Opposite Patterns of Left Ventricular Remodeling After Coronary Revascularization in Patients With Ischemic Cardiomyopathy
Role of Myocardial Viability

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Background—In patients with ischemic cardiomyopathy, left ventricular (LV) remodeling is an important prognostic indicator. The precise relation between viable myocardium, revascularization, and ongoing or reversed remodeling is unknown and was evaluated in the present study.

Methods and Results—A total of 100 patients with ischemic cardiomyopathy underwent dobutamine stress echocardiography to assess myocardial viability and LV geometry (volumes and shape). At a mean of 10.2 months and 4.5 years after revascularization, resting echocardiography was repeated to evaluate LV remodeling. Long-term follow-up (mean 5±2 years) data were obtained. According to dobutamine stress echocardiography, 44 patients (44%) were defined as viable (≥4 viable segments) and 56 as nonviable. After revascularization, 40 patients (43%) had ongoing LV remodeling and 53 (57%) did not (in 7 patients who died early after revascularization, postoperative echocardiographic evaluation was not available). On multivariable analysis, the number of viable segments was the only predictor of ongoing LV remodeling (OR 0.60, 95% CI 0.48 to 0.75; P<0.0001). The likelihood of LV remodeling decreased as the number of viable segments increased. During the follow-up, reverse remodeling was present in viable patients, whereas in nonviable patients, LV volumes significantly increased, which indicates ongoing LV remodeling. At follow-up, viable patients also showed a persistent improvement of heart failure symptoms and fewer cardiac events than nonviable patients (P<0.05).

Conclusions—In patients with ischemic cardiomyopathy, a substantial amount of viable myocardium prevents ongoing LV remodeling after revascularization and is associated with persistent improvement of symptoms and better outcome. (Circulation. 2004;110:2383-2388.)

Key Words: remodeling ■ revascularization ■ cardiomyopathy

In patients with ischemic cardiomyopathy coronary revascularization is a therapeutic option that may improve left ventricular (LV) function and prognosis.1-10 A substantial amount of viable myocardium is necessary to obtain improvement of LV ejection fraction (LVEF), symptoms and prognosis.2-10 It has been suggested that the preservation of LV geometry in patients with ischemic cardiomyopathy may also be an important end point after revascularization.11 The restoration of adequate flow to viable myocardium may prevent LV distortion and ongoing LV remodeling, and thus prevent progressive heart failure and death. Some studies showed that revascularization can prevent the LV remodeling process in patients with recent myocardial infarction and residual viability.12,13 Information on the effect of viability and revascularization on LV remodeling in patients with chronic LV dysfunction is scarce.14,15 Accordingly, in the present study, the role of myocardial viability in the LV remodeling process after revascularization has been evaluated in a large cohort of patients with ischemic cardiomyopathy. In addition, long-term clinical follow-up has been obtained.

Methods

Study Population
The study population existed of 106 patients (89 men, age 61±10 years) with ischemic cardiomyopathy and heart failure symptoms already scheduled for revascularization according to clinical criteria (symptoms, presence/absence of ischemia, and angiographic findings). Sinus rhythm was present in all patients. Ninety-eight patients (93%) had a history of myocardial infarction that had occurred ≥6 months before study entrance (median 3 years, range 0.8 to 23 years). Patients with moderate to severe valvular disease were excluded.
Study Protocol

Patients with ischemic cardiomyopathy were studied prospectively to evaluate LV remodeling, LV ejection fraction (LVEF), and cardiac events during a long-term follow-up after revascularization and their relation to myocardial viability. Before revascularization (study 1, baseline) resting 2D echocardiography was performed to assess regional wall-motion abnormalities and LV geometry, followed by dobutamine stress echocardiography (DSE) to evaluate myocardial viability. After revascularization, resting 2D echocardiography was repeated at a mean of 10.2 months (study 2) and 4.5 years (study 3) to assess changes in LV geometry. Within 2 weeks of each echocardiographic study, radionuclide ventriculography (RVN) was performed to assess LVEF by an independent technique. Before and sequentially after revascularization, functional status was evaluated by structured clinical interviews. Cardiac events were obtained during a 2- to 2-year follow-up. The local ethics committee approved the protocol, and all patients gave informed consent.

Echocardiographic Studies

All echocardiograms were performed with a Sonos-5500 device (Hewlett-Packard, PMS) equipped with a second-harmonic 1.8- to 3.5-MHz transducer. Standard views of the LV were obtained.14

Myocardial Viability

Low- to high-dose DSE (up to 40 μg · kg⁻¹ · min⁻¹ plus 2 μg of atropine, if necessary) was performed as described previously.6 Interpretation of DSE studies was performed offline from cine loops by 2 experienced observers blinded to the clinical data. Interobserver and intraobserver agreement for analysis of DSE studies was 92% and 94%, respectively.16 Regional function was scored with a 16-segment, 5-point scoring model as follows: 1, normal; 2, mildly hypokinetic; 3, severely hypokinetic; 4, akinetic; and 5, dyskinetic. The wall-motion score index (WMSI) was calculated by dividing the summed wall-motion score by the number of segments. Myocardial viability was evaluated only in severely dysfunctional segments (score 3 to 5). Segments showing a sustained improvement in wall motion up to the high dose and segments with an ischemic pattern (biphasic response or worsening of the wall motion) during DSE were considered viable.4,6,11 Segments with unchanged wall motion or with akinesis that became dyskinesia were considered nonviable.4,6 A patient was defined as viable in the presence of ≥4 viable segments and as nonviable in the presence of <4 viable segments.8 This definition is based on previous work with receiver operator characteristic curve analysis that showed that recovery of function may be predicted in the presence of ≥4 viable segments.6

LV Geometry (Volumes and Sphericity)

LV volumes and LV sphericity index (LVSI) were measured from the resting echocardiography (before and sequentially after revascularization). All measurements were performed offline in random order by 2 experienced observers blinded to patient data and study time. LV volumes were measured with the biplane Simpson’s rule.17 The end-diastolic and end-systolic volumes were indexed (LVEDVI and LVESVI, respectively) by the body surface area. LVEDVI ≤55.5 ± 5.7 mL/m² and LVESVI ≤22.1 ± 4.9 mL/m² were considered normal values.18,19 Interobserver and intraobserver variability for the measurement of LV volumes was 5% and 7%, respectively. An increase >15% in the LVEDVI or LVESVI after revascularization defined ongoing LV remodeling. Absolute changes in LVEDVI and LVESVI were expressed as the change (Δ) between the volume late after revascularization (at 4.5 years) and the baseline volume. The LVSI was derived by the ratio of LV short- to long-axis dimensions in the end-systolic apical 4-chamber view.16 The higher the LVSI, the more spherical the shape. Intraobserver and interobserver agreement for assessment of LVSI was 95% and 91%, respectively.

Assessment of Improvement in LVEF

RVN was performed at rest with the patient in the supine position after administration of 740 MBq of 99mTc. Images were acquired with a small-field-of-view gamma camera (Orbiter, Siemens Corp) oriented in the 45° left anterior oblique position with a 5° to 10° caudal tilt. LVEF was calculated from the 45° left anterior oblique view by an automated technique. An improvement in LVEF ≥5% at study 2 or 3 was considered clinically significant.6

Symptoms and Long-Term Follow-Up

At each study point, patients’ New York Heart Association (NYHA; for heart failure symptoms) and Canadian Cardiovascular Society (CCS) class (for angina) were defined by an independent physician blinded to all data. Long-term follow-up was obtained by chart review and telephone contact. Events included cardiac death, myocardial infarction, hospitalization for heart failure, and major ventricular arrhythmias (ventricular tachycardia/fibrillation). Moreover, the duration of stay in the intensive care unit after surgery was noted, as was the presence of low-output syndrome (defined as the need for high dosages of inotropic medication and/or intra-aortic balloon pumping to sustain adequate hemodynamic status).

Statistical Analysis

Continuous data were expressed as mean ± SD and compared with the Student’s t test for (unpaired) samples, as indicated. Proportions for dichotomous data were compared by χ² analysis. Repeated measurements were analyzed by 2-way ANOVA to evaluate differences across time and between different groups. Univariable and multivariable logistic regression analyses were performed to characterize predictors of ongoing LV remodeling. Categorical variables included diabetes, anterio/ inferior Q-wave myocardial infarction, persistent ST-segment elevation, ACE inhibitor and/or β-blocker therapy, and mode and completeness of revascularization. Continuous variables included age, WMSI at rest, baseline LV volumes and LVEF, and number of viable and nonviable segments. To define the predictive value for LV remodeling of each DSE pattern that indicated viable myocardium, the numbers of segments with sustained improvement, biphasic response, or worsening of the wall motion were included in the analyses. All variables entered the multivariable stage, regardless of the results of univariable analyses. Multivariable regression was then performed by stepwise backward deletion. All variables with a probability value <0.25 remained in the final model. Linear regression analysis was performed to evaluate the relation between the amount of viable myocardium and the changes (Δs) in LV volumes. Cardiac event rate was evaluated by Kaplan-Meier analysis. Differences between curves were tested with log-rank χ² statistics. For all tests, a probability value <0.05 was considered significant.

Study Population

CABG was performed in 85 patients (80%) and PTCA in 21 patients (20%). Revascularization procedures were performed within 1 month of the DSE and were complete in all but 1 patient, who had perioperative myocardial infarction (peak creatine kinase level of 2640 IU/L). Five patients who underwent associated procedures influencing LV remodeling (aneurysmectomy [n=3] and mitral valve repair [n=2]) were subsequently excluded. One patient was lost to follow-up. Cardiac death occurred early after revascularization in 6 patients, and 1 patient died of progressive heart failure at 6 months after revascularization. These patients were included in the follow-up analysis, but sequential evaluations after revascularization were not available in these 7 patients. One patient had an acute myocardial infarction 5 months after study 2. In this patient, ongoing LV remodeling was already present at study 2. Study 3 (at 4.5 years) was not performed in 8 patients who died (3 noncardiac deaths) during the follow-up period.
Baseline Echocardiographic Data
All patients presented with moderate to severe LV dilatation. LVEDVI and LVESVI were on average 108±34 and 71±31 mL/m², respectively. Analysis of wall motion showed that 939 segments (59%) were severely dysfunctional. On average, patients had 9.3±4.7 severely dysfunctional segments. During DSE, 392 segments (42%) were viable: 176 (19%) had a biphasic response, 185 (20%) had sustained improvement, and 31 (3%) had worsening of wall motion. The remaining 597 segments (58%) were nonviable.

LV Geometry After Revascularization
After revascularization, patients were divided into group 1 (40 patients [43%] with ongoing LV remodeling) and group 2 (53 patients [57%] without ongoing LV remodeling). Overall, WMSI and LVEF improved significantly after revascularization in group 2 but remained unchanged in group 1 (P<0.04). In addition, a trend toward a higher prevalence of persistent ST elevation was present in group 1. Finally, the number of viable and nonviable segments was significantly different between the 2 groups. At baseline, in groups 1 and 2, medications included ACE inhibitors in 67% and 64% of the patients, respectively (P=NS) and β-blockers in 48% and 66% of the patients, respectively (P=NS). At study 2 and 3, groups 1 and 2 were comparable for use of ACE inhibitors (82% versus 75% and 74% versus 79%, respectively, both P=NS) and β-blockers (25% versus 38% and 49% versus 52%, respectively, P=NS).

| TABLE 1. WMSI, Systolic Blood Pressure/End-Systolic Volume Ratio, and LVEF at Different Study Points |
|----------------------------------|----------------------------------|----------------|----------------|
| **Group 1 (Remodeling)** | **Group 2 (No Remodeling)** | **Study 1** | **Study 2** | **Study 3** | **Study 1** | **Study 2** | **Study 3** | **P** |
| WMSI | SBP/ESV | LVEF, % | WMSI | SBP/ESV | LVEF, % | WMSI | SBP/ESV | LVEF, % | P |
| 2.7±0.6 | 1.5±0.3 | 32±8 | 2.8±0.7 | 1.6±0.6 | 32±9 | NS | NS | NS | <0.01 |
| 2.7±0.7 | 1.6±0.7 | 31±10 | 2.6±0.7 | 2.6±1.6 | 38±11 | NS | NS | NS | <0.001 |
| 2.8±0.61 | 1.5±0.7 | 31±9 | 2.7±0.5 | 2.7±0.61.4 | 41±11 | NS | NS | NS | <0.001 |

SBP/ESVI indicates systolic blood pressure/end-systolic volume ratio.
*P<0.001, group 1 vs group 2 by ANOVA.

| TABLE 2. Baseline Characteristics |
|---------------------------------|----------------|----------------|----------------|
| **Group 1 (Remodeling)** | **Group 2 (No Remodeling)** | **Study 1** | **Study 2** | **Study 3** | **Study 1** | **Study 2** | **Study 3** | **P** |
| Age, y | Male, n (%) | CCS class | NYHA class | Hypertension, n (%) | Hypercholesterolemia, n (%) | Smoking, n (%) | Family history of CAD, n (%) | Diabetes, n (%) | History of myocardial infarction, n (%) | Q-wave myocardial infarction, n (%) | Anterior | Septal | Lateral | Inferoposterior | Persistent ST elevation, n (%) | Stenotic vessels, n | LVEF, % | Severely dysfunctional segments, n | Viable segments, n | Biphasic response, n | Sustained improvement, n | Worsening, n | Unchanged, n | **P** |
| 60±11 | 35 (87) | 2.8±0.6 | 2.6±1.2 | 29 (72) | 18 (45) | 20 (50) | 24 (60) | 4 (10) | 39 (97) | 36 (90) | 26 (65) | 14 (35) | 7 (17) | 16 (40) | 21 (52) | 2.5±0.7 | 32±9 | 8.6±4.2 | 2.2±2.2 | 1±1.6 | 0.9±1.7 | 0.3±0.7 | 6.4±4.2 | NS |
| 61±10 | 42 (80) | 2.7±0.6 | 2.9±0.9 | 38 (73) | 23 (43) | 23 (57) | 28 (53) | 4 (7) | 51 (92) | 40 (75) | 22 (41) | 13 (24) | 6 (11) | 28 (53) | 17 (32) | 2.6±0.6 | 33±9 | 9.6±5.1 | 6.1±3.3 | 2.4±2.7 | 2.7±2.4 | 0.4±0.9 | 4.2±3.4 | <0.01 |

CAD indicates coronary artery disease.
All values are presented as mean±SD or percentage (%).
both \( P=\text{NS} \). In addition, 55\% of patients in group 1 and 49\% of patients in group 2 were taking lipid-lowering drugs at baseline (\( P=\text{NS} \)). The proportion of patients taking lipid-lowering drugs in the 2 groups was similar at study 2 (48\% and 53\%) and at study 3 (both 66\%).

### Predictors of Ongoing LV Remodeling After Revascularization

The presence of anterior Q-wave myocardial infarction and the number of scar segments were positively related to ongoing LV remodeling. The sustained improvement pattern was significantly more related to the occurrence of no remodeling (OR 0.66, 95\% CI 0.5 to 0.8, \( \chi^2=10.9, \ P<0.0001 \)) than was the biphasic response (OR 0.73, 95\% CI 0.5 to 0.9, \( \chi^2=6.7, \ P<0.01 \)). The worsening pattern was not related to LV remodeling. The total number of viable segments (showing sustained improvement, biphasic response, or worsening) was the strongest univariable predictor (OR 0.62, 95\% CI 0.5 to 0.76, \( \chi^2=20.3, \ P<0.0001 \)) and the only multivariable predictor of ongoing LV remodeling (OR 0.60 for each additional viable segment, 95\% CI 0.48 to 0.75, \( \chi^2=18.7, \ P<0.0001 \); Table 3). The likelihood of ongoing LV remodeling decreased for each additional viable segment. When normal (not only dysfunctional) segments were included in the number of viable segments, this variable remained predictive of LV remodeling. Moreover, the \( \Delta \)s in LV volumes were inversely related to the number of viable segments (LVEDVI \( P<0.001 \); LVESVI \( P<0.001 \); Figure 1).

### Myocardial Viability, Remodeling, and Long-Term Follow-Up

After revascularization, the duration of stay in the intensive care unit was 1.4±2 and 3.5±2 days for viable and nonviable patients, respectively (\( P<0.005 \)). Low-output syndrome occurred in 12 viable (27\%) and 29 (52\%) nonviable patients (\( P<0.05 \)). In 2 nonviable patients, an intra-aortic balloon pump was placed.

After revascularization, the majority of viable patients (88\%) showed preserved LV volumes. Conversely, ongoing LV remodeling occurred in the majority of nonviable patients (72\%, \( P<0.0001 \) versus viable patients). On average, in viable patients, LVEDVI did not change significantly during follow-up (from 105±37 to 102±35 up to 98±48 mL, \( P=\text{NS} \) by ANOVA), whereas LVESVI decreased significantly (from 70±34 to 65±31 up to 61±39 mL, \( P<0.05 \) by ANOVA). In nonviable

![Figure 1. Relation between extent of viability and changes (\( \Delta \)s) in LV volumes after revascularization. \( \Delta \)s in LVEDVI (A) and LVESVI (B) decreased as number of viable segments increased.](image)

### Table 3. Predictors of LV Remodeling

<table>
<thead>
<tr>
<th>Predictor</th>
<th>Univariable Analysis</th>
<th>Multivariable Analysis</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>OR</td>
<td>CI</td>
</tr>
<tr>
<td>LVEDVI</td>
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</tr>
<tr>
<td>LVESVI</td>
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<td>0.9–1.1</td>
</tr>
<tr>
<td>Viable segments, n</td>
<td>0.61</td>
<td>0.5–0.7</td>
</tr>
<tr>
<td>Nonviable segments, n</td>
<td>1.16</td>
<td>1.1–1.3</td>
</tr>
<tr>
<td>Biphasic response, n</td>
<td>0.73</td>
<td>0.5–0.9</td>
</tr>
<tr>
<td>Sustained improvement, n</td>
<td>0.66</td>
<td>0.5–0.8</td>
</tr>
<tr>
<td>Worsening, n</td>
<td>0.86</td>
<td>0.5–1.5</td>
</tr>
<tr>
<td>LVEF</td>
<td>0.98</td>
<td>0.9–1.1</td>
</tr>
<tr>
<td>Inferior Q wave</td>
<td>0.61</td>
<td>0.2–1.4</td>
</tr>
<tr>
<td>Anterior Q wave</td>
<td>2.50</td>
<td>1.1–5.8</td>
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<tr>
<td>Persistent ST elevation</td>
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<td>0.7–4.5</td>
</tr>
<tr>
<td>Mode of revascularization</td>
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<td>0.6–6.2</td>
</tr>
<tr>
<td>Diabetes</td>
<td>2.88</td>
<td>0.5–16.6</td>
</tr>
<tr>
<td>WMSI</td>
<td>1.03</td>
<td>0.5–1.9</td>
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<tr>
<td>ACE inhibitors</td>
<td>1.12</td>
<td>0.4–2.6</td>
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<td>( \beta )-Blockers</td>
<td>0.49</td>
<td>0.2–1.1</td>
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<tr>
<td>Age</td>
<td>0.99</td>
<td>0.9–1.1</td>
</tr>
</tbody>
</table>
patients, LV volumes significantly increased over the follow-up period (ongoing LV remodeling; LVEDVI from 105 ± 28 to 121 ± 36 up to 131 ± 50 mL/m², LVESVI from 65 ± 24 to 80 ± 32 up to 88 ± 45 mL/m², both P < 0.001 by ANOVA). Also, LVSI improved in viable patients (from 0.60 ± 0.09 to 0.57 ± 0.1, P < 0.01 by ANOVA), whereas it worsened significantly in nonviable patients (from 0.59 ± 0.09 to 0.65 ± 0.1, P < 0.01 by ANOVA). LVEF significantly improved in 65% of viable patients compared with 25% of nonviable patients (P < 0.001). Changes in LVEF were inversely related to changes in LV EFV (P < 0.01). In addition, viable patients showed persistent improvement of symptoms during follow-up (CCS from 2.6 ± 0.6 to 1.2 ± 0.7 and NYHA from 3.1 ± 0.8 to 1.5 ± 0.5, both P < 0.001 by ANOVA). In nonviable patients, although CCS class improved persistently (from 2.8 ± 0.6 to 1.1 ± 0.9, P < 0.001 by ANOVA), NYHA class did not improve over the follow-up (from 2.9 ± 0.9 to 2.9 ± 0.7, P = NS by ANOVA). During the long-term follow-up, 1 nonviable patient underwent cardiac resynchronization therapy, and 2 patients (1 viable and 1 nonviable) received an intracardiac defibrillator.

Overall cardiac events were significantly less frequent in viable than in nonviable patients (16% versus 41%, P < 0.05; Figure 2A). A trend toward a lower frequency of cardiac death was observed in viable patients (7% versus 21%, P = 0.08). Also, cardiac events were less common in patients without LV remodeling than in those with ongoing LV remodeling (12% versus 42%, P < 0.001; Figure 2B). Cardiac death occurred less frequently in patients without remodeling (4% versus 17%, P = 0.06).

**Discussion**

Improvement in LV function, symptoms, and prognosis is likely to occur after revascularization in patients with ischemic cardiomyopathy and viable myocardium.2-6,10 The findings in the present study indicate that in these patients, substantial myocardial viability prevents ongoing LV remodeling after revascularization and is associated with improvement of symptoms and favorable long-term prognosis.

**Beneficial Effects of Revascularization in Patients With Viable Myocardium**

Several studies have demonstrated that revascularization in patients with ischemic cardiomyopathy and viable myocardium improves regional and global LV function.2-6,20,21 A substantial amount (≥25% of the LV) of viable myocardium is necessary to result in improvement in LVEF.6-10 However, it has been stated that the improvement in LV function may underestimate the benefit of revascularization and that improvement of symptoms and prognosis need to be considered.11 In particular, a recent meta-analysis demonstrated that in patients with viable myocardium, revascularization is associated with a good prognosis.22 Initial data suggested that in patients with ischemic cardiomyopathy, preservation of LV geometry and prevention of ongoing LV remodeling after revascularization can be an additional end point.14,15 Accordingly, in the present study, coronary revascularization resulted in improvement in LVEF only in 65% of viable patients, whereas ongoing LV remodeling was prevented in 88% of viable patients. Hence, after revascularization, some viable patients did improve in terms of LV remodeling, although they failed to improve in LVEF. It is known that LV remodeling after acute myocardial infarction is a major determinant of poor prognosis.23,24 In the present study, ongoing LV remodeling after revascularization of patients with ischemic cardiomyopathy was associated with a higher cardiac event rate (42%) than for patients with no remodeling (13%, P < 0.001).

**LV Remodeling After Revascularization**

Senior and coworkers14 demonstrated that coronary revascularization resulted in a significant reduction of LV volume in 32 patients with ischemic cardiomyopathy and viable myocardium. Conversely, ongoing LV remodeling occurred in viable patients treated medically.14 Dalle Mule et al15 showed a decrease in LV volumes 3 months after revascularization only in patients with substantial viability during 201Tl imaging. Interestingly, an increase in LV volumes was observed in 24 nonviable patients undergone revascularization.15 In the present study, a large number of patients were included, and more importantly, serial measurements of LV volumes were performed at different time points after revascularization. It appeared that LV volumes changed in different directions in relation to myocardial viability. In patients with minimal or absent viability, LV volumes continued to increase, which indicates ongoing LV remodeling, whereas in viable patients, attenuation or even reversion of LV dilation occurred. Uniquely, the present study demonstrated that the extent of viable myocardium was related to the occurrence and extent of LV remodeling. In the individual patient, the likelihood of LV remodeling and the absolute changes in LV volumes decreased for each additional viable segment. Among the different patterns of response to dobutamine that indicated viable myocardium, the sustained-improvement pattern was a stronger univariable predictor of no LV remodeling.
(χ²=10.9) than the biphasic response (χ²=6.7). It may well be that the duration of hibernation before study entrance affected the predictive power of the biphasic response. Also, the extent of nonviable myocardium was a univariable predictor of ongoing LV remodeling, whereas baseline volumes and resting WMSI failed to predict LV remodeling. The inclusion of only patients with moderate to severe LV dilatation and extensive wall-motion abnormalities at rest may explain this finding. Finally, β-blocker therapy before revascularization showed a trend toward a lower occurrence of LV remodeling, and the presence of anterior Q-wave myocardial infarction was a univariable predictor of ongoing LV remodeling. However, only the total number of viable segments remained predictive of LV remodeling in multivariable analysis. The beneficial effect of revascularization of viable myocardium on LV remodeling may have important prognostic implications. In the present study, patients with a substantial amount of viable myocardium, together with an improvement in LV geometry, had persistent improvement in heart failure symptoms and fewer cardiac events during the long-term follow-up. In addition, a trend toward a lower frequency of cardiac death was observed. Conversely, nonviable patients demonstrated ongoing LV remodeling without an improvement in heart failure symptoms after revascularization. Moreover, the cardiac event rate was higher in these patients (Figure 2A). Previous studies have already shown the beneficial effect of revascularization on early and mid-term prognosis.6–10 These findings further extend previous observations to a long-term follow-up and demonstrate that the benefit of revascularization in patients with substantial myocardial viability may be due, at least in part, to prevention of ongoing LV remodeling.

Study Limitations

Angiographic follow-up was not performed after revascularization. Therefore, graft closure or restenosis may have occurred in some patients, affecting LV remodeling. Moreover, during follow-up, patients received different medications according to the attending physician. The possibility that medications influencing LV remodeling (ACE inhibitors and β-blockers) may have affected the results of the present study cannot be excluded. However, at each study point, medications used were comparable in the 2 groups.

Conclusions

In patients with ischemic cardiomyopathy, substantial myocardial viability prevents ongoing LV remodeling after revascularization and is associated with improvement of symptoms and favorable long-term outcome.

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_Circulation_. 2004;110:2383-2388; originally published online October 11, 2004; doi: 10.1161/01.CIR.0000145115.29952.14

_Circulation_ is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
Copyright © 2004 American Heart Association, Inc. All rights reserved.
Print ISSN: 0009-7322. Online ISSN: 1524-4539

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