

# Herniation of the heart

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Five cases of herniation of the heart are described, of varying aetiology and presentation. In one, the pericardial deficiency is congenital. Two are traumatic—one with severe disruption of the rib cage, and one without. Two are post-operative—one early, and one late. Their aetiology, diagnosis and treatment are discussed. If the possibility of herniation is considered, the diagnosis may often be made from a plain chest radiograph. The treatment is operative, and may give relief from a critical circulatory embarrassment.

Herniation of the heart must occur through a hole in the pericardium. No such pericardial defect has been reported due to degenerative or other disease processes; the aetiologies of these herniations are, therefore, congenital, traumatic or iatrogenic.

Traumatic rupture of the pericardium, together with the 'bruit de moulin' of pneumopericardium, has been described since the first half of the nineteenth century (Bricheteau, 1844, quoted by Warburg, 1938). Recognition of the acute circulatory embarrassment which can be caused by herniation, and the possibility of relieving it, are fairly recent. Herniations following radical pneumonectomy were seen soon after intrapericardial ligation of the great vessels was practised, and Sharma, Bates, and Hurt (1959) described two cases.

We present five cases, one congenital, two traumatic, one early and one late post-operative. We hope that their comparison may help to define the signs necessary for early diagnosis.

## CASE REPORTS

**CASE 1 CONGENITAL HERNIATION** L. B., a girl, was first seen, aged 2 years, with a mild pectus excavatum which did not require correction. She was seen at three-yearly intervals, and at 7½ years review of the old films revealed a steady increase in the size of the heart, with progressive deviation into the left chest (Fig. 1). There was also a soft early systolic murmur, maximal in the pulmonary area, which had not been noted before. The second sound in the pulmonary area was late, but its components moved normally on respiration. The ECG showed a QRS axis at +120°. Cardiac catheterization gave evidence of a complex atrial septal defect with a 2:1 left to right shunt.

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This was corrected under cardiopulmonary bypass. The pericardium presented normally under a median sternotomy, but when it was opened longitudinally it was found to be absent on the left side, so that the pericardium and left pleura formed a single continuous space. The left ventricle lay horizontally across the diaphragm, its apex reaching almost to the axilla, and the left lower lobe was compressed by the heart to about one-third of its normal size. There was a free falciform margin anteriorly between the two cavities, a few millimetres deep. It arose at the left border of the inferior vena cava, and disappeared into the gap between the aorta and the pulmonary artery, which was larger than normal, so that the whole of both ventricles and the main pulmonary artery lay in the left chest. Posteriorly, both above and below, the free edges seemed to flatten out in the region of the left atrial appendage and pulmonary veins. The pericardium on the right side was normal.

When the right atrium was opened, a large sinus venosus type defect was found in the upper part of the septum, and below it was a large patent foramen ovale, with a thin residual half-valve, which was fenestrated. Both defects were closed together by a large woven Dacron patch, applied so as to include the mouths of the right pulmonary veins in the left atrium.

After this operation the child made an uneventful recovery, except that expansion of the left lower lobe remained incomplete. Now, six months later, it has improved, but the ventricular mass still lies horizontally in the left chest.

**CASE 2 TRAUMATIC PERICARDIAL RUPTURE WITH GROSS DISRUPTION OF THE RIB CAGE** A man aged 34 years, a driver of a car in a head-on collision, suffered a minor head and severe chest injury from impact with the steering wheel. At the Royal Hampshire County Hospital, Winchester, following intubation, positive pressure respiration, and transfusion totalling 2 litres, he regained consciousness and his circulation improved.

Five hours later, however, his condition deteriorated and he was transferred to the Chest Hospital.

On arrival his blood pressure was 120/70 mm. Hg and his venous pressure was slightly raised despite the very moderate transfusion. A chest radiograph showed extensive bilateral rib fractures and a displaced transverse fracture of the sternum at the level of the third space (Fig. 2). He was taken directly to the theatre. Both pleural cavities were opened through a transverse incision over the sternal fracture. There were 2.5 litres of blood in the right chest, and 1 litre in the left. Both lower lobes were badly bruised. The pericardium appeared folded and redundant and felt empty; it was found to be torn longitudinally on the left, from the origin of the pulmonary artery, anterior to the pulmonary veins, down to the diaphragm. The ventricular mass lay outside the pericardium in the left paravertebral gutter. The main pulmonary artery and both cavae were partially obstructed by the displacement of the heart, and by tension in the pericardial folds.

The heart was reduced with some difficulty into the pericardium, being now larger than its sac. The lateral tear was sutured, and a counter incision was made anteriorly, its edges being tacked to the epicardium at each side to prevent further herniation. The chest was

closed with bilateral drains, and the patient was treated for the next two weeks with anterior sternal traction and intermittent positive pressure respiration. He made an uninterrupted recovery and is now back at work full-time.

**CASE 3 TRAUMATIC PERICARDIAL RUPTURE WITHOUT DISRUPTION OF THE RIB CAGE** In July 1966 a man aged 23 years, driving alone, hit a telegraph post. On arrival at the Royal Hampshire County Hospital, Winchester, he was found to have a dislocated right hip and a right pneumothorax, though no rib fractures were seen (Fig. 3). The jugular venous pressure was not raised: the systolic pressure was 140/80 mm. Hg. The right chest was drained; the dislocated hip was reduced under general anaesthesia and he was put on traction.

Twelve hours after the accident he became pale and sweating and was seen by one of us (I. M.). His veins were full and the pulse was paradoxical. A further radiograph showed a large cardiac shadow (Fig. 4). He was transferred to the Chest Hospital. Herniation of the heart was suspected.

On arrival a radiograph showed that the pneumothorax was still present. This was corrected by apply-

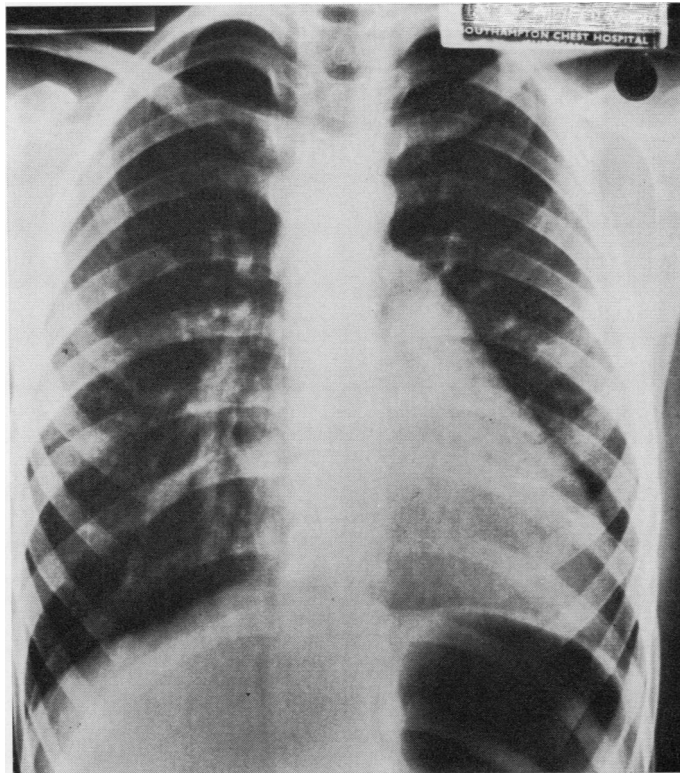
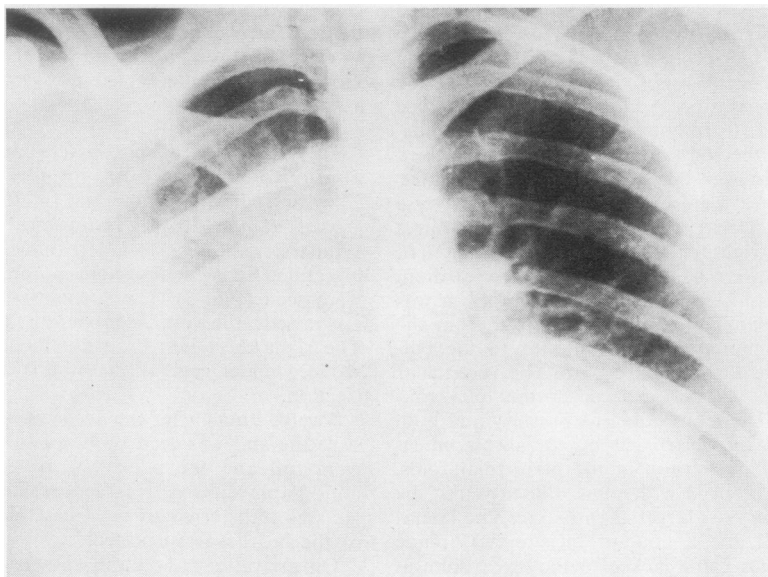
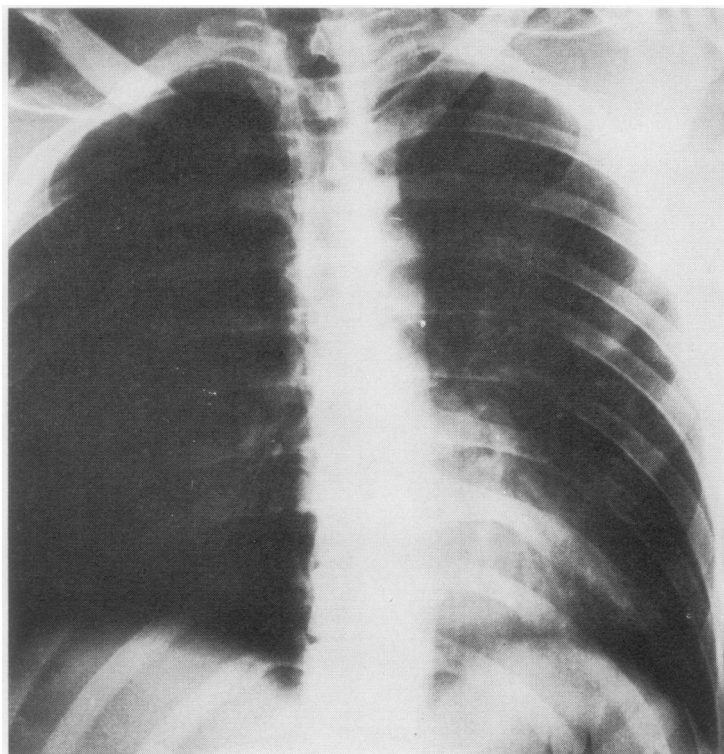


FIG. 1. Case 1, aged 7 years, before operation.



**FIG. 2.** *Case 2 before operation.*



**FIG. 3.** *Case 3 on arrival at the receiving hospital (circulation satisfactory).*



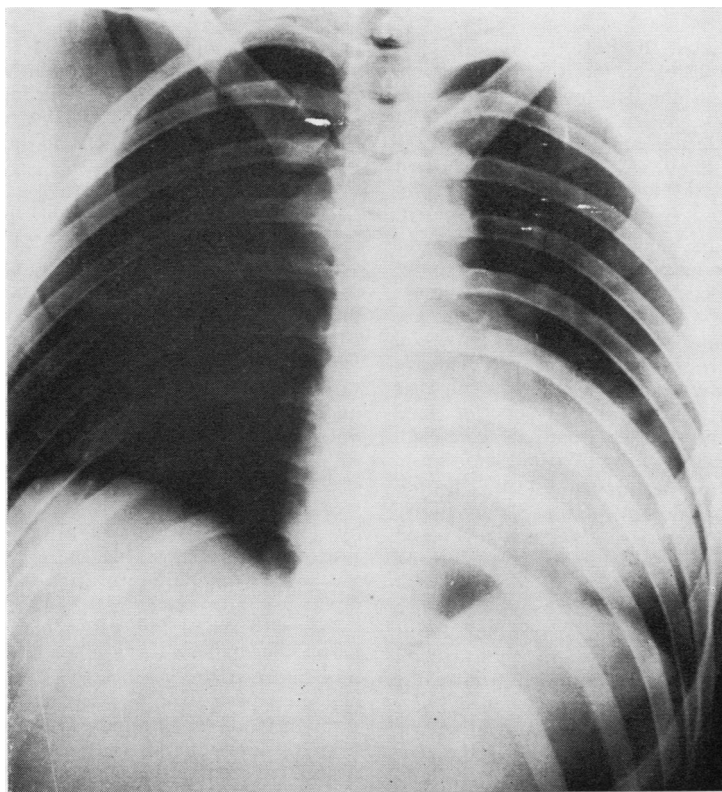


FIG. 4. Case 3, 12 hours later (veins engorged, pulsus paradoxus).



FIG. 5. Case 3, angiogram of left ventricle.

ing suction to the chest drain. His general condition improved and pulsus paradoxus disappeared. A further film showed a small residual pneumothorax, but the heart shadow was still displaced to the left. The heart sounds were faint; no murmurs were heard.

Over the next few days he maintained a good colour and stable circulation with spontaneous respiration. At this stage a diagnosis of herniated heart was thought probable, but while his condition remained stable, it was thought best not to disturb the traction on the hip by further investigation or operation. A therapeutic bronchoscopy was required on the third day. Old blood clot was aspirated from the left lower lobe, but the right side was normal. No bronchial rupture was seen.

Three weeks later his general condition remained fair, but the displacement of the heart was still present. The jugular venous pulse was raised 3 cm. with a prominent 'v' wave. The apex beat was impalpable, but a right ventricular heave could be felt to the left of the sternum. The heart sounds were faint, and there were still no murmurs. A review of the chest radiographs and ECGs showed a good correlation between the position of the heart and the ventricular axis of the ECG. Two films taken on the day of admission

showed the apex deviated to the left, but the right atrial shadow was still visible to the right of the spine. ECGs corresponding to these showed a ventricular axis of  $-75^\circ$  and  $-90^\circ$ . All subsequent films showed the whole heart in the left chest, with no atrial shadow to the right of the midline. ECGs at this stage showed a ventricular axis of  $+135^\circ$ . The earlier picture presumably corresponds to a partial prolapse of the heart.

Cardiac catheterization was undertaken. Angiograms showed the ventricular mass rotated through  $90^\circ$ , and deviated into the left chest, confirming the diagnosis (Fig. 5). There was a systolic gradient of 5 mm. Hg between the main pulmonary artery and its branches, probably due to compression. There was also evidence of gross incompetence of the tricuspid valve, in that the pressure in the right atrium and superior vena cava were the same as in the right ventricle waveform. He was thought to have ruptured tricuspid chordae.

Two weeks after injury he was taken to the theatre. Through a median sternotomy both pleurae were opened. The pericardium on the right was loose and empty. On the left it was torn longitudinally behind the phrenic nerve, and through this tear the ventricular mass protruded into the left paravertebral gutter. The superior vena cava was not obstructed, but the inferior vena cava was kinked by the pull on the pericardium. The pulmonary artery, which ran upwards and to the right from the displaced heart, was flattened by a tight band of pericardium just distal to its valve. The pericardial tear was found to extend from the pulmonary artery above, between the phrenic nerve and the pulmonary veins, down onto the diaphragm towards the inferior vena cava.

The heart was swollen and could not be reduced completely within the pericardium. A counter incision was made anteriorly, and the tear on the left was repaired. The free anterior edge of the pericardium was then tacked to the epicardium to prevent further prolapse. After reduction of the heart, the venous pressure was still very high. The tricuspid valve was palpated through a purse-string suture in the right atrial appendage. A papillary muscle was ruptured so that one cusp prolapsed into the atrium with each ventricular systole. It was felt, however, that he should be able to survive this degree of incompetence until a repair could be done under bypass as a planned procedure.

His condition remained fair for 24 hours but then deteriorated. He was returned to the theatre and the sternotomy was reopened. There was no evidence of tamponade. Emergency cardiopulmonary bypass was arranged to attempt a repair of the ruptured cusp. The valve was explored through the right atrium, and through a short transverse incision in the right ventricle near the atrioventricular groove. The papillary muscle of the anterior cusp had torn off near its origin and this site could not be identified exactly. At the second attempt, it was fixed in a position which gave a moderately competent valve. This was then reinforced by a Wooler annuloplasty. The combination gave a valve which was competent so long as the right

ventricle was not dilated (when the venous pressure was below 10 mm.), but in relative right ventricular failure the valve became grossly incompetent. Following these two operations his systemic circulation and renal function were poor, and he survived only 36 hours.

**CASE 4 HERNIATION FOLLOWING EARLY AFTER LEFT PNEUMONECTOMY** In October 1961 a man aged 37 years with an extensive carcinoma of the bronchus involving the pericardium underwent a radical left pneumonectomy. A 2-inch square of pericardium round the pulmonary veins was removed, and the veins were divided intrapericardially. The pericardium was closed loosely. The chest was drained to an underwater seal. During the night following this operation the patient's blood pressure dropped. There was no response to blood transfusion, and a radiograph was interpreted as showing a shift of the mediastinum to the left (Fig. 6). This was countered by clamping the drain and introducing air. The blood pressure improved a little, but it became obvious that the venous pressure was high, though pulsus paradoxus was not noted.

When the chest film was reviewed in the morning, herniation of the heart was diagnosed, and the patient was returned to the theatre. The pericardial sutures had given way, and the left ventricle had herniated through the hole. The right heart, and the left atrium, were obstructed and dilated. Reduction of the left ventricular mass produced an immediate improvement in the circulation. The pericardium could no longer be closed satisfactorily. The incision in it was therefore extended down to the diaphragm. The posterior edge and phrenic nerve were tacked down to the aorta, and the anterior free margin was tacked to the epicardium. Following this second procedure the patient made an uninterrupted recovery.

**CASE 5 CHRONIC LATE HERNIATION FOLLOWING MITRAL VALVOTOMY** In 1960 a 48-year-old woman (Fig. 7) underwent transventricular mitral valvotomy for rheumatic stenosis. This gave a good split without incompetence. Details of the pericardial closure are not recorded. She was well until the eleventh day, when she had paroxysmal nocturnal dyspnoea and a sharp interscapular pain. Next day the venous pressure was 15 cm.  $H_2O$ , and there was pulsus paradoxus. A chest radiograph showed an enlarged heart shadow. Pericardial effusion was diagnosed, but aspiration produced only 20 ml. of sterile bloody fluid. Next day the venous pressure was lower, and the systemic pressure higher, despite the onset of atrial fibrillation. Her discharge was delayed further by a deep vein thrombosis, but at six weeks she went home well and active.

She remained well for three years. Then, after six months of increasing breathlessness, she suddenly developed continuous retrosternal pain, dull in character, going through to the back, which lasted a week; and she fainted twice. She was readmitted for



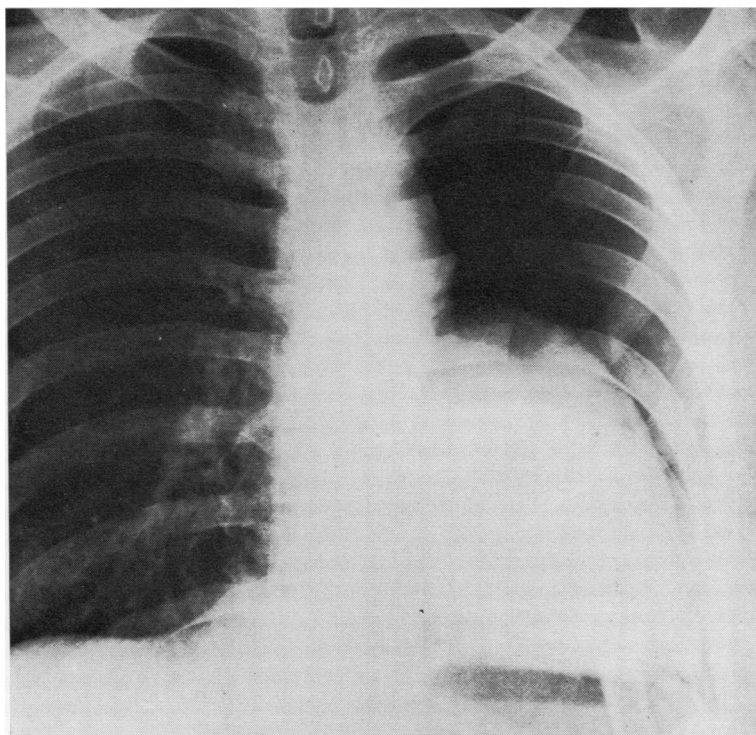


FIG. 6. *Case 4, 12 hours after left pneumonectomy (note absence of heart shadow to right of spine).*

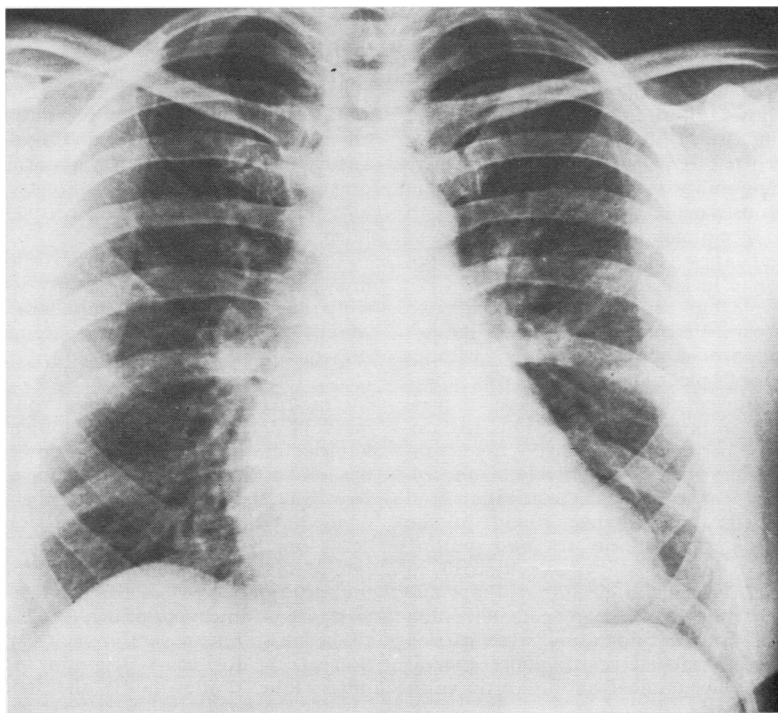


FIG. 7. *Case 5 before operation.*

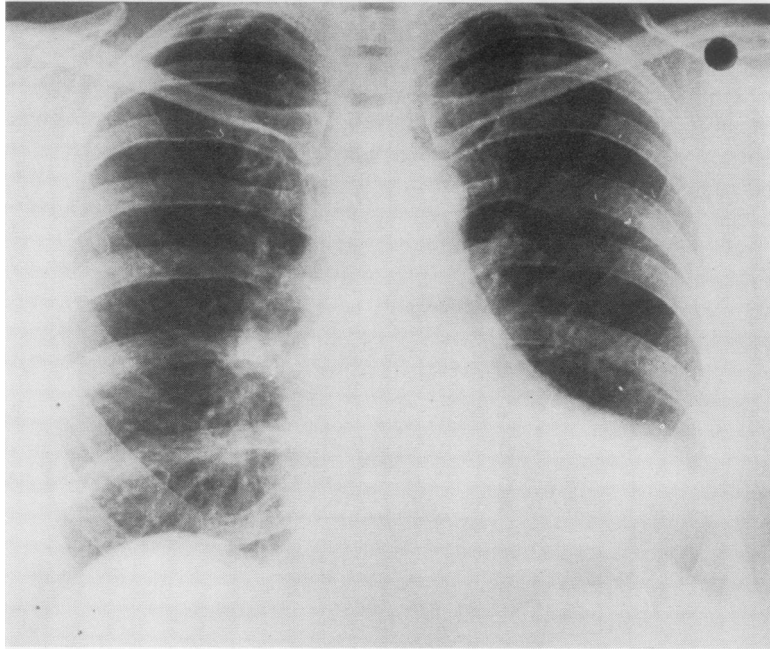


FIG. 8. Case 5 three years later (diagnosis—small apical aneurysm).

further investigation. She was in sinus rhythm with frequent ectopic beats. The venous pressure was not raised; the systemic pressure was 195/100 mm. Hg. The heart sounds were normal; there was an aortic systolic murmur but no mitral murmur nor opening snap. The ECG showed T inversion in  $V_5$  and  $V_6$  but was otherwise unchanged. Screening of the heart showed an enlarged left ventricular shadow, but there was no area of immobility or paradoxical movement (Fig. 8). The diagnosis made was a minor degree of aortic stenosis and a minimal aneurysmal dilatation of the left ventricular apex. Her chest pain was not thought to be cardiac in origin.

She returned home but two months later had two further attacks of pain, sharp and localized to the left sternal border, made worse by inspiration, but without the catch typical of pleuritic pain. During these attacks her ECG showed changes suggestive of pericarditis, which later reverted to the previous pattern. Recurrent attacks of pericarditis due to leakage from a left ventricular aneurysm were tentatively diagnosed. She was readmitted for cardiac catheterization and possible surgery. The only change found in the physical signs was the appearance of reversed splitting of the second heart sound, which was thought to denote prolonged left ventricular ejection, perhaps due to myocardial ischaemia. Left heart catheterization and left ventricular angiocardiography were carried out through the right brachial artery under general anaesthesia. The systolic gradient at the aortic valve

was only 10 mm. Hg, but the ejection curve was abnormal. It had a sharp initial rise, then an anacrotic notch, after which ejection was slow and prolonged. The angiogram showed an irregular outline to the left ventricle, there being an indentation in the posterior wall, possibly produced by pressure from without, and an irregularity of the apex. There was, however, no aneurysm and no significant abnormality of the mitral or aortic valve. The right coronary artery filled well and was large, but the left coronary artery was not filled at all.

The pre-operative diagnosis was left ventricular myocardial dysfunction, possibly ischaemic, and possibly due to localized tamponade. At operation in May 1964 the left chest was reopened through the previous incision. When the overlying lung had been freed, the apex of the heart was found to be protruding from the pericardium at the site of the original incision. Its edge formed a tough constricting ring, lying in the interventricular groove below, and crossing above the anterior descending coronary artery in front and the circumflex artery behind. The ring was divided and the heart was freed from adhesions but it could not be returned into the pericardium. The left side of the pericardium was therefore excised, leaving the sac widely open to the pleura. The phrenic nerve was tacked down posteriorly.

She made an uneventful recovery from this operation except for some delay in re-expansion of the lower lobe. Following it she has had no further

attacks of chest pain, and considerable improvement in her exercise tolerance has been maintained. Since operation her ECG has shown a complete left bundle-branch block which has obscured any evidence of improved myocardial blood supply due to the operation.

## DISCUSSION

### AETIOLOGY

*Congenital defects* Congenital pericardial defects are relatively uncommon. Ellis, Leeds, and Himmelstein (1959) collected 115 cases from the literature. Most are left-sided, and 80% are not associated with other cardiac anomalies. The defect varies from a small window to a complete absence of the pericardium. Most cause no symptoms, but small windows have been found in patients with intermittent chest pain, possibly caused by herniation of the atrial appendage. Moderate-sized, left-sided holes can clearly give rise to herniation and strangulation, and even sudden death (Sunderland and Wright-Smith, 1944). Extensive defects have been associated with sudden attacks of breathlessness, supposedly due to excessive displacement of the heart. The subject has recently been reviewed by Tubbs and Yacoub (1968).

*Trauma* Traumatic pericardial tears have been described at post-mortem following severe accidents (Kissane, 1952) but have only recently been diagnosed in living patients. Tears may follow blunt or penetrating injuries and occur whether or not the rib cage is intact. Typically, they are caused by the pericardium bursting on compression between the sternum and the spine. It is possible that such a burst may protect the heart from more serious damage, since symptomless herniation through a pericardial tear is reported (Warburg, 1938). Our second case shows that, in major trauma, injury may occur in both the pericardium and the heart. Apart from rupture of the chordae of either the mitral or the tricuspid valve, crush injuries of the heart may cause myocardial contusions which may be followed early or late by rupture of a ventricle or ventricular septum, or injury to a coronary artery or to the conducting system. Pericardial tears from blunt injury are almost confined to the left side.

*Post-operative herniation of the heart* This was first described following radical left pneumonectomy (Bettman and Tannenbaum, 1948; Yacoub, Williams, and Ahmad, 1968) and it was thought

that absence of the left lung allowed major displacement of the heart. Three of our cases illustrate the occurrence of herniae with the left lung present. Herniation may also occur following extensive pericardial resection on the right side (Higginson, 1953), if the apex manages to rotate through more than 90° past the ridge of the sternum. There is then no true strangulation, but acute distress is caused by the twisting of the cavae.

*HISTORY* Crush injury of the chest should raise the possibility of a herniated heart. Our second and third cases show that it may occur with or without obvious fractures of the rib cage. A pericardium opened at operation may, obviously, allow later herniation of the heart.

As with hernias anywhere in the body, herniation of the heart may cause no symptoms, or it may give rise to obstruction or to strangulation. Only one of our patients, the fifth, had pain clearly due to the herniation. In retrospect it seems likely to have been anginal in type, in view of the coronary obstruction seen on the angiogram.

There is a further point of interest in the histories of the two traumatic cases. Here, herniation is likely to have occurred at the moment of the accident, but in each case there was an interval of some hours before circulatory embarrassment was severe. A possible explanation for this delay may be found in another observation common to the last four cases. In all four, it was found impossible to close the pericardium after replacing the heart; and this is quite often so at the end of any heart operation. The increase in size of the heart is due to dilatation and takes some time to develop. The dilatation may be an inevitable result of the ventricle's escape from the pericardium, rather than a sign of distress in itself; one of the functions put forward for the normal pericardium is a limitation of ventricular filling in diastole. So, in a herniated heart, the displacement itself may cause little distress, until dilatation of the ventricle precipitates obstruction, and sometimes strangulation.

*PHYSICAL SIGNS* The only sign likely to be caused by displacement of the heart without circulatory distress is the absence of a palpable apex beat, and of cardiac dullness on percussion. When distress developed, the signs common to the last four cases were of low cardiac output, and raised central venous pressure, without any suggestion of overtransfusion. In case 3, and in case 5 on the twelfth post-operative day, pulsus paradoxus was also seen.



**ELECTROCARDIOGRAPHY** When satisfactory traces can be obtained, the ECG of a herniated heart may show a rotation of the ventricular axis corresponding to the anatomical displacement, as was the case in our third patient. The ECG changes in our fifth patient, who proved later to have intermittent coronary obstruction, were complex and difficult to interpret, but one might expect clearer evidence of myocardial ischaemia in some cases with strangulation.

**CHEST RADIOGRAPHS** The PA or AP chest film of a herniated heart shows a characteristic distortion of the heart shadow. The ventricular mass lies horizontally across the diaphragm in the left chest, giving a flattened boot-shape to its outline. When herniation is complete, the cardiac shadow is not seen to the right of the spine. If herniation is thought probable from the history, and from the immediate picture of circulatory distress, the chest film may give sufficient confirmation to justify immediate exploration.

**ANGIOCARDIOGRAPHY** In two of our patients, where operation was less urgent, angiocardio-graphy made or, in retrospect, could have made the diagnosis. In case 3, the diagnosis was confirmed, and evidence of an important degree of tricuspid incompetence was also provided. In case 5, a herniation had not been considered before operation. The angiogram was done with the possibility of ventricular aneurysm in mind, and this was eliminated. The positive findings were listed: irregular and variable filling of the apical half of the left ventricular cavity; an irregular filling defect of its posterior wall perhaps due to pressure from without; and an irregular ejection pattern in the aortic pressure wave-form. There was also an absence of filling of the left coronary artery. In retrospect, all these features were consistent with the herniation found.

**DIFFERENTIAL DIAGNOSIS** Without reference to the history, the presenting physical signs of low cardiac output with a raised central venous pressure might equally be caused by congestive cardiac failure, by pericardial tamponade or by herniation. Congestive failure may not seem likely following an accident to a young person, but all three diagnoses are possible following heart surgery. If pulsus paradoxus is marked, it suggests tamponade rather than congestive failure. If it is not present, the response to short sharp transfusion may help, since, even in congestive failure, unless the central

venous pressure is already above 20 cm. H<sub>2</sub>O there is likely to be an increase rather than a decrease of cardiac output. The distinction between circulatory obstruction due to pericardial fluid and to herniation may be impossible to make on clinical grounds, but a plain chest film should give the clue, if the possibility of herniation is considered.

**TREATMENT** Once a diagnosis of herniated heart is made, if the patient's circulation is embarrassed, operation offers a good chance of relief. A choice must be made between closing the pericardium securely, to prevent displacement of the heart, and leaving it so widely open that even if it is displaced, strangulation cannot occur. Dilatation of the heart, discussed above, may make a primary closure impossible. If the pericardium on the left is to be closed to prevent displacement, a counter incision elsewhere may be necessary. It is best placed anteriorly, where the sternum will help to prevent herniation. If the pericardium is left widely open, strangulation will not occur, but displacement alone may obstruct major vessels (Takita and Mijares, 1970). In these operations, the free edge of the pericardium was sewn down to the epicardium, an attempt to prevent displacement. Until adhesions form gravity probably keeps the heart in place more effectively than sutures. For the first few days after operation the patient must not lie on the open side.

In traumatic cases, injury to the heart must also be sought, and treated where possible. In our third case, a tricuspid valve replacement at the first operation would have given the patient a better chance of survival.

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