Early Intraaortic Balloon Pumping for Anterior Myocardial Infarction Without Shock

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SUMMARY Eleven patients with anterior myocardial infarction less than six hours old underwent intraaortic balloon pumping (IABP) in an attempt to control injury. Direct hemodynamic measurements excluded shock. Response to therapy was judged individually by comparison to a one-hour pretreatment period. There was no parallel control group.

Five patients responded with an 84% fall in ST elevation in one hour, with preservation of precordial R waves and good ventricular function. In contrast, six patients responded poorly, with a 40% fall in ST elevation in one hour, Q wave development and poorer residual left ventricular function.

Coronary angiography demonstrated a significant correlation between response and presence or absence of complete left anterior descending coronary artery occlusion. Early use of IABP in anterior myocardial infarction may interrupt injury, though in this series only in cases with residual left anterior descending patency.

STUDIES IN MAN have shown that certain interventions can alter acute injury during myocardial infarction. Responses to pharmacological interventions have been statistically significant, but the magnitude of change has often been small. When treatment has begun from 2–21 hours after onset of infarction, ST segment elevation has been acutely reduced by 20–40%, and other measures, such as Q wave generation and creatine phosphokinase release, have been modified. No intervention has consistently produced complete resolution of the process of injury.

We attempted to produce a maximal acute response in patients by the early use of the intraaortic balloon pump. We used continuous monitoring of the electrocardiogram to identify abrupt cessation of injury. We have further attempted to explain differences in response with coronary angiography performed soon after the onset of injury. The results suggest both exciting possibilities and exasperating limitations.

Materials and Methods

Study Outline

A homogeneous population of young patients was identified less than 6 hours after the onset of anterior myocardial infarction with persistent marked ST segment elevation. Balloon pumping at full volume was begun after demonstration that the electrocardiographic injury current was not resolving with traditional therapy. Continuous electrocardiographic monitoring was used to characterize rate and magnitude of response. Coronary angiography was then performed, permitting correlation between coronary pathology and electrocardiographic response.

Details of Study Design

Patients were selected who were under the age of 65 with pain and anterior ST segment elevation resistant to morphine, oxygen, and in many cases, nitroglycerin. The onset of infarction was considered to be the time of development of intense and steady precordial pain. In all cases an electrocardiogram confirming anterior ST segment elevation was available within 1 hour after the onset of symptoms.

A member of the study team evaluated the 12 lead electrocardiogram for minimally acceptable ST segment elevation, at least 4 mm (0.4 mV) in two standard precordial leads.

Informed consent was obtained by a principal investigator and by the senior responsible physician by discussion with the patient and the most immediate relative. The risks and possible benefits of intraaortic balloon pumping were discussed, and the option of standard therapy was offered. Ten years of experience with balloon pumping formed the basis of this proposal.

Patients were moved rapidly to a hemodynamic study area, with the last three cases taken directly to the cardiac catheterization laboratory. The study team consisted of two cardiologists, one surgeon, two nurses, a cardiac technician, and most recently, a radiology technician.

Instrumentation was performed with fluoroscopic control and included a pulmonary artery flow-directed thermal dilution catheter, a central venous line and a radial artery cannula. Precordial electrocardiographic tracings always consisted of the six standard leads (at marked sites) and continuous recording of the standard precordial lead with maximal ST segment elevation. Eighteen lead maps were also obtained in seven cases, consisting of the standard precordial leads and corresponding sites one interspace above and below.

During hemodynamic evaluation morphine and oxygen were continued, and in several instances initial doses of propranolol and increments of nitroglycerin were used. Precordial lead tracings were then repeated.
after 1 hour of evaluation and pharmacologic therapy, and if ST elevation in the six standard leads persisted, balloon pumping was begun. A 30 or 40 cc balloon pump was used. Immediate responses were noted and precordial six lead tracings were repeated at 30 minutes and one hour, producing four comparable sets: one hour before balloon pumping, immediately before balloon pumping, 30 minutes after and one hour after.

Coronary angiography was performed in all patients within six days after the onset of infarction, and in four cases within 7 hours. Two patients underwent emergency revascularization 24 hours after the onset of balloon pumping and are described below.

Post-treatment ejection fractions were obtained in 10 cases, one by blood pool scan and the others by single plane cineangiography performed two days to 10 months after infarction. Serial standard creatine phosphokinase levels were obtained, along with standard clinical evaluations and follow-up.

Results

The patients are listed with time to balloon pumping and immediate preballoon therapy in table 1. The patient listing follows the same order in all subsequent tables and illustrations. All were male. The average age was 49 years, and the average delay from onset of infarction to initiation of balloon pumping was 3 hours. This was the first infarction for 10 of the 11 patients. Patient 2 had had a previous inferior myocardial infarction. All patients had persistent chest pain despite morphine and oxygen, but none demonstrated shock or pulmonary edema. There were no precordial rubs, and there was no evidence of peripheral vascular disease that might impede balloon insertion. Electrolyte abnormalities and electrocardiographic conduction defects were not present. During the hour of hemodynamic and electrocardiographic evaluation before initiation of balloon pumping, three patients received propranolol intravenously to control tachycardia. During the last 30 minutes before balloon pumping, four received nitroglycerin sublingually or by abrupt intravenous infusion. No patient responded to either agent with a fall in ST elevation of more than 10%. The infusion of propranolol or dosage of nitroglycerin was always complete by 15 minutes before onset of balloon pumping. No further drugs were given before counterpulsation except intravenous heparin.

Hemodynamic measurements immediately before and 30 minutes after onset of balloon pumping are shown in table 2. No patient was in shock and no patient was hyperdynamic. The highest blood pressure before intraaortic balloon pumping was 155/95. Norepinephrine was infused in patient 2 because of a hypertensive reaction to morphine. After 30 minutes of balloon pumping, peak diastolic pressure recorded at

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*Depression = wedge > 15 mm Hg, cardiac index < 2.0 l/min/m² or hypotension requiring pressor.

Abbreviations: MI = myocardial infarction; IABP = intraaortic balloon pumping.

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*On norepinephrine 4 µg/min.
the radial artery increased from 78 ± 11 (sd) to 134 ± 20 mm Hg. Wedge pressure fell 3 mm Hg and heart rate was essentially unchanged.

ST elevation in the lead reflecting maximal injury was persistent for the entire group during the control hour. A gradual fall was seen from an average 0.88 to 0.79 mV (10%). In contrast, major changes were seen within minutes of full volume balloon pumping. The cause-and-effect relationship between balloon counterpulsation and ST change is exemplified by patient 2 in figures 1 and 2. In this case balloon pumping initially produced an immediate improvement in ST elevation but had to be interrupted for technical reasons. During the 5-minute interruption, full ST elevation recurred with increased chest pain (fig. 2).

Resumption of pumping abolished pain and ST elevation within 2 minutes.

The response pattern of the six standard precordial leads in patient 3 is shown in figure 3. There was pain relief and an 86% fall in ST elevation within 15 minutes. This case also exemplifies a general finding of this study, that striking ST changes were as well observed in the precordial lead with maximal ST elevation as with the sum of six precordial leads, or even with the 18 lead map. Both rate and the degree of ST fall could be followed accurately by single leads in the zone of maximal electrocardiographic injury.

Electrocardiographic responses to balloon pumping for all 11 patients could be divided into two patterns. The first pattern (patients 1–5) was considered a good
Figure 3. The six standard precordial leads in patient 3 (RB) before and 15 minutes after intraaortic balloon pumping (IABP). The sum of ST elevation in these six leads ($\Sigma ST$) fell 3.0 mV, or 86%. Note that improvement is obvious in any lead showing prominent pretreatment ST elevation.

response and consisted of obvious reduction in ST elevation within five minutes of balloon pumping, with a rapid resolution of ST elevation and pain during the first 30 minutes. The continuously monitored precordial leads for each of these good responders is shown in figure 4. For this group 30 minutes of balloon pumping reduced maximal lead ST elevation from 0.55 to 0.14 mV (75%, columns B and C), with a cumulative fall of 84% in 1 hour (column D).

The remaining patients were considered poor responders, with no immediate ST change after onset of balloon pumping. The fall in ST elevation was gradual (fig. 5). For this group, 30 minutes of balloon pumping was associated with a fall in ST elevation from 0.98 to 0.82 mV (16%, columns B and C), with a cumulative fall of 40% in 1 hour (column D). Balloon pumping was continued in all patients for an average of five days, and there was no evidence of recurrence of injury.

The pattern of Q wave evolution in the good responders is also shown in figure 4. The Q waves present before onset of pumping persisted. New Qs which developed were small (patient 4), and there was no further Q wave generation after 1 hour. All poor responders started with or developed prominent precordial Q waves. In patients 6 and 7 the Q waves evolved after 1 hour, during resolution of ST elevation, and in both cases extended out to lead V4.

The angiographic correlations are listed in table 3. Angiographically identifiable coronary lesions were seen in the left anterior descending coronary artery in all patients. These were single and focal. All good
responders showed stenosis (patients 1–5), but residual left anterior descending patency. Conversely, all but one of the poor responders (patients 6–11) showed left anterior descending occlusion. This is significant at the $P < 0.02$ level by the Fisher Exact test. This correlation holds despite delays from onset of infarction to angiography from 4 hours to six days. Collaterals to the occluded left anterior descending coronary arteries were generally poor. There was no relationship between response and the presence or absence of additional coronary disease. Also in the poor responders, the residual ejection fraction was worse and the peak creatine phosphokinase was higher ($P < 0.01$).

Patients 1 and 2 underwent revascularization 24 hours after the onset of balloon pumping. During the preoperative waiting period there was no recurrence of myocardial injury. Their ejection fractions, shown in table 3, are postrevascularization at 10 months and four months, respectively. In these two patients the peak creatine phosphokinase shown in table 3 represents the highest determination immediately before revascularization surgery.

There were no complications of balloon pumping. All patients survived hospitalization. None developed severe arrhythmias, shock or pulmonary edema. All have been followed, with an average duration of follow up 26 months. Patients 8 and 10 died at nine months and one year, respectively, of coronary events. Patients 6 and 7 have angina and patient 11 is symptomatic from poor left ventricular function. All good responders are angina-free.

**Discussion**

The 11 patients in this study were selected because of persistent severe ST elevation and chest pain early after the onset of anterior myocardial infarction. The early phase was chosen because it presents the opportunity for intervention before cell death. To further achieve a rapid clinical response, we chose intraaortic balloon counterpulsation, the most reliable technique for simultaneously increasing diastolic arterial pressure while reducing left ventricular oxygen demand. The patients were then observed continuously for rate and degree of response. Changes were judged individually by comparison to a 1-hour pretreatment period.

Two patterns were seen: 1) a marked reversal of the process within minutes, with reduction in pain and in precordial ST segment elevation of at least 50% in 30 minutes, and 2) a gradual clinical and electrocardiographic improvement not clearly differing from the natural history of infarction.

No explanation for the variable response was found in pretreatment hemodynamic status (table 2), preballoon drug usage or pretreatment electrocardiographic patterns. The difference in response, however, could be attributed to variable delay from the onset of infarction to initiation of intraaortic balloon pumping. Pumping was begun in four of the good responders within 1½ hours (table 1). One patient showed a good ST segment response after a 6-hour delay. Two poor responders, however, were pumped as early as 2 and 2½ hours, respectively. The difference in delay between these two groups is not statistically significant. Nevertheless, delay must be related in a general way to outcome, since treatment after cell death can not be restorative.

Patient 5 illustrates this point. The fall in ST segment elevation was 50% in 30 minutes, with nearly complete resolution in 1 hour and a residual ejection fraction of 0.6. The 6-hour delay in treatment for
this patient may account for the precordial Q wave development (fig. 4) and the severity of anterolateral segmental dysfunction (table 3).

The best explanation for the different response is the variation in patency of the left anterior descending coronary artery. There was residual left anterior descending patency in all good responders. However, five of six poor responders showed complete left anterior descending occlusion (fig. 6). This statistically significant correlation was maintained despite delays from the onset of infarction to angiography during which patent vessels may close and clotted vessels may recanalize. Recanalization or lysis of clot may explain the patent vessel in one poor responder (patient 9). However, in this case there was also a 6-hour delay in balloon treatment. Additionally, the fact that all good responders showed open vessels despite delays to angiography of up to four days suggests that the balloon pump may also play a role in the maintenance of coronary patency.

The response of patients 1 and 2 was rapid and complete and enzyme elevations were low. These patients

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**Figure 6.** An early 105 mm frame from the left coronary injection (right anterior oblique) in patient 11. Filming was performed 7 hours after onset of the myocardial infarction. A Swan-Ganz catheter is seen to the right and the balloon pump to the left. There is an abrupt occlusion of the proximal left anterior descending with an intraluminal filling defect, presumably thrombus. The distal left anterior descending was not filled by collateral.
were then treated as examples of prolonged ischemia with patchy necrosis, and were revascularized. The bypass procedures were not, however, performed until 24 hours after the onset of balloon pumping, demonstrating that counterpulsation can prevent recurrence of injury when there is residual coronary patency. Both patients showed improvement in ejection fraction following bypass (0.5 → 0.8, 0.5 → 0.7, respectively), with restoration of normal anterior wall motion and small or absent anteroseptal Q waves.

Anterior myocardial infarction can occur without fixed and complete left anterior descending occlusion.8 It is uncertain whether the pathophysiology involves lysis of thrombus, embolus, or spasm. In this study angiographic documentation of patency was always obtained after response to therapy, and for this reason no conclusions can be drawn concerning the left anterior descending patency at the time of maximal ST segment elevation. This especially applies to patient 3, who showed only 35% left anterior descending stenosis when studied after a delay of four days.

We have found that patients who do not have irreversible occlusion of the left anterior descending coronary artery can show a marked reduction in electrocardiographic current of injury during early treatment with the balloon pump. This rapid electrocardiographic response is associated with both preservation of precordial R waves and ventricular function. When anterior myocardial infarction is produced by left anterior descending occlusion, however, balloon treatment is less effective. Precordial Q waves evolve despite a possibly accelerated rate of ST normalization, and residual ventricular function is worse. Perfusion by collateral pathways appears to be inadequate.

Patients with anterior myocardial infarction and marked ST segment elevation are candidates for early and aggressive therapy. The process of injury can be reversed and in this study this was accomplished with IABP in some of the patients.

References


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