Selective Intracoronary Administration of Nitroprusside Before Balloon Dilatation Prevents Slow Reflow During Percutaneous Coronary Intervention in Patients With Acute Myocardial Infarction

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SUMMARY

Background: Previous studies have shown that intracoronary nitroprusside injection is safe and effective after slow reflow complicates percutaneous coronary intervention (PCI).

Objectives: We sought to determine the safety and efficacy of selective intracoronary administration of nitroprusside through the drug delivery catheter before balloon dilatation to prevent no or slow reflow during PCI for acute myocardial infarction (AMI).

Methods: We studied 120 consecutive patients with AMI treated by PCI. In 60 patients (nitroprusside group), nitroprusside (120 µg) was selectively administered through the drug delivery catheter into the distal coronary artery to reach the target lesion before balloon dilatation. Clinical and angiographic data, as well as in-hospital outcomes, of the nitroprusside group were retrospectively compared with 60 patients who had conventional PCI without nitroprusside (control group).

Results: There were no significant differences in the baseline clinical and angiographic characteristics between the 2 groups. Compared to the control group, the nitroprusside group had 1) less slow reflow during the procedure (12% versus 35%, P = 0.0025), 2) a shorter fluoroscopic time (14.4 ± 7.9 versus 18.7 ± 9.1 minutes, P = 0.0093), 3) a shorter procedure time (57.6 ± 20.6 versus 78.1 ± 26.4, P < minutes, P < 0.0001), 4) a better final TIMI flow grade (III:I:I:0 = 59:1:0:0 versus 53:6:1:0, P = 0.0284), 5) a better blush grade (III:I:I:0 = 49:10:1:0 versus 33:15:8:4, P = 0.0006), and 6) a better corrected TIMI coronary flame count (30.8 ± 13.7 versus 46.5 ± 44.7, P = 0.0102). There were no particular complications with nitroprusside use.

Conclusions: The selective intracoronary administration of nitroprusside prior to PCI is safe and well tolerated, prevents no or slow reflows, and improves reperfusion of the infarcted myocardium. (Int Heart J 2007; 48: 423-433)

Key words: Nitroprusside, Slow reflow, No reflow, Primary angioplasty, Percutaneous coronary intervention, Acute myocardial infarction
Percutaneous coronary intervention (PCI) for acute myocardial infarction (AMI) has been shown to improve acute and long-term results.\(^1\)\(^-\)\(^3\) Thus, PCI has become the standard therapy for AMI. However, in some patients, after the epicardial coronary occlusion has been resolved, the blood flow may cease or slow down dramatically. This phenomenon is called no reflow or slow reflow. No reflow or slow reflow is a serious complication of PCI performed for AMI that increases mortality and decreases left ventricular functional recovery.\(^4\)\(^-\)\(^6\) Furthermore, this phenomenon is also linked to ventricular arrhythmias, early congestive heart failure, and even cardiac rupture.\(^6\) For these reasons, it is very important to prevent no reflow or slow reflow during PCI for AMI.

The exact mechanisms that underlie the no reflow or slow reflow phenomena are not known. However, there is considerable evidence suggesting that these phenomena are mainly due to dysfunction of the microcirculation and the presence of vasospasm at the level of the resistance arterioles.\(^7\)\(^-\)\(^9\) Therefore, it is thought that improving the microcirculation would be a very useful strategy for dealing with these phenomena.

Nitric oxide strongly dilates the resistance arterioles and plays a significant role in coronary blood flow through the microcirculation.\(^10\)\(^,\)\(^11\) Nitroprusside is a direct donor of nitric oxide.\(^12\) It has been shown that intracoronary nitroprusside injection is a safe and effective technique for managing the slow reflow phenomenon once it occurs during coronary intervention.\(^13\)\(^-\)\(^15\) Therefore, we postulated that selective intracoronary administration of nitroprusside prior to balloon dilation could prevent no reflow or slow reflow. Thus, we compared the incidence of no reflow and slow reflow and microcirculation damage, as evaluated by myocardial blush grade and the corrected TIMI frame count (CTFC), in patients with AMI undergoing PCI with or without nitroprusside pretreatment.

**Methods**

**Study population:** Consecutive patients with AMI who had a PCI without nitroprusside from May 2003 to December 2004 (control group, \(n = 60\)) and those with nitroprusside from December 2004 to April 2006 (nitroprusside group, \(n = 60\)) were included. The criteria for AMI were 1) continuous chest pain that lasted > 30 minutes, 2) arrival at our hospital within 12 hours of the onset of chest pain, 3) ST-segment elevation \(\geq 0.1\) mV in 2 or more contiguous leads on the 12-lead ECG, 4) angiographic detection of a culprit lesion with diameter stenosis \(\geq 70\%\) and/or TIMI flow grade \(\leq 2\), and 5) the culprit lesion was suitable for PCI. Exclusion criteria included 1) a culprit lesion in the left main coronary artery, small branch, distal artery, or bypass graft, and 2) cardiogenic shock. Written informed consent was obtained from each subject with nitroprusside after a detailed expla-
nation of the purpose and procedures of this study. The protocol was approved by
the Institutional Review Board of Naganoken Koseiren Shinonoi General Hospi-
tal.

**Angiographic evaluations and interventional procedure:** Prior to the intervention,
all patients were given 200 mg of aspirin orally. Heparin was given intravenously
as a bolus dose of 8000 IU at the beginning of the procedure and later as required
to maintain the activated clotting time > 300 seconds. Thrombolysis was not per-
formed in any patients. Glycoprotein IIb/IIIa platelet inhibitors and clopidogrel
were not administered since they were not yet approved in Japan. Intracoronary
isosorbide dinitrate 2.5-5.0 mg was administered immediately prior to baseline
angiography and at the time of the postprocedure final angiography.

Five experienced operators performed the PCI procedures via either the
femoral (n = 5 in control group, n = 3 in nitroprusside group) or radial approach
using a 6Fr guiding catheter. The obstruction of the infarct-related artery was
crossed with a 0.014 inch guidewire, and the drug delivery catheter (FC catheter,
Tokai Medical Products, Kasugai-city, Japan) was positioned at the level of the
distal coronary artery to the target lesion. The drug delivery catheter was a flexi-
ble, multilumen catheter with side holes in the distal segment and was designed
for single-operator exchanges. Within the catheter shaft, the drug delivery lumen
and the guidewire lumen are separate, and the structure of the catheter allows the
guidewire to pass through the distal tip; the drug inserted in the catheter hub can
be released from the side hole of the distal side of the catheter. The catheter can
be used for controlled and selective infusion of solutions into target vessels. In the
nitroprusside group, nitroprusside (120 µg, diluted to 24 µg/mL) was selectively
hand-injected into the distal vascular bed to the target lesion through the drug
delivery catheter before PCI. Since the half-life of nitroprusside is several min-
utes, we repeatedly gave nitroprusside with every dilatation. In patients who
developed slow reflow despite the use of nitroprusside, an additional 120 µg
nitroprusside was given as soon as possible through the drug delivery catheter.
The PCI procedure was conducted according to standard techniques. The choice
of angioplasty, stent delivery balloon, and stent design was made by the operator
during the procedure based on a visual assessment of the reference diameter of
the vessel in which the procedure was being carried out. Intravascular ultrasound
was not available to help select balloon or stent sizes. Procedural success was
defined as the presence of less than a 20% residual stenosis postprocedure. After
the procedure, aspirin was continued indefinitely, and ticlopidine was prescribed
for patients in whom a coronary stent had been inserted.

Coronary angiograms were reviewed by two experienced interventional card-
iodiologists who were blinded to the treatment received. The antegrade flow in the
culprit vessel was determined according to the TIMI grading system, which was
assessed as previously described. Slow reflow during the procedure was defined as a decrease of $\geq 1$ TIMI flow grade after an initial successful reperfusion of the culprit artery in the absence of anatomic vessel stenosis or obstruction, flow-limiting dissection, spasm, or thrombus.

**Postprocedural assessment:** Myocardial blush grade was assessed as previously reported: grade 0, no myocardial blush or contrast density; grade 1, minimal myocardial blush or contrast density; grade 2, moderate myocardial blush or contrast density but less than that obtained during angiography of a contralateral or ipsilateral noninfarct-related coronary artery; and grade 3, normal myocardial blush or contrast density comparable with that obtained during angiography of a contralateral or ipsilateral noninfarct-related coronary artery.

The TIMI frame count method was also used to measure the coronary flow based on previously reported guidelines. The corrected TIMI frame count (CTFC) was calculated by dividing the left anterior descending artery frame count by 1.7. The frame rate of a digital cardiac image was calculated as 30 frames/s.

These analyses were independently assessed by two experienced interventional cardiologists blinded to patient data.

Creatine kinase (CK) was assessed every 6 hours during the first day of admission and then every day for 3 days.

**Statistical analysis:** Continuous variables are expressed as the mean $\pm$ standard deviation (SD), while discrete variables are presented as absolute values, percentages, or both. Continuous variables were compared with Student's $t$-test or the Mann-Whitney U test. The chi-square test or Fisher's exact test was used to compare rates of discrete variables. Results were considered statistically significant at $P \leq 0.05$.

**RESULTS**

**Baseline clinical and angiographic characteristics:** The clinical and angiographic characteristics are presented in Table I. The 2 groups were similar in age, gender distribution, incidence of coronary risk factors, and recanalization time from symptom onset. As well, target vessels and pre-TIMI flow grade were similar in the 2 groups.

**Procedural characteristics:** The procedural characteristics in the 2 groups are shown in Table II. Injection of nitroprusside selectively into the distal coronary artery was well tolerated and was free of any side effects. Some patients experienced transient hypotension for several minutes, but prolonged hypotension was not noted. The incidence of vasopressor use during the procedure was similar in both groups. In the control group, the incidence of using a distal protection device
was significantly higher than in the nitroprusside group. On the other hand, the incidence of using an aspiration device was similar in both groups. In the nitroprusside group, the incidence of slow reflow during the procedure was significantly less, and the procedure time and the fluoroscopic time were significantly shorter than in the control group. Intra-aortic balloon pumping was used in one patient in the nitroprusside group and in 2 patients in the control group. **Postprocedural analysis:** The PCI procedures were successful in all patients in both groups. There were no particular complications associated with the use of

<table>
<thead>
<tr>
<th>Table I. Baseline Clinical and Angiographic Characteristics</th>
<th>Nitroprusside group</th>
<th>Control group</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age</td>
<td>69 ± 11 (%)</td>
<td>69 ± 11</td>
<td>0.802</td>
</tr>
<tr>
<td>Male sex, %</td>
<td>37 (62%)</td>
<td>45 (75%)</td>
<td>0.1164</td>
</tr>
<tr>
<td>Diabetes mellitus, %</td>
<td>25 (42%)</td>
<td>19 (32%)</td>
<td>0.2557</td>
</tr>
<tr>
<td>Hypertension, %</td>
<td>36 (60%)</td>
<td>34 (57%)</td>
<td>0.7111</td>
</tr>
<tr>
<td>Hyperlipidemia, %</td>
<td>22 (37%)</td>
<td>20 (33%)</td>
<td>0.7019</td>
</tr>
<tr>
<td>Current smoker, %</td>
<td>22 (37%)</td>
<td>27 (45%)</td>
<td>0.3531</td>
</tr>
<tr>
<td>Recanalization time from onset, hours</td>
<td>4.8 ± 3.6</td>
<td>5.1 ± 4.6</td>
<td>0.6582</td>
</tr>
<tr>
<td>Target vessel, n</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>LAD/RCA/LCX</td>
<td>32/23/5</td>
<td>26/27/7</td>
<td>0.5289</td>
</tr>
<tr>
<td>Pre TIMI flow grade, n</td>
<td>41/11/7/1</td>
<td>42/10/7/1</td>
<td>0.8668</td>
</tr>
</tbody>
</table>

LAD indicates left descending coronary artery; RCA, right coronary artery; LCX, left circumflex coronary artery; and TIMI, Thrombolysis In Myocardial Infarction.

<table>
<thead>
<tr>
<th>Table II. Procedural Characteristics</th>
<th>Nitroprusside group</th>
<th>Control group</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>Stent use, %</td>
<td>59 (%)</td>
<td>57 (%)</td>
<td>0.3091</td>
</tr>
<tr>
<td>Distal protection device use, %</td>
<td>4 (7%)</td>
<td>19 (32%)</td>
<td>0.005</td>
</tr>
<tr>
<td>Aspiration device use, %</td>
<td>37 (62%)</td>
<td>40 (67%)</td>
<td>0.5679</td>
</tr>
<tr>
<td>Vasopressor use during procedure, %</td>
<td>8 (13%)</td>
<td>10 (17%)</td>
<td>0.6091</td>
</tr>
<tr>
<td>Slow flow during procedure, %</td>
<td>7 (12%)</td>
<td>21 (35%)</td>
<td>0.0025</td>
</tr>
<tr>
<td>Procedure time, minutes</td>
<td>57.6 ± 20.6</td>
<td>78.1 ± 26.4</td>
<td>&lt; 0.0001</td>
</tr>
<tr>
<td>Fluoroscopic time, minutes</td>
<td>14.4 ± 7.9</td>
<td>18.7 ± 9.1</td>
<td>0.0093</td>
</tr>
<tr>
<td>Post-TIMI flow grade, n</td>
<td>0/1/2/3</td>
<td>0/0/1/59</td>
<td>0/1/6/53</td>
</tr>
<tr>
<td>Postblush grade, n</td>
<td>0/1/10/49</td>
<td>4/8/15/33</td>
<td>0.0006</td>
</tr>
<tr>
<td>Post-CTFC</td>
<td>30.8 ± 13.7</td>
<td>46.5 ± 44.7</td>
<td>0.0102</td>
</tr>
</tbody>
</table>

TIMI indicates Thrombolysis In Myocardial Infarction and CTFC, corrected TIMI frame count.
nitroprusside. The post-TIMI blush grade in the nitroprusside group was significantly better than in the control group (III:II:I:0 = 49:10:1:0 versus 33:15:8:4, \( P = 0.0006 \)). In the nitroprusside group, the post-CTFC was significantly lower than in the control group (30.8 ± 13.7 versus 46.5 ± 44.7, \( P = 0.0102 \)). These beneficial effects were also shown in each target vessel even though most of the values were not significant (Table III).

None of the following differences was statistically significant. The peak creatine kinase level was 2875 ± 2214 IU/L in the nitroprusside group and 3332 ± 2567 IU/L in the control group (\( P = 0.3039 \)). Two patients in the control group were reoccluded after the procedure during the hospital admission, while no patients in the nitroprusside group experienced reocclusion. One patient in the nitroprusside group and 2 patients in the control group died in hospital due to heart failure; all 3 of these patients experienced slow reflow during the procedure. The remaining patients experienced no major adverse cardiovascular events, including reinfarction, emergent bypass surgery, and stroke.

**DISCUSSION**

The slow or no reflow phenomenon continues to be a serious complication in PCI performed for AMI and results in poor short- and long-term outcomes.1-6) In our study, 35% of the patients who had standard PCI developed slow reflow during the procedure; selective intracoronary nitroprusside before balloon dilatation safely and effectively prevented slow reflow.

<table>
<thead>
<tr>
<th>Postprocedural Characteristics in Each Target Vessel</th>
<th>Nitroprusside group</th>
<th>Control group</th>
<th>( P )</th>
</tr>
</thead>
<tbody>
<tr>
<td>Post-TIMI flow grade, ( n )</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>LAD, 0/1/2/3</td>
<td>0/0/1/31</td>
<td>0/1/2/23</td>
<td>0.2063</td>
</tr>
<tr>
<td>RCA, 0/1/2/3</td>
<td>0/0/0/23</td>
<td>0/0/2/25</td>
<td>0.1872</td>
</tr>
<tr>
<td>LCX, 0/1/2/3</td>
<td>0/0/0/5</td>
<td>0/0/2/5</td>
<td>0.2100</td>
</tr>
<tr>
<td>All, 0/1/2/3</td>
<td>0/0/1/59</td>
<td>0/1/6/53</td>
<td>0.0284</td>
</tr>
<tr>
<td>Postblush grade, ( n )</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>LAD, 0/1/2/3</td>
<td>0/1/4/27</td>
<td>2/3/3/18</td>
<td>0.1198</td>
</tr>
<tr>
<td>RCA, 0/1/2/3</td>
<td>0/0/5/18</td>
<td>2/3/10/12</td>
<td>0.0086</td>
</tr>
<tr>
<td>LCX, 0/1/2/3</td>
<td>0/0/1/4</td>
<td>0/2/2/3</td>
<td>0.1697</td>
</tr>
<tr>
<td>All, 0/1/2/3</td>
<td>0/1/10/49</td>
<td>4/8/15/33</td>
<td>0.0006</td>
</tr>
<tr>
<td>Post-CTFC</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>LAD</td>
<td>25.6 ± 9.9</td>
<td>31.7 ± 15.4</td>
<td>0.0723</td>
</tr>
<tr>
<td>RCA</td>
<td>36.8 ± 14.3</td>
<td>65.0 ± 60.5</td>
<td>0.0337</td>
</tr>
<tr>
<td>LCX</td>
<td>36.4 ± 20.2</td>
<td>30.3 ± 7.5</td>
<td>0.4745</td>
</tr>
<tr>
<td>All</td>
<td>30.8 ± 13.7</td>
<td>46.5 ± 44.7</td>
<td>0.0102</td>
</tr>
</tbody>
</table>

TIMI indicates Thrombolysis In Myocardial Infarction and CTFC, corrected TIMI frame count.
The exact mechanisms that underlie the no reflow or slow reflow phenomena are not known. However, there is considerable evidence suggesting that these phenomena are mainly due to microcirculatory dysfunction and vasospasm that occurs at the level of the resistance arterioles. Nitric oxide is a potent vasodilator in the resistance arteriolar circulation and plays a significant role in coronary blood flow through the microcirculation. In patients with slow or no reflow, nitroglycerin used to be injected into the coronary artery. To derive nitric oxide, nitroglycerin has to be metabolized by the vascular wall. However, unlike large nonresistance vessels, resistance arterioles are unable to metabolize nitroglycerin into nitric oxide. Therefore, nitroglycerin cannot be expected to play an effective role in the microcirculation. On the other hand, nitroprusside is a direct donor of nitric oxide and requires no intracellular metabolism to derive nitric oxide. It has been shown that intracoronary nitroprusside can produce coronary hyperemia. Calcium channel blockers and adenosine are two of the most common intracoronary agents used to induce hyperemia. Based on previous reports using a Doppler flow wire, the hyperemia induced by both nitroprusside and adenosine greatly exceeded the hyperemia induced by calcium channel blockers such as diltiazem, verapamil, and nicardipine. Furthermore, intracoronary nitroprusside produces an equivalent but more prolonged coronary hyperemia than adenosine. Therefore, we postulated that nitroprusside would have a beneficial effect on the slow reflow phenomenon. In fact, some previous studies have shown the positive effects of intracoronary nitroprusside injection on the no reflow phenomenon in patients not only with angina pectoris but also with AMI. However, our study differs from previous studies in a few respects. First, in the previous studies, nitroprusside was given after the appearance of slow or no reflow, whereas we administered nitroprusside before balloon dilatation prior to the appearance of slow or no reflow. Consequently, we could reduce the incidence of slow reflow. Second, in the previous studies, nitroprusside was injected into the coronary artery mainly through the guiding catheter, whereas we injected nitroprusside through the drug delivery catheter into the distal coronary artery to reach the occluded target lesion. If we had injected nitroprusside through the guiding catheter, the nitroprusside might have primarily reached the aortic root and the nonoccluded vessels. Therefore, we gave nitroprusside through the drug delivery catheter to deliver a sufficient amount of nitroprusside into the distal coronary artery to reach the occluded lesion. Given our results, our delivery method appears to be more effective than those previously reported.

In patients with AMI, the primary objective of reperfusion therapies is not only the restoration of blood flow in the epicardial coronary artery but also the complete and sustained reperfusion of the infarcted myocardium. Both the myocardial blush grade and the TIMI frame count have been used to describe myocard-
dial reperfusion in patients with restored patency of the infarct-related coronary artery. In AMI patients after PCI, the myocardial blush grade, which is the angiographic evidence of myocardial perfusion, has been shown to be related to the extent of ST-segment elevation resolution, enzymatic infarct size, nonreperfusion as defined by myocardial contrast echocardiography, left ventricular function, and long-term mortality.\textsuperscript{17,22-26} Stone, et al demonstrated that the one-year cumulative mortality was 6.8\% with a final blush score of 3, 13.2\% with a blush score of 2, and 18.3\% with a blush score of 1 or 0 ($P = 0.004$).\textsuperscript{26} We found that the final blush grade in the nitroprusside group was significantly better than in the control group. In AMI patients, the TIMI frame count after thrombolytic therapy or PCI has been shown to be an independent predictor of functional recovery, as well as in-hospital and long-term clinical outcome.\textsuperscript{18,25,27,28} According to Gibson, et al, in-hospital mortality increased from 0.0\% with a 90-minute CTFC after thrombolytic administration of 0-13 frames, to 2.7\% with a count of 14 to 40, and to 6.4\% with a count > 40 ($P = 0.003$).\textsuperscript{27} Our study indicates that selective intracoronary nitroprusside administration significantly improves the CTFC. In particular, we did not observe a CTFC of < 20 in the control group (Figure); there were significantly more patients with a CTFC > 40 in the control group than in the nitroprusside group ($n = 23$ versus 12, $P = 0.0272$). Given our results, one can expect that administering nitroprusside prior to PCI will improve not only the short-term results but also long-term outcomes.

Furthermore, in our study, selective intracoronary nitroprusside administration was safe and well-tolerated. Nitroprusside is a potent vasodilator, and the intravenous systemic administration of nitroprusside can sometimes decrease blood pressure dramatically. However, the dosage used in this study did not cause severe or prolonged hypotension that could trigger a shock state. This finding supports the previous data.\textsuperscript{13-15,20} If hypotension were to occur, the short half-life of nitroprusside protects against prolonged hypotension.

However, the use of nitroprusside did not totally prevent slow reflow. Other factors that could not be reversed by nitroprusside administration might contribute to the pathogenesis of the slow reflow phenomenon. In particular, nitroprusside has no effect on a large quantity of thrombus. If a large thrombus is present, the aspiration device must be used with nitroprusside. However, the distal protection device is not routinely necessary during PCI for all AMI patients. Previously, we had routinely used the distal protection device (PercuSurge, Medtronic, Minneapolis, USA). However, we currently do not use this device routinely, since the Enhanced Myocardial Efficacy and Recovery by Aspiration of Liberated Debris (EMERALD) trial failed to show its effectiveness in AMI patients.\textsuperscript{29} In our study, although the incidence of the distal protection device use in the nitroprusside group was significantly less than in the control group, the incidence of slow
reflow during the procedure was significantly less in the nitroprusside group than in the control group. In order to prevent slow reflow, selective intracoronary administration of nitroprusside before balloon dilatation may be more effective than the distal protection device.

Study limitations: The first limitation of this study is that it was a single-center, nonrandomized, retrospective study with a relatively small number of patients. Larger prospective randomized, multicenter studies are necessary to determine the role of selective intracoronary administration of nitroprusside before balloon dilatation. Second, glycoprotein IIb/IIIa inhibitors were not used in any of the study patients because they were not approved for clinical use in Japan. Platelet aggregates could play a significant role in the no reflow phenomenon, which may be related in part to microemboli. A previous study found that glycoprotein IIb/IIIa inhibitors reduced the incidence of no reflow. Had we been able to use glycoprotein IIb/IIIa inhibitors, we might have had different results. Third, although ST-segment resolution and myocardial contrast echocardiography results after coronary reperfusion are known to be independent predictors of functional recovery and in-hospital and long-term clinical outcome, we did not assess these...
parameters. Had we analyzed these parameters, we might have been able to obtain more complete data with respect to the improvement of reperfusion of the infarcted myocardium. Finally, we did not analyze the left ventricular size or function in the convalescent stage. In some patients left ventriculography or echocardiography was performed several days after PCI, but most patients had echocardiography only on the day after admission. Therefore, we could not compare the left ventricular size or function between the two groups.

**Conclusion:** In this study, we showed that selective intracoronary administration of nitroprusside through the drug delivery catheter before balloon dilatation is safe and well-tolerated, prevents no reflow or slow reflow, and improves reperfusion of the infarcted myocardium. Our study is the first to report the beneficial effects of selective nitroprusside administration given prior to the development of slow reflow.

**REFERENCES**