Cardiac herniation following intrapericardial pneumonectomy

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Deiraniya, A. K. (1974). Thorax, 29, 545–552. Cardiac herniation following intrapericardial pneumonectomy. Cardiac herniation is a rare and catastrophic complication of intrapericardial pneumonectomy. Untreated it is invariably fatal. This paper reports three cases of cardiac herniation following intrapericardial pneumonectomy. In two cases massive haemorrhage complicated the cardiac herniation. All three cases were re-explored with two immediate survivals. The diagnosis, aetiology, haemodynamic effects, and management of this complication are discussed, and previously reported cases are reviewed.

Cardiac herniation is a rare, highly lethal complication of intrapericardial pneumonectomy, demanding urgent treatment. The condition presents in the immediate or early postoperative period. Cardiovascular collapse is invariably present. Elevation of the jugular venous pressure and cyanosis in the drainage area of the superior vena cava are frequently noted. Diagnosis rests on an awareness of this condition, its clinical manifestation, and radiological examination.

This paper reports three patients who developed cardiac herniation following intrapericardial pneumonectomy, in two of whom massive haemorrhage complicated the herniation. A tear at the left aspect of the inferior atrio caval junction occurred as a result of cardiac herniation into the right pleural cavity in one case, and in the other the apex of the left ventricle was perforated by a fractured rib following a left pneumonectomy. A survey of the literature has not revealed similar cases. The predisposing factors and methods of prevention and treatment are discussed and previously reported cases are reviewed.

CASE REPORTS

Case 1. F. M., a 49-year-old mining inspector, was admitted to hospital on 16 March 1970 following the discovery of a hilar opacity on follow-up examination.

In August 1968 a right nephrectomy was performed for renal carcinoma. His postoperative course was satisfactory. Periodic six-monthly follow-up examinations including chest radiographs were normal until January 1970, when he was referred back with retrosternal pain and flatulence. Chest radiography revealed an isolated right hilar mass. Tomography showed a well-defined opacity intimately related to the lower lobe bronchus. The appearances were thought to be suggestive of a primary lung neoplasm. Barium meal showed a gastric ulcer.

On admission he was a fit-looking, obese, normotensive man with a well-healed nephrectomy scar. A mass 2 × 2 cm was palpable in the right supraclavicular fossa. A chest radiograph on admission showed enlargement of the hilar opacity. Bronchoscopy was normal and histological examination of the excised supraclavicular mass showed a neurolemmoma.

Thoracotomy was performed through the bed of the sixth rib. A lobulated yellowish tumour replacing the hilar lymph nodes and the lymph nodes along the lower and middle lobe bronchi was encountered. Frozen section showed metastatic renal carcinoma. A right intrapericardial pneumonectomy was performed. The pericardial defect measured 6 × 6 cm. This was not closed. A tube drain was inserted and the chest was closed. Shortly after his return to the ward the patient became restless. Intense cyanosis about the face and neck and hypotension were noted. Ventilation through an endotracheal tube did not materially improve the hypotension or cyanosis. The chest tube which had been inadvertently connected to the suction apparatus, rapidly filled with blood and asystole ensued. The wound was speedily re-opened. A massive haemothorax was evacuated. The heart had herniated through the defect into the right pleural cavity, its apex pointing towards the right paravertebral gutter. Blood was issuing from a 3 cm tear at the inferior atrio caval junction. After enlarging the defect and repairing the tear, the heart was replaced in the pericardial cavity. Despite rapid transfusion,
the inferior vena cava-right atrial junction where the tear was located (Fig. 1).

FIG. 1. Postmortem photograph showing the torn aspect of the inferior atrio caval junction. A metal probe can be seen passing from the inferior to the superior vena cava.

CASE 2. V. F., a 71-year-old woman, was admitted to hospital following the discovery of a left upper lobe opacity on a chest film. Physical examination was unremarkable. A chest film showed a large mass arising close to the hilum and extending into the anterior segment of the left upper lobe. Sputum cytology was repeatedly negative. At bronchoscopy no tumour could be seen. Operation was performed through a sixth rib posterolateral incision. A large tumour mass arising from the anterior segment of the left upper lobe and extending centrally to the hilum was found. The subaortic and left tracheobronchial glands were involved. A malignant tissue plaque involved the pleura overlying the aortic course of the recurrent laryngeal and phrenic nerves.

A radical intrapericardial pneumonectomy with deliberate division of the vagus and phrenic nerves was carried out. The resulting defect measured 7.5 × 7.5 cm. No attempt was made to close the defect. A pleural tube was inserted and the chest wound was closed. An immediate postoperative chest film showed elevation of the left leaf of the diaphragm (Fig. 2).

Sixteen hours postoperatively the patient became restless with profound hypotension and bradycardia. The jugular veins were engorged. An unusually vigorous pulsation was easily visible in the left second intercostal space in the mid clavicular line. An electrocardiogram showed ischaemic ST segment changes and complete heart block. A tentative diagnosis of cardiac herniation was made. A chest film showed an elevated diaphragm and dislocation of the heart into the left pleural cavity (Fig. 3).

An isoprenaline drip was started and the patient was returned to the operating theatre. On re-opening the chest it was apparent that the heart had herniated through the pericardial defect. The margins of the defect were firmly encircling the ventricle caudal to the atroventricular groove. The constricting ring was divided and the herniated heart was repositioned. A Dacron sheet was sutured with interrupted sutures to the edges of the defect. Isoprenaline support was required for 24 hours following re-exploration. Following discontinuation of the isoprenaline she developed nodal rhythm, which reverted to sinus rhythm after the administration of 0.6 mg atropine sulphate intravenously. She was discharged home on the fourteenth postoperative day.

CASE 3. G. P., a 65-year-old retired farm worker, presented with persistent cough and haemoptysis. Clinical examination was unremarkable. A chest radiograph revealed an opacity in the apical segment of the left lower lobe. Bronchoscopy was normal.

At operation an advanced tumour was found which had invaded the chest wall and the lung hilum. An intrapericardial left pneumonectomy was performed in which most of the pericardium overlying the left ventricle was excised together with segments of the fifth and sixth ribs. The resultant defect was not repaired. The seventh rib was fractured obliquely in the mid-axillary line by the self-retaining retractor.

Two hours after leaving theatre the patient became pale and hypotensive. The left hemithorax was dull to percussion, but as an intercostal drain had not been inserted there was no confirmatory evidence of massive intrapleural haemorrhage. A clinical diagnosis of slipped vascular ligature was made and the patient returned to theatre immediately. On re-opening the left hemithorax a massive haemothorax of approximately 2 litres was found and evacuated. The heart had dislocated into the left chest and the apex of the left ventricle was impaled on the sharp end of the fractured seventh rib. The heart was replaced in the pericardium and the ventricular tear was closed with interrupted sutures. The pericardial defect was bridged with a sheet of woven Dacron cloth and the chest was closed.

His cardiovascular status gave rise to little anxiety in the postoperative period, but the combination of
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**FIG. 2.** Immediate postoperative chest film—note elevation of left leaf of diaphragm.

**FIG. 3.** Chest film 17 hours postoperatively. Note the marked left shift of the cardiac shadow and the central tracheal air shadow. The upper border of the herniated heart can be seen in the left hemithorax.
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<th>Clinical Presentation</th>
<th>Operative Treatment = Reduction +</th>
<th>Outcome</th>
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<tr>
<td>Bettmann and Tannenbaum (1948)</td>
<td>M 53</td>
<td>L</td>
<td>Defect 5 x 5 cm. Rolling patient on to op. side</td>
<td>T</td>
<td>BP Cyanosis</td>
<td>Wide excision of pericardium</td>
<td>Recovery</td>
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<td>Kirchhoff (1951)</td>
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<td>Pericardial defect</td>
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<td>BP Acute SVC obstruction</td>
<td>Wide excision of pericardium</td>
<td>Recovery</td>
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<tr>
<td>Higginson (1953)</td>
<td>M —</td>
<td>R</td>
<td>Defect + aspiration of pneumonectomy space and inflation of left lung</td>
<td>T</td>
<td>BP</td>
<td>Wide excision of pericardium</td>
<td>Recovery</td>
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<td></td>
<td>M —</td>
<td>R</td>
<td>Small defect</td>
<td>24 hr postop.</td>
<td>Radiological evidence of herniation</td>
<td>Wide excision of pericardium</td>
<td>Recovery</td>
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<td></td>
<td>M —</td>
<td>R</td>
<td>Moderate defect</td>
<td>T</td>
<td>BP CXR = herniation</td>
<td>Wide excision of pericardium</td>
<td>Recovery</td>
</tr>
<tr>
<td></td>
<td>M —</td>
<td>R</td>
<td>Tracheal suction + very large defect</td>
<td>8 hr</td>
<td>BP Acute SVC obstruction, Transient improvement, Later deterioration and death</td>
<td>Nil</td>
<td>Died — cardiac herniation and incarceration</td>
</tr>
<tr>
<td>Dahlbäck and Nilsson (1956)</td>
<td>M 69</td>
<td>R</td>
<td>Pericardial slit + suction on chest drain</td>
<td>T</td>
<td>BP Transient improvement, Later BP and cyanosis</td>
<td>Closure of defect</td>
<td>Recovery</td>
</tr>
<tr>
<td>Neville and Jubb (1957)</td>
<td>M 53</td>
<td>L</td>
<td>Partial closure of defect and tilting patient onto op. side</td>
<td>1 hr</td>
<td>BP Acute SVC obstruction</td>
<td>Closure of defect</td>
<td>Recovery</td>
</tr>
<tr>
<td>Sharma et al. (1959)</td>
<td>M 66</td>
<td>L</td>
<td>Defect 7.5 x 6.5 cm</td>
<td>RR</td>
<td>Precordial pain, BP JVP CXR = cardiac displacement to left</td>
<td>Nil</td>
<td>Died. Pericardial defect twice op. size. Strangulated ventricles. R atrial enlargement. Congested liver</td>
</tr>
<tr>
<td></td>
<td>M 31</td>
<td>Lt</td>
<td>Moderate defect</td>
<td>RR</td>
<td>Precordial pain, CXR = dislocation of heart into left pleural cavity</td>
<td>Wide incision of pericardial defect borders</td>
<td>Death due to recurrent herniation</td>
</tr>
<tr>
<td>Walmsley (1961)</td>
<td>M 40</td>
<td>L</td>
<td>Defect 5 x 7 cm, Suction on chest drain</td>
<td>¶ hr postop.</td>
<td>BP Cyanosis, Dislocated apex beat, Transient improvement followed by relapse and VF, DC shock — SR</td>
<td>Wide incision of pericardium</td>
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</tr>
<tr>
<td>Gravel (1966)</td>
<td>M 54</td>
<td>R</td>
<td>Medium defect</td>
<td>RW</td>
<td>BP Acute SVC obstruction</td>
<td>Repair with Teflon mesh</td>
<td>Died two days postop.</td>
</tr>
<tr>
<td></td>
<td>M 59</td>
<td>R</td>
<td>Medium defect</td>
<td>4 hr</td>
<td>BP Acute SVC obstruction</td>
<td>Incision of pericardial defect borders</td>
<td>Died</td>
</tr>
<tr>
<td>Yacoub et al. (1968)</td>
<td>M 67</td>
<td>L</td>
<td>Large defect</td>
<td>RW</td>
<td>BP JVP CXR = cardiac dislocation into left pleural cavity, ECG = ischaemic changes</td>
<td>Repair of defect with pleura</td>
<td>Died</td>
</tr>
<tr>
<td>Author</td>
<td>Sex/Age</td>
<td>Side</td>
<td>Predisposing Factors</td>
<td>Onset</td>
<td>Clinical Presentation</td>
<td>Operative Treatment Reduction</td>
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<tr>
<td>Yacoub et al. (1968)</td>
<td>M 72</td>
<td>L</td>
<td>Defect 10×15 cm</td>
<td>12 hr</td>
<td>BP CXR = herniation of heart into left pleural cavity, ECG = LBBB and ischaemic changes, VF = DC shock — SR</td>
<td>Repair of defect with pleural patch</td>
<td>Died</td>
</tr>
<tr>
<td>Gates et al. (1970)</td>
<td>F 51</td>
<td>R</td>
<td>Very large defect.</td>
<td>RW</td>
<td>BP Acute SVC obstruction</td>
<td>Repair of defect with Dacron patch</td>
<td>Recovery</td>
</tr>
<tr>
<td>Takita and Mijares (1970)</td>
<td>M 58</td>
<td>R</td>
<td>Defect 5 × 2-5 cm.</td>
<td>T</td>
<td>BP Acute SVC obstruction</td>
<td>Loose closure of defect</td>
<td>Died 6 days later</td>
</tr>
<tr>
<td>Wright et al. (1970)</td>
<td>M 37</td>
<td>L</td>
<td>5 × 5 cm defect loosely closed</td>
<td>Night of op.</td>
<td>BP JVP CXR = shift of cardiac shadow to left</td>
<td>Wide incision of pericardium</td>
<td>Recovery</td>
</tr>
<tr>
<td>Levin et al. (1971)</td>
<td>F 53</td>
<td>R</td>
<td>Large defect which was enlarged to forestall herniation</td>
<td>RW</td>
<td>BP JVP CXR = right-sided herniation of the heart</td>
<td>Repair of defect using fascia lata</td>
<td>Died 2 days later</td>
</tr>
<tr>
<td>Dippel and Ehrenhaft (1973)</td>
<td>M 58</td>
<td>L</td>
<td>5 × 6 cm defect</td>
<td>RR</td>
<td>BP JVP CXR = unusual cardiac shadow in lower part of left pleural cavity</td>
<td>Nil (herniation not recognized)</td>
<td>Died</td>
</tr>
<tr>
<td></td>
<td>M 20</td>
<td>R</td>
<td>6 × 8 cm defect</td>
<td>RR</td>
<td>BP CXR = right-sided herniation of heart</td>
<td>Edges of defect sutured to myocardium</td>
<td>Recovery</td>
</tr>
<tr>
<td>Patel et al. (1973)</td>
<td>M 57</td>
<td>R</td>
<td>Defect</td>
<td>½ hr</td>
<td>BP JVP Ischaemic changes on ECG. Attempts to pass pacemaker catheter failed due to torsion at SVC-atrial junction</td>
<td>Nil. Treated as cardiogenic shock</td>
<td>Died</td>
</tr>
<tr>
<td>Present study</td>
<td>M 53</td>
<td>R</td>
<td>Defect 6 × 6 cm. Suction on chest drain</td>
<td>½ hr</td>
<td>BP Acute SVC obstruction, Haemorrhage ++</td>
<td>Re-explored on ward, attempt to suture tear and reduction of herniation</td>
<td>Died</td>
</tr>
<tr>
<td></td>
<td>F 72</td>
<td>L</td>
<td>Defect 7-5 × 7-5 cm.</td>
<td>16 hr</td>
<td>BP Bradycardia JVP. Displaced apex beat. ECG = heart block. Dislocated heart shadow</td>
<td>Repair of defect with Dacron patch</td>
<td>Recovery</td>
</tr>
<tr>
<td></td>
<td>M 65</td>
<td>L</td>
<td>Very large pericardial defect</td>
<td>2 hr</td>
<td>BP Signs of massive intrapleural bleeding</td>
<td>Closure of ventricular tear and repair of defect with Dacron cloth</td>
<td>Recovery, Died 12 days later from gastric bleeding</td>
</tr>
</tbody>
</table>

Key to symbols:
BP = hypotension
JVP = elevated jugular venous pressure
CXR = chest radiograph
SVC = obstructed central venous pressure plus cyanosis of upper half of body
SR = sinus rhythm
VF = ventricular fibrillation
T = in theatre at conclusion of operation
RR = recovery room
RW = immediately on return to ward
poor respiratory function and paradoxical movement of the left chest wall produced marked dyspnoea. One week after surgery he developed bronchopneumonia which responded to antibiotic therapy. A massive haematemesis from a duodenal ulcer led to his sudden death on the twelfth postoperative day.

DISCUSSION

Intrapericardial ligation of the pulmonary vessels has allowed a safer and more radical excision of lung tumours encroaching on the hilum or actually invading the pericardium (Allison, 1946). The resultant pericardial defect, if not repaired, however, exposes the patient to the risk of cardiac herniation into the pneumonectomy space.

Although most documented instances of cardiac herniation have occurred through surgically created defects, fatal herniation of the heart through a congenital defect, situated high up on the left side of the pericardial sac, has been reported on three occasions (Boxall, 1886; Sunderland and Wright-Smith, 1944; Bruning, 1962). Cardiac herniation through pericardial tears has resulted from severe blunt injury to the chest (Warburg, 1938; Beck, 1947; Wright, Nelson, Johnson, and McMillan, 1970). I have encountered this complication of blunt thoracic trauma in two patients, both of whom died of associated injuries. The heart prolapsed into the left pleural cavity through a longitudinal tear in the pericardium, which extended from the pericardial reflection onto the pulmonary artery to the diaphragm.

Bettman and Tannenbaum (1948) reported the first case of cardiac herniation following an intrapericardial pneumonectomy. Since then at least 24 further cases, including the three presented in this paper, have appeared in the English literature (Kirchhoff, 1951; Higginson, 1953; Dahlbäck and Nilsson, 1956; Neville and Jubb, 1957; Sharma, Bates and Hurt, 1959; Walmsley, 1961; Gravel, 1966; Yacoub, Williams and Ahmad, 1968; Gates, Sette, and Cope, 1970; Takita and Mijares, 1970; Wright et al., 1970; Levin, Penfield-Faber, and Carlton, 1971; Dippel and Ehrenhaft, 1973; Patel, Shrivastav, and Sabety, 1973). Undoubtedly further unreported and unsuspected cases must have occurred. Of the 25 cases, three were females, a reflection of the prevalence of lung cancer in males. There were 14 right-sided and 11 left-sided herniations of the heart. Seven of the former and five of the latter died, giving an overall mortality of 50%. In four cases the diagnosis was missed. All four died. Of 21 patients treated surgically, 12 survived.

The condition may present at the conclusion of the operation or in the very early postoperative period (Higginson, 1953; Dahlbäck and Nilsson, 1956; Sharma et al., 1959). All cases occurred within the first 24 hours. The onset is dramatic, with sudden profound hypotension and tachycardia. Cyanosis in the drainage area of the superior vena cava, most marked around the face and neck, and elevation of the jugular venous pressure are frequently noted, particularly in right-sided herniation of the heart (Kirchhoff, 1951; Gravel, 1966; Gates et al., 1970; Takita and Mijares, 1970).

There is no clinical evidence of respiratory obstruction, contralateral pneumothorax or tracheal shift. Oxygen administration has no effect on the cyanosis which is due to obstruction of the superior vena cava. An unduly prominent apical thrust is present in some cases of left-sided herniation of the heart (Walmsley, 1961). The expected absence of cardiac impulse in the left hemithorax in cases of right-sided herniation of the heart has, surprisingly, not been documented. Transient clinical improvement after the onset of herniation, as evidenced by sudden hypotension, occurs in some cases. All, however, relapsed into profound shock within a short time (Higginson, 1953; Dahlbäck and Nilsson, 1956; Yacoub et al., 1968).

These warning episodes should not be ignored. Radiological examination with critical appraisal of the chest film is essential.

The radiological appearance of right-sided cardiac herniation is unmistakable. Left-sided herniation of the heart is less easy to recognize. A rounded opacity in the lower part of the left hemithorax caused by the strangulated ventricular mass is visible in addition to the left shift of the cardiac shadow. There may be a constriction between the cardiac mass and the great vessels, the heart assuming a spherical shape. The apex may contact the lateral chest wall or be posteriorly directed into the posterior costophrenic sulcus (Gates et al., 1970).

The effects of cardiac herniation are due to a combination of cardiac malposition with subsequent torsion of the great vessels, obstruction to the outflow tracts of the heart, and strangulation of the prolapsed ventricles by the borders of the pericardial defect. The haemodynamic disturbance in right-sided herniation is more related to cardiac malposition. The anticlockwise rotation of the dextroposed heart results in a gross degree of torsion of the atrio caval junctions and ventricular outflow tract obstruction. We believe that the shearing stress at the medial aspect of the inferior atrio caval junction, resulting from forcible dislocation of the heart into the right pleural cavity when suction was applied to the chest tube,
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was responsible for the tear at the inferior atrio-
caval junction in our first patient. It is possible
that trauma to that particular area by the inferior
border of the defect may have contributed to the
development of the tear.

The effects of left-sided herniation on the circu-
lation result more from compression of the ventri-
cular muscle by the edges of the pericardial defect.
Increasing oedema and congestion of the herni-
ated heart caused by the constricting edges of the
defect aggravates the condition. The damage to
the myocardium soon becomes irreversible.
Electrocardiographic ischaemic changes charac-
terized by ST segment changes, widened com-
plexes, and disorders of conduction have been
documented (Yacoub et al., 1968; Patel et al.,
1973) and occasionally led to misdiagnosis (Patel
et al., 1973). Ventricular fibrillation when it
occurs may respond to DC shock (Walmsley, 1961;
Yacoub et al., 1968). Prompt relief of the herni-
ation after restoration of heart action has resulted
in survival (Walmsley, 1961).

The presence of a medium or large defect in a
pericardial cavity free of adhesions is a prerequi-
site for cardiac herniation to occur. Defects
measuring \(5 \times 5\) cm or more are the most
dangerous, although herniation has on occasions
occurred through small defects (Neville and Jubb,
1957; Takita and Mijares, 1970). The absence of
any recorded cases of late herniation is probably
the result of rapid development of adhesions
between the heart and the parietal pericardium.
Allison (1946) has shown that the edges of the
defect became adherent to the heart within three
days.

Predisposing factors have included the appli-
cation of suction to chest tubes (Walmsley, 1961;
Dahlbäck and Nilsson, 1956), positive pressure
inflation of the remaining lung while aspirating
air from the pneumonectomy space (Higginson,
1953), tracheal aspiration and coughing (Gates et
al., 1970) or changes in the position of a patient
during transport from the operating theatre to
the ward (Neville and Jubb, 1957; Takita and Mijares,
1970).

Prophylactic measures against possible hernia-
tion of the heart should be taken in all cases
where a pericardial defect has been created. Wide
excision of the pericardium is certainly no
insurance against cardiac herniation and incarce-
ration (Higginson, 1953; Levin et al., 1971;
Yacoub et al., 1968). Very small defects are pro-
bably harmless and can be closed with direct
suture. It is our belief that all medium and large
defects must be repaired. Strong pleura, if
available, can be used to close the defect but the
use of thin pleura is not reliable. Tubbs (1959)
suggested closure of the defect by a latticework
of catgut.

We have found the use of pericardial slings
fashioned from the edges of the defect and swung
across it with fixation to a point diametrically
opposite the origin of the sling effective in
medium-sized defects. More than one sling can be
fashioned so that only small insignificant gaps
remain in the pericardium. The use of synthetic
material to repair the defect is open to the
theoretical objection that it may abrade the sur-
face of the heart (Levin et al., 1971). We have
used Dacron on many occasions prophylactically
and twice in the treatment of cardiac herniation
with satisfactory results. However, its presence
may perpetuate any sepsis should the pneumonec-
tomy space become infected.

The use of fascia lata to repair the defect has
been advocated by Levin et al., (1971). A simple
technique involves the suturing of the edges of the
defect to the myocardium at \(1\) cm intervals,
avoiding the coronary vessels (Dippel and Ehren-
haft, 1973). In the treatment of the established
condition urgent operative intervention is essential
because damage to the myocardium soon becomes
irreversible. Ionotropic drug support may be re-
quired preoperatively and after the relief of the
herniation.

The reduction of the herniated heart is effected
by incising the constricting borders of the defect.

Measures to prevent recurrence of cardiac
herniation should also be carried out. We do not
favour the use of fascia lata or the method adva-
ced by Dippel and Ehrenhaft (1973). Our
objection to the use of fascia lata is based on the
fact that it is inconvenient to obtain in the course
of the operation, and that it inflicts unnecessary
trauma on the patient. We have doubts about the
security of anchorage achieved by suturing the
myocardium to the edges of the pericardial defect
as suggested by Dippel and Ehrenhaft (1973).

Simple incision of the border of the defect and
reduction of the heart is not adequate treatment,
for fatal recurrent herniation has followed its use
(Sharma et al., 1959). We do not believe that en-
larging the defect by wide excision in either right
or left-sided herniation is a guarantee against
further recurrence.

I should like to thank Mr. A. G. Norman and Mr.
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admitted, and Mr. H. C. Nohl-Oser for their help and
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REFERENCES


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