Intraaortic Balloon Counterpulsation With and Without Reperfusion for Myocardial Infarction Shock

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SUMMARY Forty patients were treated for cardiogenic shock secondary to acute myocardial infarction. Twenty-one (group 1) were treated with intraaortic balloon counterpulsation and 19 (group 2) were treated with counterpulsation and coronary artery bypass grafting. The groups were similar in age, incidence of previous infarction, initial hemodynamics and coronary anatomy.

The in-hospital mortality between group 1 (52.4%) and group 2 (42.1%) was not significantly different. The difference in long-term mortality between group 1 and group 2 was substantially different (71.4% vs 47.3%). The subset of group 2 (n = 12) that underwent reperfusion and counterpulsation within 16 hours from the onset of symptoms of infarction had a lower mortality (25.0%) than the subset (n = 7) that underwent operation more than 18 hours after the onset of symptoms (71.4%). The long-term mortality in the subset of group 2 patients operated on within 16 hours after the onset of infarction was significantly different from that in group 1 (25.0% vs 71.4%, p < 0.03). The data suggest that reperfusion with counterpulsation is more effective when carried out early. Patients who develop shock more than 18 hours after the onset of symptoms of infarction appear to benefit most if treated with counterpulsation alone.

CARDIOGENIC SHOCK is currently the most common cause of death in patients hospitalized for acute myocardial infarction. Pathologic evidence suggests that power failure subsequent to myocardial infarction is associated with a critical loss of ventricular myocardium approximating 40% of left ventricular mass. Moreover, it appears that in the setting of acute infarction, cardiogenic shock is an evolving event characterized by gradual impairment of functioning heart muscle.

Early results suggested that intraaortic balloon counterpulsation, although of temporary benefit in the management of cardiogenic shock, only marginally improved the long-term outlook for patients suffering from shock. In contrast, recent data suggest that early counterpulsation alone may offer favorable long-term results. On the other hand, early descriptions of the combined use of balloon counterpulsation with surgery for cardiogenic shock indicated substantial reduction of mortality in patients treated early after shock developed.

It was unclear, therefore, whether comparable patient groups suffering from cardiogenic shock are best benefited by mechanical assist alone or in conjunction with emergency bypass grafting. Accordingly, we reviewed our results in the treatment of cardiogenic shock by intraaortic balloon counterpulsation with and without reperfusion.

Methods

Patients and Clinical Characteristics

From December 1973 to July 1978, 883 patients (ages 31–82 years) were admitted to the Sacred Heart and Deaconess Medical Centers with a diagnosis of acute transmural myocardial infarction characterized by 1) chest pain consistent with myocardial infarction, 2) persistent ST-segment elevation progressing to Q waves greater than 0.04 second by ECG, and 3) CK-MB isoenzyme elevation. The overall mortality for transmural infarction in this group was 17%.

Definition of Cardiogenic Shock

Forty patients were treated with intraaortic balloon counterpulsation for shock associated with acute myocardial infarction. Shock was defined as 1) central systolic arterial blood pressure less than 85 mm Hg (mean blood pressure less than 70 mm Hg), 2) functional volume expansion characterized by elevation of left ventricular filling pressure ≥ 16–18 mm Hg, 3) clouded sensorium, 4) evidence of peripheral hypoperfusion on clinical exam (i.e., cold extremities with prolonged capillary filling time), 5) depressed urine flow, and 6) continued need for vasopressor support despite volume expansion. All criteria were necessary for inclusion.

Patient Groups

From this group of 40 patients with cardiogenic shock two groups were identified.

Group 1 — Intraaortic Balloon Counterpulsation Alone

Twenty-one patients (16 males and five females) with a mean age of 55.1 years (range 34–70 years) who
were admitted with acute myocardial infarction and developed cardiogenic shock were treated with intraaortic balloon counterpulsation alone. The patients were hospitalized within 10 hours after the onset of symptoms. Clinical and hemodynamic characteristics are listed in table 1. The area of infarction and mortality data are summarized in table 2. Initially, each patient was managed by routine coronary care unit protocol consisting of nitrates and morphine sulfate for pain, lidocaine for ventricular irritability, nasal oxygen and intravenous fluids. None of the patients evidenced cardiogenic shock when first observed.

The initial sign of cardiogenic shock was hypotension. Vasopressors (consisting of either dopamine or norepinephrine) were administered to establish an arterial systolic blood pressure of 100 mm Hg (monitored by intra-arterial needle). Subsequently, all patients underwent right-heart catheterization with a Swan-Ganz catheter. Continued hypotension after volume expansion was required for the diagnosis of shock. Intraaortic balloon counterpulsation was initiated in this group within an average of 9.1 hours (range 3–16 hours) from the onset of hypotension and within an average of 20 hours from the onset of infarction (range 8–48 hours).

Group 2 — Counterpulsation and Revascularization

Nineteen patients (16 males and three females) with an average age of 51.7 years (range 38–64 years) were admitted with a diagnosis of myocardial infarction. These patients manifested shock slightly earlier in their hospital course than patients in group 1 (mean 6 hours, range 2–16 hours), and within an average of 13.1 hours from the onset of infarction (range 5–25 hours). These patients underwent early surgical revascularization and intraaortic balloon counterpulsation.

Each patient underwent cardiac catheterization and was immediately taken to surgery. Initial hemodynamic data are listed in table 3. The area of infarction and mortality data are summarized in table 4. Pulmonary congestion in patients 3 and 5 precluded further volume challenge. All patients required vasopressor support during angiography. In addition, two patients were studied with intraaortic balloon support. In the remainder, intraaortic balloon counterpulsation was instituted at operation. Vasopressor support and intraaortic balloon assistance were continued postoperatively.

Selection Criteria

Forty patients received intraaortic balloon counterpulsation for coronary shock. The patients in group

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**Table 1. Hemodynamics and In-hospital Mortality of Patients Receiving Pharmacologic and Intraaortic Balloon Treatment (Group 1)**

<table>
<thead>
<tr>
<th>Pt</th>
<th>Age (years)</th>
<th>CI (l/min/m²)</th>
<th>LVFP (mm Hg)</th>
<th>SBP (mm Hg)</th>
<th>IHM*</th>
</tr>
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<tr>
<td>21</td>
<td>50</td>
<td>2.31</td>
<td>22</td>
<td>85</td>
<td>-</td>
</tr>
</tbody>
</table>

Mean 55.1 ± 8.2

*Eleven deaths (52.4%).

Abbreviations: CI = cardiac index; SBP = central systolic blood pressure; IHM = in-hospital mortality; + = survivor; — = non-survivor.

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**Table 2. Area of Infarction, In-hospital and Long-term Mortality, Incidence of Previous Myocardial Infarction and Hemodynamic Values (Group 1)**

<table>
<thead>
<tr>
<th>ECG site of infarction</th>
<th>No. of pts</th>
<th>Previous MI</th>
<th>CI (l/min/m²)*</th>
<th>LVFP (mm Hg)</th>
<th>IHM</th>
<th>LTM</th>
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<tbody>
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<td>1.93 ± 0.15</td>
<td>24.8 ± 3.6</td>
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<td>6</td>
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<td>Anteroinferior</td>
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<td>1.82 ± 0.15</td>
<td>26.0 ± 2.4</td>
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<td>5</td>
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<td>2</td>
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<tr>
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<td>1</td>
<td>2.13 ± 0.08</td>
<td>21.25 ± 0.4</td>
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<td>2</td>
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<td>1</td>
<td>2.25</td>
<td>32</td>
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<td></td>
</tr>
</tbody>
</table>

Totals 21 10 11 (52.4%) 15 (71.4%)

*Mean ± SD.

Abbreviations: MI = myocardial infarction; CI = cardiac index; LVFP = left ventricular filling pressure; IHM = in-hospital mortality; LTM = long-term mortality (6–24 months).
TABLE 3. Hemodynamics and In-hospital Mortality of Patients Receiving Pharmacologic Support, Intraaortic Balloon Assist and Saphenous Vein Bypass Grafting (Group 2)

<table>
<thead>
<tr>
<th>Pt</th>
<th>Age (years)</th>
<th>CI (l/min/m²)</th>
<th>LVFP (mm Hg)</th>
<th>EF (%)</th>
<th>SBP (mm Hg)</th>
<th>IHM*</th>
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<tr>
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<td>2.20</td>
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<td>35</td>
<td>75</td>
<td>+</td>
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<tr>
<td>19</td>
<td>50</td>
<td>2.27</td>
<td>30</td>
<td>40</td>
<td>85</td>
<td>+</td>
</tr>
</tbody>
</table>

Mean = 51.7 ± 7.1 1.89 ± 0.32 23.5 ± 5.1 29 ± 6.7 74.9 ± 7.2

*Eight deaths (42.1%).

Abbreviations: CI = cardiac index; LVFP = left ventricular filling pressure; EF = ejection fraction; SBP = central systolic blood pressure; IHM = in-hospital mortality; + = survivor; - = nonsurvivor.

1 were allocated to intraaortic balloon therapy alone because the physician initially managing the patient did not participate in the program involving acute revascularization presented elsewhere or because the patient declined therapy other than balloon pumping. Group 2 was evaluated angiographically with vasopressor support alone to eliminate the additional time required for insertion of the intraaortic balloon before angiography. The intraaortic balloon was inserted when the patient was in surgery as bypass grafting proceeded.

Exclusions

Patients with diffuse distal disease of the coronary arteries and those receiving mitral valve replacement were excluded from this study. There were no other exclusions; all patients with acute myocardial infarction and shock were included in the two groups.

Informed Consent

All patients gave informed consent. The advised consent form had been reviewed by the Human Ex-

TABLE 4. Area of Infarction, Incidence of Previous Myocardial Infarction, Hemodynamic Values, In-hospital Mortality and Long-term Mortality (Group 2)

<table>
<thead>
<tr>
<th>ECG site of infarction</th>
<th>No. of pts</th>
<th>Previous MI</th>
<th>CI (l/min/m²)*</th>
<th>LVFP (mm Hg)*</th>
<th>IHM</th>
<th>LTM</th>
</tr>
</thead>
<tbody>
<tr>
<td>Anterior</td>
<td>11</td>
<td>6</td>
<td>1.89 ± 0.29</td>
<td>24.3 ± 4.90</td>
<td>5 (45%)</td>
<td>6 (54.6%)</td>
</tr>
<tr>
<td>Anteroinferior</td>
<td>4</td>
<td>1</td>
<td>2.04 ± 0.16</td>
<td>23.2 ± 2.41</td>
<td>2 (50%)</td>
<td>2 (50%)</td>
</tr>
<tr>
<td>Anterolateral</td>
<td>2</td>
<td>1</td>
<td>1.85</td>
<td>30</td>
<td>1 (50%)</td>
<td>1 (50%)</td>
</tr>
<tr>
<td>Inferior</td>
<td>2</td>
<td>1</td>
<td>1.78</td>
<td>23</td>
<td>—</td>
<td>—</td>
</tr>
<tr>
<td>Totals</td>
<td>19</td>
<td>9</td>
<td></td>
<td></td>
<td>8 (42%)</td>
<td>9 (47.3%)</td>
</tr>
</tbody>
</table>

*Mean ± sd.

Abbreviations: MI = myocardial infarction; CI = cardiac index; LVFP = left ventricular filling pressure; IHM = in-hospital mortality; LTM = long-term mortality (6–24 months).
perimentation Committee of both hospitals. If the patient was unable to understand the nature of the interventions, family members were specifically informed that the procedures were not proved to decrease mortality and were experimental, but that the results might be of direct benefit to the patient.

Balloon Management

The AVCO and Datascope System 80 intraaortic balloon pumps were used equally in each group. A 30-ml or 40-ml intraaortic balloon was inserted through an end-to-side graft sewn to the femoral artery and advanced to the mouth of the left subclavian artery. The balloon was deflated automatically by the R wave of the ECG. In all cases, the balloon was timed to inflate at the dicrotic notch (as determined by radial or brachial arterial trace), thus ensuring maximal diastolic augmentation (counterpulsation) and appropriate timing of the balloon pump. During pumping, heparin boluses (4000 units i.v. every 4 hours) were used to maintain the partial thromboplastin time (PTT) 50–60 seconds (normally less than 35 seconds in our hospitals). PTT, arterial blood gases, hemoglobin and hematocrits were measured every 12 hours after each patient was stable. The PTT was monitored every 3½ hours until heparin therapy had been titrated to achieve the desired PTT. Low molecular weight dextran given intravenously at 10 ml/hour was used in addition to heparin therapy in two patients.

Weaning from counterpulsation was begun gradually, with each patient being “pumped” every other beat, then every third beat, and so on. If a patient became hemodynamically unstable (characterized by rising pulmonary capillary wedge pressure, falling cardiac output, decline in blood pressure) counterpulsation was resumed on a one-to-one basis. All patients received counterpulsation for 3 days or longer (range 3–13 days).

Area of New Myocardial Infarction and Incidence of Previous Myocardial Infarction

In group 1, 10 patients had suffered a previous myocardial infarction (47.6%) (table 2). Sixteen of the 21 new infarctions (76%) involved the anterior wall by ECG: nine (42%) involved the precordial leads alone (i.e., V₁ to V₄), while five had both anterior and inferior infarction, and two had anterolateral infarction. Predominantly inferior wall infarction was present in five patients.

Nine of 19 patients in group 2 (47.3%) had previous myocardial infarction (table 4). Seventeen patients (89.4%) had anterior wall involvement. Eleven of the 19 new infarctions (57.8%) involved the precordial leads alone, while four patients had both anterior and inferior wall infarction and two had anterolateral infarction. There were two inferior wall infarctions in this group.

Coronary Anatomy

A stenosis was considered significant if greater than 70% of the luminal diameter was occluded.

Coronary anatomy was available in 19 of the 21 patients in group 1. In one case, permission for autopsy was not granted, and patient 20 refused angiography after hospitalization. Of the 19 patients with known anatomy, two were catheterized before infarction and eight within 6 weeks after infarction. Nine patients were examined at autopsy.

Two patients had left main disease (100% and 90% obstruction). Left main disease was counted as two-vessel disease in this study. Five patients had three-vessel disease and 13 had two-vessel disease. One had one-vessel obstruction (a large, dominant right coronary artery). All patients had proximal disease.

Coronary angiography and ventriculography were performed preoperatively in all group 2 patients, and each had proximal disease. Left main coronary artery involvement was present in two patients (100% and 80% obstruction). Three-vessel disease was present in five patients. Eleven patients had two-vessel disease, and three patients had one-vessel involvement (two patients had proximal right coronary artery occlusion associated with a diminutive left system).

Autopsy Study

The whole heart was removed from the chest in each case autopsied. The epicardial arteries were removed, decalcified and sectioned at 1-mm intervals. Each heart was weighed and the area of infarction was grossly identified. Because the measurement of interest was the presence or absence of hemorrhage, samples were taken from both infarcted and normal myocardium. After the junction of infarcted and normal myocardium was identified, multiple samples were taken. The area that included the junction of both normal and infarcted myocardium was embedded in parafilm; serial sections were systematically examined and special attention was given to histologic features that might be consistent with extension of infarction into areas of normal myocardium.

Statistical Analysis

The t test (unpaired) was used for the analysis of continuous measurement variables and the chi-square test was used for discrete outcome variables.

Results

Comparability of Groups

The groups in this study are concurrent and appear to be comparable (table 5) in age, incidence of previous infarction, mean cardiac index, average initial systolic blood pressure, mean left ventricular filling pressure, the time interval from the onset of hypotension (the earliest marker of cardiogenic shock) to the institution of intraaortic balloon counterpulsation (mean 9.1 vs 6.0 hours) and the extent of coronary disease (2.2 vessels vs 2.1 vessels per patient). Although there were slightly more inferior infarctions in group 1, the incidence of proximal disease and the area of infarction by ECG were similar between
groups. However, the time interval from the onset of infarction to hemodynamic instability was slightly longer in group 1 than in group 2.

Mortality Between Groups

The in-hospital mortality of the groups was not significantly different (42% vs 52.4%). Anterior wall involvement carried the greatest mortality in each group (62.5% in group 1 vs 47% in group 2). Inferior wall infarction was more benign in both groups (one death vs no deaths). The subset of group 2 treated within 16 hours from the onset of symptoms showed a 25% (three of 12) hospital mortality.

Three patients in group 1 died before weaning from counterpulsation was begun. No patient was left balloon-dependent. Two patients in group 2 could not be weaned from cardiopulmonary bypass.

The long-term mortality (6–42 months) for group 1 was 71.4% (15 of 21) and 47.3% (nine of 19) for group 2. All deaths occurred within 12 months of hospitalization from progressive power failure. The difference in long-term mortality with anterior wall involvement was especially marked, as 81.2% of group 1 had died vs 52% of group 2. Further, the long-term mortality between group 1 and the subset of group 2 treated within 16 hours from the onset of infarction was significantly different (71.4% vs 25%, p < 0.03).

Mortality in Group 1

Anterior wall involvement by ECG was associated with a 62.5% (10 of 16) mortality rate, compared with predominantly inferior wall involvement, which had a 20% (one of five) mortality rate (table 2). The trend continued in the follow-up period, as the total mortality with anterior involvement rose to 81.2% (13 of 16) vs 40.0% (two of five) with inferior infarction. Of the five long-term survivors, two had previous infarction.

Neither average age nor systolic blood pressure distinguished survivors from nonsurvivors (54.6 years vs 56.2 years, 76.6 mm Hg vs 75.0 mm Hg, respectively). Likewise, the average time from the onset of clinical shock to the institution of counterpulsation did not discriminate between survivors and nonsurvivors (7.2 hours vs 9.4 hours), and the average left ventricular filling pressures (25.5 mm Hg vs 25.2 mm Hg) were not significantly different. However, the cardiac index (2.1 l/min/m² vs 1.84 l/min/m² (p < 0.05) was significantly different between survivors and nonsurvivors, possibly reflecting less myocardial injury. Of the survivors, two patients are in functional class I (by New York Heart Association criteria), two are in functional class II, and one patient is in class III. None has had cardiac surgery.

Mortality in Group 2

Patients in group 2 with anterior wall involvement suffered the greatest mortality in the hospital (47.0%, eight of 17) (table 4). The additional mortality in the follow-up period brought total mortality to 52.9% in patients with anterior involvement. Both patients with inferior infarction survived.

The average cardiac index was somewhat higher in the survivors than in nonsurvivors (2.0 l/min/m² vs 1.6 l/min/m²) (p < 0.01), but this impairment may have reflected earlier investigation from the onset of infarction in this group (11.4 hours vs 15.2 hours), as the condition of survivors and nonsurvivors alike were deteriorating rapidly. In addition, the ejection fraction was slightly higher in survivors (31.5% vs 27.6%, p < 0.05). Average age (50.3 vs 53.7 years), systolic blood pressure (74.4 mm Hg vs 76.2 mm Hg) and the left ventricular filling pressure (24.4 mm Hg vs 22.2 mm Hg) (all p = NS) were poor indicators of survival.

The major determinant of a favorable result in group 2 (table 4) appeared to be the time from the onset of infarction to the institution of treatment with bypass grafting and counterpulsation. The subset of patients treated more than 18 hours after the onset of infarction had a 71.4% (five of seven) in-hospital mortality, while the patients treated within 16 hours had a 25% (three of 12) mortality rate. No patient was treated between 16 and 18 hours from the onset of infarction. Moreover, the only long-term death occurred in the group treated more than 18 hours after onset. The subset of group 2 treated within 16 hours after the onset of symptoms also had a significantly higher cardiac index (1.99 ± 0.42 vs 1.77 ± 0.74, p < 0.05), but the mean left ventricular filling pressure, systolic arterial blood pressure, average age, area of infarction by ECG, incidence of previous infarction and the number of diseased vessels per patient were not significantly different. We do not know if the greater depression of cardiac index in the subset treated more than 18 hours after the onset of symptoms was a reflection of greater necrosis.

Of the 10 long-term survivors, four are in functional class I, five are in class I and one is in class III.

All patients (n = 4) autopsied in this group had gross hemorrhagic infarction (fig. 1), but no apparent extension of injury into area of normal myocardium. The amount of myocardium damaged approached 50% in each instance.
Discussion

This study demonstrates a striking reduction in mortality for patients operated on within 16 hours (25%) after the onset of infarction and subsequent shock. When grafting was performed more than 18 hours after the onset of symptoms, the mortality rate was disappointing (71.4%). The significance of this is unclear, but it may argue for a "critical period" for revascularization after which healing may be required, presumably accompanied by balloon support.

Groups 1 and 2 are concurrent as well as comparable in age, proximal coronary disease, incidence of multivessel disease, the incidence of previous infarction and hemodynamic measurements (table 5). The long-term mortality of each group was substantially different (47% vs 71.2%), especially when anterior wall involvement was present (52% vs 81.2%) and in the patients of group 2 operated on within 16 hours after the onset of symptoms of infarction (25% vs 71.4%, p < 0.03).

Comparison with Other Studies

The bulk of previous clinical experience8, 9, 16-19 with intraaortic balloon counterpulsation for cardiogenic shock reported only slight improvement in the long-term mortality of patients treated with this modality alone. However, recent reports suggest that the long-term prognosis for patients with coronary shock treated with counterpulsation alone may be comparable to that for patients undergoing surgery and mechanical assistance.12 Accordingly, this report was designed to investigate in concurrently treated groups whether counterpulsation alone applied in the early stage of coronary shock obviates the need for cardiac surgery.

Our results with intraaortic counterpulsation alone are comparable to those of other reported series8, 9, 16-19 and the outcome, in some respects, is more favorable. These data confirm the reports of both Johnson10 and Hagemeijer,11 in that counterpulsation in the early phase of cardiogenic shock may result in a lower hospital mortality than reported by Scheidt8 and Leinbach.18 This may be due in part to earlier application of counterpulsation after the onset of shock. The long-term survival, however, is not as favorable in that 71.4% (15 of 21) of the group died within 1 year. This figure is similar to that reported by Chatterjee20 and co-workers, who used careful afterload reduction without mechanical assistance.

Our long-term results with mechanical assistance alone are only slightly more favorable than the reports of Scheidt8 and Mueller.16 Inability to confirm the

<table>
<thead>
<tr>
<th>Table 6. Clinical and Hemodynamic Evaluation of Group 2 as Related to Time from Onset of Infarction to Bypass Grafting and Intraaortic Balloon Assist</th>
</tr>
</thead>
<tbody>
<tr>
<td>Time from onset of infarction to interventions</td>
</tr>
<tr>
<td>Clan (lit/min/m²)</td>
</tr>
<tr>
<td>LVFP (mm Hg)</td>
</tr>
<tr>
<td>SBP (mm Hg)</td>
</tr>
<tr>
<td>Clinical description</td>
</tr>
<tr>
<td>Age (years)</td>
</tr>
<tr>
<td>Area of MI by ECG</td>
</tr>
<tr>
<td>Ant</td>
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<tr>
<td>Inf</td>
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<tr>
<td>Ant + Inf</td>
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<tr>
<td>Previous MI</td>
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<tr>
<td>No. of diseased vessels per patient</td>
</tr>
<tr>
<td>One</td>
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<tr>
<td>Two</td>
</tr>
<tr>
<td>Three</td>
</tr>
<tr>
<td>IHM</td>
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</tbody>
</table>

*Mean = sd. 
†p < 0.05.

Abbreviations: CI = cardiac index; LVFP = left ventricular filling pressure; SBP = central systolic blood pressure; MI = myocardial infarction; IHM = in-hospital mortality; ANT = anterior; INF = inferior.
long-term benefits of balloon therapy alone may be
due to patient selection. In group 1, diffuse distal dis-
case was excluded from the report; otherwise, the
patients were unselected. Hagemeijer et al.\textsuperscript{11} excluded
patients with acute ischemia within 36 hours of
balloon assist, as well as those older than 65 years of
age.

Johnson and co-workers\textsuperscript{19} indicated that patients
with inferior infarction were more likely to survive
with balloon assist alone. Our data confirm that find-
ing, because three of our long-term survivors suffered
shock with inferior infarction. The mortality rate with
anterior wall involvement remained prohibitively
high, however, as only three of 16 (18.6\%) survived
after 1 year of follow-up.

Rationale for Reperfusion

Theoretically, the most direct method to limit the
extent of infarction is to restore blood flow. Ginks et
al.\textsuperscript{21} as well as Corday,\textsuperscript{22} have shown in animal
models that reperfusion after the first several hours of
coronary occlusion leads to a substantial decrease in
the extent of infarction compared with control
animals, with eventual return of functional myocar-
dium.

Early clinical data presented by Berg et al.\textsuperscript{14}
suggested that restoration of blood flow in the early
phase of acute evolving myocardial infarction carried
a low mortality. The time course of myocardial infarc-
tion in man is unknown, however. Consequently, it is
uncertain what volume of myocardium remains to be
salvaged in the shock state. The data of Dunkman\textsuperscript{12}
and Keon\textsuperscript{23} suggest that clinical cardiogenic shock
may behave differently from uncomplicated infarc-
tion, aside from the higher mortality, in that patients
responded favorably to bypass grafting well along in
the time course of infarction, indicating salvage of
substantial portions of myocardium.

The data of Sobel et al.\textsuperscript{6} may be pertinent to this
issue. They investigated enzymatically a group of
patients suffering from cardiogenic shock and found
that in the majority of cases, release of MB-CK was
an evolving event that paralleled cellular deterioration.
Moreover, the peak MB-CK values plateaued later and
the elevations were more sustained than with un-
complicated infarction, indicating that interventions
applied early in cardiogenic shock may interrupt the
progressive injury seen in their patients.

Accordingly, the Massachusetts General Hospital
group, recognizing that significantly more patients
sustaining anterior myocardial infarction develop
shock,\textsuperscript{13} have initiated an aggressive program of
beginning mechanical assistance in anterior infarction
before shock is manifest.\textsuperscript{24} Further, the same group
has recently shortened the time interval between
balloon assistance and surgery\textsuperscript{29} in an attempt to
protect ischemic but salvageable myocardium and
thereby prevent the shock syndrome.

Hemorrhagic Infarction

Bresnahan and co-workers,\textsuperscript{18} working with reper-
fusion studies in dogs, demonstrated the possible
hazard of hemorrhagic infarction if reperfusion was
carried out more than 5 hours after abrupt occlusion.
The significance of hemorrhagic infarction in our
patients is unknown. Bresnahan et al.\textsuperscript{18} described
deleterious effects due to reperfusion hemorrhage.
Their conclusion was based on augmentation of total
CK activity after reflow. Moreover, the hemorrhage
described was sampled only from the center of the
infarct zone. Recently, however, Roe et al.,\textsuperscript{27} as well as
Vatner and co-workers,\textsuperscript{28} showed that augmentation
of CK activity may be a "washout" phenomenon and
not indicative of extension of necrosis after reper-
fusion. Recent investigation of the effects of reperfusion
on infarct size did not identify extension of infarction
when measured by \textsuperscript{51}Cr-labeled red blood cells.\textsuperscript{29}

We could not find evidence in favor of extension of
infarction due to reperfusion, as all autopsy ex-
aminations indicated extensive infarction, but the
hemorrhagic areas appeared to be confined to the area
of necrosis. The unfavorable outcome of patients
autopsied appeared to reflect the extent of underlying
disease rather than aggravation of injury.

The precise role of infarctectomy as an adjunct to
surgical revascularization is poorly defined. While
Dunkman et al.\textsuperscript{12} used infarctectomy in a large portion
of their series, infarctectomy was used in only one of
our patients. It is unknown whether infarctectomy
offers any advantage over reperfusion alone. If reper-
fusion hemorrhage is proved to be truly deleterious,
infarctectomy may be helpful. On the other hand, the
significance of reperfusion hemorrhage confined to the
area of infarction is uncertain. Further, excision of
large portions of ventricular myocardium from a
severely damaged ventricle may leave the patient with
insufficient myocardial mass to support an already
failing circulation.

Clinical Implications

A controlled, randomized trial may be difficult in
such high-risk groups. We recognized the problems of
such a study and attempted to assess treatment of
comparable, concurrent groups. Our results suggest
that patients given immediate cardiac assistance with
reperfusion have improved short and long-term sur-
vival if these procedures can be performed within 16
hours after the onset of infarction.

The use of inotropic\textsuperscript{16, 18, 26} or vasodilating agents,\textsuperscript{20}
sometimes used in combination,\textsuperscript{49} has not improved
the long-term survival of these patients, possibly
because of failure to appreciate the major deter-
mnants dictating the fate of jeopardized myocardium.
Further, the inability of intraaortic balloon counter-
pulsation alone to ensure long-term survival casts
uncertainty on the value of this therapy without add-
tional measures. These data suggest that early reper-
fusion and counterpulsation offer advantages over
mechanical assist alone. Finally, without early,
aggressive treatment, the prognosis for evolving cor-
nary shock remains mostly unsatisfactory.
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