Sleep Apnea—Diagnosis and Treatment

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"Topics in Primary Care Medicine" presents articles on common diagnostic or therapeutic problems encountered in primary care practice. Physicians interested in contributing to the series are encouraged to contact the series' editors.

The syndrome of sleep apnea has been described throughout the ages, dating at least to descriptions of Dionysius the Tyrant in the fourth century BC. It is only recently that scientific investigation has led to an understanding of this previously ill-defined syndrome.

Sleep apnea is the currently accepted term for a group of heterogeneous sleep-associated respiratory disorders. The most common of these is obstructive apnea, which is defined as a cessation of airflow during continuous respiratory effort. Obstructive apnea occurs when oropharyngeal pressures become so negative as to exceed the ability of upper airway muscles to maintain patency of the oropharynx. A less common sleep-associated respiratory disorder is central apnea, which is usually defined as the cessation of airflow for at least ten seconds, while no respiratory efforts are made. Patients with central apneas probably suffer from reductions in the chemical control of breathing. Mixed apneas also occur, during which airflow ceases for at least ten seconds, associated with periods of no respiratory effort and respiratory efforts against an occluded upper airway. Finally, primary alveolar hyperventilation (PAH) leads to hypoxia and hypercapnia not associated with apneas. This disorder may be present while awake, but is usually much worse during sleep.

It has become increasingly apparent that a large portion of the adult population, especially the elderly, has mildly abnormal breathing during sleep. This may include simple snoring, a symptom often associated with polysonomographic evidence of partial upper airway obstruction, and brief, recurrent central apneas that may lead to complaints of insomnia. These patterns in their mildest forms do not cause significant hypoxemia, and therefore the need for aggressive therapy is uncertain. As further information regarding the natural history of sleep-associated respiratory disorders becomes available, we may find that such mildly abnormal breathing patterns represent different stages in the development of the sleep apnea syndrome.

These disorders are commonly associated with complaints varying from socially unacceptable snoring to nocturnal insomnia or daytime hypersomnia. More serious associated conditions may include right and left heart failure, erythrocytosis, cardiac arrhythmias (including sudden death) and even respiratory failure. It is therefore crucial to make the proper diagnosis and the appropriate therapeutic intervention.

Diagnostic Techniques

As in any diagnostic evaluation, a thorough history and physical examination are essential. Tables 1 and 2 list clinical symptoms and features common to patients suffering from sleep-associated respiratory disorders. Recognition of these characteristics should direct a clinician to more definitive testing. The affected patients usually suffer from excessive daytime sleepiness, although they may minimize this symptom. There is frequently some altered sleep behavior, most typically loud snoring, restlessness or insomnia, although enuresis and even somnambulation have been described. Progressive intellectual deterioration and personality changes are often seen. The patient is typically an obese man, although significant disease may occur in slender patients. If a woman, the patient is likely to be postmenopause. On physical examination, as many as 50% of patients may have systemic hypertension, and signs of pulmonary hypertension may be noted in as many as 30% of patients.

Daytime laboratory evaluation may not be helpful in identifying affected patients except that an elevated hemoglobin reflects the erythrocytosis related to nocturnal severe oxygen desaturation. The findings of arterial blood gas determinations and pulmonary function tests usually reflect underlying medical conditions such as chronic obstructive airway disease, obesity, congestive heart failure and neuromuscular disorders.

Flow-volume curves have characteristic shapes in some patients with obstructive apnea. A "saw-tooth" pattern is
often observed on the expiratory (and sometimes inspiratory) curve. In addition, flattening of the inspiratory curve with a ratio of forced expiratory flow to forced inspiratory flow, both at 50% of vital capacity, in excess of 1 (consistent with extrathoracic airway obstruction) may be seen. These findings in a patient with clinical features of sleep-associated respiratory disorders substantially increase the likelihood of obstructive apnea.

Various techniques have been used to study the morphology of the upper airway in attempts to predict sleep apnea. These include fiber-optic visualization of the oropharynx, fluoroscopic imaging and computed tomographic scanning of the upper airway, lateral cephalometric roentgenograms and even acoustic reflectance studies of the upper airway. These techniques are of course technically difficult, requiring considerable effort and expertise in their interpretations. Thus they are of little value in a standard medical practice.

Finally, the short-term clinical observation of sleeping patients has recently been evaluated as a screening test. Such bedside observations were found to lack specificity and sensitivity in the diagnosis of sleep-associated respiratory disorders.

It is the current consensus that nocturnal sleep polysomnography is the definitive tool for diagnosing sleep-associated respiratory disorders. Such an evaluation is best done initially in a sleep laboratory, usually in a hospital that is committed to provide high-quality studies. Such studies require a trained technician, a reasonable sleep area and various monitors and recorders, which may be expensive. Such devices are necessary to correctly stage sleep, measure respiratory effort and airflow and continuously monitor oxygenation and cardiac rhythm. Several portable home monitoring systems have recently become available and can provide useful diagnostic information in some cases, at a reduced cost to the patient. These monitors typically record cardiac rhythm, oxygen saturation and respiratory movement and lack the capability for sleep staging, flow monitoring or the exact measurement of respiratory effort. We, therefore, feel the portable monitors are most useful in follow-up studies to evaluate the success of therapy.

**Therapy**

The goals of treatment in the sleep-associated respiratory disorders are to improve alveolar ventilation during sleep, to control the significant signs and symptoms of disease and to improve overall quality of life. To accomplish this, there are several therapeutic modalities, which we have presented in algorithmic form in Figures 1 and 2.

For convenience, we present therapeutic approaches to obstructive and mixed apneas in Figure 1. One should initially exclude clearly reversible causes of these disorders. For ex-
ample, hypothyroidism can be easily treated with thyroid hormone supplementation. Adenotonsillar hypertrophy may promote anatomic narrowing of the upper airway which can be treated with tonsillectomy and adenoid excision. Other anatomic abnormalities, such as micrognathia or retrognathia, may respond less predictably to surgical procedures such as mandibular revision. Such surgical correction should, of course, only be undertaken by a specialist with expertise in the procedure and its application to this group of patients.

Other surgical procedures are directed towards less clearly identified anatomic predispositions to upper airway obstruction. A permanent tracheostomy bypasses the site of obstruction, abolishing obstructive apnea and improving nocturnal oxygen saturation. We currently reserve this procedure for patients with weight-reducing arrhythmias or those for whom other therapies have failed (often due to poor compliance). Another surgical procedure is uvulopalatopharyngoplasty, which extensively revises the soft tissues of the oropharyngeal region. This can resolve upper airway obstructions and obviate the need for a tracheostomy. It must be emphasized that the success of this procedure has been extremely variable primarily due to uncertain criteria for proper patient selection.

A recently developed nonsurgical approach to these patients is the application of continuous positive airway pressure (CPAP) to the upper airway via a nose mask. Nasal CPAP has proved to be well-tolerated and effective in reversing obstructive apnea in several studies. Reasonably priced commercial CPAP units for in-home use by patients are currently available.

There are several medical approaches to the treatment of obstructive and mixed apneas. Weight loss may be effective in most patients, but this has proved difficult to achieve or sustain in this population. Respiratory depressants such as alcohol and sedatives should be avoided in these patients, as they have been well-documented to worsen obstructive sleep apnea. Nocturnal oxygen therapy will improve oxygen saturation during sleep and in as many as 40% of patients significantly reduces apneas. Due to the high cost of oxygen, it should be used only as an adjunct to other measures directed at improving alveolar ventilation. Progesterone, a respiratory stimulant, has been used with some success in some studies, but we have found it to be useful primarily in those patients also suffering from PAH. Protriptyline is a non-sedating tricyclic antidepressant we have used in the treatment of obstructive apnea with about 20% to 30% success. Therapy is typically initiated with a dose of 5 to 10 mg at bedtime but may be increased as needed to a dose of 30 mg. Patients should then be closely monitored for the appearance of side effects (insomnia, rash, urinary retention in men and so forth) that may be intolerable, especially at higher doses.

Figure 2 shows therapeutic approaches to central apnea and PAH. There are no surgical procedures currently indicated here, although electrical pacing of the phrenic nerve or diaphragm may be done in the absence of spontaneous respiration. We have found it unnecessary to use this procedure. Alternate mechanical approaches include the use of nocturnal negative-pressure ventilation and even the use of rocking beds. It should be remembered that in some patients with combined obstructive and central apneas, obstructive episodes may actually worsen during negative-pressure ventilation and these patients may, therefore, be considered candidates for nocturnal positive-pressure ventilation.

Medical approaches to these patients are more commonly used. Weight loss, although helpful, is again difficult to achieve or sustain in this population. Oxygen should again be used primarily as an adjunct to other measures designed to improve alveolar ventilation, although it has effected a cure in some patients with central apnea, presumably due to some respiratory stimulant effects. Progesterone has been successfully used to treat central apnea and primary alveolar hypoventilation, using a dose of 20 to 40 mg three times a day. Carbonic anhydrase inhibitors are also respiratory stimulants and have been found effective in some patients. Dichlorphenamide (Daranide) may be used in doses of 50 to 100 mg twice a day, while acetazolamide (Diamox) may be used in a dose of 250 mg four times a day. Theophylline is a well-known respiratory stimulant, used frequently with success in treating recurrent apnea of prematurity. Although some researchers report limited success using theophylline in the therapy for central apnea in adults, our experience with such usage has been generally disappointing.

After a tolerable therapeutic intervention has been initiated, symptoms and features typically seen with the syndrome should improve. A follow-up nocturnal polysomnographic study is usually indicated, however, at a later date to assess effects of therapy and to direct the physician towards further intervention if necessary.

**GENERAL REFERENCES**


