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OBSTRUCTIVE SLEEP APNEA: A PROSTHODONTIC PERSPECTIVE

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ABSTRACT
Obstructive sleep apnea (OSA) is repetitive cessation of breathing during sleep. It is characterized by loss of airway patency accompanied by simultaneous respiratory effort. Patients with undiagnosed sleep apnea represent a major public health problem. Prosthodontists have a unique role in recognizing the sleep disorder and co-managing the patients along with a physician or a sleep specialist. Oral appliance therapy is an important adjunct for treatment of sleep apnea patients. This review paper aims to explain the etiology, clinical features and management of Obstructive Sleep Apnea with special reference to a prosthodontic treatment approach.

KEYWORDS: obstructive sleep apnea, polysomnography, mandibular repositioning appliance, tongue retaining devices.

I. INTRODUCTION

The vast increase in sleep related research over the past two decades has led to sleep medicine being recognized as a separate specialty.[1] Dental sleep medicine is a subset of sleep medicine where the dentist plays an important role in screening for patients who present with sleep-disordered breathing (SDB) and works with a variety of specialists to provide the best treatment possible. Some research have found a link between SDB, parafunction, movement issues related to sleep, and sleep interactions.[2] A greater understanding of them can aid in the development of a comprehensive treatment strategy for each patient.

Numerous treatment options for obstructive sleep apnea (OSA) presently exist. These treatments range from non-invasive behavioural modifications to nightly use of positive airway pressure (PAP) devices to surgical treatments that alter airway anatomy. Selection of patients for these treatments is complex given the range of variability in patient anatomy and physiology, patient preference, disease severity, and controversy with regard to the effectiveness of available treatments. The threshold for treatment is also in flux with some arguing that symptoms of sleepiness are more important than measured disease severity in most patients.[1] What can result from this is a confusing array of treatment options and treatment guidelines which leave patients and physicians unsure as to what decision is most appropriate for the individual. While this article is dedicated to application of treatment guidelines, a more detailed description of the individual components of the evaluation and treatment of OSA patients is discussed in chapters fully dedicated to them. This includes making the diagnosis of OSA, physical examination, delivering positive airway pressure, oral appliances and individual surgical treatments.

II. DEFINITIONS OF DISEASES

Sleep-disordered breathing can be viewed as a continuum from normal non-obstructed breathing at one end to a patient with sleep apnea and Pickwickian syndrome at the other.

III. ETIOLOGY AND PATHOGENESIS OF OSA

The main feature of OSA is the collapse of the tongue backwards on the pharynx during sleep. Due to this, breathing gets restricted leading to a decreased O2 and increased CO2 levels in the blood which alerts the receptors in the carotid sinus, thereby causing the patient to wake up in order to breathe normally. Alcohol is a common co-factor because of its depressant influence on respiratory muscles.[4] Due to a structurally compromised airway, these patients are predisposed to malocclusion.

In a few patients, abnormal pharynx, adeno-tonsillar hypertrophy, retrognathia and macroglossia could be the reason for the structural compromise. This structural defect is a subtle reduction in airway size that can be identified in most patients as pharyngeal crowding which can be confirmed with imaging techniques. Obesity often leads to reduced size of upper airway by increased fat deposition or a compressed pharynx due to superficial fat in the tongue.[4]

For instance, a normal patient who gains a significant amount of weight may begin to snore, develop arousals during the night and begin to move into a disease state. With additional weight gain, this patient may develop increased airway resistance or sleep apnea. A patient who consumes alcohol on a given night can also move on the scale in a similar fashion; as muscle tone decreases, obstruction increases.

IV. EVALUATION AND DIAGNOSIS

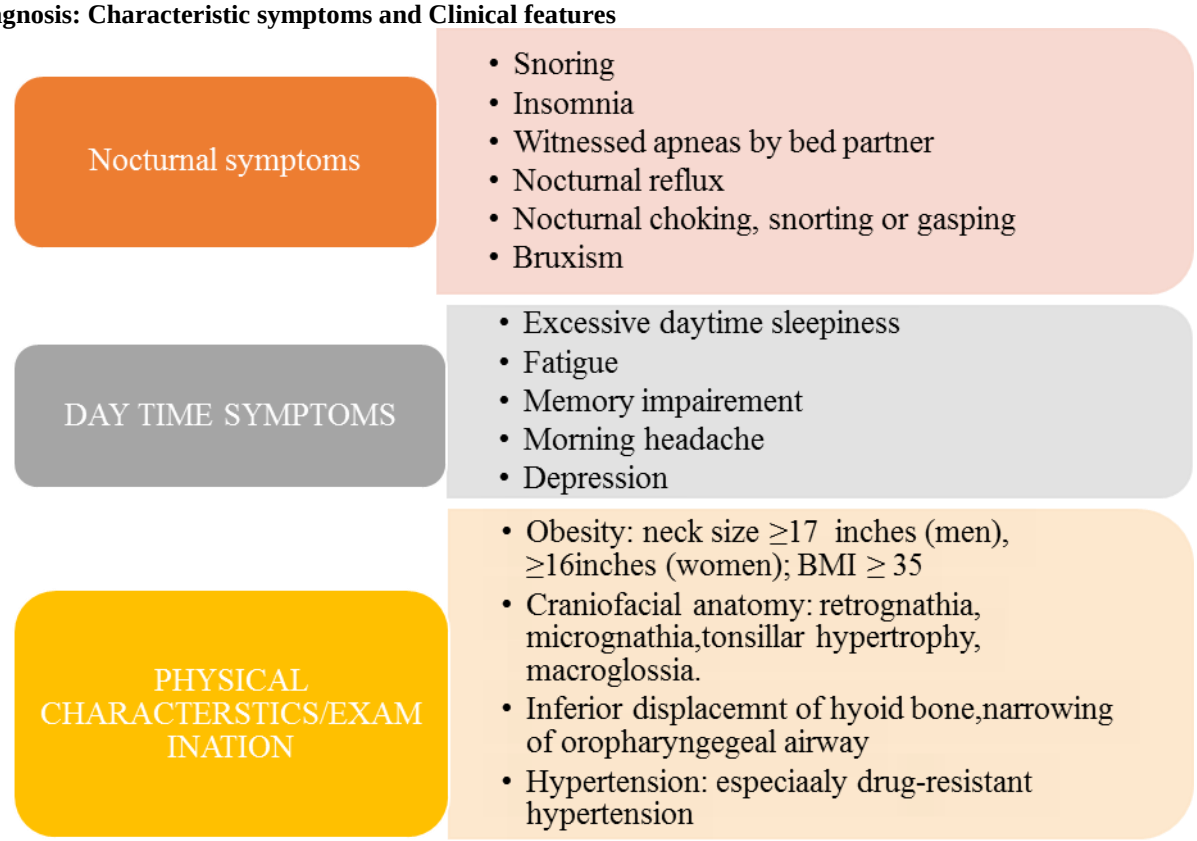
To diagnose OSA, a comprehensive sleep history, presence of specific clinical features and an objective demonstration of SDB is required.[5] High risk patients for OSA can be identified based on[6]

- Obesity (body mass index [BMI] >35)
- Type 2 diabetes
- Treatment refractory hypertension
- Pulmonary hypertension
- Nocturnal dysrhythmias
- Congestive heart failure
- Stroke
- High-risk driving populations

Systemic health evaluation can include questionnaires which are to be filled by patients at their regular appointments. The most widely used questionnaires are the Berlin Questionnaire[7] and the Epworth Sleepiness Score. The STOP-Bang questionnaire is shown to have a greater clinical utility for identifying the pretest probability of apnea.[8] If three or more items are positive, the patient is considered to be at high risk for OSA and further questions can be asked.

STOP-Bang questionnaire items
1. Snoring
2. Tiredness
3. Observed apneas
4. Hypertension
5. BMI >35
6. Age >50 years old
7. Neck >40 cm
8. Male gender

Diagnosis: Characteristic symptoms and Clinical features



Although there are numerous metrics to measure the severity of obstructive sleep apnea, disease severity is generally described in terms of the number of apneas plus hypopneas per hour of sleep, or via the Apnea Hypopnea Index (AHI) and O_2 saturation as measured on an overnight sleep study, or polysomnogram. The

polysomnogram represents a snapshot in time which may not reflect a patient's level of disease over a long period of time, but it is still the best measure of disease severity available at present.

Using polysomnography as our standard of determining disease severity, obstructive sleep apnea is represented by an AHI of greater than five events per hour and obstructive sleep apnea syndrome is defi ned as an AHI of greater than five events per hour with daytime and/or night time symptoms such as choking, gasping, snoring and daytime sleepiness.

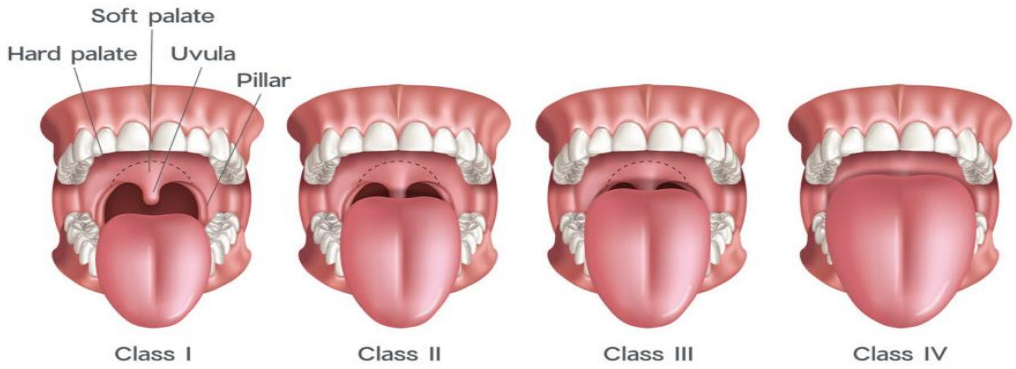
Patients can be further divided into a group based on severity.

Grading of OSA	
Severity	Grading
Mild OSA	AHI ≥5 and ≤15 per hour of sleep + symptoms or comorbidity factors
Moderate OSA	AHI ≥15 and ≤30 events per hour of sleep
Severe OSA	AHI ≥30 events per hour of sleep

Many use O_2 desaturation to modify the description of severity in sleep disordered breathing. For instance, a patient with an AHI of 12 and O_2 desaturation to 70% may be referred to as a patient with moderate OSA. Similarly, a patient with an AHI of 25 and significant desaturation may be called a patient with severe apnea. Other factors on the sleep test such as the presence or absence of REM sleep, the presence or absence of delta sleep (Stage III and IV sleep), and the percentage of time spent with oxygen saturation below 90% may also be important as well. They may help explain the patient's symptoms and may serve as a guideline as to the success of a procedure if these parameters are improved post treatment. Lastly, a respiratory effort-related arousal (RERA) is an event that occurs when a patient develops an arousal on electroencephalogram in conjunction with increasing inspiratory effort during sleep. RERAs can be added to apneas and hypopneas to create the Respiratory Disturbance Index (RDI) which many use to guide treatment.

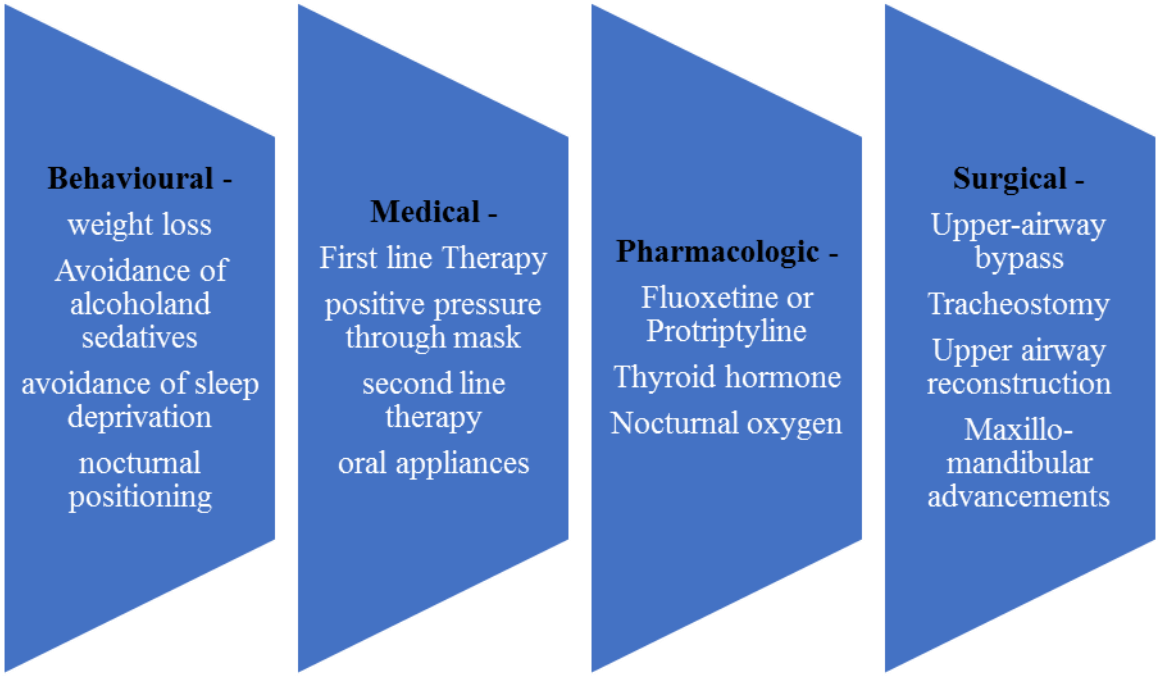
Mallampati Classification

Screening for sleep apnea should include the modified Mallampati score according to the American Academy of Sleep Medicine (AASM).6 With the patient in the upright position, the base of the uvula and the appearance of the soft palates are observed and scored accordingly. A high score (class 3 or 4) is indicative of sleep apnea.[13,14]



V. TREATMENT OPTIONS

There are four categories of treatment options available to treat patients with OSA. Every patient should be informed of the options available in each category and the treating physician and patient should review the appropriateness of treatments in each category based on individual considerations. Various brochures and informational material can be reviewed with the patient to insure that all treatment options are presented to the patient.



Initially reported in 1981, PAP provides pneumatic splinting of the upper airway and remains the standard treatment for OSA.[15,16] Various forms of PAP include Continuous PAP (CPAP), bilevel PAP, or auto titrating PAP modes. Nasal masks are often used but the effectiveness can be hampered by mouth leaks.[17] Full face masks (oronasal masks) facilitate nasal and oral breathing but if the mandible gets displaced when the interface is tightened, the tongue can be pushed posteriorly thereby worsening the apnea. Effects of PAP include alleviation of symptoms, reduced risk of traffic and workplace accidents, decreased cardiovascular comorbidities. CPAP is an effective modality for reduction of AHI. However, it has a few drawbacks such as expense, side effects with regard to the nose or face, or mask discomfort.[18]

Adjunctive treatment modalities include lifestyle modification, prevention of supine sleep position, exhalation pressure valves applied nasally, oral appliances, and upper airway surgery.[19] For patients with mild to moderate OSA who are not comfortable with a CPAP device, or those with a snoring problem oral appliance therapy is a good alternative. According to studies[20-24], even though CPAP is more effective than mandibular repositioning appliances (MRAs), patients usually prefer oral appliances over CPAP in cases where both are effective.

AASM Practice Parameters[25]

Mechanism of Action of Oral Appliances In oral appliance therapy, the tongue and the mandible are repositioned anteriorly and inferiorly which prevents the airway from collapsing. This reduces snoring and OSA by enhancing the patency of the airway by widening the lateral aspects of the upper airway.[26] The upper airway has three parts: the velo-pharynx (hard palate to tip of uvula), oro-pharynx (tip of uvula to tip of epiglottis), and hypo-pharynx (tip of epiglottis to vocal cords). The most common site of primary pharyngeal collapse in OSA is the velo-pharynx.[27,28]

Tongue retaining devices (TRD), MRAs and soft palate lifters are the three main categories of oral appliances used. Out of these, MRA is the most commonly used. The MRA covers the upper and lower teeth and positions the mandible anterior to the mandibular resting position. TRD incorporates an anterior bulb that creates a suction effect on the tongue which advances the tongue increasing the upper airway space. TRDs are preferred in cases of hypodontia, edentulism, and significant periodontal disease since these devices do not require support from teeth. MRAs are classifies as into titratable (2- piece appliance) and non-titratable (1-piece appliance) or custom-made appliances and prefabricated appliances. Studies evaluating the efficiency of various types of MRAs reveal that all custom-made devices have a comparable outcome. However, the treatment response with custom-made designs are significantly better when compared with prefabricated designs. MRAs are usually designed like a conventional orthodontic appliance, either a 1- or 2-piece type that is retained by one or both dental arches.[23]

Advantages of oral appliances

1. Nonintrusive
2. Simplicity
3. Lack of noise
4. Smaller and more portable than CPAP devices
5. Comfortable: fits inside the mouth
6. Potentially lower cost of treatment
7. Reversible treatment modality
8. No need for power source

Patient Selection Criteria for MRA Prior to treatment with MRA, the degree of sleep-related respiratory problems should be confirmed with a PSG and the dentist should be referred. Incorrect diagnosis and subsequent treatment can make the patients' condition worse. Effectiveness of MRA in children is yet to be studied conclusively, thereby limiting use of MRAs to adults only. It is not possible to tell if a patient will respond positively to oral appliance therapy.[29] Success in MRAs is associated with variables such as mild OSA, supine dependent OSA, females, and non-obesity patients.[30] Selecting ideal patients for oral appliance therapy will require further research.

Side effects can be grouped into 2 broad categories[31-35]

1. Minor in severity and temporary: they are either tolerable or tend to resolve with a short adaptive period of 6 to 8 weeks.

• TMJ pain
• Gingival irritation
• Morning after occlusal changes
• Salivation
• Tooth pain
• Loss of crown or restorations
• Myofascial pain
• TMJ sounds
• Dry Mouth
• Bad taste or odour

2. Moderate to severe and continuous: These occur at any stage during treatment and may cause intolerance and discontinuation of the appliance.

• TMJ pain
• Dry mouth
• Tooth movement: decrease in overbite and overjet, mobility of teeth, intrusion, retrusion effects
• Gingival pain
• Myofascial pain
• Tongue pain (with TRDs)
• Tooth pain
• Gagging (mostly with soft palate lifters)
• Salivation
• Skeletal changes: change in vertical condylar position, change in arch width

VI. CONCLUSION

Dentistry has a vital role to play in the identification and management of obstructive sleep apnea. It is important that when indicated, the patients be evaluated for snoring, daytime sleepiness and other signs and symptoms of OSA. American Sleep Disorders Association recommends oral appliance therapy as a treatment option for selected patients. Together with a physician and a sleep specialist, the dentist forms part of the treatment team which can effectively manage a patient with OSA.

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