APPARATUS

EFFICIENCY OF THE LEFT VENTRICLE ASSIST DEVICE HEMOPUMP IN CARDIAC FIBRILLATION


SUMMARY

We have examined in sheep the efficiency of the Hemopump during ventricular fibrillation. Circulatory arrest was induced by electrical stimulation and maintained for 30 min. Haemodynamic measurements were recorded continuously and blood samples were taken before, during and after fibrillation to determine total body and myocardial metabolic activity. All hearts were defibrillated successfully after 30 min of fibrillation. During fibrillation, the Hemopump sustained a mean arterial pressure of about 60 mm Hg with a blood flow rate of about 2.3 litre min\(^{-1}\). These perfusion conditions were sufficient for maintenance of aerobic myocardial metabolism, but with a borderline circulatory supply to the total organism.

KEY WORDS

Equipment ventricular assist device (Hemopump). Heart: circulatory arrest, fibrillation.

Intra-aortic balloon counterpulsation (IABP) is the method used most frequently for mechanical circulatory support in patients with cardiogenic shock. However, the IABP is inefficient during severe cardiac arrhythmias and low arterial pressure [1]. The Hemopump is still effective if cardiac function deteriorates [2, 3], because it produces a heart action independent of blood flow from the left ventricle into the aorta [4-6]. As with the IABP, during an emergency the Hemopump may be inserted easily via the femoral artery.

The Hemopump assist device consists of a control console, an external motor and a catheter system, which contains a drive cable and a conduit for fluid which continuously flushes the system. The catheter ends in a silicone rubber inlet cannula (26 cm long, 21-French gauge) containing the axial screw (fig. 1). The screw is driven by an external magnetic motor via the drive cable. After insertion into the left ventricle, the pump system produces a maximal volume output into the aorta of 3.5 litre min\(^{-1}\) against an arterial pressure of 75 mm Hg [7].

The aim of this study was to assess the efficiency of the Hemopump in cardiac arrest during fibrillation by assessing circulation to the heart and total body.

MATERIALS AND METHODS

Studies were performed in nine sheep (mean body weight 58 (st 11) kg, range 48-78 kg; left ventricular weight 159 (36) g). Anaesthesia was maintained with a continuous infusion of midazolam 0.25 mg kg\(^{-1}\) h\(^{-1}\) and buprenorphine 0.01 mg kg\(^{-1}\) h\(^{-1}\). The trachea of each sheep was intubated and the lungs ventilated artificially with 70% nitrous oxide in oxygen.

Left ventricular pressure was measured by a transducer-tipped catheter and other pressures (arterial, pulmonary arterial and central venous) by strain gauge transducers. Cardiac output was esti-
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FIG. 2. Original recording during the use of the Hemopump in fibrillation induced by electrical stimulation. ECG = electrocardiogram; ScvO₂ = coronary venous oxygen saturation; MBF = myocardial blood flow; P = pressure (LV = left ventricular, Ao = aortic), dP/dt = rate of left ventricular pressure increase.

TABLE I. Haemodynamic and metabolic data (mean (sd)) before, 10, 20 and 30 min during and 10 min after fibrillation. LV = Left ventricle

<table>
<thead>
<tr>
<th></th>
<th>Control</th>
<th>10 min</th>
<th>20 min</th>
<th>30 min</th>
<th>Sinus rhythm</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Haemodynamic data</strong></td>
<td></td>
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<tr>
<td>Central venous pressure (mm Hg)</td>
<td>6.6 (1.9)</td>
<td>11.6 (2.7)</td>
<td>10.6 (2.5)</td>
<td>10.5 (2.5)</td>
<td>7.1 (2.6)</td>
</tr>
<tr>
<td>Pulmonary arterial pressure (mm Hg)</td>
<td>21 (5.6)</td>
<td>11.6 (3.6)</td>
<td>10.7 (3.0)</td>
<td>10.5 (2.8)</td>
<td>18.1 (5.5)</td>
</tr>
<tr>
<td>Mean aortic pressure (mm Hg)</td>
<td>96.8 (17.9)</td>
<td>64.0 (14.2)</td>
<td>60.4 (15.8)</td>
<td>54.6 (8.1)</td>
<td>72.2 (19.7)</td>
</tr>
<tr>
<td>LV end-diastolic pressure (mm Hg)</td>
<td>11.26 (7.61)</td>
<td>4.75 (7.40)</td>
<td>1.75 (5.88)</td>
<td>1.50 (5.86)</td>
<td>13.43 (7.79)</td>
</tr>
<tr>
<td>Hemopump flow rate (litre min⁻¹)</td>
<td>4.7 (0.8)</td>
<td>2.5 (0.5)</td>
<td>2.4 (0.6)</td>
<td>2.1 (0.8)</td>
<td>2.4 (0.6)</td>
</tr>
<tr>
<td>Myocardial blood flow (ml min⁻¹/100 g LV)</td>
<td>160.1 (62.0)</td>
<td>108.9 (72.4)</td>
<td>101.2 (56.3)</td>
<td>97.8 (57.6)</td>
<td>122.1 (37.8)</td>
</tr>
<tr>
<td>Total peripheral resistance (mm Hg min ml⁻¹/kg body weight)</td>
<td>1.3 (0.5)</td>
<td>1.3 (0.4)</td>
<td>1.4 (0.5)</td>
<td>1.5 (0.5)</td>
<td>1.8 (0.7)</td>
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<tr>
<td><strong>Metabolic data</strong></td>
<td></td>
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<td></td>
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<tr>
<td>Arterial potassium content (mmol litre⁻¹)</td>
<td>3.3 (0.3)</td>
<td>3.9 (0.3)</td>
<td>3.8 (0.3)</td>
<td>3.8 (0.4)</td>
<td>3.6 (0.4)</td>
</tr>
<tr>
<td>Myocardial potassium uptake (µmol min⁻¹/100 g LV)</td>
<td>-2.0 (2.0)</td>
<td>-15.8 (8.3)</td>
<td>-8.5 (10.4)</td>
<td>-1.8 (6.6)</td>
<td>-4.3 (10.9)</td>
</tr>
<tr>
<td>Arterial lactate content (mmol litre⁻¹)</td>
<td>4.5 (1.3)</td>
<td>4.3 (1.0)</td>
<td>4.8 (1.2)</td>
<td>5.3 (1.6)</td>
<td>6.2 (1.9)</td>
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<tr>
<td>Myocardial lactate uptake (µmol min⁻¹/100 g LV)</td>
<td>51.3 (23)</td>
<td>23.5 (15.3)</td>
<td>29.9 (14.0)</td>
<td>32.9 (20.6)</td>
<td>38.3 (25.2)</td>
</tr>
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<td>Oxygen extraction ratio of lactate (%)</td>
<td>42.7 (19.26)</td>
<td>31.4 (31.68)</td>
<td>38.7 (23.72)</td>
<td>40.83 (27.0)</td>
<td>38.7 (20.7)</td>
</tr>
<tr>
<td>Coronary venous O₂ saturation (ml O₂/100 ml)</td>
<td>25.8 (9.6)</td>
<td>32.9 (14.3)</td>
<td>31.5 (12.6)</td>
<td>30.5 (12.1)</td>
<td>41.5 (14.2)</td>
</tr>
<tr>
<td>Total body O₂ uptake (ml O₂ min⁻¹/kg body weight)</td>
<td>2.6 (0.6)</td>
<td>2.0 (0.3)</td>
<td>2.1 (0.4)</td>
<td>1.8 (0.3)</td>
<td>2.0 (0.5)</td>
</tr>
<tr>
<td>Myocardial O₂ consumption (ml O₂ min⁻¹/100 g LV)</td>
<td>9.1 (1.7)</td>
<td>4.9 (1.5)</td>
<td>4.9 (1.2)</td>
<td>4.9 (1.4)</td>
<td>6.6 (1.8)</td>
</tr>
</tbody>
</table>

mated with the thermodilution technique. Measurements of serum electrolyte and lactate concentrations and oxygen content were made on pulmonary arterial, coronary venous and arterial blood samples. Myocardial blood flow (MBF) was measured using a coronary sinus flowmeter [8, 9].

The Hemopump was inserted into a large artery in the iliac region and advanced through the aorta.
under x-ray control until the bevelled tip of the inlet cannula was positioned within the left ventricle.

Fibrillation was induced by electrical stimulation and, after a 30-min observation period, sinus rhythm was re-established by electrical defibrillation. Haemodynamic data were recorded continuously throughout this period. Measurements of the metabolic data were carried out under baseline conditions and 10, 20 and 30 min after the onset of fibrillation and 10 min after sinus rhythm was induced.

RESULTS

Figure 2 shows a typical example of the change from normal haemodynamic state to ventricular fibrillation. At the beginning of fibrillation, a complete breakdown of circulation is evident. When the Hemopump was applied, arterial pressure increased and became distinct from the left ventricular pressure; after a delay, myocardial blood flow (MBF) increased. The lack of pulsation in the arterial pressure reflects the non-pulsatile blood flow produced by the Hemopump.

Data on the efficiency of the Hemopump are presented in Table I.

After 10 min fibrillation, the blood flow supplied by the Hemopump was about 2.3 litre min⁻¹, whilst arterial pressure was maintained at about 60 mm Hg. There was a decreasing trend in both values during the period of fibrillation. Total body oxygen uptake was reduced from 2.6 to 1.9 ml min⁻¹/kg body weight at the end of fibrillation. This decrease in total body oxygen uptake was accompanied by an increase in arterial lactate concentration from 4.5 to 6.2 mmol litre⁻¹.

With the Hemopump, a steady myocardial oxygen consumption of 4.9 ml min⁻¹/100 g left ventricular weight was found during fibrillation. The baseline value of 9.1 ml min⁻¹/100 g was not reached, 10 min after restoration of sinus rhythm. One hour after the end of fibrillation, baseline values were re-established.

Potassium release from the heart was evident after 10 min of fibrillation, but not at 20 min. Concomitantly, there was a marked reduction in myocardial lactate uptake at 10 min. Thereafter, an increase in lactate uptake occurred, although baseline values were not re-established.

DISCUSSION

Compared with baseline cardiac output of 4.7 litre min⁻¹, the Hemopump produced a blood flow during fibrillation of 2.3 litre min⁻¹, with no change in peripheral resistance. Correspondingly, mean arterial pressure decreased from nearly 100 to 60 mm Hg. During fibrillation, total body oxygen uptake could not be maintained at a normal value of 2.6 ml min⁻¹/kg body weight under anaesthesia; it decreased to 2.0 ml min⁻¹/kg body weight, indicating insufficient circulatory supply to the body. As a result, arterial lactate concentration increased during the period of fibrillation. After re-establishment of normal cardiac function, a maximal lactate concentration was reflected by release of lactate from the periphery.

In spite of the inadequate total blood flow, the Hemopump maintained a normal myocardial oxygen demand of 4.9 ml min⁻¹/100 g during fibrillation [10]. Aerobic metabolism was maintained as demonstrated by the normal lactate uptake and the increasing coronary venous oxygen saturation. The ratio of lactate to myocardial oxidation metabolism also supports the view that coronary blood flow was adequate to maintain normal myocardial metabolism: the value of 40%, which changed only slightly, lay in the normal range [11]. Because of this metabolic integrity, all hearts could be defibrillated successfully. Nevertheless, total blood flow to the whole body was depressed for a short time after defibrillation and baseline conditions were not reached until 1 h later. The reason for this delayed restoration of normal pump function might be catecholamine depletion during the period of fibrillation.

We conclude that the Hemopump could provide acceptable circulatory support during a 30-min period of fibrillation, thereby preventing anaerobic myocardial metabolism, although total oxygen supply to the body was inadequate.

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