Evaluation of an Extraaortic Counterpulsation Device in Severe Cardiac Failure

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A valveless, single-orifice polyurethane ventricle with a maximum stroke volume of 60 mL was implanted on the brachiocephalic artery just above the aortic arch in sheep (n = 14) to act as an extraaortic counterpulsation device. In parallel, an intraaortic balloon was placed in the descending thoracic aorta. Both devices were pneumatically driven with an intraaortic balloon pump console that was gated by the electrocardiogram to provide aortic diastolic augmentation at a stroke volume of 40 mL. To compare the efficacy of counterpulsation for each device during severe cardiac failure, biventricular block was induced by continuous infusion of esmolol (100 to 600 μg · kg⁻¹ · min⁻¹), titrated to reduce aortic flow and pressure to less than 75% of baseline. Pulsatile coronary and aortic flows were recorded with ultrasonic flow probes placed around their respective vessels. Aortic root and left ventricular pressures were recorded using micromanometers. The enhancement of hemodynamic variables for both devices were compared for optimal timing conditions, which were defined as inflation set just before the dicrotic notch and deflation bordering on isovolumetric systole. The extraaortic counterpulsation device was able to significantly augment aortic and coronary flows while simultaneously decreasing left ventricular tension time index and aortic end-diastolic pressure (p < 0.02). The intraaortic balloon pump was able to significantly reduce only tension time index (p < 0.002) to a lesser extent that the extraaortic counterpulsation device. All analysis was performed with the paired-samples t test. The extraaortic counterpulsation device greatly improves the myocardial oxygen supply-consumption ratio of the left ventricle by increasing diastolic coronary flow and reducing left ventricular wall tension during systole. The salutary effects of the extraaortic counterpulsation device greatly exceed those of the intraaortic balloon pump, and it may, therefore, provide a viable alternative for patients in pump failure after cardiopulmonary bypass.

Cardiac assist devices are used in the preoperative and postoperative care of patients undergoing cardiac operations [1, 2] or as bridges for patients awaiting cardiac transplantation. Patients with compromised ventricular function may benefit from drug therapy using digitalis, diuretics, or inotropic agents [3]. If such interventions fail, the only remaining therapy that can be offered is mechanical circulatory support, most commonly provided by the intraaortic balloon pump (IABP). Balloon counterpulsation decreases afterload and therefore myocardial oxygen consumption by the reduction of aortic end-diastolic pressure (EDP) and tension time index (TTI). It improves systemic blood pressure (despite decreased left ventricular pressure) by increasing mean diastolic pressure, which improves myocardial perfusion through the coronary circulation. The overall result is the improvement of the myocardial oxygen supply-consumption ratio of the left ventricle (LV) [2]. Intraaortic balloons (IAB) are also easily placed by percutaneous insertion through the femoral artery. Considerable experience has been obtained with the use of the IABP in the management of patients with coronary artery disease [4–8], but with much less frequency over extended periods of time while a patient is awaiting cardiac transplantation. The IABP is not without serious vascular complications and potential morbidity and mortality [9, 10]. These complications tend to be exacerbated with prolonged support times. Additionally, the IABP can require that the patient remain bedfast in a supine position, which may further reduce his or her strength and stamina.

The major drawback of the IABP is its ineffectiveness in severe cardiac failure when systolic aortic pressure drops to less than 70 mm Hg [11, 12]. This is because IABP counterpulsation produces its hemodynamic effects by altering the aortic input impedance presented to the LV. Hence, increases in aortic flow that result from institution of IABP must be supplied directly by LV output, which is markedly limited in severe cardiac failure. Additionally, the balloon’s presence in the aorta presents an impedance to blood flow during inflation and deflation, which can actually increase afterload if the IABP is not carefully
synchronized to deflate before systole [13]. Finally, the
distal placement of the balloon in the descending aorta
relative to the aortic valve can diminish the pressure pulse
created during diastole [14].

In severe cardiac failure, left ventricular assist devices
are commonly used because they bypass the LV and can
receive blood from cannulation of either the left atrium or
the apex of the left ventricle [15]. Several types of left
ventricular assist devices are available that use some
variation of standard artificial heart chamber technology.
Left ventricular assist devices produce extraaortic coun-
terpulsation directly above the aortic valve by supplying a
fixed stroke volume directly into the aorta. The proximal
location increases the efficacy of counterpulsation, and
the absence of the device in the aorta reduces some of the
timing and impedance problems associated with the
IABP. Although left ventricular assist devices are capable
of supplying a large percentage of total cardiac output,
implantation can be difficult and usually requires cardio-
pulmonary bypass.

The idea of extraaortic counterpulsation is not new, and
extraaortic counterpulsation has been shown previously
to be an effective modality of cardiac assistance. Nose and
associates [16] originally demonstrated that a small
(20 mL), single-orifice Silastic chamber anastomosed to
the aorta provided maximum hemodynamic augmentation
when placed as close as possible to the aortic valve. A
similar concept was used by Bregman [17] to augment
hemodynamics with a compliant chamber in conjunction
with an IABP. Gabbay and Frater [18] demonstrated that
a much larger device (100 mL) could provide dramatic
effects for mongrel dogs in cardiac failure. Nanas and
colleagues [19, 20] demonstrated that a smaller device
(80 mL) could provide significant augmentation when
implanted in the ascending aorta in severe cardiogenic
shock.

The purpose of this investigation was to develop a
device that would address a clinical audience in which
IABP is ineffective. In particular, the device could be used
in patients in whom insertion of an IAB is not possible, to
wean patients from cardiopulmonary bypass or balloon
dependency and possibly serve as a bridge for patients
awaiting cardiac transplantation. The design criteria were
high efficiency, ease of implantation, maximum biocom-
patibility, and low cost. To satisfy these goals, a single-
orifice, valveless, polyurethane artificial ventricle with a
maximum stroke volume of 60 mL was fabricated for use
as an extraaortic counterpulsation device (EACD). The
polyurethane components of the ventricle were vacuum-
formed on simple molds and joined together with radio-
frequency welding [21]. This produces a smooth, flexible
ventricular cavity that is highly biocompatible, easy to
manipulate, and inexpensive to fabricate and can be
pneumatically driven with conventional IABP consoles.
This combination of a flexible polyurethane architecture
and small size also facilitates device implantation. The
single-orifice valveless configuration eliminates potential
blood trauma due to the presence of valve leaflets in the
device. To evaluate the effectiveness of this new EACD,
the device was implanted on the aortic arch of sheep in
severe biventricular failure and its hemodynamic effects
were compared with those of the IABP for identical stroke
volumes.

Material and Methods

To compare the efficacy of counterpulsation for the EACD
versus the IABP, experiments were performed on 14
Suffolk sheep ranging from 50 to 60 kg in weight. After a
48-hour fast and overnight abstinence from water, all
animals were preoperatively sedated with an intramuscu-
lar injection of ketamine (10 mg/kg). General anesthesia
was induced by intravenous pentobarbital (25 to
30 mg/kg). The animals were intubated, mechanically
ventilated, and maintained with inhalation anesthesia
(oxygen 50%, isoflurane, 1.0% to 1.5%). Body tempera-
ture was maintained at 37°C with a recirculating water
blanket. The electrocardiogram was continuously moni-
tored.

Bilateral groin cut-downs were performed to gain access
to the femoral vessels. A 7F micromanometer (PC-350;
Millar Instruments, Houston, TX) catheter was placed
through a femoral artery into the ascending aorta. A 7F
Swan-Ganz catheter was placed through the ipsilateral
femoral vein into the main pulmonary arterial trunk. An
adult 40-mL IAB was placed into the thoracic descending
aorta through the contralateral femoral artery. The animal
was heparinized with 400 U/kg.

The heart was approached through a median sternot-
yomy in the usual fashion and the heart was suspended in a
pericardial cradle. Instruments were inserted into the
animal as diagrammed in Figure 1. A 20-mm transit time
ultrasonic flow probe (Transonics Systems Inc, Ithaca,
New York) was placed around the aortic root. Another
20-mm ultrasonic flow probe (Transonics) was placed
around the aortic arch just distal to the brachiocephalic
artery. The combination of both probes allowed calcula-
tion of aortic root flow, EACD flow, and net systemic
flow. A 4-mm ultrasonic flow probe (Transonics) was
placed around the left main coronary artery after sharp
dissection through the epicardial fat pad. A 5F microma-
nometer (Millar PC-350) was placed into the LV cavity
through an apical stab wound and secured by a 2-0 silk
purserting.

The brachiocephalic artery was isolated and occluded
with a vascular clamp proximally, and tied off with a 1-0
silk suture distally. It was then transected and an end to
end anastomosis was created between the brachiocephalic
artery and the Dacron graft of the EACD with a continu-
ous 4-0 polypropylene stitch. The anastomosis was placed
close to the aorta. The vascular clamp was then intermit-
tently released to fill the EACD with blood, and air was
ventilated with a 25-gauge needle through the Dacron graft.

Severe biventricular failure was then induced using
esmolol (100 to 600 μg · kg⁻¹ · min⁻¹) titrated to reduce
aortic flow to less than 75% of baseline. Esmolol was
delivered at a constant rate by an infusion pump (Lifecare,
model 4P; Abbott Labs, North Chicago, IL) adjusted to
ensure that all preparations had a average heart rate of 100
beats/min.
hemodynamic data before esmolol infusion were collected after all operative procedures were completed. The timing of counterpulsation for both devices was varied under computer control to precisely determine the times of inflation and deflation relative to the electrocardiographic R wave for each beat. Optimal inflation was determined by initially setting inflation to be coincident with the second heart sound and adjusting timing slightly earlier to smoothly obliterate the notch without causing any distortion of the systolic pressure peak. Optimal deflation was initiated at the electrocardiographic P wave and moved later in diastole so as to yield minimum aortic EDP. This point was usually defined as the border of isovolumetric systole. Late deflation was defined as any deflation timed later than optimal deflation that yields increased aortic EDP. The present study focuses on optimal timing and late deflation only; however, during each experiment an extended series of timing runs for both early inflation and late deflation were performed for both devices, in 5-millisecond increments starting from their optimum points to obtain the complete timing dependence of both devices. In each experiment, 200 to 300 runs were collected.

The derived quantities calculated from the observed hemodynamic parameters were TTI (the integral of left ventricular pressure over systole, used as an indirect measure of myocardial oxygen consumption), total coronary flow, total aortic flow, and aortic EDP. Although TTI can be rate dependent, we maintained the average heart rate at 100 beats/min for all preparations as described above. The quantities were determined for unassisted conditions and during counterpulsation obtained with each device. Percentage changes between assisted and unassisted conditions for both devices were calculated for all hemodynamic variables using the customized data analysis program. Group averages & standard errors were calculated for all animals in the study. Statistical analysis was performed on the means using the paired-samples t test to assess the significance of the changes in the hemodynamic variables observed.

All animals received humane care in compliance with the "Guide for the Care and Use of Laboratory Animals" published by the National Institutes of Health (NIH publication No. 85-23, revised 1985).

Results

Table 1 summarizes the observed changes in hemodynamic variable obtained during IABP use. Baseline values are shown before esmolol infusion to demonstrate the level of LV failure present in the preparation. It is evident that for optimal timing conditions during cardiac failure, only the average change in TTI was significant (p < 0.002). The augmentation of coronary flow approached significance (p < 0.085) with an average change of less than 15%. Changes in EDP and aortic flow were both small and of no statistical significance. These results indicate that there is a small degree of afterload reduction of the left ventricle during IABP use in severe cardiac failure and some marginal changes in coronary flow. In general, the hemodynamic effects obtained with the IABP under these conditions were not impressive.
Table 1. Hemodynamic Effects of Intraaortic Balloon Pumping

<table>
<thead>
<tr>
<th>Measurement</th>
<th>TTI</th>
<th>QA</th>
<th>QC</th>
<th>AoEDP</th>
</tr>
</thead>
<tbody>
<tr>
<td>Baseline</td>
<td>30.6 ± 2.8</td>
<td>2299 ± 247</td>
<td>136.9 ± 23.9</td>
<td>71.9 ± 7.7</td>
</tr>
<tr>
<td>Ventricular failure Unassisted</td>
<td>26.6 ± 2.5</td>
<td>1202 ± 159</td>
<td>78.1 ± 16.1</td>
<td>54.4 ± 6.4</td>
</tr>
<tr>
<td>Assisted</td>
<td>25.2 ± 2.0</td>
<td>1238 ± 188</td>
<td>88.2 ± 19.8</td>
<td>55.1 ± 5.6</td>
</tr>
<tr>
<td>% Change</td>
<td>-5.2</td>
<td>2.9</td>
<td>12.9</td>
<td>1.2</td>
</tr>
<tr>
<td>p Value*</td>
<td>0.0019</td>
<td>0.390</td>
<td>0.084</td>
<td>0.695</td>
</tr>
</tbody>
</table>

* p values determined by the paired-samples t test.

Table 2 summarizes changes in hemodynamics for the EACD. In contrast to the IABP, the changes in all hemodynamic variables were significant. Both TTI and aortic EDP were decreased significantly (p < 0.0001) with percentage changes far exceeding those obtained with the IABP. Aortic and coronary flows were both significantly increased by more than 20%, yielding greater augmentation than the IABP. It is important to note that although the absolute changes in aortic flow and EDP are not large enough to maintain adequate systemic perfusion, inotropic agents would be used in conjunction with cardiac assistance to produce adequate changes clinically. In general the results indicate that counterpulsation instituted using the EACD yielded far greater hemodynamic benefits when compared with the IABP for severe cardiac failure in both the magnitude and significance of the observed changes.

Representative tracings of hemodynamic data are used to illustrate the salient changes during EACD and IABP. Figure 2 shows solenoid, pressure, and flow waveforms for counterpulsation obtained with the EACD for optimal timing conditions. The unassisted data for aortic and LV pressures appear normal in shape and phasing. The low peak values of these pressures (approximately 66 mm Hg) indicate severe heart failure. Although the filtering of aortic pressure has removed some of the higher frequency components of the second heart sound, the inflection of the waveform at end-systole is readily apparent. Upon initiation of cardiac assistance using the EACD, optimal inflation occurs just before the second heart sound as indicated from the solenoid timing. This results in a increase in aortic diastolic pressure, which is caused by the chamber ejecting its stroke volume into the aorta. This pressure augmentation is maintained until the onset of isovolumetic systole when a vacuum is applied to the chamber. This causes a rapid filling of the chamber while the ventricle is starting its ejection into the aorta. The volume displacement due to the filling of the EACD causes a decrease in both LV and aortic EDP. Hence, afterload has been substantially reduced by this device due to the vacuum effect of the device in early systole.

The effects of the EACD on coronary and aortic flows are also apparent. For unassisted conditions, the waveforms have their normal shapes and phases when compared with aortic pressure. During EACD assistance, an increase in peak and total coronary flow occurs that coincides with the increase in aortic diastolic pressure. The aortic flow wave is obtained from the flow probe located distal to the brachiocephalic artery. In the absence of cardiac assistance, this waveform has close to a zero baseline and has its typical peak in systole. During assis-
obtained during IABP is increased by a small amount owing to afterload reduction; however, the waveform is not biphasic in nature. The IABP simply causes a volume displacement within the aortic cavity that modulates the afterload presented to the left ventricle. The IABP cannot physically pump blood and will not produce blood flow during diastole, which prevents the formation of a biphase flow wave.

Figure 4 shows the effects of EACD for late deflation timing. From the solenoid timing, it is clear that deflation is initiated during the early ejection phase of systole. In spite of this, afterload reduction still occurs as indicated by the decrease in peak values of LV pressure. The only detrimental effect of late deflation timing using the EACD is an increase in aortic EDP due to the late timing of EACD filling. Hence the filling of the EACD can decrease TTI in spite of the increase of aortic EDP because the chamber acts as a compliant blood reservoir in parallel with the aorta for the ejecting LV. This is in contrast to the late deflation data previously presented for the IABP [13]. Late deflation timing of the IABP will increase TTI due to the presence of the balloon in the aorta, which will impede aortic blood flow and increase LV afterload. Later deflation timing during IABP use can cause large left ventricular pressure spikes owing to aortic obstruction by the IAB.

Comment

Mechanical circulatory assistance is indicated in patients with reversible left ventricular failure in whom drug interventions have been unsuccessful or in those awaiting cardiac transplantation in whom pharmacological support is inadequate. Both the LVAD and the total artificial heart have been used for the temporary support of prospective transplant recipients [15] with varying degrees of success. Devices that can be implanted quickly without a complicated procedure could find use in many emergency situations where the IABP is inadequate such as the inability to wean from cardiopulmonary bypass despite support by the IABP or in cases where a weaned patient remains balloon dependent. In addition, they may also be useful in situations where the balloon cannot be inserted due to technical difficulties. The EACD satisfies these requirements because it can be anastomosed to the aorta with a tangential clamp without the need of cardiopulmonary bypass. Its flexible polyurethane construction and small volume facilitate insertion because the device can be folded up, compressed, squeezed, and held out of the way during anastomosis. The extraaortic configuration of this device does not present the same peripheral vascular complications that are associated with an IAB. In addition, the thoracic location of the EACD may allow the patient to ambulate with the IABP console. Finally, the EACD is a simple modification of a new polyurethane artificial ventricle that has been shown to operate for 6 months in calves without any thromboembolic events [16].

Intraaortic balloon pump counterpulsation has established itself as a dominant support intervention for pa-
patients with a variety of cardiac syndromes [2]. It is, however, limited in severe cardiac failure and cardiogenic shock owing to a number of arterial factors. These factors are the proper size relationship between the balloon and aortic diameter, the compliance of the aorta, and the levels of systolic aortic pressure and cardiac output. The IABP operates in a mode in which it displaces a volume of blood inside the aorta at a reasonable distance from the LV. This can lead to degradation of the IABP pressure pulse arising from energy losses due to arterial compliance. Although the IABP can create a vacuum effect during its deflation in the aorta, this effect is again strongly influenced by the aforementioned arterial factors. Since the IABP cannot physically pump blood, all increases in cardiac output that occur during IABP must be directly supplied by the LV ejection. In cardiogenic shock where systemic blood pressure and cardiac output are both low, the IABP cannot operate efficiently due to inadequate volume of arterial blood available for displacement. The IABP can also be timing sensitive, particularly in late deflation where the balloon can obstruct the aorta and actually increase TTI and myocardial oxygen consumption. This problem can have particularly detrimental effects during irregular rhythms.

In contrast, EACD will not be as strongly influenced by arterial factors relative to the IABP because the device is extraaortic. Aortic compliance will affect device filling to a small extent; however, intraaortic pressures and flows will not have a pronounced effect on the EACD. Although EACD is timing dependent, nonoptimal timing will only reduce the benefits obtained relative to unassisted conditions but will never lead to changes in hemodynamics that could potentially harm the patient.

There is one potential shortcoming that can occur during the institution of EACD. Examination of Figure 2 indicates negative flow waves due to device filling in late diastole. These negative flow waveforms indicate that not enough blood is delivered through the LV in severe cardiac failure and that the EACD requires blood volume from the aortic root. If the LV can supply blood, that volume will be taken up by the EACD and LV work will be reduced owing to the siphoning effect of the device. If LV output is low, blood that fills the device will be derived primarily from the arterial circulation and eventually the EACD will fill and eject the same volume of blood into the aorta. This increases the probability of clot formation from stasis in the low-velocity regions of the device owing to a lack of constant “washing” of the EACD with fresh blood. In this regard, both the IABP and the EACD are limited in profound LV failure because both devices derive their blood volume entirely from the LV, although the EACD was clearly more effective for the conditions tested in this study. In these instances, a left ventricular assist device is indicated because it can bypass the LV to obtain a constant fresh blood supply, which will eliminate the possibility of clot formation due to poor washing of the device and maintain adequate systemic perfusion.

This study has demonstrated the effectiveness of the EACD in severe cardiac failure. The extraaortic placement of the device just distal to the aortic valve helps to reduce the dependence of device performance on arterial factors. The EACD is not located directly in the aorta and therefore error in timing adjustments in the deflation phase will only reduce the amount of afterload reduction produced by the device but will never increase afterload greater than unassisted conditions. The EACD can physically deliver and remove a blood volume from the aorta that helps to increase its efficacy of counterpulsation. The results of this study have demonstrated that the EACD has an average beneficial effect on each individual variable of 2 to 10 times that of the IABP. If one estimates the myocardial oxygen supply–consumption ratio using cor-

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**Fig 4.** Solenoid timing, aortic pressure, and left ventricular pressure waveforms versus time in seconds for counterpulsation using the extraaortic counterpulsation device when set for late deflation timing conditions. Pressure is in millimeters of mercury. All counterpulsation was performed during cardiac failure.
coronary flow as an estimate of oxygen supply and TTI as an estimate of consumption, the EACD increases this ratio by approximately 6 times that of the IABP. This added efficacy of the EACD may prove to be useful in extreme hemodynamic deteriorations, similar to those used in this study.

In summary, the IABP has established itself as an important support modality for patients with cardiac failure. Its ease of insertion and widespread clinical experience have made the IABP an irreplaceable intervention. However, there are instances in which the insertion of the IABP is not technically feasible and situations in which the level of support is inadequate for the severity of cardiac failure. These are the situations in which the implantation of an EACD may be of clinical importance. This study demonstrates the effectiveness of the EACD implanted on the ascending aorta in cases of severe cardiac failure by reducing afterload, augmenting the coronary flow, and consequently, dramatically improving the myocardial oxygen supply-consumption ratio. In addition, the results demonstrated here support the institution of the EACD in situations in which the IABP cannot adequately support the circulation. The use of the EACD deserves further chronic animal studies followed by clinical trials.

References

DISCUSSION

DR SHLOMO GABBAY (Newark, NJ): I congratulate Dr Zelano and associates for this interesting confirmation of the previously widely published work on the extraaortic balloon pump.

I hope you are aware that the anatomy of a sheep’s aortic arch is very different from that of a human. The sheep has only one large arch vessel that supplies the head and upper limbs. One creates some difficulties by connecting the extraaortic balloon pump to the only arch vessel the animal has: first, you end up killing the animal before termination of the experiment, and second, this arrangement alters the physiology, thus forcing the pump to function in an unnatural environment. Setting up the apparatus in this fashion leaves the investigator with no control of how much blood from the head and upper limb will be affected by the counterpulsation. Why did you not connect the device on the ascending aorta end-to-side?

At this time, I would like to say that the extraaortic balloon pump connected to the ascending aorta is indeed overwhelmingly superior to the standard IABP. My study of the extraaortic balloon pump, published in ASAIO Transactions in 1981, clearly showed that bringing counterpulsation closer to the aortic valve can increase coronary flow by as much as 300%. This is striking when one realizes that the IABP only augments coronary flow by...
reproduced the same results in their laboratories and helped to prove the superior effectiveness of the extraaortic balloon pump. I must confess that I do not understand why the concept is not practiced widely in the clinical setting, and that I was unsuccessful in convincing a commercial outfit to market this device. Hopefully more publications like yours will help to speed the news of this remarkable pump. I have no doubt that in cases where the IABP is not beneficial to the patient, the extraaortic balloon pump will be more effective in helping the heart to recover from temporary ischemia.

DR ROBERT A. GUYTON (Atlanta, GA): I think Dr Kantrowitz and others would object to your calling this a new device, because this is the auxiliary ventricle that he talked about in a counterpulsation mode almost 30 years ago. I agree with the prior discussant that this has been presented before. This is clearly a refinement and a modification of this device using the concepts that we have learned from left ventricular assist devices and associated new technology.

DR ZELANO: It is also a much smaller volume than previous studies. All other studies were done using approximately 70 to 80 mL with some as high as 100 mL. This device is 40 mL; it is effective with a volume as small as 30 mL and can very easily be implanted. The other devices that were made were not practical clinically.

In response to Dr Gabbay's question, the enhancement of the hemodynamics in this study was not as pronounced as in previous studies because we performed our experiments on large animals, approximately 70 to 80 kg with a small device volume of 40 mL. Previous studies used an 80- to 100-mL device on 15- to 20-kg dogs. The great disparity in total blood volume alone more than accounts for the difference we see. Similarly, the differences between the EACD and the IABP were not as pronounced because we used equal displacement volumes for both devices.

Our use of the brachiocephalic artery in sheep was intended to make the operation more technically feasible because of the short ascending aorta in sheep. This configuration also enabled us to measure total aortic flow past the EACD. No flow is shunted to the brachiocephalic because the EACD is implanted there. The increase in sympathetic discharge from the brain is reduced by esmolol as well as ventricular contractility and the entire peripheral vasculature is dilated. We believe that we have produced worst-case conditions for testing the efficacy of counterpulsation, and both devices were alternately tested under identical conditions. Hence this study is representative of what one might obtain clinically.

INVITED COMMENTARY

Dr Zelano and associates have presented additional proof that the EACD or extraaortic balloon counterpulsation pump (EABP) is more efficient than the IABP.

In 1981 we [18] published a thorough comparative study of the EABP and the IABP. Since that time many other groups have continued the research and have published similar studies with practically identical results. It is clear that by bringing the counterpulsation closer to the heart one can increase the coronary flow substantially at the time of diastole and unload the heart at systole in a manner never seen with the IABP. The IABP has several limiting factors that can be overcome by the EABP, and there is no doubt that in many cases when the IABP fails to sustain life a simple valveless device like the EABP can improve the survival rate. As we have seen in our studies the coronary flow can be increased more than 100% using the EABP, compared with 5% to 15% using the IABP.

If the EABP device is so good, why did it not reach clinical use? Indeed, there are several reasons: the device can only be implanted by sternotomy and cannot be introduced through a catheter. For patients in cardiogenic shock, most physicians are reluctant to insert an assist device because it is a relatively invasive procedure, although this fear could be overcome if such a device was widely used and well known.

Second, there is some reservation concerning blood clot formation in a device positioned in the inflow of the large arch vessels. Presently there exist no long-term animal data to preclude this concern, and it is apparent that research must be conducted before any clinical use can be contemplated. If the patient is partially anticoagulated, however, there should be no immediate problem, and many IABPs are inserted through the ascending aorta with no sign of embolic events.

Additionally, the surgical anastomosis between the device and the aorta has not been worked out to a satisfactory level. The width and the length of the cannula are unknown factors. Dr Zelano and associates were not able to eliminate this fear in their recent study because they have cannulated the brachiocephalic artery of the sheep (the only arch vessel supplying the brain and the anterior limbs), which has a relatively large diameter. This vessel is not available in humans. The ascending aorta can be crowded after coronary bypass; therefore, additional research must be performed in this major technical area.

Finally, the industry does not have any "real" interest in sponsoring such a device and government funding was not granted in many instances.

I can only congratulate Dr Zelano and associates for continuing to work on this interesting device. Our group is working toward solving some of the technical/feasibility problems of the EABP. This instrument will, without a doubt, enter the clinical arena proving that patients in cardiogenic shock, who did not respond to the IABP, survived because the EABP was available and used. We must remember that there is nothing wrong in inserting a simple device that all investigators agree can sustain life—especially when the IABP has failed and the EABP can be activated by a widely available balloon pump machine. The patient has nothing to lose!

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