threatens the health of children. The risks are greatest for children exposed during the first two years of life. The incidence of hospitalization for bronchitis and pneumonia is higher among children of smokers. They have increased airway reactivity and greater frequency of respiratory illnesses and symptoms, including cough, phlegm production, and wheezing. About 20% of childhood asthma is caused by parental smoking.

Smoke-exposed children also have smaller rates of increase in lung function as the lung matures. Passive smoking as a child may lead to a detriment of 3% to 5% in FEV_1 levels at age 20. This is probably clinically insignificant; the question is whether subtle damage predisposes to lung disease in adulthood, such as COPD among smokers.

LUNG CANCER

The risk of lung cancer to passive smokers remains the area of greatest controversy despite the Surgeon General's estimate that 5,000 deaths per year in the U.S are due to lung cancer caused by passive smoking. Although sidestream smoke is unquestionably carcinogenic, it is uncertain whether exposure is adequate to overcome a biological threshold effect to cause cancer. It is estimated that passive smoking is equivalent to actively smoking 0.1 to 1.0 cigarette per day.

By extrapolating from the known risk of lung cancer in heavy smokers, the expected relative risk (assuming no threshold effect) from such low-level exposure is about 1.03 to 1.38. That is, a nonsmoker who experiences significant passive smoking might be expected to have a 3% to 38% increased risk of lung cancer compared to a nonsmoker who is not exposed to environmental tobacco smoke.

At least 13 epidemiologic studies in several countries have investigated the relationship between passive smoking and lung cancer. All but two or three of these studies report a modest elevation of the risk of lung cancer among involuntary smokers, but in only six studies was the positive association statistically significant. Pooled results of all studies suggest that heavy passive smoking does increase the risk of lung cancer by about 10% to 30%. This conclusion is not accepted by all experts, as most of the studies have some elements of bias, such as possible misclassification of lung cancer and passive smoke exposure, or interviewer and respondent bias.

ASTHMA

Three studies have evaluated the effects of passive smoking on persons with a history of asthma; nearly all subjects had relatively inactive or well controlled asthma when tested.

One study (Shepard et al. Environ Res 1979; 20:392–402) demonstrated no effect on lung function in asthmatic persons exposed to two hours of passive smoking in an enclosed room. However, in a similar investigation, Dahms et al (Chest 1981; 80:530–534) found a 20% decrease in FEV_1. In our assessment of the acute effects of passive smoking on lung function and bronchial reactivity, spirometry remained stable and we found no change in airway reactivity (Wiedemann et al. Chest 1986; 89:180–185).

Although these three studies overall suggest no acute respiratory risk to young asymptomatic patients with a history of asthma, some important aspects have not been evaluated, including the delayed effects of exposure, the effects of chronic exposure, and the effect on patients who have active asthma.

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APNEA: EASY TO DIAGNOSE, DIFFICULT TO TREAT

Sleep apnea, which affects 1% of the population, has far-ranging consequences, including a spouse who refuses to share the patient's bed because of loud snoring, daytime hypersomnolence that can be incapacitating, and cardiorespiratory sequelae that may be life-threatening.

CHARACTERISTICS

Apnea is defined as cessation of airflow for at least 10 seconds during sleep. The apnea index is the number of apneas per hour. A person with sleep apnea syndrome has an apnea index greater than 5, with apneas occurring during both REM and non-REM sleep.

Early symptoms of sleep apnea are nocturnal and are characterized by loud snoring and episodic breathing

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cessation. With more severe disease, symptoms—primarily hypersomnolence—occur during the day. In serious cases, there are cardiorespiratory consequences, including pulmonary and/or systemic hypertension, secondary erythrocytosis, right heart failure, and even left ventricular failure.

DIAGNOSIS

The patient with suspected sleep apnea should be referred to a sleep laboratory. Simple observation of the patient during sleep will confirm the diagnosis in 75% of cases. The overnight polysomnogram (PSG) is the gold standard of diagnosis, particularly for mild to moderate sleep apnea. Daytime PSG may be diagnostic in severe sleep apnea, but may understate the severity of sleep apnea in mild to moderate disease because these patients may not achieve REM sleep or deep stages of non-REM sleep during the day.

Polysomnography includes an electroencephalogram, electro-oculogram, and chin electromyogram. These determine whether sleep occurs and at what stages. Nasal and buccal thermistors document sleep apnea by measuring air flow. Chest and abdominal strain gauges help to differentiate mixed, obstructive, and central apneas. An electrocardiogram will detect hypoxemia-induced arrhythmias.

TREATMENT

Treatment measures have met with limited success. Weight reduction may help obese patients (who account for 60% of those with sleep apnea); but many patients are unable to achieve or maintain weight loss—and even when they do, it is not always curative. Other conservative measures include avoidance of alcohol and respiratory depressants; treatment of associated endocrine diseases such as hypothyroidism, acromegaly, and Cushing's syndrome; treatment of allergic or chronic rhinitis; and correction of anatomic obstructions such as deviated septum or large tonsils. Mild sleep apnea may respond to the patient's simply sleeping on his side.

Oxygen supplementation is variably beneficial. In a small percentage of patients, there is a decrease in daytime sleepiness and nocturnal oxygen desaturation, but apnea episodes are often prolonged, with the development of respiratory acidosis. Oxygen supplementation does not improve sleep architecture, and central apnea episodes tend to decrease while obstructive episodes—which are more dangerous—increase.

Protryptyline HCl (Vivactil) decreases desaturation that occurs during sleep and has been shown, statistically, to be beneficial. Closer examination of the data reveals that patients have less hypoxemia because the drug causes deprivation of REM sleep, where most apneas occur. Although the apnea index and saturation studies indicate improvement, in fact the patient is sleeping less well than before he started the therapy.

Nasal continuous positive airway pressure (CPAP) is a simple mechanical approach that produces dramatic results in both central and obstructive apnea. The apnea index can be reduced from as high as 46 to 1 or 2. REM sleep improves markedly and daytime hypercapnia and sleepiness are reduced. Despite the efficacy of CPAP, patient compliance is sometimes poor, even though the newer devices are less cumbersome and noisy.

Uvulopalatopharyngoplasty (UPP) involves surgical removal of redundant pharyngeal mucosa, which obstructs the airway. Although most patients improve symptomatically, only about 50% show improved polysomnography. Clearly, better methods are needed for patient selection and/or assessment of benefits.

Tracheostomy is the surgical modality most likely to provide consistent long-term benefit. It is recommended for patients with life-threatening obstructive sleep apnea syndrome if there is no other surgically correctable obstruction. Tracheostomy is disfiguring and is associated with considerable emotional morbidity. Patients who undergo the procedure—and their families—should be counseled prior to surgery and trained in tracheostomy hygiene and care.

To make decisions about therapy, evaluate the patient using a scale of increasing severity from 1) asymptomatic to 2) nocturnal sleep disruption alone to 3) daytime symptoms to 4) severe cardiorespiratory sequelae. The most conservative therapy is indicated for those at the bottom of the severity scale. If nonpharmacologic methods are unsuccessful, a trial of protriptyline is indicated. No further treatment is recommended for mild disease. In the case of moderate or severe disease, CPAP or surgical measures warrant consideration.

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