REVIEW ARTICLE

Obstructive Sleep Apnea And Orthodontics Part 1- Diagnostic Review

V.Shivasankar[1], S.Dhivakar[2], M.Kandasamy[3], S.M.Abdul Rahman[4]

ABSTRACT

Obstructive Sleep Apnea is a prevalent but under recognized chronic sleep related breathing disorder with associated substantial morbidity and mortality. Contribution from various health care specialties could lead to effective treatment benefits when considering treatment for OSA. Orthodontics is not just limited with mere alignment of the teeth and smile esthetics but it has an expanded health care role that has established a new standard of health care for OSA patients. Snoring is often an overlooked issue but there could be an underlying pathology that might be a symptom of Obstructive Sleep Apnea. This review article explores various diagnostic approaches for a definite distinction between snoring and Obstructive Sleep Apnea to formulate the ideal treatment plan.

Keywords: Obstructive sleep apnea, Orthodontics, Polysomnography, Snoring

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[1]Reader, [2]Professor, Department of Orthodontics, Rajas Dental college &Hospital, [3]Senior Lecturer, Department of Oral medicine and Radiology, [4]Senior Lecturer, Department of Oral and Maxillofacial Surgery, Kavalkinaru Jn,Tirunelveli, India

Corresponding author: V.Shivasankar, Reader, Department of Orthodontics, Rajas Dental college and Hospital,Kavalkinaru Jn,Tirunelveli

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INTRODUCTION

Obstructive sleep apnea is a chronic, progressive and disabling sleep related breathing disorder, characterized by recurrent episodes of partial or complete upper airway obstruction during sleep.[1] It is manifested as reduction in airflow (hypopnea) or complete cessation of airflow(apnea) and repetitive arousals during sleep and excessive day time sleepiness. It has a direct relationship with hypertension, glucose intolerance, cardiovascular diseases, leading to cardio metabolic morbidity and mortality.[2-4]

Modern Dentistry has crossed the boundaries of conventional oral health and plays a vital role related to the overall health of the individual.

HISTORY OF OSA

In 1918 Sir William Osler coined the term "Pickwickian" that refers to the obese, hypersomnolent patients in homage to Charles Dickens' imaginary character "Joe".

In 1956 Dr. Bickelmann et al reported that the "Pickwickian syndrome" was associated with extreme obesity and alveolar hypoventilation.[5]

In 1965, Dr .Gastault mentioned pickwickian patients had repetitive apnea events during sleep.Dr.kuhlo and colleagues suggested marked improvement in apnea patients treated with tracheostomy. Guilleminault described the total no of apnea and hypopnea episodes per hour of sleep as apnea –hypopnea index(AHI) in 1973. In 1977,Guilleminault and Dement coined the term sleep apnea syndrome in individuals with apnea ,associated with hypertension and electro cardiographic pathologies.[6]

In 1978 john remmers explained the interaction between sleep ,breathing muscles of chest, muscles of chest, muscles of the upper airway and how upper airway collapse during sleep that leads to obstructive sleep apnea. Obstructive sleep apnoea syndrome, defined as an apnoea/hypopnoea index (AHI) of 5 or more—that is,at least five apnoeic/hypopnoeic events per hour of sleep—plus reported sleepiness, is a common form of SDB. According to the American Academy of Sleep Medicine recommendations, OSA is defined with AHI >5, and it is classified as mild OSA with AHI of 5 to 15; moderate OSA with AHI of 16 to 30; and severe OSA with AHI > 30.[1]

Overweight middle-aged adult men have the highest prevalence of the disease [8,10]yet women and an increasing number of children are also affected by OSA .[9] This condition affects 2% to 4% of adults aged from 30 to 60 years; prevalence increases with age.

Term	Definition
Apnoea	Cessation of airflow of at least 10 seconds
Hypopnoea	≥50% decrease in airflow amplitude of at least 10 seconds; or <50% decrease In airflow amplitude associated with either an arousal or oxygen desaturation of ≥3%
Respiratory effort-related arousal	An event characterised by increasing respiratory effort for 1 ≥0 seconds, leading to an arousal from sleep but which does not fulfill the criteria for a hypopnoea or apnoea
Apnoea/hypopnoea index	No. of apnoea + hypopnoea episodes per hour of sleep
Respiratory disturbance index	No. of apnoea + hypopnoea episodes + arousals per hour of sleep

Table-1: Definitions of terms used in obstructive sleep apnoea[7]

Sleep apnea syndrome

Guilleminault et al. defined the term "*sleep apnea syndrome*" as at least 30 apneas/night or 5 apneas/hour and each apnea should be at least 10 seconds long. Sleep apnea is the most common sleep disorder related to breathing. There are 3 types of sleep apnea: Obstructive, central, and mixed (a combination of both forms).

In Obstructive sleep apnea ,there may be partial or complete obstruction at various level of the upper airway leads to apnea or hypopnea. There is a decrease in oxygen saturation. The throat and abdominal breathing is quite normal.

Snoring and sleep apnea

Snoring is an inspiratory noise caused by the vibrations of an enlarged and flabby soft palate. The presence of snoring alone is a poor predictor of OSA. Thus, it must be correlated with other accompanying clinical features. Similarly, snoring absence does not exclude OSA. If severe, snoring can affect social relationship and become one of the main complaints of patients. Talking to the family members can be very helpful; they can often report signs, such as apnea or falling asleep unintentionally (that the patient may be unaware of or deny). Therefore, patients can report awakening during choking episodes. But this is less common among females. Most of the obstructions were seen in the retroglossal and retropalatal tissues of the oropharynx. The base of the tongue is a common site of for hypopharyngeal obstruction in sleep apnea.

Central sleep apnea is characterised by apnea episodes during sleep with no associated inspiratory effort as a result of the absence of neural output from the respiratory centres. With CSA, oral breathing and throat and abdominal breathing all cease at the same time. Central apnea is distinguished from obstructive apnea due to the lack of respiratory effort (docume nted by EMG). This distinction is essential because treatment by the dental professional is mechanical and only effective for obstructive apnea.[11]

Mixed apnea initially started as an unobstructed centr-al apnea, followed by thoracoabdominal movem- ents with air way obstruction. It is more frequent than central apnea but less frequent compared with obstructive type.

Patho-physiology

Breathing physiologically occurs when the intercostal muscles and the diaphragm receive electrical impulses from the brain. These impulses are responsible for their contraction. During muscular contraction, the ribcage expands and create a small vacuum (negative pressure) inside the chest drawing air into the lungs. Pharyngeal abductor and dilator muscles prevents the airway collapse. In day time respiration,they are activatived rhythmically and during sleep it becomes hypotonic.

Apnea occurs when the throat muscles and tongue relax during sleep and partially block the opening of the airway. The activity of the diaphragm, the chest and the abdomen only causes a tightly sealed blockage. As of now, little is known about the compliance of the pharyngeal tissues. Nonetheless the conditions that reduce airway dimensions result in OSA.

Electromyographic studies have established the relationship between the respiratory cycle and genioglossus muscle activity. These studies are focused on the role of tongue in the occlusion of airway during sleep. It has been hypothesized that impaired genioglossal function allows the prolapse of the tongue against the posterior pharyngeal wall on inspiration during sleep.[12] Both children and adults are equally affected. However, the prevalence, etiology and pathophysiology of the disorder differ from one group to another.

OSA pathophysiological factors are usually divided into three categories, whose complex interplay may explain the variable response to treatment:

- Anatomic factors that effectively reduce airway caliber;
- Non-anatomic factors that promote increased upper airway collapsibility and include:

- mechanical factors that are passive and related to tissues properties; and
- Neurological factors that change with the state of awakening or sleep

PREDISPOSING FACTORS

Individuals with OSA are mostly obese, older males, habitual snorers and with a possible family history of enlarged tonsils and adenoids, GERD, smoking, hypothyroidism, acromegaly, nasal congestion and neuromuscular problems.

RiskFactors

Obesity,[16] Older Age, Male,habitual snorers, family history, Enlarged tonsils and adenoids, GERD, smoking, hypothyroidism, acromegaly[17,18] nasal congestion, neuromuscular are prominent risk factor.
The most common symptom of OSA is excessive day time sleepiness. To evaluate the patient sleep health, Epworth sleepiness scale would be very useful screening test. Drawbacks of this test is not able to differentiate OSA from other sleep related breathing disorder such as central apnea, restless leg syndrome, narcolepsy.[11]

Clinical Evaluation

The physical examination is frequently normal in OSA, but often presenting with obesity (defined as a body mass index greater than 28 kg/m^2) and neck diameter greater than 16 inches. The upper airway should be evaluated in all patients, particularly in non obese adults with symptoms consistent with OSA. Features associated with the presence of OSA are as follows narrowing of the lateral airway walls, which an independent predictor of the presence of OSA in men but not women, enlarged tonsils, enlarged floppy uvula, retrognathia or micrognathia, soft palate edema/erythema, high, arched hard palate.

POLYSOMNOGRAPHY[19]

The gold standard diagnostic test for OSA is the overnight in-laboratory polysomnography. It consists of various polygraphic reading from surface leads for electroencephalography, electrooculography, electro-myography, electrocardiography. Nasal pressure tran-sducer, pulse oximetry,tracheal microphone, sensors .
These recordings will identify different types of apnoeas and hyponoeas during sleep.
Polysomnography (sleep study) results can reveal the cessation of air flow for 10 seconds even with maintenance of respiratory effort, five or more episodes of apnea per hour and a decreased oxygen saturation of at least 4% during episodes. These findings are indicative of OSA.
There are several problems encountered during a sleep study. At times, a patient does not sleep long enough to obtain all the data needed. Polysomnography cannot provide data from patients who have mild OSA only at home or only after using certain medications or alcohol but who do not experience any episodes during the sleep study. Because polysomnography is expensive and labor intensive, efforts are underway to find a better method of diagnosing or screening for OSA.
Nocturnal pulse oximetry,[20] which measures a patient's oxygen saturations throughout the night. Home studies may be useful in patients who have more severe sleep apnea with significant oxyhemoglobin desaturations that are easy to document with fewer parameters. Because severe apnea is often associated with significant arterial desaturation, it may be possible to use simple and inexpensive pulse oximetry as a screening method for the most severe disease. However, negative results do not rule out the presence of a sleep disorder.
MULTIPLE SLEEP LATENCY TEST[21] - The Multiple Sleep Latency Test (MSLT) measures the speed of falling asleep. A multiple sleep latency test may also be performed to assess the level of daytime sleepiness. The average adult requires 10 or more minutes to fall asleep during the day. A mean sleep latency of less than 5 minutes is considered abnormal. The MSLT may be useful to measure the degree of excessive daytime sleepiness and to rule out other types of sleep disorders. MSLT consists of 4-5 naps of 20 minutes duration every 2 hours during the day. The latency to sleep onset for each nap is averaged to determine the daytime sleep latency. Normal daytime sleep latency is greater than 10-15 minutes. OSA is generally associated with latencies less than 10 minutes.

Cephalometric radiographs

Lateral cephalometry is widely used in clinical practice because of its relative simplicity, easy accessibility, low cost and minimal radiation. It provides a lateral radiographic view of the head and neck in a standard plane with a specific emphasis on bone and soft tissue landmarks. The pharynx is a three-dimensional structure. Hence, the patients are usually evaluated while they are awake and at an upright position. This technique unveils a variety of soft and hard tissue abnormalities that could be indicative of narrow and collapsible upper airways. Cephalometry

Nose:	Deviated septum, Enlarged turbinates ,Polyps
Nasopharynx	Enlarged adenoids,
OroPharynx [10]	Velopharynx, Retropalatal ,Mesopharynx,Tongue base, Retroglossal Retrolingual,Velum palatinum,Transpalatal
Larynopharynx [10]	Hypopharynx, Pharyngolarynx, Subpalatal, Supralaryngeal

Table-2: Possible Sites of Obstruction in OSA

Snoring
Apneic pauses (choking, gasping, snorting during the night)
Restless leg syndrome (RLS); restless sleep and increased body movements
Bruxism (nocturnal tooth grinding)[13]
Nocturnal and daytime enuresis [14,15]
Sleep position (side and stomach sleepers)or neck hyperextended[2,10,11]
Growth failure/restriction
Sleep walking or sleep terrors
Obesity

Table-3: Symptoms and predictors of OSA

explains the pathophysiology, by identifying the most noteworthy craniofacial characteristics associated with OSA.

Cephalometric Characteristics Of OSA

Common clinical and radiographic characteristics of OSA patients mentioned by Alan Lowe et al extended and forward natural head posture, lower hyoid position, Mandibular retrognathia, retruded maxilla, posterior vertical maxillary deficiency, retropositioned tongue, HOP and high mandibular plane angulation, short chin-neck line, decreased PAS on lateral cephalogram, poor definition of gonial angles, and, often Class II dental occlusion.[22]

J. M. Battagel et al compared the OSA patients and healthy control and found difference in the lengths of the mandibular body and cranial base and in cranial base angulation in OSA subjects. The width of the oropharynx was significantly narrower in this group, particularly in the post-palatal region. The area of the soft palate was increased although that of the tongue was not. Intermaxillary space length (the distance between the posterior pharyngeal wall and the tip of the lower incisor) was decreased, and thus the area in which the tongue had to function was smaller in OSA subjects.[23]

Renata L Riha et al conducted a case control study using sibling pairs , Men and women with OSAHS have a lower-set hyoid bone than do those without OSAHS.[24]

Enache et al. concluded the positive correlation between AHI and ANB angle in steiners analysis and class II antero-posterior maxillomandibular relationship can predispose to OSA[25]

Tangugsorn et al highly recommended cephalometric analysis as one of the important diagnostic tool for OSA.They analysed the craniofacial morphology in OSA patient and control group and found out significant aberrations in OSA group were shorter dimension of cranial base with slight counter-clockwise rotation and depression of clivus; shorter maxillary length with normal height; maxillo-mandibular retrognathia related to nasion perpendicular plane (N perpendicular FH) despite normal angles of prognathism,47 per cent of the OSA group had mandibular retrognathia, increased anterior lower facial height and mandibular plane angle; reduced size of bony pharynx, inferiorly positioned hyoid bone at C4-C6 level, deviated head posture with larger cranio-cervical angle.[26]

Bernard Deberry-Borowiecki conducted a detailed cephalometric analysis adult patients with obstructive sleep apnea (OSA) and age- and sex-matched controls. Statistical findings show that OSA patients are different from controls in at least five ways: their tongue and soft palate are significantly enlarged.,. the hyoid bone is displaced inferiorly.,the mandible is normal in size and position (no micrognathia or malocclusion), but the face is elongated by an inferior displacement of the mandibular body,. the maxilla is retropositioned and the hard palate elongated, the nasopharynx is normal, but the oropharyngeal and hypopharyngeal airway is reduced in area by an average of 25%, a factor that could produce or enhance OSA symptoms.[27]

Computerized tomography (CT)

Pharyngeal obstruction can be evaluated in CT scan high resolution images. Retro palatal and retro lingual areas sagittal or cross sectional images are used to identify the obstruction sites and identify the tongue size and position. Compare to MRI it has poor resolution ,it is not frequently used for UA evaluation. Advantages of the CT to visualize upper airway is the ability to scan the entire UA ,ability to combine with PSG data,avoids the use of UA anesthesia Allows visualization of events outside the pharyngeal airway. Limitations of CT is inability to record full night sleep, high radiation exposure.

Magnetic resonance imaging (MRI)

It is a non invasive method, this high contrast resolution allows scanning in multiple planes. Functional factor is different during sleep and awake

situation, sorecording is necessary in both states. Some people may find Loud noise of MRI may be a disturbance in sleep.

CONCLUSION

OSA is a common sleep related breathing disorder, which affects all age groups. In OSA pathophysiology, anatomical factors alone are not the whole story the coordination between collapsing and dilating forces is an important concept and there is increasing evidence that the quantity and pattern of ventilation plays a substantial role in airway collapse as well as the presence of upper airway neuropathology. Most reliable and gold standard test for OSA diagnosis is overnight polysomnography method. Dynamic MRI also very useful to diagnose OSA.

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