time sequence and appearance of the abscess cavity before and after the procedure strongly suggest that this was the turning point in the patient's course.

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REFERENCES

To the Editor:

I must commend Drs. Safidar and Kraman on their imaginative use of the balloon catheter and lavage by fiberoptic bronchoscopy in the management of unresponsive lung abscess. The visible drainage and improvement noted on chest x-ray film soon after the procedure almost certainly indicate that the procedure was of therapeutic benefit in this patient's course. It is possible that the use of lavage in this manner enables the bronchoscopist to overcome the restrictions of the instrument's small channel and limited suction power. Since the treatment of a lung abscess progressing under conventional therapy may be surgical,† the use of this less invasive therapeutic tool appears warranted. In the case of a slowly resolving or stable abscess, however, conservative therapy, ie postponing the diagnostic bronchoscopic procedure until the patient is in optimal medical condition, seems advisable.

The therapeutic role of fiberoptic bronchoscopy in lung disease in several clinical situations remains to be defined. While Wanner et al observed in an uncontrolled series radiologic improvement following bronchoscopy for atelectasis, Marini et al found that bronchoscopy did not add to respiratory therapy alone in a randomized controlled study. Wanner's two cases of response of lung abscess to fiberoptic bronchoscopy were incompletely described and uncontrolled, and the remainder of the literature is scanty. The need for a prospective controlled trial to evaluate the risks and benefits of this tool in the frequently occurring clinical situation of lung abscess is obvious.

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Re-Expansion Pulmonary Edema

To the Editor:

Mahajan et al in their report in the February, 1979 issue of Chest, may be over-stressing the rarity of re-expansion pulmonary edema.

A previous report1 dealt with three episodes over a three-year period out of a total of 54 pneumothoraces, an incidence of nearly 10 percent. It has been my experience that if one routinely obtains chest radiographs in patients within four hours of intubation, re-expansion edema will be seen in varying degrees of severity in about 10 percent of patients. There seems little doubt that this complication is more likely to occur in patients with large pneumothoraces where lung collapse has been prolonged for periods of three days or more and where negative pressure has been applied to the pleural cavity. In two of the episodes in the above-mentioned report, the intercostal tube was connected to a Heimlich valve without the use of negative pressure.

With respect to pathogenesis, I would agree that increased alveolar surface tension is an unlikely factor, as it has been shown that pulmonary edema is not usually associated with abnormally high surface tension forces and that edema can develop in a degassed lobe without an air-liquid interface. The probable mechanism is not only a combination of increased permeability of pulmonary capillaries to hypoxic damage, as Mahajan and others, suggest, but also the effect on these capillaries of a sudden and large increase in negative intrathoracic pressure which occurs with rapid pulmonary expansion especially with the aid of suction applied to the pleural cavity. It has been demonstrated in dogs that a large increase in negative intrathoracic pressure drew fluid from pulmonary capillaries into parenchyma and, under excessive conditions, not only plasmin but red cells left the capillaries.

The condition is unlikely to affect the prognosis in otherwise healthy individuals. In the only two fatalities reported, both patients had other severe problems compromising their survival.

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6 Warren MF, Peterson DK, Drinker CK. The effects of heightened negative pressure in the chest, together with further experiments upon anoxia in increasing the flow of lung lymph. Am J Physiol 1942; 137:641
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To the Editor:

We greatly appreciate the interest of Dr. Bernstein in our report. Dr. Bernstein does not regard re-expansion pulmonary edema as a rare entity. According to his observations, radiologic edema occurs in almost 10 percent of patients with pneumothorax when the collapsed lung is re-expanded. This figure is based on his observation of 34 patients with pneumo-
thorax when he detected radiologic evidence of pulmonary edema in three. Dr. Bernstein's experience is quite different from our combined experience of over 50 years in pulmonary medicine. The available literature also supports our observations that re-expansion pulmonary edema, indeed, is a rare happening. It was probably a coincidence that Dr. Bernstein observed re-expansion edema in three out of 34 patients treated between 1971 and 1974. Further, in another report in 1973 involving 18 patients with pneumothorax, Bernstein did not observe pulmonary edema in any patient. If the incidence of 10 percent was true, then one would have expected to see at least one case of pulmonary edema in these 18 patients. We beg to differ with Dr. Bernstein regarding the incidence of re-expansion edema being 10 percent and feel that it is in fact a rare entity.

With respect to pathogenesis, Dr. Bernstein's views are similar to ours. As mentioned in our original report, the exact pathogenesis of re-expansion pulmonary edema is not clear and is probably related to an interplay of multiple factors.

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To the Editor:

In their recent article, Mahajan, Simon and Huber (Chest 75:182-194, 1979) stressed the importance of a relatively prolonged period of pulmonary collapse in the development of re-expansion pulmonary edema. As they note, there has been only one case previously reported in which the preceding pulmonary collapse lasted less than three days. We wish to emphasize that the fact that re-expansion pulmonary edema can occur following short periods of collapse by reporting the following case.

**CASE REPORT**

A 76-white-old white woman was admitted to the hospital for elective resection of an abdominal aortic aneurysm. She gave a 40-year history of smoking one pack of cigarettes per day and had symptoms suggestive of both bronchitis and emphysema. Preoperative pulmonary function studies demonstrated forced vital capacity of 1.47 L (59 percent of predicted), FEV₁ 0.65 L (34 percent of predicted), and a residual volume of 2.93 L (282 percent of predicted). Abdominal blood gas levels preoperatively showed a PaO₂ 55 mm Hg, PaCO₂ 44 mm Hg, and pH 7.42. In spite of the obvious respiratory insufficiency, it was elected to proceed with surgery, as the aortic aneurysm was enlarging. Suitable anesthesia was provided via spinal anesthesia supplemented with light, general anesthesia. Ventilation during surgery was provided via an oropharyngeal airway. Breath sounds following airway placement were said to be bilaterally symmetrical. No complications were encountered during surgery and the patient was returned to the recovery room in satisfactory condition. A chest radiograph obtained at this time (four and one half hours after beginning of the procedure) demonstrated the left lung to be totally atelectatic presumably secondary to the endotracheal tube having been inadvertently positioned in the right mainstem bronchus. The endotracheal tube was repositioned and the patient begun on 5 cm of positive end-expiratory pressure with an FIO₂ of 40 percent. A chest film obtained two hours later showed marked reexpansion of the collapsed left lung with the appearance of indistinct vessel margins and peribronchial cuffing (Fig 1). These findings are radiographically compatible with the diagnosis of interstitial pulmonary edema and differ significantly from the pattern one would expect with the usual forms of postoperative discoid atelectasis. At this time, arterial blood gas levels (FIO₂ 40 percent) demonstrated a PaO₂ 115 mm Hg, PaCO₂ 56 mm Hg, and pH 7.38. The edema cleared progressively over the next several days and three days postoperatively the lung was entirely clear and had returned to its preoperative radiographic appearance. The patient suffered no demonstrable effects from this particular complication.

**DISCUSSION**

This case demonstrates the occurrence of presumed re-expansion pulmonary edema following a relatively short period of pulmonary collapse. Assuming that the endotracheal tube was incorrectly positioned initially, the duration of collapse would be no more than four and a half hours. Perhaps the appearance of pulmonary edema following collapse of such short duration is in some way related to the underlying emphysematous abnormalities in the lung. In the only previous case in which re-expansion pulmonary edema followed collapse of less than three days, the status of the patient's lungs (evidence of emphysema and/or bronchitis) was not commented upon.

An argument could be made that our patient's radiographic abnormality was, in fact, due to diffuse areas of atelectasis rather than to pulmonary edema and this would be difficult to entirely refute. As noted, however, radiographic appearance of indistinct vascular markings and peribronchial cuffing supports the diagnosis of interstitial pulmonary edema far more strongly than it does the diagnosis of atelectasis. The