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Diagnosis and treatment of obstructive sleep apnea

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ABSTRACT

Obstructive sleep apnoea (OSA) is a prevalent disorder characterized by repeated episodes of partial or complete upper airway collapse during sleep, resulting in intermittent hypoxia, hypercapnia, and sleep fragmentation. Common in middle-aged and older adults, its pathogenesis is multifactorial, encompassing obesity, craniofacial abnormalities, altered upper airway muscle function, pharyngeal neuropathy, and fluid redistribution to the neck. These episodes trigger sympathetic activation, oxidative stress, and systemic inflammation, contributing to daytime sleepiness, road traffic accidents, and a spectrum of cardiovascular and metabolic comorbidities such as hypertension, arrhythmias, coronary artery disease, stroke, and diabetes mellitus. The efficacy of therapy in reversing chronic sequelae remains uncertain. Continuous positive airway pressure (CPAP) is the first-line treatment in severe cases, although patient adherence varies between 60–70%.

Alternative modalities, including bi-level positive airway pressure, adaptive servo-ventilation, oral appliances, and surgical interventions, may be employed. Weight reduction and combined treatment strategies hold promise but require validation in well-designed randomized controlled trials.

Keywords: obstructive sleep apnoea, overlap syndrome, polysomnography, sleep-disordered breathing, apnea

List of abbreviation: **AASM** – American Academy of Sleep Medicine, **AHI** - apnea-hypopnea index, **COPD** – chronic obstructive pulmonary disease, **CPAP** - continuous positive airway pressure, **EEG** - electroencephalogram, **MAD** - mandibular advancement devices, **NIV** - non-invasive ventilation, **OSA** - obstructive sleep apnea, **PSG** - polysomnography, **OS** – overlap syndrome, **$PaCO_2$** – partial pressure of carbon dioxide, **SpO_2** - the oxygen saturation of arterial blood hemoglobin measured using a pulse oximeter.

Clinical indicators and associated conditions suggesting OSA

In patients who exhibit two or more signs pointing toward sleep-disordered breathing, it is recommended to gather a thorough sleep history and proceed with more in-depth diagnostic evaluations. Special attention should be given to those with medical conditions linked to a higher incidence of these breathing irregularities [1,2]. The overlap syndrome of OSA and COPD should be suspected if the patient also demonstrates indicators of hypoventilation such as morning headaches, peripheral edema, low blood oxygen levels, or unexplained polycythemia [3,4]. The overlap syndrome of OSA and COPD should be suspected when the patient also exhibits signs of hypoventilation, such as morning headaches, peripheral edema, hypoxemia, or unexplained polycythemia [5,6].

Symptoms suggesting possible sleep-disordered breathing:	Conditions associated with an increased prevalence of sleep-disordered breathing:
Snoring	Overweight or obesity (including during pregnancy)
Apneas observed by others	Resistant arterial hypertension
Non-restorative sleep	Type 2 diabetes mellitus
Morning headache	Cardiac arrhythmias, especially atrial fibrillation
Unexplained excessive sleepiness or fatigue	History of stroke or transient ischemic attack
Nocturia	Chronic heart failure
Sensation of choking during sleep	Moderate or severe asthma
Fragmented sleep or insomnia	Polycystic ovary syndrome (PCOS)
Cognitive or memory impairment	Down syndrome
	Non-arteritic anterior ischemic optic neuropathy
	Hypothyroidism
	Acromegaly

Tabel 1. Symptoms and conditions suggesting the possibility of obstructive sleep apnea

Scales used in the diagnosis of OSA

When identifying patients who may require testing for sleep-disordered breathing, it is advisable to use the STOP-BANG questionnaire alongside the Epworth Sleepiness Scale [6]. Relying solely on the Epworth Scale to determine the need for a sleep study is not recommended, as some individuals with OSA do not exhibit excessive daytime sleepiness, and multiple other, unrelated factors can also cause such symptoms [7]. In patients suspected of having the overlap syndrome (OSA combined with COPD), performing basic spirometry before and after bronchodilator administration is essential to confirm a COPD diagnosis and to establish the severity of airway obstruction [8].

Diagnostic tests

The gold standard for diagnosing OSA remains overnight polysomnography (PSG) performed under supervised conditions in a dedicated sleep laboratory, where both neurologic (electroencephalogram) and cardio - respiratory parameters are analyzed during sleep [9]. Respiratory sensors identify reductions in ventilation, which are then categorized as apneas (nearly complete cessation of airflow lasting at least 10 seconds), hypopneas (a partial decrease in airflow for at least 10 seconds), or arousals related to respiratory effort (minor airflow changes caused by heightened upper - airway resistance leading to awakenings) [10,11]. The apnea - hypopnea index (AHI), a commonly used benchmark for sleep - related

breathing disorders, is calculated by dividing the total number of apneas and hypopneas by the number of hours of sleep. According to the American Academy of Sleep Medicine (AASM), OSA is classified by AHI as mild (5 - 15 events/hour), moderate (>15 - 30 events/hour), or severe (>30 events/hour) [12].

In recent years, there has been a growing focus on simpler and more accessible diagnostic options for obstructive sleep apnoea, particularly the use of type III devices, commonly referred to as home sleep apnea tests (HSAT) [9,13,14]. These devices enable unsupervised testing in the patient's home, with polygraphy being the most frequently performed study. While polysomnography remains the gold standard for OSA diagnosis, it is often considered cumbersome, costly, and time-consuming. Consequently, home-based diagnosis and treatment methods have been increasingly explored [15]. Randomized controlled trials have demonstrated that, for certain patients, home - based approaches are as effective as laboratory - based diagnosis and management. However, it is important to note that major studies excluded patients with potentially complex conditions, such as lung disease, heart failure, or neuromuscular disorders, underscoring that home testing is not suitable for all cases [16].

Type I	**Polysomnography in a sleep laboratory** – recording of ≥7 variables, including those necessary for the assessment of sleep structure and breathing: electroencephalography (EEG), electro-oculography (EOG), electromyography (EMG), electrocardiography (ECG), pulse oximetry, respiratory effort, and airflow through the upper airways.
Type II	**Home (unsupervised) polysomnography** – recording of ≥7 parameters (as in type I devices).
Type III	Portable multi-channel monitoring devices recording 4–7 parameters, always including pulse oximetry, respiratory effort, and airflow through the upper airways
Type IV	Portable monitoring devices recording 1 or 2 parameters, always including pulse oximetry

Tabel 2. Classification of devices for diagnosing sleep-disordered breathing according to the American Academy of Sleep Medicine, the American College of Chest Physicians, and the American Thoracic Society [16].

Diagnostic tests in suspected overlap syndrome of OSA and COPD

The diagnosis of acute hypoxemic-hypercapnic respiratory failure relies on analyzing arterial blood gas (ABG) or arterialized capillary blood gas samples collected at rest [17]. For evaluating sleep - disordered breathing in patients suspected of having overlap syndrome of OSA and COPD, polygraphy serves as the primary diagnostic method, whether conducted in a hospital or at home. Incorporating transcutaneous PaCO2 monitoring into the sleep study can provide additional insights that may guide treatment decisions [8]. If the polygraphy results are negative, polysomnography (PSG) should be considered to definitively rule out OSA.

PaCO2 monitoring is the only reliable method for diagnosing hypoventilation. This monitoring during sleep should be conducted continuously using non - invasive techniques, typically through transcutaneous measurement. This approach helps track PaCO2 changes, confirm the absence of hypoventilation, and assess the progression of hypercapnia caused by repeated apneas during sleep.

These findings are critical for determining the most appropriate ventilation support, such as continuous positive airway pressure (CPAP) therapy or non-invasive mechanical ventilation (NIV) [8,13,15].

Treatment

The management of OSA is designed to relieve symptoms, improve quality of life, reduce complications, and lower mortality risk [18,19]. Treatment options encompass lifestyle changes, pharmacological interventions, continuous positive airway pressure (CPAP), oral appliance therapy such as tongue-retaining devices or mandibular advancement devices (MAD), and surgical interventions. Surgical approaches include procedures like tracheostomy, uvulopalatopharyngoplasty, maxillomandibular advancement, nasal surgery and hypoglossal nerve stimulation. Each approach is tailored to the patient's specific needs to achieve optimal outcomes [20,21].

Behavioral interventions encompass psychological support, smoking cessation, abstaining from alcohol and sedatives, engaging in regular aerobic activity, achieving weight loss, and avoiding sleeping in the supine position. These strategies target factors that can aggravate OSA symptoms [14]. Psychological support involves open communication between doctors and patients, addressing their concerns, and educating them about the link between OSA and systemic diseases. Alcohol reduces the tension of airway muscles and raises the frequency of apnea episodes during sleep [22]. Moreover, it lengthens the period of oxygen deprivation by slowing down arousal, and thus clearly hinders effective OSA treatment [23]. A prior study indicated that cigarette smoking may contribute to oropharyngeal narrowing and exacerbate the severity of OSA [24]. Additionally, exposure to secondhand smoke has been strongly linked to OSA. Smoking may worsen OSA by disrupting sleep patterns, triggering inflammation in the upper airway, and impairing the neuromuscular function and arousal responses of the upper airway [25,26]. Reducing body weight can lead to an improvement in the apnea-hypopnea index (AHI) in patients with obesity-related OSA and is recommended for all overweight or obese individuals who are not candidates for other treatment options [27]. For those with mild or no symptoms, weight loss alone may serve as an effective initial therapy. Recent research has demonstrated that in obese individuals, weight reduction is effective in decreasing tongue fat volume, which directly correlates with a decrease in the AHI [28]. Various anatomical and physiological alterations in the respiratory system can elevate the likelihood of sleep - disordered breathing when moving from a nonsupine to a supine position [29]. These include an increased loop gain, a decrease in airway size, and a reduction in functional residual lung capacity. Traditional positional therapy, commonly known as the “tennis ball technique” (TBT), requires attaching a bulky item to the patient’s back to prevent sleeping in the supine position. Although this method effectively reduces the

amount of time spent in the supine position and is both simple and cost-efficient, it frequently causes patient discomfort, resulting in low long-term compliance [30,31].

Continuous positive airway pressure is the preferred treatment for adults with symptomatic moderate to severe OSA [32]. Using CPAP for a minimum of 6 hours per night, which includes coverage of REM sleep typically occurring in the latter half of the sleep cycle, has been linked to a decreased risk of cardiovascular events [33].

It involves a motorized device that delivers filtered air through various types of facial mask interfaces at a positive pressure tailored to the individual needs of each patient. This positive pressure is maintained throughout the entire breathing cycle, helping to keep the collapsible upper airway open while the patient breathes independently. Unlike non-invasive ventilation, where alternating positive pressure assists inhalation to expand the lungs and enhance gas exchange in cases of ventilatory failure, CPAP primarily stabilizes the airway [34]. Using CPAP typically causes only mild discomfort, most commonly related to mask issues such as skin irritation, air leakage, or noise, as well as side effects from the air pressure, such as rhinitis. Despite these minor drawbacks, is regarded as a safe and effective therapy for sleep apnea and is widely adopted for managing this condition globally [32,35].

The most prevalent types of oral appliances are mandibular advancement devices (MAD) and tongue retention devices. They are recommended to use by patients with mild to moderate OSA who can't CPAP or choose to avoid its use [36,37]. These devices are made of steel plates that fit over the upper and lower teeth. The interconnected plates are adjustable, enabling the mandible to move forward in relation to the maxilla. This adjustment is designed to widen the oropharynx and velopharynx during sleep while stimulating stretch receptors to minimize airway collapse and enhance the openness of the upper airway [38]. Managing OSA with an oral appliance calls for a team-based strategy that includes both a sleep specialist and a dental professional. Mandibular advancement devices generate opposing forces on the teeth and jaw, and may place pressure on the gums and oral lining, depending on their construction. These mechanical effects can lead to discomfort, as well as dental and skeletal alterations over time [39,40].

Before the introduction of CPAP therapy, tracheotomy was a common treatment for severe OSA, as it effectively bypassed airway obstructions and significantly alleviated the condition. However, it is now rarely used in the treatment of OSA [41].

One of the most frequently performed surgeries for OSA is uvulopalatopharyngoplasty (UPPP), which involves removing the tonsils, uvula, and parts of the soft palate. Numerous modifications of UPPP have been reported. However, because UPPP alone does not reliably normalize AHI, the AASM does not endorse it as the sole treatment for moderate to severe OSA [42].

Nasal procedures (such as septal correction or polyp removal) can help reduce snoring; however, there is no evidence that nasal surgery alone effectively addresses sleep apnoea [43].

Hypoglossal nerve stimulation represents an advanced surgical intervention aimed at improving the activity of pharyngeal dilator muscles during sleep. The most commonly utilized technique and commercial implantation system involves placing the stimulating electrode on the medial branch of the right hypoglossal nerve to enhance the corresponding

side of the tongue [44]. A breathing sensor is positioned between the internal and external intercostal muscles to measure inspiratory effort, and an implantable pulse generator is implanted in the chest wall to activate the hypoglossal nerve electrode in response to respiratory effort. While hypoglossal nerve stimulation appears both effective and generally well tolerated, it remains an invasive option and is more expensive than using oral appliances or CPAP therapy [18,45].

Treatment approach for the overlap syndrome of OSA and COPD

CPAP therapy can be considered the first-line treatment if severe hypercapnia (PaCO2 <53 mm Hg) is not present. For patients with nocturnal hypoventilation and significant hypercapnia (PaCO2 ≥53 mm Hg), non - invasive ventilation (NIV) is recommended [8]. If side effects related to positive pressure therapy affecting the respiratory system occur, the use of humidification and air-warming systems should be considered. If hypoxemia persists during CPAP therapy despite the reduction of apneas, hypopneas, and hypoventilation, supplemental oxygen should be added to CPAP, while simultaneously investigating other potential causes of hypoxemia [20,34]. The decision between CPAP and NIV in the management of overlap syndrome of OSA and COPD should also take the AHI value into account. Patients with high AHI values are often more responsive to CPAP therapy. In such cases, opening the upper airways with CPAP may be sufficient to eliminate apneas and reduce hypercapnia, which in these patients is often primarily caused by the presence of apneas [8,32].

Conclusions

Obstructive sleep apnoea (OSA) is a prevalent, multifactorial disorder associated with significant cardiovascular and metabolic comorbidities. Accurate diagnosis relies on robust evaluation methods, with overnight laboratory polysomnography considered the gold standard, although home-based testing options are increasingly utilized in selected patient populations. Treatment approaches must be individualized and may include lifestyle modifications, continuous positive airway pressure (CPAP), oral appliances, or surgical interventions. CPAP remains the first-line therapy for moderate to severe OSA, improving symptoms, reducing comorbid risk, and lowering mortality when used consistently. Oral appliances offer an alternative for those intolerant of CPAP, while surgical procedures and emerging therapies like hypoglossal nerve stimulation have a role in refractory cases. In the overlap syndrome of OSA and COPD, careful selection and monitoring of ventilatory support - ranging from CPAP to non-invasive ventilation (NIV) - are paramount, guided by factors such as $PaCO_2$ levels and the apnea - hypopnea index (AHI). Future research should focus on long-term clinical outcomes, the comparative effectiveness of diagnostic methods, and strategies to enhance patient adherence and treatment precision.

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