Intraaortic Balloon Counterpulsation in a Community Hospital*

Jang B. Singh, M.D.; Pamela Connelly, R.N.; Stanley Kocot, M.D.; Peter Kotilainen, Ph.D.; Willard M. Daggett, M.D., F.C.C.P.; and Mortimer J. Buckley, M.D., F.C.C.P.

Our experience with intraaortic balloon counterpulsation in 41 patients documents the feasibility of a coordinated aggressive therapeutic approach in patients with potentially fatal complications of coronary artery disease. The use of intraaortic balloon pump in a non-cardiac surgical center has allowed hemodynamic support in the critically ill, safety in angiography, appropriate selection of patients for surgery, and safe transportation to a cardiac surgery center when indicated.

The intraaortic balloon pump (IABP) was first applied in experimental myocardial infarction in 1962. Most of the early clinical work in this field was, however, not accomplished until 1971 to 1973. Intraaortic balloon counterpulsation has been shown to improve myocardial oxygen supply by increasing aortic perfusion pressure, and hence, coronary blood flow, to decrease myocardial oxygen consumption by decreasing afterload and to improve peripheral perfusion pressure without increasing the cardiac work load. The earliest application of intraaortic balloon counterpulsation was in the management of patients with cardiogenic shock. As it became apparent that the fate of the ischemic zone in myocardial infarction depended upon the delicate balance between myocardial oxygen demand and supply, and that salvage of the ischemic myocardium was possible, the hemodynamic effects of intraaortic balloon counterpulsation seemed ideally suited for application to high risk myocardial infarction patients. With additional experience, the hemodynamic and metabolic effects of counterpulsation were also found to be desirable in the management of severe left ventricular dysfunction after cardiac surgery, management of unstable angina, and also for stabilizing patients with myocardial infarction who continue to have persistent evidence of ischemia.

The purpose of this report is to present our experience with intraaortic balloon counterpulsation in a community hospital where, at present, inhouse cardiac surgery is not available. This report is also presented to establish the feasibility of providing immediate hemodynamic support to a group of critically ill patients where it was possible to carry out diagnostic angiography safely, and thus permit further therapeutic decisions on the basis of those findings.

**Patient Characteristics**

Intraaortic counterpulsation was attempted in a total of 47 patients. This could be successfully performed in 41 patients. In six patients, the balloon could not be inserted via the iliofemoral vessels, primarily because of tortuosity of the vessels and severe atherosclerotic lesions. Of the 41 patients in whom intraaortic counterpulsation was successfully applied, 32 were men and 9 women, with an age range of 43 to 74 years (mean age 57 years). The patients were divided into the three following groups for analysis: Group A, comprised of 16 patients in cardiogenic shock; Group B, 22 patients, 13 of which had acute myocardial infarction and demonstrated recurrent ischemia within the first ten days after admission; nine presented with a history of unstable angina but without evidence of recent infarction; and Group C, three patients, two of whom had resistant ventricular arrhythmias and left ventricular failure with acute myocardial infarction. A third patient presented with noncoronary cardiogenic shock secondary to primary myocardial disease.

**Methods**

All patients in group A had hemodynamic monitoring performed with Swan-Ganz catheterization. Intraarterial monitoring was performed by cannulation of the radial artery, and cardiac outputs were obtained by the thermodilution method. Minimum criteria for cardiogenic shock were systolic pressure of less than 90 mm Hg, urine output of less than 30 ml/hour, confusion, and cold, clammy skin. This was further substantiated by a pulmonary capillary pressure of 20 mm Hg or greater and cardiac index of 2.0 L/min/sq m or less. However, cardiac output in five patients was not available before the insertion of IABP. In group A patients with cardiogenic shock, volume replacement with colloids was instituted when the pulmonary capillary pressure was below 20 mm Hg. Initial trial of vasopressor therapy was instituted in all the patients. This, however, was not carried out for longer than four hours in the patients who did not show...
hemodynamic reversibility of shock state during that period in the interest of providing early hemodynamic support to these patients. Patients with cardiogenic shock secondary to reversible cardiac arrhythmias were excluded from group A.

All patients in group B were initially treated with isosorbide starting with 10 mg (orally) q4 hours with progressive increases in dosage to a maximally-tolerated level. Beta blockade with intravenously administered propranolol was instituted to achieve a heart rate of 60 beats per minute or less, provided there was no evidence of left ventricular failure or hypotension. Most of these patients had Swan-Ganz catheters placed, enabling us to give intravenous propranolol at a dose of 0.1 mg/kg of body weight over the first 30 minutes. The patients who were already receiving propranolol had that drug increased to achieve maximal beta blockade. However, in some patients, full trial of propranolol was not possible in view of recurrent episodes of chest pain with objective evidence of ischemia and hemodynamic deterioration. The dose of propranolol in this group of patients ranged from 100 to 300 mg/day. Significant elevation of pulmonary capillary wedge pressure during an episode of chest pain or ST segment elevation or depression on ECG recorded during pain was considered objective evidence of recurrent ischemia. Ten of the 22 patients demonstrated elevation of the pulmonary capillary wedge pressure during an episode of chest pain, and all others were documented to have electrocardiographic changes during ischemic episodes. Eight patients had ST segment changes and pulmonary capillary pressure elevation during chest pain.

The AVCO intraaortic counterpulsation system with a 30 to 40 ml balloon was used in all patients, depending on the size of the patient and the size of the available vessel. The catheters were all inserted via common femoral artery cut-down under local anesthesia. Most of these procedures were carried out at bedside in the coronary care unit. The balloon was positioned in the descending thoracic aorta, just below the left subclavian artery origin. Balloon position was always confirmed by fluoroscopy and chest roentgenogram. An intravenous catheter recording from a radial artery was used for timing of balloon inflation and deflation. Intravenously administered heparin, 4,000 IU every four hours, was used in the initial patients in this study; later most of the patients received 1,000 to 1,500 IU heparin by continuous intravenous infusion. In addition to IABP, pharmacologic support with nitroprusside, dopamine, or both was instituted and continued whenever necessary. Cardiac catheterization and coronary angiography were performed in a large majority of these patients within 24 hours of insertion of the intraaortic balloon. Further decisions regarding therapy were made, depending upon the results obtained at cardiac catheterization. For the

Table 1—Group A, Shock

<table>
<thead>
<tr>
<th>Age, Sex</th>
<th>Admitting Diagnosis</th>
<th>Coronary Anatomy</th>
<th>Surgery</th>
<th>Outcome</th>
</tr>
</thead>
<tbody>
<tr>
<td>61, M</td>
<td>Ant-Lat MI</td>
<td>. . .</td>
<td>.</td>
<td>Died 2 hr</td>
</tr>
<tr>
<td>61, M</td>
<td>Inferior MI</td>
<td>. . .</td>
<td>.</td>
<td>Died on IABP</td>
</tr>
<tr>
<td>57, M</td>
<td>Inferior MI</td>
<td>Severe three-vessel disease</td>
<td>. . .</td>
<td>Weaned A &amp; W 27 mo</td>
</tr>
<tr>
<td>65, F</td>
<td>Ant-Lat MI</td>
<td>. . .</td>
<td>.</td>
<td>Died 72 hr on IABP</td>
</tr>
<tr>
<td>60, M</td>
<td>Acute inferior MI</td>
<td>Severe two-vessel disease</td>
<td>. . .</td>
<td>Died 1 yr after weaning</td>
</tr>
<tr>
<td>48, M</td>
<td>Anterior &amp; inferior MI (both acute)</td>
<td>Rt dominant high grade prox. LAD mid RCA 90%</td>
<td>Double bypass LAD &amp; RCA</td>
<td>Alive 22 mo</td>
</tr>
<tr>
<td>60, F</td>
<td>Ant-Lat infarct</td>
<td>LAD occluded, RCA occluded midportion</td>
<td>. . .</td>
<td>Weaned, died</td>
</tr>
<tr>
<td>48, M</td>
<td>Anterior infarct</td>
<td>LAD 70% proximal, circumflex 50% mid portion</td>
<td>. . .</td>
<td>Weaned A &amp; W 1 yr</td>
</tr>
<tr>
<td>74, F</td>
<td>Acute anterior-lat. infarct</td>
<td>Total occlusion of LAD</td>
<td>. . .</td>
<td>Died 5 days</td>
</tr>
<tr>
<td>58, M</td>
<td>Postero-inferior MI</td>
<td>Three-vessel disease akinetic ant wall Severe mitral regurgitation</td>
<td>Double bypass to LAD &amp; RCA mitral valve</td>
<td>15 mo A &amp; W</td>
</tr>
<tr>
<td>68, F</td>
<td>Ant-Lat MI (acute)</td>
<td>. . .</td>
<td>.</td>
<td>Died 72 hr after weaning</td>
</tr>
<tr>
<td>59, M</td>
<td>Ant-Lat MI</td>
<td>. . .</td>
<td>.</td>
<td>Died 24 hr after weaning</td>
</tr>
<tr>
<td>48, M</td>
<td>Anterior &amp; inferior MI (both acute)</td>
<td>Severe three-vessel CAD, severe MR</td>
<td>Triple CABG MVR</td>
<td>Died 1 mo, post op</td>
</tr>
<tr>
<td>38, M</td>
<td>Posterior inferior-lateral MI</td>
<td>Total occlusion dominant circumflex artery</td>
<td>. . .</td>
<td>Weaned A &amp; W 12 mo</td>
</tr>
<tr>
<td>62, M</td>
<td>Acute anterior-Lat old MI</td>
<td>Severe three-vessel CAD</td>
<td>. . .</td>
<td>Weaned Alive, died 2 mo</td>
</tr>
<tr>
<td>60, M</td>
<td>Acute ant-lat MI</td>
<td>LAD 80% proximal RCA 50% mid portion</td>
<td>. . .</td>
<td>Died 72 hr after weaning</td>
</tr>
</tbody>
</table>

CAD, coronary artery disease; RCA, right coronary artery; LAD, left anterior descending artery; MR, mitral regurgitation; MVR, mitral valve replacement
Cardiac index on hemodynamic support was $2.6 \pm 0.2$ L/min/sq m. Pulmonary capillary wedge pressure, before intraaortic balloon counterpulsation, was $21.4 \pm 2.8$ mm Hg, and after an hour of intraaortic counterpulsation, dropped to $16 \pm 1.6$ mm Hg. On the basis of the cardiac catheterization and angiographic findings, three patients were considered suitable for cardiac surgery. Two of these patients had severe mitral regurgitation and severe three-vessel coronary artery disease. One of these patients, with an acute posterior lateral myocardial infarction, had a mean pressure of 60 mm Hg, with a systolic pressure of 65 mm Hg and a diastolic pressure of 55 mm Hg. He had a pulmonary capillary mean pressure of 20 mm Hg and a cardiac index of 1.9 L/min/sq m. This patient had no urine output for 24 hours prior to transfer to this hospital, in spite of dopamine and diuretic therapy. Twenty-four hours after IABP, his aortic pressure improved to 65 to 84/42 mm Hg, with 19 mm Hg diastolic augmentation and a mean pressure of 65 mm Hg. Pulmonary capillary pressure dropped to 12 mm Hg, and cardiac index improved to 2.8 L/min/sq m. This patient had severe three-vessel disease and severe mitral insufficiency documented angiographically. Coronary artery bypass grafts to the left anterior descending and right coronary arteries and mitral valve replacement were performed.

At 23 months postoperatively, the patient is in AHA functional class 2 and is gainfully employed as an engineer. A second patient with anterior and inferior myocardial infarction with severe mitral regurgitation had a mitral valve replacement and single vessel graft to the left anterior descending artery. He survived surgery, but died one month later from renal failure. Another patient, a 42-year-old man, considered balloon dependent, underwent double coronary artery bypass graft to the left anterior descending and right coronary artery. He had an ejection fraction of 30 percent. He is alive, 30 months after cardiac surgery. He is in functional class 2 to 3. Of the 13 medically-treated patients, there were eight hospital deaths and five patients were weaned off the balloon with the assistance of intravenously given nitroprusside and dopamine which were gradually supplemented by vasodilators administered orally in large amounts. Three of these patients had inferior myocardial infarction, one anterior myocardial infarction, and one acute inferior myocardial infarction with a remote anterior myocardial infarction. The five hospital survivors have been followed for a mean period of 14 months. One patient is alive at 35 months (class 2 B), another at 24 months (class 2 C), a third patient died in the 12th month (class 2 C), and two patients are alive.
one month after weaning from the intraaortic balloon (class 2 B). The overall hospital survival in group A with cardiogenic shock is 50 percent, that is, eight out of 16 patients, and long-term survival is seven out of 16 patients, calculated to 44 percent. Five patients showed progressive deterioration, two refused angiography, in two, angiography was not carried out because of marked deterioration, four died 2 to 72 hours on IABP, and one died immediately after weaning.

**Group B, Ischemia Group (Fig 2)**

This group included 22 patients with unstable angina pectoris inadequately responsive to maximal medical therapy. Thirteen of the 22 patients had recent acute myocardial infarction with recurrent unstable angina within three weeks after infarction. All of the 22 patients (Table 3) in this group had hemodynamic assessment and coronary angiography. Six of the 22 patients had severe one-vessel coronary artery disease (five LAD, one right coronary artery), ten had severe two-vessel disease, and six had severe three-vessel disease. The ejection fraction when calculated (n = 17) preoperatively in this group of patients was 53 ± 5 percent, and left ventricular end diastolic pressure on IABP was 13 ± 1.7 mm Hg. The cardiac index was 2.5 ± 0.14 L/min/sq m. Pulmonary capillary wedge pressure before counterpulsation was 9 ± 1.6 mm Hg (NS). Sixteen of these patients were considered operable after coronary angiography had been performed, and following transfer to the Massachusetts General Hospital, they had coronary bypass surgery performed. There were no surgical deaths. One patient with Prinzmetal angina suffered a perioperative myocardial infarction. These patients have been followed for a mean period of 15 months, ranging from 15 days to 35 months, and all are alive and well. Six patients were weaned from IABP; all are alive at this time receiving medical therapy. It was possible to increase and optimize their medical regimen, and hence, allowed time for stabilization. Five of these patients had a proximal coronary artery lesion responsible for infarction and midportion or distal lesion in another coronary artery. One patient had severe three-vessel disease when he had to be weaned off because of platelet depletion secondary to associated heparin therapy. All of these patients continue receiving medical therapy.

**Group C**

Two of the three patients in group C were treated for left ventricular failure and ventricular arrhythmias. Both patients were weaned from IABP successfully. One died two months after hospital discharge. He had presented to the hospital after a massive anterolateral myocardial infarction, with a history of previous inferior myocardial infarction. Another patient with acute myocardial infarction and intractable ventricular arrhythmias was weaned successfully and is alive and well. Only moderate reductions of PVCs were seen in both cases; however, no episodes of ventricular tachycardia occurred when on IABP. The third patient, who received IABP for noncoronary cardiogenic shock due to advanced cardiomyopathy, also died six weeks later at home. On angiography he was found to have normal coronary arteries and a severely hypokinetic left ventricle.

**Complications**

One patient continued with major arterial insufficiency in one leg. Three patients suffered dissection of the iliac vessels and descending aorta during unsuccessful attempts at insertion. One patient required femoral artery thrombectomy two weeks after weaning from the balloon. There were no deaths related to any of the above complications and insertion of intraaortic balloon. The complication rate is 10 percent in the total population; however, in the group where the intraaortic balloon was success-
fully inserted, the complication rate is only 5 percent.

**DISCUSSION**

Scheidt et al., in 1973, reported the results of a cooperative clinical trial of intraaortic balloon counterpulsation in patients with cardiogenic shock. Their experience did not show any appreciable decrease in mortality from cardiogenic shock as a result of intraaortic counterpulsation. However, the majority of their patients were in very advanced stages of cardiogenic shock. Their reported hospital survival of 17 percent in a population with a predicted 100 percent mortality appeared encouraging. Willerson et al.\(^8\) described his experience with IABP in 27 patients, 23 of whom were in cardiogenic shock. Although nine of these patients were weaned from circulatory assistance, only three were discharged from the hospital. Only a small group of eight patients underwent cardiac catheterization, and only one of these patients survived. Dunkman et al.\(^9\) reviewed experience with IABP in 24 patients in cardiogenic shock, and of these, only nine could be weaned from pump support. Four patients left the hospital as long-term survivors. Sanders et al.\(^10\) suggested that intraaortic balloon counterpulsation in cardiogenic shock and acute myocardial infarction is an effective method for temporary reversal of the shock state, and commented that, unless combined with definitive surgical correction, salvage rate may be low. Lefemine et al.\(^11\) reported their experience with IABP in 94 patients. Fourteen patients were treated for cardiogenic shock including four who underwent surgery, resulting in a survival rate of 71 percent in cardiogenic shock victims. This is an unusually high number from a small series of patients. Previous experience of other authors without surgical intervention has varied from a low of 11 percent to a high of 41 percent. In this series, there was a 50 percent hospital survival in the cardiogenic shock group (and one late death).

It is our opinion that continued experience with intraaortic balloon counterpulsation has resulted in the development of pertinent specific criteria for selection of patients who can benefit from this form of mechanical assistance. Patients with moderately preserved left ventricular function and good opacification of the distal coronary arteries on angiography and significant mechanical lesions, such as mitral insufficiency and ventricular septal defect,\(^12\) are likely to benefit most from intraaortic counterpulsation, often combined with surgical correction. On the other hand, patients with a large previous myocardial infarction, previous episodes of left ventricular failure, or marked cardiac enlargement, are least likely to benefit in the long run from intraaortic counterpulsation. Balloon dependency in the latter group of patients is high, and weaning is difficult. Another probable reason for our success in the group with cardiogenic shock is the fact that we were able to wean some of these patients from IABP with careful titration of dopamine and nitroprusside over several days, after an initial period of intraaortic counterpulsation. These patients were eventually switched over to oral vasodilator therapy and then weaned from intravenously administered vasodilators. The long-term result in at least two of the three patients in group A who underwent corrective surgery is very rewarding.

In addition to the patients with cardiogenic shock, another important indication for counterpulsation is in patients with unstable angina refractory to maximal medical therapy. It was possible to stabilize all of these patients on intraaortic balloon counterpulsation and then to study them without any mortality or morbidity. Long-term follow-up of these patients, as described, is very encouraging, and one can only speculate whether it was possible to prevent cardiogenic shock in some of these patients, particularly the ones who had recurrent ischemia in areas other than a recent myocardial infarction. Out of this group, the patients undergoing surgery did so without any mortality in the immediate postoperative period. Reports that counterpulsation reduces the extent and severity of ischemic injury and perhaps opens dormant collateral vessels\(^13,14\) are promising, and raise the possibility that early use of intraaortic counterpulsation may be beneficial for patients who sustain large myocardial infarction, and may minimize infarct size and reduce eventual necrosis.

Intraaortic counterpulsation can be carried out in a coronary care unit by a well-coordinated team. In our institution, ICU nurses were trained and accepted responsibility of taking care of these patients. Two critical care nurses and one biomedical engineer provided the back-up support. The IABP insertions were carried out by thoracic surgeons at the bedside. The initial evaluation and the request for insertion of intraaortic balloon originated from a cardiac consultant. It is an effective tool for providing immediate hemodynamic support to these critically-ill patients. Cardiac catheterization and angiography can be carried out without any mortality in this high risk group of patients. Safety of these studies is enhanced by IABP. However, it is clear that only a select group of patients in cardiogenic shock are likely to benefit from this mode of therapy. In an institution providing care to a large number of patients with coronary artery disease, application of intraaortic counterpulsation is also likely to reduce...
mortality and morbidity in patients with unstable angina who are refractory to the usual forms of medical therapy. Transportation of these patients to a cardiac surgical center required the use of a specially-equipped ambulance. This ambulance is equipped to provide at least 15 amperes of power at 110 volts. It should be able to accommodate stretcher, balloon pump, ventilator and provide power output for the above as well as intravenous pumps. Coordination with the surgeon and the surgical center are of extreme importance. The surgeon is kept aware of the progress of a potential patient at all stages, and upon arrival at surgical center, appropriate arrangements for the patient are well established according to the individual needs of the patient. The patients were thus transferred without interruption of ballooning. A critical care nurse and resident accompanied the patient during transportation.

CONCLUSION

This report documents our experience in aggressive management of potentially fatal complications of coronary artery disease at a medical center where in-house cardiac surgery is not available. The use of intraaortic balloon counterpulsation is an effective and safe method for providing hemodynamic support and carrying out diagnostic angiography in high risk patients. The results of angiographic and hemodynamic studies can define a group of patients who may benefit from surgical revascularization procedures. When indicated, such patients can be transferred safely with continuing IABP support to a cardiac surgical center. This study establishes the feasibility of a coordinated, aggressive approach to the management of such patients and attempts to define criteria which may result in more optimal utilization of facilities where cardiac surgery is available.

ACKNOWLEDGMENT: The authors are grateful to Clarence C. Dumais, M.D., Richard L. Bishop, M.D., Oscar Starobin, M.D., Daniel Miller, M.D., and Edward Budsitz, M.D., for allowing us to include their patients in this report. We are also thankful to Louis Anastasia, M.D., and Gerald Carroll, M.D., who performed intraaortic balloon pump insertion in all the patients reported; and other members of the critical care team for their untiring efforts.

REFERENCES