Case Report

Obstructive Sleep Apnea And It's Management: A Prosthodontic Perspective

Ann Sales[*], Akshar Bajaj[*], Kushan Kishore Dhawan[*], Umesh Pai[+], Shobha Rodrigues[++]

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**, **soft palate lifters**.

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 studies have found a correlation between SDB, parafunction, movement disorders related to sleep orofacial pain sleep interactions.[2] A better understanding of these can help the formulation of a holistic treatment plan for each patient.

Sleep Disordered Breathing

Sleep disordered breathing (SDB) is a common medical disorder associated with important morbidities.

It can be divided into obstructive and non-obstructive breathing disorders. (Fig 1)[3]

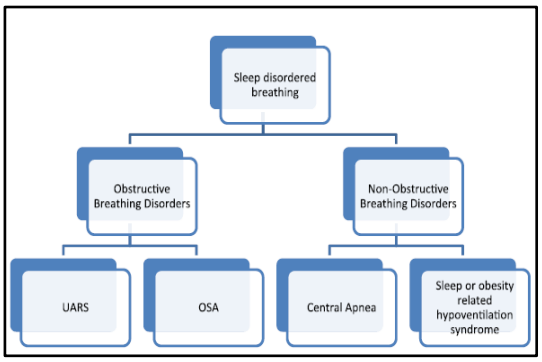

Fig. 1 Classification of SDB. UARS, upper airway resistance syndrome

Obstructive Sleep Apnea

Obstructive sleep apnea (OSA) is a sleep disordered breathing disease involving repeated obstruction of the upper airway during sleep.

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 O_2 and increased CO_2 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]

Role of edentulism in pathogenesis of OSA

Edentulism has a significant effect on the prosthodontic implications of sleep medicine and is associated with the treatment outcome of OSA. Loss of teeth can lead to the following anatomical changes:

- Decrease in vertical dimension of occlusion
- Change in position of mandible
- Change in position of hyoid bone
- Impaired function of oropharyngeal musculature such as loss of tone in soft palate and pharynx, macroglossia etc.

Diagnosis

For an OSA syndrome to be diagnosed, 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.

<u>STOP-Bang questionnaire items</u>

1. **S**noring
2. **T**iredness
3. **O**bserved apneas
4. Elevated Blood **P**ressure
5. **B**MI >35
6. **A**ge >50 years old
7. **N**eck >40 cm
8. Male **G**ender

<u>Characteristic symptoms and clinical features</u>

Nocturnal symptoms:

- Snoring
- Insomnia
- Witnessed apneas by bed partner
- Nocturnal reflux
- Nocturnal choking, snorting, or gasping
- Bruxism

- Other nocturnal symptoms: enuresis, nocturia, frequent arousals, diaphoresis, impotence

Daytime symptoms:

- Excessive daytime sleepiness
- Other daytime symptoms: fatigue, memory impairment, personality changes, morning nausea, morning headaches, depression
- Physical characteristics/examination: Obesity: neck size >17 inches (men), >16 inches (women); BMI >35
- Craniofacial anatomy: retrognathia, micrognathia, tonsillar hypertrophy, macroglossia, inferior displacement of the hyoid bone, narrowing of oropharyngeal airway
- Hypertension: especially drug-resistant hypertension

Along with medical history, self-questionnaires, and presence of clinical features[9], the diagnosis of OSA syndrome also requires demonstration of abnormal respiratory events with polysomnography (PSG) or home apnea testing.

Objective Testing

Before a dentist starts treatment, overnight sleep studies need to be conducted and interpreted by a qualified sleep physician. Polysomnography (PSG) is the "gold standard" for diagnosis of OSA. The following physiologic signals are monitored and recorded by a trained professional[9-11]:

- Electroencephalogram
- Electromyogram
- Nasal pressure
- Electrocardiogram
- Respiratory effort
- Electrooculogram
- Oxygen saturation
- Oral or oronasal thermistor
- Audio, video

AHI (apnea-hypopnea index) is the total number of apnea and hypopnea events divided by total sleep time in hours observed on an electroencephalogram. Respiratory disturbance index (RDI) is the sum total of apnea, hypopnea and RERA (respiratory effort related arousal) events divided by total sleep time in hours. Insomnia, hypersomnia, fatigue and other neurocognitive problems are comorbidities that can reduce the threshold for treatment .[12]

Grading of OSA based on AHI

Mild OSA - AHI 5 and <15 per hour of sleep with symptoms or comorbidity factors

Moderate OSA - AHI 15 and <30 events per hour of sleep

Severe OSA - AHI 30 events per hour of sleep

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]

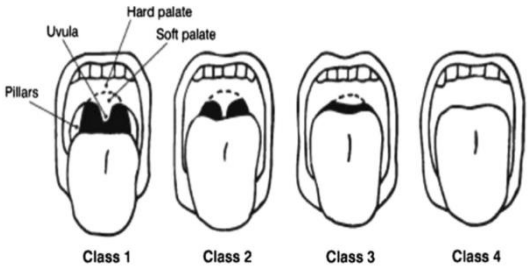

Fig. 2 Mallampati Classification

Treatment Options for OSA

Treatment indications and objectives with oral appliance therapy	
Indications	**Objectives**
Primary snorers without features of OSA	Reduce snoring to a subjectively acceptable level
Mild to moderate OSA with a preference for oral appliances, demonstrated intolerance to CPAP, poor candidates for CPAP, or failure to comply with behavioral changes	Resolution of clinical signs and symptoms of OSA Normalization of the AHI and oxyhemoglobin saturation levels
Severe OSA with initial trial of nasal CPAP Upper airway surgery may precede oral appliance therapy	Resolution of clinical signs and symptoms of OSA Normalization of the AHI and oxyhemoglobin saturation levels

A long-term, multidisciplinary management is needed for a chronic disease like OSA. Treatment includes medical, dental, behavioral and surgical options. Adjunctive treatment modalities can be used along with a primary treatment therapy to manage the condition. The patient needs to be educated regarding the severity, risk factors, modes of treatment of the disease and their role in the treatment process.[6]

Options for Treatment:

1. Positive airway pressure (PAP)
2. Upper airway surgical procedures
3. Pharmacologic treatment
4. Oral appliances
5. Behavioral modification: weight loss, alcohol avoidance, alteration of sleeping position

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]

Types of Oral Appliances

Examples of oral appliances		
Somnomed	Aveo-TSD	Adjustable Soft Palate Lifter
The silencer	Tongue-locking appliance	Silent night
Klearway	Snore guard	Snore EX
NAPA	Silent night	TPE
TAP	TheraSnore	Esmark
Herbst	Snore-no-more	HAP
SNOAR	PM positioner	Tessi
SUAD	TheraSnore	Respire

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.

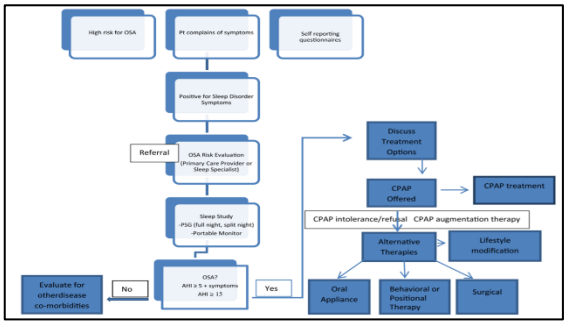

Fig. Flow chart for evaluation and treatment of patients suspected of having OSA patient

Contraindications and Side Effects of Oral Appliance Therapy

Relative dental contraindications to oral appliance therapy

Condition	Concern	Risk
Periodontal disease	Status: active or stable Concern: mobility of teeth	Reduced anchorage potential with appliance Increasing degree of mobility, and bite change with MRA Optional use of TRD
Temporomandibular dysfunction	Need to assess degree of TMD	Concern with potential aggravation of TMD and limitation of advancement potential with MRA
Number of remaining teeth	If <6–10 teeth per arch, or uneven distribution	Reduced anchorage
Protrusive capacity of the mandible	If <6 mm	Potential contraindication due to limitation of efficacy of MRA treatment
Bruxism	Patterns of wear	Early damage to appliance from overload or increase in pain with rigid appliance holding them in a fixed position
Occlusion	Number of tooth-to-tooth contacts, horizontal and vertical overjet	Reduced initial contacts will decrease patient awareness of bite change with MRA
Maximum vertical opening	If <25 mm	Inability to seat MRA
Exaggerated gag reflex	Poor adaptation potential	Inability to wear MRA

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

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.

References

1. Kryger MH, Roth T, Dement WC, editors. The principles and practice of sleep medicine. St Louis (MO): WB Saunders; 1989. p. 739.
2. Moldofsky H. Sleep and pain. Sleep Med Rev 2001;5:385–96.
3. Barewal RM, Hagen CC. Management of snoring and obstructive sleep apnea with mandibular repositioning appliances: a prosthodontic

approach. Dental Clinics. 2014 Jan 1;58(1):159-80.

4. Eliot A Phillipson. Sleep Apnea Harrison's Priciples of Internal Medicine 16th Edition, McGraw Hill Medical Publishing division ; 2005; Vol II :1573-1576.
5. Endeshaw Y.W., Katz S., Ouslander J.G., Bliwise D.L. Association of denture use with sleep-disordered breathing among older adults. J Public Health Dent. 2004;64:181–183.
6. Epstein LJ, Kristo D, Strollo PJ, et al. Clinical guideline for the evaluation, management and long-term care of obstructive sleep apnea adults. J Clin Sleep Med 2009;5(3):263–76.
7. Netzer NC, Stoohs RA, Netzer CM, et al. Using the Berlin Questionnaire to identify patients at risk for the sleep apnea syndrome. Ann Intern Med 1999;131: 485–91.
8. 9. Chung F, Subramanyam R, Liao P, et al. High STOP-Bang score indicates a high probability of obstructive sleep apnoea. Br J Anaesth 2012;108(5):768–75.
9. McNicholas WT. Diagnosis of obstructive sleep apnea in adults. Proc Am Thorac Soc 2008;5(2):154–60.
10. Practice Committee of the American Sleep Disorders Association. Practice parameters for the indications for polysomnography and related procedures. Sleep 1997;20:406–22.
11. Iber C, Ancoli-Israel S, Chesson AL, et al. The AASM manual for the scoring of sleep and associated events: rules, terminology and technical specifications. Westhester (IL): American Academy of Sleep Medicine; 2007.
12. American Academy of Sleep Medicine. International classification of sleep disorders. 2nd edition. Diagnostic and coding manual. Westchester (IL): American Academy of Sleep Medicine; 2005.
13. Nuckton TJ, Glidden DV, Browner WS, et al. Physical examination: Mallampati score as an independent predictor of obstructive sleep apnea. Sleep 2006; 29(7):903–8.
14. Friedman M, Tanyeri H, La Rosa M, et al. Clinical predictors of obstructive sleep apnea. Laryngoscope 1999;109(12):1901–7.
15. Sullivan CE, Issa FG, Berthon-Jones M, et al. Reversal of obstructive sleep apnea by continuous positive pressure applied through the nares. Lancet 1981;1: 862–5.
16. Elshaug AG, Moss JR, Southcott AM, et al. Redefining success in airway surgery for obstructive sleep apnea: a meta analysis and synthesis of the evidence. Sleep 2007;30(4):461–7.
17. Beecroft J, Zanon S, Lukic D, et al. Oral continuous positive airway pressure for sleep apnea: effectiveness, patient preference, and adherence. Chest 2003; 124:2200–8.
18. Giles TL, Lasserson TJ, Smith B, et al. Continuous positive airway pressure for obstructive sleep apnoea in adults. Cochrane Database Syst Rev 2006:CD001106.

19. Lavigne G, Cistulli P, Smith M. Sleep medicine for dentists: a practical overview. Chicago: Quintessence Publishing; 2009.
20. Ferguson KA, Ono T, Lowe AA, et al. A randomized crossover study of an oral appliance vs nasal continuous positive airway pressure in the treatment of mildmoderate obstructive sleep apnea. Chest 1996;109:1269–75.
21. Ferguson KA, Ono T, al-Majed S, et al. A short-term controlled trial of an adjustable oral appliance for the treatment of mild to moderate obstructive sleep apnoea. Thorax 1997;52:362–8.
22. Randerath WJ, Heise M, Hinz R, et al. An individually adjustable oral appliance vs. continuous positive airway pressure in mild to moderate obstructive sleep apnea syndrome. Chest 2002;122:569–75.
23. Clark GT, Blumenfeld I, Yoffe N, et al. A crossover study comparing the efficacy of CPAP with anterior mandibular positioning devices on patients with obstructive sleep apnoea. Chest 1996;109:1269–75.
24. Engleman HM, McDonald JP, Graham D, et al. Randomized crossover trial of two treatments for sleep apnea/hypopnea syndrome: continuous positive airway pressure and mandibular repositioning splint. Am J Respir Crit Care Med 2002; 166:855–9.
25. Kushida CA, Morgenthaler TI, Littner MR. Practice parameters for the treatment of snoring and obstructive sleep apnea with oral appliances: an update for 2005. Sleep 2006;29:240–3.
26. Kyung SH, Park YC, Pae EK. Obstructive sleep apnea patients with the oral appliance experience pharyngeal size and shape changes in three dimensions. Angle Orthod 2005;75:15–22.
27. Isono S, Tanaka A, Tagaito Y, et al. Pharyngeal patency in response to advancement of the mandible in obese anesthetized persons. Anesthesiology 1997;87: 1055–62.
28. Morrison DL, Launois SH, Isono S, et al. Pharyngeal narrowing and closing pressures in patients with obstructive sleep apnea. Am Rev Respir Dis 1993;148: 606–11.
29. Sutherland K, Deane S, Chan A, et al. Comparative effects of two oral appliances on upper airway structure in obstructive sleep apnea. Sleep 2011;34(4):469–77.
30. Chan AS, Sutherland K, Schwab RJ, et al. The effect of mandibular advancement on upper airway structure in obstructive sleep apnoea. Thorax 2010;65: 726–31.
31. Marklund M, Franklin KA, Persson M. Orthodontic side-effects of mandibular advancement devices during treatment of snoring and sleep apnoea. Eur J Orthod 2001;23:135–44.
32. Pantin CC, Hillman DR, Tennant M. Dental side effects of an oral device to treat snoring and obstructive sleep apnea. Sleep 1999;22:237–40.
33. Fritsch KM, Iseli A, Russi EW, et al. Side effects of mandibular advancement devices for sleep apnea treatment. Am J Respir Crit Care Med 2001;164:813–8.
34. Robertson CJ. Dental and skeletal changes associated with long-term

mandibular advancement. Sleep 2001;24:531–7.
35. Ferguson KA, Cartwright R, Rogers R, et al. Oral appliances for snoring and obstructive sleep apnea: a review. Sleep 2006;29:244–62.

*-Postgraduate student
+-Associate Professor
++-Professor and HOD

Department of Prosthodontics and Crown & Bridge
Manipal College of Dental Sciences, Mangalore
Manipal Academy of Higher Education