and least in those with the highest initial values." Sports physicians know that it is also true among healthy sedentary adults who begin training.

In regard to the small increase in VO2 max observed for their patients, the authors think that this would have been more important if they had used a treadmill instead of a cycle ergometer. We doubt this, because it is not what we have observed. Hickson and Rosenkoetter submitted 12 subjects to endurance training (cycle ergometer and running) for 40 min daily, 6 days a week for 10 weeks. The mean increase in VO2 max was 25 percent with the cycle ergometer and 20 percent with the treadmill. These findings illustrate the influence of protocol on the test result.

Concerning the opportunities for improvement of physical fitness of the most affected patients, we think that the discussion has been biased, for among the patients whose VO2 max and VO2 max are the most lowered, some are limited by important lung reshaping, while others are only confined to a sedentary lifestyle style. The latter patients will be more improved by a retraining program. The others will benefit from their training, though perhaps more in terms of psychological criteria than functional performance.

Globally speaking, we are in agreement with the conclusion of authors that pulmonary rehabilitation can reasonably be offered to any patient with chronic lung disease with the expectation that improvement can occur regardless of the severity of the underlying disease process.

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REFERENCES


To the Editor:

We find the observations reported by Drs. Menier and Talmud to be quite interesting, and we are gratified that their observations are similar to our own. They have observed many of the same benefits from a multidisciplinary rehabilitation program that we have previously reported. One of the major differences between their data and ours is that they observed a statistically significant increase in maximal oxygen consumption after pulmonary rehabilitation. As a percentage, this increase in maximal oxygen consumption is slightly less than we have observed, but it is statistically significant, presumably because of the large number of patients who were evaluated. It is possible that an even more significant increase in maximal oxygen consumption might have occurred if patients had been trained for a longer time period. A mean training duration of 4 weeks was used, whereas our program was conducted for a 9-week period. Similarly, they observed a significant increase in spirometric values and improved airway resistance measurements. Although these changes were numerically relatively small, they assumed statistical significance because of the large number of patients studied.

One other difference pointed out by Drs. Menier and Talmud is that their training protocol is for patients to exercise 5 days per week, whereas our protocol involved exercise for 3 days per week. Our sessions were generally longer than theirs, but with suitable rest periods, we have not found this to be a difficulty for our patients. Clearly, there will be some patients who cannot tolerate exercise sessions for as long as 60 min; the length of these sessions should be individualized.

Overall, however, it is reassuring and of great importance that other investigators have been able to confirm the observation that individuals of all degrees of severity stand to benefit from a multidisciplinary rehabilitation program. Based on these observations, we hope that patients with COPD will continue to be enrolled in pulmonary rehabilitation programs.

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Pulmonary Edema Associated With Radiocounter Material

To the Editor:

I was intrigued by the case report by Kozlowski and Kollef, which appeared in the August 1992 edition of CHEST. Their discussion of the putative mechanisms involved in the development of noncardiogenic pulmonary edema following administration of radiocontrast material (RCM) is praiseworthy. While the case presented could support a diagnosis of direct endothelial injury from RCM administration based on the mechanisms reviewed, it does not readily depict a clinical scenario that would result in pulmonary edema due to acute upper airway obstruction. I would like to offer an alternative hypothesis for the condition described.

The phenomenon of negative pressure pulmonary edema is believed to occur when excessive inspiratory force is directed against an obstructed upper airway (Müller maneuver), resulting in large swings in intrathoracic pressure. In most studies, the associated development of increased extravascular lung water is related to the generation of extremely negative transpulmonary pressures during the inspiratory effort. The condition most often occurs in children or relatively young healthy adults and is rare in those situations in which, for mechanical reasons, significantly negative transpulmonary pressures cannot be generated (eg, in elderly patients and in flail chest). It is exceedingly common for individuals to experience a sensation of neck "fullness" with RCM infusion, and while this patient certainly demonstrated respiratory compromise, nothing compatible with severe inspiratory stridor or Müller maneuver-like efforts is described.

The rapidity (1 min) with which the dyspnea appeared in the absence of these respiratory movements favors a direct effect of RCM on pulmonary and perhaps systemic vasculature, possibly by mechanisms similar to those seen in a variety of animal models of oxidant-mediated acute endothelial lung injury. This injury can be dose dependent, and a mild form could be expected to cause a transient acute edematous lung injury, as in the case described.

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