Sleep Disordered Breathing

Naeem Ahmad Lughmani, MD, FCCP, FAASM

ABSTRACT

Among different categories of sleep disordered breathing, obstructive sleep apnea syndrome is the most common disorder. It causes great concern due to its deleterious effects on cardiovascular and neurocognitive systems. Obstructive sleep apnea is the result of repetitive pharyngeal collapse during sleep caused by abnormalities in the anatomy of the pharynx, the physiology of the upper airway dilator muscle activity, and the stability of ventilatory control. The disorder can be diagnosed with a combination of characteristic history (snoring, excessive daytime sleepiness), physical examination (crowded upper airway with redundant tissue), and overnight polysomnography.

Repetitive pharyngeal collapse causes recurrent arousals from sleep, resulting in the excessive daytime sleepiness, and increased risk of motor vehicle and job-related accidents. The presence of hypoxemia, hypercapnia, and sympathetic stimulation with catecholamine secretion associated with sleep apnea often lead to hypertension. The risk of myocardial infarction, stroke, and congestive heart failure may also be higher in patients with obstructive sleep apnea. Differential diagnoses include central sleep apnea, central alveolar hypoventilation, and upper airway resistance syndrome. Continuous positive airway pressure is considered the treatment of choice for obstructive sleep apnea; it reduces sleepiness and long-term cardiovascular consequences.

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The sleep apnea syndromes are seen often enough that all physicians, especially primary care providers, should be familiar with the signs and symptoms and how to refer these patients appropriately. Sleep apnea is a condition characterized by repetitive episodes of breathing pauses during sleep associated with sleep disruption with arousals and/or reduction in blood oxygen saturations (Figure 1).[1] Two major categories of sleep apnea have been documented: obstructive sleep apnea and central sleep apnea. Obstructive sleep apnea is a common sleep disorder caused by repetitive episodes of upper airway obstruction during sleep. Central sleep apnea is less common and is characterized by repetitive episodes of breathing pauses because of the absence of respiratory effort lasting for at least 10 seconds. This article will focus mainly on obstructive sleep apnea.

PREVALENCE

In population-based studies of sleep apnea, 30% of men and 20% of women

Dr Lughmani is Director, Saint Charles Mercy Hospital, Sleep Disorders Center, Oregon, Ohio.
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Correspondence to: Naeem Lughmani, MD, FCCP, FAASM, Toledo Clinic Inc, 4235 Secor Road, Toledo, OH 43623.

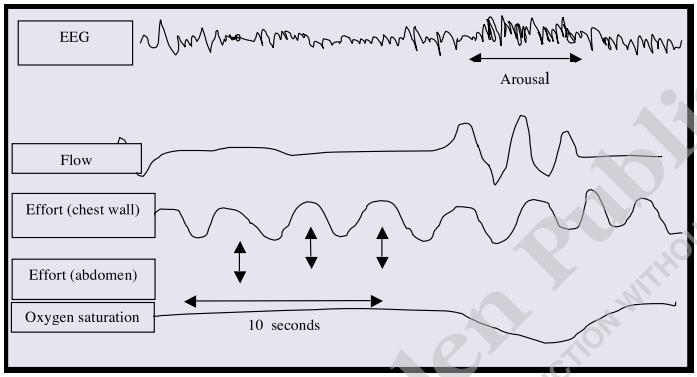
report excessive daytime sleepiness; 33% of men and 42% of women report difficulty falling asleep and maintaining sleep.[2,3] Obstructive sleep apnea is also common. Between 4% and 12% of adult men and 2% to 6% of adult women have sleep disordered breathing as defined by apnea-hypopnea index (AHI) greater than 5 per hour and excessive daytime sleepiness. The AHI is the number of apneic episodes and/or hypopneic episodes per hour. An apneic episode occurs when airflow ceases during sleep for at least 10 seconds; a hypopneic episode is a decrease in airflow lasting at least 10 seconds that is associated with a decrease in oxygen saturation and usually associated with arousal from sleep. In children, the overall prevalence of obstructive sleep apnea is 0.7% to 2%; in morbidly obese children, the prevalence is 13%.[2-4]

PATHOPHYSIOLOGY

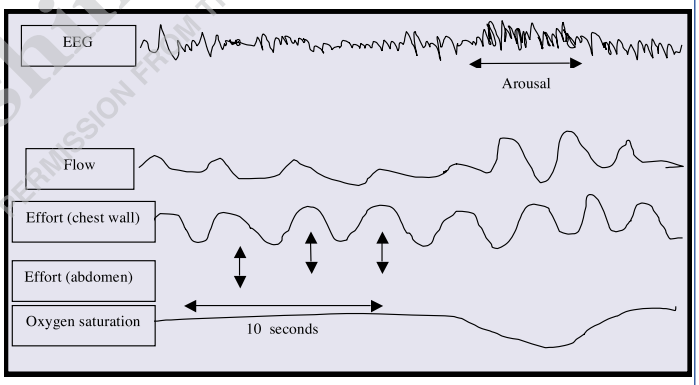
The pathophysiology of obstructive sleep apnea is complex. The most obvious pathogenetic factors include: narrowing of the upper airway from tonsils, adenoids, or both; adipose tissue due to obesity; or congenital skeletal abnormalities, such as micrognathia (abnormal smallness of the jaws, especially of the mandible). Other neural and hormonal factors also play an important role. Normally, the dilator pharyngeal muscles are activated with inspiration to increase upper airway size. New research is focusing on timing of this activation as a factor in sleep apnea patients.

Figure 2 shows a cycle of events involved in obstructive sleep apnea syndrome.[5] Structural factors (eg, genetically determined small posterior airway space, enlarged tongue, elongated soft

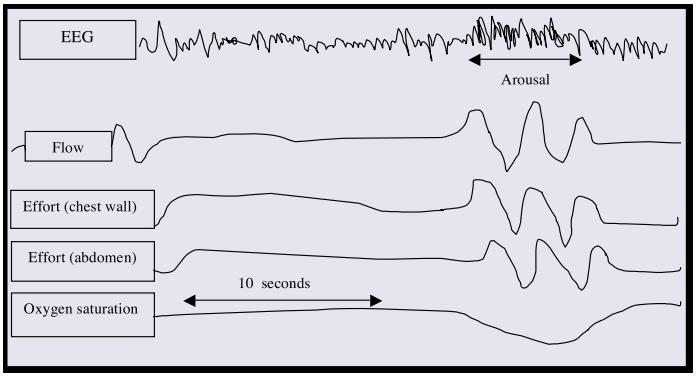
Figure 1. Different Manifestations of Sleep Disordered Breathing



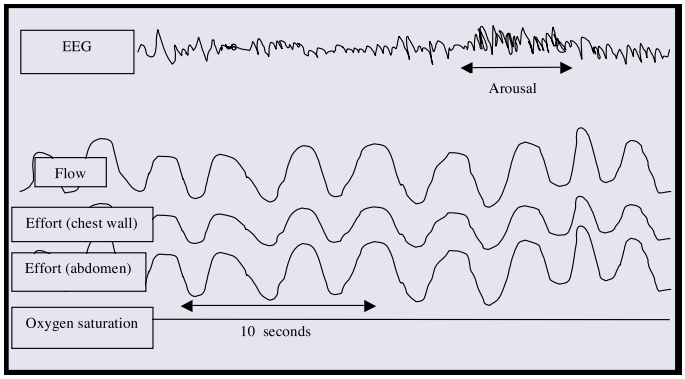
A. Obstructive Sleep Apnea. Increasing ventilatory effort is seen in the rib cage and the abdomen, despite absence of oronasal airflow. Arousal on the electroencephalographic (EEG) channel is associated with further increase in ventilatory effort. Oxyhemoglobin desaturation follows the termination of apnea. Note the paradoxical movement of chest and abdomen as the patient tries to breathe with closed airway (vertical arrows). Once the airway opens with an arousal, the breathing becomes synchronized.



B. Obstructive Hypopnea. The only difference from sleep apnea is the decrease rather than absence of oronasal flow because of less than complete collapse of upper airway.



C. Central Apnea. Note absence of airflow along with absence of chest wall and abdominal effort.



D. Upper Airway Resistance Event. Asynchronous movements of the rib cage and abdomen as well as oxyhemoglobin desaturations are not seen. Increasing ventilatory effort due to upper airway resistance results in arousal.

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palate, or neural factors, such as incoordinated pharyngeal muscle activity) can set the stage for the development of upper airway obstruction. Apnea and loud snoring ensue, leading to oxygen desaturation and stimulation of the autonomic nervous system, which contribute to the development of cardiac arrhythmias and pulmonary hypertension. Obesity contributes to apnea by narrowing the upper airway and adding a load to ventilatory activity, thereby increasing ventilation/perfusion mismatch in the lungs. The hypoxemia contributes to the production of an arousal that terminates the apnea. Sleep is thus disrupted, leading to excessive sleepiness, which increases the drive for sleep. Increased sleep in turn decreases pharyngeal muscle tone, thus increasing the tendency for upper airway collapse. Sleep disordered breathing seems to be worse in patients sleeping in the supine position and during the rapid eye movement (REM) stage of sleep.[6-8]

Physiologic Consequences

The physiologic consequences of sleep apnea are the direct result of asphyxia and sleep fragmentation, which result from repeated upper airway obstruction. Asphyxia causes hypoxia, hypercapnia, and acidosis, in addition to stimulation of the autonomic nervous system. These alterations in blood gases, along with autonomic stress, induce cardiac arrhythmias — typically bradycardia during the apneic phase because of increase in vagal tone, and tachyarrhythmias during hyperpneic phase at termination of apnea. Sinus arrest may also occur during the bradycardic phase, and atrial or ventricular tachycardia may occur during the hyperpneic phase. Systemic and pulmonary hypertension may also result.[9-15] The stress and increased drive to breathing due to airway collapse result in arousal from sleep, and breathing resumes. These arousals occur repeatedly, causing sleep fragmentation, which leads to excessive daytime sleepiness.[16]

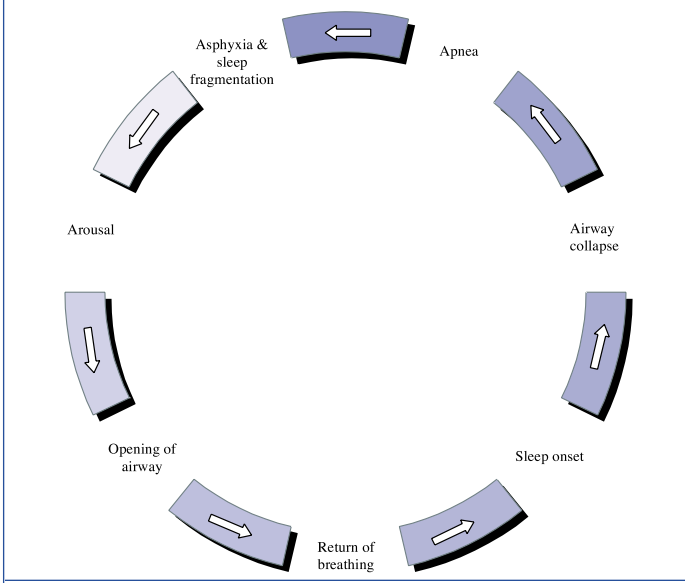
Hemodynamic consequences of obstructive sleep apnea include cyclic changes in systemic and pulmonary hypertension. Blood pressure increases during the apneic and early hyperpneic phase but reverts to normal until the next event.[17] A similar change is seen in cardiac output, which decreases during apnea and reverts to normal after the hyperpneic phase. Cor pulmonale from pulmonary hypertension and cardiomyopathy may also occur in the presence of hypoxemia during wakefulness. Until recently, the presence of cardiovascular (Table 1)[18] and cerebrovascular diseases in patients with obstructive sleep apnea was thought to be the result of shared risk factors, such as age, sex, and obesity. In the past 5 years, however, several epidemiological studies have demonstrated that obstructive sleep apnea is an independent risk factor for hypertension, probably resulting from a combination of intermittent hypoxia, hypercapnia, arousals, increased sympathetic tone, and altered baroreflex control during sleep.[9,11,12] Early recognition and treatment of sleep-related breathing disorders may improve cardiovascular function.[19-21]

About 33% of patients with obstructive sleep apnea have systemic hypertension, and 10% have pulmonary hypertension. Pulmonary hypertension tends to develop in patients who have daytime oxygen desaturation, are more obese, and have lower forced expiratory volume in 1 second and higher AHI. In a study published in 1998 by Worsnop et al, 38% of patients with treated and untreated hypertension had obstructive sleep apnea.[9] Of these patients, 44% also had increased pulmonary artery wedge pressure, thus suggesting left ventricular dysfunction. The remaining patients appeared to have primary pulmonary arterial hypertension.

Clinical Presentation

The most common symptoms of obstructive sleep apnea include loud snoring, excessive day-

Figure 2. Pathophysiology of Obstructive Sleep Apnea



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time sleepiness, observed apneas during sleep, gasping or choking awakenings, feeling tired in the morning, headache, and dry mouth in the morning (see Sidebar, page 341). The excessive daytime sleepiness results in a 7-fold higher risk of motor vehicle accidents.[22] In addition, it is associated with an increase in work-related accidents, impaired school[23] and work performance, impaired quality of life, marital problems, memory and concentration difficulties, and depression. In children, the symptoms may be different and include hyperactivity or excessive daytime sleepiness, noisy breathing, irregular body positions, ribcage retractions, and flaring of the ribs during sleep.

Age, weight gain, alcohol use, smoking, and use of sedatives increase the risk of obstructive sleep apnea. Obstructive sleep apnea is twice as likely to occur in men compared with women.[2] Other predisposing factors include anatomical abnormalities, such as retrognathia (the mandible is located posterior to its normal position in relation to the maxillae) or micrognathia (small mandible), and family history of the syndrome. Obstructive sleep apnea is associated with several disorders (Table 2).

Physical examination typically shows a large, short neck; obesity; enlarged uvula; long, soft palate; and sometimes — especially in pediatric patients — large tonsils and adenoids. In some patients, upper airway narrowing is caused by micrognathia or retrognathia. Elevated blood pressure may be present. Patients with severe obstructive sleep apnea may show evidence of right heart failure.

Table 1. Cardiovascular Consequences of Obstructive Sleep Apnea

- Bradytachycardia
- Sinus arrest
- Complete heart block
- Atrial and ventricular arrhythmias
- Systemic hypertension
- Cor pulmonale
- Pulmonary hypertension
- Myocardial infarction
- Sudden death

Data from Shahar et al.[18]

Table 2. Disorders Associated with Obstructive Sleep Apnea

- Hypothyroidism
- Acromegaly
- Marfan's syndrome
- Myotonic dystrophy
- Shy-Drager syndrome
- Amyloidosis

DIAGNOSIS

Snoring that is disruptive to a patient's life or accompanied by symptoms suggesting obstructive sleep apnea requires further evaluation. The most common test used to diagnose obstructive sleep apnea is multichannel overnight polysomnography. Practice parameters for the indications for polysomnography and related procedures have been published.[24] The variables measured in polysomnography include sleep staging (electroencephalographic leads), respiratory measures (nasal/oral airflow, chest and abdominal wall movements), electrooculography, chin electromyography, electrocardiography, oxygen saturation, limb-movement activity, position monitoring, snore recording, and video monitoring. Patients with sleep apnea typically show repeated apneas and hypopneas and sleep disruption with frequent arousals, causing sleep fragmentation. A decrease in deep and REM sleep usually occurs. Level 1 polysomnography is the test of choice for diagnosing obstructive sleep apnea. Polysomnography is also used to evaluate effectiveness of continuous positive airway pressure (CPAP) therapy; this procedure is described as CPAP titration study. Split-night study is a modification of this technique; the first 2 to 3 hours of sleep are spent without CPAP (diagnostic portion), and the remaining 4 to 5 hours are spent on titrating CPAP to find the effective pressures (treatment portion). In a selected group of patients, McArdle et al compared split-night study with standard studies and found equivalent long-term results but less healthcare utilization with the split-night study.[25]

Home studies with limited channels have been compared with polysomnograms performed in the laboratory.[26-29] Home studies have benefits in time and cost, but a large number of patients (56%) need further in-laboratory polysomnography for diagnostic reliability. Thus, home studies have a

limited role in diagnosing obstructive sleep apnea. These studies may be reasonable for those who are unable to go to a sleep laboratory because of illness, for those with severe clinical symptoms of obstructive sleep apnea when treatment is urgent and in-laboratory polysomnography is not available, and for those who need follow-up to evaluate response to treatment.[4,24] In 1999, Golpe et al showed a poor correlation between home oximetry and polysomnography.[27] The investigators also found that oximetry was more useful to confirm rather than to exclude diagnosis of obstructive sleep apnea. The greatest value of oximetry in this setting seems to be as a tool to rapidly recognize and treat patients with more severe obstructive sleep apnea who are on a waiting list for polysomnography. Chiner et al concluded, in a study in 1999, that nocturnal oximetry in patients with suspected sleep apnea/hypopnea syndrome and normal spirometric values permits the initiation of therapeutic measures in most patients.[28] In 1991, Cooper et al concluded that oximetry alone was sufficient for recognition of moderate or severe sleep apnea syndrome.[29] In routine practice, an appreciable number of equivocal results is likely, and repeated oximetry or more detailed polysomnography will then be required if clinical suspicion is high.

Disordered breathing during sleep as recognized by sleep study (polysomnography) consists of apnea, defined as a cessation or near cessation of respiration for a minimum of 10 seconds; hypopnea, defined as a reduction in airflow for a minimum of 10 seconds; and upper airway resistance events, which are episodes of increased respiratory effort due to partial upper airway obstruction. Disordered breathing during sleep is often associated with sleep fragmentation. The total number of apneas and hypopneas per hour of sleep is the AHI, also referred to as the respiratory disturbance index. The total number of arousals per hour of sleep from apneas, hypopneas, and periodic increases in respiratory effort is the respiratory-arousal index. In obstructive sleep apnea and the upper airway resistance syndrome, an increase in respiratory effort occurs as patients attempt to breathe against the obstruction of the upper airway. This increased effort can be identified by measuring an intrathoracic pressure that is more negative than is the pressure measured during unobstructed breathing. In central sleep apnea, no respiratory effort and, therefore, no airflow occurs for a minimum of 10 seconds. Central apneas rarely occur in isolation, and patients typically have both central and obstructive apneas. Distinctions may be difficult to make among obstructive, mixed, and central apneas or hypopneas. Therapy directed at treating sleep-related breathing disorders is usually effective regardless of the classification, except in rare cases when apneas are almost exclusively central.

The decision to perform a diagnostic sleep study should be based on patients' clinical presentation. Combined data from several studies suggest that excessive daytime sleepiness is a reliable predictor of the likelihood of the presence of obstructive sleep apnea syndrome and of the need for a diagnostic sleep study.[4] In a case series of 1000 patients, 84% of men and 60% of women referred to a sleep laboratory for excessive daytime sleepiness had obstructive sleep apnea.[30] Excessive daytime sleepiness can easily be measured in an office setting by documenting the responses to the Epworth Sleepiness Scale (ESS; Figure 3); a score of 11 or above is considered abnormal. Derived to measure excessive daytime sleepiness, the ESS has been validated in 4 studies by 1 investigator.[31-34] These studies provide credible evidence, including

Figure 3. The Epworth Sleepiness Scale

THE EPWORTH SLEEPINESS SCALE

Name: _____

Today's date: _____ Your age (years): _____

Your sex (male = M; female = F): _____

How likely are you to doze off or fall asleep in the following situations, in contrast to feeling just tired? This refers to your usual way of life in recent times. Even if you have not done some of these things recently, try to work out how they would have affected you. Use the following scale to choose the *most appropriate number* for each situation:

0 = would never doze
1 = slight chance of dozing
2 = moderate chance of dozing
3 = high chance of dozing

Situation	Chance of dozing
Sitting and reading	_____
Watching television	_____
Sitting, inactive in a public place (eg, a theater or a meeting)	_____
As a passenger in a car for an hour without 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 the traffic	_____

Thank you for your cooperation

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significant correlation with the multiple sleep latency test, that ESS can distinguish excessively sleepy patients from normal subjects and from those with insomnia. The ESS also documented improvement in excessive daytime sleepiness after treatment with CPAP.[32]

A history of snoring and breath holding during sleep is strongly associated with a subsequent diagnosis of obstructive sleep apnea; thus, these symptoms are reliable predictors of the need to refer a patient for sleep study.[4,35] Obesity alone is not sufficiently specific to indicate the need for sleep testing. Obesity is an indication for polysomnography only when it exists in combination with other sleep apnea symptoms.[4,24]

Differential diagnoses include central sleep apnea syndrome, central alveolar hypoventilation syndrome, and upper airway resistance syndrome (Table 3),[36-46] which are described later under **OTHER SLEEP-RELATED BREATHING DISORDERS**.

Table 3. Differential Diagnosis of Obstructive Sleep Apnea

	Obstructive Sleep Apnea	Central Sleep Apnea	Central Alveolar Hypoventilation Syndrome	Upper Airway Resistance Syndrome
Pathophysiology	Airway collapse	Instability in breathing control	Dysfunction of the respiratory center	Partial airway collapse
Symptoms				
Excessive daytime sleepiness	Present	Present	Present	Present
Snoring	Present	Not present	Not present	Present
Awakenings	Present	Present	Present	Present
Commonly associated conditions (see text)	Present (obesity, hypothyroidism, others)	Present (CHF, neurological disorder)	Present (obesity, pulmonary hypertension)	May be present (obesity)
Polysomnography				
Airflow	Compromised	Compromised	Compromised	Not compromised
Arousals	Multiple	Multiple	Possible	Multiple
Oxygen saturation	Decreased	Decreased	Decreased	Not decreased
Respiratory effort	Increased	Decreased	Decreased	Increased
Treatment	CPAP	Oxygen, acetazolamide, CPAP	NIPPV	Mandibular repositioning device, CPAP

CHF = congestive heart failure; CPAP = continuous positive airway pressure; NIPPV = noninvasive positive pressure ventilation.
Data from Exar et al[36]; Gold et al[37]; Epstein et al[38]; Jokic et al[39]; Consensus report[40]; Berger et al[41]; Hui et al[42]; Kessler et al[43]; Bonnet et al[44]; Javaheri et al[45]; Boudewyns et al.[46]

MANAGEMENT

Treatment for obstructive sleep apnea includes behavior modification along with medical interventions, surgical interventions, or both.

BEHAVIOR THERAPY

Behavior therapy for obstructive sleep apnea includes weight loss, sleeping on side position, avoiding sedatives and alcohol, avoiding sleep deprivation, elevating the head of the bed, treating nasal colds and allergies, avoiding large meals before bedtime, and abstinence from smoking.[6,7]

MEDICAL TREATMENT

Positive Airway Pressure. Medical treatment includes nasal CPAP,[47] which is the most effective form of treatment for obstructive sleep apnea. A pressure device applies preset pressures (usually through the nares), which work as an air splint to keep the upper airway patent. The amount of pressure required to overcome upper airway obstructive events and improve sleep quality is determined in the sleep laboratory by performing an overnight polysomnogram with CPAP titration.

Indications for therapy with CPAP include the following: an apnea index of at least 20 per hour or an AHI of at least 30 per hour, regardless of symptoms; an AHI of at least 10 per hour in a patient with excessive daytime sleepiness; and a respiratory arousal index of at least 10 per hour in a patient with excessive daytime sleepiness. Recent Medicare guidelines for treating obstructive sleep apnea suggest that patients with an AHI of 15 per hour and higher regardless of symptoms, and those with an AHI of 5 to 15 per hour with symptoms qualify for CPAP treatment.

Follow-up polysomnography is not routinely indicated in patients treated successfully with CPAP. In some circumstances, however, follow-up polysomnography is indicated. These include: after substantial weight loss to ascertain whether CPAP is still needed at the previously titrated pressure; after substantial weight gain in patients previously treated successfully with CPAP whose symptoms have returned despite continued therapy to ascertain whether pressure adjustments are needed; and when clinical response is insufficient or symptoms return despite a good initial response to treatment.

Patients' compliance with CPAP is between 60% and 85% based on subjective reports.[48,49] Some strategies to improve compliance include new, less restrictive masks, including nasal pillows and full oronasal masks; chin strap to decrease mouth leaks; heated humidifier to decrease nose and mouth dryness[50]; use of nasal decongestants; and bilevel positive airway pressure (BIPAP) if CPAP is not tolerable.

BIPAP works by delivering 2 different levels of positive air pressure: a higher level of pressure during inhalation and a lower level of pressure during exhalation, in contrast to the continuous delivery of positive pressure with CPAP. Automatic CPAP devices are on the horizon, which are designed to adjust pressure automatically during sleep in response to changes in airflow or ventilatory effort. If found to be consistently effective, the automatic devices may help to simplify CPAP therapy.[51-54] The effectiveness of automatic CPAP devices is yet to be established, particularly in patients with heart failure, lung disease, daytime hypoxemia, and those who may require higher pressures. Little data are available regarding the use of automated CPAP in patients with mild sleep apnea.[51] Randerath et al studied automated CPAP (APAP) in 2001 and concluded that APAP is as efficacious as constant CPAP in the treatment of obstructive sleep apnea syndrome.[52] Treatment pressure can be reduced significantly, and sleep microstructure improved with APAP. These may be the reasons for patient preference of automatic therapy. However, Farre et al tested 5 commercially available automatic CPAP devices and found the responses of the devices to apneas, hypopneas, flow limitation, and snoring were considerably different.[54] The response in some devices studied was modified by air leaks similar to the ones found in patients. The effectiveness of automatic CPAP assessed in clinical tests performed by using particular devices therefore has no general validity.

BIPAP is particularly helpful in patients who retain carbon dioxide (eg, patients with chronic obstructive pulmonary disease or central alveolar hypoventilation) and who cannot tolerate CPAP. This intolerance is usually the result of the high pressures required to splint the airways open in some patients. A population-based CPAP program consisting of consistent follow-up, troubleshooting, and regular feedback to both patients and physicians can achieve CPAP compliance rates of greater than 85% over 6 months.[49,55]

Oral Appliances. Oral appliances are receiving more attention because of recent advances that have resulted in improved efficacy.[56] They are indicated for primary snoring or mild obstructive sleep apnea syndrome that does not respond to treatment by behavioral measures. In patients with moderate-to-severe obstructive sleep apnea, oral appliances should be used only if the patient is intolerant of or refuses CPAP therapy and is not a candidate for upper airway surgery. Two categories of oral appliances are available: mandibular advancing and tongue retaining devices. Mandibular advancing devices are more popular and work by holding the mandible anteriorly in relation to the maxilla. Tongue retaining devices hold the tongue in an anterior position. Increased salivation, temporal-mandibular joint discomfort, and malocclusion are major side effects of these devices.

Pharmacologic Therapy. Pharmacologic therapy has a limited role and includes nasal steroids and antihistamines. Several medications with effects on respiratory stimulation or REM sleep suppression have been tested but have shown no beneficial effect.[57]

Surgical Treatment

Surgery is indicated to treat obstructive sleep apnea only in patients who have an underlying specific surgically correctable abnormality as the cause. Surgery may also be indicated to treat obstructive sleep apnea in patients for whom other noninvasive treatments were unsuccessful or have been rejected who desire surgery and are medically stable. In the absence of an obvious anatomic deformity, the optimal manner by which to predict a response to site-specific surgery is unclear. Universally accepted and validated clinical methods for defining the specific region of pharyngeal narrowing or collapse are lacking. Because of the complexity of airway narrowing or collapse during sleep, any one surgical procedure may not eradicate the patient's sleep apnea.

Common Symptoms Associated with Obstructive Sleep Apnea

- Loud snoring
- Excessive daytime sleepiness
- Observed apneas during sleep
- Gasping or choking awakenings
- Feeling tired in the morning
- Headache
- Dry mouth in the morning

Uvulopalatopharyngoplasty. Uvulopalatopharyngoplasty is the most popular surgical treatment for obstructive sleep apnea.[58] It was first introduced in 1981, followed by widespread use. It results in marked improvement in the AHI in 50% of unselected patients with obstructive sleep apnea. The success rate is less than 5% in patients with evidence of retrolingual narrowing or collapse. Velopharyngeal incompetence is one potential complication.

Turbinectomy. Turbinectomy and septal reconstruction alone is effective only in patients with severely impaired nasal breathing. It is, however, performed in combination with uvulopalatopharyngoplasty.

Tonsilo-adenoidectomy. Tonsilo-adenoidectomy is effective in children with obstructive sleep apnea if adenoidal and tonsillar hypertrophy is present.[18] Tonsillar hypertrophy is an uncommon cause of obstructive sleep apnea in adults, but tonsilo-adenoidectomy has been shown to be effective in carefully selected adult patients.[59,60]

Other Procedures. Laser-assisted uvulopalatoplasty is indicated for snoring and in patients with very mild obstructive sleep apnea. Other surgical procedures are performed infrequently and include genioglossal advancement, maxillomandibular advancement, tongue-base suspension, radiofrequency palatoplasty, hyoidplasty, and tracheostomy. Surgeries for weight loss, such as gastroplasty, have also been used to treat obstructive sleep apnea in morbidly obese patients.[61-66] Weight loss associated with this procedure results in significant improvement in AHI. Complications include failure to lose weight, intestinal obstruction, adhesions, and wound dehiscence. Gastroplasty should be used only in morbidly obese patients who have failed behavioral weight-reduction attempts and in whom conservative management has also failed.

Other Sleep-Related Breathing Disorders

Central Sleep Apnea

Central sleep apnea is characterized by repetitive cessation of ventilatory effort during sleep, usually with oxygen desaturation (Figure 1C). Central sleep apnea may be idiopathic, although it is most commonly associated with heart failure and neurologic and autonomic disorders. It can be managed with supplemental oxygen, acetazolamide, or CPAP.[44-47]

Central Alveolar Hypoventilation Syndrome

Central alveolar hypoventilation syndrome is characterized by ventilatory impairment, resulting in oxygen desaturation that is worsened by sleep, in the absence of abnormalities in the mechanical properties of the lungs. These patients are usually obese, with hypercarbia and hypoxia on arterial blood gases. Central alveolar hypoventilation syndrome is usually associated with diabetes mellitus, hypertension, and heart disease; pulmonary hypertension is also frequently present. Central alveolar hypoventilation syndrome can be treated with noninvasive positive pressure ventilation at night.[39-43]

Upper Airway Resistance Syndrome

Upper airway resistance syndrome is characterized by excessive daytime sleepiness with frequent arousals at night, resulting from partial collapse of the upper airway during sleep.[36-38] Unlike obstructive sleep apnea, no reduction in airflow or oxygen desaturation is present (Figure 1D). The gold standard to diagnose upper airway resistance syndrome is by polysomnography with an esophageal-pleural pressure monitor. It is a cumbersome procedure; therefore, most sleep laboratories do not use this technique but rely on a good history in conjunction with polysomnography showing repetitive arousals without apneas or hypopneas. Use of nasal cannula/pressure transducers also helps to increase the sensitivity of detecting flow limitation. In patients with a characteristic history and suspicious polysomnogram, a therapeutic trial of CPAP resulting in clinical improvements also confirms the diagnosis of upper airway resistance syndrome. This syndrome can be treated by behavior modifications as discussed earlier, in addition to definitive therapy, such as an oral appliance to reposition the mandible or tongue. CPAP is also beneficial and may be used in patients who have severe symptoms.

Summary

Sleep disordered breathing is a common problem with significant long-term morbidity that is underrecognized not only by the general public, but also by healthcare professionals. In addition to increasing morbidity, undiagnosed sleep disordered breathing affects quality of life. Primary care physicians are in a unique position to identify this serious health problem early and need to be aware of options available for diagnosing and treating these patients.

With every patient, asking a few simple questions regarding overall quality of sleep, presence of snoring, and unexplained sleepiness may help identify those with sleep disordered breathing. A formal sleep study with polysomnography is indicated in patients who have unexplained excessive daytime sleepiness or are snorers with additional symptoms, such as observed apneas and choking awakenings. Only in combination with other symptoms is obesity an indication for

polysomnography. An objective assessment of patients with excessive daytime sleepiness should be obtained routinely; the Epworth Sleepiness Scale is helpful in this regard. Home sleep studies may be reasonable for those who are unable to go to a sleep laboratory because of illness; those with severe clinical symptoms when treatment is urgent and in-laboratory polysomnography is not available; and those who need follow-up to evaluate response to treatment.

Recent Medicare guidelines for treating obstructive sleep apnea with CPAP therapy suggest that patients with an AHI of 15 per hour and higher regardless of symptoms and those with an AHI of 5-15 per hour with symptoms qualify for CPAP therapy. This mode of treatment is effective for eliminating apnea and hypopnea episodes in nearly all patients. A major long-term problem with CPAP therapy is compliance.

Other modes of treatment include surgery and oral appliances. Surgery is indicated in patients who have an underlying surgically correctable abnormality that is causing the sleep apnea, in patients for whom other noninvasive treatments have been unsuccessful or rejected, and in those who desire surgery and are medically stable. A 3-month follow-up sleep study is indicated after surgical treatment. Long-term follow-up is recommended, because there is insufficient evidence on whether immediate postoperative improvement is likely to be maintained; relapse has been reported. Oral appliances are indicated for primary snoring or mild obstructive sleep apnea syndrome that does not respond to treatment by behavioral measures. In patients with moderate-to-severe obstructive sleep apnea, oral appliances should be used only if the patient is intolerant of or refuses CPAP therapy and is not a candidate for upper airway surgery. Behavioral therapy is recommended for all patients and may be the only treatment necessary for patients with mild obstructive sleep apnea and upper airway resistance syndrome. However, CPAP therapy be considered even in these patients, as data suggests improvement in daytime symptoms after CPAP treatment.

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