Emergency Coronary Artery Revascularization: A Possible Therapy for Acute Myocardial Infarction

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SUMMARY Cardiac muscle death caused by coronary artery occlusion is a dynamic process that often takes hours or days. Emergency revascularization (saphenous vein bypass graft [SVBG]) during acute myocardial infarction (MI) can interrupt myocardial necrosis, salvage ischemic myocardium and revascularize vessels with obstructive lesions not involved in the MI. In this report we describe a preliminary experimental study of 75 patients in which emergency SVBG was the therapy for acute MI. Group 1, 16 patients, required vasoactive medications and/or intraaortic balloon pumping to maintain their blood pressure preoperatively. There was one operative death and two late deaths. Group 2 consisted of 59 hemodynamically stable patients. There were no deaths. The average preop CPK in group 1 was 892 vs 504 in group 2 (p > 0.05). Surgical techniques were routine. The average time from the onset of chest pain that continued to surgery was 6.5 hours. Forty patients were restudied. Post-vs presurgical hemodynamics revealed ejection fraction increased by 34% (p > 0.05), left ventricular end-diastolic pressure reduced by 40% (p > 0.01). End-systolic and end-diastolic volume reduced by 30% (p > 0.05), and 15% (p > 0.01), and stroke volume improved 25% (p > 0.05). Operative mortality was 1.3% and late mortality 2.8%. These results suggest that cautious continued trial of emergency SVBG in patients with evolving MI is warranted.

PROGRESSIVE cardiac muscle death resulting from coronary artery occlusion is a dynamic process that may take hours or even days. Pharmacological and mechanical intervention can favorably alter acute injury during myocardial infarction (MI).1-7 Reports indicate that improved electrical and mechanical function, with eventual limitation of myocardial necrosis, can result when surgical reperfusion is accomplished during the early phases of MI.8-12 Potential benefits of emergency revascularization during acute MI are: interruption of progressive myocardial necrosis, salvage of ischemic but viable myocardium, and revascularization of vessels with significant obstructive lesions that are not involved in the production of the acute infarction.

In this preliminary report we describe a 3-year investigative experience with 75 patients in which emergency myocardial revascularization was the selected form of therapy for acute MI.

Materials and Methods

From June 1975 through September 1978, 75 selected patients underwent emergency myocardial revascularization as the treatment of choice for acute evolving MI at Mercy Hospital, Des Moines, Iowa. All patients gave informed consent to this preliminary and experimental study.

Criteria for patient selection included: early referral to the involved physicians after onset of symptoms; documentation of evolving MI by clinical, electrocardiography, and enzymatic findings; and absence of significant chronic illnesses. All patients underwent an initial cardiologic evaluation that included coronary angiography, ventriculography and appropriate hemodynamic measurements.

During the invasive cardiologic studies, a cardiac operating room was prepared for immediate surgery. The patients were transported directly from the catheterization laboratory to the operating room.

Standard techniques for cardiopulmonary bypass included whole-body hypothermia (28-34°C), a bubble oxygenator,* and pump flow rates of 1.8-2.4 l/min. The left ventricle was not vented;14 and all coronary arterial anastomoses were performed during a single period of aortic cross-clamp. Thrombectomy of the artery perfusing the evolving MI was attempted in all cases. Myocardial protection was provided by maintaining myocardial temperature between 18-22°C by thermistor monitoring, by injecting cold cardioplegic solution into the coronary arteries via the aortic root.

Results

Clinical Findings (table 1)

Sixty-six men and nine women were studied. Average age was 51 years (range 31-76 years) and all had typical symptoms of acute MI. They had severe, unrelenting chest pain and diaphoresis. Group 1, the unstable MI group (16 patients), included 14 who were hypotensive and required vasoactive medications to maintain hemodynamic stability and two patients who were in frank cardiogenic shock that required intraaortic balloon pumping and catecholamines.15, 16 Group 2, the stable MI group, comprised 59 patients. Five of the patients required DC shock preoperatively for ventricular fibrillation but were otherwise stable.

*Cobe Laboratories, Lakewood, Colorado
Table 1. Patient Status

<table>
<thead>
<tr>
<th>No. of</th>
<th>Avg age</th>
<th>Stable</th>
<th>Anterior wall MI</th>
<th>Posterior wall MI</th>
<th>V-fib</th>
<th>Drug support</th>
<th>IABP</th>
</tr>
</thead>
<tbody>
<tr>
<td>patients</td>
<td>(years)</td>
<td>75</td>
<td>51</td>
<td>59</td>
<td>66</td>
<td>9</td>
<td>5</td>
</tr>
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<table>
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<tr>
<th>Discharge ECG</th>
</tr>
</thead>
<tbody>
<tr>
<td>Transmural MI</td>
</tr>
<tr>
<td>74 patients</td>
</tr>
</tbody>
</table>

Abbreviations: MI = myocardial infarction; V-fib = ventricular fibrillation; IABP = intraaortic balloon pumping.

Predominate ECG findings revealed hyperacute ST-segment changes compatible with 65 acute anterior wall and 10 acute posterior wall MIs (fig. 1). Presurgical enzymatic studies were consistent with early myocardial necrosis. The average preop creatine phosphokinase (CPK) in the 14 group 1 patients with hypotension was 892 IU/l. The two group 1 patients in frank cardiogenic shock, had CPKs greater than 2000 IU/l. In the 59 group 2 patients, the pre-op CPK average was 504 IU/l. (The five patients who required DC shock had an average CPK of 509 IU/l.)

All patients were completely revascularized an average of 6.5 hours from the onset of continuing symptoms (range 3.5–25 hours). At surgery there was observable noncontractile muscle in the area of MI in all cases. A clot was extracted from most MI vessels (fig. 2). These clots were usually associated with an observable ruptured plaque. No patient had any gross evidence of reperfusion injury after revascularization. The average number of grafts performed was 2.4 (range 1–6). Average cardiopulmonary bypass time was 76 minutes (range 33–179 minutes).

Postoperative management included all precautions taken for the patient having elective revascularization with one addition: bed rest was continued until the myocardial fraction of creatine phosphokinase (CKMB) became negative. No untoward arrhythmias were noted. Average hospital stay was 16 days. Discharge ECGs revealed residual transmural MIs in 39 patients, subendocardial MIs in 22 patients, nonspecific changes in eight patients, and normal ECGs in 6 patients (fig. 3). There was one hospital death — a patient in cardiogenic shock who was 16 hours into MI with a CPK greater than 3000. He required large volumes of catecholamines and intraaortic balloon pumping before catheterization and surgery. He could not be weaned from cardiopulmonary bypass. There were two sudden late deaths. Both were group 1 patients who had preoperative CPKs greater than 2000. One patient died 3 months after discharge, and
FIGURE 2. (left) Left and right anterior oblique views of filling defect in left anterior descending coronary artery (LAD). (right) Clot removed from LAD and postoperative patent graft and artery.

FIGURE 3. ECG of same patient as in figure 1, 3 days postoperatively.
Table 2. CPK and Patient Mortality

<table>
<thead>
<tr>
<th></th>
<th>CPK Preoperatively</th>
<th>Mortality</th>
</tr>
</thead>
<tbody>
<tr>
<td>Group 1—</td>
<td>2000 - Cardiogenic shock (2)</td>
<td>1 early (18%)</td>
</tr>
<tr>
<td>16 patients</td>
<td>802 - hypotensive (14)</td>
<td>2 late</td>
</tr>
<tr>
<td>Group 2—</td>
<td>504 (34)</td>
<td>(0%)</td>
</tr>
<tr>
<td>59 patients</td>
<td>509 (V-fib) 5</td>
<td></td>
</tr>
<tr>
<td>p—group 1 vs group 2</td>
<td>0.05</td>
<td>0.001</td>
</tr>
</tbody>
</table>

Abbreviation: V-fib = ventricular fibrillation.

postmortem examination revealed closure by fresh clot of the vein graft to the left anterior descending coronary artery. The second patient died unexpectedly 5 months postoperatively, and autopsy revealed that all four grafts were patent.

The early and late mortality rates were 18% for group 1 vs 0% for group 2. (p > 0.001). The total mortality rate for both groups was 1.3% early and 2.7% late. Immediate preoperative CPK in group 1 exclusive of two cardiogenic shock patients was 892 IU/l compared with 504 IU/l for group 2. (p > 0.05) (table 2)

After discharge, patients were followed via scheduled office visits. Forty patients underwent repeat cardiac catheterizations an average of 2 weeks after surgery. Seventy-two patients are alive and well, an average of 18 months (range 1–38 months) after surgery.

Catheterization Data

All patients underwent uncomplicated coronary angiography, ventriculography and hemodynamic measurements. Angiographic studies revealed an occluded or narrowed coronary artery and cardiac wall motion abnormality in its distribution, as well as diseased coronary arteries in areas other than the MI. Forty patients were restudied to date. Postoperative ventriculography revealed residual loss of localized wall motion in the area of MI if the preoperative study revealed a nonmoving, heavily trabeculated configuration in that area. All SVBG-to-MI vessels (40) were patent, and 59 of 60 SVBG-to-non-MI but diseased vessels were patent. The one closed graft was a glutaraldehyde preserved homograft vein that had been placed into a non-MI totally occluded right coronary artery that had been endarterectomized.

Each patient acted as his own control. Pre- and postsurgical hemodynamic studies were statistically analyzed by t test, and are summarized in table 3. Left ventricular end-diastolic pressure was 40% (p > 0.01) lower postoperatively than preoperatively. End-systolic and end-diastolic volumes were reduced by 30% (p > 0.05) and 15% (p > 0.1), respectively, after surgery. Postoperative stroke volume was 25% (p > 0.05) higher than preoperative values. Overall improvement in ejection fraction was 34% (p > 0.05).

Discussion

Any therapeutic measure which would result in the immediate interruption of an evolving MI and reduce or eliminate the ischemic areas associated with an infarction would represent an important contribution in the clinical management of myocardial necrosis.18, 16, 18-20

Although cardiac arrhythmias remain the most frequent cause of death after MI, the loss of functional myocardium is also a significant cause of morbidity and mortality.18, 19, 21 Histochemical studies have shown that occlusion of a coronary artery results in an ischemic area of the myocardium with central infarction interspersed by tissue with borderline perfusion.22

Although it is impossible to quantitate accurately the amount of ischemic myocardium available for salvage in man, several factors affecting infarct size are known, including: 1) duration of ischemia; 2) size of the vessel obstructed; 3) degree of obstruction; and 4) the adequacy of collateral channels.

If one or more of the factors responsible for progressive myocardial necrosis could be altered by a therapeutic intervention, clinical improvement should result. Appropriately timed and planned myocardial revascularization, combined with favorable medical management, has the potential of favorably altering some or all of the factors affecting infarct size. Successful revascularization should eliminate ischemia, improve circulation and bypass non-obstructed vessels. Most experimental studies on reperfusion of acute MI conclude that an experimental MI must be reperfused in 6 hours or less to salvage myocardium.19, 21 Most of these studies have been carried out in animals with "normal" coronary circulation. We feel that it is almost impossible to equate the experimental model to human MI. Human MI usually occurs in a heart that has chronic ischemic changes. This means that collaterals usually are pres-

Table 3. Hemodynamics Before and After Surgery

<table>
<thead>
<tr>
<th>Study</th>
<th>Preoperatively</th>
<th>Postoperatively</th>
<th>% change</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>LVEDP (mm Hg)</td>
<td>20 ± 8</td>
<td>12 ± 4</td>
<td>−40</td>
<td>&lt;0.01</td>
</tr>
<tr>
<td>EF—group 2</td>
<td>0.35 ± 15</td>
<td>0.47 ± 6</td>
<td>+34</td>
<td>&lt;0.05</td>
</tr>
<tr>
<td>ESV (ml)</td>
<td>142 ± 43</td>
<td>99 ± 17</td>
<td>−30</td>
<td>&lt;0.05</td>
</tr>
<tr>
<td>EDV (ml)</td>
<td>218 ± 63</td>
<td>187 ± 71</td>
<td>−15</td>
<td>&lt;0.1</td>
</tr>
<tr>
<td>SV (ml)</td>
<td>76 ± 19</td>
<td>95 ± 15</td>
<td>+25</td>
<td>&lt;0.05</td>
</tr>
</tbody>
</table>

Abbreviations: LVEDP = left ventricular end-diastolic pressure; EF = ejection fraction; ESV = end-systolic volume; EDV = end-diastolic volume; SV = stroke volume.
ent and that the myocardium can tolerate ischemia better than the normal myocardium. We feel that the best predictor of the efficacy of surgery, as indicated by this study, is not the time duration of the infarct, but rather how much myocardial damage has occurred.

In this study the only guide we had to indicate the extent of myocardial damage was the level of CPK and cardiac isoenzymes and the appearance of wall motion abnormalities. Our data indicate that group I patients with CPKs close to 900 IU/l did less well than group 2. We also observed that patients who on preoperative ventriculography had a nonmoving but smooth-looking wall segment, usually recovered motion in that area postoperatively. If that same akinetic area looked "trabeculated" or "irregular," then motion to that segment did not return. Thus, if a patient is having chest pain and 1) the CPK has not risen immoderately (1000 IU/l or 5 X normal); and 2) a large area of nonmoving, "heavily trabeculated" appearing myocardium is not present in the ventriculogram, then there remains salvageable myocardium (table 4).

Many reports suggest that appropriately applied medical therapy can reduce the size of an MI.1-7, 12, 18-22 This then suggests that salvageable myocardium remains in an "infarct" and that properly applied direct revascularization should also be beneficial, especially combined with medical therapy. Our clinical experience with surgical therapy in 75 patients with acute MIs is favorable. None of the patients had observable detrimental changes due to surgical intervention. Overall operative mortality was 1.4%. There were two late deaths in the entire series (2.8%). In a subgroup of group 1, 16 patients with unstable MI, all early and late deaths occurred in this higher-risk group (three of 16, 18%). Early mortality rate in group 1 was 4.8% and late was 1.3%. The 59 patients in group 2 (stable MI) had no early or late deaths, with follow-up for both groups an average of 18 months (1-38 months).

In the subgroup, the MI process was interrupted by revascularization that allowed for potential protection of ischemic myocardium still at risk. We obviously cannot know how extensive the infarct would have been had surgery not been performed. That the patients were experiencing pain at the time of surgery indicates that the infarct may still have been progressing and that we probably were not operating on small, completed infarcts. Some investigators have argued against surgical intervention for acute MI because of inordinately high surgical mortality rates. Most deaths in those studies occurred in patients that had already completed their MIs, and revascularization created reperfusion injuries.

Table 4. Best Prognosticators for Surgical Treatment

| 1. CPK less than 5 X hospital normal |
| 2. Absence of heavy, akinetic trabeculations on ventriculogram |
| 3. Continued chest pain |

Though not randomized and highly investigative, our study indicates that properly timed surgery — that is, before extensive myocardial necrosis has occurred — can be done safely with gratifying short- and long-term results.

We conclude that continued cautious investigative clinical trials of emergency revascularization in a highly selected group of patients with evolving MI is warranted.

References


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