CASE REPORTS

Delayed Recovery of Severely “Stunned” Myocardium With the Support of a Left Ventricular Assist Device After Coronary Artery Bypass Graft Surgery

CHRISTIE M. BALLANTYNE, MD, MARIO S. VERANI, MD, FACC, H. DAVID SHORT, MD, CHERYL HYATT, BSN, RN, GEORGE P. NOON, MD, FACC

Houston, Texas

A 53 year old man underwent repeat coronary artery bypass graft surgery after presenting with unstable angina. Because of intraoperative ischemia, the patient developed profound left ventricular dysfunction requiring placement of a left ventricular assist device and intraaortic balloon pump and catecholamine infusion. Serial radionuclide ventriculograms documented delayed recovery of the severely stunned myocardium with mechanical and pharmacologic support.

(J Am Coll Cardiol 1987;10:710-712)

Extensive experimental evidence in animals (1) indicates that myocardial ischemia may cause prolonged dysfunction or “stunning” that is reversible. This sequence of events, however, remains to be well documented in humans. During coronary artery bypass graft surgery, the myocardium may undergo prolonged periods of ischemia. Early evaluation of left ventricular function in this setting may overestimate the extent of irreversible myocardial damage due to “stunning” of the myocardium, which later regains function. Hemodynamic support with intraaortic balloon counterpulsation and a left ventricular assist device may allow dramatic improvement in cases that initially appear to be hopeless. The present case demonstrates objective improvement of profound and prolonged myocardial dysfunction due to perioperative ischemia, utilizing temporary mechanical and pharmacologic support.

Case Report

Clinical features. A 53 year old white man was admitted to this hospital with unstable angina pectoris. The patient had a history of prior coronary artery bypass grafting to the left anterior descending, diagonal, left circumflex and right coronary arteries 12 years earlier, with good relief of symptoms. However, 3 years before admission, he had a myocardial infarction and recently had progressive worsening of angina pectoris, including pain at rest, that required admission to another hospital in April 1986. Administration of intravenous nitroglycerin and nifedipine lessened his symptoms. Cardiac catheterization was performed and the patient was subsequently transferred to our institution for coronary artery revascularization.

Coronary angiograms demonstrated total occlusion of the proximal left anterior descending, left circumflex and right coronary arteries. The grafts to the diagonal, left circumflex and right coronary arteries were occluded. A single graft was patent to the left anterior descending artery, which provided collateral vessels to the diagonal branches and to the posterior descending coronary artery. There was 70% stenosis of the left anterior descending artery distal to the graft.

On admission, the blood pressure was 130/90 mm Hg in both arms, and the pulse was 72 beats/min and regular. The jugular venous pressure and carotid pulses were normal. The lungs were clear to auscultation. Cardiac auscultation revealed regular rhythm, normal first and second heart sounds and an S4 gallop. The electrocardiogram (ECG) showed normal sinus rhythm with ischemic repolarization changes in the anterolateral wall. The chest X-ray film was normal.

A gated radionuclide ventriculogram at rest was done on the morning of surgery using standard techniques in our laboratory, as previously reported (2). The left ventricular ejection fraction was 50%, with mild apical, septal and anterolateral hypokinesia (Fig. 1A). The left and right ventricular chambers were normal in size, and right ventricular wall motion was normal.
Operative course. On April 21, 1986, the patient had reversed autogenous saphenous vein grafts placed from the aorta to the left anterior descending, first diagonal, obtuse marginal, distal left circumflex and distal right coronary arteries under total cardiopulmonary bypass. Intraoperatively, efforts to protect the myocardium included continuous infusion of Plegisol through the aortic root and in each vein graft, as well as intermittent topical hypothermia. The total aortic cross-clamping time was 1 hour 42 minutes. Multiple attempts to wean the patient from cardiopulmonary bypass resulted in severe systemic arterial hypotension despite the insertion of an intraaortic balloon counterpulsation pump. A Biomedicus left ventricular assist device was inserted, and it pumped blood from the left atrium into the ascending aorta at a flow rate of 2.0 liters/min. The patient was then successfully weaned from the cardiopulmonary bypass pump, after a total pumping time of 4 hours 56 minutes.

Early postoperative course. After transfer to the intensive care unit, the patient remained hemodynamically stable on the intraaortic balloon pump and with the left ventricular assist device set at 3.2 liters/min, in addition to constant infusions of dopamine (5 μg/kg per min), amrinone (5 μg/kg per min), epinephrine (2.5 μg/min) and nitroglycerin (30 μg/min). Cardiac index was 2.1 liters/min per m², with a heart rate of 126 beats/min. Cardiac auscultation revealed distant heart sounds. Chest X-ray study revealed cardiomegaly and pulmonary edema. A repeat gated radionuclide angiogram 12 hours after the operation showed a dilated left ventricle with severe diffuse hypokinesia and a calculated left ventricular ejection fraction of 6% (Fig. 1B). The right ventricle was also dilated and hypokinetic. The initial postoperative ECG showed a diffuse decrease in QRS voltage. The creatine kinase MB level reached a peak of 177 IU, confirming the diagnosis of intraoperative myocardial infarction.

The patient was initially obtunded, but gradually became more alert, despite evidence of right-sided hemiparesis. A repeat radionuclide ventriculogram done 60 hours postoperatively showed no improvement in left ventricular function. However, right ventricular wall motion had returned to normal. Amrinone was stopped on the third postoperative day because of thrombocytopenia, and treatment with dobutamine, 5 μg/kg per min, was begun. The patient remained hemodynamically stable, with clinical improvement over the next few days. Repeat radionuclide ventriculography on the seventh postoperative day showed a left ventricular ejection fraction of 28%. At this time, the left ventricular assist device was removed, dobutamine was increased to 10 μg/kg per min and nitroglycerin to 126 μg/min. Cardiac index was 3.1 liters/min per m², with a heart rate of 95 beats/min. During this regimen, a repeat nuclear study on the eighth postoperative day showed a left ventricular ejection fraction of 38% (Fig. 1C). The intraaortic balloon pump was removed on the ninth postoperative day, and 3 days later the patient was weaned from dobutamine and intravenous nitroglycerin, which were replaced by topical nitrites and oral captopril. Gradual lessening of the pulmonary edema and cardiomegaly was documented by chest X-ray study. A final radionuclide angiogram, obtained on the 17th postoperative day, showed a left ventricular ejection fraction of 34%.

Late postoperative course. The patient’s recovery was slowed by right-sided hemiparesis and partial left visual field loss. Computed tomography of the brain showed right occipital and right parietal infarcts and a small left occipital infarct. However, the patient made marked improvement with physical therapy and was discharged on the 32nd postoperative day without symptoms of congestive heart failure. At that time, his mental status and speech were normal and he had only mild residual right hemiparesis. Chest X-ray study before hospital discharge showed normal cardiac dimensions and complete clearing of pulmonary edema. A predischarge ECG revealed improved QRS voltage compared with the preoperative amplitude. There were minor nonspecific repolarization abnormalities without evidence of a transmural infarction.

Discussion

This case is a dramatic demonstration that severe myocardial dysfunction after prolonged periods of ischemia may have a reversible component as a result of “stunning” in addition to an irreversible component due to myocardial necrosis. It also shows that the recovery of function may require several days, even with mechanical and pharma-
cologic support. Although several surgical series (3–5) have shown that a left ventricular assist device can benefit patients sustaining perioperative infarction and shock, documentation of serial changes in left ventricular function in human patients is lacking.

Radionuclide angiography performed 4 hours postoperatively has uncovered transient mild myocardial dysfunction in almost half of the patients undergoing coronary artery bypass graft surgery (6). The mean decline in left ventricular ejection fraction is 12%, and the values return to baseline by 7 days. The recovery of stunned myocardium in animal models is hastened by catecholamine infusion (7) and, therefore, this pharmacologic therapy may have similar benefit in humans. The use of positive inotropic drugs, however, must be tempered by the potential enhancement of postarrest myocardial damage when these drugs are used prematurely (8).

**Conclusion.** This case indicates that some patients who appear to have massive and terminal left ventricular dysfunction may instead have large amounts of reversibly stunned myocardium. Mechanical support with a left ventricular assist device and intraaortic balloon pump, concomitantly with pharmacologic support by infusion of catecholamines, can sustain life while the severe myocardial stunning gradually dissipates and the left ventricle resumes its contractile function.

We gratefully acknowledge the secretarial assistance of Maria Frias.

**References**


