Cardiovascular changes during drainage of pericardial effusion by thoracoscopy

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Background. Cardiovascular changes during drainage of pericardial effusion are not well understood, and most studies are of systemic effects and not of right ventricular performance. Thoracoscopy is not widely used to drain pericardial effusions because of haemodynamic changes in relation to the use of single lung ventilation.

Patients and methods. We studied 16 patients undergoing partial pericardiectomy for pericardial effusion, using videothoracoscopy with a low-pressure pneumothorax (6 mm Hg). Cardiac output was measured by thermodilution with the patient anaesthetized in the supine position before the procedure; in the right lateral position after a low-pressure pneumothorax had been established; and after drainage of the pericardial effusion.

Results. Before the procedure, cardiac output was low and central venous pressure and pulmonary artery occlusion pressure were increased. Systemic vascular resistance and arterial blood pressure were within normal limits. Cardiac filling pressure and pulmonary arterial pressure increased during the pneumothorax. After the drainage cardiac index increased and systemic and pulmonary vascular resistances were reduced.

Conclusions. Pericardial effusion reduces right ventricular distensibility, right and left systolic ventricular function, and cardiac output. Anaesthesia with mechanical ventilation and a low-pressure pneumothorax do not affect the circulation greatly. Drainage of the pericardial effusion allows cardiac distensibility to increase and cardiac performance changes to allow increased ejection.

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Pericardial effusion reduces cardiac diastolic filling, depending on the amount of fluid that has accumulated, the speed of accumulation, and the distensibility of the pericardium.1 Most studies of changes in the circulation have concentrated on left ventricular function, and ignored changes on the right ventricle although this is greatly affected by the increase in intrapericardial pressure.2 The effects of the drainage procedure used, particularly during thoracoscopy, are not well understood.

Videothoracoscopic drainage of pericardial effusions requires a good view of the pleural space and pericardial surface during open surgery. This can be obtained by collapse of one lung and selective ventilation of the contralateral lung using an endotracheal double-lumen tube. This has potential risks of right ventricular failure, hypoxaemia, hypercapnia, atelectasis and pneumonia. An alternative procedure is to combine general anaesthesia with tracheal intubation and a low-pressure pneumothorax. This technique can cause complications but these are less than with the open procedure.3 4 We set out to measure the changes in the circulation, especially of the right side, in patients with pericardial effusions who were undergoing drainage of cardiac effusions by thoracoscopy with a low-pressure pneumothorax.

Patients and methods

Patients

We studied 16 patients (10 females) having pericardial resection for pericardial effusion using a video-assisted technique. Patients were studied between February 1995
and May 1998. Four effusions were malignant, two were uraemic, seven idiopathic, two occurred after pericardial surgery, and one was tuberculous. The mean (range) age was 56 (18–60) yr. All patients had symptoms from the effusion such as orthopnea and venous distension.

The study was approved by the ethics committee of the hospital, and patients gave informed consent to the study. Anaesthesia was the same for all the patients. Anaesthesia was induced with the same drugs used as a continuous infusion during the procedure, but given as an initial dose, and maintained with infusions of midazolam 50 (10–150) mg kg⁻¹ h⁻¹, fentanyl 10 (5–30) μg kg⁻¹ h⁻¹, and maintained with infusions of ranitidine 50 μg kg⁻¹ h⁻¹, ketamine 2 mg kg⁻¹ h⁻¹ and fentanyl 10 μg kg⁻¹ h⁻¹. Vecuronium was used for neuromuscular block. Before surgery, we gave 500–700 ml of Ringer’s-lactate to achieve a central venous pressure (CVP) between 15 and 20 mm Hg.

### Table 1

<table>
<thead>
<tr>
<th>Variable</th>
<th>NHRR</th>
<th>Stage I</th>
<th>Stage II</th>
<th>Stage III</th>
<th>Stage IV</th>
</tr>
</thead>
<tbody>
<tr>
<td>HR (beats min⁻¹)</td>
<td>60–80</td>
<td>94 (30)</td>
<td>89 (29)</td>
<td>89 (25)</td>
<td>89 (23)</td>
</tr>
<tr>
<td>CI (litre min⁻¹ m⁻²)</td>
<td>2.8–4.2</td>
<td>2.3 (0.4)</td>
<td>2.3 (0.5)</td>
<td>2 (0.6)</td>
<td>3.8 (1.1)*** §</td>
</tr>
<tr>
<td>SVI (ml m⁻³ beat⁻¹)</td>
<td>30–65</td>
<td>27.7 (12)</td>
<td>28.6 (11.6)</td>
<td>26.1 (12.4)</td>
<td>45.5 (15.3)***</td>
</tr>
<tr>
<td>EF (%)</td>
<td>40–60</td>
<td>31.1 (7.7)</td>
<td>27.3 (6.6)</td>
<td>24.9 (8.6)</td>
<td>36.6 (9.1)***</td>
</tr>
<tr>
<td>CVP (mm Hg)</td>
<td>2–8</td>
<td>15 (4.8)</td>
<td>18.5 (4.9)*</td>
<td>21.4 (3.1)*</td>
<td>10.5 (3.9)***</td>
</tr>
<tr>
<td>PCWP (mm Hg)</td>
<td>8–12</td>
<td>17 (3.7)</td>
<td>21.1 (4.3)**</td>
<td>23.8 (3.4)*</td>
<td>14.9 (2.9)***</td>
</tr>
<tr>
<td>SAP (mm Hg)</td>
<td>100–140</td>
<td>111 (21.7)</td>
<td>124.9 (18.3)</td>
<td>128.8 (27.1)*</td>
<td>127.9 (33.3)</td>
</tr>
<tr>
<td>MAP (mm Hg)</td>
<td>70–105</td>
<td>79 (14.6)</td>
<td>89.9 (14.4)*</td>
<td>98.4 (19.9)</td>
<td>84.3 (21)*</td>
</tr>
<tr>
<td>SPAP (mm Hg)</td>
<td>15–30</td>
<td>29 (6.1)</td>
<td>33.9 (6.3)**</td>
<td>36.4 (5.7)</td>
<td>30.7 (9.3)**</td>
</tr>
<tr>
<td>DPAP (mm Hg)</td>
<td>4–12</td>
<td>17 (4.6)</td>
<td>22.8 (4.6)**</td>
<td>26.3 (3.7)**</td>
<td>16.5 (3.3)***</td>
</tr>
<tr>
<td>MPAP (mm Hg)</td>
<td>9–18</td>
<td>22 (4.9)</td>
<td>27.5 (4.3)**</td>
<td>30.3 (4.4)**</td>
<td>22.1 (5.4)***</td>
</tr>
<tr>
<td>EDVI (ml m⁻²)</td>
<td>60–100</td>
<td>87 (33)</td>
<td>98.2 (23.6)</td>
<td>97.6 (27)</td>
<td>124 (28.6)***</td>
</tr>
<tr>
<td>ESVI (ml m⁻²)</td>
<td>30–60</td>
<td>60 (24.5)</td>
<td>70.2 (19.7)</td>
<td>71.4 (17)</td>
<td>77 (19.4)</td>
</tr>
<tr>
<td>PVRI (dyn s⁻¹ cm⁻⁵ m⁻²)</td>
<td>45–225</td>
<td>204 (64.6)</td>
<td>235 (75.6)</td>
<td>288.1 (132.5)*</td>
<td>143 (56.2)***</td>
</tr>
<tr>
<td>SVRI (dyn s⁻¹ cm⁻⁵ m⁻²)</td>
<td>1500–2500</td>
<td>2310 (544)</td>
<td>2583 (767)</td>
<td>3112 (1030)*</td>
<td>1622 (516)***</td>
</tr>
<tr>
<td>RVSWI (g·m⁻³·beat⁻¹)</td>
<td>4–8</td>
<td>2.9 (1.9)</td>
<td>3.5 (2.2)</td>
<td>2.9 (1.7)</td>
<td>7.7 (3.9)***</td>
</tr>
<tr>
<td>LVSWI (g·m⁻³·beat⁻¹)</td>
<td>44–68</td>
<td>25.1 (15)</td>
<td>27.4 (13.6)</td>
<td>24.6 (14.1)*</td>
<td>45.4 (24.7)***</td>
</tr>
</tbody>
</table>

NHRV=normal haemodynamic reference values; Stage I=baseline values during supine anaesthesia; Stage II=lateral position; Stage III=during pneumothorax; Stage IV=after the procedure; HR=heart rate; CI=cardiac index; SVI=systolic volume index; EF=ejection fraction; PCWP=central venous pressure; CVP=central venous pressure; PAOP=pulmonary artery occlusion pressure; SAP=systolic arterial pressure; DPAP=diastolic arterial pressure; MAP=mean arterial pressure; PVRI=pulmonary vascular resistance index; SVRI=systemic vascular resistance index; RVSWI=right ventricular stroke work index; LVSWI=left ventricular stroke work index.

**Cardiovascular measurements**

Vascular pressures, ECG, and pulse oximetry were measured with a Kolormon–7250 (Kontron) monitor. Right ventricular performance was measured with an ejection fraction (EF) catheter (Swan–Ganz® right ventricular ejection fraction/volumetric oximetry TD catheter and Edwards REF-1 ejection fraction/cardiac output computer, SAT-2 Oximeter/cardiac output computer; Baxter, Edwards, Irvine, CA, USA) which has a rapid-response thermistor (95 ms), atrial and ventricular electrodes and four lumens. Calculations were carried out by computer (Explorer®, Baxter).

**Haemodynamic measurement technique**

The cardiac output catheter was inserted into the right internal jugular vein through an 8.5-fr introducer, advanced so that the tip had entered the pulmonary artery and...
Pericardial effusion drainage

Cardiac output was measured with injections of 10 ml of saline 0.9% at 4°C into the right atrium, through one of the two proximal lumens, just above the tricuspid valve, at end-expiration during mechanical ventilation. We used at least six injections of cold saline per measurement and discarded the smallest and largest value of cardiac output.

Initial measurements were made with the patient supine, after induction of anaesthesia and before starting the surgery, and repeated after positioning in the right lateral position. Further measurements were made after creation of the pneumothorax, and then at the end of the procedure, after drainage of the effusion and removal of the pneumothorax, with the patient supine. Comparisons were made with the previous stage and with the initial measurements. The levels of the transducers were adjusted in relation to the right atrium after moving from the supine to right lateral position.

Statistical analysis
Measurements were compared with analysis of variance for repeated measurements, using a hierarchical factorial design. The principal factor was the surgical stages. Subsequent comparison were with a paired t-test and statistical significance was taken when \( P < 0.05 \).

Results
There was no hypotension or arrhythmia during the procedure. Expired carbon dioxide, pulse oximetry and inspiratory pressures remained within normal values during the procedure. Table 1 shows the haemodynamic values obtained at the four stages of the procedure. Expected values are also shown.

Cardiac index was less, and CVP and pulmonary artery occlusion pressure (PAOP) were greater, than expected values. Arterial pressure and systemic vascular resistance index (SVRI) were within the normal range, but systolic ventricular function, both left and right, EF and right/left ventricular stroke work index (R/LVSWI) were below normal.

In the right lateral position significant increases were noted in CVP \( (P < 0.05) \), PAOP \( (P < 0.01) \), and pulmonary arterial systolic (SPAP; \( P < 0.01 \)), diastolic (DPAP; \( P < 0.001 \)) and mean (MPAP; \( P < 0.001 \)) pressures. Systemic diastolic (DAP; \( P < 0.01 \)) and mean (MAP; \( P < 0.05 \)) pressures also increased.

During the pneumothorax significant increases in MPAP and DPAP were noted, along with CVP, PAOP, pulmonary vascular resistance index (PVRI) and SVRI.

After drainage, cardiac index and EF returned to normal and the resistance values decreased appropriately. CVP and PAOP decreased, but remained greater than expected values. Measures of cardiac work suggest that distensibility of the heart increased and contractility decreased.

Discussion
Pericardial effusion limits cardiac expansion and increases atrial, ventricular, and pericardial pressures. The result is a reduction in cardiac output, and vasoconstriction to maintain the blood pressure.\(^6\)\(^-\)\(^8\) In the lateral position, intrathoracic pressures increase because of the distended pericardium and the insufflation of the pneumothorax.

Selective one-lung ventilation provides the best field for surgery,\(^9\)\(^10\) but not all patients can tolerate the hypoxaemia caused by lung collapse.\(^11\)\(^12\) Incorrect tube position, absorption atelectasis and hypoxic pulmonary vasoconstriction can aggravate this situation.\(^13\) Hypoxia and hypercapnia may cause acidosis and increase PVRI.\(^14\)\(^-\)\(^16\) Acute failure or dilatation of the right ventricle\(^17\)\(^-\)\(^19\) can further reduce left ventricular filling and cardiac output. Pulmonary collapse can occur, especially in patients with chronic lung disease.\(^9\)\(^-\)\(^11\)\(^20\)\(^21\)

Anaesthetic management by simple tracheal intubation and a pneumothorax is technically simple, and partial lung collapse allows a good view of the pericardium. We found that by avoiding complete lung collapse, arterial oxygenation can be maintained during the operation, with few postoperative complications.

The pneumothorax increases cardiac filling pressure, pulmonary arterial pressure and pulmonary resistance by a pressure effect of the pneumothorax on the mediastinum. Venous return is reduced and cardiac output is reduced by 13%. MAP does not change substantially (9%) and peripheral resistance is increased (20%). Cardiac systolic function (SVI, EF and R/LVSWI) does not change with the pneumothorax. We found that a pressure of 6 mm Hg had no adverse effects but provided good surgical conditions. A recent study\(^4\) describes reduced cardiac output with a pneumothorax of 10 or 15 mm Hg, but not with a 5 mm Hg pneumothorax.

After drainage, cardiac output is increased and MAP remains practically unaltered. Stroke volume increases because end-diastolic volume increases, although end-systolic volume increases also, so that EF, despite increasing also, does not reach the lower limits of normal.\(^22\) After drainage, the right ventricle has an increased preload and reduced afterload, and dilates, which is the main reason for the increase in cardiac index.\(^23\)

Does removal of the pericardial effusion reduce right ventricular contractility? The decreased contractility, indicated by reduced Emax, contrasts with the increase in right and left ventricular stroke work. The relationship between ventricular stroke work and end-diastolic volume (preload recruitable stroke work) is considered the most appropriate measurement of ventricular function.\(^23\)\(^24\) A decrease in the Ea:Emax ratio produces a greater ventricular contractile efficiency, as it improves ventriculo-arterial coupling.\(^24\)\(^-\)\(^27\)
The decrease in afterload does not prevent the considerable increase in stroke work, as the determining factor is the increased SVI. Cardiac performance increases as the conditions change from a situation of pressure work to ejection work.

In conclusion, in patients with pericardial effusion, right ventricular distensibility is reduced, shown by a disproportionately high pressure in relation to the existing volume, and by impaired right and left systolic ventricular function. A low-pressure pneumothorax during thoracoscopy has little effect on cardiac and respiratory function and gives an adequate surgical field. Drainage of the pericardial effusion increases cardiac distensibility and cardiac volumes.

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