Influence of Residual Perfusion Within the Infarct Zone on the Natural History of Left Ventricular Dysfunction After Acute Myocardial Infarction: A Myocardial Contrast Echocardiographic Study

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Objectives. This study used myocardial contrast echocardiography to investigate the extent of residual perfusion within the infarct zone in a select group of patients with recently reperfused myocardial infarction and evaluated its influence on the ultimate infarct size.

Background. Limited information is available on the status of myocardial perfusion within postischemic dysfunctional segments at predischarge and on its influence on late regional and global functional recovery.

Methods. Twenty patients with acute myocardial infarction were selected for the study. Patients met the following inclusion criteria: 1) single-vessel coronary artery disease; 2) patency of infarct-related artery with persistent postischemic dysfunctional segments at predischarge; 3) stable clinical condition up to 6 months after hospital discharge. All selected patients underwent coronary angiography and myocardial contrast echocardiography before hospital discharge and repeated the echocardiographic examination 6 months later. Patients were grouped according to the pattern of contrast enhancement in predischarge dysfunctional segments.

Results. In nine patients (group I), the length of segments showing abnormal contraction coincided with that of the contrast defect segments. In the remaining 11 patients (group II), postischemic dysfunctional segments were partly or completely reperfused. There was no difference between the two groups in asynergic segment length at predischarge (7.3 ± 2.5 vs. 7.2 ± 4.3 cm, p = NS). At follow-up study, asynergic segment length was significantly reduced in group II patients, whereas no changes were observed in group I patients (from 7.2 ± 4.3 to 4.7 ± 3.7 cm, p < 0.005; and from 7.3 ± 2.5 to 7.5 ± 2.9 cm, p = NS, respectively).

Conclusions. Among patients with a predischarge patent infarct-related artery, further improvement in regional and global function may be expected during follow-up when residual perfusion in the infarct zone is present.

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Several clinical trials have provided convincing evidence that thrombolytic therapy may improve the survival rate in patients with acute myocardial infarction by limiting the extent of infarct size (1-3). Whereas the rate of improvement in regional function seems to be related to the duration of coronary occlusion, the degree of functional recovery is related to the extent of necrosis (4,5). Previous studies comparing function before and after successful thrombolytic therapy showed that contractile function recovery in the salvaged tissue may require a long time ("stunned myocardium") (6,7). Although most of the improvement takes place within the 1st 7 to 10 days after reperfusion (8–11), it is not clear when it can be considered complete. Furthermore, the exact amount of viable muscle necessary for functional recovery after reperfusion therapy is not known.

Myocardial contrast echocardiography is a validated technique for the assessment of risk area soon after acute myocardial infarction (12–15). Moreover, recent studies (16–19) have demonstrated that the extent of myocardial contrast defect shortly after reperfusion in patients with myocardial infarction is a strong predictor of the degree of short-term functional recovery. However, limited information is available with regard to the status of myocardial perfusion in dysfunctional segments before hospital discharge and its influence on late functional recovery (20,21).

This study therefore used myocardial contrast echocardiography to investigate the extent of residual perfusion within the infarct area in a select group of patients with recent reperfused myocardial infarction and evaluated its influence on the ultimate infarct size.
Methods

Study patients. The study group included 20 patients (14 men, 6 women; mean [±SD] age 55 ± 6 years) admitted to the coronary care unit with transmural acute myocardial infarction diagnosed by a history of typical chest pain. ST segment elevation >2 mm in two contiguous electrocardiographic leads and a greater than threefold increase in serum creatinine kinase (CK) level. Patients were prospectively selected for this investigation if they met the following inclusion criteria: 1) single-vessel coronary artery disease without previous myocardial infarction or other cardiac disease; 2) patency of the infarct-related coronary artery with complete antegrade perfusion (Thrombolysis in Myocardial Infarction trial [TIMI] flow grade 3) (22) at predischARGE coronary angiography; 3) persistence of postschismic dysfunctional segments before discharge; 4) stable clinical condition with no major clinical events (death, angina, reinfection, coronary artery bypass graft surgery or percutaneous transluminal coronary angioplasty) up to 6 months after hospital discharge; and 4) technically adequate echocardiographic studies allowing detection of the entire left ventricular endocardial contours during cardiac catheterization. All patients were treated with intravenous thrombolytic therapy within 6 h from the onset of symptoms. Twelve patients received tissue-type plasminogen activator, and eight received urokinase, followed by heparin (infused for 3 days and then administered subcutaneously until hospital discharge).

All patients underwent coronary angiography and myocardial contrast echocardiography before hospital discharge and repeated the echocardiographic examination 6 months later (192 ± 21 days).

Informed written consent was obtained from all patients for both cardiac catheterization and contrast echocardiography protocol. The protocol was approved by the La Sapienza University of Rome Medical Ethical Committee.

Coronary artery cineangiography. Coronary angiography was performed in all patients 10 ± 3 days after the onset of acute myocardial infarction using the standard procedure and equipment. The Judkins technique was used, and multiple projections, including cranial and caudal angulations, were recorded at 30 frames/s.

Coronary angiography disclosed a single atherosclerotic lesion in the left anterior descending coronary artery in 11 patients, the left circumflex coronary artery in 7 and the right coronary artery in the remaining 2. No coronary collateral flow was present at coronary angiography.

Measurement of residual coronary stenosis. Severity of residual coronary narrowing after reperfusion was expressed in terms of percent area stenosis. The best cineangiographic images were selected and digitized by an array processor-based computer for medical image processing (Mipron System, Kontron). For quantitative analysis of coronary artery stenosis, a previously described and validated method (23,24) was used.

Myocardial contrast echocardiography. Myocardial contrast echocardiography was performed after routine predischARGE coronary angiography, by manual injection of 0.2 to 0.5 ml of sonicated 5% human serum albumin solution separately into the right and left main coronary arteries. A commercially available sonicating system was used (model W-225; Heat Systems, Ultrasones Corp.). The echo contrast agent was prepared under sterile conditions 1 h before the study according to a previously described protocol (24,25). The echo contrast agent produced by this system was recorded at hold end-tidal respiration on high fidelity videotape, from ~10 s before the injection of the contrast agent until the contrast enhancement was no longer evident. Gain settings were optimized at the beginning of each study and kept constant throughout the study.

Two-dimensional echocardiographic images were obtained by a commercially available phased-array system (Hewlett-Packard Sonos 1000) with a 3.5-MHz transducer. To allow better visualization of the left ventricle, a foam wedge was placed behind the patient’s back to tilt the chest to a 30° left lateral position. Because of technical difficulties in performing an adequate echocardiographic study from the parasternal approach during cardiac catheterization, standard apical two- and four-chamber views were always recorded.

Data analysis. Apical views were analyzed to assess risk area and regional wall motion abnormalities. For the study design, only patients with adequate antegrade coronary perfusion were selected; hence, no collateral flow was detected in our study group after contrast agent injection separately into the right and left coronary arteries (26). For this reason, only images obtained after contrast agent injection into the infarct-related artery were used for analysis. The apical view allowing the best visualization of postschismic dysfunctional segments was chosen for the predischARGE myocardial contrast echocardiographic study. The same echocardiographic view was repeated during follow-up examination. Each of these studies was analyzed in blinded manner by two experienced observers. The risk area was determined in the end-diastolic frame of the postinjection cycle showing the best delineation between contrast-enhanced and nonenhanced myocardium. To quantify the size of the risk area, the endocardial length of the contrast defect (CD) was measured, as previously described by Ito et al. (17). Similarly, to assess the extent of postschismic abnormal contractile segment, the endocardial length of the end-diastolic segment showing dyskinesia/akinesia (no inward systolic endocardial motion) was measured. The relative change in asynergic segment length from predischARGE (AS₁) to the follow-up echocardiographic study (AS₂) was defined as [(AS₂ - AS₁)/AS₁] × 100.

Patients were classified according to the pattern of contrast enhancement in predischARGE dysfunctional segment by the following formula: (AS₁ - CD)/AS₁. The presence or
Table 1. Clinical, Angiographic and Echocardiographic Data

<table>
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<tr>
<th>Pt No./Gender</th>
<th>Age (yr)</th>
<th>EF₁</th>
<th>EF₂</th>
<th>(EF₂ - EF₁)/EF₁</th>
<th>AS₁</th>
<th>AS₂</th>
<th>CD</th>
<th>(AS₂ - AS₁)/AS₁</th>
<th>(AS₁ - CD)/AS₁</th>
<th>IRA</th>
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<td>2.9</td>
<td>2.5</td>
<td>6.3</td>
<td>0.03</td>
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| Group II      |         |     |     |                 |     |     |    |                 |                 |      |
| 10/F          | 64      | 50  | 60  | 20.0            | 6.3 | 5.1 | 4.7| -19.0           | 0.25           | LAD |
| 11/M          | 56      | 30  | 40  | 33.3            | 15.2| 10.4| 9.1| -31.6           | 0.40           | LAD |
| 12/M          | 52      | 30  | 35  | 16.7            | 13.5| 10.8| 7.0| -20.0           | 0.48           | LCx |
| 13/M          | 50      | 55  | 60  | 9.1             | 9.5 | 7.3 | 4.5| -23.2           | 0.53           | LAD |
| 14/M          | 56      | 35  | 50  | 42.9            | 9.7 | 6.3 | 4.2| -35.1           | 0.57           | LAD |
| 15/F          | 52      | 50  | 60  | 20.0            | 6.2 | 4.1 | 2.4| -33.9           | 0.61           | LAD |
| 16/M          | 50      | 55  | 60  | 9.1             | 4.9 | 2.9 | 0.0| -40.8           | 1.00           | LCx |
| 17/M          | 56      | 60  | 60  | 0.0             | 4.7 | 2.6 | 0.0| -44.7           | 1.00           | LCx |
| 18/M          | 52      | 55  | 65  | 18.2            | 4.2 | 2.6 | 0.0| -38.1           | 1.00           | LAD |
| 19/F          | 50      | 60  | 75  | 25.0            | 3.6 | 0.0 | 0.0| -100.0          | 1.00           | LAD |
| 20/M          | 53      | 70  | 70  | 0.0             | 1.5 | 0.0 | 0.0| -100.0          | 1.00           | LAD |
| Mean          | 53.7    | 50.0| 57.7| 17.7            | 7.2 | 4.7 | 2.9| -44.2           | 0.71           |      |
| SD            | 4.1     | 13.0| 11.9| 13.1            | 4.3 | 3.7 | 3.2| 28.8            | 0.29           |      |

AS₁ = asynergic segment length at predischarge; AS₂ = asynergic segment length at follow-up; CD = contrast defect; EF₁ = ejection fraction at predischarge; EF₂ = ejection fraction at follow-up; IRA = infarct-related artery; LAD = left anterior descending coronary artery; LCx = left circumflex coronary artery; Pt = patient; RCA = right coronary artery.

absence of coincidence between the length of segments showing abnormal contraction and that with contrast defect was the only criterion for patient classification.

Left ventricular volume was calculated by the area-length method. Left ventricular ejection fraction was computed according to the standard formula: [{(End-diastolic volume - End-systolic volume)/End-diastolic volume}] × 100. The relative change in left ventricular ejection fraction from predischarge (EF₁) to follow-up (EF₂) echocardiographic study (in percent) was defined as [{(EF₂ - EF₁)/EF₁}] × 100.

Reproducibility of myocardial contrast echocardiography data. Reproducibility of measurements of the endocardial length of contrast defect was assessed in five patients by repeating intracoronary injections of sonicated albumin and by measuring contrast enhancement in the same region during both injections. Mean interinjection percent error was 4.2 ± 1.6%. Intraobserver and interobserver variability was determined by measuring contrast defect in 10 injections twice by the same observer and by two independent observers. Mean percent intraobserver variability for contrast defect length was 3 ± 1.2% and mean percent interobserver error was 4.8 ± 1.8%. Intraobserver and interobserver variability of the quantification of the asynergic segment length was evaluated by measuring this variable in 10 patients twice by the same operator and by two experienced operators. Mean percent intraobserver variability for asynergic segment length was 3.9 ± 1.5% and mean percent interobserver error was 4.4 ± 2.8%.

Statistical analysis. Data are expressed as mean value ± SD. Data of the two groups at predischarge were compared by one-way analysis of variance. Changes in the measured variables during follow-up were estimated by one-way analysis of variance for repeated measures. When significant differences were detected, individual mean values were compared using post hoc tests. Correlation between relative changes in asynergic segments length and ejection fraction from predischarge to follow-up and the pattern of contrast enhancement in predischarge dysfunctional segments was performed by linear regression analysis; p < 0.05 was considered significant.

Results

In-hospital data. No patient had angina or other complications before hospital discharge (Table 1). At predischarge, 14 patients were in New York Heart Association functional class I; 3 in class II and the remaining 3 in class III.

In nine patients (group I), the value for (AS₁ - CD)/AS₁
Figure 1. Bar graph showing asynergic segment length changes from predischarge to follow-up in group I and II patients. There was no difference in asynergic segment length at baseline between the two groups. At follow-up, asynergic segment length was significantly reduced in group II patients with residual perfusion in the infarct area; no changes were observed in group I patients.

Figure 2. Bar graph showing ejection fraction changes from predischarge to follow-up in group I and II patients. There was no difference in ejection fraction at baseline between the two groups. Patients with residual perfusion in the infarct area (group II) showed improvement in this variable from predischarge to follow-up (from 50 ± 13% to 57.7 ± 11%, p < 0.0001). Conversely, ejection fraction remained unchanged in group I patients.

was 0.01 ± 0.03 (range 0 to 0.06), suggesting that the length of segments showing abnormal contraction coincided with that of the contrast defect segment. In the remaining 11 patients (group II), this value was 0.71 ± 0.29 (range 0.25 to 1.00). In these patients, a contrast effect was observed at the edges of the ischemic area, suggesting that normally perfused but dysfunctioning segments were present at the lateral borders of the damaged myocardium. Consequently, the extent of contrast defect was significantly less in group II patients (7.2 ± 2.5 vs. 2.9 ± 3.2, p < 0.005). The perfusion boundary was somewhat irregular. However, contrast enhancement was never observed within the body of the ischemic area. Thus, regions with any degree of contrast enhancement were clearly distinguished from those with no contrast enhancement.

The two groups were similar with regard to mean age (58 ± 8 vs. 53 ± 4 years, p = NS), asynergic segment length at predischarge (7.3 ± 2.5 vs. 7.2 ± 4.3 cm, p = NS), mean CK and CK-MB fraction levels (490 ± 58 vs. 510 ± 64 U/liter, p = NS; 15 ± 3 vs. 17 ± 2%, p = NS, respectively), length of time from onset of symptoms to initiation of thrombolytic therapy (3.5 ± 0.9 vs. 3.2 ± 1.7, p = NS) and percent residual coronary artery stenosis after reperfusion (48 ± 15 vs. 51 ± 13%, p = NS).

Follow-up data. At follow-up study, asynergic segment length was significantly reduced in group II patients, whereas no changes were observed in group I patients (from 7.2 ± 4.3 to 4.7 ± 3.7 cm, p < 0.005; from 7.3 ± 2.5 to 7.5 ± 2.9 cm, p = NS, respectively) (Table 1, Fig. 1). An expansion of the asynergic segment was detected in three group I patients.

Finally, a close relation was found between the relative change in asynergic segment length from predischarge to follow-up and the pattern of contrast enhancement in pre-
discharge dysfunctioning segments: $y = 3.69 - 67.1x$ ($r = -0.87$, SEE = 16.2, $p < 0.0001$) (Fig. 3); only a weak correlation was found between ejection fraction changes and extent of reperfusion within the infarct area: $y = 4.4 + 13.1x$ ($r = 0.39$, SEE = 13.0, $p < 0.09$).

**Discussion**

Recently, Ito et al. (16,17) demonstrated that evaluation of the extent of myocardial contrast defect soon after reperfusion is a strong predictor of the degree of short-term functional recovery of the postischemic myocardium. The extent of contrast defect is strictly dependent on the magnitude of collateral vessel-derived residual blood flow and on the prompt achievement of coronary reflow throughout the infarct-related artery (16-19,26). These data have been recently confirmed by Clements et al. (27), who showed that the presence of anterograde or collateral flow to the infarct zone before direct angioplasty in acute evolving infarction results in a smaller infarct size after angioplasty. However, less attention has been given to the status of myocardial perfusion within postischemic dysfunctional segments at predischarge.

The present study further confirms that myocardial contrast echocardiography is very useful in assessing tissue viability after acute myocardial infarction (16-19) and that knowledge of the status of myocardial perfusion before hospital discharge may add useful clinical and prognostic information.

The coincidence between the length of segments showing abnormal contraction with that of the contrast defect segments identifies the definitive infarct size without further improvement in left ventricular regional and global function during follow-up. Thus, this pattern characterizes a subset of patients who have contractile abnormalities that are irreversible, and no further reduction in infarct size can be expected after hospital discharge. However, the presence of residual perfusion in the dysfunctional segments may be followed by a further reduction in infarct size after hospital discharge, with a slight but significant increase in global left ventricular function (Fig. 4). Thus, the natural history of regional dysfunction after effective coronary reperfusion in patients with single-vessel coronary artery disease and without clinical
evidence of restenosis during follow-up is strongly influenced by the status of residual perfusion in the infarct zone. It has been shown (28,29) that among patients surviving Q wave acute myocardial infarction, the long-term clinical outcome in those with a patent infarct-related artery is more benign than that observed in patients without coronary reflow. This study provides new information showing that among patients with a predischarge patent infarct-related artery, further improvement in regional and global function can be expected when residual perfusion in the infarct zone is present. The presence of normally perfused dysfunctional regions at the lateral borders of the damaged myocardium has been recently detected with myocardial contrast echocardiography by Nanto et al. (30) during acute and transient myocardial ischemia. Our data showed that a “functional border zone” at the lateral margins of the ischemic area can also be identified by myocardial contrast echocardiography in patients with recent myocardial infarction, suggesting that the extent of wall motion abnormalities at predischarge may overestimate the extent of the definitive infarct size.

A close relation was found between the relative change in asynergic segment length from predischarge to follow-up and the pattern of contrast enhancement in predischarge dysfunctional segments. Conversely, only a weak correlation was found between ejection fraction changes and the extent of reperfusion within the infarct area. These data further confirm that regional indexes of left ventricular function are more sensitive than global indexes in evaluating the extent of salvage and the severity of regional stunning after reperfusion (31).

Time course of functional recovery after coronary reperfusion. Several studies in patients with recent myocardial infarction have shown that functional improvement takes place largely within the 1st 7 to 10 days after reperfusion (7-11,16,17). However, experimental and clinical studies have found a further, late recovery of regional left ventricular function after acute myocardial infarction (29,31-36). Our results confirm that a further reduction in posts ischemic regional myocardial dysfunction may be observed 6 months after acute myocardial infarction. This functional improvement was mainly related to the presence of residual myocardial perfusion in predischarge dysfunctional segments. Our data are consistent with the hypothesis that in some cases myocardial stunning may persist >10 days after coronary reperfusion (31,35). The “stunned” myocardium needs time to repair damaged metabolic processes and to reconstitute high energy phosphate stores. Alternatively, the delayed recovery of left ventricular regional function after reperfusion may be explained by the presence of a high grade, flow-limiting residual stenosis that could cause persistent subendocardial ischemia, or “hibernation” (31). In the days after administration of thrombolytic therapy, gradual lysis of the thrombus or remodeling of the plaque, or both, could lessen the severity of the stenosis and bring about a gradual increase in subendocardial flow, thereby resulting in slow functional recovery (31). In this study, only patients with complete anterograde coronary perfusion at predischarge were selected. However, coronary angiography was not performed soon after thrombolytic therapy. Thus, even though all selected patients underwent thrombolytic therapy within 6 h from the onset of symptoms, the exact time of complete coronary reflow is not known. Therefore, both of these mechanisms may be responsible for the delayed functional recovery observed in this study.

Study limitations. Serial echocardiographic control studies between predischarge and follow-up were not performed. Thus, the exact time of the ultimate infarct size cannot be identified by this study.

Coronary angiography was not performed at follow-up. Thus, although all patients selected were in stable clinical condition, with no major clinical events up to 6 months after hospital discharge, the expansion in infarct size observed during follow-up in three group I patients may be due to a late asymptomatic coronary reocclusion (11,37).

A no-reflow phenomenon cannot be demonstrated in the present study. For this purpose, imaging the heart days after reperfusion might be not useful (38). However, experimental data have described an increase in the size of the no-reflow zone with time, after the moment of reperfusion (39). Thus, the size of contrast defect observed at predischarge, despite infarct-related artery patency, might represent the definitive extent of the no-reflow area. More myocardial contrast echocardiographic studies are needed to compare admission and predischarge contrast defects and to better understand whether the no-reflow state worsens over time in humans (40).

Clinical implications. Previous studies (16,17) demonstrated the usefulness of contrast echocardiography in the evaluation of myocardial reflow by performing coronary angiography in the first hours after acute myocardial infarction. The present study shows that in patients surviving acute myocardial infarction, the predischarge analysis of the ratio between myocardial contrast defect and dysfunctional segments is a simple and useful method to better distinguish still viable from necrotic myocardial regions. This analysis provides new insights into the influence of residual perfusion in the infarct area on the natural course of left ventricular remodeling after acute myocardial infarction. The presence of residual perfusion in the infarct zone may prevent infarct expansion and may allow further late reduction in the ultimate infarct size. Thus, all efforts to maintain a patent infarct-related artery and to prevent coronary reocclusion are justified.

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References


