large series of patients treated for spontaneous pneumothorax, they focus on the clinical aspects, but also list the possible mechanisms of reexpansion pulmonary edema, including increased pulmonary micrvascular permeability (PMVP).

The direct measurement of PMVP in man remains impossible, but a double-isotope scintigraphic method that provides a noninvasive index of PMVP has been developed. This method can be used to generate parametric images of the lung fields, so that regional differences in PMVP can be studied. Using this technique, we have been able to demonstrate increased PMVP in the reexpanded lobe or segment in the majority of patients in whom a large pneumothorax has been drained. These changes are also detected after drainage of large pleural effusions and after reexpansion of collapsed lung by laser bronchoscopic resection of occlusive endobronchial tumor. At present, only supportive therapy can be offered once reexpansion pulmonary edema has developed, but it is hoped that further insights into the pathophysiologic mechanisms may lead to more specific treatments.

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To the Editor:
We were pleased to learn that Dr Davies and his colleagues had read our article on reexpansion pulmonary edema (REPE).

Although REPE is clinically a very important matter, the mechanism of its occurrence has remained unexplained. We have not had the means to inspect the mechanism in detail because, as Dr Davies and his colleagues point out, the direct measurement of pulmonary microvascular permeability (PMVP) in man remains impossible.

On the basis of the results of a retrospective study of clinical cases of REPE, we postulated that age-related changes in the lung may afford some degree of protection against the development of REPE. Dr Davies and his colleagues found that PMVP increased in the reexpanded lobe or segment and proposed that increased PMVP causes REPE. These findings are very interesting and important, although they do not explain why REPE occasionally occurs in the contralateral lung. I think that our findings and the findings of Dr Davies do not clash substantially, because we did not study the mechanism of occurrence of REPE but postulated that age-related changes in the lung may afford some degree of protection against the development of REPE. I should like to ask Dr Davies and his colleagues whether there is a difference in PMVP according to age.

I agree with Davies et al that further insights into the physiologic mechanisms may lead to more specific treatments against REPE.

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Pulmonary Toxicity Following Exposure to Methylene Chloride and its Combustion Product, Phosgene

To the Editor:

The stripper strikes again! As we reported in the March 1992 issue of Chest, chemical paint removers containing methylene chloride are widely used in domestic and industrial settings where exposure to a heat source with conversion to phosgene is possible. We have now cured for an additional patient with nearly identical problems.

A 37-year-old man presented to the emergency room with complaints of shortness of breath associated with chest discomfort. His past medical history was unremarkable, and he was not a smoker. He had been using a paint remover consisting of 90 percent methylene chloride two days before coming to the emergency room.

He had been finishing a piece of furniture in a poorly ventilated garage heated with a kerosene heater. The emergency room evaluation included a chest x-ray film, which revealed extensive bilateral alveolar and interstitial infiltrates. Initial arterial blood gas analysis on room air revealed the following values: pH, 7.39; Pco2, 41 mm Hg; Po2, 50 mm Hg. The carbon monoxide level was 2.5 percent. The patient was treated with oxygen, and his symptoms improved during the next 45 to 72 h. A repeat x-ray film obtained three days later revealed marked but not complete resolution of the previously noted infiltrates. Oxyhemoglobin was 96 percent saturation on room air. At the time of discharge, the patient had a residual cough. Ten days later he was asymptomatic, and a chest x-ray film was normal.

This presentation of another patient soon after our previous report suggests that this problem occurs more frequently than we had previously suggested and may well be underreported. This certainly seems to be true in a suburban setting, where home furniture refinishing by nonprofessionals is epidemic due to current economic conditions.

We would like to again emphasize the need for complete medical evaluation following toxic exposures and for observation of the patient for a prolonged interval of time because of the potential for delayed clinical problems. We would also like to stress the need to carefully read warning labels when using paint removers containing methylene chloride and to avoid exposure to heat or open flame because of the added risk of phosgene production. Lethal concentrations have been reported within 5 to 10 min under test conditions.

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