

# PNEUMOPERICARDIUM COMPLICATING PNEUMOTHORAX THERAPY

BY

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## INTRODUCTION

While complete absence of the pericardium is a very rare abnormality, which is sometimes found during a post-mortem examination, we believe that partial deficiency resulting in a pleuropericardial communication is probably not quite as uncommon as the meagre literature on the subject would indicate.

Powell in 1869 first recorded a case of partial deficiency of the pericardium. In 1925 Moore found only sixty-four authentic cases of pericardial defect in the medical literature. Sixteen more examples have been noted by Grant (1926), Nicoli (1929), Watt (1931), Beck (1931), Egbert and Little (1935), and others. Diagnosis of this condition has been made during life on only three occasions. Ladd (1936), while operating for a diaphragmatic hernia in a two-year-old girl, found that her heart was devoid of its pericardium. Rusby and Sellors (1945) noticed a pneumopericardium when air was introduced in the left pleural sac as a preliminary to a thoracic operation, but "the full significance of this observation was not appreciated at the time," and the condition was elucidated on the operating table. Dahl (1937), while treating a case of pulmonary tuberculosis in a man aged 20, induced a left pneumothorax and on radiological examination found air in the pericardium as well as in the left pleural sac. He therefore concluded that there must have been a communication between the two cavities. This detection of a pneumopericardium during the course of artificial pneumothorax therapy is important, otherwise the possible existence of a pericardial defect remains unsuspected clinically. We report here two such cases. A third case, in which a pneumopericardium was simulated, is also described for purposes of discussion and differential diagnosis.

## CASE REPORTS

*Case 1.*—A man aged 48 was notified as a case of pulmonary tuberculosis in 1923, and received treatment in various sanatoria, the details of which are not available. In 1938 he had a slight haemoptysis. From May, 1940, to August, 1940, he was in an internment camp in Germany but spent most of the time in the camp hospital. Then he returned to England and carried on with his business till July 9, 1943, when he again began coughing up blood and was admitted to hospital under our care. The family history was not significant. On physical examination his nutritional state was found to be fairly good. His conjunctivae were pale, and he was somewhat breathless even on slight exertion. The physical signs were confined to the upper zones of both lungs, and radiography revealed extensive infiltration of the left upper zone, old scars in upper and lower lobes of the right lung, and some infiltration at the right apex. Repeated examination of the sputum for tubercle bacilli was negative. His haemoptysis soon ceased and in about three weeks his general condition improved; he remained afebrile and insisted on being discharged as he wished to get married.

Until June, 1947, he apparently remained well. On June 16 he was readmitted as an emergency because of a severe haemoptysis. He also complained that during the previous five days he had had two or three attacks of intense mid-sternal pain, described as a "tightness" radiating towards the left axilla and then down his left arm to the elbow, but later down to the finger-tips. At first the pain was brought on by physical exertion, but later it came on even when the patient was lying still. Initially it lasted one or two minutes, and he had a peculiar feeling that he was about to die. Later it lasted as long as 20 minutes.

*Examination.*—The patient was pale and anxious, with a temperature of 100° F. There was no clubbing of the fingers and the apex beat was in the normal situation. The heart sounds were distant, the rhythm was regular, the rate was 88 per minute, and there

were no significant murmurs. Blood pressure was 155/110 mm. Hg.

Movements of the chest were poor (maximum expansion  $\frac{1}{2}$  in.), but equal; vocal fremitus was increased on the left side anteriorly, and the percussion note was impaired over both apices. Breath sounds were very feeble at the right apex, and expiration was prolonged over the upper and middle zones on the left side, where there were numerous coarse râles.

*Radiology.*—An antero-posterior radiograph of the chest (Plate Ia) showed tuberculous infiltration of the upper and middle zones of the left lung and some scattered infiltration and fibrosis in the right lung.

The erythrocyte sedimentation rate was 9 mm. in the first hour (Westergren), and repeated examinations of the sputum were negative. Löwenstein culture remained sterile after six weeks. Blood Wassermann reaction was negative. Electrocardiograms were taken during an attack of pain and also a day later, and were thought to be indicative of coronary ischaemia. A blood count showed that the red cells had fallen to 2,720,000 per c.mm. of blood, and haemoglobin was 52 per cent, giving a colour index of 0.96.

As the disease was mainly unilateral a left artificial pneumothorax was induced on June 20, 1947, in order to try to control the haemoptysis. When with subsequent refills 1,000 c.cm. had been introduced, another radiograph was taken. The pneumothorax was confined to the left base but to our astonishment air was also detected in the pericardium (Plate Ib and c). However, in a few days the bleeding stopped and there were no further attacks of anginal pain. The pneumothorax was discontinued after the discovery of the pneumopericardium. Nevertheless the patient's general condition began to improve and he became afebrile. On July 24 another radiograph showed that the air at the left base and also that from the pericardium had been absorbed (Plate Ia).

At this stage it was decided to re-induce the artificial pneumothorax to observe the effect on the pericardium. Under radiological control, a needle was inserted in the same interspace as before, namely the sixth left intercostal space in the mid-axillary line (Plate Ib), and once again a left-sided pneumothorax was induced. Air was again seen in the pericardium (Plate Ic and d). Since the patient stated that he now felt perfectly well and free from pain, he insisted on his discharge.

*Follow up.*—This patient was seen again in September, 1947, and an electrocardiogram taken then was perfectly normal. By Dec. 1, 1947, he had gained over a stone in weight, felt very well, and had had no further attacks of angina.

*Case 2.*—A housewife, aged 34 years, consulted her family doctor in 1940 because of an haemoptysis. After full investigation a diagnosis of pulmonary tuberculosis was made, although at that time repeated examinations failed to reveal acid-fast bacilli

in the sputum. There was nothing noteworthy in the family history. Clinical and radiological examination showed that the disease was confined to the left mid-zone. On Sept. 2, 1940, a left artificial pneumothorax was induced. Subsequently weekly refills maintained a selective pneumothorax for four years (Plate IIIa and b). In July, 1944, she had a recurrence of haemoptysis. A further radiograph revealed that the tuberculous process had extended to the right side. The sputum had become positive. The left artificial pneumothorax was then abandoned and a right pneumothorax was induced. As soon as this was done the outline of the border of the cardiac shadow in the antero-posterior view began to change and a pneumopericardium became more evident after a sufficient quantity of air had been introduced into the right pleural cavity (Plate IIIc and d). This pneumopericardium remained evident in subsequent radiographs so long as the right pneumothorax was maintained. During the early part of 1947 the right artificial pneumothorax was allowed to obliterate. Plate IVa shows the state of affairs on March 6, 1947, there being no trace of air either in the pleural cavities or inside the pericardium.

#### DISCUSSION

*ANATOMY.*—The pericardium is like a conical money bag, the mouth of which embraces the roots of the great vessels. It is a fibroserous sac situated behind the sternum and the cartilages of the third, fourth, fifth, sixth, and seventh ribs on the left side. Anteriorly only a small portion of the pericardium comes into direct relationship with the left half of the lower portion of the body of the sternum and the medial ends of the fourth and fifth left costal cartilages. The greater part of the pericardium is separated from the anterior thoracic wall by the lungs and pleurae, and in a child, the lower extremity of the thymus. Posteriorly it rests upon the bronchi, the oesophagus, the descending thoracic aorta, and the posterior portions of mediastinal surfaces of the lungs. Laterally it is at first intimately in contact with the pleurae and then comes into relationship with the mediastinal surfaces of the lungs. The phrenic nerve and vessels pass between the pericardium and the pleura on either side. Inferiorly, it rests on, and is partly fused with, the central tendon and muscle fibres of the left side of the diaphragm.

The pericardium is composed chiefly of tough fibrous tissue lined with a serous membrane consisting of flattened cells, the latter being invaginated by the heart. Consequently the serous pericardium is divisible into a visceral and parietal portion. The visceral portion covers the heart and the great vessels, subsequently becoming continuous

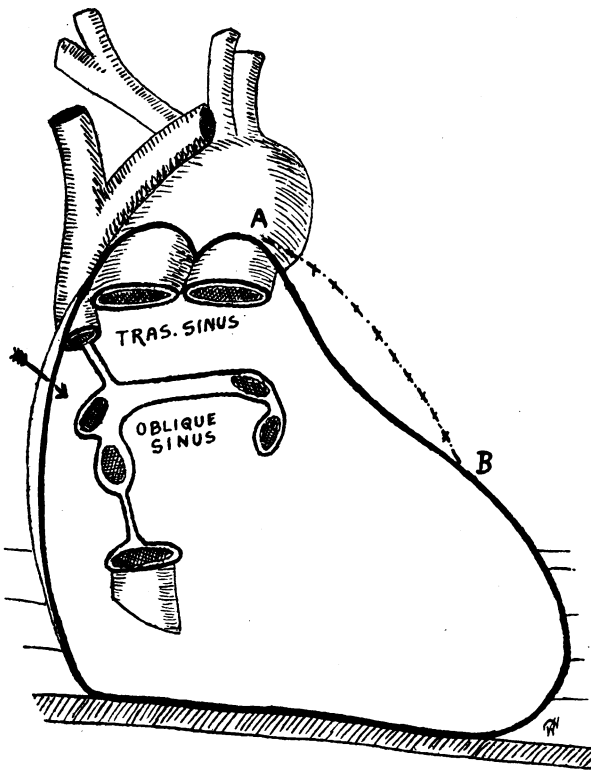


FIG. 1.—Pericardium, showing transverse sinus and oblique sinus.

with the parietal layer. Thus the transverse sinus and the oblique sinus are formed (Fig. 1).

**EMBRYOLOGY.**—The pericardium is developed from the mesodermal thickening at the cephalic end of the oval plate when the embryo is 1.25 mm. This thickening rapidly canalizes to form the intra-embryonic coelom. A little later the posterior end of this cavity opens into the extra-embryonic coelom and a portion of its anterior end is destined to become the future peritoneum.

When the simple tubular heart of early embryonic life bends on itself to form an S-shaped loop, constrictions appear in it which divide it into the sinus venosus, the primitive auricle and ventricle, bulbus cordis, and truncus arteriosus. It will be recalled that the duct of Cuvier on each side is formed by the union of the anterior and posterior cardinal veins, which drain into the sinus venosus in addition to the more medially situated vitelline and umbilical veins (Fig. 2).

When the embryo is about 4 mm. long a groove appears in the ventral wall of the pharynx. This develops into the laryngotracheal tube. From the

caudal end of this tube the right and left lung buds arise. At this stage the pericardium is comparatively a very large cavity, which is in communication with the peritoneum, and, as the lung buds expand they come into contact with the pleural canals, which ultimately become the pleurae (Fig 2). The opening in the pericardium leading into the pleural canal is the iter venosum of Lockwood, the final closure of which will separate the pleural and pericardial cavities when the embryo is about six weeks old. This closure is said to occur owing to pressure on the stretching duct of Cuvier and by the pleural canal being pushed by the growing lung tissue (Fig. 2). Complete or partial failure of this mechanism results

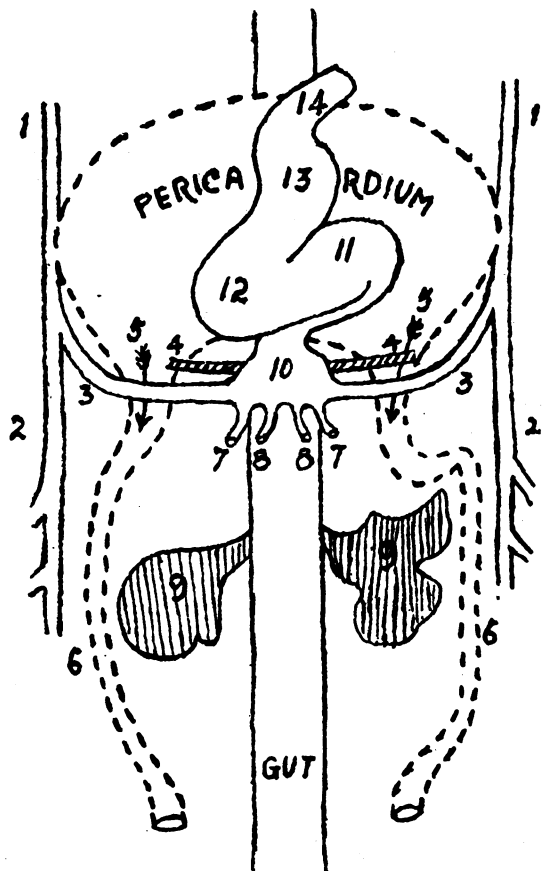


FIG. 2.—Pericardium: 1. Anterior cardinal vein. 2. Posterior cardinal vein. 3. Duct of Cuvier. 4. Septum transversum. 5. Arrow in the pleuropericardial channel (= iter venosum of Lockwood). 6. Pleural canal. 7. Umbilical vein. 8. Vitelline vein. 9. Lung. 10. Sinus venosus. 11. Primitive auricle. 12. Primitive ventricle. 13. Bulbus cordis. 14. Truncus arteriosus.

either in the absence of the pericardium or the persistence of a pleuropericardial opening. Keith speaks of a "patency of the iter venosum of Lockwood," apparently due to the lung bud somehow growing within it and preventing its closure. This theory is well supported by Rusby and Sellors' case of a 19-year-old girl who had a left bronchogenic cyst, a portion of which lodged snugly in the opening on the left side of the pericardium. Risel ascribed it to a persistence of the neurenteric canal. McGarry thought that at some stage an injury might cause a general disturbance of the coelom, resulting in multiple abnormalities, which is what was discovered in the three cases that he observed. Further, he attributed the preponderance of left-sided lesions, as compared with those on the right, to the asymmetry of the liver. Risel's theory, however, makes no attempt to explain this preponderance. There are only four instances of a right-sided deficiency in the literature, and with the exception of Egbert and Little's case, where the right side of the pericardium was missing, the other three showed a deficiency also on the left side. (We believe that our Case 2 is the second recorded example of a right-sided lesion alone.)

The duct of Cuvier and the cardinal veins on the left side normally undergo an atrophic change. In the adult they are represented by the highest left intercostal vein, the so-called ligament of the left vena cava (= the vestigial fold of Marshall), and the oblique vein of Marshall. On the right side the same embryonic structures do not atrophy but develop into superior vena cava and azygos vein. The theory of vascular atrophy put forward by Perna and later by Plaut visualizes that, if the left Cuvierian duct atrophies prematurely, then the development of the pleuropericardial region may get arrested owing to deficient blood supply, resulting in the iter of Lockwood remaining patent. On the other hand the development of large structures such as the superior vena cava and azygos veins cannot result in defective blood supply and therefore only exceptionally can a defect on the right side of the pericardium occur. The size of the pleuropericardial opening will depend on the stage of development reached at the time when the left Cuvierian duct atrophies.

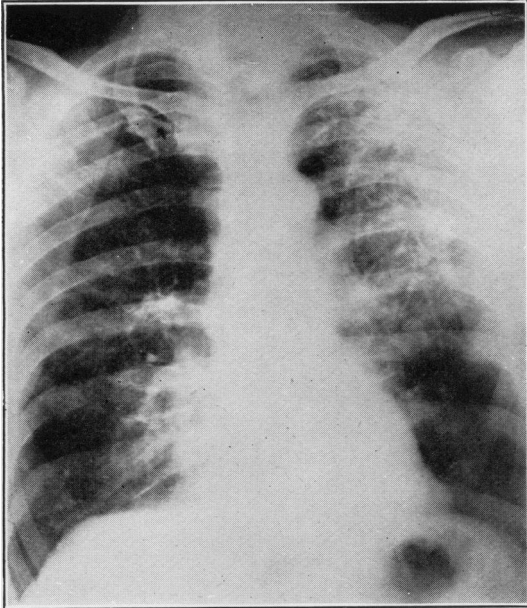
A communication between the pericardium and the pleura may occur as a result of a tuberculous gland ulcerating between the two sacs. Prof. M. J. Stewart in a personal communication informs us that "as a complication of tuberculous pleurisy or tuberculous pericarditis the condition must be very rare." As there has been no trauma to the pericardium while inducing artificial pneumo-

thorax, a congenital defect seems to be the most likely explanation of the pneumopericardium occurring in the two cases reported here. Incidentally the substernal pain, which was one of the dominant clinical features of Case 1, is of great interest. It is tentatively suggested that intermittent herniation of the myocardium through the pericardial defect producing local ischaemia might explain this symptom. In this connexion Boxall's case (1887) may be cited of a 28-year old woman who died three days after delivery, and the heart was found to have herniated through a partial deficiency of the pericardium.

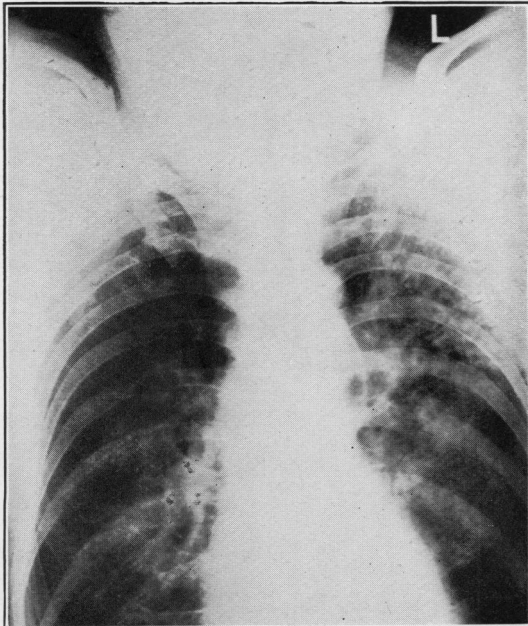
The pneumopericardium in the second case made its appearance only when an artificial pneumothorax was induced on the right side. This meant that a pleuropericardial communication existed on the right side alone—a most unusual state of affairs, as we have already noted. Furthermore, the air in the pericardium appeared at the left border of the heart in practically the same situation as in Case 1. This requires an explanation. In Fig. 1 the arrow indicates the approximate position of a congenital pericardial defect when it occurs on the right side. In the cadaver if one injects air at this point, which is just posterior to the right phrenic nerve, the air first travels over the anterior surface of the heart and then to the left. This is readily understandable for it is due to the reflections of the serous pericardium. In the living subject the relationship of the sternum and the costal cartilages to the pericardium, prevents the latter from expanding anteriorly. Thus the air passes to the left of the heart. In a silhouette of the heart the pericardium will be seen bulging between the points A and B as shown by the interrupted line in Fig. 1. It will be noticed that this is the only portion of the pericardium which can be pushed out by a relatively small pressure such as one uses in collapse therapy. A very much greater pressure is required to cause the pericardium to assume a globular shape, as might be seen in cases of artificial pneumopericardium, the appearances of and indication for which have already been described by one of us (Ellman, 1945) in connexion with tuberculosis pericarditis.

The existence of a congenital pleuropericardial communication can be inferred clinically only from radiological appearances of pneumopericardium in the presence of a pneumothorax. As we have seen, the air soon absorbs if refills are discontinued and a pneumopericardium no longer remains visible. However, it is sometimes extremely difficult to appreciate the significance of certain linear shadows seen at the left border of



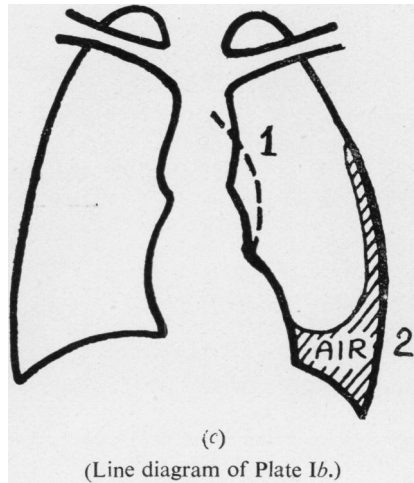


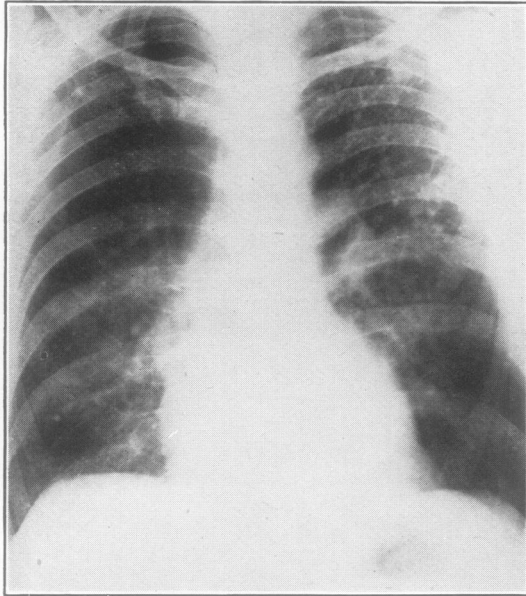
(a)



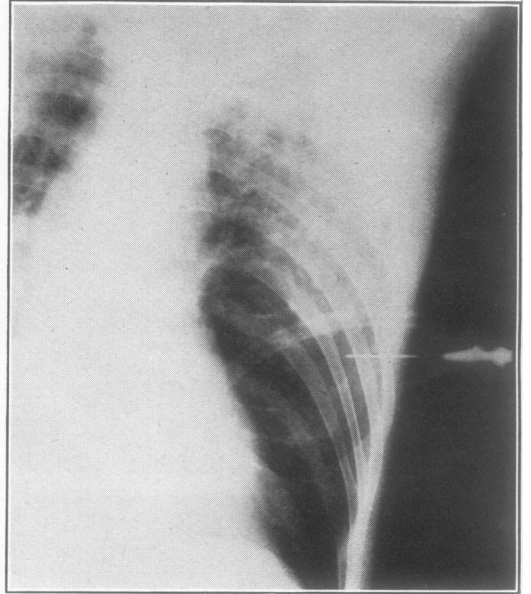
(b)

PLATE I.—Case 1 (a) Antero-posterior radiograph of the chest, taken in June, 1947, showing tuberculous infiltration of upper and middle zones of the left lung and some scattered infiltration and fibrosis in the right lung. (b) Showing air in the pericardium. (c) Pneumopericardium (1), and small basal pneumothorax (2).

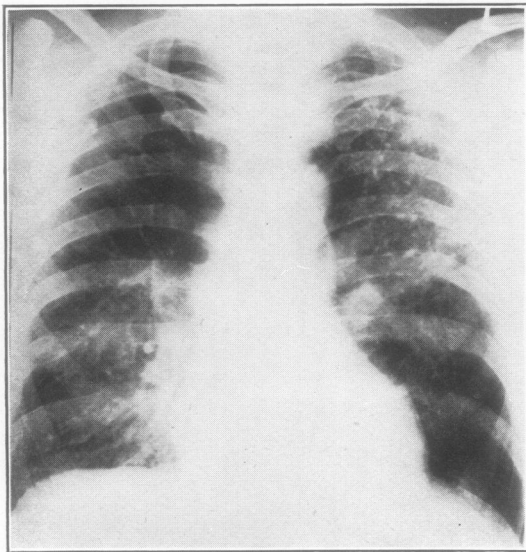




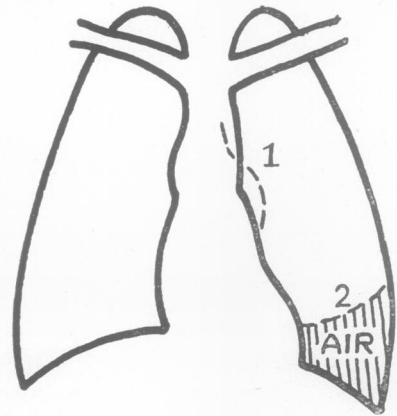
(a)



(b)



(c)

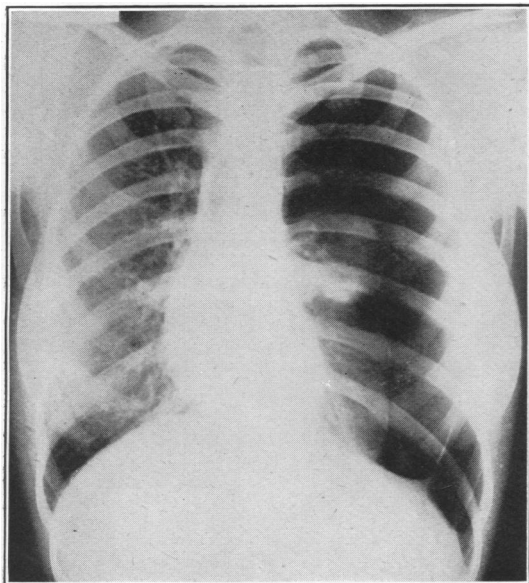


(d)

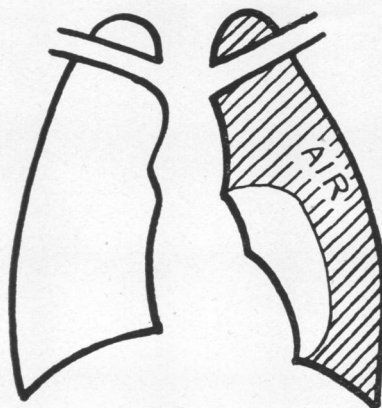
(Line diagram of Plate IIc.)

PLATE II.—Case 1 (a) Radiograph taken on July 24, 1947, showing that air at the left base and from the pericardium had been absorbed. (b) Showing needle inserted into the sixth left intercostal space in the mid-axillary line. (c) Air again in the pericardium. (d) Pneumopericardium (1), and small basal pneumothorax (2).



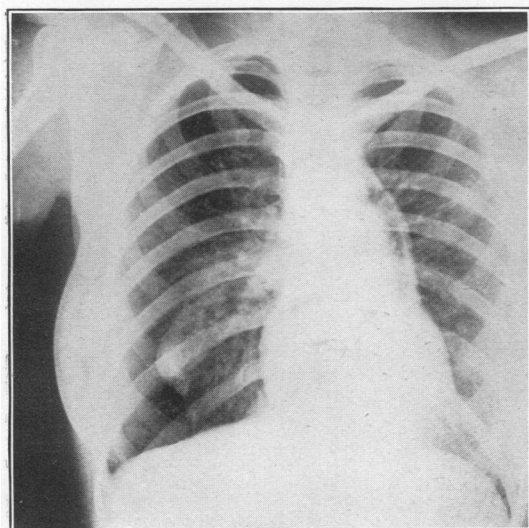


(a)

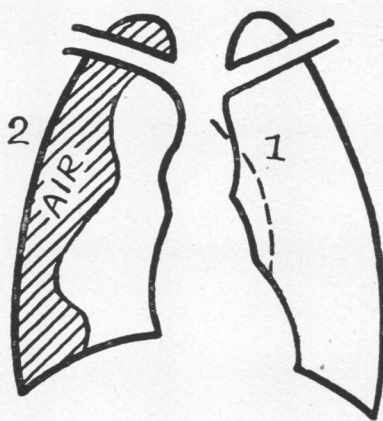


(b)

(Line diagram of P.a.e IIIa.)



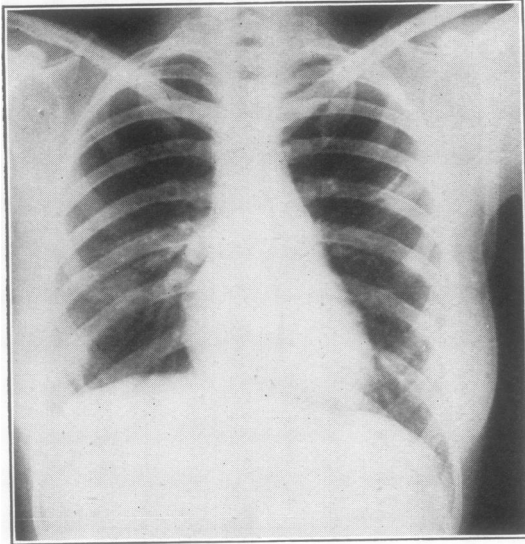
(c)



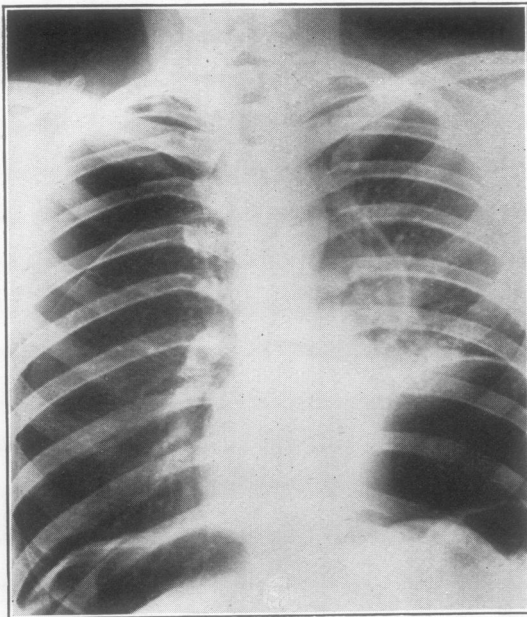
(d)

(Line diagram of Plate IIIc.)

PLATE III.—Case 2 (a) and (b) Selective pneumothorax after induction of a left artificial pneumothorax. (c) After induction of a right pneumothorax. (d) Pneumopericardium (1), and pneumothorax (2).

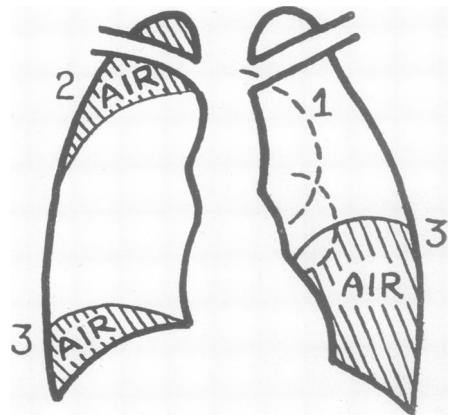


(a)



(b)

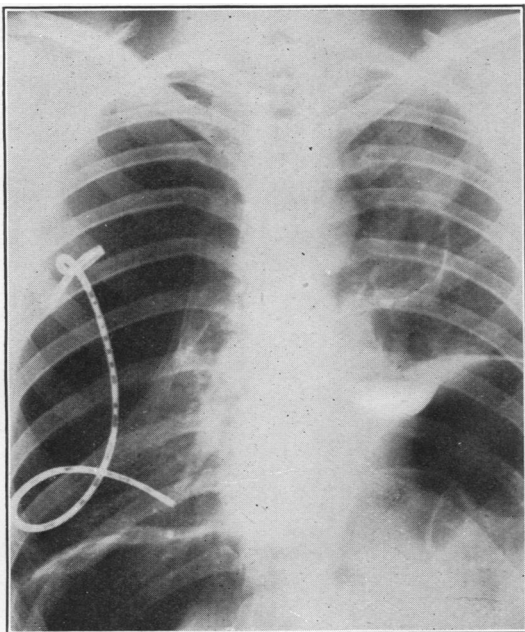
PLATE IV.—(a) Case 2: state on March 6, 1947. There is no air either in the pleural cavities or inside the pericardium. (b) Case 3: radiograph taken in February, 1946. (c) Line diagram illustrating main features in (b).



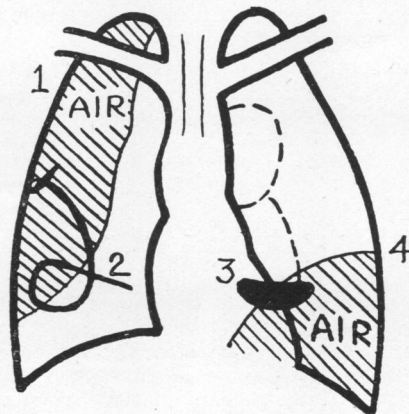
(c)

(Line diagram of Plate IVb.)



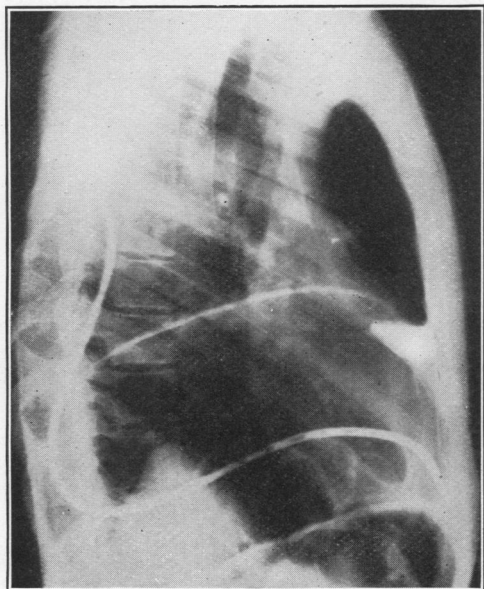


(a)

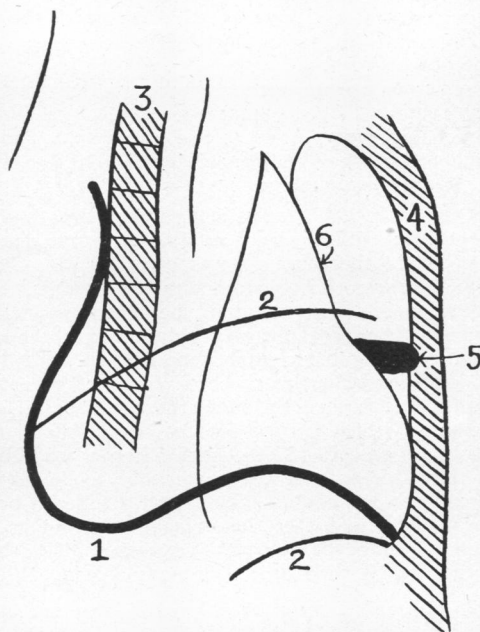


(b)

(Line diagram of Plate Va.)



(c)



(a)

(Line diagram of Plate Vc.)

PLATE V.—Case 3 (a) and (c) Radiographs taken with a Monaldi catheter in the mouth of the cavity and 15 ml. of iodized oil injected. (b) Right artificial pneumothorax (1); Monaldi catheter (2); lipiodol in "mediastinal hernia" (3); diaphragm (4). (d) Monaldi catheter (1); diaphragm (2); vertebral column (3); sternum (4); pool of lipiodol (5); pericardium (6).

the heart on radiological examination. In doubtful cases thoracoscopy is recommended, and if necessary, iodised oil should be injected into the opening to see whether it really communicates with the pericardium. The following case from Milford Sanatorium, which was kindly presented to one of us (P.E.) by Mr. W. P. Cleland, illustrates well the difficulties of differential diagnosis.

*Case 3.*—A steel metal worker, aged 22 years, had a left pleural effusion in May, 1942, and subsequent investigations led to a diagnosis of pulmonary tuberculosis. Treatment on general lines improved his condition sufficiently for him to be sent to a sanatorium. By February, 1943, he was able to walk three miles every day. His sputum, however, remained positive. He was then sent home and instructed to carry out certain prescribed exercises. Radiological examination on May 20, 1943, showed considerable infiltration of the left upper zone, and the right upper lobe contained scattered infiltration with denser lesions at the right apex. In March, 1944, right artificial pneumothorax was induced and adhesions were divided. In June attempts to induce a left artificial pneumothorax failed. On further consideration it was thought that the patient had a "mediastinal hernia." About a year later a right thoracoscopy was done by Mr. Maxwell, who considered that he had confirmed the presence of an anterior mediastinal hernia. A few days later the left phrenic nerve was crushed. Following this, treatment was continued by pneumoperitoneum. By February, 1946, there was noticeable improvement and the sputum became negative. Plate IV *b* and *c* shows the radiological findings at that time.

On Oct. 2, 1947, the patient's sputum was found to be positive and his general condition began to deteriorate. Right thoracoscopy was done by Mr. Cleland with injection of iodised oil into the "hernia." The following is a description of the thoracoscopic findings: "The neck of the opening appeared to be about 2 in. (5 cm.) in the sagittal plane by  $\frac{1}{2}$  to 1 in. (1.2 to 2.5 cm.) in the antero-posterior depth. The anterior border was formed by the sternum and costal cartilages, on which surface one could see the internal mammary vessels. The posterior border was formed by the superior vena cava, and, more to the left, the ascending aorta could be seen. In the superior border one could see the left innominate vein and its continuation into the superior vena cava. The lower border was formed by a membranous fold."

A Monaldi catheter was inserted into the mouth of the cavity, 15 ml. of iodised oil injected, and radiographs taken in various positions (Plate Va and b). Unfortunately during radiography the catheter slipped, and therefore there is no radiological indication of the position of the opening.

From a study of our own two cases and on anatomical and pathological grounds we suggest

that this is a case of an acquired interpleural communication rather than one of pericardial defect, for the position of the opening as observed by thoracoscopy is far removed from the situation where one would expect a congenital partial defect of the pericardium to occur (Fig. 1). In our two cases the linear shadow at the left border of the heart arises approximately at the aortic "knuckle" (Plates Ib and c, IIc and d, and IIIc and d), whereas in this case it seems to arise from a level beyond the upper limit of the pericardium (Plate IVb and c). Iodised oil has entered into two large spaces on the left side (Plate Va and b), but it has failed to reach the most dependent part of the pericardial sac (Plate Vc and d).

It is generally accepted that absence of the pericardium does not produce ill-effects by itself; nevertheless, in the presence of respiratory disease the subject of pleuropericardial defect runs greater risks. It is possible that a tuberculous pleural effusion may enter the pericardium and set up a tuberculous pericarditis in these cases. We suggest that minor congenital defects of the pericardium probably occur more frequently than hitherto suspected, for there is no means of making the diagnosis in healthy subjects. In order to assess the true incidence of the condition one would need to look especially for it in future post-mortem examinations. When pneumothorax therapy is begun in cases of pulmonary tuberculosis, the possibility must be kept in mind, and, in doubtful cases, investigation by thoracoscopy and injection of iodised oil should be carried out as described in Case 3. It may be advisable to abandon pneumothorax therapy if the existence of a pleuropericardial communication is established.

#### SUMMARY

Two unusual cases of what we believe to be a congenital pleuropericardial communication are described.

An explanation is offered to correlate this defect and the cardiac symptoms in one of the cases.

An experiment on the cadaver is described to show how air injected on the right side of the pericardium can be detected in a radiograph at the left border of the heart shadow.

The relevant anatomy and embryology of the pericardium is brought to mind and the various theories that have been put forward to account for the persistence of a pleuropericardial communication are recounted.

A discussion of a third case is included to illustrate difficulties in the interpretation of shadows

at the left border of the heart, which interpretation plays such an important part in the diagnosis of pneumopericardium.

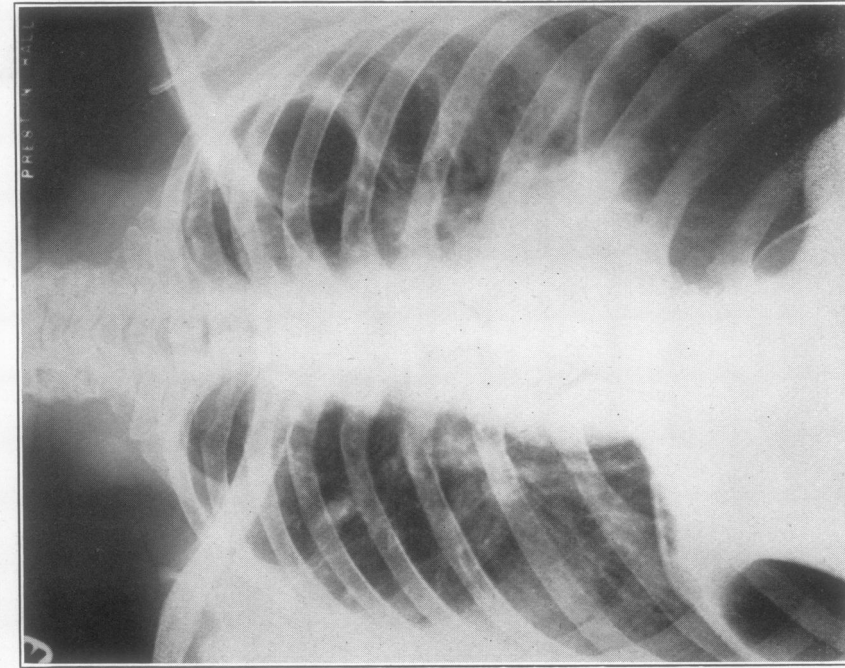
Employment of thoracoscopy for diagnostic purposes, and, if necessary, injection of iodised oil into the opening, is suggested.

We are indebted to Mr. W. P. Cleland for bringing Case 3 to our notice.

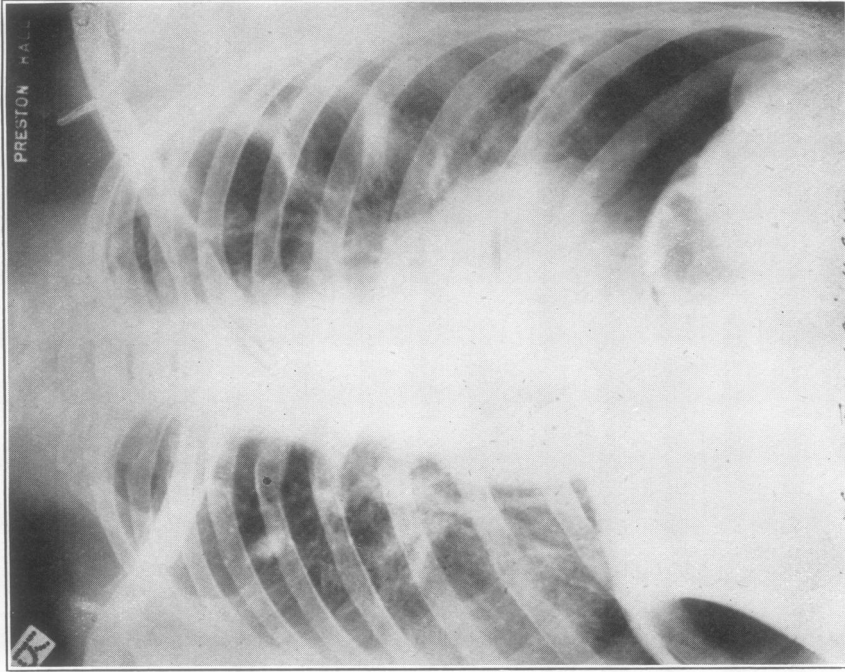
## REFERENCES

- Barsoum, H. (1935). *Brit. med. J.*, **2**, 666.  
 Bayer, R. (1938). *Z. Kreisl. Forsch.*, **30**, 411.  
 Beck, G. S. (1931). *Arch. Surg.*, **22**, 282.  
 Boxall, R. (1887). *Trans. obst. Soc. Lond.*, **28**, 209.  
 Dahl, E. (1937). *Med. Rev., Bergen*, **54**, 312.  
 Egbert, D. S., and Little, S. (1935). *Yale J. Biol. Med.*, **8**, 19.  
 Ellman, P. (1945). *Brit. Heart J.*, **7**, 147.  
 Grant, R. T. (1926). *Heart*, **12**, 371.  
 Kadin, M. (1939). *J. Mich. med. Soc.*, **38**, 503 (3 cases).  
 Keith, A. (1906-7). *J. Anat.*, **41**, 6.  
 Keith, A. (1933). *Human Embryology and Morphology*, 5th Edition, London, Arnold.  
 Ladd, W. E. (1936). *New Engl. J. Med.*, **214**, 183.  
 McGarry, R. A. (1914). *Anat. Rec.*, **8**, 43.  
 Moore, R. L. (1925). *Arch. Surg.*, **11**, 765.  
 Nicoli, L. (1929). *Bol. Soc. Med. Chir., Pavia*, **4**, 499.  
 Osgood, R., and Spector, B. (1941). *Amer. J. Dis. Child*, **61**, 1028.  
 Perna, G. (1909-10). *Anat. Anz.*, **35**, 323.  
 Plaut, M. (1913). *Frankfurt Z. Path.*, **12**, 141.  
 Powell, R. D. (1869). *Trans. path. Soc. Lond.*, **20**, 99.  
 Rusby, N. L., and Sellors, T. H. (1945). *Brit. J. Surg.*, **32**, 357-364.  
 Southworth, H., and Stevenson, C. S. (1938). *Arch. intern. Med.*, **61**, 223.  
 Watt, J. C. (1931). *Arch. Surg.*, **23**, 996.





(a)



(b)

**PLATE VI.**—(a) Postero-anterior radiograph taken on August 29, 1947. The suprahepatic effusion is visible as a dense opacity, which has not yet reached the inferior surface of the right hemidiaphragm. There is still an area of translucency between the effusion and the diaphragm. (b) Postero-anterior radiograph taken on September 14, 1947. The effusion now fully occupies the space between the superior surface of the liver and the right hemidiaphragm and spreads out over the inferior surface of the latter.