Objective: To compare the efficacy of a new intraaortic propeller pump (PP) to provide hemodynamic support to the intraaortic balloon pump (IABP) in an acute mitral regurgitation (MR) animal model.

Background: A new intraaortic PP (Reitan catheter pump; Jomed; Helsingborg, Sweden) recently has been introduced. The pump’s aim is a reduction in afterload via a deployable propeller that is placed in the high descending aorta and can be set at rotational speeds of ≤ 14,000 revolutions per minute (rpm).

Methods: In nine calves, acute MR was created by placing a vena cava filter in the mitral valve. The PP was tested at 6,000, 10,000, and 14,000 rpm and was compared to 1:1 IABP support. Cardiac output, coronary blood flow, carotid artery flow, ascending and abdominal aortic pressure, left atrial pressure, and LV pressure-volume loops were recorded.

Results: The PP caused an rpm-dependent reduction in the mean ascending aortic pressure reaching \(-10\) mm Hg \((p < 0.05)\) at 14,000 rpm. However, mean \((\pm SD)\) cardiac output did not improve \((2.6 \pm 0.7 \text{ to } 2.5 \pm 1.1 \text{ L/min}; p = \text{not significant})\), and mean diastolic coronary flow and carotid flow \((47 \pm 16 \text{ to } 35 \pm 15 \text{ centiliters/min}, p < 0.05)\) were reduced. The IABP improved cardiac output, and carotid and diastolic coronary flow.

Conclusions: In this acute MR animal model, the PP reduced afterload but left out positive effects on cardiac output, which resulted in reduced perfusion of the upper body and the coronary circulation. Therefore, the IABP gives better hemodynamic support than the new PP in calves with acute MR.

Key words: cardiogenic; heart-assist devices; intraaortic balloon pumping; mitral valve insufficiency; shock

Abbreviations: \(dP/dt_{\text{max}}\) = maximal left ventricular pressure derivative; IABP = intraaortic balloon pump; MR = mitral regurgitation; PP = propeller pump; rpm = revolutions per minute

Circulatory support by the intraaortic balloon pump (IABP) is indicated in cases of acute heart failure or cardiogenic shock that is either unresponsive to medical therapy or is accompanied by acute mitral regurgitation (MR).\(^1\)\(^2\) The mechanism of the IABP is afterload reduction and an increase in coronary perfusion.\(^1\) However, in some patients IABP support is not sufficient to maintain adequate circulation, and other circulatory support devices may be indicated.\(^1\)\(^3\)

In 2000, the intraaortic propeller pump (PP) [Reitan catheter pump; Jomed; Helsingborg, Sweden], a new device designed to continuously reduce afterload, was developed.\(^4\) It is a propeller-based pump that is placed in the high descending aorta with propeller rotational speeds of \(\leq 14,000\) revolutions per minute (rpm). Like the IABP, the aim of the intraaortic PP is to reduce pressure proximal to the pump, thereby reducing the afterload of the left ventricle. A secondary proposed benefit is the augmentation of perfusion distal to the pump. In the first human application of the PP, excellent results were reported.\(^5\) In the present
study, the efficacy of the new PP to support circulation is compared to the IABP in an animal model of cardiogenic shock due to acute MR.

**Materials and Methods**

**Animal Preparation**

All animals received humane care in compliance with the Guide for the Care and Use of Laboratory Animals of the National Institutes of Health, and the study was approved by our institution's animal ethics committee. Nine Roodbont calves with an average weight 109 kg (weight range, 76 to 148 kg) were premedicated with atropine (0.05 mg/kg subcutaneously). Anesthesia was induced with sodium thiopental (IV bolus 15 mg/kg) and was maintained with a 1:2 mixture of O₃/N₂O and 2% halothane. After the administration of the muscle relaxant suxamethonium (IV bolus 0.1 mg/kg) and analgesic buprenorfim (IV bolus 0.01 mg/kg), a left thoracotomy was performed, and ventilation was modified to give a peak end-expiration pressure of 5 cm H₂O. Heparin (IV bolus 100 IU/kg) was administered, and the activated clotting time was measured and kept at ≥ 400 s during the experiment. Monitoring included ECG, BP, oxygen saturation, and capnography. The animals were killed with an overdose of pentobarbital (IV bolus 200 mg/kg).

**Instrumentation**

Under fluoroscopy, a Swan-Ganz thermodilution catheter (Arrow International; Reading, PA) was advanced via the jugular vein. A conductance catheter, incorporating a pressure sensor (Sentron; Rhoden, the Netherlands), was placed via the right carotid artery in the left ventricle. The conductance catheter was connected to a conductance console (CD Leycom; Zoetermeer, the Netherlands), which was used in the dual-frequency mode. Solid-state pressure catheters (Sentron) were placed in the abdominal aorta via the left femoral artery, in the ascending aorta via the left carotid artery, and in the left atrium through a small incision. Flow probes (Transonic Systems; Ithaca, NY) were placed on the ascending aorta, the left carotid artery, and the left circumflex artery. The PP was placed through a 14F sheath into the femoral artery. After the measurements with PP support, a 1:1, ECG-triggered, 40-mL IABP (Datascope; Fairfield, NJ) was placed via the same sheath.

The measurement protocol was the creation of acute MR, measurement during unsupported acute MR, activation of the PP at a certain rotational speed or during IABP support, measurements during supported acute MR, relief of acute MR, and stabilization. The IABP measurements were always performed after the PP measurements.

**Acute MR Model**

As an animal model of cardiogenic shock, acute MR with a retrievable cage in the mitral valve was chosen. This model simulates severe cardiogenic shock but is also reversible, which allows multiple propeller rotational speeds to be tested, as well as pumping with the IABP without major deterioration of the hemodynamic state of the animal during the experiment.

An incision was made in the left atrium through which a retrievable steel wire cage ( Günther tulip vena cava filter; William Cook Europe; Bjaeverskov, Denmark) was placed in the mitral valve (Fig 1). The amount of MR was defined as the percentage of left ventricular stroke volume flowing toward the atrium. This percentage was calculated by subtracting the forward stroke volume (measured by an aortic flow probe) from the left ventricular stroke volume (by conductance catheter) and dividing this by left ventricular stroke volume.

**Intraaortic PP**

The new intraaortic PP is a support device with a deployable propeller (Fig 2), which is driven by a flexible central driveshaft. The other end of the driveshaft is a permanent, disk-shaped magnet, which is placed in the driving unit.
consists of a rotating magnet that can be set between zero and \( \leq 14,000 \text{ rpm} \). The PP can only operate continuously, so no triggering is needed.

The PP is placed in the descending aorta in a position similar to the intraaortic balloon. Once this position is reached, the propeller is deployed (Fig 2) and is ready for use. A solution of 20% glucose and 5 IU/mL heparin was used at 25 mL per hour to purge and lubricate the pump.

The propeller is guarded by a basket (Fig 2), which prevents contact between the propeller and the aortic wall. However, the aorta should have a diameter of at least 21 mm to prevent stenting. For this reason, the aortic diameter was measured with fluoroscopy before insertion of the PP. In all animals, the diameter was between 21 and 24 mm.

**Conductance Calibration**

Parallel conductance was determined by injecting 7.5 mL 6.5% hypertonic saline solution into the pulmonary artery. A 5-mL blood sample was collected and blood resistivity was measured by a sampling cuvette (CD Leycom; Zoetermeer, the Netherlands). The slope factor was determined by comparing the conductance-derived stroke volume with the aortic flow prior to the creation of the acute MR.

**Statistical Analysis**

Statistically significant changes between unsupported acute MR and IABP-supported or PP-supported acute MR were tested using a nonparametric, Wilcoxon signed rank test. Significance was assumed if the \( p \) value was \( < 0.05 \).

**Results**

**Instrumentation**

Placement of the new PP was possible through the femoral artery in all animals. Under fluoroscopy, the placement and deployment of the PP could be visualized clearly (Fig 2). On PP activation, a linear relationship between the gradient across the propeller (ie, the mean abdominal aortic pressure minus the mean ascending aortic pressure) and rotational speed was found (Fig 3).

In all animals, severe acute MR could be created with a mean regurgitant fraction of 65% (range, 36 to 79%). This regurgitation resulted in cardiogenic shock with low cardiac output, a decreased aortic BP, and a high left atrial pressure.

**Comparison of Baseline Values**

As the protocol dictated that the PP measurements were performed before the IABP measurements, the PP and IABP baseline hemodynamic state may have been different. To exclude this, a nonparametric Friedman test was performed on cardiac output and on mean ascending aortic BP recorded during unsupported acute MR just prior to the activation of the PP at 6,000, 10,000, and 14,000 rpm or 1:1 IABP support. No significant difference in hemodynamic state could be found.

**Hemodynamic Effects of PP and IABP**

All hemodynamic data are given in Table 1. It shows that the PP at 6,000 rpm had no significant effect on the circulation except for a small increase in abdominal aortic pressure. At 10,000 and 14,000 rpm, clear effects were visible. Afterload, mean ascending aortic pressure, and left ventricular peak systolic pressure were decreased. Left atrial pressure simultaneously decreased, but this did not result in a smaller end-diastolic volume, and therefore left ventricular preload was preserved. At 14,000 rpm, left ventricular contractility was depressed, as seen by the decrease in stroke work and maximal left ventricular pressure derivative (dP/dtmax). Cardiac output was unaffected by PP support, but mean carotid artery flow and diastolic coronary flow were decreased.

Mean coronary flow was maintained, which, together with the decreased diastolic fraction, suggested a shift from diastolic to systolic coronary flow. At 14,000 rpm, coronary resistance was significantly reduced (Table 1).

A number of these observations are seen in the example given in Figure 4, in which ascending aortic pressure, coronary flow, and carotid artery flow are depicted when the PP was switched from 14,000 to 1,000 rpm. Ascending aortic pressure and, especially, diastolic carotid artery flow recovered when the pump was switched off. Coronary flow initially increased but stabilized after 20 s. The shift from systolic to diastolic flow is also evident in this example.
Table 1—Hemodynamic Changes Before and During Support With the PP *

<table>
<thead>
<tr>
<th>Variables</th>
<th>6,000 rpm</th>
<th>10,000 rpm</th>
<th>14,000 rpm</th>
<th>IABP 1:1</th>
</tr>
</thead>
<tbody>
<tr>
<td>HR, beats/min</td>
<td>131 ± 9</td>
<td>133 ± 9†</td>
<td>134 ± 13</td>
<td>137 ± 13</td>
</tr>
<tr>
<td>CO, L/min</td>
<td>2.5 ± 0.8</td>
<td>2.5 ± 1.1</td>
<td>2.5 ± 0.6</td>
<td>2.4 ± 0.7</td>
</tr>
<tr>
<td>LVSV, mL</td>
<td>65 ± 28</td>
<td>66 ± 24</td>
<td>66 ± 30</td>
<td>68 ± 31</td>
</tr>
<tr>
<td>MR, %</td>
<td>62 ± 10</td>
<td>62 ± 11</td>
<td>70 ± 7</td>
<td>72 ± 9</td>
</tr>
<tr>
<td>Qcar, cl/min</td>
<td>45 ± 12</td>
<td>44 ± 11</td>
<td>46 ± 16</td>
<td>43 ± 12</td>
</tr>
<tr>
<td>AOPasc, mm Hg</td>
<td>51 ± 9</td>
<td>50 ± 10</td>
<td>54 ± 11</td>
<td>49 ± 12</td>
</tr>
<tr>
<td>AOPabd, mm Hg</td>
<td>51 ± 9</td>
<td>57 ± 11</td>
<td>54 ± 11</td>
<td>62 ± 12</td>
</tr>
<tr>
<td>Gradient, mm Hg</td>
<td>62 ± 10</td>
<td>62 ± 11</td>
<td>65 ± 12</td>
<td>61 ± 13</td>
</tr>
<tr>
<td>LAP, mm Hg</td>
<td>20 ± 7</td>
<td>20 ± 9</td>
<td>21 ± 11</td>
<td>19 ± 10</td>
</tr>
<tr>
<td>EDV, mL</td>
<td>101 ± 62</td>
<td>101 ± 62</td>
<td>101 ± 58</td>
<td>100 ± 60</td>
</tr>
<tr>
<td>dP/dtmax, mm Hg/s</td>
<td>729 ± 254</td>
<td>741 ± 257</td>
<td>772 ± 263</td>
<td>729 ± 313</td>
</tr>
<tr>
<td>SW, g/m</td>
<td>32 ± 19</td>
<td>33 ± 20</td>
<td>37 ± 23</td>
<td>36 ± 25</td>
</tr>
<tr>
<td>QcorMEAN, mL/min</td>
<td>59 ± 34</td>
<td>61 ± 31</td>
<td>58 ± 29</td>
<td>58 ± 31</td>
</tr>
<tr>
<td>QcorPEAK DIA, mL/min</td>
<td>218 ± 55</td>
<td>210 ± 48</td>
<td>225 ± 46</td>
<td>181 ± 62</td>
</tr>
<tr>
<td>DF, %</td>
<td>89 ± 24</td>
<td>83 ± 27</td>
<td>81 ± 16</td>
<td>67 ± 18</td>
</tr>
<tr>
<td>AOPasc/Qcor, mm Hg/min/L</td>
<td>9.5 ± 5.8</td>
<td>8.6 ± 4.2</td>
<td>9.9 ± 5.9</td>
<td>9.2 ± 5.4</td>
</tr>
</tbody>
</table>

*Values given as mean ± SD. All data shown are values averaged over 10 heart beats at end expiration breath-holding. AOPasc = mean abdominal aortic pressure; AOPasc/Qcor = index for coronary resistance (mean ascending aortic pressure divided by mean coronary flow); CO = cardiac output; DF = diastolic fraction (mean diastolic coronary flow divided by mean coronary flow); EDV = left ventricular end-diastolic volume; Gradient = difference between ascending and abdominal aortic pressure; HR = heart rate; LAP = mean left atrial pressure; LVPmax = maximal left ventricular pressure; LVSV = left ventricular stroke volume; Qcar = mean carotid artery flow; QcorMEAN = mean coronary flow; QcorPEAK DIA = peak diastolic coronary flow; SW = left ventricular stroke work; cl = centiliter.

†p < 0.05.

Switching on the IABP did not change left ventricular function as contractility (stroke work and dP/dt(max)), afterload (maximal left ventricular pressure), preload volume (end-diastolic volume), and left ventricular stroke volume all were unaffected by IABP activation. The main effect of the IABP seems to be a reduction in MR, which had a positive effect on the major hemodynamic parameters (ie, cardiac output, mean aortic pressure, and carotid flow). Furthermore, the counterpulsation increased diastolic coronary flow, although mean coronary flow was unchanged.

**DISCUSSION**

**Afterload Reduction by the PP**

In this study, we investigated a new intraventricular PP for afterload reduction. It shows that the PP is indeed able to reduce the mean ascending aortic pressure by, on average, 10 mm Hg and systolic left ventricular pressure by 6 mm Hg at 14,000 rpm (Table 1). Simultaneously, an increase in abdominal aortic pressure was observed. This gradient between ascending and abdominal aortic pressure is dependent on the rotational speed of the PP (Fig 3) and reached 26 mm Hg at 14,000 rpm (Table 1). From in vitro studies, it is known that this gradient also depends on the aortic diameter, with the PP being less effective in larger aortas. As all animals in this study had comparable aortic sizes (21 to 24 mm), this phenomenon could not be examined.

**Effect of the PP on Coronary Flow**

During PP support at high rotational speeds, diastolic coronary flow is reduced (Table 1). This is a direct result of the lower ascending aortic pressure during diastole. However, net coronary flow is unaffected. This suggests an increased systolic contribution to the flow and thus a reduced diastolic fraction, which is confirmed in Table 1. Two factors are likely to cause this increased systolic contribution to coronary flow. First, we observed a reduction in coronary resistance, which may imply coronary vasodilatation during PP support, as seen in the example in Figure 4. Second, left ventricular systolic pressure is reduced during PP support, which will lower systolic tissue pressure and decrease resistance to systolic flow.

During 1:1 IABP support, diastolic coronary flow is increased (Table 1), while net coronary flow is not. The increase in diastolic flow is known to be caused by counterpulsation. Due to the increase in mean coronary resistance, mean coronary flow does not change. This suggests the presence of vasocostric-
tion, as has been reported before in epicardial arteries in nonischemic hearts.\textsuperscript{10}

The main difference between the IABP and the PP is the counterpulsating vs continuous nature of the pumps. The continuous nature of the PP sucks blood toward the pump during diastole, which explains the reduction in diastolic coronary and carotid artery flow (Table 1, Fig 4). An obvious solution then would be to drive the PP in a counterpulsating manner. However, the driveline and the propeller itself are unable to withstand the forces generated by stopping, let alone reversing the propeller with every heart beat.

In conclusion, the PP and the IABP have opposite effects on coronary flow. The PP favors systolic coronary flow, while the IABP increases diastolic coronary flow. As diastolic flow is crucial to perfuse the subendocardial myocardium,\textsuperscript{11} it is likely that IABP support gives better subendocardial perfusion than does PP support.

**Effect of the PP on Cardiac Output**

Although the PP reduced afterload, this did not result in more cardiac output in this animal model. It is important to realize that in this acute MR model, the amount of MR and thus of cardiac output is determined by the ratio of the impedance of the aorta vs the retrograde impedance during systole. Table 1 shows that the pressure (and thus the impedance) of the aorta is lower but
that left atrial pressure (or retrograde impedance) is lower as well during PP support. The lower aortic pressure therefore does not result in less MR. In contrast, the IABP significantly reduced the amount of MR, which has a positive effect on cardiac output.

While this explains why MR is not decreased on PP activation, it does not answer the following more important question: why is left ventricular stroke volume not increased with lower afterload? First, it should be noted that although the PP creates a pressure gradient in the aorta of ≤26 mm Hg at 14,000 rpm, the average reduction in mean ascending aortic pressure is 10 mm Hg and the reduction in left ventricular systolic pressure is only 6 mm Hg (Table 1). The latter can be explained by the observation that the PP reduces pressure continuously rather than only during systole and the fact that left ventricular afterload is determined by the aortic and the atrial pressure in this MR model.

Still, on PP activation, one would expect stroke work to remain similar with the lower afterload, which would result in more left ventricular stroke volume. This does not occur and consequently, stroke work is reduced, which is statistically significant when the PP is set at 14,000 rpm. Now, the concept of preload recruitable stroke work states that a reduction in stroke work at similar preload (ie, end-diastolic volume does not change) implies less contractility. The decrease in dP/dtmax is also an indication of a loss of contractility.

We therefore conclude that left ventricular stroke volume is not increased with lower afterload because contractility of the left ventricle is reduced with propeller activation at 14,000 rpm. This reduction in contractility may be linked to the observed reduction in diastolic coronary flow.

**Redistribution of Blood by the PP**

As mentioned above, the PP did not increase cardiac output. Without an increase in cardiac output, the PP will merely shift blood from the upper to the lower body. This was confirmed by the significant reduction in carotid artery flow and the increase in abdominal aortic pressure at 10,000 and 14,000 rpm. This has an important clinical implication. In patients who are in cardiogenic shock in whom the PP is applied, cardiac output should be closely monitored, and if cardiac output does not increase, a reduction in flow to the brain should be expected.

**Study Limitations**

In this study, the PP and the IABP were tested in animals with acute MR, which is present in only 10% of cardiogenic shock patients. In patients requiring hemodynamic support due to a different pathology, continuous afterload reduction offered by the PP may be more beneficial. Further animal studies with ischemic heart disease, right ventricular failure, or aortic regurgitation models may answer this question.

Finally, this study was designed as a proof-of-principle study and not as a safety study. However, a propeller rotating at such a high speed in the aorta raises important safety issues such as hemolysis and thrombogenesis. Future studies should address this problem.

**Conclusion**

In calves with acute MR, the intraaortic PP has no significant hemodynamic effect at a setting of 6,000 rpm, and has a negative effect on general hemodynamics at 10,000 and 14,000 rpm. In contrast, the IABP at a 1:1 assist ratio has a profound positive effect on the systemic and coronary circulation. The IABP therefore provides better circulatory support than does the new PP in calves with acute MR.

**REFERENCES**


9 Spaan JA. Mechanical determinants of myocardial perfusion. Basic Res Cardiol 1995; 90:89–102