ISSN No: 2582-0559

A REVIEW ARTICLE

OBSTRUCTIVE SLEEP APNEA

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DOI: 10.37841/jidam_2022_V9_I3_04

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Received: 13.07.2022 **First Published: 07.09.2022**

Accepted: 12.08.2022 **Published: 27.09.2022**

ABSTRACT

Obstructive Sleep Apnea (OSA) is by far the common type of Sleep Disordered Breathing (SDB) that has increasingly caught the attention of dentists and Orthodontists. It has become greater public health issue than ever. It affects at least 2-4% of middle-aged population and is defined on the basis of symptoms of excessive daytime sleepiness (EDS) and objective measures of SDB. OSA is related to anatomic obstruction of upper airway, during sleep. In obese persons it occurs with comorbid conditions such as Diabetes mellitus, Arrhythmias, Heart failure, Ischemic heart diseases, Systemic and Pulmonary Hypertension and Neuro-logical complications. Untreated OSA can lead to many serious consequences. Excessive daytime sleepiness increases the risk of Motor Vehicle accidents and diminishes the quality of life. Orthodontic treatments that may reduce oral cavity volume and tongue space, are significant risk factors for OSA. Therefore, an Orthodontist should be well-aware of the symptoms of the disorder and competent enough to recognize it.

KEYWORDS: Obstructive Sleep Apnea (OSA), Sleep Sleep Disordered Breathing (SDB), excessive daytime sleepiness (EDS).

ISSN No: 2582-0559

INTRODUCTION

Sleep apnea takes its name from the Greek word *Apnoea* which means "without breathing".[1] The condition is characterized by occurrence of episodes of cessation of respiration of more than 10 seconds during sleep. Each Apnoeic episode is terminated by a few respiration accompanied by loud Snoring. Such Apnoeic episodes may occur upto 100 times per hour and lead to Hypoxemia, and eventually Pulmonary and Arterial Hypertension.[2] Sleep apnea syndrome can be of Central, Peripheral or Mixed Origin. In central sleep apnea, the respiratory muscles do not attempt to breath as a result of central nervous system disorder. Orthodontists have no role in these cases. In Peripheral/OSA, the respiratory muscles attempt to breath but are characterised by repetitive episodes of complete or partial upper airway obstruction leading to diminished or absence of airflow to the lungs. Sleep physicians may refer these cases to the orthodontist for craniofacial and upper airway evaluation with lateral cephalogram and fabrication of suitable oral appliances. Mixed sleep apnea refers to the combination of central and OSA. Oral appliance alone cannot address it effectively. OSA is related to anatomic obstruction of upper airway, during sleep. The muscles undergo relaxation and causes collapse of the soft tissues present in the back of the throat (tongue, tonsils and adenoids) which lead to upper airway blockage. Consequently, there are partial and complete pauses in breathing that lasts at least 10 seconds during sleep. The blood oxygen saturation level decreases abruptly and falls 50% or more in severe cases. The brain ultimately sends a distress signal that partially or fully wakes the person and alerts the body to breathe, causing the patient to gasp for air. This leads to snoring, multiple arousals, sleep fragmentation. As a result, affected persons have morning headaches and excessive daytime sleepiness. Patients with OSA often describes difficulty with attention, concentration and mood disturbance.[3]

PATHOPHYSIOLOGY OF OSA

Humans spend about one-third of their lives asleep, yet most individuals know little about sleep. Although its function remains to be fully elucidated, sleep is a universal need of all higher life forms including humans, absence of which has serious physiological consequences. Sleep architecture refers to the basic structural organization of normal sleep. There are two types of sleep, non-rapid eye-movement (NREM) sleep and rapid eye-movement (REM) sleep. NREM sleep is divided into stages 1, 2, 3, & 4, representing a continuum of relative depth. Each has unique characteristics including variations in brain wave patterns, eye movements, and muscle tone. Sleep cycles and stages were uncovered with the use of electroencephalographic (EEG) recordings that trace the electrical patterns of brain activity.[4]

Sleep is initiated in stage 1 NREM and progressively moves through deeper stages 2, 3, and 4 before reaching REM sleep. During these stages of NREM sleep, specific patterns of EEG activity occur. REM sleep occurs some 100 minutes later after sleep initiation. As the sleep episode progresses, REM and NREM sleep cycles alternate approximately every 90 minutes.[5] REM sleep is associated with vivid dreaming and diminished tone of the skeletal muscles of the airway and limbs. NREM sleep is associated with synchronous EEG complexes. NREM sleep constitutes about 75 to 80 percent of total time spent in sleep. The average length of the first NREM-REM sleep cycle is 70 to 100 minutes. As humans age, there are changes in sleep architecture and patterns. However, after puberty REM sleep occupies 20% to 25% of total sleep throughout life and continues alternating with NREM except in dementia. Sleep architecture can be altered by previous sleep-wake history, environmental alterations, medications, alcohol consumption, and sleep disorders.

ISSN No: 2582-0559

The sleep process normally begins with NREM sleep. In disease states such as narcolepsy sleep can begin with REM sleep. Early sleep also is associated with decreased muscle tone, which affects the limbs and support of the airway. As the process of sleep continues, there are slow asynchronous eye movements. The four stages of NREM sleep are associated with distinct brain activity and physiology.

OSA is characterized by repetitive episodes of upper airway collapse (lasting ≥10 s) during sleep. This leads to partial (hypopnea) or complete (apnea) cessation of airflow and blood gas disturbances (hypoxia and hypercapnia), despite ongoing respiratory efforts. Restoration of upper airway patency typically results in a brief awakening (arousal) from sleep. The repetitive physiological stress of OSA can lead to sleep fragmentation with consequential excessive daytime sleepiness, neurocognitive and cardiovascular sequelae. OSA is defined as more than five hypopneas or apneas per hour of sleep, combined with EDS, affects at least 4% of the adult population.[6] Obesity, male gender and increasing age are the predominant risk factors.[7] A diversity of factors is believed to contribute to OSA pathogenesis. Impaired upper airway anatomy/increased collapsibility combined with state-dependent reductions in upper airway neuromuscular function are key contributing components. However, other factors including a low arousal threshold and respiratory control instability may also contribute to inadequate upper airway muscle activation and repetitive airway collapse during sleep. The relative contribution of each of these factors probably varies considerably between individuals, implying different 'phenotypes' for OSA.[8,9]

The human upper airway is a complex structure. It is required for phonation, deglutition, and respiration. Momentary closures of the upper airway are required for swallowing and speech. Accordingly, it is intrinsically compliant. The free-floating hyoid bone anchors several pharyngeal muscles. Thus, the pharyngeal airway lacks rigid or bony support. While these features enable the upper airway to serve multiple tasks, the potential for closure and narrowing during sleep in anatomically vulnerable individuals exists. The upper airway can be differentiated into four anatomical segments: nasopharynx (between the nares and the hard palate), velopharynx (hard palate to the soft palate), oropharynx (soft palate to the epiglottis) and hypopharynx (epiglottis to the larynx). Anatomical abnormalities in one or more segments can compromise upper airway stability and contribute to narrowing/closure during sleep. Measured during wakefulness, upper airway imaging techniques including MRI, computed tomography and cephalometric measurements have been used to determine craniofacial and upper airway irregularities in OSA patients compared with healthy individuals. In general, the cross-sectional area of the various segments of the upper airway are reduced,[10] the tongue and soft tissue structures are enlarged[11], fat deposition[12] and upper airway length are increased[13] and the hyoid bone is displaced in an inferior-posterior direction[14] in OSA patients versus controls. Males also have longer pharyngeal airways than females, which may, at least in part, explain the male predisposition to OSA.[15] Together, these anatomical changes may render the upper airway more prone to collapse during sleep.

CLASSIFICATION

To grade the severity of OSA, the number of apneas plus hypopneas per hour of sleep is reported as, apnea-hypopnea index (AHI). On the basis of AHI, it is classified into, mild, moderate and severe.

An AHI of less than 5 is considered normal. An AHI of 5-15 is mild; 15-30 is moderate and more than 30 events per hour indicate severe sleep apnea.

In addition to AHI, we have to think of respiratory disturbance index (RDI) and oxygen desaturation index (ODI) together to appreciate the severity of OSA. The RDI means the average number of apnea, hypopnea,

and respiratory-effort-related arousals (RERAs) per hour of sleep. The ODI is the number of times per hour of sleep that the blood's oxygen level drop by 3% from baseline.

ORTHODONTIC EVALUATION & DIAGNOSTIC WORKFLOW FOR OSA PATIENTS

With the evolving recognition of the association between OSA and multiple comorbidities, such as systemic and pulmonary hypertension, diabetes and cardiovascular disease, the impetus for the initial screening of OSA has shifted into the primary-care physician's office.

MEDICAL APPROACH FOR DIAGNOSIS OF OSA

CHIEF COMPLAINT

The most common complaint of OSA patients is loud snoring. Listening to the patient's complaints beyond snoring may help the clinician in building up patient's trust, which is crucial in starting patient-centered care.

HISTORY TAKING

It is important to know other nocturnal symptoms like witnessed apneas and daytime symptoms like excessive daytime sleepiness, morning headache, and difficult concentration. Sleep doctors firstly focus on some important factors such as average sleep time, sleep pattern, associated insomnia, and sleep habits. Asking about smoking and alcohol consumption are of a particular importance in OSA clinic. Social history and travel history may aid in understanding patients abilities or barriers toward some treatment lines. Past medical history (like history of depression disorder, uncontrolled hypertension, diabetes mellitus, cardiac diseases, etc.) and medications history (like antidepressants, hypnotics, etc.) are also important.

QUESTIONNAIRE

Some easy-to-use questionnaires have been developed as low-cost alternatives to Polysomnography (PSG) for detecting OSA: Berlin questionnaire,[16] Epworth Sleep Scale (ESS),[17] and STOP-Bang.[18] ESS is a validated questionnaire that consists of eight items to discriminate the daytime sleepiness level of OSA patients from non-OSA patients. The STOP-Bang questionnaire includes four sleep-related questions and four additional demographic queries, for a total of eight dichotomous (yes/no) questions: snoring, tiredness, observed apnea, high blood pressure, BMI, age, neck circumference, and gender). ESS and STOP-Bang scales are easily taken by dentists.

POLYSOMNOGRAPHY

PSG is the gold standard for the diagnosis of OSA, but it is time-consuming and requires trained personnel. PSG is a non-invasive technique that involves overnight monitoring of several physiological variables including electroencephalography (EEG), eye movements (EOG), heart rhythm (ECG), and skeletal muscle activity (EMG) as well as respiratory effort, airflow, and oxygen saturation. Respiratory events can be quantified by overnight PSG study.

HOME SLEEP TEST WITH PORTABLE MONITORING DEVICE

Home Sleep Test with Portable Monitoring Device Home sleep test (HST) using a portable monitoring device can be an alternative to PSG for the diagnosis of OSA, due to the convenience, expedited diagnosis, and no need of hospitalization.

NASOPHARYNGOSCOPY

Nasopharyngeal endoscopy, nasopharyngoscopy, is the examination of the internal surfaces of the nose and throat by inserting a thin, flexible, usually fiber-optic instrument called nasopharyngoscope to detect and diagnose abnormalities in the nose and nasopharyngeal area.

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MULLER'S MANEUVER

This technique is designed to see the collapsed sites of upper airway during the inspiration with closed mouth and nose leading to the negative pressure in the chest and lungs. Introducing a flexible fiber-optic scope into the hypopharynx to obtain a view, the examiner may witness the collapse and identify weakened sections of the airway. However, the sites of obstruction with Müller's maneuver do not represent reliably the sites of obstruction during sleep.

ACOUSTIC RHINOMETRY

Acoustic rhinometry (AR) is a simple, fast, and non-invasive diagnostic tool measuring nasal cavity geometry and nasal airway change through acoustic reflection

DRUG-INDUCED SLEEP ENDOSCOPY (DISE)

Drug-induced sleep endoscopy (DISE) has been introduced as an alternative to conventional endoscopy for more accurately representing patterns of collapse during the sleeping state. DISE brings us closer to understanding the dynamic airway during sleep.

TREATMENT MODALITIES FOR OSA

LIFESTYLE MODIFICATION: WEIGHT LOSS AND SLEEP HYGIENE

Sleep hygiene recommendations include establishing a regular sleep schedule, using naps with care, not exercising physically or mentally too close to bedtime, limiting worry, limiting exposure to light in the hours before sleep, getting out of bed if sleep does not come, avoiding alcohol as well as nicotine, caffeine, and other stimulants in the hours before bedtime, and having a peaceful, comfortable, and dark-sleep environment.

POSITIONAL THERAPY

It has been long recognized that snoring patients do so most loudly in the supine position. Positional sleep apnea syndrome has been defined as an AHI during the time in supine sleep that is two or more times the AHI during sleep in the lateral position. Positional therapy uses devices like backpack, pillow, tennis balls attached to the night suit, or electrical sensors with alarm, that help patients to sleep on their side.

POSITIVE AIRWAY PRESSURE (PAP)

CPAP has been the first-line treatment for OSA due to the high efficacy in reducing sleep-disordered breathing events. In spite of the development of automatic positive airway pressure (APAP), however, lots of patients who try CPAP therapy are either completely intolerant or only partially adherent. Nowadays, the prescription of PAP to all OSA patients as a primary option is not necessary any more based on the novel concept of OSA phenotyping. If the patient has non-anatomical phenotypic causes in a progressive state of OSA, PAP would be an inevitable sole option or can be combined with other treatment modality allowing lower pressure. Otherwise, optimal treatment option needs to be considered based on the differential diagnosis of OSA phenotype.

PHARMACOLOGICAL TREATMENT

A huge number of drugs have been tested in OSA with very little success and the feasibility of an effective drug for preventing upper airway collapse is questioned. Exercise, antioxidant and anti-inflammatory drugs are currently being evaluated in comparison or in addition to CPAP.

ORAL APPLIANCES (OA)

Patients mostly prefer oral appliance (OA) to CPAP despite less reduction of AHI, due to the portability, ease of use, and better comfort. OA includes tongue-retaining device (TRD) and mandibular advancement device (MAD). Although TRD directly pulls the tongue forward during sleep to open oropharynx, it is not

ISSN No: 2582-0559

currently used because of tissue irritation, discomfort, and limited effect. MAD can be prescribed by any sleep specialist, but should be adjusted and managed by qualified dentists.

SURGICAL INTERVENTION

Tracheostomy bypasses the upper airway and is thus nearly universally successful in managing OSA. However, the significant morbidity associated with tracheostomy limits its application in the OSA population.

• Bariatric surgery is a primary surgical option in patients with morbid obesity.

• Tonsillectomy with adenoidectomy is the first-line surgical therapy for children with OSA without craniofacial anomalies.

• Nasal surgery may play a role in OSA management by improving nasal airflow. Although isolated nasal surgery is unlikely to lead to resolution of severe OSA, it may increase CPAP use and MAD adherence.

• The most common palatal surgery is Uvulopalatopharyngoplasty (UPPP), which involves removal of the tonsils, uvula, and posterior velum. Multiple variations of UPPP have been described. Due to the low success rate of 33%, UPPP is not recommended by the American Academy of Sleep Medicine (AASM) as a sole procedure for treating moderate-to-severe OSA.[19]

• Tongue base reduction surgery involves partial glossectomy or various ablative techniques to volumetrically reduce the tongue.

• Genioglossal advancement (GA) involves advancement of the genial tubercles, and may be accompanied by hyoid suspension.

• Multilevel surgery is acknowledged as an acceptable option for patients with multisite obstruction, reported surgical success rates vary widely from 22% to 78%.[20]

• Hypoglossal nerve stimulation is a relatively new addition to the array of surgical options for treatment of OSA, and is applied to the patients with poor neuromuscular responsiveness.

• Maxillomandibular advancement (MMA) is the most successful surgical intervention for OSA aside from tracheostomy. MMA has been equated to CPAP in terms of outcomes. This procedure involves advancement of both jaws and addresses airway obstruction at multiple levels; airway collapsibility decreases due to advancement of its skeletal framework.

CONCLUSION

Obstructive sleep apnea is a common disturbance with many effects on sleep and Daytime functioning. It's a medical disorder which is linked to many important adverse daytime consequences such as poor performance, accidents, hypertension, heart disease, stroke, insulin resistance and can have serious consequences if left untreated. OSA can affect adults and children at any point in the lifespan. OSA has close association with obesity and the current epidemic of obesity mean that in future, these disorders will become more prevalent, thus all orthodontists should consider incorporating OSA screening into their history-taking and clinical examination of patients. Individual orthodontists may elect to participate in the treatment and monitoring of OSA patients as appropriate and permissible under applicable laws, standards of care, and insurance coverages.

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