

Review Article

Obstructive Sleep Apnea and Its Prosthodontic Management- An Overview

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ABSTRACT

Short sleep duration and poor quality of sleep, increasingly common in our modern society, have many adverse effects on general health. Obstructive sleep apnea (OSA) is a disorder in which a person frequently stops breathing during his or her sleep. The apnea-hypopnea index (AHI) is the average number of disordered breathing events per hour. Typically, OSA syndrome is defined as an AHI of 5 or greater with associated symptoms (e.g.: excessive daytime sleepiness, fatigue, or impaired cognition) or an AHI of 15 or greater, regardless of associated symptoms.

Key Words: Obstructive Sleep Apnea, Apnea, Hypopnea, Prosthodontic management, Appliances

INTRODUCTION

Sleep is a major buffer for hormonal release, glucose regulation and cardiovascular function. [1] Sleep-disordered breathing (SDB) disrupts sleep pattern and quality. Short sleep duration and poor quality of sleep, increasingly common in our modern society, have many adverse effects on general health. Obstructive sleep apnoea (OSA) is the most common sleep disorder being diagnosed. [2]

Obstructive sleep apnea (OSA) is a disorder in which a person frequently stops breathing during his or her sleep. It results from an obstruction of the upper airway during sleep that occurs because of inadequate motor tone of the tongue and /or airway dilator muscles. [3] Obstructive sleep apnea (OSA) is a major public health problem and a social problem, affecting up to 5% of the world population and between 2% and 4% of adults in the United States. OSA has wide ranging consequences, including increased risk of motor vehicle accidents and adverse cardiovascular risk. More recently, increased risk for sudden

cardiac death during the sleeping hours as well as increased overall mortality rate among untreated individuals have been shown.

APNEA-HYPOPNEA INDEX (AHI)

An apnea is defined as cessation of airflow for 10 or more seconds. It usually indicates complete obstruction of the upper airway. A hypopnea is commonly defined as at least 30% reduction in airflow for 10 seconds [3] associated with a 4% decrease in oxygen saturation. It connotes a transient reduction in inspiratory airflow caused by increased upper airway resistance. An apnea is considered obstructive if there is continued respiratory effort despite cessation of airflow. An apnea is considered to be of central origin if there is no concurrent respiratory effort.

OSA severity

AHI < 5 Normal or primary snoring

The apnea-hypopnea index (AHI) is the average number of disordered breathing events per hour.

OSA is defined by the presence of at least 5 obstructive apneas, hypopneas, or both per hour while the patient is sleeping. OSA is commonly divided into 3 levels of severity: mild (AHI = ≥ 5 but < 15 events per hour); moderate (AHI = 15-30 events per hour); and severe (AHI = > 30 events per hour). [3] There have been reports of exacerbations of epilepsy, asthma, hypertension in patients with untreated or undiagnosed OSA. [4]

ETIOLOGY

Obesity is an important risk factor for obstructive sleep apnea (OSA). The prevalence of OSA among obese individuals is high and correlates with increasing body mass index (BMI). Among the severely obese, the prevalence of OSA ranges from 55% to 100%. In addition, obese individuals often have more severe disease as manifested by a higher apnea-hypopnea index (AHI) and lower nadir on nocturnal pulse oximetry. [5]

In addition to obesity, craniofacial anomalies like micrognathia and retrognathia, enlarged palatine tonsils, enlarged uvula, high-arched palate, nasal septal deviation, longer anterior facial height, steeper and shorter anterior cranial base, inferiorly displaced hyoid bone, disproportionately large tongue, a long soft palate, and decreased posterior airway space also predispose to obstructive sleep apnea. In addition age, ethnic background, genetic, and gender predilection, habits like consumption of alcohol, smoking, and sedatives may aggravate existing OSA. [4,6]

Risk Factors for Obstructive Sleep Apnea

Age: The increased prevalence of Sleep Disordered Breathing in the elderly appears to plateau after 65 years. Mechanisms proposed for the increased prevalence of sleep apnea in the elderly include increased deposition of fat in the parapharyngeal area, lengthening of the soft palate, and changes in body structures surrounding the pharynx. [5]

Sex: In a cross-sectional prevalence study, it shows a 4-fold higher prevalence of at least moderate OSA in postmenopausal women

as compared with premenopausal women and interestingly, in postmenopausal women taking hormonal replacement therapy, the prevalence of OSA is similar to premenopausal women. [5,6]

Influence of Tooth Loss on Obstructive Sleep Apnoea

Edentulism has been shown to produce anatomical changes in craniofacial structures and hypothesized to increase OSA.

The following anatomical changes ensue due to loss of teeth:

- 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. [7-9]

Smoking and alcohol consumption

Smoking is associated with a higher prevalence of snoring and sleep-disordered breathing (SDB). It can well be explained by the cigarette induced airway inflammation and damage which could change the structural and functional properties of the upper airway, and increasing the risk of collapsibility during sleep. Alcohol relaxes upper airway dilator muscles, increases upper airway resistance, and may induce OSA in susceptible subjects. [5]

PATHOPHYSIOLOGY OF OBSTRUCTIVE SLEEP APNEA

Pathophysiologic mechanism of snoring and OSA, although fully not understood can be explained by either the obstacle theory or the Bernoulli theory. According to the obstacle theory, an increased negative pressure during inspiration retracts the structures of the pharynx and makes them vibrate in the airflow to produce snore and possible obstruction in OSA.

The Bernoulli theory assumes that according to the principle of Bernoulli (1738), the velocity of streaming air is higher and the pressure lower at a constriction of a tube compared with the

larger part. This may cause inward suction of the pharyngeal structures in a constricted area and snores by the vibration of wall structures. [5]

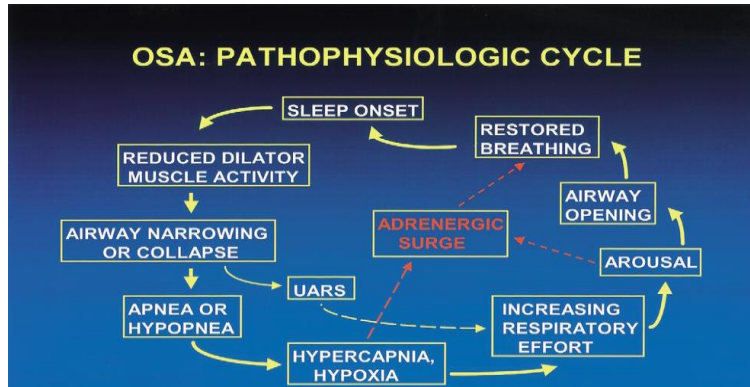


Figure 1. The recurrent pathophysiologic sequence typical of significant OSA. UARS, Upper airway resistance Syndrome.

Adopted from J Allergy Clin Immunol October 2003 [4]

SYMPTOMS OF OBSTRUCTIVE SLEEP APNEA

Patients with OSAS apart from gasping for breath at night, may have memory problems, excessive day time sleepiness, poor concentration, night drooling of saliva, depression, irritability, xerostomia, poor work performance, occupational accidents and a reduction in social interactions and other aspects of quality of life appear to be associated with untreated OSA. There have been reports of exacerbations of epilepsy, asthma, hypertension in patients with untreated or undiagnosed OSA. [4]

In particular, recent evidence indicates that OSA is associated with hypertension, ischemic heart disease, heart failure, cerebral ischemia, and cardiac arrhythmias. Consequently, in view of its high prevalence and its emerging association with cardiovascular morbidity, OSA is considered to be a major public health problem needing intervention. [3]

DIAGNOSIS

History

- Frequent awakenings
- Difficulty falling asleep
- Unrefreshing sleep
- Daytime sleepiness
- Attention, concentration, memory impairment

- Mood disturbances
- Reduced motivation, energy
- Morning headaches
- Excessive nocturia

Physical examination

- Obese
- Large neck (neck circumference >42cm in men, >37cm in women)
- Retrognathia, micrognathia
- Crowded airway
- Enlarged tonsil
- High arched palate
- Nasal deformities

Comorbid conditions

- Resistant hypertension
- Recurrent atrial fibrillation
- Stroke
- Myocardial infarction
- Pulmonary hypertension
- Chronic heart failure

INVESTIGATIONS

At present, the "gold standard" for a definitive diagnosis of OSAHS is laboratory polysomnography. Pulse oximetry has been one of the most popular monitoring techniques used in attempts at screening for sleep apnea in the home. Oxyhemoglobin indices from pulse oximetry have been used

to screen and predict sleep apnea hypopnea severity ^[10]

1. Polysomnograms

Polysomnogram (PSG) is considered the gold standard test for diagnosis of OSA. The test involves overnight recording of sleep, breathing pattern, and oxygenation. The study records analysis of apnoea, oxygen saturation, body position, change heart rate, snoring, desaturation relations, and sleep staging. The recordings include electroencephalography, electro-oculography, electromyography, and electrocardiography.

2. Lateral cephalograms

Lateral cephalograms are used to analyze skeletal and soft tissue characteristics of patients with OSA.

3. Computed tomography scanning

Compared to lateral X-ray cephalometry, CT scanning and MRI significantly improves soft tissue contrast and allows precise measurements of cross-sectional areas at different levels, as well as three-dimensional reconstruction and volumetric assessment. CT scanning has provided valuable insights into the pathophysiology of Sleep Disordered Breathing and plays a major role in its management.

4. Magnetic resonance imaging

Compared to lateral X-ray cephalometry or CT scanning MRI offers various advantages, such as excellent soft tissue contrast, three dimensional assessments of tissue structures, and lack of ionizing radiation.

5. Acoustic reflection test

Acoustic reflection test can be used to determine the airway obstruction and also the corresponding effect of mandibular advancement and protrusion on the upper airway. In this test, the sound wave is projected into the airway and is reflected back through the tube to a computer which creates graph that determines the location of the obstruction.

6. Spirometry

Spirometry is a pulmonary function test. It is a simple method of studying pulmonary ventilation by recording movements of air

into and out of lungs. The test determines the inspiratory flow rate, expiratory flow rate, forced vital capacity (FVC), ratio of forced expiratory volume in 1 s to FVC, and other ventilation rates.

TREATMENT

Treatment of sleep-disordered breathing (i.e. snoring, upper airway resistance syndrome, sleep Apnoea syndrome) can be divided into four general categories. These include: (1) Lifestyle modification i.e. weight loss, cessation of alcohol consumption, sleep position training (2) Continuous positive airway pressure (CPAP). (3) Oral appliances (4) Upper airway surgery. ^[4]

Most patients with sleep apnea are being offered nasal continuous positive airway pressure (CPAP) as the treatment of choice. However, compliance with nasal CPAP varies, and is particularly poor in non apneic snorers and those with mild sleep apnea; this group of patients have poor acceptance of CPAP. That is why oral appliances constitute an attractive noninvasive alternative for patients with sleep apnea, provided the efficacy, compliance, long-term tolerance, and satisfaction with these appliances are established. ^[4]

Originally there are three concepts for a dental appliance to modify the airway, ^[3] which could be used alone or in combination depending on where the airway obstruction occurs:

Soft palate lifting – the prosthesis lifts and/or stabilizes the soft palate, preventing vibration during sleep. ^[3]

Tongue retention – tongue-retaining devices (TRDs) incorporate an anterior hollow bulb, which generates a negative pressure vacuum when the tongue is inserted. The tongue is held forward, away from the posterior pharyngeal wall, opening up the airway. Owing to muscle anatomy, this appliance simultaneously modifies the position of the mandible. ^[3]

Mandibular repositioning – these appliances (MRAs) hold the mandible in an antero inferior position, which, as a consequence of

muscle attachment, indirectly brings the tongue forward, opening up the posterior airway. The repositioning may also stretch and reduce the collapsibility of the soft palate via its connection to the base of the tongue and increase the superior airway space. [3]

The appliances can be broadly classified into:

- Tongue repositioning devices, such as the tongue retaining device
- Mandibular advancement devices (MAD) which work by holding the lower jaw and the tongue forward during sleep
- Devices designed to lift the soft palate
- Uvula lifters, which are not in use now due to discomfort. [7,11,12]

The tongue retaining device was first developed by a physician in 1979. It is a bubble shaped device made of soft polyvinyl material. The patient's teeth rest in custom fitted grooves which are extended to form a 'bubble' that sticks out from between the lips. The patient positions his teeth in the grooves, sticks his tongue forward into the bubble until suction grabs and holds the tongue in place. Positioning the tongue forward may eliminate any obstruction caused by the base of the tongue. [13]

Mandibular Advancement Device essentially consists of a plastic mould of the teeth. Advancement of the lower teeth moves the mandible forward and opens the airway, preventing its collapse during sleep. It is relatively simple reversible and cost effective. [14]

The silencer system incorporates titanium precision attachments at the incisor level, allowing sequential 2 mm advancement of up to 8 mm, lateral movement of 6 mm, (3 mm bilaterally) and vertical pin height replacements. It is the only appliance that allows adjustment in not only an antero-posterior, but also in an 'open and closed' position as it includes a very expensive titanium metal hinge device.

The Klearway oral appliance utilizes a maxillary orthodontic expander to

sequentially move the mandible forward. Klearway is a fully adjustable oral appliance used for the treatment of snoring and mild to moderate OSA. Small increments of mandibular advancement are initiated by the patient and this prevents rapid jaw movements that cause significant patient discomfort.

The PM positioner links the upper and lower splints with bilateral orthodontic expanders. This appliance is made of a thermoplastic material which must be heated in hot tap water every night before it is placed in the mouth.

The Thornton adjustable positioner (TAP) allows for progressive 0 mm advancements of the jaw via an anterior screw mechanism at the labial aspect of the upper splint. This appliance has a separate section for the mandible and maxilla.

Modified Herbst Appliance design links upper and lower splints with a piston-post and sleeve adjustable telescopic mechanism on each side. It prevents side-to-side motion, but since the mandible is held close with small orthodontic rubber bands, opening the jaws is fairly easy.

The elastic mandibular advancement (EMA) is the thinnest and least bulky of all the appliances. It is similar to clear acrylic orthodontic retainers, and moves the jaw forward in fairly significant steps, and can be difficult to tolerate.

DUOBLOC is a Custom-made adjustable mandibular advancement device (MAD) for the treatment of obstructive sleep apnea (OSA). This MAD has attachments in the frontal teeth area that allow for progressive advancement of the mandible.

Palatal lift prosthesis significantly improved the upper airway passage dimension and thus eliminated snoring and airway obstruction and improved the overall quality of life of patient. [15]

Surgical options are also available, including removal of tissue from the posterior pharyngeal region (e.g., uvulopalatopharyngoplasty) and maxillary-mandibular advancement, in which both the

maxilla and the mandible are surgically advanced, thereby permanently enlarging the posterior pharyngeal region. Success rates of upper airway surgery vary from 24% to 86%, depending on severity, patient selection, definition of success, and type of surgery performed. Typically, a higher success rate is achieved if multilevel surgery is performed (e.g., uvulopalatopharyngoplasty followed by maxillary-mandibular advancement). The last surgical option is a tracheostomy. The AASM has published practice parameters for additional medical therapies. Neither medication nor oxygen therapy is recommended for primary treatment of OSA. Exceptions are adjunctive uses of a stimulant therapy with modafinil in those who remain adherent to OSA treatment but have residual sleepiness without any other identifiable cause and optical nasal corticosteroids in those with concurrent rhinitis. Positional therapy (in which some barrier is used to minimize supine sleep) may be acceptable as an adjunctive or secondary therapy option in those who have respiratory events predominantly in the supine position only. [7,12,16]

NEW DEVELOPMENTS

Some patients who appear to have OSA during the diagnostic test develop central sleep apnea on CPAP initiation. The incidence of this form of atypical apnea, known as *Complex Sleep Apnea Syndrome* (CompSAS), is 10% to 20%. Patients with CompSAS tolerate CPAP very poorly because of increased sleep disruptions resulting from central sleep apnea events. Although some of those with CompSAS can eventually be treated with CPAP, up to 50% will require the use of a new PAP device known as the *adaptive servo-ventilator*. Because CompSAS is difficult to diagnose and treat, patients with suspected CompSAS should be referred to a sleep center for further evaluation and treatment. Recently, a new treatment device has undergone a multicenter trial to assess efficacy. Rather than using a machine to generate PAP,

Provent uses a 1-way valve to maintain a constant pressure in the posterior pharyngeal region. The attractive feature of this device is its simplicity: it is a tape-like device worn over the nostrils at night and does not require any tubing or electricity. This product has been in the European market and has received approval by the US Food and Drug Administration; however, the results of a recently completed double-blind, prospective, multicenter trial have not yet been published. [17]

CONCLUSION

Sleep and dreams are taken for granted by those not affected by obstructive sleep apnea. Unfortunately in around 10 million population around the world, sleep is a nightly battle which leaves its victims and their bed partners fatigued, stressed and much less healthy. Untreated sleep apnea is one of the major public health issues we face in common. The emergence of dental sleep medicine as a safe and effective treatment brings hope for the millions of patients looking for alternatives to CPAP treatment.

Oral appliances used to date constitute a relatively heterogeneous group of devices for the treatment of sleep apnea and non-apneic snoring. The evidence available at present indicates that oral appliances successfully “cure” mild-to-moderate sleep apnea in 40–50% of Patients, and significantly improve it in additional 10–20%. They reduce, but do not eliminate snoring. Side effects are common, but are relatively minor. Provided that the appliances are constructed by qualified dentists, 50-70% of patients continue to use them for several years. Their effectiveness is inferior to CPAP. [7] As dental professionals, we have a significant role to play in the early diagnosis, management and care of patients suffering from sleep apnea. Oral appliances play a major role in the non surgical management of OSA and have become the first line of treatment in almost all patients suffering from OSA.

The interplay between anatomic, functional, and neural factors that influence the upper airway patency during wakefulness and sleep is still unclear. Although the role played by the prosthodontists is still in its infancy, there is much to learn and understand in the rapidly evolving field of sleep medicine. The growing interest of prosthodontists in sleep medicine has contributed immensely toward effective prevention and treatment of OSA and sleep bruxism for each patient based on his/her individual requirement. ^[18]

REFERENCES

1. Glossary of Prosthodontic Terminologies 9. J Prosthet Dent. 2017; 117(5S):e73
2. Jamie C.M. Lam & Mary S.M. Ip. Sleep & the metabolic syndrome. Indian J Med Res. 2010; 131(2): 206-16.
3. Sandeep C, Gopinadh A, Babu MS, Ravuri K. Revival of the Eclipsed: The 5th Dimension of a Prosthodontist. Int J Dent Clin. 2011;3(1):71-4.
4. Qureshi A, Ballard RD. Obstructive sleep apnea: J Allergy Clin Immunol. 2003; 112(4):643- 50.
5. Meyer J B, Knudson C R, Hall W: The sleep apnea syndrome. Part I: Diagnosis. J Prosthet Dent 1989 ;2:675-9.
6. Olson EJ, Moore WR, Morgenthaler TI, Gay PC, Staats BA. Obstructive Sleep Apnea-Hypopnea Syndrome. Mayo Clin Proc. 2003 ;78(12):1545-52.
7. Ferguson KA, Cartwright Rogers R, Nowara WS, Oral Appliances for Snoring and Obstructive Sleep Apnea: A Review. Sleep. 2006 ;29(2):244-62.
8. Hoffstein V. Review of oral appliances for treatment of sleep-disordered breathing. Sleep Breath. 2007;11(1): 1–22.
9. Lam B, Sam K, Mok WYW, Cheung MT, Fong DYT, Lam JCM, Lam D, Lorett YC, Yam, Mary SM. Randomised study of three non-surgical treatments in mild to moderate obstructive sleep apnoea. Thorax 2007;62:354–9.
10. Y. K. Tan, P. R. Lestrangle, Y. M. Luo, C. Smitha, H. R. Grant, A. K. Simonds, S. G. Spiro. Mandibular advancement splints and cpapin patients with obstructive sleep apnea: a randomized cross over trial. Eur. J. Orth.2002. 24:239-49.
11. Yoshida K. Prosthetic therapy for sleep apnea syndrome. J. Prost. Dent. 1994: 72(3): 296–302
12. LS Bennett, RJ Davies and JR Stradling. Oral appliances for the management of snoring and obstructive sleep apnoea; Thorax 1998 53: S58-64.
13. F. Gagnadoux, B. Fleury, B. Vielle, B. Pe'Telle, N. Meslier, X.L. N'guyen, W. Trzepizur And J.L. Racineux. Titrated Mandibular Advancement Versus Positive Airway Pressure For Sleep Apnoea. Eur Respir J. 2009;34:914–20.
14. Luyster FS, Buysse DJ, Strollo PJ. Comorbid Insomnia and Obstructive Sleep Apnea: Challenges for Clinical Practice and Research .J Clin Sleep Med 2010;6(2):196-204.
15. Kline CE, Crowley EP, Ewing GB, Burch JB, Blair SN, Durstine JL, Davis JM, Youngstedt SD. The Effect of Exercise Training on Obstructive Sleep Apnea and Sleep Quality: A Randomized Controlled Trial. SLEEP 2011;34(12):1631-40.
16. Reichmuth KJ, Austin D, Skatrud JB, Young B .Association of Sleep Apnea and Type II Diabetes - A Population-based Study. Am J Respir Crit Care Med. 2005; 172(12):1590-5.
17. Patel K, Moorthy D, Chan JA, Concannon TW, Ratichek SJ, Chung M, Ethan M. Balk .High Priority Future Research Needs for Obstructive Sleep Apnea Diagnosis and Treatment. J Clin Sleep Med 2013;9(4):395-402.
18. Obstructive Sleep Apnea: Role Of Oral Appliances. <http://orthocj.com/2013/05/obstructive-sleep-apnea-role-of-oral-app>

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