Nocturnal Hypoxemia in COPD

It has been well established that some COPD patients experience non-apneic episodic hypoxemia, or worsening hypoxemia, during sleep. As would be expected, those with more significant awake hypoxemia usually experience a more serious nocturnal fall in arterial oxygen saturation. Some patients with adequate awake oxygenation also develop desaturation during sleep, usually in REM sleep. Whether the occurrence of nocturnal hypoxemia in these COPD patients with adequate daytime oxygenation is a common clinical problem was not established until the work of Fletcher and co-workers (see page 604). These investigators studied the prevalence of non-apneic sleep arterial oxygen desaturation in a large group of patients. They surveyed patients with a daytime $P_{O_2} \geq 60$ mm Hg and found that approximately one-quarter of the population experienced nocturnal arterial oxygen desaturation. However, the extent of desaturation observed was not as severe; the mean SaO$_2$ nadir was 81 percent. Of interest, the COPD patients who developed desaturation did not demonstrate obvious hypoxic complications, such as pulmonary hypertension or erythrocytosis.

The crucial question raised by this work is whether any cardiovascular or neurologic sequelae result from this degree of intermittent arterial oxygen desaturation. Now that we know that sleep-related arterial oxygen desaturation occurs in a significant proportion of COPD patients with adequate awake oxygenation, we need to identify how many of these patients experiencing nocturnal desaturation have, or will develop, pulmonary hypertension. Recently, Fletcher et al presented preliminary results of right heart catheterization in a sample of patients and demonstrated that pulmonary vascular resistance (PVR) was significantly higher in the COPD patients with desaturation than in those without desaturation. The cardiac output and right ventricular ejection fraction values were not different between the groups. Since the carbon monoxide diffusion capacity was similar in each group, the increased PVR was not thought to be due to more vascular destruction or a smaller vascular space. Although acutely induced hypoxia worsened the PVR and increased the pulmonary artery pressures, the short-term administration of O$_2$ did not decrease the PVR below baseline values. Whether this laboratory finding is an early hypoxic complication, and whether increased morbidity or mortality may eventually occur in these patients is unknown. This information will take several years to accumulate, but when available, the findings will provide a significant advancement in our understanding of factors that complicate chronic lung disease.

In order to learn whether sleep-related arterial oxygen desaturation in COPD is an important variable that affects disease course, longevity and quality of life, a large number of patients will need to be studied. A NOTT$^3$ or MRC$^4$ type of controlled multicenter investigation to define the sequelae of this problem and to address the benefit of oxygen therapy in these patients might be well advised. In such a large study, patients could be stratified according to the severity of the hypoxemia and its complications and be placed into treatment and control groups. Thereby, the natural history of this process and a dose-response to oxygen therapy could be established. For several reasons, such a study would be arduous: 1) differences between control and test groups might be small, and therefore, patients would have to be followed many years; 2) many variables could not be controlled. These factors would make the establishment of definitive conclusions difficult. Courageously, Fletcher and colleagues have already started the type of investigations needed in this process. Surely, we will learn from their experience, if and when a large multicenter study becomes a reality.

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REFERENCES

1. Fletcher EC, Miller J. Fletcher J, Miller T, Costarangas C. Cardiopulmonary hemodynamics in stable COPD patients with $P_{O_2} \geq 60$ torr and nocturnal $O_2$ desaturation. Denver: World Congress on Oxygen Therapy and Home Care, February 19-21, 1987
