REVIEW ARTICLE

Obstructive Sleep Apnea: Prevalence and Diagnosis- A Review

Fahad Ahmad*, Palvi Gupta**, Manhar Khullar***

Abstract

Sleep apnea is a common disorder in which breathing is briefly and repeatedly interrupted during sleep. There are three types of sleep apnea; Central sleep apnea, Obstructive sleep apnea, and Mixed or Complex sleep apnea. Obstructive sleep apnea (OSA) being more common, is characterized by obstruction of the upper airway and subsequently cessation of breathing during sleep. Various epidemiological studies have been done in different countries to assess the prevalence of OSA which shows that it is not only common in developed but in developing countries too. Population based studies show that 2% of women and 4% of men are affected with symptomatic OSA. However prevalence of asymptomatic OSA is high which affect 20%–30% of the middle-aged population. Overweight or obese, older and the patients with craniofacial anomalies are at more risk. Excessive day time somnolence and fatigue are common complains of patient with OSA. Depression, intellectual deterioration, pulmonary hypertension, polycythemia, and cardiovascular morbidity may also develop in advance cases. Polysomnography or sleep study is gold standard for diagnosis of OSA. Endoscopic and Cephalometric examination also help in identification of site of upper airway obstruction and treatment planning. Lifestyle modification, medical and surgical options are available for the treatment. Continuous Positive Airway Pressure (CPAP) has been proved to be effective in reducing symptoms, cardiovascular morbidity and neurocognitive sequelae, but it is often poorly tolerated. Oral appliances and various surgical procedures are also indicated. Early identification and treatment of the disorder can save patients from many serious and life threatening complications as well as improve their physical, psychological and social status.

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Keywords: Obstructive sleep apnea; Epidemiology; Prevalence; Polysomnography; Endoscopy; Cephalometric.

Introduction

Sleep apnea is a disorder characterized by either complete cessation of breathing or occurrence of shallow or infrequent breathing during sleep for seconds to minute. Complete cessation of breathing is called apnea and abnormal breathing event is known as hypopnea. Sleep apnea is classified

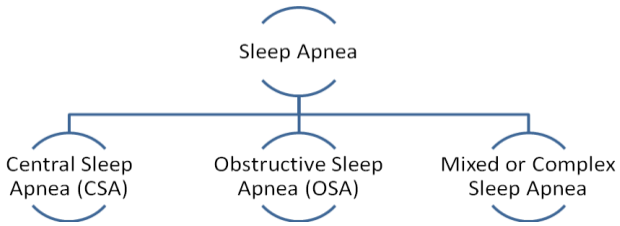
*Senior Lecturer, Department of Oral & Maxillofacial Surgery, Saraswati Dental College & Hospital, Lucknow; **Senior Resident, Department of Dentistry, Chintpurni Medical College & Hospital, Bungal, Pathankot; ***Practitioner, Gems Multispeciality Dental Clinic, Chandigarh

Address for Correspondence:
*Dr Fahad Ahmad, Department of Oral & Maxillofacial Surgery, Saraswati Dental College & Hospital, VPO Malhaur, Lucknow, U.P. -

under dyssomnia, means abnormal behavior or psychological events occur during sleep.[1]

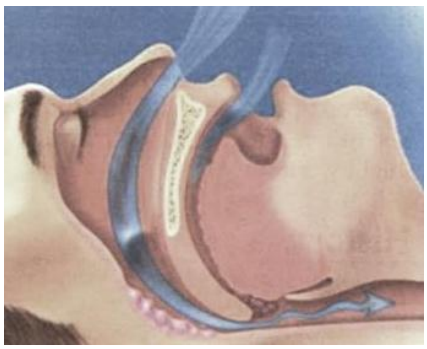
Classification

Sleep apnea can be divided into 3 types.



Central Sleep Apnea (CSA) – CSA occurs in patients with CNS insufficiency or lesion that affects the signal of neural output from the respiratory center to the diaphragm and other respiratory muscles. Conditions associated with central sleep apnea include neoplasms affecting brainstem, brainstem infarctions, bulbar encephalitis, bulbar poliomyelitis, spinal surgery, cervical cordotomy, and primary idiopathic hypoventilation. Shy-Drager syndrome, Leigh's disease, and multiple sclerosis may also cause central sleep apnea.[1,2]

Obstructive sleep apnea (OSA)– OSA is the most common form of sleep apnea. This disorder is characterized by obstruction of the upper airway during sleep resulting in repetitiveepisodes ofcessation of airflow. Inthis disorder respiratory effort, respiratory center drive, and diaphragmatic contraction are preserved which make it different from central sleep apnea (Fig 1a).[1,3]



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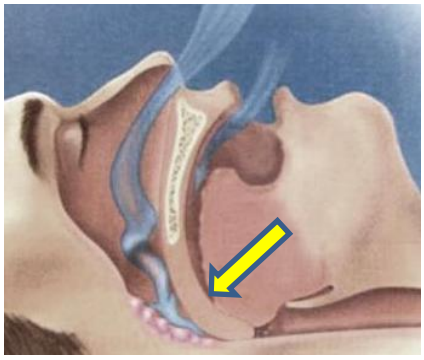

Obstructe d airway

Mixed or Complex sleep apnea–This disorder is the combination of both OSA & CSA. When OSAihs longstanding, episodes of central sleep apnea may develop. The mechanism of the loss of central respiratory drive during sleep in OSA is not clear but is most commonly related to acid–base andCO_2 feedback malfunctions stemming from heart failure. There is a many other diseases and symptoms relating to body mass, cardiovascular, respiratory, and occasionally, neurological dysfunction that have a synergistic effect in sleep disordered breathing[1,3].

Complex sleep apnea has recently been described as a novel presentation of sleep apnea. Patients with complex sleep apnea exhibit OSA, but upon application of positive airway pressure the patient exhibits persistent central sleep apnea. This central apnea is most commonly noted while on CPAP therapy after the obstructive component has been eliminated. Previously this type of apnea was managed by CPAP or Bi-level. Recently to manage this complex sleep

apnea some studies claimed that adaptive servo ventilation (ASV) modes of therapyprovide better results.[3]

Prevalence of OSA: The prevalence rates of OSA in many different countries are available now after large-scale epidemiological studies done by various clinician (Table-I). OSA is more common in male, around 2 to 3 times that of female. It is not only common in developed but developing countries also.[4,5] However, disease prevalence is more in obese, elder and subjects with craniofacial anomalies. Various epidemiological studies show that 4% of men and 2% of women aged more than 50 years suffer from symptomatic OSA (OSA with EDS and fatigue). However, OSA is often asymptomatic and the prevalence of patients with OSA, who do not present clinical symptom, might be as high as 20%–30% in the middle-aged population.[6,7] Various studies on the prevalence of OSA in different ethnic populations are given in table 1.

Table 1: Prevalence of OSA in different ethnic populations

Author	Year	Study population	Age in Years	Prevalence (%)
Gislason et al[8]	1988	Sweden	30–69	Men: 3
Young et al[9]	1993	American	30-60	Men: 4-25; Women: 2-19
Bearpark et al[10]	1994	Australia	40- 65	Overall-26
Olson et al[11]	1994	Australia		Overall -13.5
Bixler et al[12]	1998	USA (Men)	20-100	Overall - 3.3
Bixler et al[13]	2001	USA	20-100	Overall-4.7; Men: 7.2; Women: 2.2
Duran et al[14]	2001	Spain	30-70	Overall- 27; Men -28; Women-26
Ip et al[15]	2001	China (Men)	30-60	Men: 8.8
Huang et al[16]	2003	China	≥ 30	Overall: 20.4
Kim et al[17]	2004	Korea	40-69	Men -27; Women-16
Ip et al[18]	2004	China (women)	30-60	Women:3.7
Udwadia et al[19]	2004	India	25- 65	Men: 19.5
Sharma et al[20]	2006	India	30-60	Overall- 13.7;Men -19.7; Women-7.4
Vijayan & Patial[21]	2006	India	>18	Overall- 3.5; Men -4.4; Women 2.5
Kamil MA et al[22]	2007	Malaysia	30-70	Men - 8.8; Women - 5.1
Taj et al[23]	2008	Pakistan	30.4 +/- 12.	10% high risk for sleep apnea
Taj et al[24]	2009	Pakistan	>18	12.4% high risk for sleep apnea
Adewole et al[25]	2009	Nigeria	Young adult & Adult	19% high risk for sleep apnea
Reddy et al[26]	2009	India	30-65	Overall - 9.3; Men - 13.5; Women- 5.6
Tufik et al[27]	2010	Sao Paulo, **Brazil**	20-80	Overall -32.8%
Amra B et al[28]	2011	Persian	≥18	4.98% suspicious for OSA
Ansarin K et al[29]	2013	USA	≥16	Men - 9.8%; Women - 6.9%

Clinical manifestation of obstructive sleep apnea

Patients with OSA manifest variety of symptoms and clinical findings. The main complain of patients with OSA is excessive day time somnolence (EDS) and fatigue. The patients may experience serious social, economic, and emotional problems due to the EDS. It may predispose the patients to road traffic or occupational accidents. OSA patients provide history of snoring which has been present for several years.[1] In these

patients the snoring is interrupted periodically by complete cessation of airflow or apneic episode that last 30 to 90 seconds. Hyperventilation starts with a loud burst of snore or choking sound usually signals an end to the apneic episode. Other complaints, associated with OSA may be morning headaches and nausea which is related with the hypercarbia, develops with the repetitive hypo-ventilatory episodes. Depression, intellectual deterioration personality changes, andloss of libido may also develop.[30,31] The systemic hypertension which is a usual finding in OSA may be due to the catecholamine release triggered by the systemic hypoxemia.[32] In advanced cases, pulmonary hypertension, polycythemia, and cor-pulmonale may develop and become life threatening.'[32] However, most patients do not show these findings as their ventilation during wakeful periods is sufficient to prevent these abnormalities of chronic hypoxia. A prominent sinus dysrhythmia is commonly associated with the apneic episodes. The extent of bradycardia is directly proportional to the severity of the oxygen desaturation.[32,33] Increased vagal efferent tone mediates the bradycardia. The development of severe and lifethreatening medical complications from the apneic events clearly depends on the frequency, duration, and degree of hypoxemia and associated hypertensive response.[32]

Physical Findings

Obesity is the major finding in OSA. The increased body weight correlates with increased frequency of sleep apnea and hypoxemia. However, the morbidly obese, somnolent, hyperventilating patient with cor-pulmonale represents only a small number of sleep apnea patients. Lower BMI patients with OSA show more abnormal cephalometricsfindings in compare to obese people. Physical examination of OSA patients may reveal a short and thick neck, retrognathia, micrognathia, macroglossia, hypertrophy of the tonsils or adenoids, deviated nasal septum, or tumors in the nasopharynx or hypopharynx.[1,34] Both primary and secondary medical conditions are associated with OSA; owing to their effects on the upper airway anatomy. These may include tmj disorders, endocrine disorders such as myxedema, goiter, and acromegaly. Most patients with classic OSA have no identifiable craniofacial anomaly. However, there are a significant number of sleep apnea patients with craniofacial anomalies. Lowe and colleagues evaluated 25 patients of OSA and observed many alterations in craniofacial form such as posteriorly positioned maxilla and mandible, overerupted maxillary andmandibular teeth, a steep occlusal plane, proclined incisors, a steep mandibular plane, increased upper and lower facial heights, a large gonial angle,a posteriorly placed pharyngeal wall, and an anterior open bite in association with a long tongue. Bacon and colleagues studied 32 patients with sleep apnea by cephalometry and found an anteroposterior shortening of the cranial base, narrowing of the pharyngeal airwaywitha posterior facial compression, and an increased lower facial height.[1]

Diagnosis

A diagnostic evaluation of OSA includes a complete history and physical examination, fiberoptic endoscopy, radiologic evaluation, and polysomnography. Routine laboratory tests are also helpful for getting additional information. Except in severe cases, pulmonary function tests (PFT), electrocardiogram (ECG), arterial blood

gas analysis, and chest radiographs are often normal during wakefulness in sleep apnea patients. Other diagnostic tests that may be useful inevaluating sleep apnea patients include acomplete blood count (CBC), serum electrolytes, and thyroid function tests. Secondary polycythemia may be revealed by a CBC, and nocturnal carbon dioxide retention may be reflected by increased bicarbonate levels. Hypothyroidism can be identified from thyroid function test which is a contributing cause of sleep apnea.[1,2,3,35]

Clinical examination

After taking complete history from the patient and his/her bed partner, a complete clinical examination of the mouth, nasal, pharyngeal, and laryngeal areas must be performed. The main emphasis of the clinical examination should be the identification of anatomic abnormalities that may contribute to or produce obstruction during sleep. The nose is examined for a deviated nasal septum and enlargement ofthe turbinates. Micrognathia, retrognathia, and macroglossia should be examined. Tooth indentations on the lateral margin of tongue provide a clue for large tongue. Occasionally masses or tumors in the nasopharynx orhypopharynx may be found. In the pharynx, adenoid/tonsillar hypertrophy, a long soft palate, a large base of the tongue, and excess pharyngeal mucosa are potential causes of obstruction. The larynx is examined for vocal cord webs and paralysis ofthe vocal cords. OSA patients may present with any combination of these anatomic abnormalities. Clinical predictors of OSA include old age, male sex, increase BMI, Cephalometric measures, neck girth (>17 inch for male, >16 inch for female), inferiorly placed hyoid bone, Malampatti class III & IV, pharyngeal rugea or mass and craniofacial anomalies.[22,28,36]

Site of Obstruction

Various classifications have been given to describe the upper airway anatomy for OSA. These classifications are not universally accepted as physiology of airway is not considered. Although these classifications provide no aid in surgical decision making, help in documentation as well as education purpose.

Ikematsu[37] was the first to described obstruction in upper airway. On the basis ofanatomy of upper airway, he described various sites. These include six features which described oro-pharyngeal anatomy,

(a) Soft palate length ±50 mm
(b) Uvula length ±11 mm
(c) Wide uvula ±10 mm
(d) Pillar arch morphology (parallel, webbed, embedded, emerging)
(e) Oropharyngeal narrowing(anterior arch ±20 mm, posterior arch ±15 mm, shallow oropharynx ±5 mm)
(f) Enlarged tongue dorsum (oropharynx not seen with phonation)[36]

As the Ikematsu classification was difficult to use clinically. It was modified by the Fujita.

Fujita[38] classification of obstructive sleep apnea

1. Type I: Abnormalities of at the level of upper Oro Pharynx with abnormalities of the palate, uvula, tonsils, upper pharynx
2. Type II: patients with Oro Pharynx and hypopharyngeal obstructions
3. Type III: Hypo pharyngeal obstruction alone occurring form tissues of the tongue base, lingual tonsils, supraglottis and hypopharynx.

According to this classification only 20% to 25% patients have isolated oropharyngeal obstruction, 10% patients present hypopharyngeal obstruction and most of the OSA patients have mixed type of obstruction.[36,39]

Friedman developed a classification to predict the results of surgery, specifically uvulopalato-pharyngoplasty (UPPP), for patients with obstructive sleep apnea (OSA). The system includes an evaluation of palate position, tonsil size, and body mass index (BMI) to predict the success of UPPP. A modified version of the Mallampati classification is also used to evaluate palate position relative to oropharyngeal size. In the Friedman classification, the patient keeps his tongue in a neutral position, and 4 stages are used to describe the airway.[40,41]

The palate position is graded from I-IV, as follows:

- I: The uvula, soft palate, and tonsils/pillars are clearly visible.
- II: The uvula and soft palate are visible, but the tonsils are not.
- III: Only part of the soft palate is visible.
- IV: Only the hard palate is visible.

The tonsil size is graded from 0-4, as follows:

- 0+: A previous tonsillectomy has been performed.
- 1+: The tonsils are hidden within the tonsillar pillars.
- 2+: The tonsils extend to the tonsillar pillars.
- 3+: The tonsils extend beyond the pillars but not to the midline.
- 4+: The tonsils extend to the midline.

BMI is loosely included in the Friedman classification. The stages of disease are defined as follows:

- Stage I disease includes patients with palate position I or II, tonsil size 3 or 4, and a BMI of less than 40 kg/m^2.
- Stage II disease includes patients with palate position I or II and tonsil size 0, 1, or 2—or palate position III and IV and tonsil size 3 or 4—and BMI less than 40 kg/m^2.
- Stage III disease includes patients with palate position III or IV and tonsil size 0, 1, or 2.

All patients with a BMI of greater than 40 kg/m^2 are considered stage III. Patients with stage I classification are predicted to have successful results with UPPP. Stage II and III patients should have adjunctive treatment if they undergo UPPP, because the procedure does not have a high likelihood of cure. The strength of this classification system is still under review, but it has consistently shown to be a reliable indicator of prognosis after surgery.[1,2, 22, 28]

Endoscopic examinations

After application of topical anesthesia in the nasalcavity and pharynx, a fiberoptic endoscope is introduced through the nose. Insequential fashion the nasopharynx, oropharynx, hypopharynx, and larynx should be examined. The appearance and position ofthe soft palate, base of tongue, and lateralpharyngeal walls are evaluated. Changes in the position of the base of the tongue such as forward movement with protrusion ofthe mandible are noted. The appearance ofthe pharyngeal airway and degree of pharyngeal wall collapse is noted while thepatient performs a modified Müller maneuver.[1,36]

Cephalometric Examination

In OSA patient lateral cephalogram is performed routinely to evaluate any skeletal and soft tissue abnormalities. Lateral cephalometry has many

advantages such as its easy access, low cost, and low radiation exposure. Patientswith skeletal deficiencies are more likely tohave obstruction at the base of the tongueor at the level of the soft palate. According to **Riley et al** patients with OSA had an inferiorly positioned hyoid bone, a longsoft palate, and a narrowing at the base ofthe tongue.Themean mandibular plane to hyoid bone distance for normal subjects is considered as 15.4±3 mm. In OSA patients this distance increases. The mean Posterior nasal spine to tip of soft palate distance increases too which is considered 37±3 mm in normal subjects.[1,36]

Polysomnography

The gold standard diagnostic test for OSA is the overnight in-laboratory polysomno-graphy.[1,2,3,28,34,42] It involves multi-channel continuous polygraphic recording for electroencephalography (EEG), electrooculo-graphy (EOG), electromyography (EMG), electrocardiography (ECG), nasal airflow, thoracic and abdominal impedance belts for respiratory effort, pulse oximetry, tracheal microphone for snoring, and sensors for leg and sleep position. These recordings will identify different types of apnea and hypopnea as during sleep. Anapnea is defined as the complete cessation of airflow for at least 10sec. A hypopnea is definedas a reduction in airflow (30-50%) that is followed byan arousal from sleep or a decrease inoxyhemoglobin saturation (3-4%). Sleep apnea severity is assessed with apnea-hypopnea index (AHI), which is the number of apneas and hypopnea as per hour of sleep. According to the American Academy of Sleep Medicine recommendations:

- Mild OSA: 5-15 events per hour of sleep
- Moderate: 15-30 events per hour of sleep
- Severe: greater than 30 events per hour of sleep[36,43,44]

Computed Tomography

Computed tomography (CT) is an alternative to cephalometry and has been used to provide a quantitative assessment of the upper airway at various levels.[36]

Airway Manometry- Upper airway resistance measurement and manometry evaluate the pressure difference in areas with high possibility of collapse. This pressure is compared with the pleural pressure measured through the esophagus. But it is less used method due to its complexity.

Magnetic Resonance Imaging– MRI provide best resolution and detailed view of soft tissue of pharyngeal wall and its relation with airway. It produces noises and having cumbersome design of machine which make it difficult to sleep in it.[46]

Treatment

Life style modifications, weight loss and nasal continuous positive airway pressure (CPAP) are considered as the initial modes of therapy that should be initiated in obese patients with moderate obstructive sleep apnea. Currently CPAP is the most successful non-surgical treatment of OSA but compliance is poor.[33,34,47] Recently bi-level positive airway pressure(Bi-PAP) systems that allow independent regulation of inspiratory and expiratory pressures and the newest modification in CPAP systems, Auto-CPAP, have been used to more effectively treat obstructive sleep apnea and increase tolerance and compliance.[1] The use of a variety of prosthetic devices is another approach to treatment.There are various oral appliances available in

the market such as tongue-retaining device, Klearway appliance, Herbst appliance, Elastic Mandibular Advancement, Equalizer Airway Device etc. Side effects of oral appliance therapy are excessive salivation, xerostomia, soft tissue irritations, transient discomfort of the teeth and temporomandibular joint (TMJ), and temporary minor occlusal changes. Oral appliance therapy may cause permanent occlusal changes and severe TMJ discomfort. Surgical interventions are also indicated in OSA in certain conditions. It is considered as a quick cure of OSA. Currently the procedures used in the surgical treatment of obstructive sleep apnea include tracheostomy, nasal surgery, Uvulopalatopharyngoplasty (UPPP), and several orthognathic procedures.[48,49,50]

Conclusion

OSA is a morbid condition in which patients present with EDS. This uncontrolled and untimely sleep can cause serious automobile and occupational accidents. Moreover excessive day time sleepiness and fatigue may hamper professional success also. Much other co-morbidity can be arise in long term obstructive sleep apnea patients such as depression, decrease sexual desire and increase cardiovascular risk. Though this field is challenging in respect to diagnosis and treatment, evolving with new technologies and discoveries need multidisciplinary approach. Early diagnosis and treatment of the patients with OSA can improve their physical, psychological and social status.[3,4,6,7]

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