RECOGNITION OF OBSTRUCTIVE SLEEP APNEA SYNDROME

Michael J Thorpy, MD

LEARNING OBJECTIVE
After completing this activity, the learner should be able to—

- Recognize the diagnostic features of obstructive sleep apnea syndrome and understand the means of establishing the diagnosis.

INTRODUCTION
Obstructive sleep apnea syndrome (OSAS) is a disorder that is characterized by repetitive episodes of upper airway obstruction that occur during sleep. These episodes are typically either apneas, which are complete obstructions, or partial obstruction events called hypopneas.

The alteration in ventilation is associated with arousals or fragmented sleep.[1] These effects cause disturbance of sleep at night and are typically associated with excessive daytime sleepiness. In addition, there may be reduction in blood oxygen saturation and alteration in autonomic activity that lead to fluctuations in blood pressure and heart rate.[2]

When the respiratory disturbance during sleep at night is associated with excessive daytime sleepiness, the prevalence is 4% of the male population and 2% of the female population between the ages of 30 and 60 years.[3] However, OSA without symptoms occurs in approximately 24% of the adult male population and 9% of the adult female population.

DIAGNOSIS
The diagnosis of OSAS is established when there are symptoms of OSAS accompanied by objective evidence of significant respiratory disturbance during sleep at night. The symptoms can comprise of excessive sleepiness, unrefreshing sleep, insomnia, sleep interrupted by gasping and choking, observed loud snoring, or observed breathing interruptions during sleep.[1] When any of the above symptoms are associated with an apnea/hypopnea index (AHI) or respiratory disturbance index (RDI) of greater than 5,
then a diagnosis of OSAS is made. Alternatively, if the AHI or RDI is greater than 15, a diagnosis can be established in the absence of symptoms.

The AHI is the average number of apneas plus hypopneas per hour of sleep. An apnea is defined as cessation of air flow for at least 10 seconds and a hypopnea is defined as a 30% or greater drop in the respiratory excursion from baseline that lasts 10 seconds or longer and is associated with an oxyhemoglobin saturation of 3% or more from the pre-event baseline.[4] The AHI is a combination of both apneas and hypopneas per hour of sleep. The RDI is the number of apneas plus hypopneas plus respiratory effort related arousals. Respiratory effort related arousals are alterations in ventilation that cause an arousal and thereby disturbed sleep, even though they may not meet the criteria for apneas or hypopneas. It is generally regarded that severity of OSAS is demonstrated by an AHI or RDI of greater than 5 episodes an hour. Mild obstructive sleep apnea is 5 to 15 episodes per hour. Moderately severe obstructive sleep apnea is 16 to 30 and severe is greater than 30.[5]

Risk factors for OSAS include obesity, male gender, alcohol consumption, enlarged tonsils or adenoids, and family history of OSAS.

**PATHOPHYSIOLOGY**

The pathophysiology of OSAS is associated with an anatomical obstruction of the upper airway that occurs during sleep (Figure 1). There are many reasons why the upper airway may become obstructive. It may be due to a crowded oropharynx because of excessive soft tissues. It may be due to micrognathia, tonsillar or adenoidal hypertrophy, or an alteration in the size of the nasal airway with obstruction due to enlarged turbinates or polyps or small choanae. The upper airway obstruction is associated with increased collapsibility of the airway during sleep. There may also be impaired pharyngeal sensitivity that contributes to this upper airway obstruction, as well as reduction of lung volume that sets up an environment conducive to apneas in patients who have restrictive lung disease. The upper airway obstruction with collapse of the airway, the associated blood gas changes, and the loss of upper airway muscle tone during sleep create the environment for breathing instability and repetitive apneic episodes.
The flurry of events contributing to obstructive sleep apnea is sleep that causes a reduction in muscle tone, thereby precipitating the upper airway obstruction. This leads to hypoxia and possibly hypercapnia and acidosis. These blood gas changes can be associated with cardiovascular events such as arrhythmias, systemic hypertension, pulmonary hypertension, and even cor pulmonale. The blood gas disturbances contribute to an arousal that leads to fragmented sleep, leading to the excessive daytime sleepiness. When the arousal occurs, muscle tone is re-established. The patient may or may not awaken at this time, but the airway will open and the patient is able to take a few big breaths before returning back to sleep and a repeat cycle of events occurs.[6]

**Case Study:** John Morales presents to your practice complaining of fatigue and tiredness. He also is in to check his blood sugar, lipids, and blood pressure.

Medications: Lisinopril 20 g, HCTZ 25 mg, and pioglitazone 30 mg
Examination: BP 140/90, weight 315 lbs, height 5’7”. Obese, neck size: 17”, and pedal edema.
Family history of heart disease and diabetes.
Labs: FBS 90, LDL 205, triglycerides 240
Q1. What would you do next?

a. Advise him to lose weight  
b. Start a statin  
c. Have him complete an Epworth Sleepiness Scale  
d. All of the above  
e. None of the above

PRESENTATION
Patients with OSAS may present because of varied symptoms. Often patients will present because of excessive daytime sleepiness. However, for many patients, the major presenting complaint is more vague. Patients may complain of feeling tired all the time, having no energy, feeling fatigued, depressed, or not feeling rested. These symptoms need to be delved into to determine if they represent excessive sleepiness. If the patient has a tendency to fall asleep in front of the TV in the evening, or falls asleep easily as a passenger in a vehicle, or falls asleep in any sedentary situation during the daytime, then one has to consider the possibility that the patient has excessive sleepiness. This excessive sleepiness can be quantified by using the Epworth Sleepiness Scale (ESS).[7] The ESS consists of 8 questions about the chance of dozing in 8 everyday situations (from sitting and reading to in a car stopped in traffic).[7]

The ESS enables one to determine the score and, as there is a maximum of 3 per item, which is associated with a high chance of dozing, the total score for the questionnaire is 24. It is recognized that 10 or more on the ESS is associated with excessive sleepiness and 15 or more is associated with severe sleepiness. Typically patients with OSAS may have a mean score of around 16. This is very similar to narcolepsy, in which patients have a mean score of 17.5.[7]

The ESS is a very useful questionnaire that can be completed by the patient within a few minutes and a score obtained by the clinician in a few seconds. The ESS is a useful questionnaire for patients to complete in the waiting room and the clinician can use it to help in diagnosing excessive sleepiness, therefore recognizing a major symptom that contributes to OSAS or the clinician may use the form during follow-up evaluations to determine the patient’s progress after initiation of treatment.

The main symptom of OSAS is loud snoring that is often chronic and very irregular in quality. The snoring may be disturbing to the bed partner and the patient may not be aware of the snoring, as it occurs when the patient is asleep. In addition, there may be
nocturnal gasping or choking that occurs during the night that may be perceived by the patient, or, again, may only be noticed by a bed partner. Patients typically will have episodes of cessation of breathing, of which they are not aware, but these episodes may be noticed by the bed partner, who can become very concerned about the patient and concerned as to whether the patient will re-establish breathing or stop breathing completely during sleep. Patients themselves may recognize the excessive daytime tiredness and sleepiness, but in some cases, there may be some denial and, again, it may be a symptom that is most evidently recognized by a family member or associate of the patient.[8]

Other symptoms that commonly occur in patients with OSAS include morning headache or a sore or dry throat upon awakening in the morning. Some patients will be aware of having episodes of gastroesophageal reflux with heartburn or an acid taste in the mouth during the night. Sleep is typically restless, as the patient moves around a lot in association with the arousals. Patients may even fall out of bed during the night as a result of this restlessness. Nocturia and even enuresis commonly occur in patients with OSAS and this may resolve once the OSAS is treated. Because of the autonomic disturbance associated with the irregular breathing during sleep at night, there may be sympathetic changes associated with night sweats. The disturbed quality of nocturnal sleep and the daytime sleepiness contribute to the patient having impaired concentration, focus, coordination, and even memory problems. There may often be mood or behavioral changes, most typically irritability and mood fluctuations with anxiety or depressive features.

In the differential diagnosis of OSAS one has to consider simple snoring in the absence of apneic episodes, hypothyroidism which may cause tiredness and can be a risk factor for OSAS, obesity-hypoventilation syndrome (Pickwickian syndrome) which may have both obstructive and hypoventilation components, narcolepsy which can occur concomitantly with OSAS, periodic limb movement disorder and chronic insufficient sleep both of which may cause daytime sleepiness.

**CONSEQUENCES**
The clinical consequences of OSAS are associated with 2 major features (Figure 1): first, excessive daytime sleepiness; and second, cardiovascular complications. These consequences of sleep apnea cause an increased tendency for morbidity and mortality with obstructive sleep apnea.[6]
The cardiovascular consequences of OSAS can include cardiac arrhythmias, systemic and pulmonary hypertension, and cor pulmonale. The cardiac arrhythmias can occur during the autonomic disturbance that arises as a result of the breathing irregularity during sleep. There may be bradycardias during the apneic episode due to increased vagal tone and then during the hyperpnea or arousal stage; there are typically tachyarrhythmias. Atrial fibrillation is common in people with OSAS. In addition, OSAS has been associated with systemic hypertension and there is evidence that atherosclerosis and ischemic heart disease are also linked to OSAS. In severe cases of OSAS, pulmonary hypertension and cor pulmonale can occur. Because of the cardiovascular complications, particularly the cardiac arrhythmia, sudden death during sleep may occur.[9,10] As OSAS becomes more severe, there is an increasing risk of cardiovascular disease, not only coronary artery disease and congestive heart failure, but also cerebrovascular disease.[11] Hypertension has been shown to be more likely to occur in direct relationship to the severity of OSAS.[12] There is also a strong association with metabolic syndrome, diabetes, insulin resistance, and the presence of pro-inflammatory mediators and oxidative stress.[2,9]

In addition to the cardiovascular consequences of sleep apnea, there is also excessive sleepiness, which can lead to an increased likelihood of cognitive impairment[13] and this may result in motor vehicle accidents or even industrial accidents. The excessive daytime sleepiness causes impaired quality of life.[13,14] It affects relationship with the partner, professions and employment, and physical and psychological well-being.[15] There is an increased risk of psychiatric disorders, particularly depression, but also anxiety disorders and post-traumatic stress disorder.[16] Automobile accidents associated with injuries are more common in severe OSAS than suspected or mild sleep apnea syndrome.[17]

As a result of the consequences of sleep apnea syndrome, there is reduced survival, as has been shown by survival curves and this survival probability is improved with treatment of OSAS.[18]

There are certain individuals that are at greater risk for OSAS. OSAS is associated with increasing obesity, the male gender, increasing age, the use of alcohol, post-menopausal status, as well as those who smoke or have a family history of OSAS.[6]
Q2. John has an Epworth Sleepiness Scale of 15/24. What information would you want next?

a. Examine the upper airway
b. Give him a vitamin B12 injection
c. Advise him to see an endocrinologist
d. Increase his lisinopril

EXAMINATION
On examination, one should look at the upper airway to see if there is obstruction in the oropharynx. Micrognathia or retrognathia may be contributing factors to OSAS. Large neck size has been associated with sleep apnea and is more likely to occur in males with neck sizes greater than 17 inches and women with neck sizes greater than 16 inches.[21] A simple means of assessing the upper airway can be obtained by having the patient open the mouth and putting out the tongue. A scale called the Mallampati Scale can rank the severity of upper airway obstruction that contributes to OSAS (Figure 2).[19] In John, the Mallampati Scale was 3/4.

Figure 2.
On average, the odds of having obstructive sleep apnea increase more than 2-fold for every 1-point increase in Mallampati Scale.[19]

<table>
<thead>
<tr>
<th>Class I</th>
<th>Class II</th>
<th>Class III</th>
<th>Class IV</th>
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<tbody>
<tr>
<td>Full visibility of tonsils, uvula, and soft palate</td>
<td>Visibility of hard and soft palate, upper portion of tonsils and uvula</td>
<td>Soft and hard palate and base of the uvula are visible</td>
<td>Only hard palate visible</td>
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</tbody>
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Any patient who has symptoms suggestive of sleep apnea, particularly excessive sleepiness and snoring, should be suspected of having OSAS and particularly if it is associated with conditions that are comorbid with obstructive sleep apnea, such as hypertension and metabolic syndrome. The physical findings that are suggestive of sleep apnea consist of obesity or large neck and a small airway. In severe OSAS right heart failure can occur which can be evidenced by cardiopulmonary examination and pedal edema.

A simple screening tool may be useful to detect sleep apnea syndrome in patients.[20] The Stop-Bang questionnaire is a simple tool that assesses the primary features of OSAS (Figure 3). If patients have answered yes to 3 or more of the items, then they have a high likelihood of having OSAS and should be referred for diagnostic testing. Coupled with the ESS the Stop-Bang can enable both the main features of the disorder and the severity of the sleepiness to be determined in a particular patient.

![Figure 3. STOP - BANG Questionnaire](image)

Q3. John has a very narrow upper airway and admits to being a loud snorer. What would you do next?

a. Refer for a polysomnogram
b. Send him to a psychiatrist
c. Arrange a cardiac stress test
d. Send him to an otolaryngologist
e. All of the above
f. None of the above
INVESTIGATIONS
The diagnosis of OSAS is established by an all night polysomnography. OSAS is determined if the patient with sleepiness has an AHI of greater than 5. Patients with impaired circulation time or central nervous system lesions such as cerebrovascular disorders are at risk of having central sleep apnea which is characterized by repetitive episodes of cessation of breathing during sleep that is not due to upper airway obstruction. However, some patients with OSAS can have central apneic episodes in addition to obstructive episodes and these may respond to treatment of the upper airway obstruction.

Although there are a variety of forms of diagnostic testing, the full in-laboratory polysomnography with more than 7 channels of recording that measures not only EEG, EOG, chin EMG, ECG, airflow, respiratory effort, and also oxygen saturation, is the most accurate way of diagnosing sleep apnea. There are various forms of sleep testing that comprise fewer channels of recording and diagnostic accuracy falls as the number of channels measured decrease. There are high false negative rates reported with home testing, but home testing may be useful for patients who are unable to attend an in-laboratory polysomnographic evaluation. Another value of the in-laboratory testing is that patients can be videoed and any abnormal events that occur during the night can be documented. If portable monitoring and home testing is performed, it should be done under the auspices of an accredited comprehensive sleep medicine program of the American Academy of Sleep Medicine.[22] As it is beyond the scope of this article the reader is referred to the AASM guidelines for details of when to perform and how to interpret portable monitoring studies.[22]

Q4. The sleep study comes back with a result of an apnea/hypopnea index of 32 and a lowest oxygen saturation of 76%. What is your next step?

a. Send to an otolarygologist
b. Recommend positive airway pressure evaluation
c. Recommend weight loss
d. All of the above
e. None of the above

Treatment will be discussed in detail in a subsequent article but it should be noted that OSAS is best treated by positive airway pressure (PAP). However the patient needs to undergo a sleep study with titration of continuous PAP to determine if it is effective and whether the patient can tolerate the therapy. An all night study with PAP would be
advised. However, the patient should also be referred for an otolaryngological study of the upper airway to determine if there are any treatable upper airway obstructions. Weight loss recommendations are essential for all patients with obstructive sleep apnea.
References:


