A VALVELESS HIGH STROKE VOLUME COUNTERPULSATION DEVICE RESTORES HEMODYNAMICS IN PATIENTS WITH CONGESTIVE HEART FAILURE AND INTRACTABLE CARDIOGENIC SHOCK AWAITING HEART TRANSPLANTATION

The paraaortic counterpulsation device is a round pumping chamber with one valveless opening 20 mm in diameter and a 100 ml stroke volume. The paraaortic counterpulsation device was implanted on the ascending aorta of three male patients with intractable cardiogenic shock. Patients were assisted for 4 hours and 8 and 54 days, respectively; the first patient died as a result of nonresponding peripheral vasodilation and the other two died of septic shock. The two patients who were assisted for 8 and 54 days were conscious and able to function in a limited manner during the mechanical assistance. Discontinuation of the mechanical support for a few seconds was followed by low systolic arterial pressure (30 to 60 mm Hg) and syncopal episodes. Biochemical tests and autopsy results in these patients showed no evidence of blood cell destruction, thrombus formation, brain infarction, or other distal emboli. In conclusion, satisfactory hemodynamic effects, excellent biocompatibility, and simplicity of the implantation procedure in these patients encourage the use of the paraaortic counterpulsation device as a bridge to heart transplantation. (*J THORAC CARDIOVASC SURG* 1996;111:55-61)

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The high mortality rate of patients with cardiogenic shock and the limited number of donor hearts¹⁻⁷ necessitate the use of assisting devices that have the capacity of undertaking most or all the work of the failing heart.⁸⁻¹⁷ The complexity of the existing devices and the attendant high risk and complications^{13, 16-18} stress the need for continuation of investigation for new developments. The paraaortic counterpulsation device (PACD) is a new high stroke volume counterpulsation device.¹⁹⁻²⁰ It has been more effective in the failing heart than the intraaortic balloon pump (IABP) when implanted on the abdominal aorta.²¹ Its implantation on the ascending aorta was effective in restoring the hemodynamics in severe experimental cardiogenic shock^{22,23} and became ineffective only when the

systolic arterial pressure was low. It is noteworthy that a counterpulsation device is more effective when implanted on the ascending rather than on the abdominal aorta.²⁴ The biocompatibility of the PACD in chronic experiments was shown to be better than that of the total artificial heart.²⁵ In this report, the implantation procedure and the hemodynamic effect and biocompatibility of the PACD in its first clinical applications are evaluated.

Patients and methods

PACD. The PACD (Fig. 1) is a round pumping chamber connected by a screw-connector to a spiral polytetrafluoroethylene graft 3 to 5 cm in length with one valveless orifice 20 mm in diameter. It has a 100 ml stroke volume when fully inflated and an ejection fraction of 65%. The air space of the device is separated by three polyurethane filaments from the blood space. It was made with the use of the same technique and materials used for the Utah Artificial Heart (Symbion, Inc., Salt Lake City, Utah). A gas conduit leads from the pumping chamber to the Datascope 82 driving system (Datascope Corp., Montvale, N.J.). This driving system pumps at a 7 to 8 Pi driving pressure and almost zero vacuum pressure. The zero vacuum pressure was obtained by means of a safety valve added on the Datascope 82 driving system. This valve was electrically adjusted to open during the vacuum phase of the driving system. The driving Datascope 82 system was adjusted on the base of the electrocardiogram to provide aortic diastolic augmentation. During the sys-

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Received for publication Dec. 27, 1994.

Accepted for publication March 16, 1995.

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0022-5223/96 \$5.00 + 0 12/1/64938

Fig. 1. Schematic presentation of the PACD.

tolic phase of the left ventricle, the pressure into the device was near zero, thus blood from the left ventricle and the aorta entered the device; during the diastolic phase of the left ventricle, blood was ejected from the device into the ascending aorta.

Implantation procedure. After midsternotomy, the chest was opened and the graft was anastomosed to the right lateral wall of the ascending aorta at a 30- to 45-degree angle with the proximal part of the aorta. The air conduit was placed subcutaneously and was brought out on the anterior lateral chest wall at the level of the seventh intercostal space. The gas conduit was connected to the intraaortic balloon driving system. The vacuum pressure was near zero, but for safety purposes an electrically triggering exit valve was placed and activated when the driving system was in the vacuum phase. The R wave was detected by means of a lead of the surface or the epicardial electrocardiogram, and the driving system was synchronized to provide aortic diastolic augmentation.

Patients. Three men, 57, 32, and 33 years of age, had cardiogenic shock which was intractable to conventional treatment including the IABP and were assisted by the PACD for 4 hours and 8 and 54 days, respectively.

Results

Patient No. 1. A 57-year-old man underwent emergency coronary artery bypass grafting because of recent myocardial infarction and episodes of angina at rest after myocardial infarction. He received two vein grafts on the left anterior descending coronary artery and the first diagonal branch. He was weaned from extracorporeal circulation with the assistance of the IABP, and high doses of adrenaline infusion (40 μ g/min) were given. He was assisted continuously with the IABP for 2 days, but higher doses of inotropic support were required to keep

him alive. During this period he was clinically in cardiogenic shock with systolic arterial pressure less than 85 mm Hg, had a heart rate of more than 130 beats/min, was totally anuric for the last 24 hours, and had an arterial oxygen tension around 63 mm Hg (inspired oxygen fraction = 80% and peak end-expiratory pressure = 10 cm H₂O). At that time the PACD was implanted to interrupt the vicious cycle of the shock. Anesthesia, induced to implant the PACD, resulted in intractable peripheral vasodilation; even higher doses of adrenaline infusion did not restore hemodynamics, and the patient died 4 hours after PACD implantation. After the patient's death, no clots were found within the device. Infections were not detected around the device or around its driving line.

Patient No. 2. A 32-year-old man with a 6-month history of progressively impaired dyspnea and ankle edema was admitted to our hospital because of intractable heart failure and recurrent syncopal episodes caused by ventricular fibrillation. Two months before his last admission, while he was receiving medical treatment with digoxin 0.25 mg/day, enalapril 5 mg/12 hours, furosemide 160 mg/day, and potassium chloride, he was evaluated for heart transplantation. His electrocardiogram showed normal sinus rhythm, low voltage in limb leads, and nonspecific ST-T changes. The chest roentgenogram showed an obvious congestion and enlarged cardiac shadow. The echocardiogram showed a markedly dilated left ventricle (left ventricular end-diastolic dimension = 78 mm) and left atrium (57 mm),

regurgitation of the mitral and tricuspid valves, and markedly decreased contraction of the left and right ventricles. Ejection fraction was 9% by nuclear ventriculography. His hemodynamics were indicative of left and right ventricular failure (Table I), and his right ventricular biopsy specimen was indicative of cardiomyopathy. Forty days before his last admission he had pneumonia, and during hospitalization he had cardiogenic shock with pulmonary congestion, abdominal pain, and ankle edema requiring high doses of dobutamine (15 $\mu\text{g/kg/min}$) plus dopamine (15 $\mu\text{g/kg/min}$). During that period he also had recurrent syncopal episodes caused by ventricular fibrillation. The patient was transferred to our hospital to undergo mechanical assistance as a bridge to heart transplantation. On admission he was found in cardiogenic shock with orthopnea, abdominal pain, generalized edema, and a large decubitus ulcer. The combination of high intravenous doses of furosemide and high doses of inotropic drugs initially stabilized the patient's condition, but on hospitalization day 5 he had cardiogenic shock requiring mechanical assistance with the IABP. Discontinuation of the IABP was necessary after 10 days of mechanical support because of severe leg ischemia, which was followed by relapse of the cardiogenic shock with mental deterioration and anuria. At that time the patient was not responding to high doses of dobutamine and dopamine, and adrenaline infusion was required to keep him alive. Finally, the PACD was implanted. Partial clamping of the ascending aorta during graft anastomosis did not affect the patient's hemodynamics. After the operation, normal hemodynamics and diuresis were restored. Heparine 1200 IU/hr was administered intravenously after the first 8 postimplantation hours up to postimplantation day 3, and thereafter the patient received a low dose of acenocoumarol to maintain a prothrombin time of 1.4 to 1.6 times the control value. The patient was extubated 24 hours after implantation and was well, communicating, and capable of limited function during the period of mechanical assistance. Here it must be pointed out that, 4 days before PACD implantation, blood cultures showed multiresistant *Acinetobacter calcoaceticus*. The same microorganism was grown in the blood cultures on postimplantation day 3. He died on postimplantation day 8 of septic shock. No hemolysis or platelet destruction was detectable during the whole period of mechanical assistance with the device, and autopsy showed no evidence of either distal embolus in any organ or

Table I. Right heart catheterization of the second patient (2 months before his last admission) and the third patient (hospitalization day 6)

	Patient	
	Second	Third
Age (yr)	32	33
Right atrial pressure, mean (mm Hg)	11	17
Right ventricular pressure, systolic/ end-diastolic (mm Hg)	65/10	58/15
Pulmonary artery pressure, systolic/ diastolic/mean (mm Hg)	65/34/40	58/28/46
Pulmonary capillary wedge pressure, mean (mm Hg)	33	33
A-VO ₂ difference (cm ³ %)	3	10.4
Arterial blood pressure, systolic/dia- stolic/mean (mm Hg)	90/70/78	105/ 70/80
Cardiac output (L/min)	5.8	2.3
Systemic vascular resistance (IU Wood units)	11.4	27.4
Pulmonary arterial vascular resis- tance (IU Wood units)	1.2	4.7

A-VO₂, Arteriovenous oxygen difference.

thrombus formation into the body of the PACD. No infections of the tissues around the device or the driving line were present.

Patient No. 3. A 33-year-old man was admitted to our hospital because of orthopnea, abdominal pain, weakness, and ankle edema. The patient had been free of symptoms up to 2 years before admission when he was hospitalized for pulmonary edema. After initial improvement, he had exertional dyspnea that became progressively worse, and for 2 months before his current admission he had paroxysmal nocturnal dyspnea, ankle edema, fatigue, and weakness.

On admission the clinical examination showed a well-developed, well-nourished orthopneic male patient in acute distress. Blood pressure was 90/50 mm Hg, pulse rate was 140 beats/min and regular, respiration was 20 breaths/min, jugular venous pressure was estimated to be more than 20 cm H₂O, and the temperature was 38° C. He had no cyanosis. A systolic low left parasternal heave was palpable, and the apical pulse was also palpable on the fifth and sixth intercostal spaces to the left of the midclavicular line and was sustained and increased. Physical signs of left and right heart failure and mitral regurgitation were present. The electrocardiogram showed normal sinus rhythm, left ventricular hypertrophy, and intraventricular conduction defect. Chest roentgenograms showed a markedly enlarged heart. The echocardiogram showed markedly di-

Fig. 2. Tracing of the aortic pressure, obtained from the third patient with the PACD, functioning on every heart beat for the first four cardiac cycles and cessation of its function after that, and the IABP, functioning on every other heart beat. It is obvious that the IABP alone was ineffective and that the simultaneous use of the two devices provided higher aortic diastolic augmentation than any of the devices alone. *AOP*, Aortic pressure; *S*, systolic pressure; *I*, aortic diastolic augmentation provided by the IABP; *P*, aortic diastolic augmentation provided by the PACD; *P+I*, aortic diastolic augmentation provided by the PACD and the IABP.

lated left (left ventricular end-diastolic dimension = 78 mm) and right ventricles (right ventricular end-diastolic dimension = 36 mm) and left atrium (44 mm), regurgitation of the mitral and tricuspid valves, and diffuse and markedly decreased contraction of the left and right ventricular walls. Radionuclear ventriculography showed a left ventricular ejection fraction of 6%. Coronary arteriography before admission had shown normal coronary arteries and diffuse and markedly decreased contraction of the left ventricle with severe mitral regurgitation. The patient received high doses of diuretics (230 ± 290 mg/day, intravenous furosemide) and dobutamine (8 ± 3 μ g/kg/min). After 6 days of intensive treatment his condition improved clinically, and he underwent right heart catheterization (Table I). On hospitalization day 11, patient was supported by mechanical assistance with the IABP because of cardiogenic shock. He responded with significant improvement, which lasted for 9 days, after which the patient again showed signs of progressive cardiogenic shock despite assistance with the IABP, high doses of dobutamine, and additional administration of high doses of dopamine. The use of adrenaline drip was necessary to keep the patient alive. The creatinine level increased to 3.6 mg/dl, and urea was 250 mg/dl, while potassium was 6.4 mEq/L, and sodium was 120 mEq/L. At that time, on hospitalization day 21, the PACD was implanted. During the assistance with the IABP pump, heparin 1200 IU/hr was administered intravenously and was continued to be administered on the same dose after

Fig. 3. A chest roentgenogram of the third patient after implantation of the PACD seen on the right side of the cardiac shadow. The IABP is also seen.

the implantation of the PACD. Partial clamping of the ascending aorta during graft anastomosis did not affect patient's hemodynamics.

During the first 4 hours the patient showed impressive hemodynamic improvement with an increase of mean aortic pressure from 52 to 85 mm Hg and a urine output to 100 ml/hr starting from anuria. Six hours after the operation the course was complicated by massive mediastinal bleeding which resulted in oligemic shock. Heparin administration was discontinued for 12 hours. Thereafter, heparin was administered up to postimplantation day 3, and afterwards the patient received low-dose acenocoumarole to maintain a prothrombin time of 1.4 to 1.6 times the control value. Normal hemodynamics were restored after 4 hours with blood transfusion and crystalloid fluid administration; however, the patient had acute renal insufficiency and later required peritoneal dialysis which lasted for the rest of his clinical course. On postimplantation day 43 the patient had pneumonia and, after that, septic shock; he died 54 days after PACD implantation.

The patient was assisted for 3 days with two devices, the PACD and the IABP (Fig. 2). The IABP was withdrawn on postimplantation day 3 (Fig. 3), and the patient was assisted only with the PACD thereafter (Fig. 4). During the 54 days of mechanical assistance, interruption of function of the PACD for a few seconds was followed by syncopal episodes with low systolic arterial pressure

Fig. 4. *Upper tracing:* Radial arterial pressure (RAP), obtained on the day after implantation from the third patient, with the PACD functioning as follows: (1) on every heart beat, (2) on every other heart beat, (3) on every third heart beat. *Lower tracing:* RAP, obtained on the day after implantation from the third patient, with the PACD functioning on every heart beat followed by temporary interruption. DP, Driving pressure; S, systolic pressure; P, aortic diastolic augmentation provided by the PACD.

(30 to 60 mm Hg) (Fig. 4). In this patient, because of the prominent right heart failure, the administration of inotropic agents was necessary to keep adequate hemodynamic conditions. The patient was able to communicate and had minimal function (e.g., he could eat and sit in a chair).

No hemolysis was detectable, and hematocrit value was 40% before device implantation, was 36% after the first week, and remained on the same level during the entire course of assistance with the PACD. Lactate dehydrogenase had not increased. Indirect bilirubin was within normal limits, and plasma-free hemoglobin was undetectable after the first postimplantation week.

Extensive autopsy showed a right lung infiltrate (lower lobe). No evidence was found of distal embolus in any organ (brain, kidneys, etc.) or of thrombus formation within the "cavity" of the PACD. The device was easily removed from its implantation side, and no infections were detected on the tissues surrounding the device and the driving line.

Discussion

This article describes the initial clinical experience with the PACD. Devices which perform on the same principles include the following: The IABP, which has become the most popular assisting device, the implanted mechanical auxiliary ventricle, and the dynamic aortic patch.^{26, 27} With the exception of the IABP, the other devices are no longer in clinical use. These devices required a major operation for their implantation, and the obtainable hemodynamic benefits were similar to those of the IABP.

The implantation procedure of the PACD in these patients has proven to be a simple and fast procedure. The implantation was performed with partial clamping of the ascending aorta and required about 30 minutes from the time that the aorta was clamped until the device started assisting the patient. The partial clamping of the aorta was not followed by hemodynamic compromise. Its hemodynamic effects, even when the device was implanted on the abdominal aorta, were superior to those of the IABP.²¹ A similar device implanted on the

brachiocephalic artery in sheep proved to have salutary hemodynamic effects greatly exceeding those of the IABP.²⁸ Furthermore, the implantation of the PACD on the ascending aorta is expected to provide further hemodynamic improvement.²²⁻²⁴ However, this matter necessitates further investigation. The biocompatibility of the PACD has been shown to be better than the total artificial heart in experimental animals.²⁵ In this limited clinical experience the hemocompatibility was excellent according to the hematologic and biochemical tests. Autopsy results were negative for infarctions on any organ or thrombus formation in the device. The absence of valves in the device may have accounted for these effects. The recommended anticoagulation therapy could be as follows: (1) intravenous dextran-40, 10% solution at the rate of 50 ml/hr, should be started 2 hours before implantation and continued for 12 hours after the implantation. Intravenous methylprednisolone (250 mg) should be administered to prevent potential anaphylactic reactions to dextran. (2) The device must be prefilled with heparinized (5000 IU) dextrose 5% before its connection to the graft. (3) When dextran is discontinued, continuous infusion of heparin 1200 IU/hr must be given for 3 days. (4) Thereafter, patients must be treated with aspirin 100 mg/day and acetocoumarole to maintain a prothrombin time of about 1.5 times of the control value.

It is noteworthy that the effect of the PACD on human blood cells seems to be better than expected compared with that of the experimental animal studies. This effect is probably due to higher thrombogenicity of animals compared with human beings.²⁹

These findings and the fact that two patients remained without anticoagulants for up to 12 hours leads to the assumption that anticoagulant therapy is probably not absolutely necessary for this device. On the other hand, the mechanical auxiliary ventricle, after its implantation in two patients, was not used further because of thromboembolism from the prosthesis.²⁶⁻²⁷ Regarding the hemodynamic effects, it was impressive to note that PACD was able to restore hemodynamics and keep the patient alive for up to 54 days while the left ventricle without the mechanical support could not generate a systolic blood pressure higher than 30 to 60 mm Hg. Two of three patients, while assisted with the PACD, were conscious and able to communicate well, speak, and eat, and the patient with the longest survival was able to stand up and sit in a chair. The PACD assisted the failing left ventricle to a degree almost

equal to that provided by other left ventricular assist devices. However, this device cannot take over all of the work of the ventricle for more than a few minutes.³⁰ On the other hand, the total artificial heart and some ventricular assist devices can substitute the work of the heart. Some common problems of the ventricular assist devices and the total artificial heart are the major operation required for their implantation, the technical challenge and the time required for insertion, the increased cost, and the high risk for thromboembolic episodes. The presence of prominent right heart failure in the third patient required the administration of inotropic agents to preserve adequate hemodynamics. However, in cases of severe biventricular failure with evidence of systemic congestion and cardiogenic shock, the biventricular support systems are probably more suitable. Implantation of the PACD in three patients and assistance for up to 54 days provided some evidence that it can overcome some of the problems of the ventricular assist devices and the total artificial heart. It also points out the stronger effect of large-volume counterpulsation versus the smaller volume achieved by the IABP. It seems that this device has a place in the armament of the left ventricular assist devices, and it may be preferable to other devices in cases of intractable heart failure or severe cardiogenic shock (systolic arterial pressure more than 50 to 60 mm Hg) with predominant impairment of the left ventricular function. The earlier in the patient's course the PACD is implanted, the better the outcome of the patient is expected to be because the patient will recover easier from the implantation procedure and the hemodynamic improvement will prevent the failure of other organs.

The PACD implanted on the ascending aorta of three patients proved to be able to restore hemodynamics for up to 54 days, and its biocompatibility was excellent. These results showed the significance of using high stroke volume for the effectiveness of the counterpulsation technique and encourage the use of the PACD as a bridge to heart transplantation.

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