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 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

1 Kassanoff AH, Martirosian MG. Postpericardiotomy and post-myocardial infarction syndrome presenting as noncardiac pulmonary edema. Chest 1991; 99:1410-14

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. Somewhere, 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 acenocoumarol was given after a myocardial infarction. In January 1989 an extrapleural effusion suggestive of tuberculous empyema was diagnosed. Despite tuberculosiastic therapy, the effusion was seen to have grown in December 1989 (Fig 1). Pleuropulmonary 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.