ORGAN PERFUSION WITH HEMOPUMP DEVICE ASSISTANCE WITH AND WITHOUT INTRAAORTIC BALLOON PUMPING

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Objective: Our objective was to analyze the potential advantage of combining an intraaortic balloon pump with a transthoracic Hemopump device (Medtronic, Inc., Minneapolis, Minn.) (Nimbus Medical, Inc., Rancho Cordova, Calif.). **Methods:** Twelve sheep underwent implantation of a transthoracic Hemopump device and an intraaortic balloon pump. In the first series ($n = 6$), we analyzed the influence of the counterpulsation on the performance of the Hemopump device. In the second group ($n = 6$), hemodynamic changes, myocardial wall thickening, organ perfusion, and myocardial perfusion (determined with colored microspheres) were analyzed under the following conditions: (1) control situation, (2) during application of coronary stenosis, (3) during support with the Hemopump device, and (4) during support with the Hemopump device combined with intraaortic balloon pump support. **Results:** In the first series, we found that addition of counterpulsation reduced output with the Hemopump device by $11.1\% \pm 6\%$. In the second series, it was shown that coronary stenosis significantly reduced contractility (rate of pressure change and wall thickening) but did not cause hemodynamic collapse. Myocardial blood flow was significantly reduced in the poststenotic subendocardial regions (mean subendocardial blood flow dropped from 78 ± 33 to 24 ± 17 ml/min/100 gm; $p = 0.0486$). Support with the Hemopump device alone improved the ratio of subendocardial to subepicardial blood flow, but endocardial underperfusion remained (analysis of variance, $p < 0.001$). The Hemopump device with an intraaortic balloon pump completely restored perfusion in poststenotic regions. Peripheral organ perfusion did not change during ischemia or mechanical support. **Conclusions:** The association of balloon counterpulsation with the Hemopump device reduces the Hemopump output by 11% and increases myocardial blood flow to ischemic regions. Perfusion to peripheral organs remains unaltered. The transthoracic Hemopump device combined with an intraaortic balloon pump is an ideal support system for the ischemic, failing heart. (J Thorac Cardiovasc Surg 1997;114:243-53)

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Intraaortic balloon pumping is an established method of supporting the failing heart. Intraaortic balloon pump (IABP) devices have been continuously improved since Moulopoulos, Topaz, and Kolff¹ introduced counterpulsation by means of an intraaortic balloon in 1962. Experimental work has shown that balloon counterpulsation reduces left ventricular work and increases myocardial perfusion.²⁻⁴ The concept of the Hemopump device (Medtronic, Inc., Minneapolis, Minn.) is completely different. This device is a miniature rotary blood pump introduced into the left ventricular cavity through the aortic valve. It aspirates the blood from

Fig. 1. Experimental protocol setup. 1, Hemopump flow is diverted through Y-graft and measured by electromagnetic flowmeter; 2, intraaortic balloon is inserted through femoral artery.

the left ventricular cavity and expels it in the ascending aorta. The most frequently used and most reliable cannula is the transthoracic cannula, which is introduced through a graft sutured on the ascending aorta. This transthoracic Hemopump device is a powerful pump, with a rotational speed of 26,000 rpm producing a maximum pump flow of 5.1 L/min. The performance of this nonpulsatile blood pump, however, is dependent on the actual pressure differences between inflow and outflow of this pump. With a mean aortic pressure of 70 mm Hg in the clinical situation, the pump thus produces approximately 4 L/min. The Hemopump device increases myocardial blood flow by decreasing ventricular end-diastolic pressure and by increasing mean aortic blood pressure.⁵ Most clinicians prefer the less invasive IABP as the first choice for mechanical support in cases of low cardiac output because the use of the transthoracic Hemopump device requires a sternotomy for both insertion and removal. The transthoracic Hemopump device is considered a

“second-line” mechanical support device for patients who do not recover despite the use of an IABP. An IABP is thus often already in place when the Hemopump device is introduced. The transthoracic Hemopump device and the IABP inserted through the groin can easily be combined, so we studied whether the combination of these devices could be beneficial.

Materials and methods

Animal instrumentation. Twelve sheep (mean weight 69.8 ± 15 kg) were anesthetized with intravenous pentobarbital (3 mg/kg), intubated, and mechanically ventilated with 60% oxygen. Anesthesia was maintained with 0.5% to 2% halothane, and 0.2 mg/kg piritramide was administered every 40 minutes. All animals received humane care in compliance with the “Guide for the Care of and Use of Laboratory Animals” prepared by the Institute of Laboratory Animal Resources and published by the National Institutes of Health NIH Publication No. 86-23, revised 1985.

A triple-lumen central venous line was inserted into the jugular vein, and a fluid-filled pressure catheter was placed in the carotid artery. Both were placed through a cutdown in the left side of the neck. Left thoracotomy was performed through the fifth intercostal space. Heparin was administered at 300 U/kg. When the pericardium was opened, 100 mg lidocaine (Xylocaine) was administered. The left groin was incised and a 40 ml intraaortic balloon (Datascope Inc., Paramus, N.J.) was introduced through the femoral artery. The balloon was positioned in the descending thoracic aorta. A fluid-filled pressure line was inserted in the left atrium and a Mikro-Tip catheter transducer (Millar Instruments, Inc., Houston, Tex.) was placed in the left ventricle through the apex. All pressure transducers were connected to a Triton pressure module (Triton, San Diego, Calif.). Two different instrumentation procedures were performed: one group of six animals was implanted with instruments for accurate measurements of flow through the Hemopump device (protocol 1); the other group of six animals was implanted with instruments for hemodynamic and organ perfusion analysis (protocol 2).

Protocol 1. In the first group of six animals, the ascending aorta was encircled and a 12 mm Dacron polyester fabric Y-graft was sutured to the aorta. A second graft of the same size was sutured to the descending aorta, and the grafts were interconnected with an inflow electromagnetic flowmeter of $\frac{3}{8}$ inch (Cliniflow II, model FM 701 D; Carolina Medical Electronics, Inc., N.C.; Fig. 1). A transthoracic Hemopump cannula was transvalvularly placed in the left ventricle through the side arm of the Y-graft. The side arm was occluded by the occluders provided with the Hemopump device, diverting the produced Hemopump flow through the flowmeter back to the descending aorta (Fig. 1). The metal outflow part of the Hemopump device was positioned in the graft, and a suture around the graft secured the pump in this position and prevented leakage of flow along the Hemopump device into the ascending aorta.

Fig. 2. Experimental protocol. Colored microspheres are injected at five different times. Modes of mechanical support (Hemopump device alone and Hemopump device with IABP) alternated between experiments.

Protocol 2. In the second group of six animals, a 12 mm Dacron polyester fabric graft was sutured to the ascending aorta for introduction of the transthoracic Hemopump cannula. The lateral branch of the left coronary artery was visualized in the atrioventricular groove near its origin. An electromagnetic flow probe was positioned around it and connected to a Nyhon Kohden (Tokyo, Japan) flowmeter. A second electromagnetic flowmeter (model 1401; Skalar, Delft, The Netherlands) was placed around the pulmonary artery for continuous measurement of cardiac output. A set of ultrasonic transit-time crystals consisting of one endocardial crystal and one epicardial crystal was sutured to the posterolateral region of the heart. These crystals were connected to a sonomicrometer module (Triton, San Diego, Calif.) for measurement of wall thickening.

Experimental protocols. In the first protocol ($n = 6$), we wanted to analyze the effect of the IABP on the flow production by the Hemopump device. The flow through the graft (Hemopump flow) was measured for the seven different rotational speeds provided by the Hemopump console. Flow and pressure measurements were analyzed from beat to beat and flows were expressed in relation to pressure. Aortic pressure, left ventricular pressure, first derivative of the left ventricular pressure, electrocardiograph tracing, aortic flow, and Hemopump flow were recorded on a Nyhon Kohden chart recorder. Aortic flow, left ventricular pressure, first derivative of the left ventricular pressure, Hemopump flow and aortic flow were on-line registered on computer by means of Labview software (National Instruments, Austin, Texas). These curves were analyzed and stored in their numeric form. Measurements were repeated with the balloon pump working in an electrocardiographically triggered 1-to-1 mode. At the end of the experiment, the heart was opened to confirm the intraventricular position of the Hemopump.

In the second protocol ($n = 6$), we focused on hemodynamic changes, organ perfusion, and myocardial perfusion during Hemopump support alone and with IABP support. Myocardial wall thickening, left ventricular pressure, first derivative of the left ventricular pressure, arterial blood pressure, coronary flow, cardiac output, and left atrial pressure were continuously recorded on an eight-channel chart recorder (Nyhon Kohden). After stabiliza-

tion, a first set of colored microspheres was injected into the left atrium (Fig. 2).

A stenosis was applied to the lateral branch of the left coronary artery. This stenosis was tightened until a drop of 50% in the baseline coronary flow in that vessel occurred and was then left untouched for 10 minutes. After 5 minutes of stenosis, a second set of colored microspheres was injected through the left atrial catheter and the mechanical support was started (Fig. 2). Five minutes later, a third injection of microspheres was given. The stenosis was relieved and time was allowed for all parameters to stabilize. Then the same stenosis was applied again and a fourth set of microspheres was injected after 5 minutes. This time the second condition of mechanical support was started, and 5 minutes later the fifth set of microspheres was given. The modes of mechanical support (Hemopump device alone and Hemopump device with IABP) were alternated in sequence of use.

Myocardial and organ flow measurement with colored microspheres. Nine million 15 μ m diameter polystyrene microspheres of five different colors (white, red, yellow, blue, and violet) were injected on five different occasions during the experiment. The microspheres were injected in a volume of 3 ml over 30 seconds. Arterial reference blood was withdrawn during 90 seconds from the aorta at a flow rate of 10 ml/min. On termination of the experiment, 1 gm tissue samples were isolated from lungs, cerebrum, cerebellum, spleen, liver, pancreas, stomach, small bowel, large bowel, renal cortex, renal medulla, skin, and muscles.

The hearts were removed, trimmed of excess fat, and cut in five slices along the short axis. Each slice was then cut and unrolled into a myocardial strip (Fig. 3). Three right ventricular and nine left ventricular regions were identified and biopsy samples were taken from each zone from the subepicardium as well as from the subendocardium. The second slice (counting from the base of the heart) from each animal was processed for quantification of the colored microsphere content. The colored microsphere content was determined by the methods described by Kowallik and coworkers,⁶ Rudolph and Heymann,⁷ and Wieland and colleagues.⁸

Data analysis. Computer sampling of the signals was performed at a rate of 1000 samples per second. Logging

Fig. 3. The heart is cut into five slices. Each slice is unrolled into a myocardial strip. Colored microsphere content is analyzed in subendocardial and subepicardial biopsy samples from three right ventricular (*rv*) and nine left ventricular (*lv*) regions. Regions *lv4*, *lv5*, and *lv6* are ischemic regions when the lateral branch of the left coronary artery is stenosed.

Fig. 4. Mean Hemopump flow ($\pm$ standard error) without (closed circles) and with (open circles) IABP for different rotational speeds. Difference is significant when pump is turned off and at speed 1.

of the data covered 1 second of data per log. Thus, depending on the animal's heart rate, one or two heartbeats were analyzed at a time. Data were analyzed with the Statistica statistical software package (Statsoft Inc.,

Tulsa, Okla.). Organ flows and hemodynamic data are presented as means plus or minus the standard deviation. Myocardial blood flow values in different zones of the heart were analyzed with analysis of variance (ANOVA) for repeated measurements. Hemodynamic data were analyzed with ANOVA for repeated measurements and with a one-way multivariate ANOVA for differences between modes of mechanical support. Paired comparisons with control values were performed with the two-tailed paired Student's *t* test.

Results

Influence of the IABP on the performance of the Hemopump device. The IABP reduced the performance of the Hemopump device. Fig. 4 shows the mean flow produced at the different rotational speeds by the Hemopump device and by the combination of the Hemopump device with the IABP. In the control situation, with the Hemopump device switched off, a backflow was measured through the cannula (-0.39 L/min). This backflow was further increased to -0.49 L/min by the IABP (increase of the backflow by 20%). When the Hemopump device was working, the IABP reduced the flow by 11.1% at all rotational speeds. This reduction of the Hemopump flow by the IABP was only significant in the control situation and at low speed.

Fig. 5. Left ventricular pressure, aortic blood pressure, Hemopump flow and electrocardiographic (ECG) tracing during Hemopump run with 1 in 2 counterpulsation assist. Diastolic pressure (*arrow*) augmented by the intraaortic balloon causes a drop in Hemopump flow (*arrow*). Systolic peak flows remain unchanged.

The increased diastolic blood pressure increased the pressure head of the Hemopump device and reduced diastolic pump flow. This is shown in Fig. 5. With the IABP in a 1-in-2 frequency, one can clearly identify the reduced diastolic flow production in the IABP-assisted beat versus the unassisted beat.

Hemodynamic changes, unloading, and contractility. Systolic arterial blood pressure, diastolic arterial blood pressure, cardiac output, and left atrial pressure are shown in Fig. 6. The influence of the coronary stenosis on overall hemodynamics was mild. There was no significant drop in total cardiac output or perfusion pressure. The use of the Hemopump device significantly increased the diastolic

blood pressure (from 55 ± 15 to 75 ± 22 mm Hg; $p = 0.0088$), but the combination of Hemopump device and IABP was even more effective in increasing the diastolic blood pressure (from 56 ± 11 to 88 ± 6 mm Hg; $p < 0.00001$). Left atrial pressures were increased during ischemia and reduced as soon as the mechanical support commenced. There was no difference in the reduction of the left atrial pressure between modes of mechanical support.

The maximum and minimum values of the first derivative of the left ventricular pressure changed significantly during the experiment (Fig. 7). There was a severe drop in contractility when the stenosis was applied, and the heart was further unloaded when the

Fig. 6. Hemodynamic changes during ischemia and mechanical support. **A**, Systolic blood pressure; **B**, diastolic blood pressure; **C**, left atrial pressure; and **D**, cardiac output for Hemopump device alone (*closed circles*) and Hemopump device with IABP (*open circles*). Only diastolic blood pressure during assistance with both Hemopump device and IABP shows significant change (*asterisk*).

Hemopump device was started. The association of the IABP did not influence this pattern. The wall-thickening fraction (Fig. 8) was significantly reduced during ischemia. This measurement of contractility was not altered by assisting the heart with the intraventricular Hemopump device nor with the Hemopump device and IABP.

Peripheral organ perfusion. Peripheral organ perfusions are shown in Table I. The perfusion was measured by colored microspheres before the appli-

cation of the stenosis, 5 minutes after the application of the stenosis, and during mechanical support (with the stenosis still applied). Major changes in pulmonary and splenic perfusion were noted during the different conditions. We consider lung perfusion to be an unreliable measurement in an open-chest experiment, however, because pulmonary manipulations are frequent. Similarly, the ovine spleen act as a blood pooling organ and adjusts for rapid fluid changes. We therefore also do not consider

Fig. 7. Maximum and minimum values of first derivative of left ventricular pressure (dp/dt max and min) for Hemopump device alone (closed circles) and Hemopump device with IABP (open circles). Significant differences from control value are indicated by asterisks.

Fig. 8. Wall thickening of lateral wall of left ventricle for Hemopump device alone (closed circles) and Hemopump device with IABP (open circles). Significant difference from control value is indicated by asterisk.

Table I. Mean organ blood flow in milliliters per minute per 100 gm of tissue ($\pm$ standard deviation)

	Control	Ischemia	HP alone	Ischemia	HP + IABP
Lung	117 $\pm$ 21	146 $\pm$ 81	158 $\pm$ 82	127 $\pm$ 66	248 $\pm$ 123
Cerebrum	72 $\pm$ 28	55 $\pm$ 28	72 $\pm$ 43	84 $\pm$ 66	80 $\pm$ 28
Cerebellum	51 $\pm$ 27	45 $\pm$ 24	54 $\pm$ 10	59 $\pm$ 39	63 $\pm$ 24
Spleen	75 $\pm$ 60	20 $\pm$ 10	36 $\pm$ 11	20 $\pm$ 14	27 $\pm$ 16
Liver	28 $\pm$ 5	19 $\pm$ 10	20 $\pm$ 9	16 $\pm$ 3	19 $\pm$ 9
Renal cortex	202 $\pm$ 40	175 $\pm$ 60	177 $\pm$ 97	180 $\pm$ 70	178 $\pm$ 90
Renal medulla	27 $\pm$ 13	26 $\pm$ 16	23 $\pm$ 8	21 $\pm$ 9	19 $\pm$ 9
Stomach	20 $\pm$ 10	22 $\pm$ 18	31 $\pm$ 17	16 $\pm$ 9	14 $\pm$ 8
Small bowel	42 $\pm$ 15	36 $\pm$ 25	36 $\pm$ 8	31 $\pm$ 10	35 $\pm$ 16
Pancreas	43 $\pm$ 15	37 $\pm$ 15	60 $\pm$ 46	41 $\pm$ 22	44 $\pm$ 18
Large bowel	22 $\pm$ 12	23 $\pm$ 17	25 $\pm$ 20	22 $\pm$ 13	15 $\pm$ 10
Muscle	7 $\pm$ 6	5 $\pm$ 4	5 $\pm$ 4	4 $\pm$ 3	2 $\pm$ 1
Skin	9 $\pm$ 7	7 $\pm$ 6	11 $\pm$ 9	5 $\pm$ 4	5 $\pm$ 3

No significant differences for the different conditions. HP, Hemopump.

the splenic perfusion data to be clinically useful. There were no statistically significant changes in organ perfusion between the control values and the ischemic values (hemodynamics did not change). Also, the mechanical support did not alter peripheral organ perfusion. There was no difference in peripheral organ perfusion between the Hemopump device alone and the combination of Hemopump device with IABP.

Myocardial perfusion. Myocardial perfusion was measured by the colored microsphere technique in different regions of the heart, both in the subendocardium and in the subepicardium. We

thus obtained the subendocardial blood flow, the subepicardial blood flow, and the ratio of subendocardial to subepicardial blood flow. Figs. 9 through 11 show the means of the different animals in the same myocardial regions for three different conditions: (1) control value before the stenosis was applied, (2) the ischemic value measured 5 minutes after application of the stenosis, and (3) the assisted value measured 5 minutes after the start of mechanical support (with the stenosis still in place). These curves are presented for subendocardial blood flow (Fig. 9), subepicardial blood flow (Fig. 10), and the ratio of subendocardial to subepicardial

Fig. 9. Subendocardial blood flow in 12 different regions of myocardial slice (see Fig. 3). Regions from right ventricle are *rva*, *rvl*, and *rvp*; *lv1* through *lv9* are from left ventricle. Each panel shows myocardial perfusion in control situation (closed diamonds), during ischemia (open circles), and during mechanical support (closed squares). Asterisks indicate significant differences from the control value. **A**, Hemopump device with IABP; **B**, Hemopump device alone.

flow (Fig. 11) for assistance with the Hemopump device alone and with the IABP.

After application of the stenosis on the lateral branch of the left coronary artery, the myocardial blood flow was statistically significantly changed in the subendocardial regions (ANOVA, $p < 0.001$). This drop in blood flow was seen in the posterolateral zones of the left ventricle (Fig. 9). The mean subendocardial blood flow in these regions dropped from 78 ± 33 to 24 ± 17 ml/min/100 gm ($p = 0.0486$). The changes in the epicardial blood flow (Fig. 10) were not statistically significant (ANOVA, $p = 0.0704$; mean from 64 ± 26 to 57 ± 40 ml/min/100 gm, $p > 0.2$). As a consequence, the ratio of subendocardial to subepicardial blood flow (Fig. 11) was significantly reduced (ANOVA, $p <$

Fig. 10. Subepicardial blood flow in 12 different regions of myocardial slice (see Fig. 3). Regions from right ventricle are *rva*, *rvl*, and *rvp*; *lv1* through *lv9* are from left ventricle. Each panel shows myocardial perfusion in control situation (closed diamonds), during ischemia (open circles), and during mechanical support (closed squares). Asterisks indicate significant differences from the control value. **A**, Hemopump device with IABP; **B**, Hemopump device alone.

0.001; mean from 1.23 ± 0.11 to 0.48 ± 0.26 , $p = 0.0022$).

Assistance with the Hemopump device improved myocardial flow to a small extent (Figs. 9, A, 10, A, and 11, A). The mean subendocardial blood flow in the ischemic regions rose from 24 ± 17 to 44 ± 40 ml/min/100 gm ($p > 0.2$), the subepicardial flow rose from 45 ± 31 to 55 ± 45 ml/min/100 gm, and the ratio rose from 0.48 ± 0.22 to 0.85 ± 0.59 ($p > 0.2$). For the subendocardial myocardial perfusion, the ANOVA still showed a significant difference from the control situation ($p < 0.001$; Fig. 9, A).

Support with the combination of the Hemopump device and IABP improved myocardial

blood flow to a greater extent (Figs. 9, B, 10, B, and 11, B). Subendocardial blood flow improved from 24 ± 17 to 63 ± 57 ml/min/100 gm ($p > 0.2$), epicardial blood flow remains unchanged from 57 ± 40 to 57 ± 44 ml/min/100 gm, and the ratio increased from 0.48 ± 0.28 to 1.26 ± 1.23 ($p = 0.1750$). As a consequence, there was no significant difference from the control situation (ANOVA for subendocardial flow $p > 0.2$).

Discussion

Clinical practice has led us to the combined use of the transthoracic Hemopump device and the IABP.⁹ In cases of postcardiotomy heart failure, the IABP remains the first choice as a mechanical support device. The device is easily introduced through the femoral artery, has an acceptable complication rate,^{10,11} and has been shown to be beneficial by increasing myocardial perfusion and reducing left ventricular work.²⁻⁴ In cases of persistent low cardiac output despite pharmacologic support and IABP use, we consider the transthoracic Hemopump device to be the first choice as a left ventricular assist device. This pump is powerful (produces up to 5 L/min), also is easily introduced, and has proved to be technically reliable, as opposed to the femoral cannulas of the Hemopump device. With the IABP already in place, we introduced the transthoracic Hemopump device and thus created the combination of these devices. We suspected a possible beneficial effect on myocardial flow and were intrigued by the gradual weaning possibilities (first weaning from the Hemopump device and then from IABP).

This experimental study proves our theoretic hypothesis to be sound. First, the increase in diastolic blood pressure created by the IABP reduced the Hemopump flow. Second, the higher diastolic perfusion pressure created an increase in myocardial blood flow in ischemic regions. With the colored microsphere technique, we showed that the Hemopump device and IABP combination returned subendocardial flow in ischemic regions to normal.

Hemodynamic effects. We did not intend to cause hemodynamic collapse by the applied coronary stenosis. On the contrary, our intention was to show the specific effects of the assisted circulation on myocardial perfusion. In a model of cardiogenic shock, the simple increase of cardiac output and arterial blood pressure by the Hemopump device should itself influence the myocardial perfusion. We therefore preferred to create a stenosis with 50% flow reduction, rather than a complete occlusion. In consequence we did not study

Fig. 11. Ratio of subendocardial to subepicardial blood flow in 12 different regions of myocardial slice (see Fig. 3). Regions from right ventricle are *rva*, *rvl*, and *rvp*; *lv1* through *lv9* are from left ventricle. Each panel shows myocardial perfusion in control situation (closed diamonds), during ischemia (open circles), and during mechanical support (closed squares). Asterisks indicate significant differences from the control value. **A**, Hemopump device with IABP; **B**, Hemopump device alone.

the effects of cardiogenic shock. Total cardiac output and blood pressure did not significantly change during the created "ischemia." The effects of the Hemopump device alone and with IABP support were therefore minimal; there was no change in total cardiac output.

Organ perfusion. We could not identify any effect on peripheral organ perfusion. In this series of experiments, the myocardial stenosis was only applied with the purpose of creating an ischemic subendocardial region. We did not intend to cause myocardial failure and subsequent shock. The so-called "ischemic values"

of peripheral organ perfusion thus do not differ from the control values. However, the fact that assistance with the Hemopump device alone and with IABP did not alter this organ perfusion is important. It proves that the organ perfusion in the assisted situation (with the nonpulsatile Hemopump device) is similar to that in the normal or unassisted situation. Associating an IABP and adding to the pulsatility of the flow did not change the organ perfusion. This finding underlines our clinical experience, where several of our patients with nonpulsatile assistance had good organ function.⁹

Myocardial perfusion. Several series have shown that myocardial perfusion is increased by support with the Hemopump device.^{5,12,13} These experiments in dogs studied myocardial perfusion after occlusion of the left anterior descending coronary artery. Such an occlusion will always impair the hemodynamic status of the animal, and the beneficial effect of the Hemopump device on myocardial perfusion is therefore due in part to the treatment of the cardiac shock. The coronary stenosis used in our experiments did not alter cardiac output or perfusion pressures. The changes in myocardial blood flow in this study were thus due purely to the unloading capacities of the Hemopump device. We caused a significant drop in subendocardial perfusion during ischemia. The Hemopump device improved the ratio of subendocardial to subepicardial blood flow, and this effect was caused by the decreased end-diastolic left ventricular pressure,³ as also illustrated by the decrease in left atrial pressure (from 12.5 ± 4.8 to 8.4 ± 5 mm Hg). Moreover, the Hemopump device strongly unloaded the heart (decrease in first derivative of pressure from 1089 ± 448 to 505 ± 277 ; $p < 0.001$) and therefore reduced myocardial oxygen need. The Hemopump device increased the subendocardial blood flow in the ischemic areas to a small extent (from 24 ± 17 to 44 ± 40 ml/min/100 gm; $p > 0.2$), and a significant underperfusion compared with the control situation remained (ANOVA, $p < 0.001$).

Perfusion pressure (and particularly diastolic perfusion pressure) is the most important parameter for coronary flow in the ischemic, vasodilated vascular bed. The association of the IABP with the Hemopump device combined both advantages, unloading and high diastolic pressure, to create an almost ideal situation. Coronary flow in the ischemic subendocardial region was returned completely to normal (no statistically significant difference from the control situation; ANOVA, $p > 0.2$) and the unloading features of the Hemopump device remain unchanged. In other words, myocardial flow was re-

turned to normal and oxygen need was reduced; one could hardly assist the ischemic heart in a better way.

To validate our experimental results in the clinical situation, the studied animals' weights needed to be similar to those of human beings. Otherwise, the effect of the mechanical support systems could have been overestimated. We therefore chose to use sheep. One anatomic difference between human beings and sheep must be considered in this experimental setup. The ovine descending aorta is relatively small in comparison to the human aorta. This means that the effect on the ovine heart of an IABP in the descending aorta remains rather moderate in comparison with that in the human heart. Taking this into consideration, it is possible that the IABP effect on the heart will be more pronounced in human beings than we were able to demonstrate in these sheep.

This physiologic insight has shown us that the Hemopump device with IABP is an ideal support system in cases of ventricular failure from ischemia. However, the addition of the IABP is not always beneficial. It neither contributes to nor reduces the peripheral organ perfusion, and it reduces the flow produced by the Hemopump device by approximately 10%. In all cases in which the myocardial perfusion is no longer of importance (irreversible damage), the association of an IABP with the Hemopump device is not indicated.

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