factors contributing to the development of obstructive sleep apnea syndrome

Age and sex also play a significant role in determining OSAS risk. The prevalence of the disorder increases notably in middle age and beyond, with men being at higher risk compared to women.[4,6] However, after menopause, the risk in women also rises significantly due to hormonal changes. This phenomenon can be explained not only by biological differences but also by structural variations in upper airway anatomy.[2,3]

In recent years, genetic predisposition has increasingly gained attention. For example, polymorphisms in matrix metalloproteinase (MMP-1, MMP-3, MMP-9) genes may affect tissue elasticity and inflammatory processes, contributing to the development of OSAS.[22] Similarly, SIRT1 gene variants are thought to facilitate disease onset via oxidative stress and metabolic regulation mechanisms.[21] These findings demonstrate that OSAS is a reflection not only of environmental conditions but also of a significant genetic foundation.

Additionally, craniofacial anatomical anomalies play a crucial role in shaping the risk profile. Conditions such as retrognathia, macroglossia, or nasal obstruction lead to upper airway narrowing, which facilitates apnea formation.[12,14] In pediatric populations, adenotonsillar hypertrophy stands out as one of the most frequently encountered risk factors.[3] These findings suggest that OSAS should be considered a significant health concern not only in adults but also in children.

Lifestyle-related factors must also be acknowledged. For instance, alcohol consumption and the use of sedative medications reduce muscle tone during sleep, thereby promoting airway collapse.[1] Cigarette smoking may exacerbate upper airway narrowing by increasing mucosal inflammation.[8] As such, lifestyle modifications are not only important during treatment but are also critical in preventive strategies.

Another emerging aspect in recent years is the influence of environmental factors. Evidence suggests that global warming may contribute to increased OSAS prevalence, indicating that the condition should be addressed not only from an individual

risk perspective but also at societal and environmental levels.[9] Indeed, rising temperatures may impair sleep quality and intensify symptoms.

In light of all these findings, it is clear that the risk factors for OSAS are not isolated but interrelated and often overlapping. Therefore, risk assessment must be comprehensive for each individual, and personalized prevention and treatment strategies should be developed. This approach may help prevent the onset of the disorder and improve the clinical course of existing cases.

CLINICAL FEATURES AND SYMPTOMS

OSAS is a significant sleep disorder characterized by a wide spectrum of symptoms and clinical manifestations that negatively impact an individual's quality of life. The most commonly reported complaints include loud and intermittent snoring, witnessed breathing pauses during sleep (witnessed apnea), and excessive daytime sleepiness. These symptoms not only prevent individuals from achieving restorative sleep but also lead to serious societal consequences such as reduced attention, impaired cognitive performance, and increased risk of occupational accidents (**Table 1**).[15]

In a recent large-scale study, the clinical features of OSAS were categorized into three main groups:

- Patients experiencing excessive daytime sleepiness and attention deficits
- Cases with significantly impaired sleep quality
- Individuals who present with cardiometabolic comorbidities despite the absence of prominent symptoms

Furthermore, treatment preferences among patients may vary, underscoring the importance of personalized approaches in clinical management.[16]

OSAS is a complex condition characterized by a broad range of clinical manifestations that extend beyond snoring, witnessed apnea, and excessive daytime sleepiness. Common neurocognitive complaints in patients include morning headaches, difficulties in attention and concentration, forgetfulness, and irritability.[15] Additionally, nocturia (frequent nighttime urination), excessive night sweating, sudden awakenings with a feeling of choking, and dry mouth in the morning are among the typical symptoms.[17] Gender-specific differences are also noteworthy; women often report psychiatric symptoms such as depression, anxiety, and insomnia, whereas men are more likely to present with cardiometabolic complications.[16]

Recent studies have shown that the presence of OSAS in the perioperative period increases sensitivity to anesthesia and raises the risk of respiratory and cardiovascular complications.[18] These findings highlight the clinical heterogeneity of OSAS and emphasize the need for a multidisciplinary approach in both diagnostic and therapeutic processes.[19]

OSAS AND ASSOCIATED COMORBID CONDITIONS

OSAS is not limited to episodes of apnea and hypopnea during sleep but is also closely associated with the development and progression of various systemic diseases. Therefore, OSAS should not be considered merely a sleep disorder, but rather a clinical condition with multifaceted effects (**Figure 2**).[6,8]

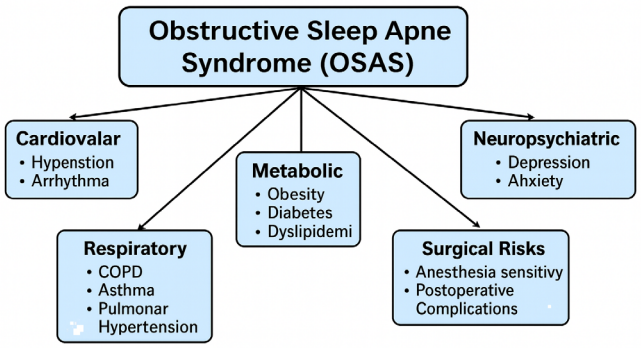

Figure 2. Relational flowchart of obstructive sleep apnea syndrome and its associated comorbid conditions

Cardiovascular Comorbidities

One of the most prominent and well-studied associations of OSAS is with the cardiovascular system. Repeated episodes of hypoxemia continuously activate the sympathetic nervous system and lead to vascular dysfunction. As a result, OSAS emerges as a major risk factor for the development of resistant hypertension.[20] Clinical observations show not only difficulty in controlling blood pressure in these patients, but also an increased susceptibility to cardiovascular diseases. Research has revealed that severe cardiac problems such as coronary artery disease, arrhythmias, and heart failure occur more frequently in individuals with OSAS.[6,16] This suggests that the respiratory disturbances experienced during sleep become a sustained cardiovascular burden throughout the day.

Metabolic and Endocrine Comorbidities

In addition to cardiovascular effects, OSAS also has profound impacts on metabolism. Fragmented sleep and hypoxemia-induced oxidative stress contribute to insulin resistance, laying the groundwork for the development of type 2 diabetes.[23] Many patients experience both conditions simultaneously, complicating the treatment process. Obesity lies at the center of this relationship-it is both the most significant risk factor for OSAS and a condition that OSAS itself may worsen through hormonal and metabolic mechanisms.[23] Moreover, hormonal imbalances are noteworthy. Elevated aldosterone levels have been found to correlate with the severity of OSAS, which is clinically important, particularly in hypertensive patients.[17] Thus, OSAS evolves beyond being merely a sleep disorder, becoming a multifaceted endocrine-metabolic dysregulation syndrome.

Table 1. Symptoms of obstructive sleep apnea syndrome and their clinical significance

Symptom	Definition	Clinical significance
Snoring	A loud and disturbing sound caused by turbulent airflow due to narrowing of the upper airway.	Commonly observed in the general population, disrupts sleep quality, and negatively affects social life.[11]
Witnessed apnea	Periods during sleep when breathing completely stops or significantly decreases, observed by a bed partner.	A direct indicator of apnea, plays a critical role in the diagnostic process.[11]
Excessive daytime sleepiness	Recurrent sleep episodes, lack of attention, and persistent fatigue during the day due to non-restorative sleep.	Leads to occupational and traffic accidents and significantly impairs quality of life.[11]

Respiratory and Inflammatory Comorbidities

OSAS also significantly affects the respiratory system. When combined with obesity and inflammatory processes, it can play a key role in the development of acute respiratory distress syndrome (ARDS).[16] Additionally, when coexisting with chronic obstructive pulmonary disease (COPD), the clinical picture worsens-a condition referred to in the literature as "overlap syndrome." This syndrome is associated with deeper hypoxemic episodes and a poorer prognosis.[6] For this reason, the presence of OSAS should always be evaluated in individuals with respiratory diseases.

Neuropsychiatric and Neurocognitive Comorbidities

Another critical but often overlooked dimension of OSAS is its neuropsychiatric and cognitive consequences. Chronic sleep fragmentation and recurring nocturnal hypoxemia directly impact brain function, leading to symptoms such as forgetfulness, difficulty concentrating, and reduced attention span.[15] Emotional aspects are equally important. Depression and anxiety are more prevalent in individuals with OSAS than in the general population. Interestingly, while depressive symptoms and insomnia are more pronounced in women, men are more frequently affected by cardiometabolic problems.[19] These differences suggest the influence of both gender-based biological mechanisms and sociocultural factors.

Perioperative Risks

The comorbidities associated with OSAS are not limited to chronic diseases; they also pose significant risks during surgical procedures. These patients have increased sensitivity to anesthesia, as well as higher rates of airway complications and cardiovascular instability.[18] Therefore, diagnosing OSAS prior to surgery is crucial for preventing perioperative complications.

Public Health Perspective

Considering all these associations, it becomes clear that OSAS transcends being a merely individual health issue. When coupled with cardiometabolic, respiratory, and psychiatric conditions, it contributes substantially to the overall burden of disease in the population.[4,8] Furthermore, rising obesity rates and global environmental changes indicate that the prevalence of OSAS may continue to grow in the coming years.[9] Consequently, OSAS should not only be a priority for clinicians but also for public health professionals.

DIAGNOSTIC METHODS

The diagnostic methods used for OSAS aim to evaluate both the clinical symptoms and objective sleep data.

Polysomnography

Polysomnography is considered the gold standard diagnostic method for OSAS. It is a comprehensive sleep study that simultaneously records brain electrical activity (EEG), eye movements (EOG), muscle tone (EMG), heart rhythm (ECG), respiratory airflow, thoracoabdominal effort, and oxygen saturation during sleep.[1,2] This allows for the objective identification of sleep stages as well as the number, duration, and severity of apneic and hypopneic episodes. From a clinical perspective, polysomnography not only determines disease severity based on the Apnea-Hypopnea Index (AHI), but also reveals the patient's sleep architecture, arousal responses, and cardiorespiratory effects.[3,11] Because of these features, in-lab polysomnography remains an indispensable tool for differential diagnosis, treatment planning, and long-term follow-up of OSAS.

During polysomnography, electroencephalography (EEG) plays a central role in assessing sleep stages. The brain's activity across various frequency bands reflects transitions between wakefulness and sleep cycles (**Table 2**).

- Alpha waves (8-13 Hz) typically emerge when the eyes are closed and the person is awake but relaxed. They diminish during sleep onset.[1]
- Beta waves (13-30 Hz) are higher in frequency and are prominent during active mental processing and REM sleep.[2]
- Theta waves (4-7 Hz) are a hallmark of the N1 sleep stage and become dominant during light sleep.[3]
- Delta waves (0.5-4 Hz) are characteristic of deep slow-wave sleep (N3) and play a critical role in physiological restoration, memory consolidation, and metabolic repair processes.[4]

The regular recording of these brain waveforms enables detailed analysis of sleep architecture and allows for the objective detection of micro-arousals and disruptions in sleep continuity in disorders such as sleep apnea.[1,11,24] Thus, polysomnography not only evaluates respiratory parameters but also reveals the neurophysiological dynamics of the brain during sleep.

In the diagnosis of OSAS, polysomnography makes it possible to differentiate between apnea types by recording not only changes in airflow but also thoracoabdominal effort. Different types of sleep apnea can be identified based on patterns of airflow and thoracoabdominal effort:

In obstructive sleep apnea, airflow ceases due to upper airway collapse, while thoracic and abdominal muscles continue to exert effort. This pattern is characterized by visible respiratory effort without effective air passage into the lungs.[25]

In central sleep apnea, the issue is not mechanical obstruction of the airway but a cessation of respiratory drive from the brainstem. As a result, both airflow and thoracoabdominal effort are absent simultaneously.[26]

Table 2. Frequency ranges and clinical significance of EEG waves

Wave type	Frequency range	Associated sleep stage and function
Alpha waves	8-13 Hz	Occur during relaxed wakefulness with closed eyes; diminish during sleep onset.[1]
Beta waves	13-30 Hz	High frequency; prominent during active mental processing and REM sleep.[2]
Theta waves	4-7 Hz	Characteristic of N1 stage; dominant during light sleep.[3]
Delta waves	0.5-4 Hz	Associated with deep slow-wave sleep (N3); essential for physiological restoration, memory consolidation, and metabolic repair.[4]

EEG: Electroencephalography

Mixed apnea involves both mechanisms; it typically begins with the lack of effort seen in central apnea, followed by obstructive respiratory effort.

Hypopnea is defined by a significant reduction in airflow without complete cessation, accompanied by oxygen desaturation.

Recognizing these patterns is crucial for accurate classification of sleep disorders and for guiding clinical management.

Home Sleep Apnea Testing (HSAT)

HSAT has emerged as a more accessible and practical alternative, especially for high-risk individuals. While polysomnography remains the gold standard, HSAT devices are increasingly used due to their ease of use, home-based application, and lower cost to healthcare systems.[25] These tests typically record parameters such as airflow, oxygen saturation, heart rate, and thoracoabdominal effort to evaluate the presence and severity of sleep apnea.

Importantly, home testing allows for assessment in a natural sleep environment, which may yield more clinically relevant insights.[26] However, HSAT may not be suitable for patients with comorbidities or suspected complex sleep disorders. Selecting the appropriate patient population for HSAT is essential to prevent false negatives and to ensure diagnostic reliability.[25,26]

Screening Tests

Screening tools are essential for supporting clinical assessment in the diagnosis of OSAS. One of the most widely used tools is the Epworth Sleepiness Scale (ESS), which allows patients to subjectively evaluate their daytime sleepiness. The scale assesses the tendency to fall asleep during routine daily activities such as sitting, reading, watching TV, or being a passenger in traffic. The resulting score provides valuable clues about the severity of sleep disorders.[25] In individuals suspected of having OSAS, high ESS scores often prompt further diagnostic testing such as polysomnography.[26] Although ESS is entirely subjective and not diagnostic on its own, its ease of use and rapid assessment capability make it a valuable screening tool to clarify a patient's clinical disorders.[25,26]

CLASSIFICATION OF OSAS

OSAS is classified during the diagnostic process based on the AHI, following international standards. According to the guidelines of the American Academy of Sleep Medicine (AASM), an AHI value between 5 and 14 is defined as mild OSAS, between 15 and 29 as moderate OSAS, and 30 or above as severe OSAS (**Table 3**).[27]

This classification not only indicates the frequency of apneic and hypopneic events but also helps guide the clinical course, treatment necessity, and evaluation of associated comorbid conditions.

Moreover, the international classification of sleep disorders (ICSD-3) considers OSAS as a distinct clinical category, emphasizing that diagnostic criteria should include not only objective measurements but also symptomatic features-such as excessive daytime sleepiness, snoring, and witnessed apneas.[27] This approach highlights that the condition should not be evaluated solely through numerical parameters but must also consider its impact on quality of life and functional capacity. In doing so, international classification systems provide clinicians with diagnostic clarity while also enhancing the comparability of data across different populations.

TREATMENT APPROACHES

Lifestyle Modifications

The first-line approach in the management of OSAS often begins with lifestyle modifications. This is because the condition is not only influenced by medical devices or surgical interventions but also significantly shaped by an individual's daily habits. As such, lifestyle changes are considered a fundamental therapeutic strategy that directly impacts both the onset and progression of the disease.

Weight control is one of the most critical components in OSAS management. Excess body weight-particularly the accumulation of fat tissue around the neck-narrows the upper airway and increases the risk of apnea. Clinical observations have shown that weight loss not only provides mechanical relief but also improves metabolic processes, leading to a significant reduction in the AHI.[25] Therefore, adjusting dietary habits and achieving an ideal body weight are essential not only for OSAS but also for overall health.

Another important factor is alcohol consumption. Alcohol intake, especially at night, reduces pharyngeal muscle tone, making the airway more susceptible to collapse. This results in prolonged apnea episodes and more pronounced oxygen desaturation.[26] Thus, patients are advised to limit or completely avoid alcohol.

Similarly, smoking is a detrimental lifestyle factor in OSAS. Cigarette smoke increases inflammation in the upper respiratory tract, causing mucosal edema and narrowing of the airways. This can exacerbate both the frequency and severity of apnea events.[25] Smoking cessation is therefore a crucial step not only for pulmonary health but also for improving sleep quality.

Sleep hygiene should also not be overlooked. Regular sleep schedules, sufficient sleep duration, and a proper sleep environment help alleviate symptoms in OSAS patients. Moreover, adapting to a lateral sleeping position is particularly effective in mild cases. Sleeping on the back can cause the tongue base to fall backward due to gravity, whereas side-sleeping reduces this effect and supports better airflow.[26]

Finally, regular physical activity is a powerful preventive component among lifestyle modifications. Exercise not only

Table 3. OSAS within the framework of the ICSD-3

AHI	Severity	Description
5-14 events/hour	Mild OSAS	Associated with light symptoms; may affect sleep quality and daytime function.
15-29 events/hour	Moderate OSAS	More frequent events; often causes noticeable daytime sleepiness and reduced quality of life.
≥30 events/hour	Severe OSAS	High frequency of events; linked to significant health risks, including cardiovascular and metabolic disorders.

OSAS: Obstructive sleep apnea syndrome, ICSD-3: International classification of sleep disorders, AHI: Apnea-Hypopnea Index

facilitates weight loss but also strengthens respiratory muscles, supports respiratory function, and reduces cardiometabolic risks.[25] Therefore, lifestyle interventions play a pivotal role not only in symptom control but also in improving the long-term health outcomes of OSAS.

Medical Treatment

In the management of OSAS, medical approaches aim to alleviate symptoms and prevent complications by targeting the underlying pathophysiology of the condition. Within this context, oral appliances and positive airway pressure (PAP) devices stand out as effective and customizable therapeutic alternatives.

Role and limitations of oral appliances: Oral appliances are a non-surgical treatment option primarily recommended for patients with mild to moderate OSAS. These devices typically function by repositioning the mandible anteriorly, thereby mechanically maintaining patency of the pharyngeal airway and reducing the risk of upper airway collapse.[28] They serve as a viable alternative for patients who cannot tolerate or do not adhere to CPAP or BiPAP therapy. However, their effectiveness may be limited in severe cases of OSAS and they are not recommended for individuals with temporomandibular joint disorders.[28] Long-term use may be associated with side effects such as jaw pain and alterations in dental alignment. Therefore, careful patient selection and a multidisciplinary approach to monitoring the treatment process are essential.

PAP therapy: This is considered the first-line treatment for OSAS. Continuous CPAP devices deliver a constant and positive pressure throughout the night to keep the airway open.[29] Due to its simplicity and efficacy, this method is widely used. Regular use of the device and close patient monitoring are essential to ensure adherence to therapy. Tracking the adaptation process and integrating digital monitoring systems can directly influence the success of CPAP therapy.[30]

Some patients may struggle to adapt to a fixed pressure. In such cases, Bilevel Positive Airway Pressure (BiPAP) devices can be used as an alternative. BiPAP delivers different pressure levels during inhalation and exhalation, which allows for a more comfortable sleep experience.[30] This modality may be particularly effective in patients with advanced OSAS or those suffering from hypoventilation syndrome.

Surgical Treatment Options

OSAS is a condition with highly variable responses to treatment. A one-size-fits-all approach may not be effective for every patient; therefore, an individualized treatment strategy is essential. While PAP therapy is the first-line recommendation, some patients may not adapt to this method, or they may not achieve sufficient symptom relief despite regular use of the device. In such cases, particularly in patients with prominent anatomical narrowing, surgical interventions come into consideration.[26]

The main goal in evaluating surgical options is to determine the location and degree of anatomical obstruction in the upper airway and to develop a structured intervention plan for these areas. For this purpose, diagnostic tools such as imaging methods, flexible endoscopy, and, when necessary, drug-induced sleep endoscopy (DISE) are used. The surgical approach not only offers a mechanical solution but can also enhance the success of PAP therapy.[26]

One of the most commonly performed procedures is uvulopalatopharyngoplasty (UPPP), which aims to enlarge the airway by removing excess tissue from the soft palate, uvula, and posterior pharyngeal wall. This method is particularly effective in patients with oropharyngeal-level obstruction.[26] However, because this surgery addresses only a limited anatomical region, it may not be sufficient on its own in some cases.

In more extensive and deeper airway obstructions, advanced surgical procedures such as maxillomandibular advancement (MMA) are employed. MMA involves advancing both the maxilla and mandible forward to create a permanent expansion of all segments of the upper airway and has a high success rate in cases with multilevel obstruction.[27]

Additionally, in patients with significant tongue base obstruction, targeted interventions such as glossectomy, genioglossus advancement, or hypoglossal nerve stimulation may be utilized. Nasal surgeries such as septoplasty and turbinate reduction, although not direct treatments for OSAS, can support treatment success by improving PAP compliance or correcting nasal obstruction.[27]

The success of surgical interventions is closely related to proper patient selection, appropriate surgical techniques, and postoperative follow-up processes. However, it should be remembered that surgery is always an invasive option, and the decision-making process should be shaped within the framework of patient education and multidisciplinary evaluation. Since each individual's anatomical structure and clinical needs differ, surgical treatment must also be equally personalized.

Emerging Developments and Future Treatment Options in OSAS

In recent years, remarkable innovations have emerged in the treatment of OSAS, driven by scientific and technological advancements. These innovations are designed both to enhance treatment response and to improve patient comfort. Considering the multifaceted nature of the disease, future treatment options are becoming increasingly diverse.

Neurostimulation and Electrical Modulation

Hypoglossal nerve stimulation (HNS) has emerged as an effective alternative, particularly for patients who cannot tolerate CPAP or require surgical intervention due to anatomical factors. Experimental studies on animal models have demonstrated that this method reduces the number of apneic episodes and improves oxygen levels.[33] These findings suggest that the physiological effects of HNS contribute not only to muscle activation but also to systemic oxygenation.

Moreover, engineering-based research on the energy efficiency and volumetric performance of implantable neurostimulator systems indicates that future generations of these devices will be longer-lasting and more user-friendly.[34] These developments may offer significant clinical advantages in terms of mobility and quality of life.

Pharmacological Approaches

While pharmacotherapy has not yet become a routine clinical practice in OSAS treatment, drug development targeting the underlying pathophysiological mechanisms has gained momentum. Recent studies have shown promising results

from drug combinations aimed at parameters such as reduced upper airway muscle tone, ventilatory instability, and arousal threshold.[35] These novel pharmacological strategies may play a complementary role, particularly in multidisciplinary treatment plans.

Advanced Diagnostic and Monitoring Technologies

Low-contact systems designed to reduce patient burden during the diagnostic process and increase accessibility are also gaining attention. Notably, ongoing studies are exploring cost-effective and efficient home-based diagnostic systems that combine millimeter-wave radar technology with pulse oximetry data.[36] These technologies have the potential to improve both accuracy and accessibility in the diagnosis of OSAS.

TREATMENT APPROACHES ACCORDING TO CLINICAL STAGES OF OSAS

Determining treatment strategies based on the stage of OSAS is crucial for ensuring effective and sustainable interventions tailored to individual disease progression. Each stage of OSAS presents different clinical needs in terms of symptom severity and complication risk. Addressing these differences not only improves patient quality of life but also yields meaningful improvements in long-term health outcomes.

Treatment Approach in Mild OSAS

Mild OSAS is defined as having an AHI between 5 and 15 and is typically characterized by less severe symptoms. At this stage, the primary approach is based on lifestyle modifications and conservative interventions. Behavioral measures such as weight loss, avoiding alcohol and sedatives that reduce upper airway tone, quitting smoking, and refraining from sleeping in the supine position are recommended as the first-line strategies.[29]

If symptoms are not adequately controlled through these measures, or if the patient experiences persistent complaints such as excessive daytime sleepiness or morning headaches that impair quality of life, alternative medical treatments should be considered.

At this point, the use of oral appliances emerges as an effective option. These devices aim to increase upper airway patency by repositioning the mandible forward and can significantly reduce the frequency of apneas and hypopneas during the night.[28] Being portable, quiet, and non-invasive, oral appliances enhance patient comfort, though they should be used cautiously in patients with temporomandibular joint disorders. In mild OSAS, they may offer better patient adherence compared to more intensive treatments such as CPAP.[28]

Although PAP devices are not usually the first choice for mild OSAS, in some clinical situations, their use may become medically necessary. For instance, if significant daytime sleepiness is detected through standardized tests like the Epworth Sleepiness Scale and it negatively affects the patient's functionality and quality of life, PAP therapy may be initiated.[29] Similarly, in patients with treatment-resistant hypertension, PAP can be beneficial in suppressing increased sympathetic tone associated with OSAS.[29]

PAP therapy is also recommended in patients with cardiac comorbidities-particularly those with congestive heart failure or atrial fibrillation-due to its beneficial effects in reducing cardiac workload. Moreover, for patients who have experienced cerebrovascular events such as stroke, PAP treatment may support neurological recovery during the post-ischemic phase.[29]

Lastly, for individuals in occupations requiring high alertness-such as pilots and commercial drivers-initiating PAP therapy may be necessary even in the presence of mild OSAS to prevent potential work-related accidents.[29] These examples clearly demonstrate that treatment planning should be guided not only by the AHI score but also by the broader clinical context.

Treatment Approach in Moderate OSAS

Moderate OSAS refers to clinical cases where the AHI falls between 15 and 30. Treatment at this stage should be more structured and individualized. According to the clinical guidelines recommended by the AASM, PAP therapy is suggested as the first-line treatment for patients in this group.[29]

The most commonly used form of PAP is the CPAP (Continuous Positive Airway Pressure) device, which delivers a constant and steady positive pressure to the patient's upper airway throughout the night. This mechanism prevents airway collapse, thereby reducing the frequency of apneas and hypopneas, and significantly improving both sleep quality and daytime alertness.[29] CPAP therapy is a standard approach in moderate OSAS, aiming to relieve symptoms and reduce long-term cardiometabolic risks.[31]

However, not all patients can easily adapt to CPAP therapy. Discomfort with the mask, noise, or psychological unease may cause some to discontinue treatment. In such cases, BiPAP (Bilevel PAP) devices, which allow separate adjustments for inspiratory and expiratory pressures, may provide a more comfortable experience. Alternatively, for individuals with suitable anatomical structures and milder symptoms, oral appliances can be considered.[31]

Another important factor influencing treatment decisions in moderate OSAS is the presence of comorbidities. Systemic conditions such as hypertension, type 2 diabetes, and obesity should be considered during treatment planning, and the potential benefits of PAP therapy evaluated within this context.[29] Moreover, enhancing treatment adherence through patient education and regular follow-ups should be a priority to improve clinical outcomes.

Treatment Approach in Severe OSAS

A diagnosis of severe OSAS is made when the AHI exceeds 30. In this patient group, not only sleep quality but also cardiovascular, metabolic, and neurological systems can be significantly affected. Therefore, treatment at this stage requires a more aggressive and multidisciplinary approach.[29]

In severe OSAS, CPAP therapy is the first-line and most strongly evidence-supported treatment option. CPAP applies constant positive pressure to the upper airway during sleep, preventing airway collapse and ensuring continuous breathing. This mechanism reduces the frequency of apnea-hypopnea episodes and provides significant improvement in symptoms such as daytime sleepiness, cognitive impairment, and hypertension.[29]

However, the success of CPAP therapy depends not only on prescription but also on patient adherence and consistent use of the device. Therefore, telemetric monitoring systems

developed to track treatment adherence play a crucial role in increasing its effectiveness and sustainability. Today, many CPAP devices transmit cloud-based data, allowing both healthcare professionals and patients to regularly monitor usage duration and pressure parameters.[30]

Some patients may not tolerate CPAP for various reasons. In such cases, alternative positive pressure systems such as BiPAP (Bilevel Positive Airway Pressure) may be used. BiPAP delivers different pressure levels during inhalation and exhalation and is often better tolerated in patients with hypoventilation or coexisting respiratory disorders. In cases where CPAP or BiPAP fails to control symptoms and in patients with prominent anatomical obstructions, surgical interventions (e.g., uvulopalatopharyngoplasty) may be considered.[29]

In conclusion, treatment planning for severe OSAS should be based not only on the AHI score but also on the patient's symptom profile, lifestyle, comorbidities, and adherence potential. Patient education, device usage guidance, and regular follow-up are key components of successful treatment.

The management of OSAS is most effective when approached through multidisciplinary and holistic strategies. Although treatment options vary according to disease severity, lifestyle modifications remain a cornerstone of care at all levels. These include weight control, reduction of alcohol and tobacco use, and adjustments in sleep position.

Beyond lifestyle measures, medical therapies such as PAP devices, oral appliances, and pharmacological interventions can be applied. When indicated, surgical approaches may also be considered. Importantly, treatment should not only be guided by severity classification but also tailored to the individual's anatomical features, physiological characteristics, and lifestyle factors. Such personalized care ensures both efficacy and long-term adherence, supporting better clinical outcomes and improved quality of life.

CONCLUSION

Although OSAS often presents with seemingly "simple" symptoms such as snoring or pauses in breathing during sleep, it hides a complex condition that can lead to serious health issues. These silent obstructions during sleep not only disrupt sleep quality but also negatively impact many areas-from cardiovascular health and metabolic balance to mood and daily performance. Therefore, OSAS should be approached as a systemic disorder that affects not just nighttime rest but an individual's overall quality of life. In this context, early diagnosis is of vital importance. A timely evaluation can prevent numerous serious complications such as hypertension, heart disease, stroke, or depression. The earlier treatment begins, the greater the improvement in the patient's quality of life and overall health. However, effective treatment is not limited to a device or surgical intervention. Managing OSAS is a multidisciplinary process that requires collaboration among various fields, including otolaryngology, pulmonology, cardiology, nutrition, and psychology. This teamwork not only accelerates diagnosis but also enhances treatment adherence and helps patients feel understood and supported. In conclusion, managing OSAS should aim not only to treat a disorder but also to holistically improve a person's sleep, health, and quality of life. Through awareness, early action, and interdisciplinary collaboration, a breath that stops during sleep can be transformed into a life renewed with health.

ETHICAL DECLARATIONS

Referee Evaluation Process

Externally peer-reviewed.

Conflict of Interest Statement

The authors have no conflicts of interest to declare.

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Author Contributions

All of the authors declare that they have all participated in the design, execution, and analysis of the paper, and that they have approved the final version.

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