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Obstructive Sleep Apnea Syndrome: Perspective on risk factor and management.

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Abstract

People who are physically prone to the disorder obstructive sleep apnea (OSA) have upper airway collapse while they are asleep, which frequently results in hypoventilation, hypoxia, and awakenings from sleep. Daytime hypersomnolence, cognitive impairment, cardiovascular illness, metabolic dysfunction, respiratory failure, and pulmonary hypertension are only a few of the disorder's significant clinical manifestations. The most typical complaint is a loud, annoying snore that is interrupted frequently, along with sleep disturbances, excessive daytime sleepiness (EDS), or fatigue. Due to the nocturnal nature of the respiratory problems, OSAS may go undiagnosed for a considerable amount of time, but the effects can be observed in daytime function impairment. The burden of OSA and its consequences on public health is anticipated to rise as obesity rates, a key risk factor for OSA, rise. In this piece of writing, we go through the reasons behind OSA's onset and the concomitant neurocognitive and cardiometabolic conditions that it causes as well as the various therapeutic alternatives for treating OSA, including new pharmacotherapeutic advancements.

Keywords: Obstructive Sleep Apnea Syndrome, Obesity, Oral appliances, Anatomical factors.

Introduction

The grave medical disorder known as obstructive sleep apnea syndrome (OSAS) is characterised by recurrent partial or total obstruction of the upper airway while sleeping (1). Obstructive sleep apnoea (OSA) is a common condition that has a strong link to obesity. It is characterised by recurrent partial or total collapse of the upper airway while you are sleeping, which impairs the exchange of gases and disturbs the quality of your sleep. An elevated risk for congestive heart failure, hypertension, myocardial infarction, and coronary artery disease are all frequently linked to OSAS (2). Additionally, it has detrimental immediate health effects such excessive daytime sleepiness (EDS), which can eventually reduce cognitive function and alertness (3). Obstructive sleep apnea is increasingly being shown to be an independent risk factor for a poor cardio-metabolic profile (4), in addition, despite the fact that the majority of the causal role and mechanisms are still not fully understood, it has been linked to an increase in cardiovascular and cerebrovascular morbidity and death (5). The majority of persons affected are still undiagnosed despite recent developments in diagnostic technologies in this area and increasing public awareness of obstructive sleep apnea (6). Obstructive sleep apnea syndrome (OSAS) is a sleep condition that, by definition, is characterised by frequent breathing cessation during sleep or by prolonged absence of breathing lasting 10 seconds or longer despite an effort to breathe.

An individual has obstructive sleep apnoea-hypopnoea syndrome (7) if they meet criteria A, B, and/or C in addition to criteria C. The following are the criteria:

A. Prolonged daytime sleepiness that cannot be attributed to any other causes.

B. Two or more symptoms of daytime fatigue, choking or gasping while sleeping, poor concentration, frequent awakenings from sleep, and unrefreshing sleep that cannot be explained by other circumstances.

C. Breathing obstructions during sleep are detected by overnight monitoring in at least 5 instances per hour.

A diagnosis of OSAS can be made if there are five or more apnea/hypopnea episodes per hour of sleep, along with clinical symptoms like insomnia, daytime sleepiness, hypertension, etc., or if there are at least 15 events per hour without any additional symptoms, according to the American Academy of Sleep Medicine. When there are 5 to 15 events per hour, OSAS is considered mild, 30 or more events per hour is considered moderate, and more than 30 events per hour is considered severe (8).

Classification

Central sleep apnea: In this form, airflow at the mouth and nostrils ceases simultaneously with termination of respiratory muscle activity.

Obstructive sleep apnea: it is a common form of the condition. It is characterised by upper airway blockage induced by sleep that prevents airflow while maintaining respiratory centre drive, respiratory effort, and diaphragmatic contraction. (9)

Mixed sleep apnea: This condition combines obstructive and central apnea. It starts with a central apnea episode in which there is no airflow at the mouth or nostrils and no respiratory muscle activity is visible. When the pattern is complete, there is cessation of airflow at the mouth and nostrils, with an episode of obstructive apnea. (9,10)

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The severity of obstructive sleep apnea (OSA) based on the apnea hypopnoea index (AHI).

	OSA severity
AHI < 5	Normal or primary snoring
5 < AHI < 20	Mild
20 < AHI < 40	Moderate
AHI > 40	Severe

Etiology

Old age, male sex, and obesity are the key risk factors for obstructive sleep apnea, while the underlying mechanisms are yet unknown.

Age: After the age of 65, it appears that the prevalence of sleeping disorders breathing in the elderly increases(11). Numerous research aim to pinpoint the precise reason for the impact of ageing on sleep apnea, but no definitive findings have been made so far.

The lengthening of the soft palate, increased fat deposition in the parapharyngeal region, and modifications to the body's structure in the area around the pharynx area have all been proposed as potential causes of the increasing occurrence of obstructive sleep apnea in the elderly(12)

Sex: It is unclear why men experience OSA at a higher rate than women. It can be related to the upper airway's anatomical and functional characteristics as well as to the ventilatory response during sleep arousals(13). According to imaging studies, men have more fat deposited around their pharyngeal airways than women (14).

Obesity: Obesity, or visceral obesity, is the main risk factor for the development of OSA. It is believed to be linked to anatomical changes that increase adiposity around the pharynx and body, which predispose to upper airway blockage during sleep.

Family history and genetic predisposition: It is believed that genetic and familial aggregation factors contribute to the emergence of OSA. Family susceptibility to OSA rises directly with the number of affected relatives, with first-degree relatives of people with OSA having a 1.5–2.0 increase in relative risk when compared to those without OSA(15,16)

Craniofacial anomalies: The upper airway's structural components may change its mechanical characteristics. The variance in OSA risk among various racial and ethnic groups may be partially explained by variations in cranial morphology. Particularly in people who are not obese, craniofacial anomalies have a significant role in the development of OSA.(17)

Smoking and alcohol consumption: It has been established that both smoking and alcohol use increase the risk of OSA. Snoring and sleep-disordered breathing are more common among people who smoke(18,19). In the Winconsin Sleep Cohort Study, current smokers were substantially more likely to have moderate or worse OSA than never smokers (odds ratio, 4.44) (20). It makes perfect sense given that smoking causes airway inflammation and injury, which may alter the morphological and functional characteristics of the upper airway and raise the danger of collapsibility during sleep. Alcohol consumption can lengthen apnoea episodes, reduce arousals, increase the frequency of occlusive episodes, and exacerbate hypoxemia; however, the underlying mechanisms remain unclear. (21)

Anatomical factors: It is believed that obesity and the growth of soft tissue structures in the upper airway are the primary causes of OSA. Additionally, upper airway irregularities are known to dramatically enhance the risk of it collapsing while the individual is sleeping, even if they are not obese. The anatomical defects causing airway obstruction are made more complex by additional

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factors, such as physiologic and functional failure of the upper airway dilating muscles.

Physical Examination: The clinician should conduct a thorough head and neck examination on each patient and evaluate their respiratory, cardiovascular, and neurological systems. The procedure of detecting physical examination findings that are consistent with OSA is thoroughly reported in the literature (22). Obesity (body mass index [BMI] R30 kg/m2), craniofacial abnormality, increased neck circumference (>17 in in men; >16 in in women), anatomic nasal airway deformity, elongated/enlarged soft palate, high-arched/narrow palate, hypertrophic tonsils and adenoids, lateral peritonsillar narrowing, and macroglossia are all physical findings that are suggestive of OSA. During the examination, specific attention should be directed to any potential locations of upper airway blockage. External inspection and speculum or intranasal examination can both be used to assess the nasal complex.

Imaging: Nasopharyngoscopy, cephalometrics, computed tomography (CT), and magnetic resonance imaging (MRI) are the main upper airway imaging techniques. These imaging techniques have been applied to study how breathing, losing weight, dental appliances, and surgery affect the upper airway. The airway and surrounding soft tissue structures can be measured in three dimensions using MRI and CT (23). New imaging methods using computational fluid dynamics (CFD) are now available for assessing the upper airway in OSAS and perhaps forecasting therapeutic treatments. The computers are being used in this extremely complex bioengineering process (24).

Discussion

1. Pharmacological and Non- surgical treatment:

Modulation of monoamine and serotonin: The non-sedating tricyclic antidepressant protriptyline has been linked in studies to a reduction in OSA symptoms (25) Compared to placebo, protriptyline 20 mg administered orally at bedtime for two weeks significantly decreased REM sleep and increased nocturnal oxygenation. The length and frequency of apnea while sleeping, however, remained unchanged (26). According to an early study, L-tryptophan, a precursor to serotonin, caused a mild reduction in OSA (27). Serotonergic drugs are now receiving a lot of interest as a result of this. Serotonin (5-hydroxytryptamine [5-HT]) is known to affect upper airway dilator motor neurons and sleep induction.

Acetylcholine mechanisms: Agents that modulate cholinergic activity provide as an example of an alternate strategy that may be related to sleep-stage modulation. The control of the respiratory drive is mediated by acetylcholine, one of the major neurotransmitters active during REM sleep. Acetylcholine has a key role in the secretory activity of the upper airway mucosa, which may lower airway compliance and, consequently, collapsibility during sleep. Chemosensitization can also be brought on by central cholinergic processes. Reduced thalamic cholinergic nerve terminal density and the severity of OSA were shown to be correlated in a study of patients with multi-system atrophy(28).

Amphetamine with theophylline: Theophylline affects breathing in a variety of ways, including centrally inhibiting adenosine and stimulating diaphragm contraction. In a recent uncontrolled trial of oral theophylline in 13 patients with compensated heart failure and periodic breathing, these effects on central

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apnea were verified (29). This study showed an improvement in oxygenation, a reduction in mostly central events by over 50%, and no adverse effects on sleep.

GABA and glutamate mechanisms: Sabeluzole, a potential glutamate antagonist, decreased hypoxemic episodes in a small double-blind, controlled research conducted in individuals with moderate to severe OSA (30).

Clinical protocol for oral appliance therapy:

The management of oral appliances (OAs) in patients receiving treatment for snoring or OSA is advised using the following therapeutic regimen. (31)

- A thorough physical examination. Patients who are completely edentulous might not be ideal for mandibular repositioning therapy because they might lack sufficient intraoral retention to keep the device in place while they sleep. There should be no limitation in jaw movements. Some people may not be eligible for an OA if they have a history of significant temporomandibular joint (TMJ) pathology or chronic joint pain.
- Before starting therapy with an OA, a full examination of the diagnostic criteria for OSA must be performed during an overnight polysomnogram. Cephalometric radiographs, initial dental radiographs such as a panoramic or full mouth survey, and diagnostic plaster models are important records of a dentist's initial assessment.
- The choice of appliance is chosen, considering the pros and cons of tongue retainers versus mandibular repositioners as well as whether a boil-and-bite or custom device is needed.
- After fabrication, the dentist should fit the appliance and make any necessary adjustments to ensure patient comfort. The patient must then be shown how to use the device and given training.
- The attending dentist should schedule recall appointments every six months for the first two years if the OA has been proven to be successful and the patient is at ease. The condition of the occlusion should be assessed at every appointment. The dentist should also keep an eye on TMJ and dental status, fit, comfort, and subjective efficacy.
- To monitor OA wear, efficacy, and possible adverse effects, regular follow-up appointments must be planned at least once a year. OAs are known to last for anywhere from two to three years.

Compliance And Adverse Effects

Depending on the type of appliance, the severity of the disease, and even patient management, the compliance and side effects of OA treatment may vary. Except in one study, when a compliance monitor showed that the OA was worn for a mean of 6.8 hours each night, compliance is frequently assessed subjectively (32). Studies have found compliance rates to range greatly, from as low as 4% to as high as 82% following a year of therapy (33,34). 75% of the patients in a study with 630 patients reported compliance after 12 months of treatment, according to Marklund and colleagues(35). According to research, compliance rates ranged from 48% to 90% after 2 to 5 years of follow-up (36-38). According to reports, adverse effects and insufficient snoring reduction are the two biggest causes for stopping treatment (39). The majority of OA-related side effects are typically minor and temporary, and they most frequently include dry mouth, excessive salivation, mouth or tooth soreness, muscular tenderness, and jaw stiffness. TMJ issues that are severe and persistent are uncommon.

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In assessing OA side effects over a period of more than 5 years, long-term side effects have more recently been discussed. Almeida and colleagues(40,41) demonstrated that OAs used for a mean time of 7.3 years have a substantial impact on occlusal and dental structures using a titratable appliance (Klearway). Most of the changes in craniofacial features were related to significant tooth movements.

2. Surgical modalities for obstructive sleep apnea:

Nasal obstruction: Nasal obstruction has been linked to snoring and OSA. Increased resistance causes turbulent flow in the nasal cavity, prompts oral breathing, and encourages pharyngeal airway oscillation, all of which can contribute to snoring. Hypoxia and sleep apnea are two additional effects of nasal blockage, together with increased negative pressure and functional restriction of the pharyngeal airway (6). The nasal polyps, deviated nasal septum, and hypertrophic inferior turbinates are the most often observed and pertinent anatomic anomalies. Surgery is used to surgically remove nasal polyps, along with cauterization if necessary. This procedure aims to reduce snoring and OSA characteristics by enhancing turbulent airflow via the nasal passages and promoting more laminar airflow. An obstruction caused by hypertrophic turbinates can significantly reduce airflow through one or both nares. When one nostril is blocked from breathing, the airflow on the opposite side becomes turbulent and may result in loud snoring. The patient becomes obligated to mouth breathe if both nares are blocked, which may further change the functional dynamics of the upper airway and put the patient at risk for obstruction at other levels. Turbinectomy and radiofrequency ablation of the nose may be used as treatment options at this stage (42,43).

Oropharyngeal Obstruction

Uvulopalatopharyngoplasty (UPPP): Patients with snoring and OSA are more likely to have obstruction at the level of the tonsilar, tonsillar pillars, and soft palate. Here, surgery aims to widen and open the oropharyngeal airway and remove tissue that is obstructive or redundant in order to lessen airflow resistance.

Laser-assisted uvulopalatoplasty (LAUP): Laser surgery has also been used to remove elevated or swollen tissues from the oropharynx and to lower the uvula's height vertically. Kamami, a French surgeon, developed the laser-assisted uvulopalatoplasty (LAUP) technique in the 1980s to remove only the uvula and the distal region of the soft palate, sparing the muscle uvulus. The uvula and a specific region of the palate were laser-vaporized over the course of several brief treatments. He hypothesised that scar contracture would cause the pharynx to enlarge and thus lessen the soft palate's redundancy(44,45).

Radiofrequency (RF) tissue ablation or volumetric tissue reduction: Ionic agitation caused by radiofrequency causes frictional heating of the tissue surrounding an electrode. When the electrode tip is inserted into soft tissues, localised ablation of the soft tissue due to heat results in volume loss and stiffness of the tissues. Until clinical success is noted with a decrease in snoring noises or in the degree of obstruction, these procedures might be performed several times

Tonsillectomy: Obstructive tonsils, while uncommon in adults, are an essential component of the structure of the upper airway and may play a significant role in obstructive sleep apnea. Numerous research back up the idea that larger tonsils significantly contribute to oropharyngeal airway blockage. Tonsillectomy may be

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effective in severe obstructive tonsillitis instances and is likely to improve upper airway blockage. Tonsillectomy may be beneficial at any age (46).

Recent Advances

Transoral robotic surgery for OSA: A minimally invasive telerobotic system enables superb 3D visualization, flawless precision, and the absence of tremor during transoral robotic surgery for OSA.

Conclusion

Sleep apnea is a prevalent disorder that has a variety of implications on everyday functioning and quality of sleep. Numerous significant adverse daily effects, including poor performance, accidents, hypertension, heart disease, stroke, and insulin resistance are associated with obstructive sleep apnea. These diseases will likely grow more common in the future due to their strong relationship to obesity and the present obesity epidemic, thus doctors must be aware for them and cautious in their assessment of sleep. To that aim, routine inquiries about sleep ought to cover topics including snoring, daytime tiredness , observed apneic episodes, nocturia, sleep length, and sleep quality. Currently, the primary goal of treating OSA patients should be to eliminate apneas by individualised therapy with the long-term objective of restoring normal quality of life and avoiding or delaying the development of comorbidities. The patient must be an integral component of lifestyle modification programs with the ultimate objective of losing weight and increasing physical activity in order to aid reach this aim, especially in overweight or obese people. There is currently insufficient high-quality evidence to support managing asymptomatic OSA to lower cardiovascular and cerebrovascular events.

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