

# Tension hydrothorax—an unrecognized danger

KEITH RABINOV, MYRON STEIN<sup>1</sup>, AND HOWARD FRANK

*From the Departments of Radiology, Medicine, and Surgery, Beth Israel Hospital, Boston, and Harvard Medical School, Boston, Mass., U.S.A.*

Shortness of breath and slight mediastinal shift are not uncommon in the presence of a large pleural effusion. It is not generally recognized, however, that in certain circumstances pleural fluid may accumulate under such greatly increased pressure that it results in gross mediastinal shift and irreversible respiratory and circulatory changes. The following is a report of such a case of fatal 'tension hydrothorax'.

## CASE REPORT

C.A., a 43-year-old woman, suffered increasingly severe dyspnoea, weakness, and right chest pain for two months.

The physical findings included marked shortness of breath with a respiratory rate of 40/min., pulse 120/min., evidence of a large right pleural effusion, and a temperature of 103° F. A blood serum protein determination showed albumin 1.4 g./100 ml. and globulin 4.7 g./100 ml.

A chest radiograph (Fig. 1) showed a large right pleural effusion. The mediastinum was in the midline. It had previously been displaced slightly to the left, but this was corrected on several occasions by thoracentesis, which revealed non-diagnostic serosanguineous fluid. Bronchoscopy, scalene node biopsy, and needle biopsy of the pleura were negative. Later attempts at thoracentesis failed and dyspnoea increased with a respiratory rate as high as 56/minute. During a radiological examination done in the supine position the patient suddenly became unresponsive, and pulse and blood pressure could not be obtained. Because of the gross displacement of the heart and mediastinum to the left (Fig. 2) it was concluded that the large pleural effusion was under greatly increased pressure. Resuscitative measures restored the circulation and consciousness temporarily. Arterial blood studies revealed  $P_{CO_2}$  70 mm. Hg and pH 7.21. The respiratory rate was 36/min. and tidal volume 260 ml. These findings were interpreted as being consistent with acidosis and extreme hypoventilation. An emergency thoracotomy was performed.

On entering the pleura, serosanguineous fluid was found under such pressure that it spurted to a height of 10 in. (25.4 cm.). The entire pleura was grossly thickened by tumour and fibrinous clot. The right lung was completely collapsed. A frozen section of the pleura revealed undifferentiated carcinoma. Several litres of free and partly loculated pleural fluid were removed, relieving the mediastinal displacement and compression of the opposite lung. However, blood pressure could not be maintained and the patient died shortly after the operation. Permission for post-mortem examination was not obtained. Analysis of the chest fluid revealed a protein value of 4.8 g./100 ml.

## DISCUSSION

Several mechanisms are thought possibly to be related to the accumulation of pleural fluid under a pressure estimated to be 10 in. (25.4 cm.)  $H_2O$ .

1. The normal pleural fluid dynamics present a modest osmotic-hydrostatic pressure gradient preventing the accumulation of pleural fluid (Agostini and Mead, 1964). Calculations indicate that a net pressure as high as 60 cm.  $H_2O$  may have been available for the formation of pleural fluid in this patient, largely due to the diminished serum albumin and the high protein value of the pleural fluid.

2. The entire pleural surface was involved by tumour, providing a very large surface for exudation of fluid.

3. Lymphatic drainage of fluid may have been compromised by the extensive tumour involvement.

Accumulation of a large amount of pleural fluid under such greatly increased pressure had two effects which became critical when fluid could no longer be removed by thoracentesis.

- a. Marked mediastinal shift and compression of the lung occurred, causing severe hypoventilation and respiratory acidosis.

- b. Pressure of the pleural fluid on the heart and great vessels inhibited central venous return, causing reduced cardiac output and circulatory collapse and metabolic acidosis.

<sup>1</sup>Present address: Department of Biological and Medical Sciences, Brown University and Rhode Island Hospital, Providence, Rhode Island

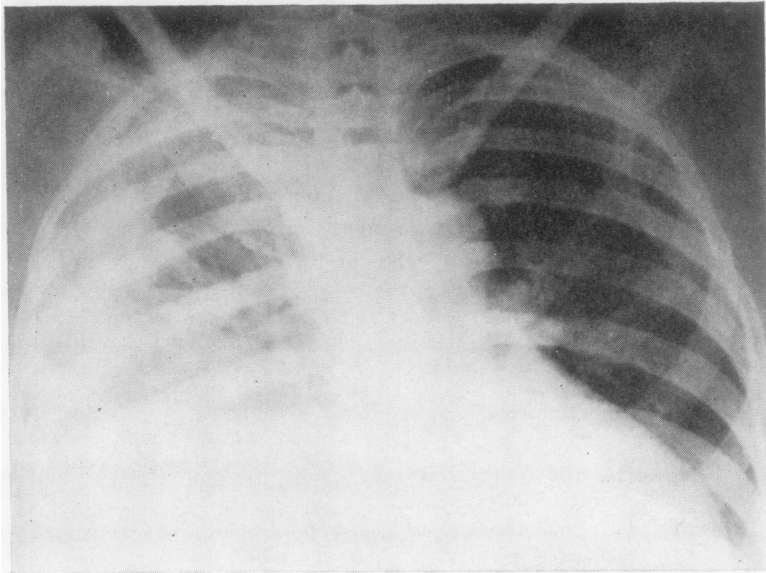


FIG. 1. *Postero-anterior chest film shows partly loculated hydropneumothorax on the right. The mediastinum is in the midline.*

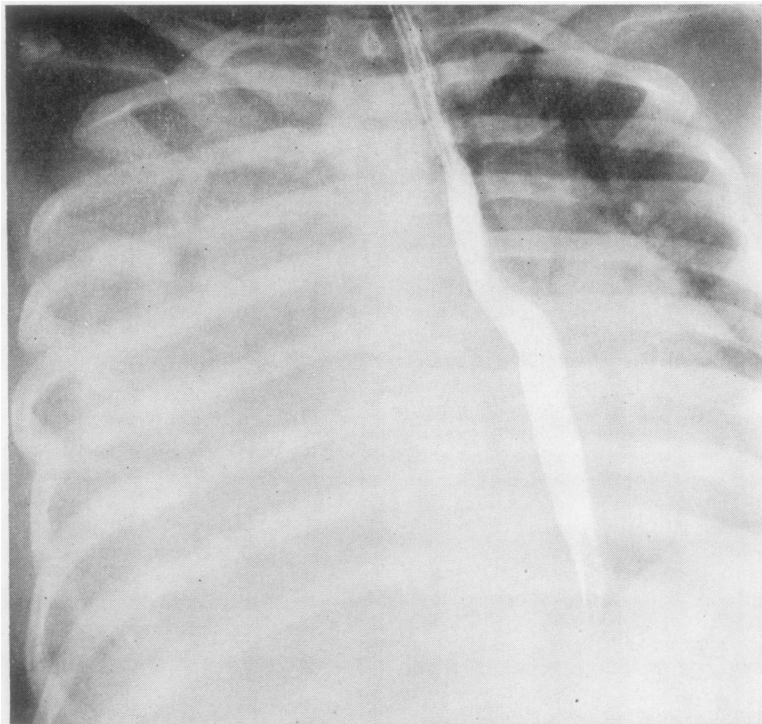


FIG. 2. *Supine chest film taken 11 days after the radiograph shown in Figure 1. There is a very large pleural effusion on the right with a marked shift of the heart and mediastinal structures to the left. Note the oesophagus outlined by barium.*

SUMMARY

A case is described in which a malignant pleural effusion accumulated under greatly increased pressure, causing sudden irreversible respiratory and circulatory collapse. The possible pathophysiological mechanisms are discussed.

Tension hydrothorax, although less common than tension pneumothorax, has similar adverse

physiological consequences. Radiological evidence of a marked mediastinal shift suggests the danger of tension hydrothorax and indicates the need for immediate therapy.

REFERENCE

- Agostini, E., and Mead, J. (1964). Statistics of the respiratory system. In *Handbook of Physiology, Section 3: Respiration*, vol. I, pp. 387-409. American Physiological Society, Washington, D.C.