Review Article

A Review-Obstructive Sleep Apnea

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ABSTRACT:

Obstructive sleep apnea (OSA) is characterized by recurrent episodes of partial or complete collapse of the upper airway during sleep, resulting in reduced or absent airflow lasting for at least 10 seconds and associated with either cortical arousal or a fall in blood oxygen saturation. OSA is associated with major comorbidities including daytime somnolence, impaired cognition, poor quality of life. OSA is an independent risk factor for a variety of cardiovascular diseases such as hypertension, atrial fibrillation, etc. Specialized diagnostic modalities include CBCT, CT, MRI, cephalometry. The gold standard is polysomnography. Management include behavioral modification, weight loss, medication, continuous positive airway pressure, oral appliance therapy, and surgical procedures. This article briefly describes the pathophysiology and its effects on the day to day life, various diagnostic modalities and management of OSA.

KEY WORDS: Lasers, Dentistry, laser efficiency, patient care, laser application

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INTRODUCTION:

Recurrent episodes of partial or total collapse of the upper airway during sleep, resulting in decreased (hypopnea) or absent (apnea) airflow lasting for at least 10 seconds, are the hallmark of obstructive sleep apnea (OSA). These episodes are linked to either low blood oxygen saturation or cortical arousal. About 25% of individuals in the US have OSA, which is a major cause of excessive drowsiness that lowers quality of life, impairs performance at work, and increases the risk of motor vehicle crashes [1]. Unusually loud snoring is a sign of OSA. The patient alternates between awake and sleep as a result of these episodes, resulting in a cyclical breathing pattern and fragmented sleep.

Respiratory episodes usually last 20–40 seconds and in extreme situations might occur more than 100 times per hour. Major co morbidities linked to OSA include daytime somnolence, reduced cognitive function, low quality of life, and an elevated risk of car accidents. There is sufficient data to conclude that OSA stands alone as a risk factor for a range of unfavorable cardiovascular outcomes. At least 2-4% of adults suffer with the clinical condition, which is defined as more than five abnormal breathing disruptions (hypopneas or apneas) per hour of sleep accompanied with symptoms of daytime sleepiness [2].

In fact, increases in body mass index, accumulation of adipose tissue, and neck circumference are strong indicators of the disease; additionally, the prevalence of OSA is two to three times higher in men than in women, and in older people (>65 years old) as opposed to middle-aged people (30–64 years old)[2]. Obesity is the primary epidemiologic risk factor.

Though most of India's 1.2 billion people live in rural areas, increased connectivity and technological and infrastructural advancements have forced them to embrace an urban lifestyle.

It causes the prevalence of smoking and illnesses like cardiovascular disease which are closely linked to OSA—to rise gradually.

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India has a high prevalence of daytime sleepiness and snoring, which contributes to the misdiagnosis of many OSA sufferers. One significant noncommu-nicable public health risk is undiagnosed OSA.

PATHOPHYISIOLOGY:

The upper respiratory tract in humans is a special, multifunctional system that helps with breathing, food and liquid swallowing, and speech. As a result, the airway is made up of several muscles and soft tissues but has no hard or skeletal support.

Anatomically speaking, a narrower upper airway tends to collapse more frequently than a wider one. The levator and tensor palatine muscles, which advance and elevate the soft palate, as well as the geniohyoid and stylopharyngeus muscles, which oppose medial collapse of the lateral pharyngeal walls, help to prevent posterior collapse of the tongue by contracting with each inspiration. The genioglossus muscle is the most significant upper airway dilator muscle.

Most people with OSA have a constricted upper airway, which is typically caused by fat buildup in the Patients with OSA appear to use protective reflexes, which raise upper airway dilator muscle activity to preserve airway patency, to make up for an anatomically damaged upper airway while they are awake. The relationship between pharyngeal architecture and a decreased capacity of the upper airway dilator muscles to sustain a patent airway during sleep is the pathophysiology of OSA [1,2]. Figure 1 represents Schematic presentation of the typical pathophysiological sequence that occurs in OSA.

RISK FACTORS:

The major risk factors for OSA include advanced age, male gender and obesity.

1) **Age:** It seems that the severity of sleep disorder breathing reduces with age, however the prevalence of the condition among the elderly seems to rise beyond 65 years of age. The lengthening of the soft palate, changes in the body structures surrounding the pharynx, and increased fat deposition in the parapharyngeal area are some of the mechanisms suggested to explain the increased occurrence of sleep apnea in the elderly.
2) **Gender:** The reason why men are more likely than women to have OSA is unknown. It can be related to anatomical and functional features of the upper airway and in the ventilatory response to the

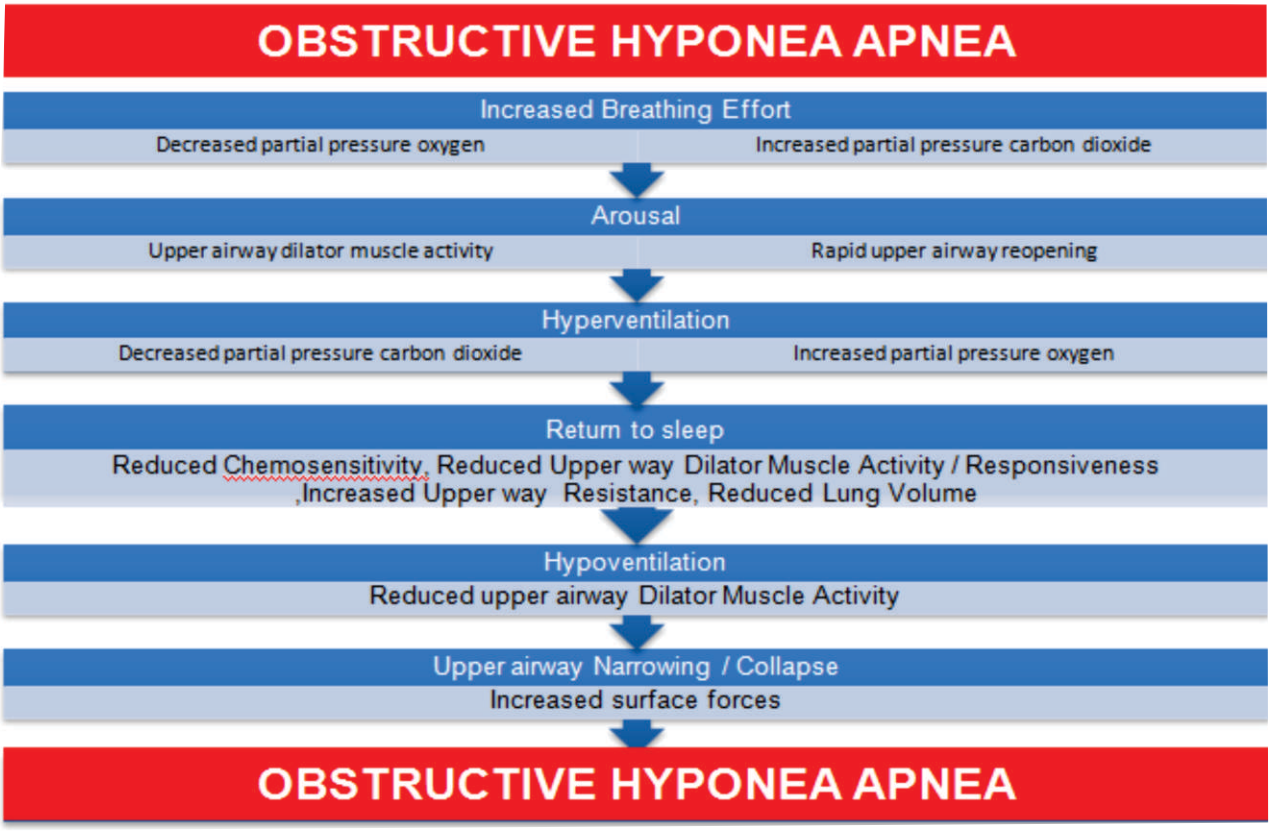

Figure 1: Schematic representation of the typical pathophysiological sequence that occurs in OSA

arousals from sleep. Compared to women, men have more fat deposited around their pharyngeal airways. There could be a hormonal component to the tendency toward irregular breathing as you sleep.

3) **Obesity:** Obesity is the primary risk factor for developing OSA. It is believed to be linked to anatomical changes that increase body and pharyngeal adiposity, which predisposes to upper airway blockage during sleep.
4) **Genetic predisposition and family history:** It is believed that genetics and familial aggregation factors contribute to the development of OSA. Given that obesity and OSA are closely related and that obesity itself tends to accumulate in families, it is plausible that the genetics of obesity plays a role in the familial aggregation of OSA.
5) **Craniofacial Anatomy:** The upper airway becomes narrower due to the enlarged tongue, soft palate, and inferiorly positioned hyoid bone, which increases the likelihood of the airway collapsing.
6) **Alcohol and tobacco use:** Research has indicated that both alcohol and tobacco use are risk factors for OSA. Snoring and sleep-disordered breathing are more common among people who smoke. Smoking causes inflammation and damage to the airways, which may alter the upper airway's anatomical and functional characteristics and raise the possibility of collapsibility when you sleep. Alcohol raises upper airway.
7) **Nasal Obstruction:** Air naturally enters our bodies through the nasal passages. Any blockage in these channels causes restricted airflow, which is most noticeable when you're sleeping and results in apneas and other OSA symptoms [3,4,].

CLINICAL PRESENTATION:

Loud and persistent snoring is one of the most prevalent night time symptoms of OSA. It is brought on by the upper airway closing considerably during sleep, which restricts airflow and causes turbulence. In the adult population as a whole, snoring is extremely prevalent; 25%–30% of women and 40%–45% of men regularly experience this problem. People who have OSA snore more frequently and intensely with time, and this is frequently related to factors like smoking, weight gain, nasal obstruction, or using muscle relaxants.

Because of the fragmentation of sleep, daytime drowsiness is one of the most prevalent nocturnal symptoms of OSA. Other well-known symptoms of daytime sleepiness include fatigue, difficulty in concentrating, memory loss, mood changes, and decreased libido. Morning headaches and awakening with a sore throat or dry tongue are possible additional symptoms [5].

ASSESSMENT:

Inquiring about snoring, breathing pauses at night, and extreme exhaustion or sleepiness during the day should be part of the systems assessment because of the high prevalence of OSA and the fact that patients frequently do not mention sleep issues to doctors [1].

CLINICAL APPROACH:

Health professionals should not disregard the risk factors of OSA and should detect and diagnose this disorder. OSA screening should be based on sleep-oriented history and physical examination in conjunction with objective tests.

The Berlin Questionnaire, designed for use in the general care context, and the STOP-Bang questionnaire, intended for preoperative screening, are two of the various questionnaires for determining OSA risk. The Epworth Sleepiness Scale is widely used in both clinical practice and research to assess sleepiness, but has low sensitivity for OSA[1].

For any patient experiencing extreme tiredness, excessive drowsiness, or unrefreshing sleep, an OSA test is advised. Patients with morning headaches, nocturnal gastric reflux, unexplained nocturia, or frequent nocturnal awakenings should be evaluated for testing, especially if they also exhibit snoring, observed nocturnal apneas, or an overweight body habit [1].

DIAGNOSTIC TOOLS:

A patient with OSA is diagnosed using a limited number of exact steps. A polysomnography test and a home sleep apnea test are two of these techniques. The most reliable and accurate diagnostic methods are sleep studies, which are the subject of both.

In a hospital, a polysomnography is a test that captures the patient's numerous body movements and functions while they are asleep. The gold standard for diagnosing OSA is this testing procedure. The apnea hypopnea index (AHI), which is derived from polysomnography, is the most significant statistic. By connecting the values of apneas and hypopneas, it illustrates the severity of OSA. It is computed as follows: total sleep time in minutes divided by the

product of all hypopneas and apneas multiplied by sixty. A score of less than five indicates negligible or no OSA. Mild results are those that are 5 or higher but less than 15. A moderate AHI is one that is more than or equal to 15 but less than 30. Lastly, severe OSA is correlated with an outcome of at least 30.

OTHER DIAGNOSTIC TOOLS:

A home apnea test is also a sleep test. Unlike the polysomnography, however, a home apnea test is taken at home and can only test for OSA[5,6,7].

Other diagnostic tools are also available for the diagnosis purpose of OSA such as electoencephalography, electrooculography, electrocardiography, electromyography, pulse oximetry, optical coherence tomography, MRI, CT scan.

DIAGNOSIS: The diagnosis is based on clinical presentation, assessment and diagnostic tools.

MANAGEMENT OF OSA:

The treatment of open-mouth breathing (OSA) involves various measures such as behavioral modification, medication, continuous positive airway pressure, weight loss, oral appliance therapy (using devices to retain tongue or advance the mandible or orthodontic appliance), and surgical procedures (tracheostomy, uvulopalatopharyngoplasty, laser-assisted uvulopalatoplasty, surgically assisted rapid maxillary expansion, maxillomandibular advancement, and stimulation of the hypoglossal nerve).

Behavioral therapies target variables that could increase the risk of OSA. It is advised that all patients with OSA abstain from alcohol and sedatives. Losing weight can improve airway patency for certain patients by reducing snoring and apneic episodes. For certain patients with sleep apnea, avoiding the supine position while they sleep may lessen the frequency of episodes.

NONSURGICAL TREATMENTS:

The most successful strategy to treat OSA is continuous positive airway pressure (CPAP), which is typically used as the initial course of treatment for patients. By functioning as a pneumatic splint to elevate and maintain constant pressure along the upper airway during inspiration and expiration, the mechanism of action enhances the quality of sleepiness [7].

ORAL APPLIANCES (OA):

Patients who do not respond to CPAP or who are not a good fit for it should use OA. Patients who prefer OA over CPAP with mild-to-moderate severity should also use OA. The design elements of commercially available OA vary from one another. Variations mostly concern how much the patient's dentition is customized. Reduction of oxygen desaturation episodes, reduction of AHI, and reduction of symptoms are the metrics used to quantify the effectiveness of OA. In general, two-thirds of patients who get OA therapy report a clinically significant improvement in their symptoms [8].

MEDICATIONS:

Even with consistent use of CPAP at effective pressures, medication can be utilized as an adjuvant to reduce residual daytime drowsiness in patients with OSA. One such drug that aids in wakefulness and alertness is modafinil (Provigil; Cephalon, Frazer, PA). Headache and jitters are the most frequent side effects of this drug [8].

MECHANISM OF ACTION OF MODAFINIL:

Modafinil is a central nervous system (CNS) stimulant that does not include amphetamines and has the ability to increase wakefulness. It is well known that modafinil has little to no in vivo affinity for the norepinephrine (NE) or serotonin (5HT) transporters and is a poor inhibitor of dopamine reuptake. On the other hand, once modafinil was administered, there were higher levels of NE and 5HT in the prefrontal cortex and hypothalamus, which could be an indirect result of increased extracellular dopamine.

Modafinil cannot be injected intravenously because it is insoluble in aqueous solutions and easily absorbed when taken orally [9].

SURGICAL INTERVENTION:

The extremely low documented propensity of modafinil to produce the euphoric effects of conventional psychostimulants (such as cocaine and amphetamine) is one of its possible advantages. Since modafinil is soluble in water and easily absorbed when taken orally, it cannot be injected intravenously Modafinil is a central nervous system (CNS) stimulant that does not include amphetamines and has the ability to increase wakefulness.

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Patients with severe OSA should use it. Maxillomandibular advancement, in which the maxilla and mandible are simultaneously advanced to widen the retropalatal and retrolingual airway, is one of the most successful surgical procedures for addressing OSA. Uvulo-palatopharyngoplasty (UPPP), which entails the removal of the tonsils, uvula, and posterior velum, is an additional option. The lingual tonsil surgery and hyoid suspension myotomy procedure are two surgical techniques that can be used to prevent the posterior collapse of the tongue musculature; in this case, the tongue size will be altered by intrinsic reduction, radiofrequency or plasma frequency ablation, or partial glossectomy[9].

CONCLUSION:

Sleep disordered breathing is an extraordinarily common condition. Pharyngeal during sleep as a consequence of abnormal structural anatomy and loss of muscle tone during sleep is the defining event. Defined obstructive sleep apnea occurs in 2% to 4% of middle aged adults. Sleep apnea is associated with a wide variety of health concerns including a lower quality of life, neurocognitive disfunction, medical/ cardiovascular morbidity especially related to cardiovascular outcomes, and increased mortality risks. A wide variety of therapies have been described to treat sleep disordered breathing. These range from modifying predisposing conditions, reducing medical risk factors (such as obesity), appliances (oral, nasal, and CPAP), and finally upper airway surgeries. Nasal CPAP is the most common medical treatment and has revolutionized treatment since its introduction by Sullivan in 1981. It is of complete importance to raise awareness of this disorder among community members and increase its recognition in health sectors to help in the early diagnosis and intervention for OSA.

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Conflicts of Interest

There are no conflicts of interest.

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