To the Editor:

I am pleased to see that Dr Schachter and Dr Domingo and colleagues confirm our observation relative to an increase in arterial $P_{O_2}$ occurring three to six months after initiation of oxygen therapy in stable patients with COPD.1 However, Dr Schachter takes issue with the recommendation that supplemental oxygen not be discontinued when the arterial $P_{O_2}$ increases as a result of the beneficial effects of oxygen therapy. Dr Schachter further comments that in his experience once the patient has been able to discontinue the supplemental oxygen, the hypoxemia and cor pulmonale do not recur, barring an acute exacerbation of the COPD.

First of all, it is extremely uncommon for patients with a $P_{O_2}$ of 55 mm Hg or less (when clinically stable) not to experience subsequent exacerbations of their COPD, even with what would be a minor respiratory illness for normal individuals. Even more important is the fact that the multicenter clinical trial funded by the National Institutes of Health (the NOTT study)2 found that patients with an arterial $P_{O_2}$ of 55 mm Hg or less (or 56 to 59 mm Hg with cor pulmonale or erythrocytosis) who qualified after only three weeks of observation experienced a significant reduction in mortality that was evident by the end of one year and that persisted for the three-year period of study. None of these patients was retested, and there is no evidence or assurance that discontinuing oxygen during the course of this study would have resulted in the same survival statistics for these individuals. It also should be noted that all of the patients in our study who had an increase in $P_{O_2}$ at six months continued to demonstrate desaturation with walking, indicating that they would have experienced periods of desaturation during their daily living cycle without supplemental oxygen.

The bottom line is that the only conclusive scientific data currently available indicate that if patients qualify for long-term oxygen therapy in a stable clinical state, then continued oxygen therapy results in improved survival and an improved quality of life. Until a multicenter study specifically examines those patients who show improvement in $P_{O_2}$ attributable to the beneficial effects of the therapy and it has been demonstrated that stopping therapy does not jeopardize any of the positive results found in the NOTT study, then it is my strong conviction that termination of therapy is not scientifically justified. It may be that some patients can safely terminate oxygen therapy after a period of time if the $P_{O_2}$ increases, but currently we are uncertain about the effect of exercise or nocturnal desaturation in these individuals and what would be the long-term prognosis when compared with a group of similar patients who are allowed to continue oxygen therapy. The findings of the NOTT study appear to be conclusive as far as the study was taken, and until new data from controlled studies are available the lives and welfare of patients with COPD and hypoxemia should not be jeopardized on the basis of uncontrolled observations that patients appear to do well as long as they do not experience an acute exacerbation of their disease. After all, we were unable to recognize the significant benefit of nearly continuous oxygen (19 h/d) over nocturnal oxygen until a multicenter study was conducted. Let us not discard these findings simply because we now recognize that oxygen has a reperative effect after months of therapy.

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Pulmonary Embolism Associated with Postoperative Deep Breathing

To the Editor:

I would like to report a unique association of pulmonary embolism with incentive deep-breathing exercises. A 52-year-old man underwent right total hip arthroplasty (THA) in 1977 and left THA in 1984. The patient was recently hospitalized for revision of the right THA. His past medical history was significant only for a 55-pack-year history of smoking. On the night prior to surgery he received 5 mg of coumadin and was placed on a coumadin protocol to achieve a prothrombin time of 1.3 to 1.5 times control in the postoperative period.

On postoperative day 1 bilateral atelectasis was noted on the chest radiograph. Treatment with albuterol via metered-dose inhaler and incentive spirometry (Air-eze Incentive Deep Breathing Exerciser, Sherwood Medical, St Louis) was begun. The next day, while the patient was performing deep-breathing exercises, he experienced the acute onset of dyspnea and left-sided chest pain.

An arterial blood specimen showed a pH of 7.46, a $P_{O_2}$ of 35 mm Hg, and a $P_{CO_2}$ of 76 mm Hg while receiving oxygen by face mask with an $F_{O_2}$ of 50 percent. A heparin bolus of 5,000 U was given, followed by a continuous intravenous infusion at 1,000 U/h. A ventilation-perfusion scan was interpreted as being indicative of low probability for pulmonary embolism. A subsequent pulmonary artery angiogram demonstrated two intraluminal filling defects in the left lower lobe consistent with acute pulmonary embolism.

Venous Doppler examinations of lower extremity deep veins have demonstrated phasic respiratory flow-wave patterns during quiet breathing.1 The Valsalva maneuver has been shown to eliminate these venous Doppler flow-wave signals with significant accentuation of the flow signals following termination of the maneuver.1 Pulmonary embolism due to presumed dislodgment of lower extremity thrombus has been shown to occur with the act of defecation and performance of the Valsalva maneuver.2

A similar dislodgment of thrombus induced by a hydrostatic pressure gradient presumably occurred in the present case during deep breathing and the generation of negative intrapleural pressures. The importance of this observation is unknown. In postoperative patients with documented deep vein thrombosis and a high risk for pulmonary embolism, alternative methods of preventing atelectasis, such as the application of nasal continuous positive airway pressure, may be preferred.1

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