nodeficiency syndromes and mechanisms of lung infection and injury.

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Sleep Apnea: When Does Better Become Benefit?

Implicit in the physician's decision to treat is recognizing impairment or impending impairment of the patient's well-being and the expectation of improving longevity and/or quality of life. Until recently, the only treatment for obstructive sleep apnea was tracheostomy. This procedure, while definitively alleviating obstructive apnea and excessive daytime sleepiness, often has significant medical and psychological morbidity. Therefore, other therapeutic alternatives, including protriptyline, continuous positive airway pressure via nasal mask (n-CPAP) and uvulopalatopharyngoplasty (UPPP), have been tried. Unlike tracheostomy, these therapies frequently provide only partial reductions in obstructive apnea and nocturnal hemoglobin oxygen desaturation. Without question, the abolition of malignant cardiac dysrhythmias and disabling daytime sleepiness represent therapeutic success. Less obvious is how to determine the level of benefit derived from varying degrees of amelioration of apnea and hypoxia in those patients without such dysrhythmias or disabling sleepiness. The problem is even more difficult in asymptomatic patients with sleep apnea who come to medical attention because of spousal observations (truly silent sleep apnea). In these individuals, the physician must decide if any therapy will be beneficial.

Given the current level of knowledge, clinicians and investigators can only make assumptions regarding the significance of reductions in apnea frequency and nocturnal hypoxia following therapeutic intervention. For example, in assessing the results of UPPP, several groups of investigators have identified therapeutic success or response in terms of a specified percent increase in the nadir of hemoglobin oxygen saturation during sleep. Examination of these data, however, reveal that despite the lack of response or success, some patients continued to have considerable hypoxia during sleep following this surgical procedure. Similarly, while nocturnal hemoglobin oxygen saturation is generally increased following the administration of protriptyline and n-CPAP, hypoxia to variable degrees still occurs in some patients. In this context, it is disturbing that, in animals, 8-16 hours per day of hypoxia at levels comparable to those observed in some sleep apnea patients who seemingly responded to therapy results in sustained elevation of pulmonary artery pressure and alterations in ventricular wall thickness. In dogs, repetitive hypoxic challenges, similar in degree to that observed in some patients after treatment for sleep apnea, causes progressive increments in pulmonary artery pressure. These results may not be totally applicable to the patient with sleep apnea whose hypoxic events are characteristi-
cally much shorter, although more frequent. They do, however, provide a basis for concern regarding what constitutes significant improvement in oxygenation during sleep in these individuals following therapy. However, in the final analysis, as indicated by Wynne,10 the degree and duration of hypoxia which is harmful has yet to be established.

Changes in apnea frequency have also been employed to assess therapeutic results,11 as in the study by Conway et al in this issue (see page 385). Complicating the application of this criterion, however, is the question of what constitutes a pathologic frequency of apnea. Indeed, several investigators have suggested that normal values may vary, depending on such factors as age and sex.12,13

Finally, claims of therapeutic effectiveness in sleep apnea patients have been made on the basis of subjective improvement in daytime alertness. This practice is questionable, since subjective improvement in daytime alertness following therapy often occurs despite the absence of polysomnographically documented amelioration of sleep-disordered breathing.14 For a variety of psycho-social reasons, patients may not admit to the lack of improvement in daytime sleepiness following therapy. Objective studies such as multiple sleep latency tests are needed, particularly in clinical research, to evaluate changes in daytime sleepiness following therapeutic intervention.

In summary, important information about the clinical and physiologic significance of varying degrees of sleep apnea and nocturnal hypoxia is presently unavailable. Such data must be obtained from carefully designed, long-term, multi-center cooperative studies. Only then can the indications for therapeutic intervention and the criteria for therapeutic benefit be delineated.

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Dipyridamole Thallium Testing
An Alternative Form of Stress Testing in Patients Unable to Exercise

But the patient cannot exercise? This is not an uncommon question facing physicians managing patients with suspected or documented coronary artery disease. The inability to evaluate noninvasively the extent and severity of exercise-induced ischemia has limited management of these patients to decisions based predominantly on a history of symptomatic angina and, if available, coronary arteriographic findings. In patients undergoing exercise testing, physiologic impairment due to underlying anatomic coronary disease may include exercise-induced ST segment changes, reversible thallium-201 perfusion defects post exercise, or fall in radionuclide left ventricular ejection fraction during bicycle exercise. Until recently, those unable to exercise were mostly evaluated on the basis of prior pattern, ie, frequency and severity of chest pain.

Recent reports14 have demonstrated the usefulness and safety of dipyridamole-thallium imaging in categorizing patients into high and low risk subsets for future cardiac events. It has previously been shown1 that intravenous dipyridamole-thallium scintigraphy has a 93 percent sensitivity and an 80 percent specificity for the detection of coronary disease. Leppo and colleagues1 studied 51 patients recovering from acute myocardial infarction and found that of all variables