Outline of Diagnosis of Obstructive Sleep Apnea in Adults

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Abstract

Background: Sleep-disordered breathing means sleep disorders that involve difficulty breathing during sleep. It refers to momentary, often cyclical, cessations in breathing rhythm (apneas) or momentary or sustained reductions in the breath amplitude (hypopneas), sufficient to cause significant arterial hypoxemia and hypercapnia. These apneas and hypopneas are accompanied either by 1) a compromised, often completely closed, extra-thoracic upper airway ("obstructive" event); 2) a marked reduction or cessation of brain stem respiratory motor output ("central" event); or 3) a combination of central and obstructive events. Symptoms of OSA can be classified into those manifesting during sleep and those present during awake fullness. Several methods had been designed to evaluate the upper airway and to investigate the severity and location of its obstruction.

Keywords: Obstructive Sleep Apnea, diagnosis

Introduction

Sleep-disordered breathing means sleep disorders that involve difficulty breathing during sleep. It refers to momentary, often cyclical, cessations in breathing rhythm (apneas) or momentary or sustained reductions in the breath amplitude (hypopneas), sufficient to cause significant arterial hypoxemia and hypercapnia. These apneas and hypopneas are accompanied either by *1*) a compromised, often completely closed, extra-thoracic upper airway ("obstructive" event); *2*) a marked reduction or cessation of brain stem respiratory motor output ("central" event); or *3*) a combination of central and obstructive events **(1).**

The obstructive sleep apnea (OSA) results from interaction between the unfavorable anatomic upper airway susceptibility and the sleep-related changes in upper airway function. The wakeful state provides compensatory neuronal activation of dilator muscles of the hypopharynx. At sleep onset, this activation will be lost. Thus, in an anatomically compromised collapsible pharynx, the airway narrows and/or collapses resulting in a degree of OSA **(2).**

The prevalence of OSA is two to three times greater in males (4%) than females (2%). Moreover, the reported prevalence rates vary widely, and asymptomatic sleep apnea is more common than symptomatic, clinically significant obstructive sleep apnea **(3)**.

The pathophysiological causes of OSA likely include upper airway anatomy, the ability of the upper airway dilator muscles to respond to respiratory challenge during sleep, the propensity to wake from increased respiratory drive during sleep, the stability of the respiratory control system, and the potential for state-related changes in lung volume to influence these factors **(3)**.

Olsen and **kern (4)** believed that partial or total nasal obstruction can lead to hypopnea and apnea while **Young et al (5)** explained that OSA is most prevalent in the human in part because the hyoid bone, a key anchoring site for pharyngeal dilator muscles, is not rigidly attached to skeletal structures. Thus the human pharynx has no rigid support except at its extreme upper and lower ends were it is anchored to bone (upper) and cartilage (larynx); therefore, pharyngeal cross-sectional area will vary with lumen pressure.

Huang et al (6) showed that enlargement of soft tissue structures both within and surrounding the airway contributes significantly to pharyngeal airway narrowing in most cases of OSA. **Pevernagie et al (7)** added that volumetric time overlapped magnetic resonance imaging (MRI) or computer tomography (CT) images strongly implicate the thickness of the lateral pharyngeal walls as a major site of airway compromise, as the airway is narrowed primarily in the lateral dimension in the majority of OSA patients.

Schwab et al (8) showed that airway obstruction can occur in many areas of the nasopharynx, oropharynx and hypopharynx. The airway narrowing is a dynamic process, varying markedly among and within subjects and often includes the retroglossal and hypopharyngeal areas **(8)**.

Although non-obese individuals may suffer from OSA, obesity is the main epidemiologic risk factor. Several factors can be contributed as underlying mechanisms. They include generalized hypotonia and other factors that narrow the upper airway, such as a large tongue, enlarged tonsils, and increased total soft tissue in the pharynx or a retro-positional.Moreover, reposition of fat around the pharyngeal airway is likely to increase the collapsibility of the pharyngeal airway. Also, fat deposition around the abdomen leads to reductions in functional residual capacity, which would be predicted to reduce lung volume tethering effects on the upper airway. Finally, obesity has been associated with functional impairment in upper airway muscles **(9)**.

Schwartz et al (10) reported that central, or visceral, obesity is associated with the greatest risk for OSA. This suggests that factors other than pure mechanical load may contribute to the pathogenesis of respiratory disturbances during sleep. The concept is emerging that visceral fat depots, which represent a rich source of humoral mediators and inflammatory cytokines, can impact on neural pathways associated with respiratory control. Perhaps the well-studied adipocyte-derived factor; leptin, affecting respiratory control **(11)**.

Jose et al (12) stressed that maxillofacial injuries might be associated with life-threatening complications such as blockage of the airway which can occur immediately, later on, or after treatment of the initial injury**.** The situation may be aggravated by diminished consciousness, alcohol, and/or drug intoxication, as well as altered laryngeal and pharyngeal reflexes, making the patient vulnerable to the risk of aspiration. Furthermore, the condition may be complicated by the presence of broken teeth, dentures, foreign bodies, avulsed tissues, multiple mandibular fractures, and massive edema of glottis which can cause a direct threat to the airway.

During inspiration, the size of the pharyngeal lumen depends on the balance between the narrowing force that results from the present air pressure (below atmospheric pressure) and the dilating force generated by the contracted small muscles that attached to the upper airway. The normal contraction of these muscles with each inspiration stabilizes the floppy walls of the pharynx. At sleep onset, there is a reduction in pharyngeal luminal area and upper airway muscle activity, both of which are exaggerated in OSA. In general, any pathological change or normal variant that narrows the upper airway when awake will predispose the individual to obstructive apnea or hypopnea when asleep **(13).**

It was reported that muscles of the pharynx might become hypotonic during sleep to the degree that became unable to prevent the collapse of the airways resulting in difficulty breathing. These disruptions to breathing lead to intermittent blood gas disturbances (hypercapnia and hypoxemia) and surges of sympathetic activation. Loud snoring becomes a typical feature and in most cases it is associated with a brief awakening

from sleep (arousal). These events result in a cyclical breathing pattern and fragmented sleep as the patient oscillates between wakefulness and sleep. In severe cases respiratory events can occur more than 100 times per hour and typically each event lasts 20–40 seconds. They added that although it is a physiologic phenomenon of little consequence in healthy individuals, it may promote upper airway narrowing in susceptible individuals.

Moreover, the route of breathing during sleep appears to influence salivary flow and perhaps as a result may affect surface tension. Nasal breathing may reduce, and oral breathing may increase, surface tension forces of the liquid lining of the upper airway thus influences pharyngeal patency and perpetuate severity of OSA (**14)**.

Occasionally, obstructive sleep apnea can be caused by less common medical problems, including hypothyroidism, acromegaly and renal failure. Moreover, post-polio syndrome can result in inadequate neuromuscular control of the upper airway and lead to obstructive sleep apnea. Restrictive lung disease from scoliosis has also been associated with the disorder. In addition to the neurocognitive effects (e.g depression and gastroesophageal reflux disease; the major long-term sequelae to severe OSA should be assessed through use of CPAP treatment. They are: a) Cardiovascular: daytime systemic hypertension, stroke, or cardiac arrhythmias. b) Cerebrovascular infarction. c) Insulin sensitivity and homeostasis of glucose regulation **(1).**

Symptoms of OSA can be classified into those manifesting during sleep and those present during awake fullness.

Table 1: Symptoms of OSA; adapted from **Victor (1999)**.

During sleep	**While awake**
Loud snoring / snoring	Daytime sleepiness
Witnessed apnea by bed partner	Non-restorative sleep
Awaking with chocking	Lack of concentration
Nocturnal restlessmess	Cognitive deficits
Vivid , strange or threatening dreams	Changes in mood
Gastro-oesophageal reflux	Morning headache
Insomnia with frequent awakenings	Dry mouth
Nocturia	Impotence or decreased libido
Hypersalivation , teeth grinding	
Diaphoresis (sweating)	

Several methods had been designed to evaluate the upper airway and to investigate the severity and location of its obstruction **(8)**.It includes:

1- **Physical examination:** It includes reporting the general characteristics, mental status, blood pressure, height and weight, and collar size. A neck circumference greater than 16 inches in a woman or greater than 17 inches in a man correlates with an increased risk for the disorder **(15)**.
2- **Nasal examination:** is critical for diagnosing or treating OSA (12**)** as it is could identify potential anatomic sites of obstruction, such as a deviated septum, enlarged adenoids, swollen nasal turbinates, polyps, or other mass lesions.
3- **Pulmonary function testing:** is mandatory in patients who have chronic obstructive pulmonary disease or chronic interstitial lung disease as they may have worsening ventilatory function, as well as oxygen desaturation, during sleep **(13).**
4- **Mandibular alignment and dental occlusion.**
5- **Imaging methods:** They include:
   - **Somnofluoroscopy**: to evaluate the area of pharyngeal collapse and maximal airway narrowing during sleep. Also, it can predict surgical success in some patients undergoing uvulopalatopharyngoplasty (UPPP). However, its use remains limited especially in patients with obstruction distal to the velopharyngeal region **(16).**
   - **Magnetic resonance imaging** (MRI): allows for dynamic imaging of the airway with good resolution of the airway, soft tissues, and fat.
   - **Cephalometry**: may be used to evaluate the hyoid position and the craniofacial deficiencies (**17)**.
6- **Evaluation of excessive daytime sleepiness:** usually done through **Epworth Sleepiness Scale** (ESS): The subject was asked to grade the likelihood of falling asleep in each of eight everyday situations. Each one of these situations is scored from zero to three. An ESS score of greater than 11 out of 24 indicates daytime sleepiness, irrespective of age. Disadvantage of this scale is that it is a subjective scale that can be prone to misinterpretation by the patient with the result of high scores that may be due to causes other than OSA **(18)**.

Table 2: Epworth Sleepiness Scale **(18)**.

Situation	Chance of dozing
Sitting and reading	
Watching TV	
Sitting inactive in a public place (e.g. a theater or a meeting)	
As a passenger in a car for an hour without a break	
Lying down to rest in the afternoon when circumstances permit	
Sitting and talking to someone	
Sitting quietly after a lunch without alcohol	
In a car, while stopped for a few minutes in traffic	

Scoring	
0	No chance of dozing
1	Slight chance of dozing

2	Moderate chance of dozing
3	High chance of dozing

7- **Overnight polysomnography (PSG)**: It is the gold standard for the diagnosis and characterization of breathing-related sleep disorders**.** In this technique, multiple physiologic parameters are measured while the patient sleeps in a laboratory. They include measurement of oral and nasal airflow, respiratory effort, and pulse oximetry, along with a 2-lead electro-cardiogram, leg and chin electro-myelogram, electro-oculogram, and 2-lead electro-encephalogram. It helps us to classify sleep-related breathing disorders with calculation of the apnea \ hypopnea index as follows:
   - Obstructive apnea: continued muscle activity in the setting of cessation of airflow.
   - Central apnea: absent muscle and airflow activity.
   - Mixed apnea.
   - Hypopnea: drop in airflow by at least 50% and associated with oxygen desaturation.
   - Apnea \ hypopnea index (AHI): equals the total number of apneas and hypopneas divided by the total sleep time in hours. It helps in defining and grading the severity of sleep-disordered breathing.

PSG also used to evaluate the physiologic consequences of the respiratory events; specifically, associated oxygen desaturations, cardiac dysrhythmias, and sleep fragmentation. In patients in whom the findings on the overnight PSG are normal and the etiology of the daytime sleepiness remains unknown. These tests are conducted in a sleep Lab. with all psychoactive medications stopped for at least 2 weeks prior to test **(19).**

8- **Home evaluation:** used to screen patients for OSA. They are less expensive than laboratory testing and are certainly more convenient for patients. However, they do not measure as many physiologic parameters as formal laboratory tests. In addition, it is not possible to adjust the equipment during the night if this becomes necessary. Overall, home evaluation is useful when the results are clearly positive as negative results do not rule out the presence of a sleep disorder **(20).**

9- **The oropharynx and nasopharynx:** should be evaluated properly as a variety of anatomic features can have an impact on airflow limitation at this level. They include large tonsils, a long soft palate, a large uvula, a pharyngeal flap, posterior pharyngeal stenosis and scarring, a redundant fold, and tumors. Moreover, the relationship of the tongue, palate, and posterior pharyngeal wall should be estimated as they can cause posterior pharyngeal crowding and predisposition to OSA **(21)**. They could be done through**:**

a- Fiber-optic Nasal Endoscopy with Müller's Maneuver (FNMM):

Awake FNMM is usually performed by a single surgeon for investigation of retropalatal and retrolingual collapse. All patients should taught how to perform Müller's maneuver and given the opportunity to practice before nasal endoscopy **(22).**

All individuals would be examined underwent topical nasal administration of aerosolized 4% lidocaine and 0.25% phenylephrine solutions for adequate pre-procedure decongestion and topical anesthesia. Subsequently, patients will placed in a sitting position while a flexible fiber-optic nasal endoscope passes through 1 nostril into the nasopharynx. **Müller's maneuver** then performed by maintaining maximal inspiration with an open glottis against closed oral and nasal airways. The same maneuver and assessment then performed with the nasal endoscope placed past the velopharynx into the oropharynx for a measurement of retrolingual collapse **(22).**

The estimated degree of airway collapse will described qualitatively as a percentage change in cross-sectional airway area and divided into quartile groups of <25%, 25% to 50%, 50% to 75%, and >75% at each level. Most authors considered an obstruction of >75% to be significant enough to warrant surgical alteration at that particular level of airway **(23).**

Because FNMM is performed during wakefulness, there are limitations to its ability to accurately detect and predict the severity of airway collapse that occurs during sleep**.** Moreover, **Friedman et al. (24)** reported that there is no diagnostic gold standard existed for FNMM. So, the ability of the FNMM technique to predict the site and severity of airway collapse has been questioned as studies demonstrate disparity against objective measurements such as flex tube reflectometry and nasopharyngeal pressure measurements.

b- Drug-Induced Sleep Endoscopy Method (DISE):

It is an upper airway endoscopic evaluation method which allows assessment of upper airway collapse under sedation. It represents a diagnostic tool indicated in patients with snoring or OSA, in whom non-CPAP therapy is being considered. Anticholinergics can be considered to reduce upper airway secretions to aid better assessment **(25)**.

It is usually performed in the operating room by an expert surgeon. The target level of sedation is that of light sleep with arousal to tactile but not vocal stimulation. So, the use of benzodiazepines and other sedating medications is strictly prohibited. Once sedation is achieved, a flexible fiberoptic nasal endoscope will pass through the nose for inspection of the entire upper airway. The key to reporting and grading of the DISE findings is that it should include levels of collapse, the pattern of collapse (lateral, anteroposterior or concentric), as well as the degree of obstruction and vibration at the nose, retropalatal area, retroglossal area, lateral wall of the entire airway. Observation of at least 2 or more cycles (progression from snoring to obstruction or oxygen desaturation to the resumption of breathing) for each segment of upper airway was suggested to diagnose an upper airway collapse. Dynamic collapse is roughly described in terms of a percentage cross-sectional area change based on anteroposterior and lateral axes of collapse. The estimated degree of airway collapse at each level is usually recorded as >25%, 25% to 50%, 50% to 75%, and >75% **(22).** However, use of nasal local anesthesia and decongestant, whilst may ease scope insertion, could potentially alter nasal resistance and upper airway dynamics **(25)**.

Although findings of DISE have been shown to have good inter user and test-retest reliability as well as good correlation with the respiratory disturbance index, an over-estimation of severe airway collapse had been reported. **Huntley et al. (26)** referred it to the sedative use that characterizes DISE procedure.

Furthermore, there is a growing number of DISE classification systems which increase subjectively in the analysis of upper airway obstruction. The two popular systems are the VOTE system and the NOHL system **(26)**.

VOTE classification: It provides a clear and simple framework for robustly and accurately analysing upper airway obstruction at four anatomical levels: velum, oropharynx, tongue base and epiglottis. The severity of obstruction can be classified as 0, no obstruction (no vibration, < 50%); 1, partial obstruction (vibration 50–75%) and 2, complete obstruction (collapse, > 75%)**.** The configuration of obstruction at the velum level can be in anteroposterior, lateral or concentric direction. Oropharynx obstruction occurs in the lateral and concentric direction. Tongue base obstruction occurs only in the anteroposterior configuration. The configuration of epiglottis obstruction occurs in the anteroposterior direction **(27)**.

NOHL classification system is focused on assessing the severity and configuration of upper airway obstruction based on four anatomical zones: nose, oropharynx, hypopharynx and larynx. The severity of obstruction was categorised in conjunction with the Müller maneuver grading system. Grade 1 is an upper airway obstruction between 0 and 25% airway collapse and also defined as no collapse during Müller

manoeuvre. Grade 2 is an upper airway obstruction between 25 and 50% airway collapse. Grade 3 is an upper airway obstruction between 50 and 75% airway collapse. Grade 4 is an upper airway obstruction greater than 75% airway collapse and also defined as a total pharyngeal wall collapse during Müller manoeuvre. A generalist approach of anteroposterior, lateral and concentric configuration of obstruction was applied to the nose, oropharynx and hypopharynx. The laryngeal obstruction was assessed as positive or negative at the supraglottic and glottic level. The key advantage of NOHL classification is the provision of a scoring framework for upper airway obstruction during awake and sleep state. Its limitation is that two of these anatomical zones (nose and larynx) play significantly reduced roles in upper airway obstruction **(28)**.

Unfortunately, there is no standardized framework for deciding, planning and predicting surgical outcome based on any current DISE classification system which increases the likelihood of suboptimal surgical treatment decision, planning and outcomes for OSA management. Also, there is a growing divergence in analytic analysis between different clinical and research centers **(29).**

Soares et al. (22) showed a significant difference between FNMM and DISE in the identification of severe retrolingual collapse. They stressed that further scrutiny of each diagnostic endoscopic technique is warranted.

Furthermore, **Wang et al. (30)** explored the correlation between clinical explorations, including modified Mallampati score and Müller's maneuver, with DISE findings regarding retrolingual obstruction. They reported a significant discrepancy between retrolingual airway collapse evaluated by modified Mallampati score and Müller's maneuver. In addition. The modified Mallampati score was more correlated with DISE regarding retrolingual obstruction compared to Müller's maneuver.

10-Friedman Tongue Position:

Friedman tongue position (FTP) is a grading system that grades the tongue position in relation to tonsil, uvula, soft and hard palate. It provides an indication of the degree of tongue base obstruction as well as the size of the tongue. The patient should be asked to avoid sticking their tongue out, and instead to have it inside the mouth in its natural and neutral position. The rationale behind this is that the resting tongue position during sleep is not held in a protruded position. The patient should be asked to open his or her mouth repeatedly for at least five times, without protruding their tongue, so that the most accurate level of tongue position can be assessed **(24).**The higher the Friedman tongue position grading (III, IV), the narrower the posterior airway space. FTP can be used in conjunction with findings from other evaluation methods to guide surgical management of tongue base obstruction **(31)**.

11-Assessement of lingual Tonsil Hypertrophy:

Lingual tonsil hypertrophy can cause significant hypopharyngeal collapse and increase the severity of OSA. Evaluation of lingual tonsil is performed, in multiple positions, with flexible nasoendoscopy either in the patient's awake state or during drug-induced sleep endoscopy **(24).**

Moreover, the upper airway assessment can be performed and enhanced by the Malampati score, the hyoid-mental distance and the hyoid-thyroid cartilage distance. If they are shortened, it implies a difficult airway **(32).**

Table (3): Friedman tongue position.

Friedman tongue position (FTP) stage	**Description**
FTP I	Entire tonsils/tonsillar pillar, uvula, soft palate and hard palate are visualized.
FTP IIa	Partial view of tonsils, but entire uvula, soft and hard palate are visualized.
FTP IIb	Tonsil and uvula are not seen. Only uvula base, soft palate, and hard palate are seen.
FTP III	Tonsils and uvula are not seen, but soft palate and hard palate are seen.
FTP IV	Only hard palate is visualized.

Table (4): Lingual tonsil grading.

Lingual tonsil grading	**Description**
Grade 0	Complete absence of lymphoid tissue on tongue base
Grade 1	Lymphoid tissue scattered over tongue base
Grade 2	Lymphoid tissue covering entire tongue base but with limited vertical thickness
Grade 3	Lymphoid tissue covering entire tongue base with a raised thickness of approximately 5–10 mm between 25–27% of epiglottis height
Grade 4	Lymphoid tissue 1 cm or more in thickness Vertical height rising above the tip of the epiglottis.

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