Early versus late ST-segment resolution and clinical outcomes after percutaneous coronary intervention for acute myocardial infarction

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Background. **Absence of complete ST-segment resolution (STR) after percutaneous coronary intervention (PCI) for ST-segment-elevation myocardial infarction (STEMI) is a determinant of mortality. Traditionally, STR is determined on the coronary care unit (CCU) 60 to 90 minutes after the initiation of reperfusion therapy. We studied the prognostic value of STR immediately after PCI.**
Methods. **We analysed 223 consecutive patients with STEMI and successful PCI. Continuous ECG data were collected during PCI and at 30 minutes after arrival on the CCU (mean time 81±17 minutes after reflow of the culprit artery). Patients were divided into three groups: patients with complete STR immediately after PCI ('early'), patients with complete and persistent STR at 30 minutes on the CCU, but not immediately after PCI ('late') and patients without STR. One-year follow-up was obtained for death and rehospitalisation for major adverse cardiac events. Cox proportional hazards regression was used to evaluate the association between STR and outcome.**
Results. **Early STR occurred in 115 (52%) and late STR in 43 (19%) patients. Patients with early or late STR had a lower incidence of one-year cardiac death than those without STR (1.9 vs. 9.2%; p=0.02). In contrast, rehospitalisation occurred more frequently in patients with early or late STR (20.3 vs. 6.2%; p=0.009). As compared with patients without STR, early and late STR had a similar prognostic value (hazard ratios [95% confidence interval] for cardiac death 0.40 [0.08-2.03] and 0.25 [0.03-2.08]).**
Conclusions. **We found no (major) change in prognostic value of STR during the 0 to 90 minutes time window after PCI. (Neth Heart J 2010;18:416-22.)**

Keywords: ST-Segment Resolution; Percutaneous Coronary Intervention; Acute Myocardial Infarction

Primary percutaneous coronary intervention (PCI) restores epicardial coronary flow in the vast majority of patients with acute ST-segment elevation myocardial infarction (STEMI). However, despite successful primary PCI, a substantial number of patients show signs of suboptimal myocardial reperfusion, manifested by persistent ST-segment elevation and incomplete ST-segment elevation resolution (STR). Absent or incomplete STR after PCI is a strong predictor for impaired left ventricular function and adverse clinical outcomes.[1-8]

After fibrinolysis for STEMI, the electrocardiogram (ECG) for measuring STR is usually taken 60 to 90 minutes after onset of therapy.[9-11] After primary PCI, the first ECG that is suitable for evaluation of STR is usually done on the coronary care unit (CCU). In previous studies, the timing of the ECG after primary PCI for determination of STR is highly variable. It ranges from the time of arrival on the CCU, approximately 30 minutes after PCI, to another 30 minutes or even several hours later.[1-7]

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Earlier studies showed that STR at 30 minutes after PCI correlated better with other markers of myocardial perfusion than STR at 60 to 90 minutes.[12,13]

However, the optimal time to measure STR after primary PCI is unknown. In addition, in case of suboptimal myocardial reperfusion, therapeutic options have to be considered as soon as possible, preferably during the PCI procedure. Therefore, in the present study we used continuous ten-lead ST monitoring,[14,15] and measured STR at the end of the PCI procedure (early STR) and compared it with STR measured at 30 minutes after arrival on the CCU (late STR). We evaluated the predictive value of early versus late STR for (cardiac) death and rehospitalisation for major adverse cardiac events (MACE) at one year.

Methods

Patients

The study population consisted of consecutive patients with STEMI (>1 mm ST-segment elevation in ≥2 limb leads or >2 mm ST-segment elevation in ≥2 precordial leads). In case of posterior myocardial infarction >2 mm ST-segment depression in ≥2 precordial leads) admitted for primary PCI at the department of cardiology of the VU University Medical Center in Amsterdam, the Netherlands. Patients were included after successful PCI, defined as sustained patency of the occluded epicardial coronary artery with TIMI flow grade ≥2, within six hours after symptom onset. Patients were excluded in case of a history of myocardial infarction in the same vascular territory, if continuous ECG registration failed or if the ECG could not be used to measure STR (left bundle branch block, pacemaker or ventricular rhythm). In addition, patients who already showed reperfusion at the start of the PCI procedure, defined as resolved symptoms with complete STR, were excluded.

All patients received acetylsalicylic acid (450 mg IV) and heparin (5000 IU IV) before the procedure. Administration of abciximab or intracoronary adenosine was left to the discretion of the operator. After the procedure all patients received clopidogrel 75 mg daily with a loading dose of 300 mg orally. In 216 (97%) of patients in the study population, one or more coronary stents were implanted.

ST-segment resolution

During and after the procedure, patients were monitored with a continuous ten-lead ECG registration (ST-Guard®, Medtronic, Santa Rosa). An ECG was derived and stored every minute. Automated ST-segment deviation (μV) was measured on line for each single lead at 60 ms after the J point. Percentage STR (100% minus (ST elevation after PCI/ST-segment elevation before PCI) x 100%) was calculated for the single lead with maximal elevation. ST-segment depression in leads V1 to V4 was used in case of posterior infarction.[16]

STR was determined based on the ECG at the beginning and the end of the PCI procedure and at 30 minutes after arrival on the CCU (mean time 81±17 minutes after reflow of the culprit artery). Complete STR was defined as >70% STR or <70% without residual ST-segment elevation (<1 mm in non-anterior leads and <2 mm in anterior leads), while no STR was defined as <70% STR with residual ST-segment elevation.[6] Patients were divided into three groups according to the presence or absence of STR: complete and persistent STR immediately after PCI, ('early'), complete and persistent STR at 30 minutes on the CCU, but not immediately after PCI ('late') and no or incomplete STR at 90 min after PCI ('without').

Endpoint definition

One-year follow-up data were collected on (cardiac) death and rehospitalisation for MACE, defined as nonfatal infarction, unstable angina or re-revascularisation. We contacted patients by telephone, the patient's general practitioner, as well as the referring physician to obtain information on these events. The primary endpoint was cardiac death, which was defined as death that could directly be related to loss of heart function. Secondary endpoints were all-cause death, and hospitalisation for MACE.

Statistical analyses

Statistical analysis was performed with SPSS 15.0 (SPSS, Inc, Chicago, Illinois). Categorical variables are summarised as numbers and percentages and continuous variables are presented as mean values ± one standard deviation (SD). Differences in baseline characteristics according to STR classification were studied by χ^2 or Fisher's exact tests (categorical data) and analysis of variance (ANOVA) and Mann-Whitney tests (continuous data).

The incidence of study endpoints over time was studied according to the method of Kaplan-Meier, and differences in the incidence according to the STR classification were evaluated by log-rank tests. The association between STR and study endpoints was further analysed by Cox proportional hazard regression. We obtained crude, unadjusted hazard ratios (HR), and HRs that were adjusted for age and infarct localisation. HRs are reported together with their 95% confidence intervals (CI). All statistical tests were two-sided, and a p value <0.05 was considered statistically significant.

Results

Between January 2003 and April 2004, 223 patients met the inclusion criteria. A total of 115 (52%) patients had early STR, and another 43

Table 1. Baseline and procedural characteristics.

Variable	Early STR	Late STR	Without STR	P value
Patients, n (%)	115 (51.6)	43 (19.3)	65 (29.2)	
Age, years	61±13	66±13	66±14	0.03
Male sex, n (%)	80 (70)	28 (65)	46 (71)	0.81
Culprit artery, n (%)	36 (31)	18 (42)	38 (58)	<0.01
- LAD				
- LCx	21 (18)	4 (9)	6 (9)	
- RCA	58 (50)	21 (49)	21 (32)	
Post PCI TIMI 3 flow, n (%)	111 (97)	37 (86)	47 (72)	<0.01
Medication				
- Abciximab, n (%)	77 (67)	24 (56)	44 (68)	0.37
- Adenosine, n (%)	28 (24)	12 (28)	14 (22)	0.75
ST elevation, μV				
- Pre PCI*	323 (190; 541)	424 (249; 649)	463 (320; 722)	<0.01
- Post PCI*	78 (9; 136)	224 (122; 302)	302 (224; 449)	<0.01
- CCU*	58 (9; 97)	92 (58; 161)	273 (181; 351)	<0.01

Continuous data are expressed as mean ± standard deviation. Categorical data are expressed as percentage. P values indicate the difference between the three groups. * Data are expressed as the median and interquartile range because of skewed distribution. STR=ST-segment resolution. LAD=left anterior descending coronary artery, LCx=left circumflex coronary artery, RCA=right coronary artery, PCI=percutaneous coronary intervention, CCU=coronary care unit.

(19%) had late STR. Hence, STR at 30 minutes after arrival on the CCU was obtained in 71% of patients. There were differences in baseline clinical and procedural characteristics between patients with early and late STR: patients with late STR were older and TIMI flow post-PCI was worst in these patients. Larger differences were found in the baseline profile between those with and without STR. Patients without STR were older, more often had the culprit lesion in the left anterior descending artery and TIMI flow grade 3 after PCI was (considerably) more often obtained in patients with (early) STR than in those without (table 1).

The event-free survival curves according to presence or absence of STR show that more patients died in the group without STR from cardiac causes (figure 1, p=0.049) as well as from overall causes (figure 2, p=0.03). Figure 3 shows that rehospitalisation occurred more frequently in the early STR group (figure 3, p=0.02).

During one-year follow-up, 15 patients died and 36 were rehospitalised for MACE. As table 2 demonstrates, patients with early or late STR had a significantly lower incidence of one-year cardiac death than those without STR (1.9 vs. 9.2% events; p=0.02). Although no difference in cardiac mortality was found between the three groups (p=0.06), total mortality was higher in the group without STR (13.8 vs. 3.8%; p=0.02). As compared with patients without STR, early and late STR had a similar prognostic value (HRs for cardiac death 0.40 [0.08-2.03] and 0.25 [0.03-2.08]) (table 3).

Early and late STR was associated with an increased risk of rehospitalisation for MACE (table 2; p=0.009). After adjustment for age and infarct localisation, the HR for rehospitalisation for early STR was 3.76 (1.29 to 11.02) and for late STR 2.19 (0.62 to 7.78) (table 3).

Discussion

Immediately after the percutaneous intervention, approximately half of the patients had complete and sustained STR, whereas about two thirds of the patients had complete STR at 30 minutes after arrival on the CCU. Our data suggested that, within this time window, there is no major change in prognostic value of early versus late STR with respect to all-cause mortality, cardiac mortality, or rehospitalisation for adverse cardiac events during one-year follow-up.

This improvement of STR over time after reperfusion, from 54% early STR to 71% late STR, is caused by a gradual, spontaneous recovery of the ST-segment. This finding is consistent with earlier studies[17-20] and has important consequences, in particular in considering additional therapies in persistent ST-segment elevation after PCI. Currently, additional therapies, such as vasodilators, glycoprotein IIb/IIIa receptor antagonists or an intra-aortic balloon pump, are mainly ap-

Table 2. Early ST resolution and clinical endpoints.

	Early STR n=115	Late STR n=43	Early + late STR n=158	Without STR n=65	P value
Cardiac mortality, (%)	2 (1.7)	1 (2.3)	3 (1.9)	6 (9.2)	0.02
Noncardiac mortality (%)	2 (1.7)	1 (2.3)	3 (1.9)	3 (4.6)	0.36
Total mortality (%)	4 (3.5)	2 (4.7)	6 (3.8)	9 (13.8)	0.02
Rehospitalisation (%)	26 (22.6)	6 (14.0)	32 (20.3)	4 (6.2)	0.009
- Nonfatal infarction	3 (2.6)	1 (2.3)	4 (2.5)	0	0.56
- Unstable angina	9 (7.8)	0	9 (5.7)	0	0.04
- Revascularisation	18 (15.7)	5 (11.6)	23 (14.6)	4 (6.2)	0.08
- PCI	11 (9.6)	1 (2.3)	12 (7.6)	1 (1.5)	0.12
- CABG	11 (9.6)	4 (9.3)	15 (9.5)	3 (4.6)	0.22
- TVR	10 (8.7)	4 (9.3)	14 (8.9)	4 (6.2)	0.50
Total MACE	26 (22.6)	7 (16.3)	33 (20.9)	10 (15.4)	0.34

Data are expressed as numbers (%). P values indicate the difference between early + late STR versus without STR. STR=ST-segment resolution, PCI=percutaneous coronary intervention, CABG=coronary artery bypass grafting, TVR=target vessel revascularisation, MACE=major adverse cardiac event.

Table 3. Hazard ratios for cardiac and total mortality and rehospitalisation according to STR.

	Cases/ subjects	Hazard ratio (CI)	
		Model 1*	Model 2*
Cardiac mortality			
- Without STR	6/65	1 (reference)	1 (reference)
- Early STR	2/115	0.25 (0.05-1.28)	0.40 (0.08-2.03)
- Late STR	1/43	0.27 (0.03-2.22)	0.25 (0.03-2.08)
Total mortality			
- Without STR	9/65	1 (reference)	1 (reference)
- Early STR	4/115	0.36 (0.11-1.20)	0.53 (0.16-1.76)
- Late STR	2/43	0.37 (0.08-1.72)	0.34 (0.07-1.59)
Rehospitalisation			
- Without STR	4/65	1 (reference)	1 (reference)
- Early STR	26/115	4.17 (1.45-12.05)	3.76 (1.29-11.02)
- Late STR	6/43	2.27 (0.64-8.04)	2.19 (0.62-7.78)

* Model 1 adjusted for age; model 2 adjusted for age and infarct localisation (anterior versus non-anterior). STR=ST-segment resolution.

plied in case of no reflow.[21,22] However, even though angiographic evidence of reflow may be present, absence of STR is a negative prognostic marker and must be considered as a guide to initiate additive, abovementioned, therapies as well. Studies investigating the effect of pharmacological or mechanical therapies to optimise myocardial reperfusion should take spontaneous late STR into account in nearly half of the patients with persistent ST elevation directly after PCI.

As shown in table 1, patients with late STR and without STR had more single lead ST-segment elevation before PCI compared with patients with early STR (424 μV, 463 μV and 323 μV respectively, $p<0.01$). A possible explanation is that more ST-segment elevation pre-PCI reflects more intense myocardial ischaemia, for example due to distal embolisation, causing less STR after restoring blood flow in the epicardial coronary vessel. In addition, more ST-segment elevation before reperfusion can

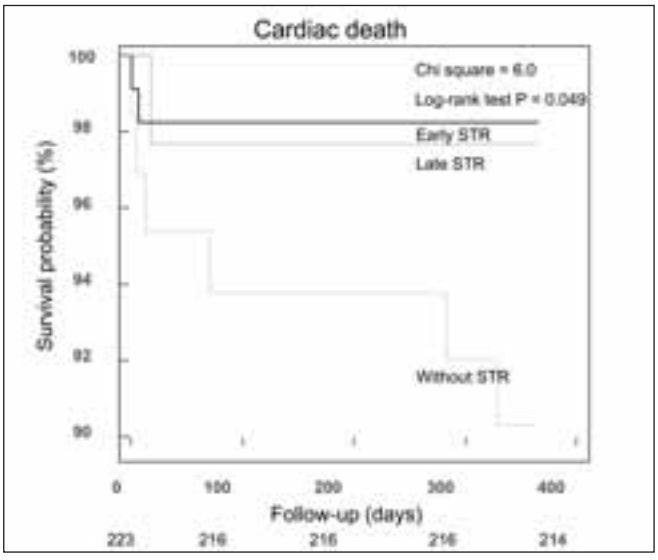

Figure 1. Event-free survival curve according to ST-segment resolution (STR) classification.

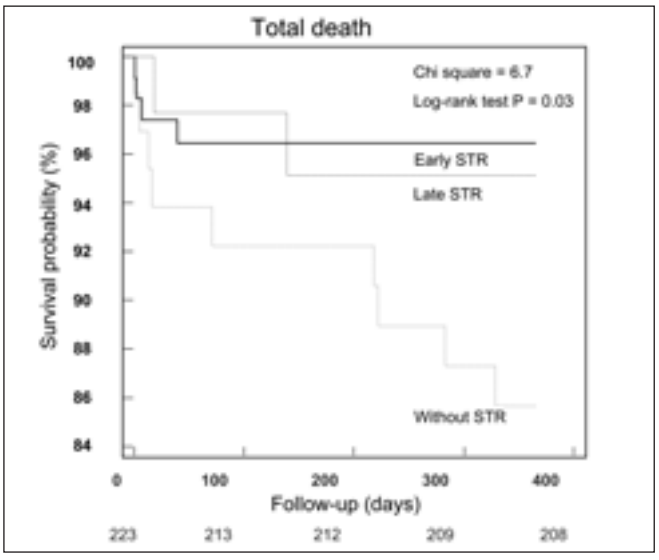

Figure 2. Event-free survival curve according to ST-segment resolution (STR) classification until the occurrence of death of any cause.

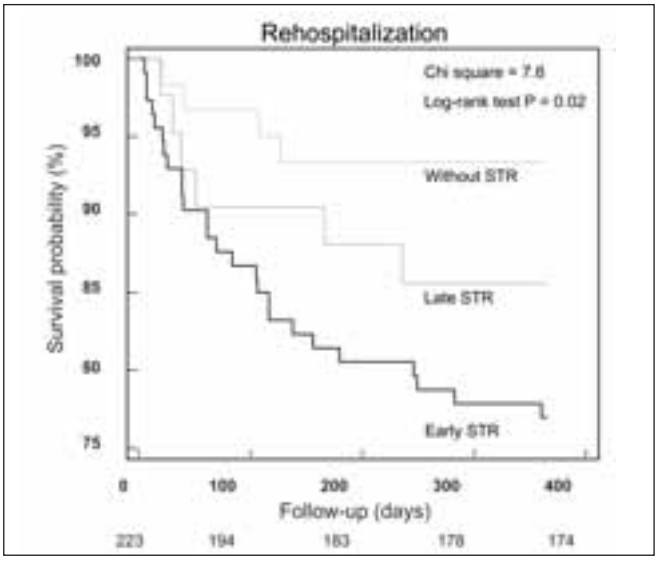

Figure 3. Event-free survival curve according to ST-segment resolution (STR) classification until the occurrence of rehospitalisation.

reflect more irreversible myocardial cell damage, for instance due to a lack of collateral blood supply or less preconditioning, with less myocardial tissue rescue and less STR after reperfusion.

One-year survival free from cardiac death was not significantly different in patients with early STR compared with late STR, but in patients without STR, cardiac death was significantly higher. This first finding is unexpected considering the fact that persistent ST-segment elevation after PCI is thought to reflect ongoing myocardial cell damage and thus should be associated with larger final infarct size, lower left ventricular ejection fraction and higher mortality. It is possible that persistent ST-segment elevation after successful PCI is not equal to longer ischaemic time or that the time between early and late STR in our study was not long enough to find a significant difference. Moreover, our study may be too small to detect small but clinically relevant differences. Future studies are needed to investigate the effect of delayed recovery of the ST segment after primary PCI on microvascular reperfusion, no reflow area and infarct size.

Another finding in this study was a significantly higher incidence of rehospitalisation in the early and late STR group compared with patients without STR (20.3 vs. 6.2%, p=0.009) caused by a higher incidence of nonfatal myocardial infarction, unstable angina and revascularisation. Bainey et al. also found a higher likelihood for recurrent acute myocardial infarction and unstable angina[23] as well as De Lemos et al. who reported that more STR was associated with higher rates of recurrent myocardial infarcts.[24] We can hypothesise that STR after primary PCI is associated with more extensive residual myocardial viability. Restenosis or re-occlusion (stent thrombosis) of these target vessels may therefore lead to more symptoms and rehospitalisation compared with target vessels without myocardial viability in its territory. However, there was no significant difference between the incidence of target vessel revascularisation in patients with STR compared with those without STR (8.9 vs. 6.2%, p=0.50) that would have supported this hypothesis. In addition, unknown confounding factors could have contributed to the association of no-STR after primary PCI with less rehospitalisation, for instance differences in certain patient baseline and therapeutic characteristics.

In the one-year results of the Horizons-AMI

study, MACE (defined as death, reinfarction, ischaemia-driven target vessel revascularisation and stroke) was 10.2 to 12.9%[25] compared with 15.4 to 22.6% in our study. Nontarget vessel revascularisations could partly explain these differences, but it is unknown how many of our patients had ischaemic-driven target vessel revascularisation. However, in our study period, only the target vessel was treated by primary PCI using bare metal stents and staged PCI procedures were only performed in case of clearly demonstrated ischaemia by nuclear tests, which was rarely the case.

Study limitations

Our study population consisted of relatively low-risk STEMI patients, because high-risk patients (e.g. shock, respiratory failure, previous myocardial infarction and failed PCI) were excluded. This may have caused an underestimation of the incidence of cardiac death and MACE in STEMI patients in the general cardiology clinic. In addition, patients with persistent conduction or rhythm disturbances could not be used for determination of STR. Data concerning blood pressure, heart frequency, myocardial infarct size (cardiac enzyme release) and left ventricular function were not available so that we could not correct for all known confounders. We can therefore not exclude a residual bias in the effect estimates (HRs). The number of patients we studied was relatively small. Consequently, clinically relevant associations between STR status and patient outcome might have been missed due to this relatively small sample size resulting in few events during follow-up. Therefore, we warrant further large sample studies to conform our data.

Conclusions

We found no (major) change in the prognostic value of STR during the 0 to 90 minutes time window after PCI for one-year follow-up on cardiac death and MACE. Therefore, for initiation of additional therapy in case of suboptimal myocardial reperfusion after primary PCI, STR determined at the end of the procedure is a useful guide. ■

References

1 van 't Hof AW, Liem A, de Boer MJ, Zijlstra F. Clinical value of 12-lead electrocardiogram after successful reperfusion therapy for acute myocardial infarction. Zwolle Myocardial infarction Study Group. Lancet. 1997;350:615-9.
2 Claeys MJ, Bosmans J, Veenstra L, Jorens P, De Raedt H, Vrints CJ. Determinants and prognostic implications of persistent ST-segment elevation after primary angioplasty for acute myocardial infarction: importance of microvascular reperfusion injury on clinical outcome. Circulation. 1999;99:1972-7.
3 Matetzky S, Novikov M, Gruberg L, Freimark D, Feinberg M, Elian D, et al. The significance of persistent ST elevation versus early resolution of ST segment elevation after primary PTCA. J Am Coll Cardiol. 1999;34:1932-8.
4 Santoro GM, Antoniucci D, Valenti R, Bolognese L, Buonamici P, Trapani M, et al. Rapid reduction of ST-segment elevation after successful direct angioplasty in acute myocardial infarction. Am J Cardiol. 1997;80:685-9.
5 Brodie BR, Stuckey TD, Hansen C, VerSteeg DS, Muncy DB, Moore S, et al. Relation between electrocardiographic ST-segment resolution and early and late outcomes after primary percutaneous coronary intervention for acute myocardial infarction. Am J Cardiol. 2005;95:343-8.
6 Buller CE, Fu Y, Mahaffey KW, Todaro TG, Adams P, Westerhout CM, et al. ST-segment recovery and outcome after primary percutaneous coronary intervention for ST-elevation myocardial infarction: insights from the Assessment of Pexelizumab in Acute Myocardial Infarction (APEX-AMI) trial. Circulation. 2008;118:1335-46.
7 McLaughlin MG, Stone GW, Aymong E, Gardner G, Mehran R, Lansky AJ, et al. Prognostic utility of comparative methods for assessment of ST-segment resolution after primary angioplasty for acute myocardial infarction: the Controlled Abciximab and Device Investigation to Lower Late Angioplasty Complications (CADILLAC) trial. J Am Coll Cardiol. 2004;44:1215-23.
8 Schroder R. Prognostic impact of early ST-segment resolution in acute ST-elevation myocardial infarction. Circulation. 2004;110(21):e506-10.
9 Gibson CM, Karha J, Giugliano RP, Roe MT, Murphy SA, Harrington RA, et al. Association of the timing of ST-segment resolution with TIMI myocardial perfusion grade in acute myocardial infarction. Am Heart J. 2004;147:847-52.
10 Johanson P, Jernberg T, Gunnarsson G, Lindahl B, Wallentin L, Dellborg M. Prognostic value of ST-segment resolution-when and what to measure. Eur Heart J. 2003;24:337-45.
11 Schroder R, Dissmann R, Bruggemann T, Wegscheider K, Linderer T, Tebbe U, et al. Extent of early ST segment elevation resolution: a simple but strong predictor of outcome in patients with acute myocardial infarction. J Am Coll Cardiol. 1994;24:384-91.
12 Terkelsen CJ, Norgaard BL, Lassen JF, Poulsen SH, Gerdes JC, Sloth E, et al. Potential significance of spontaneous and interventional ST-changes in patients transferred for primary percutaneous coronary intervention: observations from the ST-MONitoring in Acute Myocardial Infarction study (The MONAMI study). Eur Heart J. 2006;27:267-75.
13 Watanabe J, Nakamura S, Sugiura T, Takehana K, Hamada S, Miyoshi H, et al. Early identification of impaired myocardial reperfusion with serial assessment of ST segments after percutaneous transluminal coronary angioplasty during acute myocardial infarction. Am J Cardiol. 2001;88:956-9.
14 Krucoff MW, Green CE, Satler LF, Miller FC, Pallas RS, Kent KM, et al. Noninvasive detection of coronary artery patency using continuous ST-segment monitoring. Am J Cardiol. 1986;57:916-22.
15 Krucoff MW, Johanson P, Baeza R, Crater SW, Dellborg M. Clinical utility of serial and continuous ST-segment recovery assessment in patients with acute ST-elevation myocardial infarction: assessing the dynamics of epicardial and myocardial reperfusion. Circulation. 2004;110:e533-9.
16 Schroder K, Wegscheider K, Zeymer U, Tebbe U, Schroder R. Extent of ST-segment deviation in a single electrocardiogram lead 90 min after thrombolysis as a predictor of medium-term mortality in acute myocardial infarction. Lancet. 2001;358:1479-86.
17 Cura FA, Escudero AG, Berrocal D, Mendiz O, Trivi MS, Fernandez J, et al. Protection of Distal Embolization in High-Risk Patients with Acute ST-Segment Elevation Myocardial Infarction (PREMIAR). Am J Cardiol. 2007;99:357-63.
18 Stoel MG, Marques KM, de Cock CC, Bronzwaer JG, von Birgelen C, Zijlstra F. High dose adenosine for suboptimal myocardial reperfusion after primary PCI: A randomized placebo-controlled pilot study. Catheter Cardiovasc Interv. 2008;71:283-9.
19 Stone GW, Webb J, Cox DA, Brodie BR, Qureshi M, Kalynych A, et al. Distal microcirculatory protection during percutaneous coronary intervention in acute ST-segment elevation myocardial infarction: a randomized controlled trial. JAMA. 2005;293:1063-72.

20 De Carlo M, Wood DA, Webb JG, Gerckens U, Cortese B, Grube E, et al. Adjunctive use of the Rinspiration system for fluidic thrombectomy during primary angioplasty: the Rinspiration international registry. Catheter Cardiovasc Interv. 2008;72:196-203.
21 Fischell TA. Pharmaceutical interventions for the management of no-reflow. J Invasive Cardiol. 2008;20:374-9.
22 Lee KW, Norell MS. Management of 'no-reflow' complicating reperfusion therapy. Acute Card Care. 2008;10:5-14.
23 Bainey KR, Senaratne MP. Is the outcomes of early ST-segment resolution after thrombolytic therapy in acute myocardial infarction always favorable? J Electrocardiol. 2005;38:354-60.
24 De Lemos JA, Antman EM, Giugliano RP, Morrow DA, McCabe CH, Charlesworth A, et al. Very early risk stratification after thrombolytic therapy with a bedside myoglobin assay and the 12-lead electrocardiogram. Am Heart J. 2000;140:373-8.
25 Mehran R, Lansky AJ, Witzenbichler B, Guagliumi G, Peruga JZ, Brodie BR, et al. Bivalirudin in patients undergoing primary angioplasty for acute myocardial infarction (HORIZONS-AMI): 1-year results of a randomised controlled trial. Lancet. 2009;374:1149-59.