Nonsurgical Coronary Artery Recanalization In Acute Transmural Myocardial Infarction

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SUMMARY In 41 consecutive patients with an acute transmural myocardial infarction (AMI) admitted within 3 hours after the onset of symptoms, we tried to recanalize the occluded coronary artery by an intracoronary infusion of streptokinase (SK) (2000 units/min). SK infusion was preceded by (1) an intracoronary injection of 0.5 mg nitroglycerin to rule out coronary artery spasm, (2) an attempt to recanalize the vessel mechanically with a flexible guidewire, and (3) an intracoronary injection of plasminogen (500 units) to increase the efficacy of the subsequent SK infusion. Coronary angiography revealed a total coronary artery occlusion in 39 patients and a subtotal occlusion in two patients. In 30 patients (73%), the occluded coronary artery was successfully recanalized within 1 hour (mean 29 ± 15 minutes), resulting in prompt contrast filling of the previously occluded vessel. An arteriosclerotic stenosis always remained at the site of the occlusion. Nitroglycerin opened the occluded coronary artery in one patient, contrast injection in seven patients and guidewire perforation in four of the 15 patients, in whom it was attempted. In 18 patients the occluded coronary artery was recanalized by intracoronary SK infusion alone. After the initial opening of the occluded coronary artery, subsequent SK infusion markedly reduced the degree of stenosis and visible thrombi disappeared. Clinically, recanalization was associated with significant relief of ischemic chest pain.

None of the successfully recanalized patients died, including three patients with cardiogenic shock. Recanalization, however, did not prevent myocardial infarction, as shown by new Q waves and/or R-wave reduction in 24 of the 30 patients and by the rise in serum CPK with an early peak, indicating CPK washout by coronary artery reperfusion. Repeat angiography 7–21 days later revealed a patent coronary artery in 12 of 15 successfully recanalized patients. The left ventricular ejection fraction had significantly improved, from 37 ± 5% to 47 ± 4% (mean ± SEM). Failure of recanalization in 11 of 41 patients may be explained by the absence of coronary artery thrombosis or poor SK penetration of the thrombus because of its distal location or SK runoff into nonaffected arteries.

Thus, in patients with acute myocardial infarction the occluded coronary artery can be rapidly recanalized in 73% of the patients by an intracoronary infusion of streptokinase.

ENCOURAGED by the first positive results suggesting that an acutely occluded coronary artery can be rapidly recanalized with a catheter, we attempted coronary artery recanalization in all patients with an acute myocardial infarction (AMI) admitted to our hospital, provided the infarct was estimated to be no older than 3 hours. Animal experiments have shown that coronary artery reperfusion within this period reduces infarct size.4,6 In this paper, we describe the procedure itself and its effect on coronary artery anatomy. Randomized studies may be required to determine if and to what extent jeopardized myocardium can be saved by this approach.

Methods

Patient Selection

Patients who arrived at the emergency room of the Hamburg University Hospital with a history and electrocardiographic evidence of AMI were considered candidates for recanalization if the duration of ischemic chest pain was less than 3 hours and significant ST-segment elevation was present on the ECG with no changes in the QRS complexes.

Informed consent for recanalization was obtained from the patient and his relatives; in patients in cardiogenic shock, consent was obtained only from the patient’s relatives.

Protocol

Recanalization was performed on a 24-hour basis. In preparation for the procedure, patients with cardiogenic shock were intubated and artificially ventilated. The patients were heparinized with 10,000 units as a i.v. bolus. To avoid significant bradycardia during coronary angiography, in most patients a pacing catheter was placed in the apex of the right ventricle. In patients with left-heart failure, a Swan-Ganz catheter was advanced to the pulmonary artery. The coronary artery judged to be occluded, based on the distribution of ST-segment elevation on the ECG, was catheterized by a second investigator using a #8F Judkins catheter. In most cases, the “infarct-vessel” was easy to recognize and was either totally or subtotally occluded. The acute nature of a total occlusion was often characterized by a poor washout of the contrast medium (meglumine and sodium diatrizoate) from the area proximal to the occlusion. A subtotal occlusion was defined as stenosis that permitted only markedly delayed filling of the distal artery. After visualizing the
occlusion, nitroglycerin, 0.5 mg, was slowly injected into the occluded artery to rule out coronary artery spasm as a cause of the acute occlusion.\(^7\) Then, we tried to recanalize the "infarct vessel" mechanically by passing a 0.032-inch guide wire with movable core through the occlusion.\(^8\) If not easily successful, mechanical recanalization was abandoned and 500 units of human plasminogen (investigational drug, Behring Werke) were slowly injected into the occluded artery to increase the efficacy of the subsequent intracoronary streptokinase (Streptase, Behring Werke) infusion.\(^9\) Two thousand units of streptokinase per minute dissolved in Ringer's solution were then infused through the Judkins catheter into the occluded coronary artery at a rate of 4 ml/min. At 15-minute intervals, the effect of the streptokinase infusion was checked angiographically. The infusion was continued until the artery was maximally patent. In our experience, an infusion for more than 2 hours has not proved to be effective. After 2 hours of intracoronary streptokinase infusion, a systemic fibrinolytic effect was present. To complete the examination, the non-affected coronary artery and the left ventricle were visualized. The patient was then transferred to the Intensive Care Unit with the pacing catheter and the Swan-Ganz catheter left in place. The pigtail catheter was pulled back into the abdominal aorta, where it was left for 24 hours.

In one patient with an occlusion of the distal right coronary artery, the technique was slightly modified. The streptokinase infusion was given through a \#3F catheter, which was advanced through the Judkins catheter into the midportion of the right coronary artery. In another patient, with evidence of continued coronary artery spasm, nitroglycerin (2 mg/hour) was added to the intracoronary streptokinase infusion.

All patients were anticoagulated during their hospital stay and given long-term anticoagulation thereafter, except for two patients in whom anticoagulants were stopped because of old age. Coronary and left ventricular angiography were repeated later.

**Results**

**Clinical Characteristics**

Coronary artery recanalization was performed in 41 patients, ages 32–82 years (mean 64 years). According to the protocol, the time from the onset of symptoms to admission was never more than 3 hours (average 83 ± 56 minutes). In all patients, the ECG on admission showed more than 2 mm of ST-segment elevation. Fifteen patients had an anterior or anterolateral AMI, and 26 an inferior AMI. Six patients were in cardiogenic shock with severe hypotension, signs of peripheral hypoperfusion and an elevated pulmonary capillary wedge pressure. Blood samples for enzyme determinations were obtained on admission, but we did not wait for the results. Later, we learned that serum CPK was still within the normal range in all but one patient, in whom it was elevated to 210 units (normal < 70 units).

**FIGURE 1.** Right coronary arteriograms in the left anterior oblique 30\(^\circ\) position in inferior acute myocardial infarction. (A) Subtotal occlusion of the proximal right coronary artery (RCA) with two filling defects (arrows), presumably representing coronary artery thrombosis. (B) During contrast injection, the lower defect moves peripherally. (C) The contrast injection caused a new (embolic) occlusion. (D) After 45 minutes of intracoronary streptokinase infusion, the distal occlusion and the remaining proximal filling defect have disappeared and the artery fills rapidly and completely. A significant stenosis persists in the proximal RCA.
Coronary Artery Recanalization

Before the summarized data of the whole group are presented, the angiograms of individual cases are discussed.

Case 1 (fig. 1)

In this 81-year-old female patient with inferior AMI 2 hours after the onset of symptoms, the proximal right coronary artery was subtotally occluded. Below the site of occlusion, two filling defects were seen (fig. 1A). During contrast injection, the lower defect moved toward the periphery of the vessel (fig. 1B), where it caused a new, complete occlusion (fig. 1C). After 45 minutes of intracoronary streptokinase infusion, the distal occlusion and the filling defect that had proximally remained had disappeared. A stenosis at the site of the previous occlusion still remained (fig. 1D).

Case 2 (fig. 2)

The coronary angiogram of this 73-year-old woman with inferolateral AMI and cardiogenic shock showed a total occlusion of the proximal right coronary artery (fig. 2A). Contrast injection alone opened the vessel. A stenosis at the site of the occlusion became visible, with an oval-shaped filling defect below the stenosis (fig. 2B). The filling defect disappeared after intracoronary streptokinase infusion (fig. 2C). Within 2 hours after recanalization, the symptoms of shock were reversed.

Case 3 (fig. 3)

The angiograms of this 40-year-old patient with an inferior AMI 2.5 hours after the onset of chest pain show a combined mechanical and pharmacologic recanalization. Despite significant wall irregularities, the occlusion of the distal right coronary artery (fig. 3A) was passed with a 0.032-inch flexible guidewire (fig. 3A), which created a small channel through the occlusion (fig. 3C). After 45 minutes of intracoronary streptokinase infusion, this channel had become wider and shorter (fig. 3D). The control angiogram 6 weeks later, however, showed reclosure of the right coronary artery at the same point.

Case 4 (fig. 4)

The acute angiogram of this 71-year-old patient with inferolateral AMI who died in cardiogenic shock on the second day after admission showed a distal right coronary artery occlusion similar to that in case 2. The occluded artery, however, did not open despite

Figure 2. (A) Proximal right coronary artery (RCA) occlusion in a 73-year-old patient with inferolateral acute myocardial infarction and cardiogenic shock. (B) After contrast injection, the RCA is patent. A stenosis with a filling defect (acute thrombus) below the stenosis is visible. (C) The angiogram after intracoronary streptokinase infusion reveals the disappearance of the thrombus and faster filling beyond the residual obstruction.
2 hours of intracoronary streptokinase infusion. The postmortem examination revealed a high-grade arteriosclerotic stenosis and an acute thrombus immediately behind the stenosis.

**Case 5 (fig. 5)**

This 45-year-old patient with distal right coronary artery occlusion had an inferior AMI in the hospital. A #3F catheter was advanced through the Judkins catheter into the midportion of the right coronary artery to administer the streptokinase closer to the occlusion. After 15 minutes of streptokinase infusion, the right coronary artery was patent.

**Case 6 (fig. 6)**

In this 64-year-old male patient, routine coronary and left ventricular angiograms were performed because of mitral valve disease 2 days before the
anterior AMI. The angiograms showed a high-grade proximal left anterior descending artery (LAD) stenosis. One hour after the onset of infarct, the LAD was found to be occluded at the site of this stenosis. Intracoronary nitroglycerin did not open the occlusion. After 30 minutes of intracoronary streptokinase infusion, prompt contrast filling of the distal RCA appears.

**Figure 5.** (A) Distal right coronary artery (RCA) occlusion 90 minutes after the onset of inferior acute myocardial infarction. (B) Advancement of a small Teflon catheter into the midportion of the RCA. (C) After 15 minutes of intracoronary streptokinase infusion through the small catheter, the vessel opened. (D) After another 30 minutes of streptokinase infusion, prompt contrast filling of the distal RCA appears.

**Figure 6.** A sequence of four left coronary arteriograms in anterior acute myocardial infarction (double oblique projection, right anterior oblique 30° + 25° caudal tilt). (A) Routine coronary angiography obtained 2 days before the infarct shows a significant proximal left anterior descending coronary artery (LAD) stenosis. (B) One hour after the onset of infarct, the LAD is completely occluded at the point of the stenosis. (C) Thirty minutes after intracoronary streptokinase infusion, the LAD is patent again. A filling defect (thrombus) is present behind the stenosis. (D) The thrombus has disappeared after another 30 minutes of intracoronary streptokinase infusion. The original stenosis appears to be more severe than before the infarct.
infusion, the occluded coronary artery was patent. A filling defect below the stenosis was visible. It disappeared after another 30 minutes of intracoronary streptokinase infusion.

Case 7 (fig. 7)

The left coronary angiogram of this 32-year-old patient with an extensive anterior AMI showed a proximal occlusion of the LAD, which was opened by an intracoronary injection of 0.5 mg of nitroglycerin. A stenosis and the typical defect behind the stenosis became visible. After 15 minutes of intracoronary streptokinase infusion, there was a tendency toward reocclusion despite i.v. nitroglycerin infusion and sublingual nifedipine. Only after nitroglycerin (2 mg/hour) had been added to the intracoronary streptokinase infusion did the artery remain patent. The filling defect behind the stenosis disappeared after 45 minutes of streptokinase infusion. The intracoronary nitroglycerin infusion was continued until an aortocoronary bypass operation could be performed.

The occluded vessel, which was the right coronary artery in 16 patients, the LAD in 15 patients and the circumflex coronary artery in 10 patients, was successfully recanalized in 30 of 41 patients. Successful recanalization always resulted in prompt contrast filling of the artery distal to the site of the occlusion. In all cases, an arteriosclerotic obstruction remained after recanalization.

Recanalization was achieved by (1) an intracoronary injection of nitroglycerin in one patient (a remaining filling defect disappeared after subsequent intracoronary streptokinase infusion); (2) intracoronary contrast injection with subsequent intracoronary streptokinase infusion in seven patients; (3) guidewire perforation of the occlusion and subsequent intracoronary streptokinase infusion in four patients; and (4) intracoronary streptokinase infusion alone in 18 patients. In patients in whom nitroglycerin, contrast injection or the guidewire were initially successful, further widening of the stenosis or the disappearance of visible filling defects was achieved by subsequent intracoronary streptokinase. The mean duration of intracoronary streptokinase infusion required for successful recanalization was 29 ± 15 minutes. A filling defect (figs. 1, 2, 6 and 7) was noted in 11 of the 30 successfully recanalized patients. It always disappeared after intracoronary streptokinase infusion.

Recanalization was not successful in 11 of the 41 patients. The causes of failure are not completely clear. Two patients with a proximal LAD occlusion on the acute angiogram and no response to streptokinase and nitroglycerin died on the second day of the infarct due to cardiogenic shock. A high-grade arteriosclerotic obstruction, but no thrombus, was found on autopsy. In a patient who had distal right coronary artery occlusion and poor contrast runoff from the area proximal to the occlusion, the postmortem examination revealed a thrombotic occlusion (fig. 4). Three more patients with distal right coronary artery occlusion and poor runoff of the contrast medium from the preocclusion area did not respond to streptokinase. In four unsuccessful cases, the circumflex coronary artery was involved. The average time of intra-

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**Figure 7.** Left coronary arteriograms in a 32-year-old patient with an extensive anterior acute myocardial infarction. (A) Left anterior descending coronary artery (LAD) occlusion at the bifurcation. (B) Opening of the LAD immediately after 0.5 mg of nitroglycerin injection into the left coronary artery. A stenosis and a thrombus (better seen in C) become visible. (C) After 15 minutes of intracoronary streptokinase infusion there is a tendency toward reocclusion. (D) After adding nitroglycerin to the intracoronary streptokinase infusion, the LAD remains patent and the thrombus is dissolved.
coronary streptokinase infusion in the failure group was 99 ± 17 minutes.

Subsequent Catheterization

Nineteen of the 41 patients were recatheterized 7–21 days after the AMI. Fifteen belonged to the successfully recanalized group and four to the unsuccessful group. In the latter, the initial occlusion was still present. In the group of successfully recanalized patients, the recanalized coronary artery was patent in 12 of 15 patients. Despite adequate anticoagulation, it was totally reoccluded in two patients and subtotally reoccluded in another. As evident from a second peak in serum CPK, reocclusion most likely had occurred during the first days of the acute infarct.

Clinical Effects of Recanalization and Further Patient Management

None of the successfully recanalized patients died, including three patients with cardiogenic shock. Successful recanalization was always associated with significant relief of ischemic chest pain.

Serum CPK rose rapidly immediately after recanalization, reaching an early peak 13 ± 1 hours (± SEM) after the onset of the infarct. Peak serum CPK was 712 ± 93 units on the average. In the unsuccessfully recanalized group, peak serum CPK was 816 ± 98 units. Here, the peak was reached 26 ± 4 hours after the onset of the infarct. A significant reduction of ST-segment elevation in the 12-lead surface ECG was always noted immediately after successful recanalization. New Q waves and/or R-wave reduction, however, appeared in 24 of 30 recanalized patients.

Only one of the 30 successfully recanalized patients had severe left-heart failure after recanalization, which required afterload-reducing agents in addition to diuretics. No patient required anti-anginal drugs. Twenty of the 30 successfully recanalized patients were treated medically. Three of them had enzymatic and electrocardiographic evidence of reinfarction within 10 days after the AMI. Two other patients had to be readmitted because of suspected reinfarction 6 and 8 weeks after the acute event. Reinfarction was ruled out in these patients. The remaining 15 patients have been asymptomatic for 3–12 months.

Reinfarction after successful recanalization led us to advocate early bypass surgery in successfully recanalized patients. So far, 10 patients, mean age 53.7 years (range 28–67 years), underwent uneventful bypass surgery 1–7 days after recanalization. A hemorrhagic infarct was not seen at the time of surgery.

In the unsuccessfully recanalized group, three of the 11 patients died in cardiogenic shock 2 days after the AMI. Six had to be kept on diuretics because of intermittent pulmonary congestion. Postinfarction angina was not noted in these patients.

The global left ventricular ejection fraction, measured acutely and 7–21 days later, showed a significant increase, from an average of 37 ± 5% to 47 ± 4% in the 11 successfully recanalized patients who could be restudied. Nine of the 20 nonoperated patients refused to be reinvestigated. In patients in whom recanalization failed, the ejection fraction deteriorated (fig. 8).

Complications

Seven patients had ventricular fibrillation during the procedure, two immediately after contrast injection and five during the phase of reperfusion, but independent of contrast injection. Defibrillation reestablished normal sinus rhythm in every case. In one patient, a small side branch of the right coronary artery was perforated with the guidewire. Contrast injection revealed a subepicardial depot of contrast medium. The procedure was terminated before the streptokinase infusion, with no further complications. Except for minor hematomas at the puncture site, no bleeding complications were observed.

Discussion

The purpose of this investigation was to determine whether an acutely occluded coronary artery could be rapidly recanalized with a catheter. The technique used for recanalization was an infusion of streptokinase into the occluded coronary artery, preceded by
intracoronary administration of nitroglycerin and an attempt to recanalize the vessel mechanically. Streptokinase itself is not a fibrinolytic agent. It combines with plasminogen to form an activator complex, which transforms plasminogen to plasmin. Plasmin lyases fibrin. Plasminogen has a high affinity for fibrinogen and fibrin and therefore is adsorbed onto the thrombus. The high plasminogen concentration enables a more rapid local lysis. Therefore, we injected plasminogen into the occluded coronary artery before the intracoronary streptokinase infusion.

The study group included consecutive patients with an acute transmural myocardial infarction admitted to our hospital within 3 hours after the onset of symptoms. They had severe ischemic chest pain unresponsive to sublingual nitroglycerin and significant ST-segment elevation on the ECG, with no changes in the QRS complexes. All but one patient had no enzyme elevation on admission, confirming the early stage of the infarct. The angiographic findings in these patients were reproducible. In patients with acute inferior myocardial infarction, either the right or the circumflex coronary artery was occluded. The LAD was found to be occluded in patients with an acute AMI. The acute nature of the occlusion was often recognized by no or poor washout of the contrast medium from the preocclusion area of the vessel.

The occluded coronary artery was successfully recanalized in 73% of the patients. In only one patient (fig. 7), did intracoronary nitroglycerin open the artery, suggesting that spasm contributed to the occlusion. After the artery had become patent, an arteriosclerotic stenosis and a filling defect behind the stenosis were seen. The filling defect disappeared after intracoronary streptokinase infusion. Therefore, the defect probably represented coronary artery thrombosis. Thus, in this patient 2 hours after the onset of symptoms, three factors contributed to the proximal LAD occlusion: an arteriosclerotic stenosis, coronary artery spasm and coronary artery thrombosis. In seven of the 30 successfully recanalized patients, the occluded coronary artery was unexpectedly opened by the intracoronary injection of contrast medium, although we do not know what caused this. The forceful intracoronary injection itself may have swept the thrombus away from the site of occlusion, or coronary artery spasm may have been relieved due to the vasodilating effect of the contrast medium. In all instances of successful contrast injection, a thrombus became visible behind the site of the occlusion and disappeared after streptokinase infusion.

The third method used before intracoronary streptokinase infusion was mechanical recanalization using a flexible guidewire. This procedure, which may carry a risk, was successful only in four of the 15 patients in whom it was attempted. In 18 patients, an intracoronary streptokinase infusion alone opened the occluded coronary artery; this made it difficult to determine the value of the other measures, because streptokinase alone might have had the same effect. We believe, however, that a small channel through the occlusion created by any method improves the penetration of the thrombus by a locally administered streptokinase infusion. Therefore, streptokinase may have always resulted in further opening of the artery or the disappearance of visible thrombi in patients in whom the occluded artery was opened by any of the preceding measures.

In 11 of the 41 patients, recanalization failed despite an intracoronary streptokinase infusion that was four times longer than that in the successfully recanalized group. The causes of failure are not known. In two of the three patients who died the day after the procedure, the autopsy revealed a high-grade arteriosclerotic stenosis but no thrombus. Either spasm or a thrombus that was dissolved later may have caused the complete occlusion on the acute angiogram. In the third patient (fig. 4), the postmortem examination revealed an acute thrombus behind an organic stenosis of the distal right coronary artery. Particularly poor runoff of contrast medium from the area proximal to the occlusion in this patient suggested that the streptokinase did not reach the thrombus in a high enough concentration to dissolve it. A similar situation of a distal right coronary artery occlusion with poor runoff of the contrast medium was found in three more patients in whom recanalization failed.

On the basis of our findings, it is tempting to speculate about the pathophysiology of AMI. Undoubtedly, the infarct is caused by coronary artery occlusion. An arteriosclerotic obstruction forms the basis for the occlusion. Visible thrombi that disappear after intracoronary streptokinase infusion were present in 27% of the patients. If we accept a positive streptokinase effect as indirect evidence of coronary artery thrombosis, 2-4 hours after the onset of AMI a thrombus was present in 73% of the patients. This frequency would be consistent with pathologic studies that demonstrate coronary artery thrombosis in 54-96% of the cases. We do not know when the thrombus occurred in our patients, but at the time of the study it contributed significantly to the occlusion. Recent papers suggest that coronary artery spasm may be the initial event in AMI. Spasm, however, could be demonstrated with certainty in only one patient. Spasm could have been the first step in the acute occlusion with secondary thrombus formation. However, 2-4 hours after the onset of symptoms, coronary artery thrombosis appears to be the more important mechanism of coronary artery occlusion. Theoretically, there would be no need to introduce spasm as an obligatory step in thrombus formation. Local hemodynamics behind an arteriosclerotic stenosis would also easily explain the formation of a thrombus.

Coronary artery recanalization did not open the occluded artery completely. A significant arteriosclerotic stenosis remained at the site of the occlusion. This condition probably represents the state of the coronary artery, as it had been before the acute occlusion (fig. 6). Despite the remaining stenosis, there was prompt contrast filling of the distal artery and a normal washout of the contrast medium, suggesting that recanalization resulted in some degree of coronary artery reperfusion. Because of the stenosis, the distal perfusion pressure may have been lower than
aortic pressure, reducing the likelihood of hemorrhagic infarct.

The value of nonsurgical coronary artery recanalization depends on whether a significant amount of ischemic myocardium threatened by infarction can be saved by this method. From studies of patients with variant angina we know that early coronary artery reperfusion prevents myocardial necrosis. However, reperfusion 2-4 hours after the onset of infarct as achieved by our method may be too late. In fact, our data reveal that myocardial necrosis was not completely prevented, as evident from the ECG and the rise of serum CPK. When considering CPK values, one has to consider that CPK washout after coronary artery reperfusion may have contributed significantly to the amount of CPK released from the infarcted myocardium. The early peak in serum CPK would be consistent with such a washout phenomenon. Apart from these objections, there is strong evidence that coronary artery recanalization is beneficial. Invariably, ischemic chest pain disappeared after successful recanalization. Left ventricular ejection fraction significantly improved after successful recanalization, from an average of 37 ± 5% to 47 ± 4%. The clinical course of the successfully recanalized patients was surprisingly uncomplicated, with a mortality rate of 0%, including three patients with cardiogenic shock. In these three patients, recanalization resulted in a dramatic reversal of the shock symptoms. A recent observation in an individual patient supports the potential benefit of the method. In this patient, with an acute inferolateral myocardial infarction and cardiogenic shock, recanalization was associated with the disappearance of the shock. Three days later the patient again complained of anginal chest pain. The ECG showed ST-segment elevation associated with a secondary rise in serum CPK from 800 to 1300 units. Repeat angiography showed reclosure of the right coronary artery at the same location. Thus, reclosure resulted in reinfarction. The amount of reinfarcted myocardium probably represents the amount of myocardium that was saved by the first procedure.

Our study demonstrates that in patients with an acute transmural myocardial infarction of less than 3 hours' duration the acutely occluded coronary artery can be rapidly recanalized via catheter. There is evidence that jeopardized myocardium can be saved by this approach. However, further studies are needed to determine the ultimate benefit of the method.

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