Review Article

Sleep Apnea and Dental Implications -A Review

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How to cite this article: M.Veerakumari, Anjibabu Merneedi M., R. Sudhakara Reddy,N.Rajesh, Sleep Apnea and Dental Implications -A Review 2023;11(2)

Abstract

Obstructive sleep apnea (OSA) is an increasingly common, chronic, sleep-related breathing disorder.The prevalence of obstructive sleep apnea (OSA) continues to rise due to change in life patterns.The most common presenting symptom of OSA is excessive sleepiness and is associated with a 2- to 3-fold increased risk of cardiovascular and metabolic disease. The key contributors to OSA pathogenesis include a narrow, crowded, or collapsible upper airway “anatomical compromise” and “non-anatomical” contributors such as ineffective pharyngeal dilator muscle function during sleep, a low threshold for arousal to airway narrowing during sleep, and unstable control of breathing. Effective treatments include weight loss and exercise, positive airway pressure, oral appliances that hold the jaw forward during sleep, and surgical modification of the pharyngeal soft tissues or facial skeleton to enlarge the upper airway. This review summarizes the latest knowledge ondifferent contributors to OSA with a focus on emerging clinical tools.

Key words - Obstructive sleep apnea, upper airway physiology, positive airway pressure; snoring. Oral appliances

Introduction

Sleep disordered breathing (SDB) is a term which includes simple snoring, upper airway resistance syndrome (UARS), and sleep apnea.Obstructive sleep apnea (OSA) is characterized by recurrent episodes of partial or complete collapse of the upper airway during sleep, resulting in reduced (hypopnea) or absent (apnea) airflow lasting for at least 10 seconds and associated with either cortical arousal or a fall in blood oxygen saturation. Patients present with various symptoms, although almost all complain of snoring, witnessed breathing pauses,and excessive day time sleepiness. Simple snoring is a common complaint affecting 45% of adults occasionally and 25% of adults habitually and is a sign of upper airway obstruction. (1) OSA is approximately 25% of adults in the US and is a major cause of excessive sleepiness, contributing to reduced quality of life, impaired work performance,

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and increased motor vehicle crash risk.(2)OSA is associated with an increased incidence of hypertension, type 2 diabetes mellitus, atrial fibrillation, heart failure, coronary heart disease, stroke, and death.(3)OSA can be diagnosed with either home- or laboratory-based sleep testing, and effective treatments are available. This review provides an update on the epidemiology, pathophysiology, diagnosis, and management of OSA.

Epidemiology

The prevalence of OSAS is around 4% for men and 2% for women in the age-group of 30-60 years. OSA is associated with overweight and obesity.(4) Among individuals aged 30 to 49 years with a body mass index (BMI) less than 25, the prevalence of OSA among men is 7.0% and among women is 1.4%, compared with 44.6% among men and 13.5% among women with a BMI of 30 to 39.9.7.(5,6)

Pathophysiology

OSA is characterized by repetitive partial or complete collapse of the upper airway during sleep, resulting in episodic reduction (hypopnea) or cessation (apnea) of airflow despite respiratory effort. Contraction of upper airway dilator muscles is necessary to maintain airway patency during inspiration. The most important upper airway dilator muscle is the genioglossus muscle, which contracts with each inspiration to prevent posterior collapse of the tongue, assisted by the levator and tensor palatini muscles (advancing and elevating the soft palate) and the geniohyoid and stylopharyngeus muscles (opposing medial collapse of the lateral pharyngeal walls). (7) Most people with OSA have a narrow upper airway, typically caused by fat deposition in the parapharyngeal fat pads and pharyngeal muscles,(8)or abnormalities in craniofacial structure **(Figure 1).**

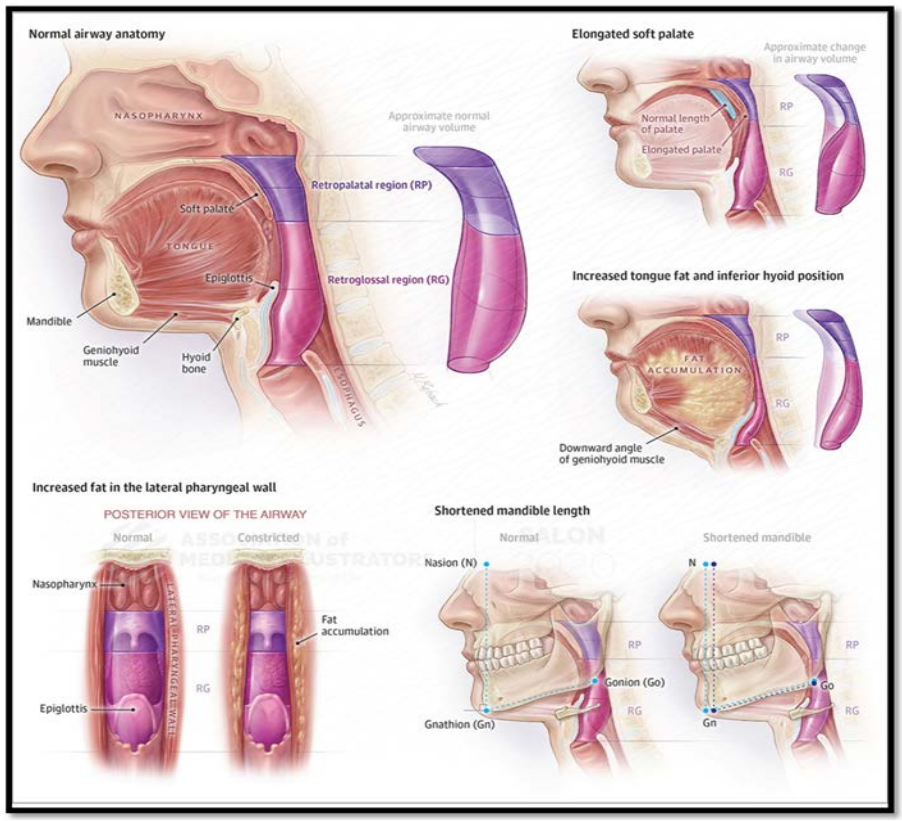


Figure -1 Anatomical features contributing to OSA

These abnormalities include both clinically evident anatomic abnormalities, such as micrognathia and retrognathia, or subtle radiographic findings, such as inferior positioning of the hyoid bone and shorter mandibular and maxillary length, which result in a small maxillomandibular volume.(9) Obstructive apneas and hypopneas result in large changes in intrathoracic pressure, intermittent hypoxemia, and arousal from sleep. Although these arousals generally do not wake the patient, this sleep fragmentation is the primary cause of excessive sleepiness in individuals with OSA. Intermittent hypoxemia, particularly with concomitant hypercapnia, activates the sympathetic nervous system

system and is the major contributor to both acute and chronic elevation of blood pressure. Repetitive episodes of hypoxemia increase reactive oxygen species, which may further contribute to vascular disease, metabolic abnormalities, and inflammation.(7)

Clinical Features

Excessive sleepiness is reported by 15% to 50% of people with OSA identified through general population screening.(10) While some patients experience awakenings accompanied by gasping or choking, awakenings without accompanying symptoms are more typical.(11) Patients with OSAS may have memory problems, excessive day time sleepiness, difficulty in concentrating, night drooling of saliva, depression, irritability, xerostomia, gasping for breath at night, and witnessed apneas. 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 andhypertensionin patients with untreated or undiagnosed OSA. Motor vehicle accidents in untreated OSAS patients is reported to be two or three times higher than in matched control drivers. (12,13)

Assessment and Diagnosis

OSA can be made on History, Examination, various modalities like Questionnaires,Imaging modalities, Polysomnography, AHI, Split-night testing, Oximetry, Home sleep apnea testing etc...intensive and inconvenient for the patient.(21)

A complete history obtained regarding snoring, day time sleepiness, association with systemic complications was obtained and evaluated. Questionnaires like Berlin Questionnaire (developed for use in the primary care setting) and the STOP-Bang questionnaire (developed for preoperative screening) helps in preoperative assessment. The Epworth Sleepiness Scale is widely used in both clinical practice and research to assess sleepiness, but has low sensitivity for OSA.(14-17)

Examination of oral and upper airway may include identification of anatomic abnormalities, such as tonsillar hypertrophy, macroglossia, or retrognathia, occlusion, periodontal status, tooth mobility, parafunctional habits, wear facets (generalized / isolated), DMFT, charting, recording of the sensitivity of teeth, tori, and the amount of overbite and overjet present. The dental, skeletal midlines, and temporomandibular joint (TMJ) status have to be recorded prior to analysis of sleep apnea. The Mallampati score **(Figure-2)**

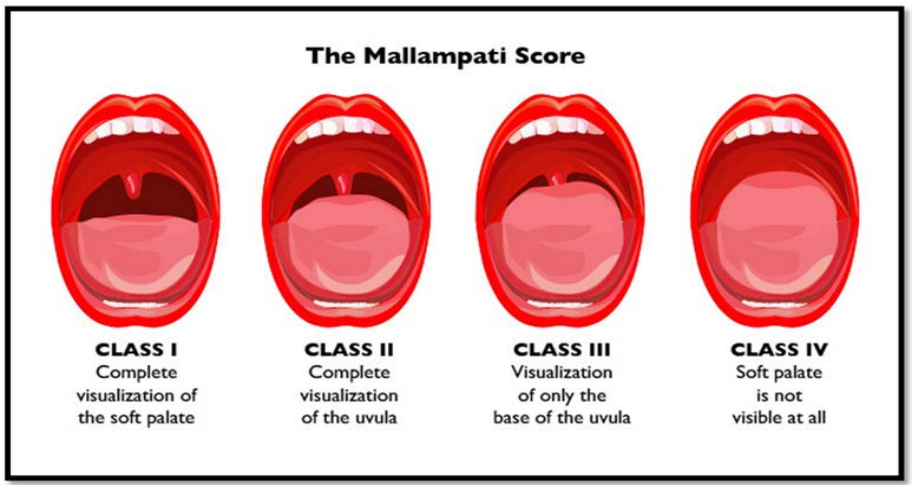


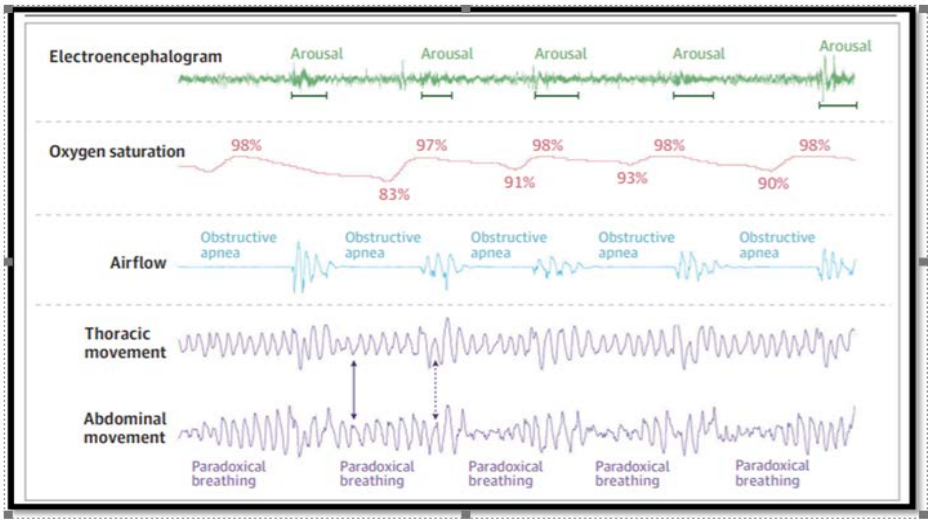
Figure-2 Mallampati Score

(Grades 1-4) can be used as a predictor for determining the severity of sleep apnea, particularly in cases where an enlarged tongue may seem to be the cause for airway obstruction.18-19

A number of Imaging modalities like acoustic reflexion, fluoroscopy, nasopharyngoscopy, cephalometry, MR imaging, and both conventional and electron-beam CT scanning have been used to assess the airway. When specific problems like TMJ dysfunction are present and an oral appliance is being planned, specific imaging of the TMJ should be done. Cephalometrics could be used if the practitioner wishes to evaluate the airway dimension, evaluate cranial or skeletal structures, or plan for orthognathic surgery; for example, SNA and SNB angles and posterior airway space are decreased and PNS-P (length of soft palate) are increased in OSAS.(20)

If the clinical and radiographic evaluation suggests OSA, diagnostic confirmation requires overnight testing.

The standard diagnostic test is laboratory-based Polysomnography, during which both sleep and respiratory parameters aremonitored **(Figure 3).**



A typical laboratory-based polysomnogram includes measures of (1) Airflow through the nose using a nasal cannula connected to a pressure transducer or through the nose and mouth using a thermal sensor; (2) Respiratory effort using thoracic and abdominal inductance bands; (3) Oxygen hemoglobin saturation by finger pulse oximetry; (4) Snoring using a microphone affixed over the trachea or by filtering out low-frequency signals from the nasal cannula-pressure transducer system; (5) Sleep stage and arousal using electroencephalogram, electrooculogram, and chin electromyogram; (6) Electrocardiogram findings; (7) Body position; and (8) Leg movement. Laboratory-based testing is labor-intensive and inconvenient for the patient.[21]

OSA severity is typically quantified using the AHI. An AHI less than 5 events per hour is considered normal, 5 to 14.9 is considered mild, 15 to 29.9 is considered moderate, and at least 30 is considered severe OSA. Differences in how hypopneas are defined affect the AHI value and a lack of consistency in event definition complicates the interpretation of sleep test results and highlights the importance of considering symptoms and comorbid illnesses when making treatment decisions. [22]

Home sleep apnea testing is increasingly used to diagnose OSA, and consists of measures of airflow, respiratory effort, and oxygen saturation, but not measures of sleep or leg movements. The sensors are self-applied by the patient at home following instruction from a technologist or via an instructional video. Home sleep apnea testing has both high sensitivity (79% [95% CI, 71% 86%]) and specificity (79% [95% CI, 63%-89%]).[23]

Treatment Options

Various treatment modalities are proposed based on the severity of the sleep disorder, preference of the patient, the patient's general health, and the preference and experience of the team members the first and simplest option is behavior modification; this would be followed by insertion of oral devices suited to the patient, especially in those with mild to moderate OSA. CPAP and surgical options are chosen for patients with moderate to severe OSA.

BEHAVIOR MODIFICATION

Behavioral measures include abstinence from alcohol, avoiding supine sleep position, regular aerobic exercise, and weight loss.Changing the sleep position from the supine position to the side position; by placing a tennis ball or by positioning a pillow such that they cannot roll on to their back (positional training). The avoidance of alcohol and sedatives for 3 h before sleep causes a depressing effect on the central nervous system. They also act as muscle relaxants, reducing airway patency.[24-26]

CONTINOUS POSITIVE AIRWAY PRESSURE (CPAP)

Positive airway pressure (PAP) is the primary therapy for individuals with symptomatic OSA of any severity. PAP devices deliver pressure to the airway through a mask worn over the nose or the nose and mouth.

This pressure acts as a splint to prevent airway collapse during inspiration. PAP normalizes AHI in more than 90% of patients while wearing the device. Benefit depends on adherence to therapy, with more hours of use per night associated with greater symptom improvement and greater blood pressure reduction.[27-28]

ORAL APPLIANCES

Oral appliances were used by Robin to treat glossoptosis in infants with micrognathia as early as 1905. There is sporadic mention of dental devices for prevention of snoring in patent records before 1980. In 1991, The American Academy of Sleep Dentistry was formed for the education and certification of dental sleep-disorders specialists. In 2000, a section on oral appliances was created in the Academy of Sleep Medicine. [29-30]

The American Academy of Sleep Medicine (AAOSM) has recommended oral appliances for use in patients with primary snoring and mild to moderate OSA. It can also be used in patients with a lesser degree of oxygen saturation, relatively less day time sleepiness, lower frequency of apnea, those who are intolerant of CPAP, or those who refuse surgery. [31]

Oral devices are basically thermoplastic materials with retainers and supports and are usuallycustommade.

A. Mandibular repositioning or advancement devices (MRD/MAD)

E.g., Herbst appliance / snoreguard / silencer. [32]

Which may betitratable They function by engaging one or both of the dental arches to modify mandibular protrusion; Fabrication of appliance requires dental impressions, a centric relation record, and protrusive record.

B. *Tongue repositioning or retaining devices (TRD),* E.g., snorex.[33]

C. *Soft-palatelifters.*

D. Tonguetrainers. [12]

E. *A combination of Oral Appliance and CPAP*

The combination of CPAP with Oral appliance helps in eliminating the disadvantages of CPAP. This combination helps in delivering the pressurized air directly into the oral cavity and eliminates the use of head gear or nasal mask and avoids the problems of air leaks and the claustrophobia. [34]

How the Oral Appliances Work

Oral appliances are worn only during sleep and work to enlarge the airway by moving the tongue (anteriorly) or the mandible to enlarge the airway. It is hypothesized that these appliances may also affect upper airway muscle tone and thus decrease their collapsibility. Movement of the tongue or **Mandible anteriorly** can increase the cross-sectional size of the airway and hence oral appliances help in increasing the airway size **(Figure-4).**

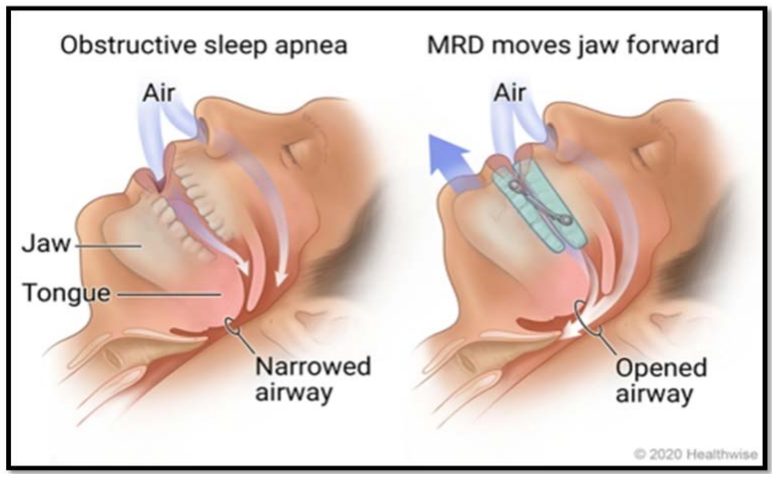


Figure-4 Oral Appliance -MAD

Activation of the upper airway dilator muscles by the appliance could cause a decrease in airway collapsibility and this may contribute to preservation of airway patency during sleep, A Tongue-retaining device **(Figure -5)**

Figure -5 Tongue Retaining Device

is a custom-made soft acrylic appliance that covers the upper and lower teeth and has an anterior plastic bulb. It uses negative suction pressure to hold the tongue in a forward position inside the bulb. By holding the tongue in a forward direction through its attachment to the genial tubercle, it stabilizes the mandible and hyoid bone, thus preventing retrolapse of the tongue. These devices, reverse pharyngeal obstruction both at the level of the oropharynx and the hypopharynx, thereby enlarging the airway and reducing snoring and the related apnea. [35] Soft palate trainers and tongue posture trainers are rarely used. [12]

Goals of Oral Appliance

The treatment goal should be a decrease of about 50% of the initial AHI or to less than 10 events/ hr.Oral appliances improve the blood oxygen saturation levels as they relieve apnea in 20-75% of patients. They reduce AHI to < 10 events per h or bring about 50% reduction in AHI. Oral appliances also reduce the AHI to normal in 50-60% of patients.

Advantages

The main advantages of using oral appliances are that there is good patient compliance and the appliances are noninvasive and relatively inexpensive; they can also be easily carried anywhere by the patient.

Side effects and complications

Dental malocclusion (21%), TMJ pain (15%), and TMJ dislocation (<5%) are the side effects of MRDS. Other side effects include excessive salivation, tongue dryness, tooth pain, posterior open bite, and insomnia. The overall incidence of side effects with MRDs is reported to be 25-60%, though these side effects were often mild and resolved with adjustment of the device.[32] Complications with oral appliances include limited degree of lateral freedom during jaw movements. Recalls are necessary at a minimum at 2 weeks, 1 month, and thereafter every 6 months. The appliances are retained tightly by the remaining dentition and place almost orthodontic like forces on the teeth. They may also become loose or can distort or break and hence maintenance is mandatory.[36]

SURGICAL PROCEDURES

The most common surgical procedures for managing OSA modify upper airway soft tissue, including palate, tongue base, and lateral pharyngeal walls. The most extensively studied procedure is *Uvulopalatopharyngoplasty,* which involves resection of the uvula and part of the soft palate. Other procedures include lateral wall pharyngoplasty and tongue reduction procedures. The bony structures of the face can also be modified to manage OSA.

Maxillomandibular advancement, in which the upper airway is enlarged via Lefort I Maxillary and Bilateral Mandibular Osteotomies with forward fixation of the facial skeleton by approximately 10 mm causes mean reduction of 80% in AHI, consistent with a mean (SD) change of −47.8 (25.0) events per hour. Hypoglossal nerve stimulation is a newer surgical procedure that increases pharyngeal dilator muscle tone during sleep.[37-41]

FUTURE STUDIES

OSA is common and the prevalence is increasing. Daytime sleepiness is among the most common symptoms, but many patients with OSA are asymptomatic. The treatment of sleep apnea requires requires a skilled multidisciplinary team. The dentist can also identify a patient with symptoms of snoring and OSA and refer him/her for medical and sleep evaluation. Dentist can treat OSA either by surgical procedures or oral appliance. As oral appliance is noninterventional, it is most accepted by patients. The Association of American Sleep Disorders has published guidelines about the appropriate use of oral appliance therapy and defines the respective roles of the physician and the dentist in this type of care. Further research is necessary for advancement of newer Oral Appliances allow greater lateral jaw movement, cover all of the dentition, and provide better retention. Adjustable (titratable) appliances allow the clinician to titrate the amount of mandibular protrusion in order to obtain an adequate treatment response.

Declarations

Ethics approval and consent to participate

Obtained from institutional ethics committee

Consent for publication:- I hereby give the consent for publication on behalf of all the authors

Availability of data and material: The data and material was obtained from the department after obtaining the permissions from authorities

Competing interests: None

Funding: Self Funded

Authors' contributions:
All TheAuthours Collectively Performed The Study

Acknowledgements: None

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