data are never used in clinical decision making.[19]

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Screening for Sleep Apnea?

Breathing during sleep has only recently become of interest to the physician. In 1958, Robin[1] observed, "The sleeping patient is still a patient. His disease not only goes on while he sleeps, but indeed may progress in an entirely different fashion from its progression during the waking state." The first reports of obstructed respiration during sleep were made in 1965.[2,3] In the subsequent 24 years, much has been learned about sleep-disordered breathing,[4-7] but large gaps in our understanding persist. Fundamental questions about the true incidence of the disorders, their natural history, and their prognosis are among them. As we consider ways in which to answer these, it seems to us an appropriate time to raise the further question of the methods by which practitioners will undertake their evaluation of patients with sleep-disordered breathing. At present, it is recommended that the initial laboratory evaluation be done overnight in a sleep laboratory, observed by a trained technician. Even though patients are not considered inpatients, establishing (or excluding) the diagnosis by polysomnogram is expensive, and, in addition, questions about the effect of the artificial environment on the measurement persist.[8] The need for an effective and acceptable home screening tool is evident.

The apparent incidence of obstructive sleep apnea varies greatly across diagnostic groups. In population surveys an incidence of as little as 1 percent[9] or as great as 5 percent has been reported. Such surveys used questionnaires, with obvious drawbacks. More directed studies invariably found greater incidences in specific populations. Peter and colleagues[10] reported that 20 percent of patients with suspected coronary artery disease have >10 apneas/h, all with accompanying hypoxia or cardiac irregularities, and fully 6 percent have "high sleep apnea activity," which they defined as >100 apneas/night. Furthermore, the incidence of high sleep apnea was 35 percent in 20 patients with reduced cardiac ejection fractions (although this group may have had periodic breathing as a result of heart failure). Similarly high incidences were reported among the elderly, in whom as many as 37 percent are affected,[11] while four groups of investigators in the United States found significant sleep apnea in 30 percent of patients with essential systemic hypertension.[12-15] However, a population study in the United Kingdom did not confirm this.[16] In spite of

this, the overwhelming evidence is that sleep apnea syndromes are common.

If common, how significant? Data on prognosis are limited. Guilleminault and Dement[4] in their classic monograph identified a subgroup of patients with obstructive sleep apnea who refused tracheostomy and in whom an excessive mortality was quickly found. More recently Kryger and Roth[17] reported on the outcome in a larger untreated group of 385 followed up over nine years. The mortality in those with an apnea index of >20 was much greater than those with an index of <20. Treatment of another group of 25 by nasal continuous positive-airway pressure was effective, and no deaths were reported. Part of the increase in mortality may be due to the propensity of patients with sleep apnea for motor vehicle accidents.[18,19]

Thus, it seems that sleep apnea is common, potentially serious, and can be effectively treated, important criteria to satisfy in considering screening for a disease. In addition, the screening tool must be sensitive, specific, and economical. Candidates for such screening tests are logically those elements that physicians already use in making the diagnosis: the patient's history; perhaps the physician's findings; and the nonneurophysiologic components of the polysomnogram, specifically the ECG, measurements of airflow and respiratory effort, and oxyhemoglobin saturation.

A scoring system based on the history has been developed by a number of groups. The Hawaii Sleep Questionnaire survey of adult clinic visitors[20] evaluated the usefulness of a self-report measure of sleep apnea. Items from the questionnaire were weighted and summed to yield an apnea score. This score was validated against polysomnographic apnea indices and shown to have a sensitivity of 71 percent and a specificity of 91 percent. Stopped-breathing during sleep and loud snoring are particularly suggestive symptoms.[21]

Among the individual elements of the polysomnogram, three seem particularly attractive:

1. *The ECG:* A convincing report by Guilleminault et al[22] identified cyclical variations in heart rate in the sleep apnea syndrome, with all patients having typical cyclical changes in R-R interval due to alternating bradycardia (the diving reflex) and tachycardia. The pattern is pathognomonic but requires a special computer program to analyze the Holter monitor recording. In spite of the frequency with which such monitoring is done, an informal survey of local cardiology departments revealed that none analyzed the recording for this abnormality.

2. *Oximetry:* Data from the continuous measurement of oxyhemoglobin saturation can be displayed and summarized together with heart rate. Personal experience has shown that the cyclical bradycardia/tachycardia referred to before is readily apparent. This makes the study doubly valuable, for even without sufficient hypoxemia to appreciably alter oxyhemoglobin saturation, pulse rate will often change with obstructive apneas. When hypoxemia is prominent, the records can be pathognomonic.

3. *Tracheal sound:* the *sine qua non* of apnea is absence of airflow. Many devices are used to measure this including the thermistor, pressure transducer, and gas analyzer. Tracheal sound has been shown to effectively identify apneas[23] and computer hardware and software have been developed.[24]

Severe sleep-disordered breathing is likely to be detected by any of these methods. However, using only one, the sensitivity may not be sufficient. In addition, if the tracing is normal, can one be sure that the patient actually slept? The absence of wrist movement has been shown to correlate well with sleep time, and has been proposed as an adjunct to oximetry, for example. It should be noted, however, that in some patients with sleep apnea the characteristic lack of movement seen in sleep is lost because of sleep disruptions, to the extent that wrist activity recording has itself been suggested as a screening test. In patients for whom a clinical suspicion of sleep apnea exists, a specificity of 95 percent and a sensitivity of 89 percent has been reported.[25]

As information about sleep-disordered breathing is disseminated, the public and physicians will more often ask who is affected. Referral to a sleep laboratory of all the potential patients among snorers (30 percent male population), somnolents (5 percent), and hypertensives (20 percent) may not be the first best approach. As with other expensive technologies, we should consider limiting its use to those most likely to benefit. In most other diseases the gold standard test (which is often invasive) is not used until simpler screening studies have been performed to select an appropriate population. Examples of this approach include the exercise ECG before coronary angiography and the perfusion lung scan before pulmonary angiography. It seems to us that the same approach could properly be considered in the case of sleep-disordered breathing.

Screening of patients beforehand is, we believe, indicated, and every effort should be made to define the most practical and acceptable tool(s). From among those discussed and currently available, it seems that pulse oximetry (with display of saturation and heart rate variations) may be the best candidate. At a cost of <$100, this is more appropriate than Holter monitoring, which costs considerably more (about $600). A history and physical examination will always be available and should, of course, be used to supplement the study by calculating a clinical score, one for each of severe hypersomnolence, loud snoring, observed apneas, obesity, and hypertension. False negative oxi-

metric study results have been found in individuals with a high score (3.8 ± 1.3, mean $\pm$ SD), while the score is low (2.5 ± 0.6) in the truly negative.[21]

Because of the potential impact of unrecognized significant sleep-disordered breathing, every effort must be made to identify those affected as well as to define more completely the natural history. To do so will require a tool simpler than polysomnography. In the early days the observation of apneas in a sleeping patient sufficed,[26] but this specific and sensitive method would be too dependent on wake-sleep schedules (the physician's as well as the patient's), and a consensus about screening must be achieved.

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Antibiotic Studies in Pneumonia

Pitfalls in Interpretation and Suggested Solutions

Pneumonia continues to offer a major diagnostic and therapeutic challenge for the clinician. Lower respiratory tract infections constitute a major usage group for antibiotics, and the commercial success of a new agent virtually requires that it carries an indication for these infections. Consequently, many new antimicrobial agents continue to be introduced for lower respiratory tract infections with a corresponding number of clinical studies being performed, making it increasingly difficult for the practicing physician to select the appropriate antimicrobial with confidence.

The readers of *Chest* are well aware of the difficulty in conducting antibiotic studies in pneumonia. The clinical diagnosis is often not straightforward. Fever, chills, and cough do not always signify pneumonia. Markings on chest x-ray films can suggest infection but may also be consistent with congestive heart failure, pulmonary embolus, tumor, old scarring, or an immunologic process.

The major problem is that the determination of the etiologic agent in pneumonia is fraught with difficulty. The most commonly used criterion is the isolation of a microorganism from the sputum culture. However,