Obstructive Sleep Apnea: A complete Review for Dentists
Dr. Ajay Ghanshyambhai Patel B.D.S., M.D.S.*

Absdent Orthodontic and Implant Center, Opposite JK Park Bus Stop, Chandlodiya, Gota, Ahmedabad Gujarat, India

DOI: 10.36347/sjds.2021.v08i10.003 | Received: 16.10.2021 | Accepted: 20.11.2021 | Published: 25.11.2021

*Corresponding author: Dr. Ajay G Patel

Abstract
Obstructive Sleep Apnea is a complex disorder and has the characteristic finding of collapse of the upper airway during sleep. The effects of OSA may be widespread and can affect the cardiovascular, pulmonary, and neurocognitive systems. OSA has higher prevalence in men than women. The clinical symptoms can help to identify patients with OSA but it is also common to have no signs and symptoms in the initial stages. Obesity can be a contributing factor for OSA. OSA is identified as a major factor for cardiovascular morbidity such as systemic and pulmonary hypertension, heart failure, atrial fibrillation, and other arrhythmias. The screening for OSA includes the use of symptom questionnaire, CBCT for airway measurements, and confirmed by polysomnography. The management primarily includes the use of continuous positive airway pressure, orthodontic options, and surgical options.

Keywords: Sleep Apnea; Obstructive; Airway Obstruction; sleep disordered breathing.

INTRODUCTION
Obstructive sleep apnea (OSA) is a respiratory disorder caused due to upper airway obstruction during sleep [1]. The decrease in tone of upper airway muscles while sleeping results in a collapse of the upper airway. This occurs mostly during the inspiration phase of breathing that leads to intermittent occurrences of hypopnea and apnea [2, 3]. During these episodes, the arterial oxygen saturation levels decrease and this can result in autonomic dysregulation [3]. The changes occurring due to the occurrence of apnea and hypopnea result in chronic conditions in cardiovascular, pulmonary, and neurocognitive systems [2, 3]. Obesity has been presumed to be associated with this condition. Bickelmann et al. described obesity hypoventilation syndrome (OHS) in a report regarding an obese business executive who presented to the hospital with the complaint of excessive daytime sleepiness [4]. The broader condition is known as sleep-disordered breathing which covers many conditions such as obstructive sleep apnea, central sleep apnea, and obesity hypoventilation syndrome.

In the current literature, OSA is the most prevalent and clinically significant sleep disordered breathing disorders. OSA is known to be linked with multiple conditions such as hypertension, atrial fibrillations, heart failure, cerebrovascular accidents, pulmonary hypertension, and others [2, 5, 6]. The goal of this review is to compile and better understand the impact of OSA on other organs and how to manage it.

Prevalence and risk factors
OSA has higher prevalence in males than females [1, 2]. The worldwide prevalence of OSA has been obtained from numerous countries such as Brazil, Germany, Spain, China, Switzerland, USA and it has been extrapolated from this data that about 1 billion people between the age of 30-65 years have OSA [7]. Of these people with OSA, 425 million are presumed to have moderate to severe OSA [7]. The prevalence of SDB for patients from 30 years to 50 years of age is found to be 10% in North America and 3% among women from the same age bracket; and 17% in men from 50 years to 70 years of age and 9% in women in the same age bracket [3]. Numerous risk factors are associated with OSA and many of these can be observed by a thorough examination. The most important factor for OSA is obesity and high body mass index. A linear correlation has been observed between OSA and obesity [1, 6]. Other factors such as having a neck circumference of more than 17 inches in men and 15 inches in women, males, age of more than 50 years increase the risk for OSA [1, 2]. Some contributing factors for OSA could be menopause, neuropathy, myopathy, craniofacial anatomical structure, family history, smoking, nasal congestion [2, 5, 8]. Patients with syndromic conditions may suffer from OSA.
example, patients with acromegaly may develop OSA due to macroglossia that is large tongue, and they can also develop central sleep apnea due to altered respiratory control.

Clinical Symptoms
The clinical symptoms of OSA play a vital role in diagnosis and identification of patients with OSA. But the symptoms vary in frequency, intensity, and severity in different patients. The usual clinical symptoms are fatigue, increased daytime sleepiness, snoring, drooling of saliva, nocturnal gasping, nocturnal choking, headaches, sleepiness while driving. Patients with OSA have a higher change of motor vehicle accident and collisions [9]. OSA can contribute to cardiovascular disease. OSA produces a chronic inflammatory state, which causes increased atherosclerotic changes in the blood vessels of the patient. In addition, OSA leads to fragmented sleep, intermittent hypoxemia, hypercapnia. These changes stimulate increased activity of sympathetic system, systemic inflammatory changes, and higher oxidative stress. The final results of the changes is that endothelial dysfunction and metabolic dysfunction which results in higher cardiovascular disease. OSA can also cause systemic hypertension. It has been found that if the OSA is treated and the apnea-hypopnea index is decreased, then a decrease in the mean blood pressure can be observed [10, 11]. In patient with heart failure, a higher prevalence of sleep disordered breathing is observed [12]. In a study by Paulino et al., it was found that prevalence of obstructive sleep apnea was as high as 70 percentages [13]. OSA can act as contributory factor for acute coronary syndrome. OSA causes many changes in vascular system including intermittent hypoxemia, development of acidosis, increased blood pressure, and sympathetic vasoconstriction leading to acute coronary syndrome [14]. Atrial fibrillation is also found to be linked with OSA. Infant, atrial fibrillation is the most common arrhythmia found associated with OSA. The prevalence of atrial fibrillation with OSA is found to be 5 percentae [15]. Also, patients with OSA are more likely to develop atrial fibrillation post coronary artery bypass graft surgery [16].

Diagnosis of OSA
The clinical symptoms of OSA are key in identifying and screening the patients for potential OSA. It should be noted that no signs and symptoms are also common in OSA patients. Screening questionnaires can help in identifying such patients in an outpatient setting. For symptomatic patients, it can be determined whether they should be referred for polysomnography.2 Polysomnography is the diagnostic method used to confirm the presence of OSA. CBCT analysis of airway can identify the minimal cross-sectional area and any areas of obvious obstruction in airway. 2D radiographs should not be used for this purpose as 2D radiographs give erroneous images with changes in head position and this can lead to misdiagnosis [17]. The questionnaires used for screening OSA patients are sensitive but not very specific [18, 19]. The questionnaires used for identifying patients with OSA are STOP-bang include questions on snoring, tiredness, apnea, blood pressure, BMI, age, neck circumference, and gender. The berlin questionnaire has ten sections with three different categories and include data on snoring, fragmented sleep, feeling sleepy while driving, apnea, hypertension, BMI [19]. After screening with such questionnaires, the patients who are at high risk are referred for polysomnography. Polysomnography provides the apnoea to hypopnea index of the number of episodes during the sleep period [20]. Artificial Intelligence can be used to analyze radiographs, and provide an interpretation to the investigator [21]. If enough data for OSA tests is fed into machine learning algorithms, the process of diagnosis of OSA can be automated in the future.

Management of OSA
Continuous positive airway pressure (CPAP) is the primary management strategy for OSA. CPAP is used to decrease the symptoms of sleepiness and improve the quality of life of patient with moderate to severe OSA [2, 22]. CPAP provides positive airway pressure during sleep and prevent the collapse of the upper airway [20]. Other options for management of OSA are dietary habits, regular exercise, and weight loss which can reduce both the symptoms and severity of OSA. Bariatric surgery can be used an option in extreme cases and can result in significant improvement [23]. Other options include mandibular advancement appliances which posture the mandible, or tongue forward. Mini-screw assisted rapid palatal expansion (MARPE) appliances have been shown to increase the airway volume [24]. The dimensions of upper airway can be affected with bone-borne MARPE or tooth-bone-borne MARPE [25]. Such appliances can lead to extrusion of molars and some changes in occlusion but no significant effects on the condyle position [26]. Appliances are also available that can stimulate the soft palate during sleep, although not found to be very effective [27]. When the maxilla is moved forward with protraction, it can have some beneficial effects on airflow [28, 29]. Osteoperforation result in increased inflammatory response and more tooth movement and can be used in combination with such orthodontic treatment options [30]. Surgical options include tonsillectomy, uvulopalatopharyngoplasty, surgery for reduction of tongue size, as well as maxillomandibular advancement surgery [31]. Dental appliances such as mandibular advancement devices can expand the airway and stabilize the airway and decrease the collapse and can be used when patients do not want CPAP appliance [31, 32].

CONCLUSIONS
OSA affects different organs of the body. OSA could present initially with cardiovascular and neurological symptoms rather than respiratory
symptoms. It is valuable to use clinical judgement and to identify such patients with clinical symptoms and having them fill out the screening questionnaires. The final diagnosis of OSA can be performed with polysomnography and the management is undertaken with different methods depending on the physician, dentist, and patient interaction.

REFERENCES


