Initial Experience with Transluminal Recanalization of the Recently Occluded Infarct-Related Coronary Artery in Acute Myocardial Infarction – Comparison with Conventionally Treated Patients

K. P. Rentrop, M.D., H. Blanke, M.D., K. R. Karsch, M.D., H. Kreuzer, M.D.

Department of Internal Medicine, Division of Cardiology, University of Goettingen, Goettingen, W. Germany

Summary: In 7 patients, the recently occluded infarct-related vessel was recanalized by transluminal catheter techniques during acute myocardial infarction (Group A). 4 patients had single-vessel disease, 2 patients two-vessels disease and one, involvement of three vessels. Control angiography was performed in 6 patients, 8 days to 7 months later. Changes of coronary artery anatomy and left ventricular function were compared with a group of 9 conventionally treated patients, who were found to have occlusion of the infarct-related vessel in the acute stage (Group B). Five Group B patients had one-vessel disease, 3 patients two-vessel disease and 1 patient, involvement of all three vessels. In the chronic stage, all transluminally recanalized vessels were found to be patent in Group A. There was spontaneous recanalization of the infarct vessel in 4 of 9 Group B patients. In Group A, the length of the akinetic segment (AKS) decreased significantly ($p < 0.05$) from $145.4 \pm 48.5 \text{ mm}$ to $73.2 \pm 73.4 \text{ mm}$ (mean $\pm$ SD). Volume parameters did not change significantly. In Group B, length of the AKS did not change significantly, EDVI increased significantly from $81.1 \pm 19.8$ to $106.8 \pm 40.6 \text{ ml/m}^2$ ($p < 0.05$); ESVI increased significantly from $41.7 \pm 13.7 \text{ ml/m}^2$ to $66.8 \pm 37.9 \text{ ml/m}^2$ ($p < 0.01$). In the acute stage, length of the AKS and volume parameters did not differ significantly between the two groups. In the chronic stage, AKS was significantly shorter (A: $73.2 \pm 63.4 \text{ mm}$; $144.9 \pm 59 \text{ mm}$ ($p < 0.0025$)) and EF was significantly higher (A: $54.6 \pm 11.6\%$; B: $40.9 \pm 14.5\%$ ($p < 0.05$)) in Group A. Peak CPK was lower in Group A (A: $1009 \pm 827 \text{ U/l}$; B: $1324 \pm 655 \text{ U/l}$), but this difference did not achieve statistical significance. Results of this pilot study suggest that transluminal recanalization in the early phases of acute myocardial infarction might result in limitation of myocardial injury. However, further research will be needed to improve the technique and to test its results.

Keywords: transluminal recanalization, acute myocardial infarction, spontaneous recanalization, left ventricular function, akinetic segment, coronary angiography, coronary artery thrombosis, coronary heart disease

Introduction

Transluminal recanalization of a totally occluded coronary artery was first successfully performed with a guide wire in a patient with impending infarction due to a catheter-related complication (46). The total occlusion in this patient was probably brought on by thromboembolic material, dislodged from the catheter into the preexisting high-degree stenosis. A parallel could be drawn between this case and noncatheter-induced, spontaneous infarction, since most autopic studies seem to indicate acute thrombotic occlusion of a high-degree lesion as the cause of myocardial infarction in the majority of patients (6, 9, 11, 14). For this reason, it would seem plausible to try to limit myocardial injury in the early stages of spontaneous, evolving infarction by means of transluminal recanalization. However, several problems with this approach become evident at the present time:
1. Acute coronary angiography, a prerequisite, is still felt to be contraindicated during acute myocardial infarction by some authors (8). However, findings of other authors (3, 26, 31, 37, 38) as well as our own (47), seem to indicate that acute coronary angiography, if performed properly, does not increase mortality or cause deterioration of hemodynamics.

2. To the present time, no standard equipment for transluminal recanalization is available. Danger of inflicting injury on the coronary artery by intraluminal manipulation with a stiff guide wire is obvious. A compromise needed to be found between the degree of stiffness required to perforate fresh thrombotic material, and degree of flexibility required to decrease danger of injury to the coronary intima. In animal experiments, various wires and catheter materials were tested (Cook Company) until a practicable compromise seemed to have been found.

In this paper, the results of the first pilot study of transluminal recanalization in patients with acute infarction will be presented and compared with data obtained in a conventionally treated group.

Material and Methods

Patients

Informed consent was obtained from all patients. Conventional therapy was discussed with the patients, as well as risks and potential benefits of acute angiography and possible subsequent measures such as intraaortic balloon pumping (IABP), aortocoronary bypass surgery, and in a subgroup, transluminal recanalization. The patients were taken immediately, thereafter, to the cardiac catheterization laboratory, and the surgical team was alerted for a possible emergency procedure.

Table I Clinical data of 7 patients in whom transluminal recanalization was performed during acute myocardial infarction and of 9 conventionally treated control patients. For further clinical details on patients A1 and A5, see (46, 39).

<table>
<thead>
<tr>
<th>pt. No.</th>
<th>sex</th>
<th>age</th>
<th>beg. of symptoms before angio (h)</th>
<th>ECG</th>
<th>CPK (U/l) start</th>
<th>max</th>
<th>IABP (h)</th>
<th>acute complications</th>
<th>diagnosis</th>
<th>date of angiography</th>
<th>date of angio chronic</th>
</tr>
</thead>
<tbody>
<tr>
<td>Group A</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>1*</td>
<td>♂</td>
<td>45</td>
<td></td>
<td></td>
<td>35</td>
<td>359</td>
<td></td>
<td>asystole</td>
<td>EMI</td>
<td>6/13/78</td>
<td>6/21/78</td>
</tr>
<tr>
<td>2</td>
<td>♂</td>
<td>31</td>
<td>24</td>
<td></td>
<td>1106</td>
<td>1106</td>
<td>37</td>
<td>ventricular fibrillation</td>
<td>EMI</td>
<td>7/3/78</td>
<td>7/27/78</td>
</tr>
<tr>
<td>3</td>
<td>♂</td>
<td>39</td>
<td>2</td>
<td></td>
<td>97</td>
<td>1258</td>
<td>24</td>
<td></td>
<td>EMI</td>
<td>7/12/78</td>
<td>1/10/79</td>
</tr>
<tr>
<td>4</td>
<td>♂</td>
<td>65</td>
<td>11</td>
<td></td>
<td>1900</td>
<td>2700</td>
<td>56</td>
<td></td>
<td>EMI</td>
<td>9/4/78</td>
<td>11/13/78</td>
</tr>
<tr>
<td>5*</td>
<td>♂</td>
<td>60</td>
<td>4</td>
<td></td>
<td>22</td>
<td>280</td>
<td></td>
<td></td>
<td>EMI</td>
<td>10/9/78</td>
<td>10/16/78</td>
</tr>
<tr>
<td>6</td>
<td>♂</td>
<td>59</td>
<td>3</td>
<td></td>
<td>56</td>
<td>716</td>
<td></td>
<td></td>
<td>EMI</td>
<td>10/22/78</td>
<td></td>
</tr>
<tr>
<td>7</td>
<td>♂</td>
<td>58</td>
<td>11</td>
<td></td>
<td>433</td>
<td>644</td>
<td></td>
<td>ventricular fibrillation</td>
<td>EMI</td>
<td>12/3/78</td>
<td>1/3/79</td>
</tr>
<tr>
<td>Group B</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>1</td>
<td>♂</td>
<td>57</td>
<td>10</td>
<td></td>
<td>54</td>
<td>581</td>
<td>42</td>
<td>saddle-embolism 5th day</td>
<td>imp.shock</td>
<td>5/3/78</td>
<td>6/15/78</td>
</tr>
<tr>
<td>2</td>
<td>♂</td>
<td>38</td>
<td>21</td>
<td></td>
<td>1598</td>
<td>1814</td>
<td>130</td>
<td></td>
<td>EMI</td>
<td>6/27/78</td>
<td>8/14/78</td>
</tr>
<tr>
<td>3</td>
<td>♂</td>
<td>53</td>
<td>10</td>
<td></td>
<td>50</td>
<td>461</td>
<td>28</td>
<td></td>
<td>EMI</td>
<td>7/28/78</td>
<td>8/14/78</td>
</tr>
<tr>
<td>4</td>
<td>♂</td>
<td>54</td>
<td>25</td>
<td></td>
<td>50</td>
<td>1380</td>
<td>48</td>
<td></td>
<td>EMI</td>
<td>8/12/78</td>
<td>11/15/78</td>
</tr>
<tr>
<td>5</td>
<td>♂</td>
<td>55</td>
<td>7</td>
<td></td>
<td>140</td>
<td>660</td>
<td>20</td>
<td>ventricular fibrillation</td>
<td>imp.shock</td>
<td>9/14/78</td>
<td>11/20/78</td>
</tr>
<tr>
<td>6</td>
<td>♂</td>
<td>62</td>
<td>3</td>
<td></td>
<td>160</td>
<td>2350</td>
<td>130</td>
<td></td>
<td>EMI</td>
<td>9/14/78</td>
<td>11/10/78</td>
</tr>
<tr>
<td>7</td>
<td>♂</td>
<td>54</td>
<td>1</td>
<td></td>
<td>60</td>
<td>1160</td>
<td></td>
<td>ventricular tachycardia</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>8</td>
<td>♂</td>
<td>54</td>
<td>2</td>
<td></td>
<td>165</td>
<td>1710</td>
<td></td>
<td>AV-block II</td>
<td>EMI</td>
<td>11/23/78</td>
<td>1/6/79</td>
</tr>
<tr>
<td>9</td>
<td>♂</td>
<td>52</td>
<td>2</td>
<td></td>
<td>424</td>
<td>1738</td>
<td></td>
<td></td>
<td>EMI</td>
<td>12/14/78</td>
<td>1/18/79</td>
</tr>
</tbody>
</table>

pt. No. = patient number; CPK = creatine phosphokinase; start = CPK immediately prior to angiography; max = peak value after angiography; IABP (h) = duration of intraaortic counterpulsation in hours; ST † = ST segment elevation; RBBB = right bundle branch block; EMI = evolving myocardial infarction; imp. shock = impending shock.
Transluminal recanalization of a totally occluded infarct vessel has been attempted in thirteen patients since 6/13/78. The occlusion was localized in the proximal segment of a major coronary artery in all patients. In seven of these patients, it was possible to reach, and pass, the occlusion. These seven patients are presented in this study as Group A. There were 6 males and 1 female. Average age was 51.0 ± 12.7 years (Table I). Onset of symptoms was 7.8 ± 8.3 h prior to angiography (Table I). In 5 patients the diagnosis was hemodynamically stable, evolving infarction, in 2 patients impending shock (Table I). The first patient of this group (A 1) sustained his total occlusion on the catheter table; the others were admitted to the hospital because of “spontaneous” infarction. The ECG, taken immediately prior to catheterization, showed signs of acute infarction in all patients except for the one in whom occlusion of the right coronary artery (RCA) occurred during angiography (Table I). In 6 patients, control angiography could be performed after 8 days to 5 months. In only one of them (A 1) aortocoronary bypass surgery was performed between acute and control angiography.

Group B is made up of the 9 patients in our total patient population, studied during acute infarction, who fulfilled the following criteria:

1. Proximal total occlusion of a major vessel as cause of the acute infarction,
2. no history of previous infarction,
3. no transluminal recanalization,
4. control angiography after four weeks to seven months,
5. no heart surgery between acute and control angiogram.

There were 7 males and 2 females. Average age was 53.2 ± 6.4 years (Table I). Onset of symptoms was 6.5 ± 6.5 h prior to angiography (Table I). Five of these 9 patients, 4 with evolving infarction, one with impending shock, were studied between April 9, 1978, and August 12, 1978, at a time when equipment for transluminal recanalization was still unavailable or being tested. Four patients, 3 with evolving infarction, one with impending shock, were studied since September 1978. In these patients, transluminal recanalization was not attempted, since there seemed to be unfavorable anatomical conditions, such as localization of the orifice of the coronary artery, making placement of a catheter difficult, or localization of the total occlusion in or beyond a tortuosity of the vessel increasing the danger of perforating the intima.

The ECG showed signs of acute infarction in all patients of Group B prior to angiography.

Coronary Angiographic Studies

Coronary angiography was performed by the same technique in both groups. In addition to the catheterization team, there was one cardiologist exclusively responsible for the monitoring and treatment of the patient before, during, and after catheterization. Cineangiography (35 mm film, 75 frames/s for ventriculography, 25 frames/s for coronary angiography) was performed by the Sones technique (52). Biplane left ventriculography was performed by injection of 20 to 40 ml of Urographin 76% (Schering), with a flow of 8 to 10 ml/s. In 2 patients (B 2, B 4) IABP was begun before ventriculography (Table I); in 7 patients, the balloon was inserted before ventriculography, and counterpulsation was started immediately after ventriculography and prior to coronary angiography (Table I). In one Group B patient (B 6) counterpulsation was begun after coronary angiography (Table I).

Both coronary arteries were visualized selectively by injection of 8–10 ml of Urographin 76% in at least two projections. In order to minimize the amount of contrast media used in patients studied without IABP, the number of coronary injections was decreased by biplane filming.

Re-angiography in the chronic stage was begun with biplane left ventriculography. Subsequently, both coronary arteries were visualized in at least two projections.

Transluminal Recanalization

There is no standard technique for transluminal recanalization of totally occluded coronary arteries as of yet. Three different approaches were used:

1. A Sones or Shirey end-hole catheter (USCI) was advanced subselectively into the infarct-related vessel immediately to the site of total occlusion (Fig. 1a). A straight, or slightly curved 0.32" to 0.38" guide wire (Cook), with the movable core pulled back by 5 cm – 10 cm, was advanced 1 cm – 2 cm through the occlusion (Fig. 1b). The guide wire was withdrawn and the result of the recanalization was controlled by subselective injection of contrast media (Fig. 1c). Only in patient A 1 was the guide wire stiffened (46). In patient A 5 who had a very proximal total occlusion of the RCA, the Sones catheter slipped through the occlusion, inadvertently, before the guide wire had been advanced (39). This case (A 5), however, must not be viewed as an example of technique No. 1, since it is our intention to avoid blind perforation with large diameter instruments.

2. A preshaped guiding catheter (Cook) with an internal diameter of 1.9 mm was placed transfemorally or transbrachially into the ostium of the occluded coronary artery. The soft end of a straight, or slightly curved, 0.32" to 0.38" movable core guide wire was advanced subselectively to the site of the occlusion. The position of the guide wire could be controlled by injection of contrast media through the large lumen of the guiding catheter, around the guide wire. During advancement in the coronary artery, the movable core of the guide wire was withdrawn at least 5–10 cm. If the obstruction could be reached without problems, the soft tip of the guide wire was advanced beyond the obstruction (Fig. 2). The movable core retracted throughout the procedure. If problems arose negotiating tortuosities of the coronary artery with the guide wire, the procedure was immediately discontinued after a final control injection.
Fig. 1 a-f Transluminal recanalization in patient A5. Left coronary artery in LAO half axis projection. a) Subselective injection into the totally occluded LAD via a Shiray end-hole catheter; b) advancement of a special 0.38" guide wire beyond the total occlusion; c) visualization of the distal LAD after transluminal recanalization; d) control angiogram of the left coronary artery after 5 months. e) left ventricle in endystole in the RAO projection during the acute stage of infarction; f) left ventricle in endsystole, RAO projection, after 5 months.
3. A preshaped guiding catheter (Cook; Schneider Medintag) with an internal diameter of 1.9 mm was placed transbrachially or transfemorally into the ostium of the occluded vessel (Fig. 3). An end-hole recanalization catheter (Cook) with long taper and an external diameter of 0.7 to 1 mm at the tip and 1.7 mm at the shaft, was advanced through the guiding catheter subselectively into the occluded vessel. A 0.18" guide-wire (Cook) could be passed through the recanalization catheter. The recanalization catheter was attached to a pressure transducer. Pressures

Fig. 2 a

Fig. 2 b

Fig. 2 c

Fig. 2 a–c Transluminal recanalization in patient A7. a) Total occlusion of the LAD, RAO projection in acute infarction; b) Guide wire advanced through the total occlusion into the distal lumen of the LAD via a guiding catheter; c) control angiography of the left coronary artery, RAO projection after 1 month.

Fig. 3 a

Fig. 3 b

Fig. 3 Tranluminal recanalization in patient A6. a) A special guiding catheter is placed into the ostium of the totally occluded RCA. A long taper recanalization catheter is advanced beyond the total occlusion to the bifurcation, b) contrast media is injected in the distal RCA. A filling defect, attached to the site of the total occlusion, which represents a distal thrombus, is seen surrounded by contrast media.
were continuously recorded during advancement of this catheter, in order to assure correct placement of the tip in the lumen of the vessel. The exact position of the catheter could be controlled by small injections of contrast media. If the occlusion could be reached without any technical difficulties, the catheter was advanced by about 1.5 additional centimeters beyond the occlusion, and a small amount of contrast media was injected in order to assure correct positioning of the tip of the catheter in the distal lumen of the occluded vessel. Subsequently, the catheter was advanced far into the occluded vessel (Fig. 3) in order to dilate the occlusion. The result of the recanalization was controlled by selective injection of contrast media through the guiding catheter after pull-back of the recanalization catheter.

Medication

Intravenous nitroglycerin (NTG) was started at a dose of 1.5–6 mg/h prior to angiography in all patients except for one (B 6), who was hemodynamically unstable and treated with IABP. The NTG infusion was continued throughout the study and in the intensive care unit, until normalization of CPK, after which a maintenance therapy of isosorbide dinitrate (ISDN), 40–80 mg per day orally, was started (Table II). All patients received a bolus of heparin 3500–10000 U, i.a., at the beginning of angiography. In those patients who were treated by counterpulsation, systemic heparinization was maintained until removal of the balloon (Table II). The other patients received low-dose heparin subcutaneously (s.c.) for the first

<table>
<thead>
<tr>
<th>Table II</th>
<th>Medication in 7 patients in whom transluminal recanalization was performed during the acute stage of infarction (A) and in 9 conventionally treated infarct patients (B).</th>
</tr>
</thead>
<tbody>
<tr>
<td>pt. No.</td>
<td>parent. NTG (mg/h)</td>
</tr>
<tr>
<td></td>
<td>angio</td>
</tr>
<tr>
<td><strong>Group A</strong></td>
<td></td>
</tr>
<tr>
<td>1</td>
<td>3.0</td>
</tr>
<tr>
<td>2</td>
<td>6.0</td>
</tr>
<tr>
<td>3</td>
<td>3.0–6.0</td>
</tr>
<tr>
<td>4</td>
<td>1.5</td>
</tr>
<tr>
<td>5</td>
<td>3.0</td>
</tr>
<tr>
<td>6</td>
<td>1.5–3.0</td>
</tr>
<tr>
<td>7</td>
<td>4.5</td>
</tr>
</tbody>
</table>

| **Group B** | | | | | | | | | | |
| 1 | 1.5–4.5 | 3.0–6.0 | 10000 | 1000 | 3x5000 | 180 days | | 4th day | 4th day | 1st day | 15 |
| 2 | 4.5–12 | 3.0–9.0 | 10000 | 1000 | 3x5000 | 24 days | | 4th day | 4th day | 2nd day | 15 |
| 3 | 1.5 | 1.5 | 10000 | 1000 | 3x5000 | 21 days | | 4th day | 4th day | 15 |
| 4 | 3.0–6.0 | 6.0 | 10000 | 1000 | 3x8000 | 40 | | 4th day | 4th day | 15 |
| 5 | 3.0–6.0 | 4.5–6.0 | 10000 | 1000 | 3x5000 | permanent | | 70 | 45 |
| 6 | 1.5 | 3.0–6.0 | 3500 | 1000 | 3x5000 | permanent | | 4th day | 4th day | 15 |
| 7 | 4.5 | 4.5–6.0 | 3500 | 18 days | 3x8000 | permanent | | 50 | 15 |
| 8 | 4.5 | 4.5–6.0 | 3500 | 3x6000 | 3x6000 | permanent | | 70 | 45 |
| 9 | 1.5 | 1.5 | 3500 | 1000 | 3x5000 | 42 days | | 4th day | 4th day | 7.5 |

Parent. NTG = parenteral nitroglycerin; ISDN = isosorbide dinitrate; ICU = intensive care unit.
few days, and subsequently coumadin, during the period of ambulation. Anticoagulation was discontinued at the time of hospital discharge in 3 patients of Group A and in 3 patients of Group B (Table II).

In all patients except one (B 4) in Group B, long-term therapy with a beta-blocker (pindolol) was started at varying intervals after acute angiography (Table II). At the time of control angiography, all patients were taking ISDN, and all but one (B 4) a beta-blocker. After ventriculography and before coronary angiography, one capsule of NTG was administered sublingually.

Calculations

Severity of coronary artery lesions was assessed by standard criteria (19). The presence of collaterals to the infarct area was noted, without an attempt of quantification. Volumes and local wall function were determined from biplane left ventriculograms. Only a sinus beat not immediately preceded by an extrasystole was evaluated. Abnormalities of local wall function were categorized according to the criteria of Herman et al (24). Length of the akinetic segment (AKS) was calculated in mm. Left ventricular enddiastolic (EDS) and endsystolic volumes (ESV) were calculated using the area length method of Dodge et al (13). From the angiographically determined volumes, stroke volume (SV = EDV - ESV) and ejection fraction (EF = SV/EDV) were derived.

Aortic and left ventricular pressures were recorded via the fluid-filled catheter with a Statham P 23 DB manometer. All pressures were obtained during medication as indicated above. In those patients, who were treated with the intraaortic balloon, pressures were obtained prior to counterc pulsation, with the exception of patient B 4.

The diagnosis of a myocardial infarct was based on the criteria of the World Health Organization (59, 60). Blood for serial determination of CPK (54) was obtained every 70 min until normal values (< 70 U/l) were reached.

Data presented are mean values ± standard deviation. Statistical analysis was carried out by means of the paired t-test, the Wilcoxon test or Student's t-test for non-paired data. P values above 0.05 were considered not significant (N. S.).

Results

Coronary Angiographic Findings

Acute Angiography. In 4 patients of Group A, the LAD was the totally occluded infarct vessel, the RCA in the other 3 patients. In these 3 patients, the RCA was dominant (Table III).

In 7 patients of Group B, the LAD was the totally occluded infarct-related vessel, the dominant RCA and the dominant left circumflex, in one patient each.

There were collaterals to the infarct area in 4 patients of Group A and in 6 patients of Group B (Table III).

Four patients of Group A had single-vessel disease, 2 patients 2-vessel disease, and 1 patient, involvement of all 3 coronary arteries (Table III). In Group B, single-vessel disease was found in 5 patients, 2-vessel disease in 3 patients, and 3-vessel disease in 1 patient.

Immediate Angiographic Results of Transluminal Recanalization. Immediately after recanalization, there was approximately 90% narrowing at the site of the previous total occlusion in 6 patients of Group A. In 1 patient (A 5), there was an 80% lesion at the site of previous obstruction.

The quality of distal filling varied widely. In some patients the distal segments could be visualized only by selective injection of contrast media into the infarct vessel. In others, injection into the ostium of the coronary artery resulted in good distal filling. In patients A 5 and A 6 a distal filling defect was seen, suggestive of a thrombus (Fig. 3). In patient A 5 the filling defect was far away from the lesion, in the distal right coronary artery, at the site to which the tip of the Sones catheter had been advanced. In patient A 6, the segments of the right coronary artery beyond the total occlusion were visualized by injection of contrast media through the recanalization catheter. A constant filling defect of about one and a half centimeters in length, surrounded by contrast media, was seen attached to the total occlusion (Fig. 3).

Changes of Coronary Angiographic Findings in the Chronic Stage. In all 6 patients of Group A, who could be restudied, there was prompt filling of the distal segment of the previously totally occluded infarct vessel (Table III). In patient 1, prompt filling of the distal segments was also achieved through the saphenous vein graft, which had been constructed during the acute stage, after transluminal recanalization. In patients 3, 4, 5 and 7 of Group A, the lumen at the site of the previous obstruction was clearly larger in the chronic stage than immediately after recanalization (Fig. 1).

In the acute stage, 3 of the restudied patients showed collaterals to the infarct area; in the chronic stage no collaterals were seen in these patients (Table III).

In Group B, 4 of the 9 previously totally occluded vessels showed antegrade filling of the distal segments. The lesions seen at the site of the previous total obstruction were between 80% and 95% (Table III). In one of the 4 patients with spontaneous recanalization, collaterals to the infarct area disappeared in the chronic stage. Two patients, who did not recanalize the total obstruction, developed new collaterals, which were not present in the acute stage (B 4, B 8).

Non-infarct-related vessels did not show any major changes in either of the groups.
Table III  Coronary angiographic and left ventriculographic findings in the acute and chronic stage of myocardial infarction in (A) 7 patients in whom transluminal recanalization was performed during the acute stage and (B) in 9 conventionally treated patients.

<table>
<thead>
<tr>
<th>pt. No.</th>
<th>IRV</th>
<th>coll</th>
<th>vessels</th>
<th>al</th>
<th>ap</th>
<th>inf</th>
<th>sept</th>
<th>post</th>
<th>lat</th>
<th>PLV/ PLVED mmHG</th>
</tr>
</thead>
<tbody>
<tr>
<td>A</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>1</td>
<td>RCA 100</td>
<td>+</td>
<td>LAD 60-70</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>2</td>
<td>LAD 100</td>
<td>+</td>
<td>H-A</td>
<td>D</td>
<td>n</td>
<td>A</td>
<td>n</td>
<td>105/22</td>
<td></td>
<td></td>
</tr>
<tr>
<td>3</td>
<td>LAD 100</td>
<td>+</td>
<td>D</td>
<td>D</td>
<td>n</td>
<td>D</td>
<td>n</td>
<td>110/10</td>
<td></td>
<td></td>
</tr>
<tr>
<td>4</td>
<td>LAD 100</td>
<td>+</td>
<td>CX 80</td>
<td>A</td>
<td>D</td>
<td>n</td>
<td>A</td>
<td>110/30</td>
<td></td>
<td></td>
</tr>
<tr>
<td>5</td>
<td>RCA 100</td>
<td>+</td>
<td>LAD 80</td>
<td>n</td>
<td>n</td>
<td>A</td>
<td>n</td>
<td>110/13</td>
<td></td>
<td></td>
</tr>
<tr>
<td>6</td>
<td>RCA 100</td>
<td>+</td>
<td>LAD 80</td>
<td>n</td>
<td>H</td>
<td>A</td>
<td>n</td>
<td>H-A</td>
<td>110/15</td>
<td></td>
</tr>
<tr>
<td>7</td>
<td>LAD 100</td>
<td>+</td>
<td>LMCA 40</td>
<td>D</td>
<td>D</td>
<td>n</td>
<td>A</td>
<td>n</td>
<td>120/17</td>
<td></td>
</tr>
<tr>
<td>B</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>1</td>
<td>LAD 100</td>
<td>+</td>
<td>D</td>
<td>D</td>
<td>n</td>
<td>D</td>
<td>n</td>
<td>100/5</td>
<td></td>
<td></td>
</tr>
<tr>
<td>2</td>
<td>CX 100</td>
<td>+</td>
<td>LAD 60</td>
<td>H</td>
<td>H</td>
<td>A</td>
<td>n</td>
<td>A</td>
<td>100/25</td>
<td></td>
</tr>
<tr>
<td>3</td>
<td>LAD 100</td>
<td>+</td>
<td>A</td>
<td>D</td>
<td>n</td>
<td>A</td>
<td>n</td>
<td>160/20</td>
<td></td>
<td></td>
</tr>
<tr>
<td>4</td>
<td>LAD 100</td>
<td>+</td>
<td>RCA 70</td>
<td>H</td>
<td>D</td>
<td>n</td>
<td>115/15</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>5</td>
<td>RCA 100</td>
<td>+</td>
<td>H-A</td>
<td>A</td>
<td>n</td>
<td>n</td>
<td>n</td>
<td>140/5</td>
<td></td>
<td></td>
</tr>
<tr>
<td>6</td>
<td>LAD 100</td>
<td>+</td>
<td>LMCA 50</td>
<td>D</td>
<td>D</td>
<td>H</td>
<td>A</td>
<td>85/23</td>
<td></td>
<td></td>
</tr>
<tr>
<td>7</td>
<td>LAD 100</td>
<td>+</td>
<td>RCA 90</td>
<td>D</td>
<td>D</td>
<td>n</td>
<td>H</td>
<td>95/4</td>
<td></td>
<td></td>
</tr>
<tr>
<td>8</td>
<td>RCA 100</td>
<td>+</td>
<td>LAD 40</td>
<td>A</td>
<td>A</td>
<td>n</td>
<td>A</td>
<td>110/20</td>
<td></td>
<td></td>
</tr>
<tr>
<td>9</td>
<td>RCA 100</td>
<td>+</td>
<td>LAD 40</td>
<td>n</td>
<td>n</td>
<td>A</td>
<td>n</td>
<td>A</td>
<td>130/10</td>
<td></td>
</tr>
</tbody>
</table>

IRV = infarct-related vessel; coll = collaterals to the infarct area; RCA = right coronary artery; LAD = left anterior descending artery; CX = left circumflex artery; al = anterolateral; ap = apical; inf = inferior; sept = septal; post lat = posterolateral segment of the left ventricle; n = normal wall motion; H = hypokinesia; A = akinesia; D = dyskinesia; PLV = systolic left ventricular pressure; PLVED = end-diastolic left ventricular pressure.

**Left Ventricular Function**

*Left Ventricular Function During the Acute Stage.* In all patients, abnormalities of local wall motion were found which corresponded to the localization of the acute infarct in the ECG (Table I and III) and the perfusion area of the totally occluded vessel (Table III).

The length of the akinetic segment was $145.4 \pm 48.5$ mm in Group A and $124.3 \pm 36$ mm in Group B (Fig. 4). EDVI was $88.4 \pm 21.2$ ml/m² in Group A and $81.1 \pm 19.8$ ml/m² in Group B. ESVI was $48.4 \pm 12.1$ ml/m² in Group A and $41.7 \pm 13.7$ ml/m² in Group B. SVI was $40.0 \pm 12.7$ ml/m² in Group A and $39.6 \pm 11.6$ ml/m² in Group B. Ejection fraction (EF) was $45.0 \pm 6.5\%$ in Group A and $48.9 \pm 9.5\%$ in Group B (Fig. 5). None of the differences between the groups was significant.

*Changes of Left Ventricular Function in the Chronic Stage.* In Group A, changes of local and global left ventricular function could be evaluated in only 5 patients, since patient 6 refused restudy, and patient 1 who infarcted on the catheterization table, did not have a repeat ventriculogram after total occlusion of his right coronary artery. The length of the AKS decreased markedly in 3 of the 5 patients (Table I); in one patient it decreased slightly, and in one patient it did not change. The mean value of the AKS decreased significantly in Group A from $145.4 \pm 48.5$ mm to $73.2 \pm 63.4$ mm ($p < 0.05$) (Fig. 4).

In Group B, the AKS was shorter in the chronic stage in 2 patients, remained approximately the same in 1 patient, and lengthened in 6 patients (Fig. 4). The mean value of...
the AKS increased significantly from $124.3 \pm 36.6$ mm to $144.9 \pm 59$ mm. In the chronic stage, the AKS was significantly ($p < 0.0025$) longer in Group B than in Group A (Fig. 4).

In Group A, EDVI did not change significantly in the chronic stage of infarction. In Group B there was a significant rise of EDVI from $81.1 \pm 19.8$ ml/m$^2$ during the acute stage to $106.8 \pm 40.6$ ml/m$^2$ in the chronic stage ($p < 0.05$) (Fig. 5).

ESVI did not change significantly in Group A, whereas in Group B, there was a significant ($p < 0.01$) increase of ESVI from $41.7 \pm 13.7$ ml/m$^2$ in the acute stage to $66.8 \pm 37.3$ ml/m$^2$ in the chronic phase. ESVI remained unchanged in only 2 patients of Group B, patients in whom length of the AKS decreased.

Stroke volume index did not change significantly in either group.

There was a rise of EF in Group A from $45. \pm 6.5\%$ to $54.6 \pm 11.6\%$. However, this increase was not statistically significant. In the medically treated patients EF decreased significantly ($p < 0.05$) from $48.9 \pm 9.5\%$ in the acute stage, to $40.9 \pm 14.5\%$ in the chronic stage (Fig. 5). There was a slight increase of EF in 3 Group B patients only. In 2 of these patients the AKS shortened. Whereas there was no significant difference of EF between Groups A and B during the acute stage, EF was significantly lower in Group B during the chronic stage (Fig. 5).
K. P. Rentrop et al.: Transluminal Recanalization of Coronary Arteries in AMI 101

Fig. 6 Creatine phosphokinase (CPK) immediately before acute coronary angiography (Pre-A) and peak values (max). A, in 7 patients in whom transluminal recanalization was performed during acute infarction; B, in 9 conventionally treated patients.

CPK

CPK increased from the preangiographic value of 521 ± 722 U/l to the peak value of 1009 ± 827 U/l in Group A and from 300 ± 506 U/l to 1324 ± 655 U/l in Group B (Fig. 6).

Complications

There was no early or late mortality. At the time of submission of this publication, all patients of the study were alive and free of major symptoms.

There were no complications in our patients due to transluminal recanalization. However, in animal experiments there were intima perforations whenever tortuosities of a vessel could not be negotiated with ease in the use of all three procedures.

During angiography ventricular fibrillation occurred in 5 patients: in 3 patients of Group A, and in 2 patients of Group B. Defibrillation was immediately successful in all cases.

The pressures at the beginning of acute coronary angiography are summarized in Table I. There was a pathological elevation of left ventricular filling pressure during medication with parenteral NTG in 4 patients of Group A, and in 5 patients of Group B. Injection of contrast media resulted in a transient decrease of systolic pressure and sometimes in a transient increase of left ventricular filling pressure. In no case was there permanent worsening of pressures. On the contrary, in those patients treated with IABP, filling pressures decreased during the study.

All patients, except for patient A 1 who infarcted on the catheterization table, had symptoms typical of myocardial infarction upon hospital admission. Symptomatic improvement was achieved in all patients prior to angiography by a combination of intravenous sedatives (diazepam), infusion of NTG and, if needed, opiates. Sometimes there was a transient worsening of angina pectoris after injection of contrast media, which lasted 5 min at most. In no case was there permanent worsening of symptoms, however, evaluation of angina pectoris in these patients is problematic due to the medication.

Discussion

Complications

In agreement with other authors (3, 26, 28, 30, 36, 37, 38), we found that hospital mortality is not increased and hemodynamics are not worsened by coronary angiography in acute infarction, provided certain precautions are observed (47).

1. In hemodynamically unstable patients, coronary angiography was performed after stabilization with intravenous sedatives, which is probably the consequence of several factors.

2. In hemodynamically stable patients with evolving infarction who were studied without IABP, the amount of contrast media was restricted to a maximum of 0.5–1.0 ml/kg body weight.

3. A preload increase due to contrast media was avoided by intravenous infusion of NTG.

4. Treatment and monitoring, before, during, and after coronary angiography, was maintained at coronary care unit level.

There were no complications due to intracoronary manipulations with guide wires or catheters, which is probably the consequence of several factors.

1. Wires used for intracoronary manipulations had a soft tip.

2. Catheters used for intracoronary manipulations had a long taper with an end-hole for pressure control.

3. If tortuosities of the coronary artery could not be negotiated relatively easily with a slightly curved guide wire or catheter, the procedure was discontinued, since animal experiments had shown a high incidence of intima perforations in these situations.
Spontaneous Recanalization in Medically Treated Patients (Group B)

In 4 of 9 patients of Group B, spontaneous recanalization of the totally occluded infarct vessels occurred. Two mechanisms could explain the spontaneous recanalization of totally occluded vessels in myocardial infarction.

1. Oliva, and Breckinridge (40) and Helfant (22) suggest that spasm superimposed upon high-degree lesions might play a causative role in acute myocardial infarction. However, we did not see any spontaneous recanalization of total occlusions or changes of lesion severity during coronary angiography in patients who received high doses of parenteral NTG.

2. Most pathological findings seem to indicate that in the majority of cases, acute infarction is due to thrombotic obstruction or narrowing of high-degree atherosclerotic lesions (6, 9, 11, 14). In this study the presence of thrombotic material at the site of acute coronary occlusion could be shown by means of coronary angiography in two patients for the first time.

There are pathological findings which indicate that occluding coronary thrombosis can be partially or completely dissolved in 2–16 weeks after infarction (10, 38).

In animal experiments, complete resolution within 30 days of totally, to subtotally, occluding thrombosis was observed (57). In humans, coronary emboli, which occurred as a complication of coronary angiography, were seen to lyse spontaneously within 5½–8 weeks (44). Henderson et al observed progressive regression of an LAD lesion in a patient with anterior wall infarction, by performing serial coronary angiograms in the chronic stage of infarction (23). The changes of the coronary artery morphology from the acute to the chronic stage of infarction have not yet been analyzed by serial angiograms. Our findings seem to substantiate the pathologic–anatomical hypothesis, that there is spontaneous recanalization of a considerable percentage of totally occluded infarct vessels in the healing phase, due to spontaneous lysis of thrombotic material. To which degree this process was influenced by heparinization in our patients cannot be determined.

Transluminal Recanalization

In 7 of 13 patients with acute proximal total occlusion of a major vessel resulting in myocardial infarction, it was possible to reach the site of the lesion with a special guide wire or a thin catheter, and to pass the instrument through the lesion into the distal lumen of the vessel. Control angiography in the chronic stage of infarction possible in 6 of the 7 patients, revealed patency of the recanalized occlusion in all 6 patients. In 5 of them, the lumen at the site of the previous obstruction was clearly larger in the chronic stage than immediately after transluminal recanalization. The most likely interpretation of this observation is that the mechanically-created canal through the acutely occluded thrombus was, subsequently, enlarged by spontaneous resolution of thrombotic material.

Since permanent recanalization of the total occlusion was found in only 4 of the 9 medically treated patients, but in all of the mechanically recanalized patients, it might be concluded, that the rate of recanalizations can be increased by mechanical intervention in the acute stage of infarction. However, this hypothesis must be substantiated by further research.

Changes of Left Ventricular Function in Medically Treated Patients (Group B)

In comparison to the acute stage of infarction, there was a significant increase of enddiastolic as well as endsystolic volume in the chronic stage. There was a significant decrease of ejection fraction, whereas stroke volume did not change significantly as compared to the acute stage of infarction. This is in agreement with the results of Theroux et al who found “progressive dilatation and increase in function” of the surviving myocardium during the healing phase of infarction in animal experiments (55). Bergmann found hypertrophy of the surviving myocardium (4). Increase of enddiastolic volume might also be due to increase of fiber stretch during the chronic stage of infarction, however our data do not permit a differentiation between changes of fiber stretch and growth processes of the surviving myocardium.

There were 2 patients in the medically treated group, in whom ejection fraction did not decrease and endsystolic volume index did not increase during the chronic stage of infarction. These are the 2 patients, in whom the akinetic segment had shortened as compared to the acute stage. This finding is in agreement with the observations of Bardet et al (2) and Farcot et al (15) who, in a larger series of patients, observed an improvement of pump function in those patients who had improvement of local wall function in the infarct area; in patients, in whom wall motion of the infarct area did not change or deteriorated, there was a significant increase of the endsystolic volume and decrease of ejection fraction.

Relationships Between Changes of Local Wall Function and Coronary Artery Morphology in Group B

The length of the akinetic segment decreased in the chronic stage in 2 of the 9 conventionally treated patients. In both patients there was collateral flow to the infarct area during the acute stage of infarction. A number of clinical (21, 32, 58), pathologic-anatomical (6, 18) and experimental findings (45, 48, 53) indicate that the extent of myocardial injury can be limited by collateral flow. Therapy with nitrates and IABP could have been an additional factor limiting infarct size (5, 7, 12, 17, 20, 25, 31, 33, 34, 43, 50, 51, 56). On the other hand, there were col-
latterals to the infarct area in 6 of the 9 patients during the acute stage of infarction. In 4 of these patients, the length of the AKS increased, although these patients had received the same therapy as the other 2 patients with decreased akinetic segment.

In the 2 patients with shortened AKS, there was a spontaneous recanalization of the infarct vessel in the chronic stage. However, spontaneous recanalization occurred in 2 additional patients of the medically treated group. One of these 2 patients had also had collateral flow to the infarct area during the acute stage of infarction. Our findings do not permit a detailed analysis of the complex interrelationships between changes of local wall function, collateral flow in the acute stage, therapy by nitrates and IABP, and spontaneous recanalization.

**Changes of Left Ventricular Function in Group A as Compared to Group B**

Volume parameters did not change significantly in Group A, whereas the length of AKS decreased significantly in these patients. These changes resulted in two significant differences between Groups A and B in the chronic stage of infarction.

1. The AKS was significantly shorter in the transluminally recanalized patients as compared to the medically treated group. This difference is paralleled by a lower peak value of CPK in the Group A patients.

2. Ejection fraction was significantly higher in the transluminally recanalized patients than in the medically treated group. These differences did not exist during the acute stage of infarction.

In the acute stage of infarction, there were collaterals to the infarct area in only 2 of the 5 Group A patients, in whom changes of left ventricular function could be evaluated, whereas in the medically treated group there were collaterals in 6 of 9 patients. There were no differences in medical treatment or use of counterpulsation between the two groups.

The most plausible explanation for the differences of left ventricular function between the two groups in the chronic stage seems to be variation of the fate of total occlusion in the infarct-related vessel. The infarct-related vessel was recanalized in all patients of Group A, but only in 4 of 9 patients of Group B. In Group A, recanalization of the infarct-related vessels was achieved sooner after total occlusion than in Group B.

There is a close analogy between transluminal recanalization of a totally occluded vessel during evolving infarction in humans and reperfusion experiments, which were performed by a number of investigators in animals (1, 27, 29, 35, 42, 45, 49). These animal experiments have shown that with increasing duration of coronary occlusion, irreversible injury progresses from the subendocardial to the subepicardial myocardium. In dogs, the presence of a subepicardial zone of ischemic but viable myocardium, which is available for salvage for at least three hours, has been demonstrated. The mechanism of subendocardial to subepicardial progression of injury is related to the transmural distribution of coronary collateral flow.

Although there is no experimental model which completely simulates the development of coronary heart disease and collaterals in humans, the histological findings of **Page et al** seem to substantiate the concept of progressive myocardial necrosis in the periinfarction zone in humans (41). **Page et al** found areas of myocardial necrosis of varying histological ages in patients who died of cardiogenic shock.

**Conclusion**

Our findings seem to indicate that transluminal recanalization in the early phases of myocardial infarction might result in salvage of jeopardized myocardium. However, no definite conclusions can be drawn, in view of the small number of cases of this pilot study. Further research will be needed to improve the technique, and to test its results.

**Acknowledgment**

The authors gratefully acknowledge the technical assistance of A. Henniges, U. Spaar, E. Rohrmoser, and the editorial assistance of C. Bauer.

**References**


27. Kane JJ, Murphy ML, Bissett JK, de Soya N, Doherty JE, Straub KD: Mitochondrial function, oxygen extraction, epicardial ST segment changes and titrated digoxin distribution after reperfusion of ischemic myocardium. Am J Cardiol 36, 218 (1976)


35. Mathur VS, Quinn GA, Burris WH: Maximal revascularization (reperfusion) in intact conscious dogs after two to five hours of coronary occlusion. Am J Cardiol 36, 252 (1975)


60. WHO-Regional Office for Europe: Ischemic heart disease register, report of the fifth working group. Copenhagen (1971)