Effect of High-Dose Intracoronary Adenosine Administration During Primary Percutaneous Coronary Intervention in Acute Myocardial Infarction
A Randomized Controlled Trial

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Background—Coronary microvascular dysfunction is frequently seen in patients with ST-elevation myocardial infarction after primary percutaneous coronary intervention. Previous studies have suggested that the administration of intravenous adenosine resulted in an improvement of myocardial perfusion and a reduction in infarct size. Intracoronary adenosine (bolus of 30 to 60 μg) is a guideline-recommended therapy to improve myocardial reperfusion. The effect of intracoronary adenosine during primary percutaneous coronary intervention has not been investigated in a large randomized trial.

Methods and Results—Patients presenting with acute ST-elevation myocardial infarction were randomized to 2 bolus injections of intracoronary adenosine (2 × 120 μg in 20 mL NaCl) or placebo (2 × 20 mL NaCl). The first bolus injection was given after thrombus aspiration and the second after stenting of the infarct-related artery. The primary end point was the incidence of residual ST-segment deviation <0.2 mV, 30 to 60 minutes after percutaneous coronary intervention. Secondary end points were ST-segment elevation resolution, myocardial blush grade, Thrombolysis in Myocardial Infarction flow on the angiogram after percutaneous coronary intervention, enzymatic infarct size, and clinical outcome at 30 days. A total of 448 patients were randomized to intracoronary adenosine (N = 226) or placebo (N = 222). The incidence of residual ST-segment deviation <0.2 mV did not differ between patients randomized to adenosine or placebo (46.2% versus 52.2%, P = NS). In addition, there were no significant differences in secondary outcome measures.

Conclusions—In this randomized placebo controlled trial enrolling 448 patients with ST-elevation myocardial infarction, administration of intracoronary adenosine after thrombus aspiration and after stenting of the infarct-related artery did not result in improved myocardial perfusion. (Circ Cardiovasc Intervent. 2009;2:00-00.)

Key Words: myocardial infarction ■ adenosine ■ angioplasty ■ reperfusion

Primary percutaneous coronary intervention (PCI) is the preferred treatment to achieve reperfusion of the infarct-related artery in patients with ST-elevation myocardial infarction (STEMI).1,2 Despite restoration of epicardial flow, impaired myocardial perfusion is frequently seen after primary PCI and results in a larger infarct size and increased mortality.3 In addition to embolization of atherothrombotic material,4 vasoconstriction, platelet aggregation, neutrophil adhesion and myocardial edema, and necrosis are major contributors to microvascular dysfunction.5 Large randomized trials have suggested that a high-dose intravenous administration of adenosine during intervention for acute myocardial infarction may result in a reduction in infarct size.8,9 Two clinical studies have documented that intracoronary administration of adenosine is safe in patients with acute myocardial infarction and may result in a lower incidence of no reflow.10,11 Improved myocardial reperfusion as assessed by ST-segment elevation resolution after primary PCI was observed in a cohort of 79 patients treated with intracoronary adenosine compared with 200 historical controls12 and in a randomized trial of 51 patients.13 Although intracoronary administration of adenosine in a dose of 30 to 60 μg is recommended for the treatment of microvascular dysfunction during primary PCI in STEMI guidelines,14 large prospective randomized trials are lacking. The aim of this study was to investigate the effect of...
Methods

Population
The ADenosine Administration during and after Primary percutaneous coronary intervention in acute myocardial infarction Trial was a single-center, prospective randomized open trial with blinded evaluation of end points. All consecutive patients presenting to the University Medical Center of Groningen suspected of an acute STEMI and candidates for primary PCI were eligible for participation. Inclusion criteria were symptoms of chest pain suggestive for myocardial ischemia for at least 30 minutes, a time from onset of symptoms of <12 hours before hospital admission, and an ECG showing ST-segment elevation of $>0.1$ mV in 2 or more leads. Exclusion criteria were the presence of cardiogenic shock, existence of a life-threatening disease with a life expectancy of <6 months, receiving pharmacotherapy for chronic obstructive pulmonary disease, or no informed consent. After coronary angiography, patients were randomized when PCI was indicated. Patients treated conservatively or with emergency coronary artery bypass grafting were not enrolled. The study was approved by the medical ethics committee of our institution.

Randomization and Treatment
After the performance of an initial coronary angiography, patients who met the eligibility criteria were randomized (1:1) to 2 high-dose bolus injections of intracoronary adenosine ($2 \times 120 \mu g$ in 20 mL 0.9% NaCl) or placebo ($2 \times 20$ mL 0.9% NaCl) after thrombus aspiration and after stenting. For adenosine, we used Adenocor (Sanofi-Synthelabo BV, Maassluis, The Netherlands). To protect the microcirculation against embolization of atherothrombotic material and to restore epicardial coronary flow, manual thrombus aspiration was performed (Export Aspiration Catheter, Medtronic Corporation, Santa Rosa, Calif). After restoration of epicardial flow, intracoronary nitroglycerine ($400 \mu g$) was administered through the guiding catheter, followed by the first bolus of intracoronary adenosine or placebo through the guiding catheter. After stenting of the infarct-related lesion and an additional injection of nitroglycerine, the second bolus intracoronary adenosine or placebo was administered.

All patients received aspirin (500 mg), heparin (5000 IU), and clopidogrel (600 mg) after confirmation of ST-segment elevation on the first ECG. During primary PCI, patients received the glycoprotein Ib/IIa inhibitor abciximab (0.25 mg/kg), if not contraindicated. Additional heparin was administered guided by the activated clotting time. The standard treatment after primary PCI included aspirin, clopidogrel ($\geq 1$ month), $\beta$-blockers, lipid-lowering agents, and angiotensin-converting enzyme inhibitors or angiotensin-II receptor blockers.15

End Points and Definitions
The primary end point was the incidence of residual ST-segment deviation $<0.2$ mV on the 12-lead ECG made at 30 to 60 minutes after primary PCI. Residual ST-segment deviation was calculated as the sum of residual ST-segment elevation and depression in all leads, with the magnitude of ST-segment elevation or depression measured at 60 milliseconds from the J-point. The residual ST-segment deviation was categorized as $<0.2$ mV, $0.2$ to $0.5$ mV, $0.5$ to $1.0$ mV, or $>1.0$ mV. ST-segment elevation resolution was measured by comparing the sum of ST-segment elevation on the postintervention ECG with that on the ECG at presentation, and categorizing the ST-segment resolution as complete ($>70\%$), partial ($30\%$ to $70\%$), or no resolution ($<30\%$).16 In addition, the presence of new Q waves was assessed on the ECG after PCI, defined as an initial negative deflection of the QRS complex of $>0.1$ mV and $>40$ milliseconds in an ECG lead related to the myocardial area involved in the STEMI. In addition, all the pathological Qs associated with prior myocardial infarction were assessed.17

Angiographic Analysis
Coronary angiograms were reviewed by 2 observers blinded for treatment allocation and clinical data. Angiographic records were obtained before primary PCI, after thrombus aspiration, after stenting, and after the second administration of intracoronary adenosine or placebo. On the initial angiogram, the site of the infarct-related coronary lesion reflecting the myocardial territory at risk was assessed. In the right coronary artery and circumflex artery, the infarct-related lesion was classified as proximal or as distal in the infarct-related artery. In the left artery descendens, lesions were classified as proximal, mid, or distal. On the initial angiogram and on the angiogram after stenting, thrombolysis in myocardial infarction flow grade,18 and angiographic evidence of thrombus in the infarct-related lesion, and distal embolization were assessed. Angiographic evidence of thrombus was evaluated according to the criteria as previously described by Mabin et al.19 Distal embolization was defined as a filling defect, with an abrupt cutoff in the vessel located distally from the infarct-related coronary lesion.4 In addition, myocardial blush grade was assessed on the angiogram after stenting. The evaluation of myocardial blush grade was performed as described by van’t Hof et al.:0, no myocardial blush; 1, minimal myocardial blush or contrast density; 2, moderate myocardial blush or contrast density, but less than that obtained during angiography of a contraor ipsilateral non–infarct-related coronary artery; and 3, normal myocardial blush or contrast density, comparable with that obtained during angiography of a contra or ipsilateral non–infarct-related coronary artery.

Quantitative Blush Evaluation
To evaluate the immediate effect of intracoronary adenosine or placebo on myocardial perfusion, we compared myocardial perfusion on the angiogram after stenting and on the angiogram performed directly after the second bolus administration of intracoronary adenosine or placebo. Analysis of myocardial perfusion was performed by computerized quantitative blush evaluation (QuBE), as described earlier.20 QuBE was measured blinded to clinical data and treatment allocation by 2 observers. The QuBE program loads the coronary angiogram and allows the operator to select the run for quantification of the myocardial perfusion. On the selected run after stenting, the operator indicates a polygonal shape that contains the distal infarct-related area in which the myocardial perfusion should be measured. The same polygonal shape is copied to the run that is made after the second bolus intracoronary adenosine or placebo. By quantifying the myocardial contrast density on each frame and by identifying the sum of greyvalues over time, a continuous value is calculated. This value allows a fine-grained assessment of myocardial perfusion, which correlates well with angiographic and clinical outcome measures.20

Biomarkers Reflecting Infarct Size
Serum creatine kinase (CK) and myocardial band of CK (CK-MB) measurements were collected in all patients during their stay in our hospital. The first analysis was performed before or during the primary PCI. At the coronary care unit, blood samples were taken following a standardized protocol at 3, 6, 9, 12, 18, 24, 36, and 48 hours after primary PCI. The infarct size was defined as the maximum level of CK and CK-MB that was measured.

Follow-Up
Follow-up data at 30 days after primary PCI were collected from hospital records, written questionnaires, and telephone interviews. Reinfarction was defined as the onset of recurrent symptoms of ischemia combined with new ST-segment elevations and/or a second increase of serum CK or CK-MB to at least twice the upper limit of the normal range. Target vessel revascularization was defined as PCI or bypass grafting of the infarct-related coronary artery.

Side Effects
In previous studies, side effects of adenosine have been documented during and after intravenous administration.4-21 In this study, side
effects were registered by the interventional cardiologist during and directly after infusion of the bolus intracoronary adenosine or placebo. Side effects specifically documented were flushing, hypotension, bradycardia, ventricular tachycardia, bronchospasm, first- or second-degree atrioventricular block, dyspnea, and chest pain.

Sample Size and Statistical Analysis
The number of patients included in this study was based on previous observations of residual ST-segment deviation after primary PCI, in which 53.1% of patients had residual ST-segment deviation <0.2 mV. To detect an increase of 25% of residual ST-segment deviation <0.2 mV in patients treated with intracoronary adenosine, with 80% power and a 2-sided significance level of 0.05, a total number of 450 patients were required.

We performed an analysis of data by intention to treat. Categorical variables are presented as frequency values and proportions and were compared by the χ² test or Fisher exact test. Continuous normally distributed variables are presented as mean values and standard deviations and were compared with the 2-tailed Student t test. For skewed distributed variables, median values with interquartile range (IQR) are shown, and the variables were compared with the use of the Mann–Whitney U test. For all analyses, P values of <0.05 were defined as significant. Statistical analysis was performed using the Statistical Package for Social Sciences version 16.0 (SPSS Inc., Chicago, Ill) 16.0. The authors had full access to the data and take responsibility for its integrity. All authors have read and agree to the manuscript as written.

Results
A total of 507 patients were diagnosed as STEMI and candidates for primary PCI in our hospital between May 2007 and July 2008. After the initial angiography, 448 patients met the inclusion criteria and were randomized to high-dose intracoronary adenosine (N=226) or intracoronary placebo (N=222; Figure 1). Baseline and procedural characteristics did not significantly differ between patients randomized to intracoronary adenosine and intracoronary placebo, except for diastolic blood pressure before PCI (75.9±13.9 versus 73.1±13.6, P=0.033; Table 1). A detailed analysis of the site of the infarct-related coronary lesion showed that the myocardial territories at risk were comparable in size in patients randomized to intracoronary adenosine and intracoronary placebo. Thrombolysis in myocardial infarction flow 0 or 1 was present in 142 of 226 patients (62.9%) randomized to intracoronary adenosine and in 130 of 222 patients (58.6%) randomized to placebo (P=NS).

Myocardial Reperfusion
In 415 of 448 patients (92.6%) residual ST-segment deviation could be assessed on the ECG after primary PCI. The median time from the start of the PCI procedure to the ECG made after the procedure was 44 minutes (IQR, 35 to 70 minutes). Residual ST-segment deviation <0.2 mV was observed in 96 of 208 patients (46.2%) randomized to intracoronary adenosine and in 108 of 207 patients (52.2%) randomized to intracoronary placebo (P=NS; Figure 2). In addition, ST-segment elevation resolution >70% was present in 129 of 189 patients (68.3%) randomized to intracoronary adenosine and in 128 of 193 patients (66.3%) randomized to intracoronary placebo (P=NS). Myocardial blush grade 3 was observed in 64 of 224 patients (28.6%) randomized to intracoronary adenosine compared with 77 of 218 patients (35.3%) randomized to intracoronary placebo (P=NS).

Quantitative Blush Evaluation
Paired angiograms performed after stenting and directly after the second bolus intracoronary adenosine or placebo were available for 318 patients (71.0%). The median time interval between the two angiograms was 56 seconds (IQR, 46 to 73 seconds). After stenting, the median QuBE value was 11.3 (IQR, 8.4 to 14.3) in patients randomized to adenosine and 11.8 (IQR, 8.3 to 15.0) in patients randomized to placebo (P=NS; Table 2). After PCI, distal embolization was observed in 27 of 223 patients (12.1%) randomized to intracoronary adenosine and in 19 of 220 patients (8.6%) randomized to intracoronary placebo (P=NS).

Biomarkers Reflecting Infarct Size
Enzymatic infarct size could be analyzed in 446 of 448 patients. The median number of measurements was 6 (IQR, 5

Figure 1. Flow diagram: randomization, treatment, and angiographic recordings during primary PCI. CABG indicates coronary artery bypass grafting; i.c., intracoronary; MBG, myocardial blush grade; nitro, nitroglycerine.

Angiographic Results
After primary PCI, thrombolysis in myocardial infarction flow 3 was present in 212 of 225 patients (94.2%) randomized to intracoronary adenosine and in 208 of 222 patients (93.7%) randomized to placebo (P=NS; Table 2). After PCI, distal embolization was observed in 27 of 223 patients (12.1%) randomized to intracoronary adenosine and in 19 of 220 patients (8.6%) randomized to intracoronary placebo (P=NS).
The maximum measurement of CK was 1213 U/L (IQR, 571 to 2720) in patients randomized to intracoronary adenosine and 1190 U/L (IQR, 478 to 2163) in patients randomized to intracoronary placebo (P/H11005 NS; Table 2). Time to maximum CK value was 6.6 hours (IQR, 3.7 to 10.0) in patients randomized to intracoronary adenosine and 7.9 hours (IQR, 5.6 to 11.8) in patients randomized to intracoronary placebo (P=NS). In addition, we did not observe a significant difference in maximum value of CK-MB (176 [IQR, 85 to 305] versus 153 [IQR, 62 to 268]). Time to maximum CK-MB was 6.2 hours (IQR, 3.3 to 9.0) versus 6.9 hours (IQR, 3.9 to 10.0) (P=NS).

### Side Effects

During and directly after administration of intracoronary adenosine or placebo, side effects were seen more frequently in patients randomized to intracoronary adenosine compared with intracoronary placebo (Table 3). Hypotension occurred in 24 of 168 patients (14.3%) randomized to intracoronary adenosine and in 2 of 160 patients (1.3%) randomized to intracoronary placebo (P/H11005 0.001). Furthermore, transient first-degree and second-degree atrioventricular block were seen in, respectively, 16 of 166 patients (9.6%) and 31 of 168 patients (18.5%) randomized to intracoronary adenosine, compared with both 2 of 159 patients (1.3%) randomized to intracoronary placebo (P=0.001 and P<0.001). All side effects disappeared within 2 to 3 minutes, and no clinical sequelae were observed.
Table 2. Outcomes of Patients Randomized to Intracoronary Adenosine or Placebo

<table>
<thead>
<tr>
<th>Outcome Characteristics</th>
<th>Adenosine (N=226)</th>
<th>Placebo (N=222)</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>Angiogram post-PCI, n/N (%)</td>
<td>2/222 (0.9)</td>
<td>1/222 (0.5)</td>
<td>NS</td>
</tr>
<tr>
<td>TIMI flow post-PCI</td>
<td>11/225 (4.9)</td>
<td>13/222 (5.9)</td>
<td>NS</td>
</tr>
<tr>
<td>DE post-PCI</td>
<td>2/223 (12.1)</td>
<td>19/220 (8.6)</td>
<td>NS</td>
</tr>
<tr>
<td>ECG post-PCI, n/N (%)</td>
<td>168/211 (79.6)</td>
<td>166/208 (78.9)</td>
<td>NS</td>
</tr>
<tr>
<td>Enzymatic infarct size (IQR)</td>
<td>31/223 (12.1)</td>
<td>208/222 (93.7)</td>
<td>NS</td>
</tr>
</tbody>
</table>

NS indicates not significant; DE, distal embolization; TIMI, Thrombolysis in Myocardial Infarction; TVR, target vessel revascularization.

Clinical Outcome at 30 Days

At 30-day follow-up, 3 patients (1.3%) randomized to intracoronary adenosine had died compared with 2 patients (0.9%) randomized to intracoronary placebo (P=NS; Table 2). Reinfarction occurred in, respectively, 3 (1.3%) and 1 (0.5%) patient. Target vessel revascularization was performed in 8 patients (3.6%) randomized to adenosine and in 7 patients (3.2%) randomized to placebo (P=NS).

Discussion

This prospective randomized controlled trial demonstrated that intracoronary administration of high-dose adenosine after thrombus aspiration and after stenting did not improve myocardial perfusion in patients with STEMI. The incidence of residual ST-segment deviation <0.2 mV, ST-segment elevation resolution, thrombolysis in myocardial infarction flow after PCI, myocardial blush grade, enzymatic infarct size, and clinical outcome at 30 days after PCI did not significantly differ between patients randomized to intracoronary adenosine or intracoronary placebo. Even with QuBE, a quantitative measure of myocardial perfusion, no effect of intracoronary adenosine administration was observed in the myocardium related to the target coronary artery.

In AMISTAD-II, the effect of intravenous administration of adenosine in patients with STEMI undergoing primary PCI or thrombolysis was investigated and did not show an improvement on clinical outcome.8 Furthermore, Micari et al23 did not find an improvement in microvascular reflow measured by myocardial contrast echocardiography directly after PCI, but the infarct size was smaller in the patients treated with intravenous adenosine at 3 to 5 days after PCI. Both studies suggested that the beneficial effect of adenosine on infarct size depended on whether time to reperfusion was <3 to 4 hours after the onset of ischemic symptoms.9,23

Several retrospective and prospective clinical studies with intracoronary administration of adenosine have shown favorable effects during primary PCI. These studies documented that intracoronary infusion of adenosine resulted in an increased coronary blood flow,10 a reduction of no reflow,11 and an improvement of signs of myocardial reperfusion on the ECG after PCI.12,13 However, these favorable results should be interpreted with caution because these results are limited by a small sample size, potential selection bias, or a nonrandomized trial design.

The physiological effects of adenosine are multifactorial and still not fully understood. Adenosine is endogenously produced by dephosphorylation of adenosine monophosphate, mainly by the cardiac myocytes during myocardial ischemia. Within a few seconds of ischemia, levels of interstitial adenosine increase and cause arteriolar vasodilation, depending on the degree of ischemia.24 In addition to vasodilatation, experimental studies have documented that...
adenosine has a protective effect on many mechanisms involved in myocardial ischemia and infarction, such as inhibition of platelet aggregation, inflammatory cell activation, generation of oxygen free radicals, and decreasing cellular calcium overload.5

Why does high-dose intracoronary adenosine not improve myocardial perfusion in patients with STEMI treated with primary PCI? The first explanation may be that, because of the high level of endogenous production of adenosine during myocardial ischemia, maximal vasodilatation was already present in our patients. In animal models, it has been described that levels of adenosine may increase to levels producing maximal coronary arteriolar dilatation during ischemia.25 Therefore, our study may be “negative” because of the absence of vasodilatory reserve during and after reperfusion by PCI.

Second, the dose of the bolus intracoronary adenosine may have been inadequate to induce maximal vasodilatation. However, flow measurements have shown that near-maximal vasodilatation is achieved with a dose of 16 μg intracoronary adenosine in humans.26 In a more recent study, doses >100 μg intracoronary adenosine are suggested as adequate for fractional flow reserve measurements.27 Compared with the recommended bolus intracoronary adenosine of 30 to 60 μg in STEMI guidelines,14 it can be concluded that the intracoronary dose in this study was sufficient to achieve a maximal treatment effect. Higher cumulative doses have been published, but only in trials that used continuous intracoronary infusions. Nevertheless, this may well in part explain the difference between our findings and those of Marzilli et al10 and Stoel et al13 Contrariwise, the dose of intracoronary adenosine may have been too high, leading to side effects in 20% of patients. However, all side effects occurred during or immediately after infusion of intracoronary adenosine and disappeared within 2 to 3 minutes without clinical sequelae.

As a third explanation, it can be discussed whether adenosine infusion was performed in the right time window. Previous studies have described that the beneficial effect of adenosine is limited to the first 3 to 4 hours after onset of ischemic symptoms.9,23 As the median time to reperfusion in this study was 170 minutes, it is clear that we have enrolled many patients within this time window.

Finally, in the contemporary treatment of patients with STEMI, the systematic performance of thrombus aspiration and the administration of the glycoprotein IIb/IIIa inhibitor abciximab during primary PCI has resulted in an improvement of myocardial reperfusion.22,28 Thrombus aspiration and abciximab were not systematically used in previous studies. Compared with previous trials, the contemporary treatment may have left limited opportunities for further improvement of myocardial perfusion.

**Limitations**

In this study, several limitations should be recognized. Randomization to adenosine or placebo was performed after the initial coronary angiogram, and the interventional cardiologists were aware of administrating a bolus intracoronary adenosine or placebo. In addition, the study was powered on a functional end point, making the number of patients too small for interpretation of clinical end points. Furthermore, quantitative evaluation of the immediate perfusion effect of adenosine or placebo with the QuBE program was performed in only 71% of patients. Because the QuBE program was developed during the initial months of the inclusion period of ADenosine Administration during and after Primary percutaneous coronary intervention in acute myocardial infarction Trial, interventional cardiologists became experienced in systematically performing paired analyzable angiograms during the first months of inclusion. Finally, the timing of intracoronary adenosine infusion may not have been optimal in this study. It has been suggested previously that the administration of adenosine to the ischemic myocardium before reperfusion therapy has a maximal beneficial effect on ischemia myocardial injury.29 Because intracoronary adenosine was administered directly after the performance of thrombus aspiration, circulating levels of exogenous adenosine were not elevated in the distal microvasculature before reperfusion.

**Conclusions and Implications**

Intracoronary administration of high-dose adenosine as adjunctive therapy to primary PCI, including thrombus aspiration and abciximab, did not improve myocardial perfusion in patients with STEMI. In addition, no reperfusion effect could be measured directly after infusion of adenosine or placebo, suggesting that no vasodilatory reserve is present during myocardial ischemia. Further improvements in myocardial reperfusion should focus on pharmacological therapies with other modes of action.

**Disclosures**

None.

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**CLINICAL PERSPECTIVE**

Primary percutaneous coronary intervention (PCI) is the preferred treatment to achieve reperfusion of the infarct-related coronary artery in patients with ST-elevation myocardial infarction. However, impaired myocardial perfusion is often seen after primary PCI. Previous studies have suggested that the administration of intravenous adenosine may improve myocardial perfusion and reduce infarct size. The effect of intracoronary adenosine during primary PCI has not been investigated in a large randomized trial. In this study, 448 consecutive patients presenting with ST-elevation myocardial infarction were randomized to 2 bolus injections of intracoronary adenosine (2×120 μg in 20 mL NaCl) or placebo (2×20 mL NaCl). The first bolus injection was given after thrombus aspiration and the second after stenting of the infarct-related artery. The administration of intracoronary adenosine as adjunctive therapy during primary PCI did not result in an improvement of myocardial perfusion, as assessed on coronary angiogram (myocardial blush grade) and ECG (ST-segment elevation resolution and residual ST-segment deviation), or enzymatic infarct size. No myocardial perfusion effect could be measured directly after infusion of adenosine or placebo, suggesting that no endothelial-dependent vasodilatory reserve is present during myocardial ischemia and reperfusion by PCI. In addition, the contemporary treatment of patients with ST-elevation myocardial infarction, including the systematic performance of thrombus aspiration and the administration of abciximab, may have left limited opportunities for further improvement of myocardial perfusion. Therefore, for further improvement of outcome after primary PCI for ST-elevation myocardial infarction, we should focus on pharmacological therapies with other modes of action.
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