Percutaneous Transluminal Coronary Angioplasty Improves Survival in Acute Myocardial Infarction Complicated by Cardiogenic Shock

Linda Lee, MD, Eric R. Bates, MD, Bertram Pitt, MD, Joseph A. Walton, MD, Nathan Laufer, MD, and William W. O'Neill, MD

Modest survival benefits have been reported in patients with acute myocardial infarction complicated by cardiogenic shock who were treated with early surgical revascularization or thrombolytic therapy. To determine whether coronary angioplasty improves survival, 87 patients with cardiogenic shock complicating acute myocardial infarction at the University of Michigan, Ann Arbor, Michigan, from 1975 to 1985 were retrospectively analyzed. Patients in group 1 (n=59) were treated with conventional therapy; patients in group 2 (n=24) were treated with conventional therapy and angioplasty. Extent of coronary artery disease, infarct location, and incidence of multivessel disease were similar between groups. Hemodynamic variables including cardiac index, mean arterial pressure, and pulmonary capillary wedge pressure were also similar. The 30-day survival was significantly improved for group 2 patients (50% vs. 17%, p=0.006). Survival in group 2 patients with successful angioplasty was 77% (10 of 13 patients) versus 18% (two of 11 patients) in patients with unsuccessful angioplasty, (p=0.006). The findings suggest that angioplasty improves survival in cardiogenic shock compared with conventional therapy with survival contingent upon successful reperfusion of the infarct-related artery. (Circulation 1988;78:1345–1351)

Cardiogenic shock remains a lethal complication of acute myocardial infarction.1–7 Pathological studies demonstrate extensive myocardial necrosis approximating 40% of the left ventricular mass associated with thrombotic occlusion of the infarct-related coronary artery.8–11 Standard therapeutic strategies not designed to establish reperfusion of the infarcted artery have yielded disappointing results with hospital mortality rates of 80–90%.12–25 DeWood and associates26 suggested that early surgical revascularization of the infarcted artery may salvage jeopardized myocardium and improve survival. Kennedy and coworkers27 demonstrated a modest survival benefit in patients treated with intracoronary streptokinase.

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Percutaneous transluminal coronary angioplasty is less invasive and more rapidly applied than surgical revascularization. Angioplasty affords higher acute reperfusion rates with lower residual stenoses than does thrombolytic therapy.28 This 10-year review of our experience in treating acute myocardial infarction complicated by cardiogenic shock was undertaken to determine whether acute reperfusion of ischemic myocardium with coronary angioplasty altered prognosis.

Patients and Methods

Patients

From July 1975 to July 1985, 109 patients were admitted to the University of Michigan Medical Center with the diagnosis of acute myocardial infarction complicated by cardiogenic shock. Patients admitted from 1983 to 1985 were candidates for acute coronary angioplasty. Myocardial infarction was characterized by: 1) chest pain consistent with myocardial ischemia; 2) ST segment elevation of at least 1 mm in limb leads or at least 2 mm in precordial leads; and 3) creatine kinase MB isoenzyme elevation. Cardiogenic shock was defined as 1) a systolic blood pressure less than 80 mm Hg without inotropic or...
intra-aortic balloon pump support; 2) a systolic blood pressure greater than 90 mm Hg with inotropic or intra-aortic balloon pump support; and 3) evidence of adequate volume expansion demonstrated by a pulmonary capillary wedge pressure greater than 12 mm Hg. Twenty-six patients did not meet study criteria: five patients had sepsis; six had no evidence of myocardial infarction before death or at autopsy; 10 did not have adequate hemodynamic measurements; and five patients had shock responsive to volume expansion. Eighty-three patients composed the final study group. Fifty-nine patients (group 1) were treated without angioplasty in the coronary intensive care unit. All patients underwent right-heart catheterization and were treated with sympathomimetics, vasodilators, cardiac glycosides, antiarrhythmics, and intra-aortic balloon counterpulsation at the discretion of the staff cardiologist. Twenty-four patients (group 2) underwent angioplasty of the infarct-related artery, with subsequent admission to the coronary care unit for conventional therapy. Eight percent of patients in both group 1 (five of 59) and group 2 (two of 24) had a pulmonary capillary wedge pressure greater than 12 mm Hg but less than 18 mm Hg (p=NS). The pulmonary capillary wedge pressure was greater than 18 mm Hg in the remaining patients.

**Angioplasty**

Angioplasty was performed with (United States Catheter and Instrument [USCI]) guiding catheters and USCI steerable balloon systems. After initial coronary angiograms were obtained, attempts were made to pass the balloon catheter system across the culprit lesion. When successful, the balloon catheter was serially inflated until the translesional pressure gradient was less than 20 mm Hg. If the gradients could be reliably measured, contrast injections were used to assess arterial patency. Angioplasty of only the infarcted artery was attempted in each patient. The decision to perform coronary angioplasty was made by the interventional cardiologist who based his decision on the anatomic characteristics of the coronary arteriogram. Patients with acute myocardial infarction complicated by cardiogenic shock were not candidates for thrombolytic therapy and were excluded from investigational studies involving those agents. One patient in group 2 received intra-coronary streptokinase after successful angioplasty for treatment of residual thrombus.

**Statistical Analysis**

All numerical results are expressed as mean±SD. Tests for differences between the means were performed with two-tailed Student's t test for analytical data. Discrete variables were analyzed with the χ² test or Fisher's exact test where appropriate. A p value less than 0.05 was considered statistically significant. Cumulative survival was calculated by the life-table method. Differences between survival curves were analyzed by the logrank method.

**Results**

**Clinical Data**

There were no differences between group 1 and group 2 with respect to age, gender, history of hypertension, previous myocardial infarction, angina, or coronary artery bypass grafting (Table 1). Group 1 patients reported a higher usage of digoxin compared with group 2 patients (19% vs. 0%, p=0.02). Use of nitrates and β-blockers before hospitalization was similar between groups. Both groups had a high incidence of anterior wall infarction. Lateral wall infarction occurred infrequently. Four patients in group 1 had indeterminate infarct locations secondary to the development of either a new left bundle branch block (two patients), or atrioventricular dissociation (two patients). Myocardial infarction, however, was confirmed by a rise in creatine kinase MB isoenzymes in these patients. Dopamine was instituted with equal frequency in both groups; l-norepinephrine, however, was used more frequently in group 1 patients (33% vs. 8%, p=0.05), whereas dobutamine was used significantly more often in group 2 patients (50% vs. 26%, p=0.05). Intra-aortic balloon counterpulsation was initiated in 31% of group 1 patients and 88% of group 2 patients (p=0.001). No patient in group 1 underwent coronary artery bypass surgery during the hospitalization, whereas four patients in group 2 underwent bypass graft surgery. Two patients underwent surgery immediately from the catheterization laboratory, one because of dissection of the left anterior descending artery and the other because of inability to cross the lesion. Both patients died. Peak creatine kinase MB isoenzyme activity was significantly higher in group 2 patients compared with group 1 patients (5,888±5,607 vs. 3,151±2,969 IU, p=0.01). Group 2 patients also demonstrated a tendency toward earlier peak of creatine kinase MB activity (16±12 vs. 30±32 hours, p=0.07). The duration of chest pain to development of cardiogenic shock was longer in group 1 patients (47±53 vs. 28±30 hours), but this difference was not statistically significant. In group 2 patients, angioplasty was attempted at 20±32 hours after the onset of cardiogenic shock (range, 2–95 hours). In patients who had successful angioplasty (n=13), 60% were reperfused within 12 hours of the onset of shock. Life-threatening arrhythmias documented by electrocardiogram occurred with high frequency in both groups. Ventricular tachycardia occurred in 51% of group 1 patients and 46% of group 2 patients. Cardioversion was required in 36% of group 1 patients and 38% of group 2 patients.

**Hemodynamic Data**

Hemodynamic variables obtained during cardiogenic shock were similar. No statistically significant differences were found with respect to heart rate, systolic blood pressure, right atrial pressure, pul-
monary artery pressure, pulmonary capillary wedge pressure or cardiac index (Table 2).

30-Day Survival

Thirty-day survival (Figure 1) was significantly improved for group 2 patients compared with group 1 patients (50% vs. 17%, p<0.006). Between 1983 and 1985, acute cardiac catheterization was attempted in 30 of 40 (75%) patients, with coronary angioplasty attempted in 24 patients (60%). Two of the 16 (12.5%) patients treated with conventional therapy survived. Between 1975 and 1982, eight of 43 (19%) patients treated with interventional therapy survived; therefore, survival in patients not treated with coronary angioplasty remained constant throughout the study period (19% vs. 12.5%, p=NS). Subgroup analysis of angioplasty patients (Table 3) was performed to determine which factors affected survival. The left anterior descending artery was the infarcted artery in more of the nonsurvivors, but the difference was not statistically significant (83% vs. 67%, p=NS). Univariate analysis demonstrated that survivors of angioplasty had a significantly lower incidence of multivessel disease compared with nonsurvivors (42% vs. 83%, p=0.04). Survivors demonstrated a tendency toward improved hemodynamic variables with lower filling pressures and higher cardiac indexes than nonsurvivors. Figure 2 shows that 10 of 13 patients with successful angioplasty survived. In contrast, only two of 11 patients with unsuccessful angioplasty survived (77% vs. 18%, p=0.006).

Discussion

This study suggests that angioplasty improves survival in the setting of myocardial infarction complicated by cardiogenic shock. Mortality in the conventionally treated group was 84%, whereas mortality in the angioplasty group was 50% (p=0.006). When angioplasty was successful in achieving reperfusion, mortality was further reduced.

### Table 1. Clinical Data

<table>
<thead>
<tr>
<th></th>
<th>Group 1 (n=59)</th>
<th>Group 2 (n=24)</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (yr) (mean±SD)</td>
<td>62±12</td>
<td>59±12</td>
<td>NS</td>
</tr>
<tr>
<td>Male (%)</td>
<td>68</td>
<td>67</td>
<td>NS</td>
</tr>
<tr>
<td>Medical history (%)</td>
<td></td>
<td></td>
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<tr>
<td>Hypertension</td>
<td>36</td>
<td>33</td>
<td>NS</td>
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<tr>
<td>Myocardial infarction</td>
<td>46</td>
<td>33</td>
<td>NS</td>
</tr>
<tr>
<td>Angina</td>
<td>10</td>
<td>25</td>
<td>NS</td>
</tr>
<tr>
<td>Coronary artery bypass graft</td>
<td>5</td>
<td>4</td>
<td>NS</td>
</tr>
<tr>
<td>Medication (%)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>(before hospitalization)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Digoxin</td>
<td>19</td>
<td>0</td>
<td>0.02</td>
</tr>
<tr>
<td>Nitrates</td>
<td>20.3</td>
<td>33</td>
<td>NS</td>
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<tr>
<td>β-Blockers</td>
<td>17</td>
<td>29</td>
<td>NS</td>
</tr>
<tr>
<td>Infarct location (%)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Anterior</td>
<td>63</td>
<td>67</td>
<td>NS</td>
</tr>
<tr>
<td>Inferior</td>
<td>31</td>
<td>30</td>
<td>NS</td>
</tr>
<tr>
<td>Lateral</td>
<td>0</td>
<td>4</td>
<td>NS</td>
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<tr>
<td>Pressor support (%)</td>
<td></td>
<td></td>
<td></td>
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<tr>
<td>Dopamine</td>
<td>89</td>
<td>83</td>
<td>NS</td>
</tr>
<tr>
<td>l-norepinephrine</td>
<td>33</td>
<td>8</td>
<td>0.05</td>
</tr>
<tr>
<td>Dobutamine</td>
<td>26</td>
<td>50</td>
<td>0.05</td>
</tr>
<tr>
<td>Intra-aortic balloon pump</td>
<td>31</td>
<td>88</td>
<td>0.001</td>
</tr>
<tr>
<td>Laboratory</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Creatine kinase (IU)</td>
<td>3,152±2,937</td>
<td>5,888±5,607</td>
<td>0.01</td>
</tr>
<tr>
<td>Symptom duration</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Chest pain to shock (hr)</td>
<td>47±53</td>
<td>28±30</td>
<td>NS</td>
</tr>
<tr>
<td>Shock to reperfusion (hr)</td>
<td>20±32</td>
<td>. . .</td>
<td>. . .</td>
</tr>
</tbody>
</table>

Values are mean±SD.

### Table 2. Hemodynamic Data

<table>
<thead>
<tr>
<th></th>
<th>Group 1 (n=59)</th>
<th>Group 2 (n=24)</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>Heart rate</td>
<td>99±25</td>
<td>103±26</td>
<td>NS</td>
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<tr>
<td>Systolic blood pressure (mm Hg)</td>
<td>74±11</td>
<td>75±12</td>
<td>NS</td>
</tr>
<tr>
<td>Right atrial pressure (mm Hg)</td>
<td>14±8</td>
<td>13±5</td>
<td>NS</td>
</tr>
<tr>
<td>Pulmonary artery pressure (mm Hg)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Systolic</td>
<td>43±13</td>
<td>42±10</td>
<td>NS</td>
</tr>
<tr>
<td>Diastolic</td>
<td>25±8</td>
<td>23±6</td>
<td>NS</td>
</tr>
<tr>
<td>Pulmonary capillary wedge pressure (mm Hg)</td>
<td>25±8</td>
<td>24±5</td>
<td>NS</td>
</tr>
<tr>
<td>Cardiac index (l/min/m²)</td>
<td>1.8±0.7</td>
<td>2.0±0.8</td>
<td>NS</td>
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</tbody>
</table>
to 23%. Although conventionally treated patients were drawn from 1975 to 1985, while angioplasty patients were treated between 1983 and 1985, the two groups were similar with respect to demographic data, hemodynamic variables, extent of coronary disease, and infarct location. It is possible that a positive patient selection bias existed for group 2 patients because they survived to the catheterization laboratory and could be considered for angioplasty. Group 2 patients had a slightly lower incidence of previous myocardial infarction (33% vs. 46%) and shorter duration of chest pain to the onset of cardiogenic shock (28 vs. 47 hours). However survival rates in those patients eligible for angioplasty from 1983 to 1985 but treated conventionally were not different than survival rates for patients treated conventionally from 1975 to 1983. This is a retrospective study, and unrecognized factors may in part account for the differences in outcome between conventionally treated and angioplasty-treated patients.

Cardiogenic shock develops in 5% of patients hospitalized with acute myocardial infarction.\textsuperscript{29,30} Despite dramatic improvements in coronary care, the 80–90% in-hospital mortality associated with cardiogenic shock remains nearly unchanged.\textsuperscript{31–34} Decreased myocardial perfusion and progressive myocardial necrosis amplify the severity of the original lesion perpetuating the vicious cycle of the shock state.\textsuperscript{35–38} Standard drug therapy with sympathomimetic amines has not improved survival in cardiogenic shock. Although β-adrenergic agonists increase both the force and velocity of myocardial contraction, this positive inotropic effect is achieved at the expense of increased myocardial oxygen consumption.\textsuperscript{39–43} Mueller and colleagues\textsuperscript{44} demonstrated that isoproterenol improved total cardiac performance but caused a deterioration in myocardial cell metabolism despite an increase in coronary perfusion. Intra-aortic balloon counterpulsation for shock has produced little long-term benefit. Hagemeijer et al\textsuperscript{45} reported a 1-year mortality of 71% (15 of 21 patients died). This mortality is comparable to that reported by Chatterjee et al,\textsuperscript{46} who used pharmacological afterload reduction with intra-aortic balloon pump assistance.\textsuperscript{46} Scheidt and coworkers\textsuperscript{19} reported similar results in a controlled cooperative study; mortality at one year was 90% in 87 patients. DeWood and associates\textsuperscript{26} first suggested that coronary reperfusion with emergent coronary bypass

### Table 3. Acute Angioplasty

<table>
<thead>
<tr>
<th></th>
<th>Survivor (n=12)</th>
<th>Nonsurvivor (n=12)</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>Infarct artery (%)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Left anterior descending</td>
<td>66</td>
<td>83</td>
<td>NS</td>
</tr>
<tr>
<td>Right coronary</td>
<td>17</td>
<td>9</td>
<td>NS</td>
</tr>
<tr>
<td>Left circumflex</td>
<td>17</td>
<td>9</td>
<td>NS</td>
</tr>
<tr>
<td>Multivessel disease (%)</td>
<td>42</td>
<td>83</td>
<td>0.04</td>
</tr>
<tr>
<td>Hemodynamics (after intervention)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Right atrial pressure (mm Hg)</td>
<td>9±4</td>
<td>11±9</td>
<td>NS</td>
</tr>
<tr>
<td>Pulmonary capillary wedge pressure (mm Hg)</td>
<td>15±5</td>
<td>22±10</td>
<td>0.08</td>
</tr>
<tr>
<td>Cardiac index (l/min/m²)</td>
<td>2.5±0.9</td>
<td>1.5±0.6</td>
<td>0.06</td>
</tr>
</tbody>
</table>

Data are mean±SD.
grafting and intra-aortic balloon pumping improved outcome in acute myocardial infarction complicated by cardiogenic shock. They reported a 74% long-term mortality (6–42 months) in patients treated with balloon pump assistance alone. However, when balloon assistance was coupled with emergent surgical revascularization to reestablish coronary perfusion within 16 hours of infarction in selected patients, mortality was reduced to 25% (p<0.03). Leinbach et al in a small cohort of seven patients demonstrated a 58% mortality rate with surgical reperfusion in the setting of shock. Mundth and coworkers reported a mortality rate of 56% in patients with cardiogenic shock who underwent emergent bypass graft surgery after intra-aortic balloon pump assistance.

Percutaneous transluminal angioplasty is a rapid and effective method of establishing coronary patency and a widely used alternative to coronary bypass graft surgery. Successful reperfusion with coronary angioplasty in the setting of acute myocardial infarction approaches 80–90%. Although our success rate in establishing reperfusion in this early group of shock patients was only 54%, current reperfusion rates in this population are greater than 70% because of improvements in catheter design and increased operator experience. The disappointing results with respect to survival in the group of patients with multivessel disease (83% mortality) despite successful angioplasty suggests that angioplasty is best reserved for patients with single-vessel disease. In patients with multivessel disease and cardiogenic shock, early coronary artery bypass graft surgery should be considered. Interestingly, our data suggests that the time frame during which benefit may be derived from reperfusion strategies in acute myocardial infarction complicated by cardiogenic shock may be somewhat longer than that reported for uncomplicated myocardial infarction. Although 40% of our patients underwent angioplasty within 4 hours of symptom onset, improved survival was documented in patients undergoing angioplasty many hours after this critical period. In animal studies, Hochman and Choo demonstrated a beneficial effect of late reperfusion on limitation of infarct expansion and aneurysm formation independent of myocardial salvage. Force et al demonstrated that late coronary reperfusion leads to an acute reduction in diastolic and systolic infarct expansion in the dog model. Furthermore, delayed reperfusion may improve the rate and extent of infarct healing.

Thrombolytic therapy offers an attractive rapid method of establishing reperfusion in patients with cardiogenic shock. However, Mathey et al and Rentrop et al have demonstrated low rates of recanalization in shock patients with intracoronary streptokinase and no improvement in mortality over conventional therapy. The Gruppo Italiano per lo Studio della Streptochinasi nell'Infarto Miocardico (GISSI) trial also demonstrated a high mortality in patients with cardiogenic shock treated with intravenous streptokinase alone. Angioplasty can be performed quickly and with a high rate of success in this patient group. Although this study is retrospective and uncontrolled, the data strongly suggest that urgent angioplasty may dramatically reduce mortality in the setting of acute myocardial infarction complicated by cardiogenic shock, particularly with single-vessel disease. Randomized, controlled studies will be required to definitively determine the role of angioplasty therapy in this patient population.

References
3. Swan HJC, Danzig R, Sukumalchantra Y: Current status of treatment of power failure of the heart in acute myocardial...


KEY WORDS • cardiogenic shock • myocardial infarction • coronary angioplasty
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