Sleep and breathing disorders: the genesis of obstructive sleep apnea

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Sleep encompasses approximately a third of our lives; however, the physiology of sleep is not widely understood. Data suggest that sleep plays a restorative role in physiologic mechanisms and that long-term disruption of sleep may contribute to the development of disease. Nearly a third of the adult population is chronically afflicted by sleep disorders, and substantial economic loss is attributable to these disorders in terms of lost time, inefficiency, and accidents. Of the sleep disorders, obstructive sleep apnea (OSA) is one of the more common, clinically affecting up to 5% of the adult population. Obstructive sleep apnea contributes to the development of disease and has an adverse impact on daytime functioning in those affected by the disease. This article reviews basic sleep physiology, how these physiologic mechanisms are disrupted by OSA, and some of the techniques for treating patients with this disorder.

(Key words: sleep disorders, obstructive sleep apnea, daytime sleepiness, continuous positive airway pressure, circadian rhythm)

Sleep encompasses approximately a third of our lives; however, the physiologic processes active during sleep or sleep’s role in maintaining physiologic homeostasis is largely unknown. Until the early 1980s, the physiologic need for sleep had not been convincingly established. Now, data suggest that sleep plays a restorative role in physiologic mechanisms and long-term disruption of sleep may contribute to the development of disease. Data regarding the prevalence of sleep disorders suggest that nearly a third or more of the adult population is chronically affected by sleep disorders and a substantial loss in terms of time and accidents is related to these disorders.

Estimates from the United States suggest that 10% to 15% of the general population have frequent daytime sleepiness, while some select groups may approach 35%.

Sleep-related breathing disorders are one of the most common disorders that may affect sleep and cause excessive daytime sleepiness; obstructive sleep apnea (OSA) is the major disorder in this class. In the general population, these disorders are not trivial and they are often complicated by other disorders, medical conditions, or behavioral issues. Symptomatic OSA affects between 2% and 4% of women and 5% to 9% of men, depending on the criteria used.

Although there appears to be an “at-risk” population who is not symptomatic, up to 9% of women and 24% of men have the physiologic hallmarks. This may be especially important as these individuals may be at risk for other disorders.

Recent studies conducted through the National Institutes of Health have begun to define the relationships between OSA and cardiovascular disease. One of the major findings is that OSA contributes an independent risk for the development of cardiovascular disease after accounting for other known risk factors. Consequently, the identification of these disorders and their treatment may help to prevent morbidity and mortality. The prevalence of these disorders poses significant issues for the primary care physicians.

Sleep physiology
Basic sleep physiology, although rarely discussed in osteopathic medical schools, is essential in the understanding of OSA and related disorders. Sleep is classified in two major states: non–rapid-eye-movement (non-REM) sleep and REM sleep. Non-REM sleep comprises stages 1, 2, 3, and 4. Stages 3 and 4 comprise slow-wave sleep and are characterized as deep sleep. As one progresses from stage 1 to stage 4, sleep becomes deeper and the number of slow waves increases. These stages give way to the development of REM sleep, that stage of sleep in which the majority of dreams occur. During REM sleep, the stimuli that create dreams also cause signals to be generated down the motor pathways of the brainstem. Were it not for a secondary mechanism, these signals would initiate motor activity consistent with the dream content. The simultaneous activation of an inhibitory pathway causes muscle atonia in the majority of the skeletal muscles and prevents people from acting out their dreams. For individuals who rely on the skeletal muscles, and especially the accessory muscles, the muscle atonia compromises ventilation and may result in hypoventilation or apnea. The characteristics of each of these sleep stages are briefly outlined in Table 1.

Sleep architecture
The pattern of sleep stages that occurs during a night’s sleep constitutes the sleep architecture. Typically, an individual progresses from stage 1 to stage 2 to slow-wave sleep and then to REM sleep in a recurring pattern. Each cycle, from the lighter stages of sleep through the end of REM, typically takes 60 to 90 minutes. As the night progresses, each
cycle contains less slow-wave sleep and more REM sleep. The amount of each sleep stage and the amount of sleep required by an individual changes with age. Very young children require 14 to 16 hours of sleep, with such requirement declining to 8 to 10 hours for teenagers and young adults. Slow-wave sleep and REM sleep predominate. As individuals move into adulthood, their typical sleep requirement decreases into the range of 6 to 9 hours. Some individuals may require more sleep or less sleep, but they represent less than 5% of the population. Most commonly, individuals who are getting fewer than 6 hours of sleep each night are sleep deprived.

Circadian patterns
The timing of sleep is important in the overall assessment of sleep disorders. Physiologic rhythms cycle across the course of a single day. For most individuals, the duration of these rhythms, referred to as “circadian rhythms,” is about 26 hours. These internal rhythms must be reset each day to maintain consistency with the environment. This resetting occurs through a process of entraining mediated by three primary processes. The first is exposure to light on awakening. The light stimulates neural signals from the eye through the suprachiasmatic nucleus that helps to regulate our internal “clock” and biologic rhythms. The second mechanism is the pattern of daily activities. The stimulation arising from these activities and our interactions with other people reinforces the sleep-wake cycle. The final mechanism involves patterns of eating. Food is a very potent stimulus with regard to our sleep-wake

<table>
<thead>
<tr>
<th>Stage</th>
<th>Background EEG</th>
<th>EMG</th>
<th>EOG</th>
<th>Special characteristics</th>
</tr>
</thead>
<tbody>
<tr>
<td>■ Wake†</td>
<td>Mixed frequency with more than 50% of the epoch alpha waves</td>
<td>Relatively high tonic</td>
<td>Eye movements and blinks</td>
<td>May observe beta waves in EEG</td>
</tr>
<tr>
<td>■ Stage 1†</td>
<td>Low voltage, mixed frequency, less than 50% alpha waves, predominance of 2-Hz to 7-Hz activity</td>
<td>Tonic EMG less than wake</td>
<td>SREMs in early portion</td>
<td>Occasional vertex sharp waves in EEG; absence of spindles and K complexes</td>
</tr>
<tr>
<td>■ Stage 2</td>
<td>Low voltage, mixed frequency may have some slow-wave activity</td>
<td>Similar to stage 1 tonic EMG</td>
<td>Absence of REMs or SREMs</td>
<td>Intermittent K complexes† and/or sleep spindles‡</td>
</tr>
<tr>
<td>■ Stage 3</td>
<td>Slow-wave activity (&lt;2 Hz) of 75 µV amplitude in 20% to 50% of the epoch</td>
<td>Similar to stage 1 tonic EMG</td>
<td>Absence of REMs or SREMs</td>
<td>Sleep spindles and K complexes may or may not be present</td>
</tr>
<tr>
<td>■ Stage 4</td>
<td>Same as stage 3 but greater than 50% of the epoch consists of delta waves</td>
<td>Same as stage 3</td>
<td>Same as stage 3</td>
<td>Same as stage 3; clearly identifiable K complexes are rare</td>
</tr>
<tr>
<td>■ Stage REM†</td>
<td>Low voltage, mixed frequency,‡ 5-Hz to 7-Hz “sawtooth” waves frequently seen but not required</td>
<td>Low voltage, tonic EMG, lower than preceding stage‡</td>
<td>Episodic REMs (Phasic REM)‡</td>
<td>Absence of sleep spindles and K complexes; may see intermittent alpha wave activity</td>
</tr>
</tbody>
</table>

Key: EEG = electroencephalogram; EMG = electromyogram; EOG = electro-oculogram; REM = rapid eye movement; SREM = slow rolling eye movement.
*A scoring epoch is typically 30 seconds. If paper systems are used, the paper speed is 10 mm/s.
†For more details and exceptions see criteria in Rechtschaffen A, Kales A. A Manual of Standardized Terminology, Techniques and Scoring System for Sleep Stages of Human Subjects. Los Angeles, Calif: Brain Information Service/Brain Research Institute, University of California; 1968.
‡Characteristic that must be present.
### Table 2
**Physiologic Changes in Respiratory Control With Sleep**

<table>
<thead>
<tr>
<th>Variable</th>
<th>Stage 1 sleep</th>
<th>Stage 2 sleep</th>
<th>Slow-wave (stages 3 and 4) sleep</th>
<th>Rapid-eye-movement sleep</th>
</tr>
</thead>
<tbody>
<tr>
<td>Source of control</td>
<td>Metabolic</td>
<td>Metabolic</td>
<td>Metabolic</td>
<td>Nonmetabolic</td>
</tr>
<tr>
<td>Respiratory pattern</td>
<td>Periodic</td>
<td>Regular</td>
<td>Regular</td>
<td>Irregular</td>
</tr>
<tr>
<td>Central apneas/hypopneas</td>
<td>Often</td>
<td>Rare</td>
<td>Absent</td>
<td>Frequent</td>
</tr>
<tr>
<td>Response to metabolic stimuli</td>
<td>Variable</td>
<td>Mild decrease</td>
<td>Mild decrease</td>
<td>Moderate decrease</td>
</tr>
<tr>
<td>Chest wall movement</td>
<td>Phasic</td>
<td>Phasic</td>
<td>Phasic</td>
<td>Occasionally paradoxical</td>
</tr>
</tbody>
</table>

### Table 3
**Characteristics of Respiratory Events**

<table>
<thead>
<tr>
<th>Respiratory event</th>
<th>Duration</th>
<th>Airflow</th>
<th>Effort</th>
<th>Desaturation</th>
<th>Arousal</th>
</tr>
</thead>
<tbody>
<tr>
<td>Obstructive apnea</td>
<td>At least 10 s</td>
<td>Absent at some point in the event</td>
<td>Proportionately greater than flow; crescendo effort common</td>
<td>Not required</td>
<td>Not required</td>
</tr>
<tr>
<td>Central apnea</td>
<td>At least 10 s</td>
<td>Proportional to respiratory effort; absent at some point in the event</td>
<td>Absent or proportionally decreased with airflow</td>
<td>Common, but not required</td>
<td>Not required</td>
</tr>
<tr>
<td>Hypopnea</td>
<td>10 to 120 s, longer should be hypoventilation</td>
<td>Decreased by 50% relative to most recent baseline airflow</td>
<td>Proportionately greater than flow; crescendo effort common</td>
<td>Usually required if there is no arousal</td>
<td>Usually required if there is no desaturation</td>
</tr>
<tr>
<td>Respiratory event–related arousal*</td>
<td>At least 10 s; often several minutes</td>
<td>No significant change from baseline</td>
<td>Slight increase, may crescendo to end of event</td>
<td>Not required</td>
<td>Required; usually cyclic</td>
</tr>
<tr>
<td>Cheyne-Stokes respiration</td>
<td>Series may last 15 to 30 min or more</td>
<td>Varies proportionate with the respiratory effort; may include apnea at lowest point</td>
<td>Crescendo-decrescendo pattern</td>
<td>Usually mild cyclic desaturations, but not required</td>
<td>Not required</td>
</tr>
</tbody>
</table>

*Associated with upper airways resistance syndrome (UARS).*
mechanisms. These three mechanisms are often referred to as Zeitgebers, or “time givers.” These internal mechanisms affect sleep onset, the patterns of sleep, and the timing of REM sleep.

Sleep deprivation
Factors that limit sleep or fragment sleep functionally cause sleep deprivation. The major effect of sleep deprivation is to cause excessive sleepiness; however, studies by Rechtschaffen and colleagues have shown that sleep is required for maintenance of health. Individuals who are sleep deprived consistently show moodiness, decrements in memory, difficulty in concentration, and progressive increases in sleepiness. Such changes are often dependent on the type of sleep deprivation (total versus selective) and the amount of sleep deprivation. Some disorders such as OSA may result in selective REM deprivation. Over time, the tendency for REM to occur increases, which may result in an accentuated amount of REM sleep during the recovery phase; this effect is commonly referred to as “REM rebound.” A short sleep latency and increased sleep efficiency are characteristic of sleep deprivation.

Respiratory control
As an individual makes the transition from wake to sleep, the respiratory control relationships change (Table 2). With the onset of sleep, the central mechanisms controlling blood levels of carbon dioxide and oxygen allow functionally higher and lower levels, respectively. The theoretic reason for these changes is a shift to metabolic control of respiration and a change in the set points for both gases. The set point changes allow the carbon dioxide to rise by 2 torr to 3 torr and the oxygen saturation to fall by 2% to 3%. Rapid transitions from wake to sleep can cause sleep-onset central apneas. This form of central apnea should generally be considered a normal finding in the otherwise healthy adult.

Etiology and pathophysiology of obstructive sleep apnea
The pathophysiologic mechanisms that account for sleep-related breathing disorders result from physiologic changes that occur with the onset of sleep and may be made worse by a supine position. Etiologic mechanisms include neuro-muscular weakness (eg, amyotrophic lateral sclerosis), abnormal control of respiration (eg, congestive heart failure), partial or complete airway obstruction (eg, OSA), and disorders associated with airway disease (eg, asthma). The major disorder primarily associated with sleep is OSA. This disorder is characterized by recurrent narrowing or closure of the upper airway (Figure 1), leading to repeated apneas, hypopneas, or respiratory arousals that are often associated with desaturations and fragmentation of sleep.

The source of the problem relates to the structure of the oropharynx and functional interrelationships involving the pharyngeal muscles. The pharyngeal muscles comprise two functional groups: a pharyngeal dilator group and a pharyngeal constrictor group. At the initiation of each breath, the pharyngeal dilator mechanism is activated, thereby maintaining the patency of the pharynx throughout inspiration. In the majority of cases of OSA, the pharyngeal dilator mechanism is dysfunctional or there are physical impediments to airflow that intermittently obstruct airflow during sleep.

Structural abnormalities such as micrognathia, macroglossia, and large tonsils may also contribute to the development of sleep apnea. The increase in body fat that is common in OSA results in airway narrowing, which may further predispose to upper airway obstruction. In this regard, obesity should be considered a contributor to OSA, but not a common etiologic mechanism.

With sleep onset, the pharyngeal muscles relax, leading to an obstructive respiratory event (ie, apnea or hypopnea). Apneas may be categorized as obstructive apneas, mixed apneas, or central apneas (Table 3). Obstructive and central forms of hypopneas may also be seen. Both apneas and hypopneas must have a duration of at least 10 seconds (Table 3). Reductions in airflow longer than 120 seconds are typically characterized as hypoventilation. The exact amount of

![Figure 1. Major sites of airway closure in obstructive sleep apnea.](http://jaoa.org)

Foresman • Sleep and breathing disorders: the genesis of obstructive sleep apnea
the decrease in airflow necessary to identify the reduction varies; however, a minimum reduction of 30% to 50% is necessary in most circumstances because of technical limitations of the equipment used to measure airflow. In some sleep laboratories, the reduction in airflow must be coupled with a desaturation or arousal in order to score the event. The choice of criteria for scoring respiratory events varies widely, and no one definition has been accepted as a universal standard.4

The number of apneas that occurs per hour of sleep is referred to as the apnea index (AI). The number of apneas plus hypopneas that occurs per hour of sleep is referred to as the apnea-hypopnea index (AHI). In some instances, the respiratory-disturbance index (RDI) may be substituted for the AHI; however, the criteria for respiratory events has changed during the past 10 years,4 and proposed changes in the definition will likely alter the validity of such substitutions in the future. Typically, an AHI or an RDI greater than 5 is abnormal. In the past, some authors suggested that this number did not become clinically significant until the RDI was greater than 20. More recent data from the Sleep Heart Health Study,2 however, has provided other findings that support this contention by showing that an AHI of 5 is closely associated with the development of disease. These data also suggest that sleep apnea may progress from mild to severe disease over time (Figure 2). Therefore, symptomatic patients with an abnormal RDI should be treated.

There are several adverse cardiovascular consequences of obstructive respiratory events. Sympathetic increases occurring with these events and the reactive tachycardia often cause a transient rise in blood pressure. Over time, the increases in blood pressure become more persistent and develop into hypertension and other cardiovascular disorders. Although recurrent hypoxia is common in OSA, pulmonary hypertension is not. It is a relatively rare complication more commonly associated with chronic hypoxemia and hypoventilation.

Clinical features
The most common features of OSA are excessive daytime sleepiness, loud snoring, witnessed apneas, morning headaches, frequent nocturnal arousals, and weight gain. Usually, patients present with the history of increasing daytime sleepiness present for the past 2 to 5 years, increasing weight, and decreasing ability to perform typical activities. Patients or their significant other often report that sleep is quite restless and associated with frequent arousals related to snoring or snoring. They usually awaken unrefreshed and often take naps during the day or fall asleep spontaneously. The sleepiness associated with OSA can lead to accidents, interfere with the activities of daily living, impair work performance, and lead to general decline in satisfaction that is often perceived as depression. Additional symptoms or complaints may relate to declines in vision, poor memory, irritability, dry mouth, chronic fatigue, and impotence.

Frequently, individuals with OSA are moderately obese with a relatively narrow oropharynx and an increase in neck girth. Men are two times more likely than women to have OSA. These individuals may have structural deformities that contribute to the disease, such as macroglossia, micrognathia, or an enlarged uvula. Occasionally, nasal obstruction, nasal polyposis, structural defects of the nose, or allergies may also contribute to airway obstruction. Ventilation may also be impaired as the result of moderate obesity and its effect on breathing when supine. Further physical examination often reveals evidence of lower extremity edema and hypertension. Cardiovascular disease, diabetes, or hyperlipidemia is frequently noted in these patients. The family medical history frequently reveals that other family members have either OSA or a history of excessive sleepiness and snoring.

Typical laboratory findings
The definitive test for suspected sleep apnea usually involves polysomnography. Polysomnography is performed to verify the diagnosis of OSA and to rule out other disorders.5 These studies include physiologic measurements of eye movement, electroencephalographic recordings, oronasal airflow, pulse oximetry, electrocardiographic activity, chin muscle activity, and snoring. Other physiologic measurements may be included, depending on the diagnoses under consideration. Recently, a wide array of multichannel recording devices has been developed for use in sites outside of the sleep laboratory. The recordings of the majority of these devices are not sufficient to make a diagnosis of OSA. The use of these devices has been reviewed and clinical recommendations on their use published.6

Another testing procedure, the Multiple Sleep Latency Test (MSLT), has been developed to assess for sleepiness and narcolepsy.7 The MSLT is performed using methods similar to those for the overnight polysomnogram; however, multiple short naps are taken. The
naps typically are less than 20 minutes and are assessed for the time to sleep onset and the occurrence of any sleep-onset REM periods. In preparation for an MSLT, patients may be instructed to discontinue taking medications or alter their sleep period for several weeks. Also, an overnight polysomnogram is performed before the MSLT to rule out other disorders and to verify the amount of sleep immediately preceding the MSLT. The performance standards and indications for the MSLT have been reviewed elsewhere.8

Treatment and management

Once the diagnosis of OSA has been confirmed, an appropriate treatment regimen needs to be developed. Therapy should first be directed at the primary disorder, and then, consideration should be given to secondary or confounding disorders. Simply treating the patient with OSA without consideration of associated illnesses, behaviors, or circadian disturbances usually results in an inadequate treatment regimen, incomplete resolution of symptoms, and the patient’s noncompliance.

The most common treatment modality for OSA is positive airway pressure (PAP). This modality applies air pressure to the upper airway either through the nose or through the nose and mouth by use of a full-face mask. The air pressure in the upper airway displaces the airway walls outward, providing a pneumatic splint to the areas of obstruction. If effectively applied, this treatment modality will typically relieve the obstruction in patients with OSA. Two major patterns for delivering PAP are routinely used to treat OSA: continuous PAP (CPAP) and bi-level PAP. In both of these delivery patterns, the pressure delivered to the patient during exhalation must be sufficient to maintain airway patency and not allow complete collapse of the oropharynx. These two forms differ in one significant respect: the bi-level form increases its pressure during inspiration when the tendency to collapse the airway is the greatest. This form allows the use of lower pressures during end-exhalation and often increases comfort.

In more recent years, variations on CPAP have been attempted to improve tolerance, increase adherence, adjust to day-to-day variations in the severity of OSA, and provide for improved monitoring of CPAP use. More recently, machines that can automatically vary the applied pressure have been developed, so-called autotitrating CPAP. These machines are good for initiating CPAP but have not replaced the sleep laboratory in the optimal determination of CPAP pressures.

In general, CPAP is a less-expensive modality than bi-level PAP. Bi-level PAP is more expensive because of an additional mechanism necessary to enable the bi-level delivery process. The more sophisticated versions of CPAP machines are slightly to moderately more expensive than standard machines, but significantly less than bi-level machines. Overall, each of these devices has its own advantages, and no one device represents the universal alternative for all situations.

The choice of masks used to apply CPAP or bi-level PAP is important in the appropriate care of the patient with OSA. The masks are of three major formats: the nasal mask, the full-face mask, and nasal prongs or pillows. Each of these formats has its own advantages and disadvantages. The mask should be chosen to optimize tolerance and to minimize complications.

To adequately treat an individual with OSA, adequate pressure settings must be used. Most centers will attempt to determine an adequate pressure setting using a titration trial. Titration studies are frequently performed on a night after the study diagnostic for OSA. Some centers perform the diagnostic phase and the titration phase during the same study when they have appropriate patients. This type of study is referred to as a split-night study. Usually, this study requires that a patient have a minimum of 30 respiratory events or apneas within the first 2 to 3 hours of the study, which allows sufficient time to perform the titration phase of the study. Overall goal of the properly performed titration study is to optimize sleep while minimizing the side effects and complicating factors involved in the administration of CPAP.

Tracheostomies have been shown to be an effective therapeutic intervention for OSA. Studies performed after the introduction of nasal CPAP demonstrated an improvement in mortality with both CPAP and tracheostomy. Today, a tracheostomy may be an appropriate intervention for those individuals whose OSA cannot be well controlled with CPAP or bi-level PAP, or those who did not tolerate PAP interventions. Surgical interventions such as uvulopalatopharyngioplasty (UPPP), hyoid advancement, and mandibular advancement are potential alternatives.10 The reduction in respiratory events associated with these interventions, either alone or in combination, is significantly less than that associated with CPAP; however, most series show 40% to 50% of patients have reduced the number of respiratory events by half. To date, no reliable test exists to determine which patients will benefit from UPPP or other surgical interventions. Also, individuals treated with surgery have a tendency to have recurrence of OSA 3 to 5 years after the surgery has been completed. Laser uvulopalatopharyngioplasty (LAUP) has been evaluated; however, it appears to be an ineffective modality for treating OSA. Oropharyngeal appliances are best used with individuals who have mild OSA or in situations in which patients do not have access to their CPAP for short periods.11 The choice of these alternative modes of therapy requires knowledge of the patients’ condition, the severity of their sleep apnea, the tolerance to previously attempted therapeutic interventions, and the patients’ preference. No one modality works for all patients, and the failure of a modality such as CPAP should not preclude its future use.

Weight loss is rarely a cure for OSA, but it frequently reduces the severity of
the disease and may reduce the CPAP needed for effective control of respiratory disturbances. Some patients will have clear worsening of their respiratory events when in the supine position. In these instances, positional retraining rather than an increased CPAP may be an effective supplement. Supplemental oxygen may be necessary to control desaturations in some patients.

The treatment of young children with OSA may vary somewhat. For this reason, young children and neonates should be studied only in selected centers. Also, surgical interventions may be more common and more effective in children than in adult patients. Children of an appropriate size and stature may also be well treated with CPAP.

Patient education, health promotion, quality of life, and public policy
Once a diagnosis of OSA is made, then patient education is necessary to avoid complications and optimize compliance with physician recommendations (Figure 3). Patients with untreated or inadequately treated OSA have an increased risk of accidents. The laws involving OSA vary from state to state and may be dependent on the occupation of the afflicted patient. In some circumstances, reports may need to be forwarded to the appropriate administrative body such as the Federal Aviation Authority or Department of Transportation. Once under adequate treatment, most professional pilots and drivers will require yearly updates in order to maintain their operational status. Regardless of occupation, every patient should be cautioned with regard to the risk of accidents, and follow-up should be tailored accordingly.

Health and public policy
Important issues with regard to public policy and OSA include the development of cardiovascular disease and the prevention of accidents. Recent data now suggest that OSA is not only a cause of hypertension, but it is also an independent risk factor for the development of cardiovascular disease. The relative risk for the development of cardiovascular disease is approximately 1.2 to 1.5 and is likely to increase with advancing age.

To date, little is known about the effects of treating OSA with regard to preventing the development or progression of cardiovascular disease. Despite this paucity of knowledge, it is likely that effective interventions will have a significant impact. With regard to accidents, it has been clearly shown that effective treatment of OSA reduces the risk of accidents. Recent efforts by the National Institutes of Health and several agencies, including the American Academy of Sleep Medicine, have begun to address these issues.

Comment
In summary, the key points of this article are as follows:
- Sleep-related breathing disorders are common in the general population. Approximately 2% to 5% of the population are symptomatic and meet criteria for these disorders.
- Patients with sleep-related breathing disorders commonly present with excessive daytime sleepiness.
- Patients with cardiovascular disorders have a greater likelihood of having OSA than the general population, and OSA may worsen their cardiovascular disease.
- Diagnosis of sleep-related breathing disorders depends on some simple questioning of the patient and ordering the appropriate diagnostic studies (eg, polysomnography).
- Therapy for OSA usually incorporates CPAP, which is effective in most individuals, but may include surgery, weight loss, and other modalities.
- Treatment should address behaviors related to sleep (eg, smoking, drinking) and the patterns of sleep (eg, shift work, limited sleep).

References