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Extended Use of Intra-aortic Balloon Pumping in Peripartum Cardiomyopathy

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A patient with perinatal cardiomyopathy was greatly benefited physiologically by 7 weeks of intra-aortic balloon pumping. This experience documented that extended pumping can be carried out with only manageable complications. Although the patient survived the hospitalization, she died shortly thereafter of intractable congestive failure. Perinatal cardiomyopathy is a potentially reversible condition. Ventricular assistance by intra-aortic balloon pumping may be sustaining during continued systemic treatment of this entity. Further evaluation for longevity of more advanced congestive heart failure from cardiomyopathies needs further clinical trial.

WIDE EXPERIENCE reported by many authors has documented the usefulness of intra-aortic balloon pumping as an adjunct to the treatment of various forms of cardiogenic shock.[2,8,11,13] In spite of the now frequent use of this technique for short term assist of patients with myocardial infarction, the limitations of balloon pumping are still being defined. The length of time that balloon pumping can be safely used, the maximum pump run producing long term patient benefit, and the usefulness in more chronic forms of cardiogenic shock remain to be elucidated. The suggestion that recovery from perinatal cardiomyopathy may be expected if the patient survives the initial episode of heart failure prompted this attempt to assist a terminal patient with this disease. Experience in treating this patient provides important information about the limits of intra-aortic balloon pumping.

Case Report

M.M., a 43-year-old Caucasian primagravida, was transferred to St. Luke's Hospital on October 8, 1973. Although her life had been characterized by good health, her pregnancy had been complicated by congestive heart failure and pulmonary edema, and macrocytic anemia, treated with digoxin, diuretics and procaine amide.

Physical examination and chest x-ray confirmed the presence of congestive failure and pulmonary edema. BUN was elevated and creatinine normal. She was treated with digoxin, furosemide, salt restriction, and bed rest. With the exception of her rising BUN and creatinine, she did well.

Thirty days after admission, a 5 pound, 13½ ounce healthy male infant was delivered by cesarean section. The patient's postoperative course was characterized by worsening heart failure.

She became more dyspneic with increasing BUN (Fig. 1). Five days after delivery she became cyanotic, disoriented, and developed Cheyne-Stokes respirations with severe proteinuria. Pulmonary edema became worse and apneic spells became longer. Her operative incision separated and was reclosed. On the eleventh day post "C" section the patient was only semiconscious. She became jaundiced and developed the murmur of mitral insufficiency. Fibrinolysis developed with a platelet count of sixty thousand and a fibrinogen of 127 mg%. Prothrombin time was control 12, patient 15, and PTT was normal. Transfusion and epsilon amino caproic acid were used to control her coagulopathy. High dose steroids were added. Although her blood pressure remained 100 mm Hg, the patient was rapidly deteriorating, with cerebral, liver, renal, cardiac and pulmonary failure in spite of maximal medical management. Intra-aortic balloon pumping was started the 14th day with a 40 cc Avco intra-aortic balloon catheter inserted through the right femoral artery using local anesthesia. Systemic heparinization was not used.

Postoperatively, mixed venous oxygen saturation rose immediately from 40 to 58%. Epsilon amino caproic acid was continued. With a digoxin level of 2.8 micrograms she developed multiple arrhythmias, including both supraventricular arrhythmias and ventricular fibrillation. Multiple drugs and cardioversion were required. Her mental status returned to normal over the next two days. Steroids were tapered and her coagulopathy resolved. Hyperalimentation was started. The BUN fell rapidly after a single dialysis and remained low. After 22 days the renal and liver failure had resolved, her pulmonary edema had cleared somewhat, and her mental status had become normal. Attempts to wean

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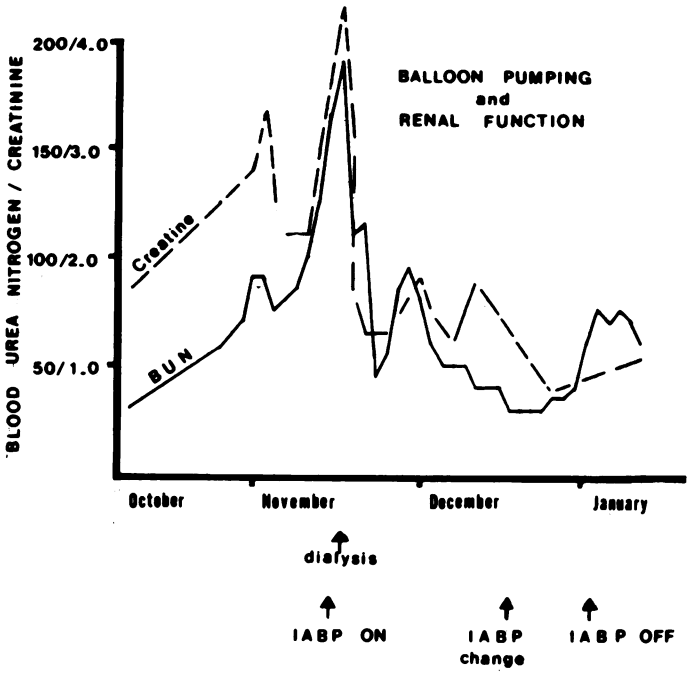


FIG. 1. The effect of intra-aortic balloon pumping on BUN and creatinine. Intra-aortic balloon pumping sustained a pronounced decrease in BUN and creatinine.

her off the pump were made over several days, but when the assist interval was reduced to 1:4 she became confused and disoriented and 1:1 pumping was resumed. Mental clarity returned. The balloon catheter tube developed a leak and was repaired using silastic bathtub caulking and plastic tubing. Our practice of having the patient sit in a chair undoubtedly contributed to the tube failure.

Ischemia of both lower extremities developed and heparin was started. Arteriogram the following day showed pronounced clot formation around the catheter and in both legs (Fig. 2). The catheter was removed and a new one placed in the left femoral artery. Clots were removed from the aorta and both femoral arteries using Fogarty catheters. Postoperatively, pedal pulses were restored and profuse diuresis ensued. Systemic heparinization was instituted until the following week when significant bleeding developed from the suture line requiring reoperation and one additional suture. Aspirin and dipyridamole were used from that time. Cardiac catheterization showed severe left ventricular dysfunction. No coronary artery or valve pathology was found. LVEDP was 34 and cardiac output on the pump was 2.2 L/M. With the discontinuance of the intra-aortic balloon pump LVEDP was 44 and C.O. 1.8 L/M.

Since clots were seen in the arm vessels, she was weaned from the balloon pump over the next several days. She became disoriented, with absent peripheral pulses, little blood pressure and severe metabolic acidosis. Severe clotting in her peripheral vessels precluded further balloon pumping, so the catheter was removed, and clots were removed from her aorta and left femoral artery using Fogarty catheters. Postoperatively, shock and metabolic acidosis resolved, and confusion cleared. Cardiac compensation remained marginal on maximal therapy until her transfer to a local hospital where she died 3 weeks later of intractable congestive failure. No autopsy was obtained.

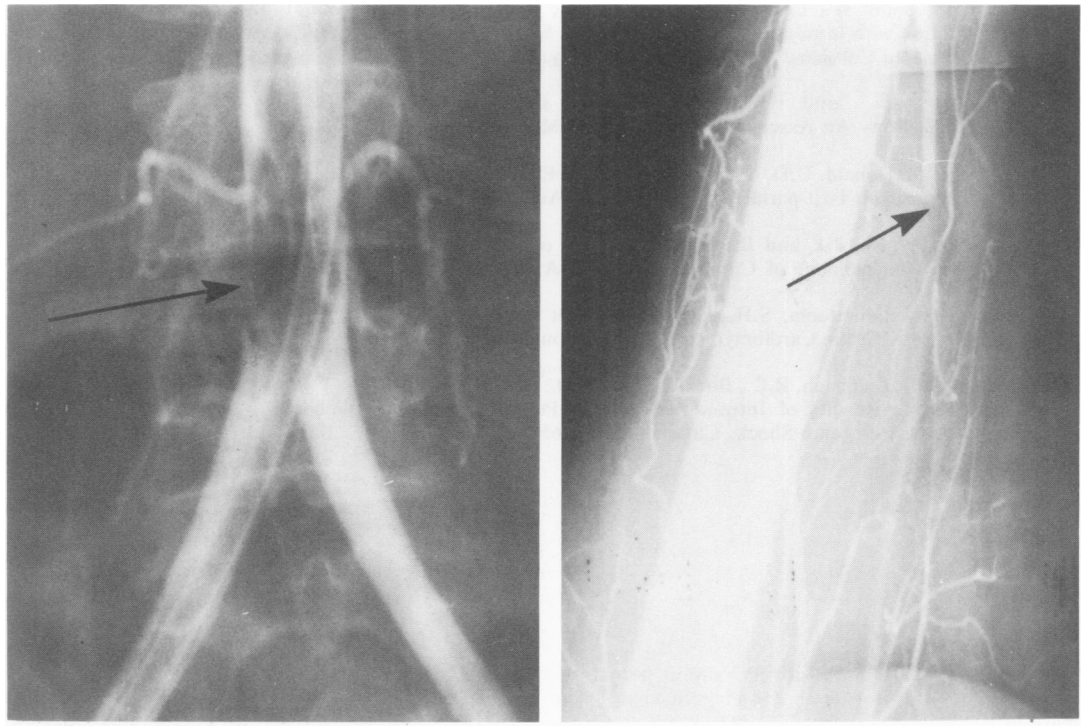
Discussion

Perinatal cardiomyopathy is an unexplained development of cardiomegaly and congestive failure during pregnancy or within 3 to 4 months following its termination. No previous cardiac disease is usually present.[1,4,6,9,10,12] The condition is often fatal, carrying a mortality of 30 to 60 per cent. Affected patients die of intractable congestive heart failure, arrhythmia, or embolization. Symptomatic medical therapy seems to have little effect on the course of the disease. No specific therapy, other than cardiac transplantation, is currently available.

The etiology of this disease is unknown. Nutritional deficiency, infections and auto-immune process have been implicated.[4] Treatment has been largely symptomatic with dietary deficiencies corrected and prolonged bed rest.[5]

Many authors have emphasized the variable nature of this disease. Although the mortality is high, some patients recover entirely.[1,10] DeMakis et al.[6] divided their patients into two groups. The first, though initially quite ill, recovered a normal-sized heart within 6 months. Their prognosis was good, though 5 of 8 who had subsequent pregnancies had mild recurrences of the disease. The second group, sometimes showing transient improvement, retained cardiomegaly. Eighty-five per cent of these patients eventually died of intractable congestive failure. In more severly and acutely ill patients, the intra-aortic balloon pumping may be used to further reduce the afterload on the diseased heart in an attempt to improve the advanced cardiac failure.

Intra-aortic balloon pumping often produces the same dramatic relief of cardiogenic shock in patients with myocardial infarction as it did in the patient reported. The physiologic mechanisms for this improvement have been carefully documented.[3,7] Not only does the balloon pump increase cardiac output, but it also increases coronary perfusion. By decreasing the afterload on the left ventricle, ventricular work is decreased as much as 25%. Most of these physiologic benefits apply as well to the heart failing because of cardiomyopathy. Increased cardiac output was undoubtedly responsible for the prompt resolution of hepatic, renal, pulmonary and cerebral failure in this patient. With other systems failing because of poor peripheral perfusion, increasing acidosis and hypoxia also aggravated cardiac decompensation.[11] Increasing stroke volume and relief of ventricular work could "buy time" for possible recovery of myocardial function. Since many of the physiologic benefits to the myocardial infarction, septic shock[2] and cardiomyopathy patients are the same, the dramatic initial response can be expected. Each of these problems requires a relatively brief period of support before either type of patient recovers or expires. Lack of an endpoint and the potential need for long term support are undoubtedly the reasons balloon pumping has not previously been applied to the other major cause of cardiogenic shock—cardiomyopathy.



FIGS. 2a and b. Aortogram showing clot formation around intra-aortic balloon catheter and in the right leg (arrow).

Little is known about the time limits of intra-aortic balloon pumping. How long can the pump be left in place without life-threatening complications? Is there a time beyond which pumping cannot be expected to produce long term benefit to the patient? Many complications have been associated with balloon pumping. We have seen infections, thrombosis, ischemic legs, bleeding, and false aneurysm formation. During this 7 week "pump run," the patient's main problems were thrombotic, necessitating removal of the first balloon catheter at 32 days, and the second at 49 days. Though it has not been our routine to use anticoagulation therapy on these patients, anticoagulation was begun when the second catheter was inserted. Bleeding required discontinuation of heparin, but aspirin and dipyridamole were continued until thrombus formation required removal of the second catheter. There was no evidence of balloon failure and helium leak during this long pump run. The consequences of thrombus formation were easily controlled using Fogarty catheters. Our experience with this patient shows that safe pumping can be carried out for as long as 7 weeks.

During its evolutionary phase, pumping was carried out for only 24 hours before the patient was considered pump dependent. We have been more persistent with this form of assist than many because some of our patients on the balloon pump up to 3 weeks have benefited. With this philosophy, dependence has not been a problem. Although we have enough survivors to justify pumping until a complication or catastrophe ensues, the question of a time limit beyond which there is no long term benefit remains unanswered. This patient received no long term benefit although she was discharged with improvements in the hemodynamics and concurrent "normalization" of multi-system failures.

Conclusion

Long term intra-aortic balloon pumping was carried out in a patient with perinatal cardiomyopathy and cardiogenic shock to provide sustained benefit by preventing advanced cardiac decompensation with secondary hepatic and renal failure. The patient's condition was improved during the period of assist and the feasibility of balloon pumping for as long as 7 weeks was demonstrated. Whether this type of treatment is recommended for all cardiomyopathies needs further therapeutic evaluation.

Addendum

Since submission of this report, a 54-year-old Caucasian man, who arrived at Denver General Hospital in ventricular fibrillation due to cardiomyopathy, was resuscitated into cardiogenic shock and assisted with the balloon pump for 6 days. Following discharge from the hospital he did well for a month when he had another episode of ventricular fibrillation from which he could not be resuscitated.

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