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OBSTRUCTIVE SLEEP APNEA AND ITS IMPACT ON ATHLETES AND SPORTS PERFORMANCE – THE REVIEW

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ABSTRACT

Obstructive sleep apnea (OSA) is a prevalent and underdiagnosed condition with significant systemic health implications. It is characterized by recurrent upper airway collapse during sleep, leading to intermittent hypoxia, sleep fragmentation, and an array of comorbidities. Among the risk factors, obesity is the most common, but other factors such as sex, age, tonsillar hypertrophy, and the presence of comorbid conditions also play a role. Untreated obstructive sleep apnea (OSA) can lead to cardiovascular diseases, diabetes, strokes, and even sudden death. Diagnosis is based on medical history, clinical examination, and most importantly, polysomnographic testing. Treatment includes lifestyle modifications, continuous positive airway pressure (CPAP) therapy, and in some cases, surgical interventions. This literature review synthesizes current evidence regarding the pathophysiology, clinical presentation, diagnostic approaches, and therapeutic options for OSA in adults.

KEYWORDS

Obstructive Sleep Apnea, Sleep-Related Breathing Disorders, Apnea, Continuous Positive Airway Pressure (CPAP)

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Introduction

More than one billion people worldwide suffer from sleep-related breathing disorders, with obstructive sleep apnea being by far the most common form. Obstructive sleep apnea (OSA) is a common chronic sleep-related breathing disorder characterized by repetitive upper airway collapse during sleep, which causes sleep fragmentation, oxygen desaturation, and excessive daytime sleepiness.[1] Pharyngeal collapse could be complete (causing apnoea) or partial (causing hypopnoea).[2] Although in the past OSA prevalence was estimated as being 2%–4% among adults, more recent studies have suggested prevalence of OSA of 56% and of moderate to severe OSA of 37%.[3] The accepted criteria for OSA severity rank it according to the apnea-hypopnea index (AHI) as mild, 5–15 events/h, moderate, >15–30 events/h, and severe, >30 events/h.[3,4] AHI is an important indicator in the diagnosis of sleep apnea, calculated by dividing the number of apnea and hypopnea episodes by the number of hours of sleep. The global prevalence of OSA is increasing, paralleling the rise in obesity and aging populations.[2] Despite its significant health burden, OSA remains largely undiagnosed, particularly among certain demographics due to health inequities and limited access to specialized care.[4,5]. Factors such as reduced access to healthcare, lack of awareness, and socioeconomic barriers result in delayed diagnosis and inadequate treatment.[5] Although several treatments exist, they are often either poorly tolerated or only partially alleviate abnormalities. Thus, improvement of patient adherence to existing treatments and development of new treatments (or combinations of treatments) are needed.[2]

Patophysiology

The primary mechanism in OSA is the collapse of the pharyngeal airway due to impaired neuromuscular control and anatomical predisposition.[3] Factors such as obesity, craniofacial abnormalities, and reduced muscle tone contribute significantly to airway collapsibility.[6,7] Anatomical constraints, such as macroglossia, adenotonsillar hypertrophy, or retrognathia, exacerbate upper airway obstruction. In particular, supine position has been identified as a trigger for airway collapse due to gravitational effects on soft tissues.[7] Recent perspectives emphasize the multifactorial pathogenesis, including muscle dysfunction and ventilatory control instability.[8] Moreover, alterations in chemosensitivity and ventilatory responses during sleep reduce upper airway patency.[7] Dewald (2022)[8] has highlighted the importance of evaluating muscular tone and functionality, suggesting that structural interventions should be paired with neuromuscular retraining for optimal results.

It is believed that inflammation and OSA are strongly related, systemic inflammatory markers levels are upregulated in subjects with OSA. Several studies have highlighted the role of oxidative stress in the genesis of endothelial damage in both adult and pediatric OSA patients. Many studies are being conducted on the use of biomarkers for the diagnosis of OSA, based on the high levels of pro-inflammatory factors.[6] Sex differences also influence OSA expression, with varying symptom profiles and risk factors between men and women.[9]

Risk Factors

Obesity and male sex are major risk factors for obstructive sleep apnoea. Obesity could increase the likelihood of airway collapse by directly affecting the anatomy of the upper airway.[2] Several large epidemiological studies have demonstrated a strong association between weight gain and an increase in the odds of developing OSA. More recently, clinicians are recognising that the development of OSA and its subsequent sleep fragmentation may contribute to accelerated weight gain. Many patients report rapid increases in weight in the year prior to OSA diagnosis.[10]

Nevertheless, clinicians need to be aware of other risk factors, such as increasing age, perimenopausal or postmenopausal status in women, craniofacial abnormalities (e.g., small mandible, retruded mandible, palatine tonsillar hypertrophy, deviated nasal septum, elongated uvula), genetic factors and ethnic origin, which affect craniofacial anatomy, allergies and upper respiratory tract infections, alcohol and sedative use, and smoking.[2,10,15]

Clinical Presentation

Typical symptoms include loud snoring, observed apneas, excessive daytime sleepiness, morning headaches, and non-restorative sleep. In adults, the Epworth Sleepiness Scale (ESS) is often used to quantify daytime sleepiness, aiding in clinical assessment. The ESS is used for self-assessment; the patient completes a questionnaire in which they rate the likelihood of falling asleep in eight common daily life situations. Many patients also report mood disturbances, cognitive impairments, and decreased libido. Moreover, there may also be a sensation of shortness of breath during sleep, heart palpitations, increased sweating, and frequent urination at night. Dryness in the mouth upon waking is caused by mouth breathing. In pediatric populations, OSA manifests differently, often with hyperactivity, poor academic performance, and behavioral issues, necessitating specialized diagnostic and therapeutic approaches.[6]

OSA has well-established associations with cardiovascular diseases, including systemic hypertension, coronary artery disease, stroke, and arrhythmias.[4,11,15] The inflammatory cascade triggered by intermittent hypoxia contributes to endothelial dysfunction and atherosclerosis. It also plays a role in metabolic syndrome, with a bidirectional relationship with type 2 diabetes mellitus, contributing to poor glycemic control and insulin resistance.[3,12] There is growing evidence of a bidirectional relationship between OSA and comorbidity, especially for heart failure, metabolic syndrome and stroke.[12] Data from small human studies suggest fluctuations in blood pressure can influence upper airway tone by demonstrating inhibitory changes on electromyogram (EMG). This suggests that reduction in blood pressure may improve airflow and reduce OSA severity.[12]

Epidemiological data suggest OSA is also associated with depression, especially among children and adolescents at high risk.[13] Thus, comprehensive screening strategies are needed across all age groups. Furthermore, OSA is linked to neurocognitive dysfunction, depression, and increased all-cause mortality. Gresova et al. (2023)[14] underscore the public health burden posed by untreated OSA, noting its role in increasing traffic accidents, workplace injuries, and healthcare costs.

In women, OSA is often underdiagnosed or misdiagnosed due to differences in clinical presentation and sleep study findings compared to men. Women with OSA experience a greater decline in quality of life and more frequently report daytime fatigue, lack of energy, morning headaches, mood disturbances, and nightmares compared to men. AHI is not a sufficient criterion for assessing clinical severity in women, as OSA in women presents with a lower AHI, shorter apnea episodes, and a lower prevalence of supine position-related OSA.[9]

OSA increases perioperative morbidity due to heightened sensitivity to anesthetic agents, risk of airway collapse, and postoperative hypoventilation. Obstructive sleep apnoea presents a unique perioperative challenge. Patients with obstructive sleep apnoea have up to a four-fold risk of difficult intubation.[16] Preoperative screening using STOP-BANG or other tools, along with intraoperative monitoring, is critical.[16] Chambers et al. (2022)[16] recommend implementing perioperative care protocols for patients with diagnosed or suspected OSA to reduce adverse outcomes.

Diagnosis

The gold standard for diagnosis remains overnight polysomnography (PSG), which records multiple physiological parameters during sleep.[17] PSG is conducted in a hospital setting. The examination allows for the assessment of brain activity (EEG), heart rhythm (EKG), body movements, body position, blood oxygen levels, abdominal and chest respiratory movements, airflow through the upper airways, and the recording of snoring. The results are analyzed by a specialist who determines the type and severity of the apnea. However, PSG is expensive and time consuming, which hinders its utility for the prevention, early diagnosis and treatment of OSA.[18] Home sleep apnea testing (HSAT) offers a more accessible option for diagnosing moderate to severe OSA in patients without significant comorbidities.

Innovations like drug-induced sleep endoscopy (DISE) offer dynamic assessment of airway collapse, guiding surgical decision-making.[19] (DISE) involves endoscopic upper airway examination during unconscious sedation, identifying structures that contribute to airway obstruction.[1] DISE not only provides clinicians with valuable information about the sites, pattern and degree of obstruction but also provides profound insights into the upper airway collapse mechanism during sleep.[19]

OSA causes changes in metabolism associated with increased anaerobic metabolism and oxidative stress, as well as disrupted lipid metabolism. Biomarker studies and metabolomic profiling are being explored for phenotype-specific diagnosis and management.[18]

Ioachimescu (2024)[20] noted that metrics like oxygen desaturation index (ODI) may serve as effective tools in stratifying disease severity, especially in remote or resource-limited settings.

Treatment Options

Lifestyle interventions, including weight loss, positional therapy (such as avoidance of the supine position during sleep), and alcohol and sedatives avoidance, are foundational in OSA management.[1] Intensive lifestyle intervention in persons with OSA and obesity is associated with both weight loss and sustained reduction in the AHI, with resolution of OSA in many persons.[3]

CPAP therapy (continuous positive airway pressure) remains the standard of care, significantly improving symptoms and quality of life. [1] Nonetheless, adherence remains a challenge, prompting exploration of alternative modalities. CPAP is the most effective OSA treatment, although mandibular advancement devices are easier for patients to use. Oral appliances that advance the mandible (e.g., use of tongue-retaining devices or use of orthodontic or mandibular advancing appliances) can be beneficial in mild to moderate cases.[17,21] Lower efficacy may be balanced by greater adherence to this form of treatment compared with continuous positive airway pressure (80%–90% v. 50%–70%).[17] Surgical options, including uvulopalatopharyngoplasty (UPPP), which involves removal of the tonsils, uvula, and posterior velum, and hypoglossal nerve stimulation, are considered for patients refractory to conservative treatment.[1,17] Surgeons surgically implant an upper-airway stimulation device in OSA patients who have difficulty tolerating or adhering to CPAP therapy. Neurostimulation for stability of the upper airway during sleep was proposed as a less invasive and more effective option for selected patients.[1] Aggressive surgeries—eg, maxilla-mandibular advancement—are probably more effective than simple surgeries, but many patients would rather avoid major surgery for obstructive sleep apnoea.[2]

Pharmacotherapy, myofunctional therapy, and neuromuscular retraining represent emerging strategies with potential to complement existing modalities.[8,17] Martinez-Garcia (2022)[22] advocates for a multidimensional approach to therapy, integrating clinical phenotyping to personalize care. Clinical phenotyping would be based on evaluating the severity, disease activity, and the impact of the disease on the patient's life.[22] We must be aware that, in the world of progress towards precision medicine and personalized treatment, OSA lags behind other respiratory diseases and that the scientific sleep community should focus its efforts on reversing this situation as soon as possible.[22]

Residual daytime sleepiness despite effective treatment of OSA may occur. Causes include chronic sleep restriction, central sleep apnea that comes to light with therapy using continuous positive airway pressure, another sleep disorder (e.g., narcolepsy), chronic medical or psychiatric conditions (e.g., depression) or adverse effects owing to medications (e.g., sedatives, antihistamines, β-blockers). About 5% of patients have persistent sleepiness and may benefit from drugs to promote wakefulness.[17]

The Impact of Obstructive Sleep Apnea on Physical Performance and Athletes

Obstructive sleep apnea (OSA) can significantly affect physical performance, recovery, and overall well-being, making it a relevant concern not only for the general population but also for physically active individuals and elite athletes.

Several studies have shown that individuals with untreated OSA demonstrate decreased maximal oxygen uptake (VO_2 max), increased daytime sleepiness, reduced reaction times, and impaired decision-making, which may compromise both training capacity and competitive outcomes.[23,24] Additionally, growth hormone secretion can be disrupted in OSA patients, negatively influencing muscle recovery and adaptation to training.[25]

In the context of sports science and medicine, integrating OSA screening and management into athlete health programs may serve as a key factor in optimizing performance and prolonging careers, particularly in sports where recovery and respiratory efficiency are crucial.

Conclusions

Obstructive sleep apnea (OSA) is a common yet significantly underdiagnosed sleep-related breathing disorder with substantial systemic health consequences. Obstructive sleep apnoea represents a sizable public health and economic burden. A comprehensive approach encompassing accurate diagnosis, individualized therapy, and public health strategies is necessary to address its growing burden. OSA remains a major public health challenge, and improving its detection and treatment adherence is essential to reducing complications and improving patient outcomes. Collaborative efforts across disciplines and continued innovation in diagnostics and therapeutics are essential to reduce the prevalence and impact of this condition.

Disclosure

Author's Contribution

Conceptualization - Kinga Wiechnik, Methodology - Kinga Wiechnik, Joanna Wrona, Aleksandra Kaźmierczyk, Jędrzej Kęsik, Daria Madycka, Kacper Michta, Małgorzata Słaboń, Karol Stępniak, Wiktor Telega, Aleksandra Wójcik; Software - Kinga Wiechnikk, Joanna Wrona, Aleksandra Kaźmierczyk, Jędrzej Kęsik, Daria Madycka, Kacper Michta, Małgorzata Słaboń, Karol Stępniak, Wiktor Telega, Aleksandra Wójcik; Check - Kinga Wiechnik, Joanna Wrona, Aleksandra Kaźmierczyk, Jędrzej Kęsik, Daria Madycka, Kacper Michta, Małgorzata Słaboń, Karol Stępniak, Wiktor Telega, Aleksandra Wójcik; Formal analysis - Kinga Wiechnik, Joanna Wrona, Aleksandra Kaźmierczyk, Jędrzej Kęsik, Daria Madycka, Kacper Michta, Małgorzata Słaboń, Karol Stępniak, Wiktor Telega, Aleksandra Wójcik; Investigation - Kinga Wiechnik, Joanna Wrona, Aleksandra Kaźmierczyk, Jędrzej Kęsik, Daria Madycka, Kacper Michta, Małgorzata Słaboń, Karol Stępniak, Wiktor Telega, Aleksandra Wójcik; Resources - Kinga Wiechnik, Joanna Wrona, Aleksandra Kaźmierczyk, Jędrzej Kęsik, Daria Madycka, Kacper Michta, Małgorzata Słaboń, Karol Stępniak, Wiktor Telega, Aleksandra Wójcik; Data curation - Kinga Wiechnik, Joanna Wrona, Aleksandra Kaźmierczyk, Jędrzej Kęsik, Daria Madycka, Kacper Michta, Małgorzata Słaboń, Karol Stępniak, Wiktor Telega, Aleksandra Wójcik; Writing (rough preparation) - Kinga Wiechnik, Joanna Wrona, Aleksandra Kaźmierczyk, Jędrzej Kęsik, Daria Madycka, Kacper Michta, Małgorzata Słaboń, Karol Stępniak, Wiktor Telega, Aleksandra Wójcik; Writing (review and editing) - Kinga Wiechnik, Joanna Wrona, Aleksandra Kaźmierczyk, Jędrzej Kęsik, Daria Madycka, Kacper Michta, Małgorzata Słaboń, Karol Stępniak, Wiktor Telega, Aleksandra Wójcik; Visualization -Kinga Wiechnik, Joanna Wrona, Aleksandra Kaźmierczyk, Jędrzej Kęsik, Daria Madycka, Kacper Michta, Małgorzata Słaboń, Karol Stępniak, Wiktor Telega, Aleksandra Wójcik; Supervision - Kinga Wiechnik; Project administration - Kinga Wiechnik;

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