Smoking, Carboxyhemoglobin Levels, and Oxygen Therapy

To the Editor:

In the article by Lilker et al entitled "Portable Oxygen in Chronic Obstructive Lung Disease with Hypoxemia and Cor Pulmonale: A Controlled Double-Blind Crossover Study" (Chest 68:236-241, 1975), a very important factor was either not mentioned or not considered. Nine patients with severe chronic obstructive pulmonary disease (COPD) and severe hypoxemia were studied. It does not state anywhere in the study whether or not these patients were smoking during the time of the study, and neither oxygen saturation nor carboxyhemoglobin levels were measured. Of the nine patients, three patients had improvement in exercise capacity. The authors noted that although oxygen therapy corrected the hypoxemia, there was no significant change in the red blood cell count, hemoglobin level, or reticulocyte count. The authors concluded that further studies are needed to evaluate the patients who show a striking improvement while receiving portable oxygen therapy and yet are not clearly separated from the others. Lilker et al stressed the need for studying differences in the reactivity of the pulmonary vascular bed and left ventricular failure.

In our experience, it is not uncommon for patients, even with severe COPD, to continue to smoke heavily and have markedly elevated carboxyhemoglobin levels. Many of these patients may have secondary polycythemia with severe hypoxemia; however, it is unlikely that oxygen therapy will benefit them because the desaturation is due to binding of hemoglobin with carbon monoxide. Recently, other patients have been described with polycythemia secondary to heavy smoking, so that this is a real cause of reversible secondary polycythemia. In any study designed to determine the effects of chronic low-flow oxygen administration, an accurate smoking history and measurement of carboxyhemoglobin level or actual oxygen saturation must be done. A difference in response may have been due to the fact that the three patients who seemed to respond were nonsmokers, whereas the other six patients continued to smoke heavily. In patients who continue to smoke heavily, it would not be expected that oxygen would bring about a significant improvement of their desaturation. We feel this is a critical point to include in any study such as that presented by Lilker et al. Perhaps if they had data on smoking histories, carboxyhemoglobin levels, or oxygen saturations, they did not present it due to space limitations; however, if they neglected this factor, perhaps reassessing their patients in this way may shed light on what separates the responder from the nonresponder.

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REFERENCES


To the Editor:

I thank Dr. Goldman for his constructive criticism. To our knowledge, none of the patients smoked during the entire three months of the study, as judged by what they told us, what their families told us, and the observations made by the physician on his weekly visits, as well as the patient's reassessments while in the hospital.

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Corticosteroids and Pericarditis of Acute Myocardial Infarction

To the Editor:

In an article entitled "Pericarditis of Acute Myocardial Infarction" (Chest 67:647-653, 1975), Toole and Silverman describe the use of corticosteroids for the symptomatic treatment of pericarditis associated with acute myocardial infarction. They indicate that this therapy is without major complications and provides the advantage of decreased narcotic use. Bulkley and Roberts described a patient treated with...