Incomplete Resolution of ST-Segment Elevation Is a Marker of Transient Microcirculatory Dysfunction After Stenting for Acute Myocardial Infarction

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Methods and Results—Fifty patients with ≤12-hour AMI underwent successful primary angioplasty and systematic stenting with a Doppler guidewire. Patients with incomplete (<50%) STR 60 minutes after TIMI 3 flow was restored had flow velocity features suggestive of severe microcirculatory dysfunction, including a higher incidence of early systolic retrograde flow (41% versus 9%, \( P = 0.007 \)) and lower coronary flow velocity reserve (CVR, 1.3 versus 1.6, \( P < 0.001 \)). CVR improved immediately after stenting in patients with ≥50% STR but not in patients with <50% STR. There was a significant correlation between STR and poststenosis CVR. At 3 months, CVR was similar in patients with <50% and ≥50% STR. However, left ventriculography indicated lower global (42% versus 55%, \( P = 0.001 \)) and regional (16% versus 20%, \( P = 0.03 \)) left ventricular ejection fractions and 201Tl rest-redistribution scintigraphy indicated a larger infarct size (34% versus 16% 201Tl defect, \( P = 0.007 \)) in patients with <50% STR.

Conclusions—After successful primary angioplasty with systematic stenting, <50% STR is a marker of severe albeit transient microcirculatory dysfunction in patients with AMI and is associated with more extensive myocardial damage.

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Key Words: myocardial infarction ■ angioplasty ■ stents ■ electrocardiography ■ microcirculation

In up to 40% of patients with acute myocardial infarction (AMI), successful recanalization of the culprit coronary artery is associated with suboptimal myocardial reperfusion (no reflow) caused by microcirculatory damage and distal embolization.1 Rapid ST-segment resolution (STR) after primary angioplasty for AMI is associated with improved long-term survival and preserved left ventricular (LV) function.2,3 Even though STR may be an index of microcirculatory function, the relation between STR and other more complex techniques directly evaluating microcirculatory function remains to be fully determined.4 Doppler guidewires have been used extensively in the AMI setting.5–11 Specific coronary flow velocity patterns7,11 and a severe impairment of coronary flow velocity reserve (CVR)10 have been documented in patients with AMI with no reflow after primary angioplasty.

In the multicenter FRench Optimal STenting (FROST)-2 study, we used Doppler guidewires to investigate the relation between STR and microcirculatory function in patients with AMI undergoing successful primary angioplasty and systematic stenting.

Patient Population

Patients referred for primary angioplasty were enrolled in the study if they fulfilled the following criteria: (1) symptoms consistent with AMI lasting for ≤12 hours; (2) ST-segment elevation ≥2 mm in ≥2 contiguous leads; and (3) angiographic evidence of total occlusion, that is, Thrombolysis In Myocardial Infarction (TIMI) 0 or 1 grades, of proximal or mid segments of the left anterior descending artery or a dominant right coronary artery. Only patients with successful angioplasty (defined as correct stent deployment, stable TIMI 3 flow, and <30% residual stenosis at the occlusion site) and no additional >50% stenosis on the infarct-related coronary artery (IRA) were included. Patients were not eligible if they had ≥50% left main coronary artery stenosis; 3-vessel coronary artery disease; unfavor-
able anatomy precluding stent implantation; hemodynamic instability requiring the use of intra-aortic balloon pump or intravenous inotropic support, vasodilators, or β-blockers; sustained arrhythmia; LV hypertrophy criteria on baseline ECG, defined as Sokolow-Lyon voltages >35 mV; and history of diabetes mellitus or AML. The study was approved by the Human Research Committee of CHU Angers, and all patients gave written informed consent.

Fifty-nine patients were included in 8 trained coronary care units (CCU). Nine patients were excluded from analysis for the following reasons: One patient had persistent TIMI 2 flow after stenting; in 4 patients, 1 of the 2 ECGs was missing or not interpretable because of the development of a left bundle-branch block; and in 4 patients, the quality of Doppler flow velocity recording during primary angioplasty was suboptimal. Hence, the data from 50 patients were analyzed.

Electrocardiographic Analysis
The first ECG was obtained immediately before angioplasty and the second 60 minutes after restoration of TIMI 3 flow. ST-segment elevation was measured 20 ms after the J-point. The sum of ST-segment elevations (2STE) was measured in leads I, aVL, and V1 through V6 for inferior infarctions and in leads II, III, aVF, V5, and V6 for inferior infarctions. Patients with ≥50% reduction in 2STE on the second ECG were classified in the “≥50% STR” group, whereas the other patients were classified in the “<50% STR” group. The 2 ECGs were analyzed in a blinded fashion by 2 cardiologists. Classification was identical for the 2 readers in 42 of 50 patients (84%). Discrepancies were resolved by consensus repeat analysis for the 8 remaining patients.

Primary Angioplasty, Quantitative Coronary Angiography, and LV Function
Each patient received aspirin (>250 mg IV) and heparin (70 IU/kg IV) before angioplasty. The use of GP IIb/IIIa blockers was left to the discretion of the operator. The contralateral artery was first injected, and collateral vessels were graded according to Rentrop’s classification.12 Occlusion of the IRA was crossed by using a 0.014-inch, Doppler-tipped guidewire (FioWire, EndoSonics). Balloon angioplasty was performed to achieve TIMI 3 flow and <50% residual stenosis by visual assessment. A Bard XT stent (15- or 19-mm length, 3.0- or 3.5-mm diameter) was then implanted so that residual stenosis was <30%. Additional stents could be implanted when required. The occurrence of angiographic complications—for example, acute occlusion of the IRA or major side-branch, distal embolism, or coronary dissection of grade >B according to the classification of the National Heart, Heart, Lung, and Blood Institute,13 at any time during angioplasty—was noted. After angioplasty, patients received aspirin plus ticlopidine or plus clopidogrel for 4 weeks, then aspirin alone.

Coronary angiography was repeated at 3 months, using the same projections. In addition, LV angiograms were acquired in the 30° right anterior oblique projection.

Quantitative coronary angiography and evaluation of LV function were performed off-line in a core laboratory with the computerized Sigma Cardio system (Traitement Synthèse Image).14 Global LV ejection fraction (LVEF) was measured by using the area-length method. Regional LVEF in the infarct zone was determined by using an area-based method in which the specific contribution of 8 LV segments (antero inferior: segments 1 to 5; inferior infarct: segments 5 to 8) to the global LVEF is measured.

Coronary Flow Velocity Measurements
Coronary flow velocity and CVR were measured during primary angioplasty and 3-month follow-up angiogram with the Doppler guidewire connected to a real-time spectrum analyzer (FloMap, EndoSonics).9 The Doppler probe was positioned ≥20 mm distal to the occlusion site to avoid poststenotic turbulence. Flow-dependent dilation of epicardial coronary arteries was abolished by intracoronary injection of 1 mg lidocaine.

Five minutes after the last balloon inflation, prestent CVR was measured after intracoronary injection of 18 μg adenosine as the ratio of hyperemic average peak velocity (APV<sub>max</sub>) to resting APV (APV<sub>rest</sub>). Measurements were repeated until 2 consecutive values varied by ≤10%. The higher of the 2 values was used in the analysis. After stent implantation, CVR measurements were repeated after APV trends had reached a plateau for ≥5 minutes. Immediately before withdrawal of the guidewire from the IRA, final Doppler flow velocity spectrum was recorded for off-line analysis.7 The presence of an early systolic retrograde flow (defined as a negative signal ≥10 cm/s, lasting ≥60 ms and visible on ≥3 consecutive cardiac cycles) was noted and the following variables were measured: systolic flow duration time (ms), maximum systolic peak velocity (cm/s), average systolic peak velocity (cm/s), diastolic flow duration time (ms), maximum diastolic peak velocity (cm/s), and average diastolic peak velocity (cm/s). Reference CVR (CVR<sub>ref</sub>) was then measured in a nonstenosed coronary artery. Relative CVR (CVR<sub>rel</sub>) was calculated as the ratio of poststenosis CVR in the IRA to CVR<sub>ref</sub>.15 At 3-month angiographic follow-up, measurements of coronary flow velocity and CVR were repeated at the same locations in the IRA and the reference artery. If angiographic restenosis, defined as in-stent stenosis ≥50%, had occurred, balloon angioplasty was systematically performed to achieve a residual stenosis <30%, and flow velocity studies were performed ≥5 minutes after angioplasty. All flow velocity data were recorded on super-VHS videotape and reviewed off-line by 2 cardiologists.

Determination of Myocardial Infarct Size
Myocardial infarct size was first approximated by peak serum creatine kinase (CK) activity measured on blood samples obtained every 6 hours for 48 hours. In addition, rest-redistribution 201<TI myocardial scintigraphy was performed at 3 months and myocardial infarct size was measured in a nuclear medicine core laboratory. Briefly, reconstructed slices were displayed by using a bull’s eye polar map, defect was delineated with a 60% isocountour level of the maximal pixel value, and infarct size, measured by the 201<TI defect size, was expressed as a percentage of the whole myocardial area.16

Statistical Analysis
Continuous variables are expressed as medians (25th to 75th percentiles). Comparisons of hemodynamic and flow velocity variables at different time points (presten, poststen, and follow-up) in the ≥50% STR and <50% STR groups were performed by a 2-way repeated-measures ANOVA, which tested the effect of STR (between factor), the variations over time (within factor), and the interaction of both. If the global test showed a significant effect or interaction, a 1-way repeated-measures ANOVA with post hoc Fisher protected least-squares difference test was performed to study specifically the variations over time of hemodynamic and flow velocity variables in the ≥50% STR and <50% STR groups, and a factorial ANOVA was performed to study the effect of STR on individual variables. Other continuous and categoric variables were compared by using the Mann-Whitney U test and χ² analysis, respectively. The relation between continuous variables was studied by means of simple regression analysis. A value of P<0.05 was considered statistically significant. All statistical analyses were performed with StatView 5.0 software (SAS Institute Inc).

Results
Clinical Characteristics
Seventeen patients (34%) had <50% STR despite successful angioplasty and stenting. These patients were more likely to have an anterior myocardial infarction and less likely to have pre-AMI angina, defined as episodes of chest pain at rest ≤7 days before AMI (Table 1).
Primary Angioplasty and Angiographic Data

All the patients had successful stent implantation, with similar pain–to–TIMI 3 times (Table 2). Angiographic complications were more frequent in patients with <50% STR. Abciximab was used only as a bailout treatment in 5 of 50 patients with angiographic complications, of whom 1 had ≥50% STR and 4 had <50% STR (P=0.02).

Follow-up angiography was performed 95 (84 to 110) days after AMI in 31 of 33 patients with ≥50% STR (2 patients refused the procedure) and 14 of 17 patients with <50% STR (2 in-hospital deaths, and 1 patient refused the procedure). In-stent restenosis was found in 6 of 31 (19.3%) and 3 of 14 (21.4%) patients with ≥50% and <50% STR, respectively (NS). In each case, successful balloon angioplasty was performed before flow velocity measurements.

Hemodynamic Data and Coronary Flow Velocity Reserve

After balloon angioplasty but before stenting, CVRs in the IRA were low and similar in patients with ≥50% and <50% STR (Table 3). After stent implantation, patients with ≥50% STR had a greater increase of APV peak (+42% [6 to 82] versus +22% [0 to 42] P=0.01) and CVR (+25% [5.5 to 38.8] versus +7% [−13 to 15], P=0.01) than patients with <50%

### TABLE 1. Baseline Clinical Characteristics

<table>
<thead>
<tr>
<th></th>
<th>≥50% STR</th>
<th>&lt;50% STR</th>
<th>P</th>
</tr>
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<tbody>
<tr>
<td>No. (%)</td>
<td>33 (66)</td>
<td>17 (34)</td>
<td></td>
</tr>
<tr>
<td>Male, n (%)</td>
<td>27 (82)</td>
<td>14 (82)</td>
<td>NS</td>
</tr>
<tr>
<td>Age, y</td>
<td>55.5 (49–67.5)</td>
<td>50 (46–67.5)</td>
<td>NS</td>
</tr>
<tr>
<td>Anterior AMI, n (%)</td>
<td>18 (54.5)</td>
<td>14 (82.3)</td>
<td>0.05</td>
</tr>
<tr>
<td>Hypertension, n (%)</td>
<td>9 (27.2)</td>
<td>6 (35.3)</td>
<td>NS</td>
</tr>
<tr>
<td>Dyslipidemia, n (%)</td>
<td>19 (57.5)</td>
<td>8 (47)</td>
<td>NS</td>
</tr>
<tr>
<td>Current smoker, n (%)</td>
<td>26 (78.8)</td>
<td>12 (70.6)</td>
<td>NS</td>
</tr>
<tr>
<td>Treatments before AMI</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Nitrates, n (%)</td>
<td>1 (3)</td>
<td>1 (5.9)</td>
<td>NS</td>
</tr>
<tr>
<td>β-Blockers, n (%)</td>
<td>4 (12.1)</td>
<td>2 (11.8)</td>
<td>NS</td>
</tr>
<tr>
<td>ACE inhibitors, n (%)</td>
<td>3 (9.1)</td>
<td>0</td>
<td>NS</td>
</tr>
<tr>
<td>Statins, n (%)</td>
<td>3 (9.1)</td>
<td>0</td>
<td>NS</td>
</tr>
<tr>
<td>Pre-AMI angina, n (%)</td>
<td>17 (51.5)</td>
<td>3 (17.6)</td>
<td>0.02</td>
</tr>
<tr>
<td>Pain-to-CCU time, min</td>
<td>162.5 (117.5–244)</td>
<td>195 (116–286)</td>
<td>NS</td>
</tr>
<tr>
<td>∑STE before angioplasty, mm</td>
<td>10 (6–18)</td>
<td>10 (7–16.5)</td>
<td>NS</td>
</tr>
<tr>
<td>∑STE 60 min after TIMI 3, mm</td>
<td>3 (1–6)</td>
<td>8 (6–15)</td>
<td>&lt;0.0001</td>
</tr>
</tbody>
</table>

∑STE indicates sum of ST-segment elevations in infarct leads.

### TABLE 2. Angiographic Data During Primary Angioplasty

<table>
<thead>
<tr>
<th></th>
<th>≥50% STR</th>
<th>&lt;50% STR</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>LAD, n (%)</td>
<td>18 (54.5)</td>
<td>14 (82.3)</td>
<td>0.05</td>
</tr>
<tr>
<td>TIMI 0 at baseline, %</td>
<td>100</td>
<td>100</td>
<td>NS</td>
</tr>
<tr>
<td>TIMI 3 after angioplasty, %</td>
<td>100</td>
<td>100</td>
<td>NS</td>
</tr>
<tr>
<td>Multivessel disease, n (%)</td>
<td>23 (69.7)</td>
<td>8 (47)</td>
<td>NS</td>
</tr>
<tr>
<td>Rentrop grade ≥2, n (%)</td>
<td>14 (42.4)</td>
<td>4 (23.5)</td>
<td>NS</td>
</tr>
<tr>
<td>Reference diameter before stent, mm</td>
<td>3 (2.54–3.23)</td>
<td>3.1 (2.92–3.2)</td>
<td>NS</td>
</tr>
<tr>
<td>MLD before stent, mm</td>
<td>1.78 (1.38–2.08)</td>
<td>2 (1.7–2.2)</td>
<td>NS</td>
</tr>
<tr>
<td>% Stenosis before stent, %</td>
<td>33 (28–49.5)</td>
<td>31.6 (27.3–54)</td>
<td>NS</td>
</tr>
<tr>
<td>Reference diameter after stent, mm</td>
<td>3.1 (2.80–3.3)</td>
<td>3.1 (2.9–3.3)</td>
<td>NS</td>
</tr>
<tr>
<td>MLD after stent, mm</td>
<td>2.6 (2.28–2.89)</td>
<td>2.63 (2.57–2.9)</td>
<td>NS</td>
</tr>
<tr>
<td>% Stenosis after stent, %</td>
<td>14.7 (7.8–23.1)</td>
<td>14.2 (11–20)</td>
<td>NS</td>
</tr>
<tr>
<td>Length of stented segment, mm</td>
<td>15 (15–19)</td>
<td>15 (15–19)</td>
<td>NS</td>
</tr>
<tr>
<td>Use of abciximab,* n (%)</td>
<td>1 (6.5)</td>
<td>4 (23.5)</td>
<td>0.02</td>
</tr>
<tr>
<td>Intra-aortic balloon pump, n</td>
<td>0</td>
<td>0</td>
<td>NS</td>
</tr>
<tr>
<td>Angiographic complications, n (%)</td>
<td>8 (24.2)</td>
<td>11 (64.7)</td>
<td>0.007</td>
</tr>
<tr>
<td>Pain–to–TIMI 3 time, min</td>
<td>220 (178.7–299.2)</td>
<td>246 (181.5–341.2)</td>
<td>NS</td>
</tr>
</tbody>
</table>

*LAD indicates left anterior descending coronary artery; MLD, minimum lumen diameter.

*All bailout.
STR, resulting in higher poststent APVpeak and CVR in patients with ≥50% STR. There was a significant correlation between STR and poststent CVR (Figure 1). In the 2 groups, APVpeak and CVR improved over time, and at 3 months the differences were no longer significant. Reference CVR was also low during AMI and improved over time, without difference between patients with ≥50% and <50% STR. At all time points, CVRrel was only mildly reduced and similar in the 2 groups.

Flow Velocity Patterns
Flow velocity patterns were studied after stent implantation (Table 4). Patients with <50% STR had a higher incidence of early systolic retrograde flow, shorter systolic and diastolic duration times, and lower maximum and mean systolic peak velocities.

In-Hospital and 3-Month Follow-Up
Two patients with <50% STR died 2 and 4 days after angioplasty of acute coronary reocclusion and heart failure, respectively. There was no additional death during the 3-month follow-up. Eight of 17 patients with <50% STR (47%) but only 4 of 33 patients with ≥50% STR (12.1%) had ≥1 episode of congestive heart failure (Killip grade ≥2, P = 0.007).

Left Ventricular Function and Myocardial Infarct Size
Patients with ≥50% STR had lower peak CK activity (2636 U/L [1472 to 3598] versus 4190 U/L [2559 to 6974], P = 0.006). At 3 months, these patients had smaller scintigraphic infarct size and higher global and regional LVEF than patients with <50% STR (Figure 2). We found a positive correlation between peak CK activity and scintigraphic infarct size (r = 0.5, P = 0.001).

Discussion
The prognostic value of STR has been extensively studied in thrombolysis trials showing that patients with AMI with incomplete STR are more likely to have persistent coronary artery occlusion17 and large infarct size18 and are at higher risk for death and congestive heart failure.19 However, even after restoration of a TIMI 3 flow using combinations of thrombolitics and abciximab20 or primary angioplasty, 2,3 30% to 50% of the patients still have incomplete STR, suggesting that microcirculatory perfusion in the infarct zone may be more severely impaired in these patients than in patients with complete STR.

We found that 34% of patients with AMI had <50% STR after successful primary angioplasty, in keeping with previous studies.2,3 In addition, we established a relation between STR and previously validated7,10,11 flow velocity parameters of microcirculatory function. Specifically, we found that patients with <50% STR after stenting had a higher incidence of early systolic retrograde flow, shorter systolic and diastolic duration times, lower maximum and mean systolic peak velocities, and lower CVR than patients with ≥50% STR, all indicative of microcirculatory no reflow. At 3

![Figure 1. Correlation between STR 60 minutes after primary angioplasty and poststent CVR.](image-url)
months, CVR had improved and was similar in the 2 groups, suggesting that microcirculatory lesions in patients with <50% STR may be, in a large part, reversible. Of note, CPRref was also low after stenting, a phenomenon that may be related to diffuse α-adrenergic stimulation in the AMI setting,21 and improved at 3 months. Hence, CPRref was only mildly reduced and remained stable over time.

In our study as well as in previous studies,2 patients with complete STR were more likely to have an anterior infarct (82% versus 54%, P = 0.05). Whether microcirculatory reperfusion is intrinsically better in inferior versus anterior infarcts is unknown. We did not find a higher incidence of angiographic complications in anterior versus inferior infarcts (34% versus 44%, NS). However, a Rentrop grade >2 was found in 22% of anterior infarcts versus 61% of inferior infarcts (χ², P = 0.005), hence microcirculation may be less exposed to ischemia as the result of better collateral flow before reperfusion of inferior infarcts. More importantly, poststenotic CPR in the IRA was lower in anterior versus inferior infarcts (1.4 [1.2 to 1.5] versus 1.7 [1.6 to 2.1], P = 0.0001), and the presence of an early systolic retrograde flow was found only in anterior infarcts, suggesting that microcirculatory function is more depressed in anterior infarcts after reperfusion. Whether the higher prevalence of anterior infarcts in patients with incomplete STR is a bias for selecting larger infarcts due to a larger baseline risk area is unclear. However, the similar magnitude of baseline ST-segment elevation in patients with complete and incomplete STR does not support this hypothesis.

Prior studies with angiographic myocardial perfusion blush22 or myocardial contrast echocardiography23 suggested that incomplete STR after primary angioplasty might be pathophysiologically linked to microcirculatory no-reflow. However, these techniques rely on qualitative grading of microcirculatory perfusion, whereas quantitative Doppler velocimetry allowed us to draw a positive correlation between STR and poststenot CVR. In addition, in the present study, all the patients had an occluded culprit coronary artery before angioplasty, and stents were systematically implanted at the occlusion site. In contrast, in the prior studies, many patients had a patent culprit coronary artery before angioplasty22 and stenting was not mandatory,22,23 leading to potential under-estimation of STR and suboptimal myocardial perfusion caused by persistent flow-limiting epicardial stenosis, respectively.

The acute effects of stents on myocardial perfusion are still debated.24 In the present study, median CVR improved after stenting by 25% in patients with ≥50% STR but by only 7% in patients with <50% STR, suggesting that stents may have a beneficial effect on myocardial perfusion, which is more apparent in patients with preserved microcirculatory function. However, in 5 of 33 (15%) patients with ≥50% STR and of 17 (29%) patients with <50% STR, individual CVR actually decreased after stenting. Potential mechanisms for this impairment of microcirculatory perfusion after stenting include distal embolism,25 endothelial dysfunction,26 and α-adrenergic stimulation.21

The prognostic importance of poor microcirculatory reperfusion and incomplete STR has been previously underlined.1–3 Our study is in keeping with these previous findings in that we found that patients with <50% STR had a 4-fold–higher incidence of congestive heart failure, lower global and regional LVEF, and larger enzymatic and scintigraphic infarct size. Hence, incomplete STR is a simple, inexpensive, bedside marker of severe albeit transient microcirculatory dysfunction after stenting in patients with AMI and identifies a high-risk subset of patients who may require extra care. Alternatively, the value of STR as a surrogate end point in clinical trials testing the efficacy of adjunctive therapies aimed at preventing no-reflow was recently suggested20 and warrants further investigation.

Our results should be interpreted with caution. First, we used the prespecified 50% STR cutoff, whereas the 70% cutoff has been proposed.4 In the present study, patients with ≥70% STR (n = 25) had higher CVR (1.7 [1.5 to 1.9] versus 1.4 [1.3 to 1.5], P = 0.02), higher global (56% [50 to 67] versus 48% [42 to 57], P = 0.04) and regional (21% [16 to 23] versus 15% [14 to 17], P = 0.07) LV ejection fractions, and smaller scintigraphic infarct size (16% [11 to 19] versus 26% [12 to 42], P = 0.04) than patients with STR between 50% and 70% (n = 8), suggesting an incremental improvement of microcirculatory perfusion at this higher cutoff. However, by using the 70% cutoff, our results remained unchanged (data not shown). Second, it is unclear whether the marginal difference of time to treatment observed in our study between patients with ≥50% and <50% STR was biologically meaningful, even though we found no relation between, on the one hand, pain-to-CCU or pain-to–TIMI 3 times, and, on the other hand, STR or poststenot CPR (data not shown). Finally, our findings in patients with AMI successfully revascularized with primary angioplasty and systematic stenting cannot be extrapolated to patients with AMI with other forms of revascularization or no revascularization at all.

Appendix

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References
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