



SLEEP APNEA SYNDROME: ETIOPATHOGENESIS, CLINICAL MANIFESTATIONS, CONTEMPORARY DIAGNOSTIC APPROACHES, AND EVIDENCE BASED THERAPEUTIC STRATEGIES

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Abstract

Sleep apnea syndrome (SAS), particularly obstructive sleep apnea (OSA), has become one of the most significant challenges in modern medicine, extending beyond the fields of cardiology and endocrinology. Sleep apnea syndrome is a chronic, multifactorial disorder characterized by recurrent episodes of partial or complete cessation of breathing during sleep. This condition not only impairs normal sleep quality but also contributes to the development of severe cardiovascular, endocrine, neurological, and metabolic complications. This review comprehensively discusses the etiological factors and pathogenetic mechanisms underlying sleep apnea syndrome, the diversity of its clinical manifestations, contemporary diagnostic approaches, and current evidence-based treatment strategies. Particular emphasis is placed on continuous positive airway pressure (CPAP) therapy, which remains the gold-standard treatment for moderate-to-severe obstructive sleep apnea.

Keywords: Sleep apnea syndrome; obstructive sleep apnea; polysomnography; continuous positive airway pressure; intermittent hypoxia; apnea-hypopnea index; cardiovascular complications.

Introduction

Sleep-related disorders have become an increasingly important public health concern worldwide. Among these conditions, sleep apnea syndrome (SAS) is one of the most prevalent yet substantially underdiagnosed disorders. Epidemiological studies indicate that obstructive sleep apnea occurs approximately two to three times more frequently in men than in women. The prevalence of obstructive sleep apnea among middle-aged adults is estimated to be approximately **24% in men** and **5% in women**.

Sleep apnea is characterized by recurrent episodes of complete or partial upper airway obstruction lasting at least **10 seconds**, accompanied by intermittent oxygen desaturation. Disease severity is commonly assessed using the **Apnea-Hypopnea Index (AHI)** and the **Oxygen Desaturation Index (ODI)**, which quantify the hourly frequency of apnea/hypopnea events and oxygen desaturation





episodes, respectively. Clinically significant events are generally associated with a reduction in oxyhemoglobin saturation of at least **3–4%**.

The insidious onset and gradual progression of symptoms frequently delay diagnosis, resulting in prolonged exposure to intermittent hypoxia and sleep fragmentation. Consequently, untreated sleep apnea syndrome is associated with impaired work performance, increased risk of motor vehicle accidents, reduced quality of life, and early development of cardiovascular diseases.

Approximately **50%** of patients with sleep apnea syndrome develop arterial hypertension and ischemic heart disease. Furthermore, obesity—particularly abdominal obesity—is one of the strongest predisposing factors for obstructive sleep apnea, with insulin resistance playing a substantial role in disease progression and severity.

Etiopathogenesis

Sleep apnea syndrome is a multifactorial disorder resulting from the interaction of anatomical, neuromuscular, metabolic, and neurophysiological mechanisms. Based on the underlying pathophysiology, sleep apnea is classified into three major subtypes: **obstructive sleep apnea (OSA)**, **central sleep apnea (CSA)**, and **mixed sleep apnea (MSA)**. Although these conditions differ in their primary mechanisms, all are characterized by recurrent episodes of respiratory interruption during sleep, leading to intermittent hypoxemia, hypercapnia, sleep fragmentation, and systemic physiological disturbances.

Obstructive Sleep Apnea

Obstructive sleep apnea is the most common form of sleep-disordered breathing and is characterized by repetitive partial or complete collapse of the upper airway during sleep despite preserved respiratory effort. Airway obstruction primarily occurs at the level of the pharynx due to excessive relaxation of the soft palate, tongue base, and pharyngeal dilator muscles.

During normal sleep, physiological reduction in upper airway muscle tone occurs. However, in susceptible individuals, this relaxation becomes excessive, resulting in upper airway collapse. The negative intrathoracic pressure generated during inspiratory efforts against an occluded airway further aggravates airway obstruction, producing repeated episodes of apnea and hypopnea.

Obesity is considered one of the strongest risk factors for obstructive sleep apnea. Excess adipose tissue deposited around the neck and upper airway narrows the pharyngeal lumen and increases airway collapsibility. Craniofacial abnormalities, enlarged tonsils, macroglossia, nasal obstruction, smoking, alcohol consumption, and sedative medications further contribute to airway narrowing and disease progression.

Repeated episodes of airway obstruction produce intermittent hypoxemia, hypercapnia, oxidative stress, systemic inflammation, and sympathetic nervous system activation. These pathological processes promote endothelial dysfunction, metabolic dysregulation, insulin resistance, and persistent arterial hypertension, thereby markedly increasing the long-term risk of cardiovascular and cerebrovascular diseases.

Consequently, obstructive sleep apnea should not be regarded solely as a respiratory disorder but rather as a complex neuromuscular and anatomical dysfunction with significant systemic





consequences. Early diagnosis and timely interventions aimed at maintaining upper airway patency have been shown to substantially reduce cardiovascular morbidity and metabolic complications.

Central Sleep Apnea

Unlike obstructive sleep apnea, central sleep apnea is a neurophysiological disorder characterized by transient cessation of respiratory effort resulting from impaired central regulation of breathing rather than upper airway obstruction. In this condition, the airway remains anatomically patent; however, respiratory drive generated by the brainstem is temporarily interrupted.

The respiratory rhythm is tightly regulated through interactions between the medullary respiratory centers, peripheral and central chemoreceptors, arterial carbon dioxide (PaCO_2), oxygen tension, and cardiovascular function. Instability within this feedback system results in periodic fluctuations in respiratory drive, ultimately producing recurrent episodes of central apnea.

Central sleep apnea frequently develops in patients with heart failure, cerebrovascular disease, opioid use, or neurological disorders. In chronic heart failure, prolonged circulation time delays communication between pulmonary gas exchange and central chemoreceptors. Consequently, increased sensitivity to carbon dioxide leads to unstable ventilatory control, cyclic hyperventilation, hypocapnia, and transient suppression of respiratory drive, culminating in recurrent episodes of central apnea.

Recent investigations indicate that individualized therapeutic strategies targeting the underlying cardiovascular or neurological disorder can significantly reduce the incidence of central sleep apnea and improve long-term clinical outcomes.

Mixed Sleep Apnea

Mixed sleep apnea represents a combination of central and obstructive mechanisms. Typically, an apnea episode begins with the absence of respiratory effort characteristic of central sleep apnea and subsequently progresses to upper airway obstruction, resembling obstructive sleep apnea.

The coexistence of these mechanisms reflects a more complex pathophysiological process involving both impaired neural respiratory regulation and increased upper airway collapsibility. Patients with obesity, enlarged neck circumference, craniofacial abnormalities, chronic nasal obstruction, smoking, and alcohol consumption are at particularly high risk of developing mixed sleep apnea.

The recurrent cycles of hypoxia, reoxygenation, and sleep fragmentation associated with mixed sleep apnea contribute to oxidative stress, systemic inflammation, endothelial dysfunction, and autonomic imbalance. These mechanisms substantially increase the risk of hypertension, cardiac arrhythmias, myocardial infarction, stroke, insulin resistance, and reduced overall quality of life.

Overall, accumulating evidence suggests that sleep apnea syndrome should be recognized as a systemic multisystem disorder rather than merely a nocturnal breathing abnormality. A comprehensive understanding of its underlying pathogenetic mechanisms provides the foundation for early diagnosis, individualized treatment, and prevention of long-term cardiovascular and metabolic complications.





Clinical Manifestations

The clinical presentation of sleep apnea syndrome is highly heterogeneous and varies according to disease severity, duration, and the presence of associated comorbidities. Symptoms are generally classified into **nocturnal** and **daytime manifestations**, both of which significantly impair quality of life and are associated with increased cardiovascular and neurocognitive morbidity.

Nocturnal Manifestations

The most recognizable symptom of obstructive sleep apnea is **persistent loud snoring**, which results from turbulent airflow through a partially collapsed upper airway. Although snoring alone is not diagnostic of sleep apnea, habitual loud snoring, particularly when accompanied by witnessed breathing pauses, strongly suggests the presence of obstructive sleep apnea. This symptom is especially common among individuals with obesity, increased neck circumference, and excessive adipose tissue surrounding the upper airway.

A hallmark feature of both obstructive and central sleep apnea is **recurrent episodes of apnea during sleep**. In obstructive sleep apnea, respiratory effort persists despite complete or partial upper airway collapse, whereas in central sleep apnea both airflow and respiratory effort temporarily cease because of impaired respiratory drive. These episodes are frequently terminated by transient arousal from sleep, restoring ventilation but simultaneously disrupting normal sleep architecture.

Many patients experience **sudden nocturnal awakenings accompanied by a sensation of choking or suffocation**. These events are primarily triggered by intermittent hypoxemia, fluctuations in arterial carbon dioxide levels, and activation of peripheral and central chemoreceptors. The resulting sympathetic nervous system activation produces abrupt awakenings associated with tachycardia, anxiety, and respiratory distress.

Additional nocturnal manifestations include **restless sleep, excessive nocturnal sweating, dry mouth upon awakening, nocturia, sleep talking, and frequent changes in sleeping position**. Repeated arousals prevent progression into restorative slow-wave and rapid eye movement (REM) sleep, leading to chronic sleep fragmentation.

Daytime Manifestations

The most common daytime consequence of sleep apnea syndrome is **excessive daytime sleepiness (EDS)**, which results from recurrent sleep interruption and poor sleep quality. Patients frequently report an irresistible tendency to fall asleep during routine daily activities, including reading, watching television, attending meetings, or even driving. Excessive daytime sleepiness represents one of the principal factors contributing to occupational accidents and motor vehicle collisions among individuals with untreated sleep apnea.

Chronic intermittent hypoxia and recurrent sleep fragmentation also impair **attention, concentration, executive functioning, learning capacity, and memory**. Many patients describe persistent mental fatigue, reduced work productivity, diminished academic performance, and difficulty performing cognitively demanding tasks.

Morning headaches are another common complaint and are thought to result from nocturnal hypoxemia, hypercapnia, cerebral vasodilation, and impaired sleep continuity. These headaches





typically improve within several hours after awakening but may become chronic in patients with severe untreated disease.

Sleep apnea syndrome exerts profound effects on psychological well-being. Patients frequently develop **irritability, emotional instability, anxiety, depressive symptoms, decreased motivation, and impaired social functioning**. Persistent sleep disruption alters normal neurotransmitter regulation and contributes to neurocognitive dysfunction, thereby negatively affecting overall mental health.

Systemic Complications

Long-standing untreated sleep apnea syndrome is associated with widespread multisystem complications. Recurrent cycles of intermittent hypoxia and reoxygenation promote oxidative stress, systemic inflammation, endothelial dysfunction, and sustained sympathetic activation, all of which accelerate cardiovascular disease progression.

Patients with moderate-to-severe sleep apnea demonstrate a substantially increased risk of **arterial hypertension, coronary artery disease, chronic heart failure, atrial fibrillation and other cardiac arrhythmias, myocardial infarction, pulmonary hypertension, and ischemic stroke**. Furthermore, sleep apnea contributes to metabolic abnormalities, including **insulin resistance, type 2 diabetes mellitus, dyslipidemia, and metabolic syndrome**, creating a bidirectional relationship between obesity and cardiometabolic disease.

Emerging evidence also indicates that chronic sleep apnea is associated with accelerated cognitive decline and an increased risk of mild cognitive impairment and dementia through mechanisms involving chronic cerebral hypoxia, neuroinflammation, and microvascular injury.

Taken together, these findings emphasize that sleep apnea syndrome should be regarded as a **systemic chronic disease** with significant respiratory, cardiovascular, neurological, metabolic, and psychological consequences rather than simply a disorder of nocturnal breathing.

Clinical Implications

Because the clinical manifestations of sleep apnea syndrome often develop gradually and remain unrecognized for many years, clinicians should maintain a high index of suspicion in patients presenting with habitual snoring, witnessed apneas, excessive daytime sleepiness, resistant hypertension, obesity, or unexplained cognitive impairment. Early recognition of these symptoms facilitates timely diagnosis, reduces disease progression, and significantly lowers the risk of long-term cardiovascular and neurocognitive complications.

Modern Diagnostic Approaches

Early and accurate diagnosis of sleep apnea syndrome is essential for preventing disease progression and reducing the risk of long-term cardiovascular, cerebrovascular, and metabolic complications. Since the clinical manifestations of sleep apnea are often nonspecific and develop gradually, diagnosis requires a combination of comprehensive clinical assessment, standardized screening tools, physical examination, and objective sleep studies.





Clinical Evaluation and Risk Assessment

The diagnostic process begins with a detailed medical history focusing on nocturnal and daytime symptoms. Particular attention should be paid to habitual loud snoring, witnessed apneic episodes, nocturnal choking, excessive daytime sleepiness, morning headaches, impaired concentration, and the presence of cardiovascular or metabolic comorbidities.

Physical examination should include assessment of well-established anatomical and anthropometric risk factors. Obesity remains one of the strongest predictors of obstructive sleep apnea. Patients with a body mass index (BMI) ≥ 34 kg/m² and a neck circumference exceeding 44 cm have a substantially increased likelihood of upper airway collapse during sleep.

Evaluation by an otorhinolaryngologist is recommended to identify structural abnormalities that may contribute to upper airway obstruction. Common findings include nasal septal deviation, adenoid hypertrophy, chronic hypertrophic tonsillitis, enlarged tonsils, nasal polyps, turbinate hypertrophy, and other craniofacial or upper airway abnormalities that may compromise airflow.

Polysomnography: The Gold Standard

Overnight polysomnography (PSG) is universally recognized as the gold-standard diagnostic method for sleep apnea syndrome. This comprehensive examination simultaneously records multiple physiological parameters throughout sleep, allowing precise identification, classification, and severity assessment of sleep-related breathing disorders.

Polysomnography typically includes continuous monitoring of:

- electroencephalography (EEG) for sleep staging;
- electrooculography (EOG);
- electromyography (EMG);
- electrocardiography (ECG);
- airflow through the nose and mouth;
- thoracic and abdominal respiratory movements;
- arterial oxygen saturation (SpO₂);
- body position; and
- snoring intensity.

Simultaneous analysis of these physiological variables enables clinicians to distinguish obstructive, central, and mixed sleep apnea while evaluating their frequency, duration, and physiological consequences.

Diagnostic Criteria

According to current international sleep medicine guidelines, respiratory events are classified as follows:

Apnea

Apnea is defined as a $\geq 90\%$ reduction in airflow lasting at least 10 seconds. Depending on the presence or absence of respiratory effort, apnea episodes are categorized as obstructive, central, or mixed.

Hypopnea





Hypopnea is characterized by a $\geq 30\%$ reduction in airflow lasting at least 10 seconds, accompanied by either an oxygen desaturation of at least 3–4% or an electroencephalographically confirmed arousal from sleep.

Apnea–Hypopnea Index

Disease severity is quantified using the Apnea–Hypopnea Index (AHI), which represents the average number of apnea and hypopnea events occurring per hour of sleep.

Severity is generally classified as follows:

AHI (events/hour)	Disease Severity
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5–14	Mild OSA
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15–29	Moderate OSA
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≥ 30	Severe OSA
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The Oxygen Desaturation Index (ODI) serves as an additional indicator of disease burden by measuring the hourly frequency of oxygen desaturation episodes. Higher ODI values correlate strongly with intermittent hypoxia, cardiovascular risk, and disease severity.

Screening Questionnaires

Validated clinical questionnaires provide an effective initial assessment of patients at risk for sleep apnea syndrome.

Among these, the Epworth Sleepiness Scale (ESS) is the most widely used instrument for evaluating excessive daytime sleepiness. The questionnaire measures the patient's likelihood of falling asleep during eight common daily situations.

An ESS score greater than 10 suggests clinically significant daytime sleepiness and indicates the need for further diagnostic evaluation.

Additional validated screening instruments include the STOP-Bang Questionnaire, the Berlin Questionnaire, and the NoSAS Score, all of which improve risk stratification in both primary care and sleep medicine settings.

Pulse Oximetry

Overnight pulse oximetry represents a valuable, inexpensive, and non-invasive screening tool, particularly in settings where polysomnography is not readily available.

Repeated nocturnal episodes of oxygen desaturation strongly suggest sleep-disordered breathing. Although pulse oximetry cannot reliably distinguish obstructive from central sleep apnea, it provides important information regarding the severity of nocturnal hypoxemia and helps identify patients who require comprehensive polysomnographic evaluation.

Upper Airway Endoscopy and Imaging Studies

Flexible nasopharyngoscopy and upper airway endoscopy play an important role in identifying anatomical sites of airway obstruction. These examinations are particularly valuable when surgical intervention is being considered.

Radiological imaging, including computed tomography (CT) and magnetic resonance imaging (MRI) of the upper airway, brain, or thorax, may be indicated in selected patients with suspected structural abnormalities, neurological disorders, or central sleep apnea. Imaging studies also facilitate preoperative planning and differential diagnosis in complex cases.





Cardiovascular Evaluation

Because sleep apnea syndrome is strongly associated with cardiovascular disease, patients should undergo comprehensive cardiovascular assessment. Electrocardiography, ambulatory blood pressure monitoring, echocardiography, and other investigations may be indicated depending on the clinical presentation.

Particular attention should be given to identifying resistant hypertension, cardiac arrhythmias, chronic heart failure, pulmonary hypertension, and ischemic heart disease, as these conditions frequently coexist with moderate-to-severe sleep apnea and significantly influence prognosis and therapeutic decision-making.

Future Perspectives in Sleep Apnea Diagnosis

Recent advances in digital health technologies have expanded the diagnostic landscape of sleep medicine. Home sleep apnea testing (HSAT), wearable biosensors, artificial intelligence–assisted signal analysis, and machine-learning algorithms are increasingly being incorporated into clinical practice. These innovations have the potential to improve accessibility, reduce healthcare costs, and facilitate earlier diagnosis while maintaining acceptable diagnostic accuracy in appropriately selected patients.

Nevertheless, despite these technological advances, laboratory-based polysomnography remains the reference standard for confirming the diagnosis, determining disease severity, differentiating sleep apnea subtypes, and guiding individualized treatment strategies.

Clinical Significance

Accurate diagnosis of sleep apnea syndrome requires an integrated approach combining clinical evaluation, standardized screening questionnaires, objective sleep studies, and multidisciplinary assessment. Early identification of affected individuals enables timely initiation of evidence-based therapy, significantly reducing cardiovascular morbidity, neurocognitive impairment, metabolic dysfunction, and overall mortality.

Treatment Strategies

The primary objectives of sleep apnea syndrome management are to eliminate or significantly reduce apnea and hypopnea episodes, restore normal nocturnal oxygenation, improve sleep quality, alleviate daytime symptoms, and prevent long-term cardiovascular, cerebrovascular, metabolic, and neurocognitive complications. Because sleep apnea syndrome is a heterogeneous disorder with diverse etiological mechanisms, treatment should be individualized according to the type and severity of disease, underlying anatomical abnormalities, associated comorbidities, and patient-specific characteristics.

Lifestyle Modification

Lifestyle intervention represents the cornerstone of treatment for all patients with sleep apnea syndrome, particularly those with mild disease or obesity-related obstructive sleep apnea.

Weight reduction is among the most effective non-invasive interventions. Even modest weight loss has been shown to decrease upper airway collapsibility, improve respiratory mechanics, reduce the Apnea–Hypopnea Index (AHI), and enhance overall sleep quality. In overweight and obese





individuals, comprehensive weight management programs incorporating dietary modification, regular physical activity, and behavioral counseling should be strongly encouraged.

Patients should also receive education regarding proper sleep hygiene, including maintaining regular sleep schedules, obtaining adequate sleep duration, and avoiding sleep deprivation. Alcohol consumption and sedative medications should be minimized or discontinued whenever possible, as these substances further reduce upper airway muscle tone and increase the frequency and duration of apnea events.

Smoking cessation is equally important because tobacco use contributes to chronic upper airway inflammation and edema, thereby exacerbating airway obstruction during sleep.

Continuous Positive Airway Pressure (CPAP) Therapy

Continuous Positive Airway Pressure (CPAP) therapy remains the **first-line treatment and the gold standard** for patients with moderate-to-severe obstructive sleep apnea.

CPAP devices deliver a constant level of positive airway pressure through a nasal or oronasal mask throughout the respiratory cycle. This continuous pressure acts as a pneumatic splint that prevents collapse of the upper airway during sleep, thereby maintaining airway patency and ensuring uninterrupted ventilation.

As a result, CPAP therapy effectively:

- eliminates recurrent upper airway obstruction;
- markedly reduces apnea and hypopnea episodes;
- restores normal nocturnal oxygen saturation;
- decreases intermittent hypoxemia and hypercapnia;
- improves sleep architecture;
- reduces sympathetic nervous system activation; and
- enhances daytime alertness and cognitive performance.

Numerous randomized controlled trials have consistently demonstrated that CPAP therapy reduces the Apnea–Hypopnea Index by approximately **70–90%**, significantly improves excessive daytime sleepiness as measured by the **Epworth Sleepiness Scale**, lowers systolic and diastolic blood pressure, and substantially improves health-related quality of life.

Long-term adherence to CPAP therapy has also been associated with reduced risks of resistant hypertension, atrial fibrillation, myocardial infarction, ischemic stroke, and cardiovascular mortality. Owing to its excellent efficacy and favorable safety profile, CPAP remains the preferred therapeutic option for the majority of patients with moderate-to-severe obstructive sleep apnea.

Nevertheless, treatment success depends heavily on patient adherence. Appropriate mask fitting, humidification, patient education, behavioral support, and regular follow-up are essential for maximizing long-term compliance and therapeutic effectiveness.

Oral Appliance Therapy

Mandibular advancement devices (MADs) represent an effective alternative for patients with mild-to-moderate obstructive sleep apnea who are unable to tolerate CPAP therapy.

These custom-made intraoral devices advance the mandible anteriorly during sleep, enlarging the upper airway and reducing pharyngeal collapse. Although generally less effective than CPAP in reducing AHI, mandibular advancement devices have demonstrated significant improvements in





snoring, daytime sleepiness, and patient-reported quality of life, particularly among carefully selected patients.

Positional Therapy

Some individuals exhibit **position-dependent obstructive sleep apnea**, in which respiratory events occur predominantly in the supine position. Positional therapy aims to prevent sleeping on the back through behavioral techniques or specialized positioning devices.

Although positional therapy alone is rarely sufficient for severe disease, it may provide meaningful clinical benefit in selected patients with mild positional sleep apnea.

Surgical Treatment

Surgical intervention may be considered when anatomical abnormalities of the upper airway significantly contribute to obstruction or when conservative therapies fail to achieve adequate disease control.

Common surgical procedures include:

- **Tonsillectomy**
- **Uvulopalatopharyngoplasty (UPPP)**
- Nasal septoplasty
- Turbinate reduction
- Adenoidectomy
- Maxillomandibular advancement surgery

These procedures aim to enlarge the upper airway, reduce airflow resistance, and improve nocturnal ventilation. Careful preoperative evaluation, including upper airway endoscopy and imaging studies, is essential for selecting appropriate surgical candidates.

Management of Central Sleep Apnea

Treatment of central sleep apnea differs fundamentally from that of obstructive sleep apnea because therapy should primarily address the underlying disorder responsible for impaired respiratory control. Management may include optimization of chronic heart failure, treatment of neurological disease, reduction or discontinuation of opioid medications, correction of metabolic abnormalities, and supplemental oxygen therapy in selected cases.

Patients with persistent central sleep apnea despite optimal medical management may benefit from advanced ventilatory support modalities such as **adaptive servo-ventilation (ASV)** or bilevel positive airway pressure (BiPAP), provided no contraindications exist.

Emerging Therapeutic Approaches

Recent advances in sleep medicine have introduced several promising treatment modalities for patients who are unable to tolerate conventional CPAP therapy.

Among these, **hypoglossal nerve stimulation** has emerged as an effective option for carefully selected individuals with moderate-to-severe obstructive sleep apnea. Electrical stimulation of the hypoglossal nerve during inspiration activates the genioglossus muscle, thereby preventing upper airway collapse without the need for continuous positive airway pressure.





Additional areas of ongoing research include pharmacological modulation of upper airway muscle activity, personalized ventilatory support guided by artificial intelligence, and implantable neurostimulation technologies. Although these approaches remain under active investigation, they represent promising future directions in the management of sleep-disordered breathing.

Comprehensive Long-Term Management

Successful treatment of sleep apnea syndrome extends beyond the elimination of respiratory events. Long-term management requires regular clinical follow-up, monitoring of treatment adherence, reassessment of disease severity, optimization of cardiovascular risk factors, and management of associated metabolic and psychological comorbidities.

A multidisciplinary approach involving sleep medicine specialists, pulmonologists, cardiologists, neurologists, otorhinolaryngologists, endocrinologists, nutritionists, and primary care physicians is essential for achieving optimal clinical outcomes.

Clinical Perspective

Current scientific evidence consistently identifies **continuous positive airway pressure (CPAP) therapy** as the most effective evidence-based treatment for moderate-to-severe obstructive sleep apnea. When combined with lifestyle modification and individualized management of contributing risk factors, CPAP therapy substantially improves sleep quality, reduces daytime symptoms, lowers cardiovascular risk, and enhances long-term survival. Early diagnosis, individualized treatment planning, and sustained patient adherence remain the key determinants of successful management and improved quality of life in patients with sleep apnea syndrome.

Conclusion

Sleep apnea syndrome is a highly prevalent, multifactorial, and chronic disorder that represents a significant challenge for contemporary healthcare systems. Beyond being a nocturnal breathing disorder, it should be recognized as a systemic disease with profound respiratory, cardiovascular, neurological, endocrine, and metabolic consequences. The recurrent cycles of intermittent hypoxia, sleep fragmentation, oxidative stress, and sympathetic nervous system activation constitute the principal pathophysiological mechanisms responsible for disease progression and the development of long-term complications.

Early recognition of characteristic clinical manifestations, combined with timely application of evidence-based diagnostic methods, is essential for improving patient outcomes. **Polysomnography** remains the reference standard for diagnosing sleep apnea syndrome, enabling accurate differentiation between obstructive, central, and mixed forms of the disease while providing objective assessment of disease severity. The integration of validated screening questionnaires, overnight pulse oximetry, upper airway evaluation, and comprehensive cardiovascular assessment further enhances diagnostic accuracy and facilitates individualized treatment planning.

Current evidence consistently supports **continuous positive airway pressure (CPAP) therapy** as the first-line treatment for patients with moderate-to-severe obstructive sleep apnea. By maintaining upper airway patency throughout sleep, CPAP effectively reduces apnea and hypopnea events, restores normal oxygenation, improves sleep architecture, alleviates excessive daytime sleepiness,





and significantly decreases the risk of cardiovascular morbidity and mortality. When combined with lifestyle modification, weight management, and treatment of underlying comorbidities, CPAP therapy substantially improves both short-term clinical outcomes and long-term quality of life.

Successful management of sleep apnea syndrome requires a multidisciplinary, patient-centered approach involving sleep medicine specialists, pulmonologists, cardiologists, neurologists, otorhinolaryngologists, endocrinologists, and primary care physicians. Such collaboration enables comprehensive evaluation, individualized therapeutic decision-making, and long-term monitoring of treatment adherence and disease progression.

Future advances in sleep medicine, including home-based diagnostic technologies, wearable monitoring systems, artificial intelligence–assisted analysis, and novel neurostimulation therapies, are expected to further improve the early detection and personalized management of sleep apnea syndrome. Continued clinical research and implementation of evidence-based strategies will be essential for reducing the global burden of this increasingly prevalent disorder and improving long-term health outcomes. Increasing physician awareness and expanding access to early diagnostic services remain fundamental priorities for reducing the global burden of sleep apnea syndrome.

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