Obstructive sleep apnea syndrome, part 1: Identifying the problem

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Abstract: Obstructive sleep apnea-hypopnea syndrome (OSAHS) is a common, yet often overlooked, form of symptomatic sleep-disordered breathing. OSAHS is a cause for concern for several reasons, one of which is its association with cardiovascular disease. Risk factors include obesity, hypertension, and upper airway malformations. Diagnostic clues include habitual snoring, witnessed apneas, choking arousals, excessive daytime sleepiness, and large neck circumference. Polysomnography is the definitive diagnostic test; it provides objective documentation of apnea and hypopnea. Since OSAHS may contribute to adverse postsurgical events, consideration of this syndrome should be part of the preoperative assessment of patients. (J Respir Dis. 2006;27(4):144-152)

Sleep-disordered breathing refers to a group of clinical syndromes with the common characteristic of altered ventilation during sleep.1 There is overlap between central and obstructive disordered breathing syndromes. For example, mixed apneas and conversion of obstructive apnea to central apnea with application of positive airway pressure suggest a shared pathophysiologic basis.2

Obstructive sleep apnea-hypopnea syndrome (OSAHS) is a form of sleep-disordered breathing that has emerged as a major health issue. The neurobehavioral and cardiovascular consequences of OSAHS underscore the importance of prompt recognition. In this article, the first of 2 parts, we will provide some background information about OSAHS. We will then offer a practical plan for the clinical evaluation. In the second part of this article, to be published in a future issue of The Journal of Respiratory Diseases, we will discuss our approach to managing OSAHS.

OVERVIEW

Normally, upper airway diameter, tonic pharyngeal muscular activity, and compensatory reflex dilator mechanisms are attenuated during the transition from wakefulness to sleep.3 The impact of these changes can be increased in the presence of certain upper airway abnormalities and clinical risk factors, such as obesity, nasal congestion, smoking, sleep deprivation, and sleeping in a supine position.4,5 The critical constriction occurs behind the uvula and soft palate or the tongue (Figure). Repeated attempts to breathe against the resistance caused by an occluded pharynx lead to oxyhemoglobin desaturation, hypercapnia, and pronounced swings in intrathoracic pressure. Snoring is a repetitive sound caused by the vibration of restricted upper airway structures during sleep. A respiratory effort-related arousal (RERA) is a sequence of breaths occurring over a span of at least 10 seconds, in which increasingly vigorous respiratory efforts--made against a narrowed upper airway--end with an arousal before the onset of apnea-hypopnea.1 Upper airway resistance syndrome (UARS) is characterized by the occurrence of 10 or more RERAs per hour and the presence of excessive daytime sleepiness, and it is considered to be part of the OSAHS spectrum (Table 1).1,6 Apnea is defined as airflow cessation that lasts for 10 seconds or longer. The definition of hypopnea is more variable. Events are considered obstructive when respiratory efforts are constant or increasing, and they are considered central when respiratory efforts are absent. Mixed apneas may begin as central events and terminate with crescendo efforts against an obstructed airway. Hypopneas are temporary reductions in airflow of at least 10 seconds’ duration. An obstructive etiology is inferred from concurrent dyssynchronous movements of the chest and abdomen, flattening contour of the airflow signal, or crescendo snoring.7 The definition of obstructive hypopnea has varied over time. The Clinical Practice Review Committee of the American Academy of Sleep Medicine (AASM) and the Centers for Medicare & Medicaid Services (CMS) define hypopnea as airflow reduction of at least 30% that lasts for 10 seconds or longer and results in at least 4% oxygen desaturation.8 Some laboratories may also mandate an associated arousal.

It is important to understand how your affiliated sleep facility defines hypopnea, since even a subtle change in the criteria--requiring just a 2% oxygen desaturation, for example--can significantly
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Influence detection rates. The distinction between RERAs and hypopneas may sometimes be simply a matter of the degree of associated oxygen desaturation. The number of apneas and hypopneas per hour of sleep, as recorded by polysomnography, is the apnea-hypopnea index (AHI). From the perspective of the CMS, persons with OSAHS have an AHI of at least 15 or greater than 5 if hypertension, ischemic heart disease, stroke, excessive daytime sleepiness, impaired cognition, mood disorder, or insomnia is present. Although the AHI is an oversimplified criterion for defining OSAHS, no better alternative exists. Epidemiology OSAHS is common, affecting about 5% of adults. As many as 20% of adults may have an AHI greater than 5 but without any symptoms. The prevalence may level after age 65. About twice as many men as women have OSAHS, although obesity and menopause tend to reduce this difference. OSAHS often escapes detection. Earlier estimates were that 80% to 90% of patients with OSAHS had not received the diagnosis. Effects of OSAHS The physical effects of OSAHS—marked intrathoracic pressure changes, oxyhemoglobin desaturation, hypercapnia, and sleep arousal—have significant consequences. Neuropsychological dysfunction, for example, occurs even in persons who have mild OSAHS, although there is significant individual variability. Persons who have OSAHS are at increased risk for motor vehicle accidents, with an odds ratio of 2.5. OSAHS also contributes to cardiovascular disease. Large cross-sectional and longitudinal studies show a dose-dependent link between a high AHI and hypertension, probably because of chronically heightened sympathetic nervous system activity. OSAHS may also be associated with congestive heart failure and stroke. In the Sleep Heart Health Study, a cross-sectional assessment of more than 6000 outpatients, those persons with the highest AHI had an adjusted odds ratio of self-reported cardiovascular disease of 1.42 (95% confidence interval, 1.13 to 1.78), with the strongest links to heart failure and stroke.

Results of a recent cohort observational study revealed an association between OSAHS and stroke or death with an adjusted hazard ratio of 1.97 (95% confidence interval, 1.12 to 3.48). Those with the highest AHI at baseline incurred the highest risk for the composite end point of stroke or death. In addition, bradyarrhythmias, tachyarrhythmias, and atrial fibrillation are associated with OSAHS, and atrial fibrillation is twice as likely to recur after cardioversion if OSAHS remains untreated. OSAHS also is linked to impaired glucose tolerance independent of obesity. Other possible consequences of OSAHS include erectile dysfunction, nocturia, gastroesophageal reflux, and morning headaches. Although these are nonspecific features, they may be clues to the diagnosis of OSAHS when they respond only partially to standard treatment. Diagnosis The diagnosis of OSAHS is complicated by the high incidence of its principal features. In the United States, about 45% of men and 25% of women are habitual snorers, two thirds of adults are overweight, and more than 30% of the population report excessive daytime sleepiness; yet only 1 in 20 middle-aged adults has OSAHS. Whether the symptoms of OSAHS are significantly different in men than in women is debatable. Clinical evaluation Sometimes the patient—either independently or at a bed partner's urging—comes to you directly seeking help for OSAHS. Other times, you might suspect OSAHS during a routine examination or while evaluating hypertension or other cardiovascular problems. When taking the history, ask about the quality of sleep. Is the patient waking up often at night? Has he or she noticed any breathing disturbances that occur during sleep? Is he or she snoring intensely? Persons who do not snore are unlikely to have OSAHS. Also ask whether the patient experiences choking during the night. (You might need to ask the bed partner about this.) Excessive daytime sleepiness, especially while driving, is another strong clue to OSAHS. However, such drowsiness may have other causes—most notably, insufficient sleep. Is the patient getting enough sleep each night? Is he or she working prolonged or rotating work shifts that may make it more difficult to get adequate sleep? Are there indications of other sleep disorders, such as restless legs syndrome or cataplexy? During the physical examination, note obvious factors that could contribute to OSAHS, such as hypertension and obesity (Table 2). Look for head and neck conditions that may compromise upper airway patency. These include nasal obstruction (septal deviation, polyps), a low-draping soft palate, an elongated uvula, a prominent tongue base, tonsillar hypertrophy, and retrognathia. Does the patient have redundant neck tissue? Neck circumference over 17 inches in men or over 16 inches in women is highly correlated with OSAHS. Routine evaluation for pulmonary arterial hypertension is not recommended. Significant pulmonary arterial hypertension in a person with OSAHS usually indicates concurrent risk factors, including
left-sided heart disease, underlying lung disease, or nocturnal oxygen desaturation. Obese persons can also have pulmonary hypertension. Hypercapnia is uncommon in persons who have uncomplicated OSAHS. However, consider checking arterial blood gases if there is concurrent pulmonary disease, neuromuscular or chest wall disorder, or medically complicated obesity.

Thyroid function testing is generally unnecessary unless the patient's clinical features suggest a thyroid disorder. Consider a thyroid evaluation when there are no obvious findings to account for symptoms of OSAHS.

Presurgical patients

Untreated OSAHS in patients who are scheduled for surgery may increase complications and length of hospital stay. Therefore, a high index of suspicion for OSAHS should be maintained when assessing patients preparing for surgery. If you suspect significant OSAHS and clinical circumstances are allowable, postpone surgery until a sleep evaluation has been completed. If surgery cannot be postponed, sedatives or narcotics and analgesics or anxiolytics need to be used very cautiously during surgery. Patients need appropriate cardiorespiratory monitoring and may be candidates for empiric treatment with continuous positive airway pressure (CPAP) to ensure adequate oxygenation.

Oximetry

Clinical impression alone lacks accuracy. Overnight oximetry (which measures the oxygen saturation in arterial blood, pulse rate, respiration rate, tidal volume, cardiac output, and blood pressure) is most helpful in the assessment of patients in whom clinical suspicion of OSAHS is low. The reported capabilities of oximetry vary widely because of differences in criteria, populations, and oximeter types. With proper data collection, frequency, and analysis, the sensitivity and negative predictive value of overnight oximetry exceed 90%, while specificity averages 50%, for diagnosing OSAHS. A normal overnight oximetry study excludes significant OSAHS, but not UARS (since RERAs do not cause oxyhemoglobin desaturations), from the diagnosis. It also does not rule out other causes of excessive daytime sleepiness.

Oximetry reports must be visually inspected and interpreted by clinicians who know how to differentiate artifact from the "saw-toothed" (cyclical) oscillations in oxyhemoglobin saturation caused by apneas and hypopneas. Patient selection may influence the performance characteristics of oximetry. The oxygen desaturation caused by apneas and hypopneas will probably be more pronounced in patients who have underlying cardiopulmonary disease or obesity. Thus, oximetry is expected to be more sensitive in these patients than in healthy, thin patients.

Polysomnography

The current definitive test for OSAHS is polysomnography, which evaluates sleep and cardiorespiratory function via electroencephalography, electro-oculography, chin electromyography, airflow, thoracoabdominal movement, oxygen saturation, and electrocardiography. Polysomnography is indicated for persons who have intermediate to high probability of having OSAHS. In some circumstances, primary care physicians may refer their patients directly for polysomnography.

A preliminary assessment by a sleep medicine specialist is recommended for patients with the following characteristics:

- The presence of significant cardiopulmonary disease.
- The presence of medically complicated obesity or neuromuscular or chest wall disorders.
- Other sleep disorders: insomnia, narcolepsy, restless legs syndrome.
- Special needs: neurodegenerative conditions, employment in commercial transportation, strong initial aversion to CPAP.

The traditional approach to polysomnography calls for a full-night diagnostic procedure, followed by a full-night titration of therapy (usually CPAP). The expense and inconvenience of this approach, however, has spurred a quest for alternatives. Split-night polysomnography, which combines an initial diagnostic segment with a latter-half CPAP titration in the same night, is an acceptable alternative, according to the AASM, if the following criteria are met: an AHI of at least 40 is documented during a minimum of 2 hours of diagnostic polysomnography (or an AHI of 20 to 40, based on clinical judgment); CPAP titration is carried out for more than 3 hours; and polysomnography documents that CPAP eliminates apneas and hypopneas during rapid eye movement (REM) and non-REM sleep, including during REM sleep in the supine position.

If you strongly suspect OSAHS and the results of the first polysomnogram are normal, consider arranging for a second test. About 10% of patients with an AHI of less than 5 during the first night have an AHI of at least 5 during a second polysomnogram.

Polysomnography systems are now available that try to improve convenience and comfort by recording a limited number of cardiorespiratory parameters. Some of these devices can be used at home without the assistance of a technologist.
The proper role for portable polysomnography monitors, however, remains a matter of significant debate. A comprehensive review conducted jointly by the AASM, American College of Chest Physicians, and American Thoracic Society concluded that there was insufficient evidence to support the use of limited, portable monitoring devices in an unattended setting (performed in the home, with no technologist present) for confirming or ruling out OSAHS. However, monitoring in an attended setting (performed in the sleep laboratory, with a technologist present) may be a whole-night diagnostic option, provided that the data are manually reviewed by qualified personnel, are applied to patients who do not have significant comorbid conditions, and are not used for titration of CPAP.

The CMS mandates that for CPAP reimbursement purposes, the diagnosis of OSAHS must be established by a facility-based polysomnogram, not one obtained in the home or in a mobile facility. Sleep medicine specialists should manually review the polysomnographic data and generate the report (see “Interpreting the polysomnography report”). The arousal index, oxyhemoglobin saturation data, and sleep architecture complement the AHI in revealing the physiologic impact of apnea and hypopnea on the patient.

References: REFERENCES

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