believe that the authors have made a strong case for its use. Perhaps if they collect more data over a period of years and define more closely its indications and contraindications, one would be more inclined to accept their argument.

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A Postpericardiotomy and Postmyocardial Infarction Syndrome Presenting as Noncardiac Pulmonary Edema

To the Editor:

In the June 1991 issue of Chest, Kassanoff and Martirosian \(^1\) reported three cases of acute pulmonary edema, which, as they indicate, probably represented an autoimmune response associated with abnormal capillary permeability. Their valuable report should be further clarified.

First, since diastolic ventricular function was not measured, it may be incorrect to conclude that these were cases of "acute pulmonary edema within two to three days after cardiac injury that could not be ascribed to impaired ventricular function." Ventricular function, taken as a whole, must be measured as a whole; the measures like hemodynamics and ejection fraction are incomplete descriptors.

Second, I am curious about the title of the article and some of the discussion. This syndrome, which appears to be unique, is described in the title as "Postpericardiotomy and Postmyocardial Infarction Syndrome" with no basis other than a possible autoimmune response following cardiac injury with elevated sedimentation rates. To avoid misleading readers, perhaps they should have called their report something like "A Postmyocardial Injury Syndrome." That would avoid implying that this form of pulmonary edema is a component of what Dressler described (now quite rare) and what Engle and colleagues (cited by the authors) have carefully investigated—two classic syndromes that include some element of active pericardial involvement, conspicuously lacking in these three fascinating patients.

These remarks are made for clarification, rather than in criticism, of a very valuable report.

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Reference


To the Editor:

Our article was submitted with the title "A Postpericardiotomy and Postmyocardial Infarction Syndrome Presenting as Noncardiac Pulmonary Edema," which is the way the article is listed in the table of contents. Somewhat, the limiting adjective "A" was omitted from the title of the article itself. Unfortunately, I failed to make the necessary correction when I received the galley proof. The grammatical determiner "A" was intended to emphasize the point that the three cases presented a different type of postpericardial or postmyocardial infarction injury, certainly a variant from Engle's and Dressler's descriptions. Whether there is a common immunologic thread between the three entities remains to be elucidated.

Dr Spodick is quite correct in emphasizing the fact that ventricular function cannot be accurately determined without diastolic ventricular function measurement. The three cases, by necessity, were evaluated at the bedside; in the first case the ejection fraction was 60 percent on echocardiogram, and in the second and third cases the postoperative left atrial pressures were normal. Thus, on the basis of the measurements available to us and therapeutic observation, we felt it reasonable to assume that the pulmonary edema was not due to congestive heart failure. Our observations do indeed need clarification through careful laboratory study, but based on our clinical observations, as Dr Spodick emphasizes, we may be dealing with an entirely different entity, probably immunologic in origin.

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Extrapleural Hematoma as a Late Complication of Collapse Therapy for Tuberculosis

To the Editor:

We observed an apparently spontaneous extrapleural hematoma in a 65-year-old patient treated with extrapleural pneumothorax from 1948 to 1951 for right apical tuberculosis. A residual pleural thickening remained unchanged until 1987 when aceclofenac was given after a myocardial infarction. In January 1989 an extrapleural effusion suggestive of tuberculous empyema was diagnosed. Despite tuberculostatic therapy, the effusion was seen to have grown in December 1989 (Fig 1). Pleuropericardial decortication performed in March 1990 disclosed a clotted extrapleural hematoma that was negative for microorganisms and malignancy. Recovery and one-year follow-up were uneventful.

Figure 1. Chest x-ray film shows a large extrapleural effusion at the site of previous extrapleural pneumothorax.
Late complications of collapse therapy generally present as pyogenic, caseous, or mycotic empyema at the site of the previous pneumothorax. Hemorrhagic effusion associated with pleural malignant change has been reported. To our knowledge, extrapleural hematoma without malignancy has not yet been described.

Our patient's medical history excluded external violence or traumatic thoracentesis. Although prothrombin values were within the therapeutic range, transient overtanticoagulation most probably enhanced pleural bleeding. We believe that pleuropulmonary decortication was the appropriate treatment, suppressing the pocket that might have become infected by repeated centesis or tube thoracostomy and allowing full pulmonary expansion.

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REFERENCES

Increase of Intraocular Pressure during Nasal CPAP

To the Editor:

Nasal continuous positive airway pressure (CPAP) was first described by Sullivan et al and has proved to be of great value for the treatment of patients with obstructive sleep apnea (OSA). The therapeutic instent of nasal CPAP is due to its good results and security, with very few complications described. To our knowledge, secondary increase of intraocular pressure during treatment with nasal CPAP has not been previously reported in the literature. We present the case of a patient with OSA and glaucoma in whom the intraocular pressure increased during nasal CPAP treatment.

A 62-year-old obese man was studied because of daytime sleepiness, loud snoring, and nocturnal arousals with apnea episodes. Glaucoma in the right eye had been diagnosed, and he had received the pertinent treatment. Respiratory functional testing revealed a moderate obstructive ventilatory defect (FVC, 82 percent; FEV1, 56 percent; FEV1/FVC, 55 percent). Values obtained at blood gas analysis were as follows: PO2, 65 mm Hg; PCO2, 45 mm Hg; pH, 7.36. All-night polysomnography demonstrated severe OSA, with an apnea-hypopnea index of 40. Treatment with nasal CPAP was initiated at a pressure of 3 cm H2O and was then increased on the following nights until a pressure of 10 cm H2O was reached. The patient reported pain in the glaucomatous eye. The intraocular pressure in both eyes was measured at baseline and after 15 min of nasal CPAP at 10 cm H2O. An increase of 7 mm Hg in intraocular pressure in the glaucomatous eye was documented; no changes appeared in the healthy eye.

Nasal CPAP produces an increase in pressure in the oropharynx, keeping the airway open during sleep. The pressure of nasal CPAP can be transmitted through the esophagus and has been useful in reduction of gastroesophageal reflux. The relation between intrathoracic and intracranial pressure has also been studied in OSA. Transmission of intrathoracic pressure to the intracranial space occurs through the venous system or directly to the cerebrospinal fluid space via the thoracic vertebral foramina. Impairment of cerebral blood outflow across valveless veins and an increase in cerebrospinal fluid pressure can increase cerebral venous and intracranial pressures. The increase in intraocular pressure might be due to a similar pathogenetic mechanism associated with failure of the diseased eye to regulate the pressure.

To our knowledge, no other published studies corroborate our findings, but it seems that nasal CPAP treatment increases intraocular pressure in patients with glaucoma, which suggests that glaucoma should be considered a relative contraindication for nasal CPAP. Further evidence is needed to support this possibility.

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Therapeutic Pulmonary Artery Catheterization

To the Editor:

I read with interest in the June 1991 issue of Chest the article by Steingrub et al on therapeutic pulmonary artery catheterization. It is curious that groups 1 and 2, for whom the reviewers were "uncomfortable with care," had the lowest mortality (although not statistically significant) compared with the "comfortable with care" and "optimal management" groups. Why is this so? Does it imply that therapy makes no difference in chance of survival in these critically ill patients? Rather, I think it implies that (1) we need a better idea of what it is we measure and should measure; and (2) we need controlled studies to compare different therapies to determine optimal management.

First we may ask what does pulmonary artery wedge pressure (PAWP) measure? It is commonly used as a surrogate for fluid status. Yet Shipp et al showed that there is a poor correlation between PAWP and measured blood volume. Schuster and Haller showed that there is a poor correlation between PAWP and extravascular lung water. There was a better correlation when capillary permeability was included.

More important than cardiac output is oxygen delivery (the product of cardiac output, hemoglobin concentration, and oxyhe-