Experimental hemodynamic studies with a permanent ventricular assist device

The dynamic aortic patch

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echanical circulatory assistance by means of diastolic pressure augmentation has proved beneficial in the management of left ventricular failure.1-4 Intra-aortic phase-shift balloon pumping, a temporary form of this mode of assistance, has proved successful in the treatment of acute left ventricular failure5,6 secondary to myocardial infarction. A permanently implanted assist device, the dynamic aortic patch (DAP), which is similar to the balloon pump in function, was developed. This study describes the hemodynamic effects of the DAP in dogs.

Materials

The DAP consists of an ellipsoidal silicone rubber pumping chamber encased in Dacron velour (Fig. 1). A silicone rubber, pneumatic conduit encased in Dacron velour connects the pumping chamber through a transcutaneous connector to an external driving unit. Conductive polyurethane, having a permanent negative surface charge which enhances the growth of a pseudointima, backs the Dacron velour that lines the blood interface9,10 (Fig. 2). The DAP has an approximate stroke volume of 15 c.c.1

Nineteen experiments were performed in 10 anesthetized dogs ventilated with the chest open. Through an incision in the sixth or seventh left intercostal space, the chest was opened and snares were passed around the proximal and distal descending thoracic aorta. The intercostal vessels were tied off at their origin. By means of a roller pump, left heart bypass from the left atrium to the femoral artery was instituted. A longitudinal incision equal to the length of the DAP was made in the thoracic aorta. The DAP was sewn into the aorta with a double row of continuous everting sutures, and the suture line was reinforced with Dacron felt. Sutures were placed so that the addition of the patch did not appreciably increase the diameter of the aorta (Fig. 3).

Large-bore polyethylene catheters were placed in the central aorta via the carotid artery and in the right atrium and left ventricle by direct puncture. Biotronex flow probes were placed around the aortic root and left circumflex coronary artery. The

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Fig. 1. Extravascular surface of dynamic aortic patch booster.

Fig. 2. Vascular interface of dynamic aortic patch booster.

Fig. 3. Dynamic aortic patch booster sewn into the thoracic aorta.

electrocardiogram was recorded via needle electrodes applied subcutaneously to the limbs. Flow probes were connected to a Biotronex 610 electromagnetic flowmeter system, and pressure catheters were connected to Statham strain gauges. Electrocardiogram, right atrial pressure, left ventricular pressure, central aortic pressure, central aortic flow, and left circumflex coronary artery flow were simultaneously displayed and recorded on a direct-writing Sanborn recorder. Hemodynamic parameters were measured with the patch off (control) and compared with the patch on (pumping).

Cardiac output was determined from the aortic flow curve. Stroke volume was taken as cardiac output divided by heart rate per minute. The tension time index was derived from the integrated area under the left ventricular pressure curve. Left ventricular stroke pressure work (SPWLv) in gram-meters per beat was calculated from the formula:

$$SPWLv = \frac{(SV) \ (MAP) \ (1.35)}{1,000},$$

where SV is stroke volume and MAP is mean arterial pressure.

Mean systolic ejection rate (MSER) in milliliters per second was calculated from the formula:

$$MSER = \frac{SV}{DS},$$
Fig. 4. Sequential hemodynamic changes with the dynamic aortic patch booster static (off) and dynamic (on).

where DS is the duration of systole in seconds.

Systemic vascular resistance (SVR), expressed in resistance units, was calculated from the formula:

\[ SVR = \frac{MAP}{CO} \]

where CO is cardiac output.

All data were subjected to statistical analysis. The significance of the differences between the control (static patch) and the experimental (dynamic patch) was determined by a paired t test.

Results

Fig. 4 represents the sequential flow and pressure tracings with the patch static (con-
Table I. Hemodynamic changes with the dynamic aortic patch active

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Per cent change from control</th>
<th>Probability value</th>
</tr>
</thead>
<tbody>
<tr>
<td>TTI</td>
<td>-23.3</td>
<td>&lt; 0.01</td>
</tr>
<tr>
<td>SPWLV</td>
<td>-17.5</td>
<td>&lt; 0.01</td>
</tr>
<tr>
<td>LVPP</td>
<td>-23.2</td>
<td>&lt; 0.01</td>
</tr>
<tr>
<td>SVR</td>
<td>-32.1</td>
<td>&lt; 0.01</td>
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<tr>
<td>CO</td>
<td>+16.2</td>
<td>&lt; 0.01</td>
</tr>
<tr>
<td>MDP</td>
<td>+18.7</td>
<td>&lt; 0.01</td>
</tr>
<tr>
<td>CBF</td>
<td>+41</td>
<td>&lt; 0.01</td>
</tr>
<tr>
<td>MSER</td>
<td>+36.1</td>
<td>&lt; 0.01</td>
</tr>
</tbody>
</table>

Legend: TTI, Tension time index. SPWLV, Left ventricular stroke pressure work. LVPP, Left ventricular peak pressure. SVR, Systemic vascular resistance. CO, Cardiac output. MDP, Mean diastolic pressure. CBF, Circumflex blood flow. MSER, Mean systolic ejection rate.

trol) and dynamic (pumping) in a normotensive animal.

Table I summarizes the hemodynamic changes that occurred when the DAP group was compared to the control group. The tension time index decreased 23.3 per cent (S.D. 11), systemic vascular resistance decreased 32.1 per cent (S.D. 12), left ventricular peak pressure decreased 23.2 per cent (S.D. 14), and the stroke pressure work of the left ventricle decreased 17.5 per cent (S.D. 10). Increases in the mean aortic diastolic pressure and cardiac output were 18.7 per cent (S.D. 14) and 16.2 per cent (S.D. 8), respectively. Left circumflex coronary blood flow increased 41.0 per cent (S.D. 27), and the mean systolic ejection rate increased 36.1 per cent (S.D. 11).

All data were statistically significant at values of p < 0.01.

Discussion

A device that works on the principle of diastolic augmentation, in order to be hemodynamically effective, must fulfill the following criteria: It must consistently be accurately timed and driven so that it will effectively (1) reduce the left ventricular afterload and hence the myocardial oxygen consumption, and (2) increase the total coronary blood flow. In addition to a favorable hemodynamic response, a permanently implanted left ventricular support device should be nonthrombogenic and promote the development of a pseudointima at the blood interface. It should be nontraumatic to the blood elements and be durable.

The DAP, a permanent left ventricular assist device, fulfills the above-mentioned criteria. The driving unit is portable and simple to operate as previously reported. Other studies in this laboratory indicate that the DAP creates negligible blood trauma, has minimal thromboembolic potential, and enhances the growth of a pseudointimal layer at the blood interface.

This study indicates that the DAP is hemodynamically effective as a permanent left ventricular assist device. The decreases in stroke pressure work of the left ventricle, tension time index, left ventricular peak pressure, and systemic vascular resistance indicate that the DAP can improve myocardial function by reducing myocardial tension and hence oxygen demands. The simultaneous increases in the mean systolic ejection rate, coronary blood flow, mean diastolic pressure, and cardiac output point out a generalized metabolic and hemodynamic improvement.

Summary

The hemodynamic effects of a DAP for permanent ventricular assistance by diastolic augmentation were studied in dogs.

Favorable increases in cardiac output, mean diastolic pressure, coronary blood flow, and the mean systolic ejection rate combined with favorable reductions in the tension time index, stroke pressure work of the left ventricle, systemic vascular resistance, and left ventricular peak pressure indicate that the DAP is hemodynamically effective.

The favorable cardiovascular response combined with the ability of the blood interface of the DAP to form a pseudointima indicate that it might be clinically applicable as a permanent left ventricular support device.

REFERENCES

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