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Contractile Reserve of Dysfunctional Myocardium After Revascularization: A Dobutamine Stress Echocardiography Study

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Objectives. **We sought to investigate the effects of revascularization on the contractile reserve of dysfunctional myocardium.**

Background. **The improvement in dysfunctional but viable myocardium after revascularization is frequently less than expected from the amount of contractile reserve detected on dobutamine stress echocardiography. The fate of the contractile reserve, when it does not result in an adequate contractile recovery, is unknown.**

Methods. **Basal contraction and contractile reserve of infarct zones were assessed by dobutamine stress echocardiography in 21 postinfarction male patients before and >3 months after revascularization (30 infarct zones; mean ± SD left ventricular ejection fraction 35 ± 8%). An infarct zone wall motion score index (WMSI) was calculated.**

Results. **Before revascularization, contractile reserve was present in 14 infarct zones (12 patients) and absent in 16 (9 patients). After revascularization, ejection fraction increased by 5 ± 4% ($p < 0.01$) in patients classified as positive for contractile reserve and remained unchanged in those classified as negative. New York Heart Association classification improved in 58.3% and 22.2% of patients, respectively. Basal contraction improved in eight zones with previous contractile reserve (57.1%) and in one zone without (6.3%) ($p < 0.01$). Contractile reserve was still evident in 13 zones with previous contractile reserve (93%; 8 with contractile recovery), and it developed in 6 zones without (38%; none with contractile recovery). WMSI values after revascularization were decreased from values before revascularization during low dose dobutamine in zones with and without previous contractile reserve ($p < 0.01$ and < 0.05, respectively).**

Conclusions. **After revascularization, contractile reserve is maintained or even increases in viable infarct zones that do not recover as expected. It may also develop in some infarct zones judged not to be viable before revascularization. This increased contractile reserve may play a role in the functional improvement of patients after revascularization.**

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Contractile reserve during dobutamine stress echocardiography is being used to detect viable myocardium and to predict recovery of regional left ventricular (LV) contraction after revascularization (1–8). However, dysfunctional but viable myocardium may show less improvement in basal contraction after revascularization than that predicted by contractile reserve (3). This phenomenon may be due to the inherent limitations of dobutamine stress echocardiography or, alternatively, it may be an expression of histologic patterns, in which the extent and distribution of fibrosis are unfavorable to contractile recovery in viable tissue but allow a positive inotropic response to catecholamine stimulation. On the other hand, in the presence of a flow-limiting coronary stenosis, dysfunctional but viable LV segments may show a modest inotropic response to dobutamine because of the early occurrence of ischemia (9). The fate of the contractile reserve in the dysfunctional but viable myocardium that remains asynergic after restoration of flow is not known.

In this study, we investigated the effects of revascularization on the contractile reserve of asynergic (infarcted) myocardium by performing dobutamine stress echocardiography in postinfarction patients before and >3 months after revascularization. Specifically, we analyzed 1) whether contractile reserve is maintained in presumably viable infarct zones showing no improvement in basal contraction after revascularization, and 2) whether it develops in infarct zones judged not to be viable before revascularization. We also analyzed changes in basal LV ejection fraction and in New York Heart Association (NYHA) functional class after revascularization in patients with and without contractile reserve in infarct zones.

Methods

Patients. We prospectively enrolled 35 consecutive patients with previous (>1 month) myocardial infarction sched-

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Abbreviations and Acronyms

ECG	=	electrocardiogram, electrocardiographic
LV	=	left ventricular
NYHA class	=	New York Heart Association functional class
SPECT	=	single-photon emission computed tomography (tomographic)
Tl-201	=	thallium-201
WMSI	=	wall motion score index

uled to undergo revascularization of one or more infarct zones. Detection of contractile reserve in infarct zones by dobutamine stress echocardiography was not a prerequisite for revascularization. We excluded patients who had suboptimal echocardiographic images, previous coronary artery bypass graft surgery or percutaneous transluminal coronary angioplasty or who required surgery for valvular or LV aneurysm. We subsequently excluded 14 patients: 5 whose infarct-related artery was unsuitable for grafting, 5 who had a >10% increase in serum creatinine kinase-MB fraction postoperatively and 4 who refused follow-up studies. The study cohort thus constituted 21 patients, all men, aged 42 to 76 years (mean age ± SD 60.6 ± 9.2 years).

All patients had a previous history of at least one Q wave myocardial infarction. On the basis of electrocardiographic (ECG) Q waves and the site of dyssynergy on echocardiography, infarction was classified as anterior in 11 patients, inferior in 1 patient and anterior plus inferior in 9 patients. Seventeen patients had received thrombolytic treatment during at least one previous infarction. In the total group the most recent infarct had occurred a mean of 129 ± 105 days (range 32 to 365) before the study. In the nine patients with more than one infarct, the oldest infarct had occurred 597 ± 400 days (range 150 to 1,100) before the study. Nineteen patients (90.5%) had angina pectoris not completely responsive to medical treatment; 10 patients were in NYHA class II and 11 in class III or IV; 4 patients (19.0%) had diabetes mellitus, and 11 (52.4%) had systemic hypertension. Mean LV ejection fraction was 35 ± 8%.

Coronary angiography was performed 8 ± 4 days before dobutamine stress echocardiography. All patients had at least one significant coronary artery stenosis (>70% of maximal lumen diameter) of a major epicardial artery. Ten patients had three-vessel disease, 7 two-vessel and 4 one-vessel. All 21 patients had disease of the left anterior descending coronary artery, and in 15 the right and in 11 the left circumflex coronary artery was also involved. No major clinical events occurred between the time of coronary angiography and the preoperative studies. Seventeen patients underwent bypass surgery and four (i.e., those with single-vessel disease) underwent coronary angioplasty. The revascularization procedure was complete in each patient.

Study protocol. Dobutamine stress echocardiography and rest/redistribution thalluium-201 (Tl-201) single-photon emission computed tomography (SPECT) were performed in all patients 6 to 8 days before revascularization and were repeated ≥3 months (mean 5.2 ± 0.9) after. Studies were performed after an angina-free period of ≥3 days, after overnight fasting and in a random sequence 24 h apart. Beta-adrenergic blocking agents were withdrawn 48 h before the test without adverse effects. Other medications (i.e., different combinations of nitrates, calcium channel antagonists, digitalis, diuretic drugs and converting enzyme inhibitors) were not administered on the morning of the test. All patients gave written informed consent to the protocol, which was approved by the Ethics Committee of our institution.

Dobutamine stress echocardiography. Dobutamine stress echocardiography was performed with a Toshiba 270 system provided with a 2.75-MHz transducer and recorded on a 0.75-in. (1.9-cm) videorecorder for subsequent analysis. Images were acquired from parasternal long- and short-axis views and from apical four- and two-chamber views, with the patient in the left lateral recumbent position.

After baseline echocardiography, dobutamine infusion performed with use of a mechanical pump, was begun at a starting dose of 5 μg/kg body weight per min for 5 min, followed by a dose of 10 μg/kg per min for 3 min (low dose dobutamine). The infusion rate was then increased by 10 μg/kg per min at 3-min intervals up to a maximal dose of 40 μg/kg per min (high dose dobutamine). Atropine (1 to 2 mg) was added at the end of the last step if the target heart rate had not been achieved. ECG lead II was monitored continuously throughout the test. An ECG, cuff blood pressure and heart rate were recorded at baseline, at the end of each dose and during the recovery period. End points for interrupting the infusion were 1) achievement of 85% of the maximal predicted heart rate; 2) chest pain or dyspnea not tolerated; 3) development of wall motion abnormalities in a remote region or worsening contraction in previously asynergic segments; 4) ST segment depression ≥2 mm; 5) ventricular arrhythmias (Lown grade 3 to 5); 6) severe hypertension (>230/130 mm Hg) or hypotension (decrease in systolic blood pressure ≥40 mm Hg). In the event of demonstrable ischemia, intravenous metoprolol or nitrates, or both, were administered.

Quantitative echocardiographic analysis. Echocardiograms performed before and after revascularization were interpreted side by side by two observers who did not know which images preoperative were, ^{201}Tl SPECT findings or clinical outcome and by a third observer in case of disagreement. LV ejection fraction was calculated at baseline by use of the biplane Simpson method. Regional wall motion was analyzed qualitatively by utilizing a 16-LV segment model with the following scoring system: 1 = normal contraction (≥5-mm endocardial excursion, ≥25% systolic thickening), 2 = hypokinesia (<5-mm endocardial excursion, <25% systolic thickening); 3 = akinesia (absence of endocardial excursion and wall thickening), and 4 = dyskinesia (paradoxic outward motion in systole) (10). Intraobserver and interobserver variabilities in wall motion score were calculated in a sample of 86 dyssynergic segments (41 akinetic or dyskinetic and 45 hypokinetic segments) in the 1st 10 consecutive patients with use of

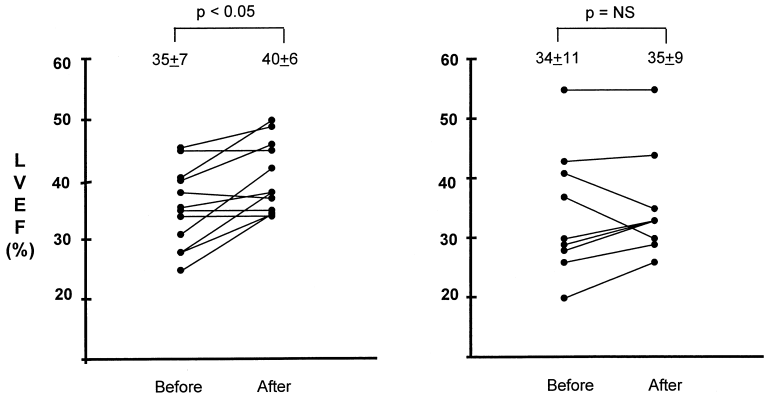


Figure 1. Changes in left ventricular ejection fraction (LVEF) before and after revascularization in 12 patients classified as positive (**left**) and 9 as negative (**right**) for contractile reserve in infarct zones.

the percent coefficient of variation. These scores were 2 + 2% and 4 + 2%, respectively, for wall motion at rest and 4 ± 3% and 5 ± 3%, respectively, for wall motion response during low dose dobutamine infusion. Infarct zones for anterior and inferior (posterolateral) infarcts were constructed according to the theoretic maximal area at risk (11). The apical inferior and apical lateral segments were considered to be overlapping. Thus, each infarct zone comprised nine segments. In the presence of two infarct zones, the apical inferior and apical lateral segments were attributed to each zone. An infarct zone wall motion score index (WMSI) was calculated at baseline and during low and high dose dobutamine as the sum of scores in segments in the infarct zone divided by the number of segments scored (11).

Changes in the score of individual segments from baseline to low and high dose dobutamine infusion were analyzed. Only the central portion of the segment was considered. An infarct zone was judged to have both *contractile reserve* (comparing WMSI during low dose dobutamine with baseline WMSI) and *contractile recovery* (comparing baseline WMSI after revascularization with that before revascularization) when 1) wall motion improved in at least two pertinent contiguous asynergic segments (of which at least one was akinetic or dyskinetic), and 2) infarct zone WMSI decreased by ≥0.22 (11). Homozonal and remote *contractile worsening* were defined by an increase in WMSI ≥0.22 in the infarct or remote regions, respectively, in comparison with WMSI during low dose dobutamine. Patients with at least one infarct zone showing contractile reserve before revascularization were classified as positive for contractile reserve; those with no infarct zone showing contractile reserve before revascularization were classified as negative.

^{201}Tl imaging. SPECT imaging was performed 15 min and 4 h after thallium injection (74 MBq intravenously) (at rest with the patient supine) with use of a wide field of view general purpose gamma camera (ELSCINT, Apex 409). The camera was rotated over a 180° arc with 6° increments, each exposure lasting 45 s. The acquisition matrix was 64 × 64; the zoom factor was 1.2. Reconstruction of transaxial slices was performed by filtered back-projection with use of a Butterworth filter (cutoff frequency 0.35 cycles/pixel; order 5). A series of short-axis, horizontal long-axis and vertical long-axis slices was generated. For quantitative analysis, the circumferential profile curves were generated by using short-axis slices from apical to basal slices to create a bull's-eye polar map of percent myocardial thallium uptake in which a maximal count was normalized as 100%. To match regional thallium uptake with regional wall motion, the LV myocardium was divided into 16 segments, and infarct-related zones were constructed for anterior and inferior (posterolateral) infarcts, as with the echocardiographic method. An infarct zone mean percent thallium uptake was then calculated as the sum of percent uptakes in each segment divided by the number of segments considered (12).

Data analysis. Data are presented as mean value ± SD. Comparison of quantitative variables was accomplished by using analysis of variance for repeated measures. To assess statistical significance, post-hoc comparisons were evaluated by the Newman-Keuls test. The chi-square test was also used, when appropriate. The extent of association between multiple infarct zones in the same patient was analyzed by the contingency coefficient C. Significance of changes was analyzed by the McNemar test for paired data. A p value < 0.05 was considered significant.

Results

Before revascularization. Twelve of the 21 patients were classified as positive for contractile reserve in infarct zones (6 in NYHA class II, 4 in class III and 2 in class IV); 9 were classified as negative (4 in class II, 3 in class III, and 2 in class IV). There was no difference in LV ejection fraction between the two groups ($p = 0.79$) (Fig. 1). Bypass surgery was performed in 17 patients (26 revascularized infarct zones) and coronary angioplasty in 4. Of the 17 patients submitted to bypass surgery, 8 were classified as positive and 9 as negative for contractile reserve. All four patients submitted to coronary angioplasty were classified as positive for contractile reserve.

Dobutamine stress echocardiography. *General data*. No complications occurred during dobutamine stress echocardiography. Dobutamine infusion was interrupted before the maximal dose was reached in seven patients (in three because of homozonal contractile worsening and in four because of new

Table 1. Heart Rate and Blood Pressure Changes During Dobutamine Stress Echocardiography Before and After Revascularization*

	Patients Classified as Positive for Contractile Reserve (n = 12)		Patients Classified as Negative for Contractile Reserve (n = 9)	
	Before	After	Before	After
Heart rate (beats/min)				
Baseline	68 ± 19	71 ± 13	67 ± 18	70 ± 14
Low dose dobutamine	79 ± 14	80 ± 13	80 ± 14	79 ± 15
High dose dobutamine	124 ± 19	130 ± 22	122 ± 18	132 ± 24
Systolic blood pressure (mm Hg)				
Baseline	126 ± 19	136 ± 23	128 ± 18	134 ± 26
Low dose dobutamine	129 ± 22	138 ± 25	130 ± 23	136 ± 23
High dose dobutamine	136 ± 23	152 ± 30	138 ± 22	148 ± 31

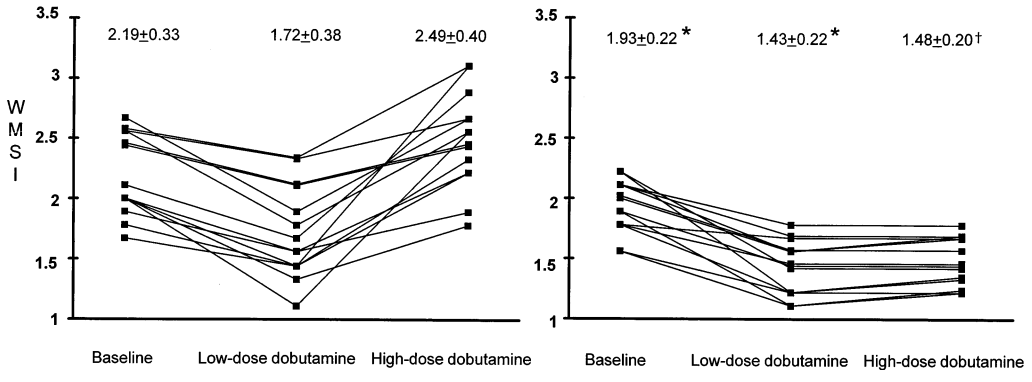
*There were no significant differences in either patient group between values before and after revascularization. Values presented are mean value ± SD.

remote dyssynergy). The peak dose of dobutamine achieved was 35 ± 9 and 31 ± 11 μg/kg per min in patients with and without contractile reserve, respectively ($p = 0.37$). No significant differences in heart rate and systolic blood pressure achieved were observed during administration of the test in either group (Table 1).

Infarct zones. In the nine patients with more than one myocardial infarction, different infarct zones showed independent behavior, as indicated by the contingency coefficient C (0.056) and McNemar test (chi-square = 4.08, $p < 0.05$). Consequently, we utilized the infarct zone as the unit of analysis. Fourteen infarct zones showed contractile reserve and 16 did not. A significant difference in infarct age was present between infarct zones with and without contractile reserve (79 ± 53 days [range 32 to 180] vs. 410 ± 431 days [range 90 to 1,100]; $p < 0.01$). WMSI changes during dobutamine testing in infarct zones with and without contractile reserve are shown in Figures 2 and 3, respectively. WMSI at baseline was greater in infarct zones with contractile reserve than in those without ($p < 0.01$). Compared with baseline value, during low dose dobutamine WMSI decreased in infarct zones with contractile reserve (−21.7 ± 10.3%, $p < 0.001$) and was unchanged (−2.8 ± 5.5%, $p = 0.16$) in those without. Compared with the value during low dose dobutamine infusion, WMSI during high doses increased in infarct zones with (+30.2 ± 14.0%, $p < 0.001$) and without (+17.5 ± 11.0%, $p < 0.001$) contractile reserve. Contractile worsening occurred during high dose dobutamine in 28 infarct zones. The individual responses to dobutamine of LV segments in infarct zones with and without contractile reserve are shown in Figures 4 and 5, respectively. The mean Tl-201 uptake was similar in infarct zones with (56 + 8%) and without (53 + 8%) contractile reserve ($p = 0.09$).

After revascularization. At follow-up (5.2 ± 0.9 months after revascularization), an improvement in NYHA functional class had occurred in 7 (58.3%) of the 12 patients previously classified as positive for contractile reserve (3 in NYHA class I, 7 in class II and 2 in class III) and in 2 (22.2%) of the 9 previously classified as negative (2 in class I, 2 in class II, 3 in class III and 2 in class IV). Compared with prerevascularization values, LV ejection fraction increased by 5 ± 4% in patients previously classified as positive for contractile reserve ($p < 0.01$) and remained unchanged (+1 ± 4%, $p = 0.67$) in those previously classified as negative (Fig. 1). Thus, after revascularization, LV ejection fraction was greater in the group previously classified as positive ($p < 0.001$). LV ejection

Figure 2. Infarct zones (n = 14) with contractile reserve: changes in WMSI on dobutamine stress echocardiography before (**left**) and after revascularization (**right**). Individual and mean values (±SD) are shown. *$p < 0.01$, † $p < 0.001$ versus value before revascularization.



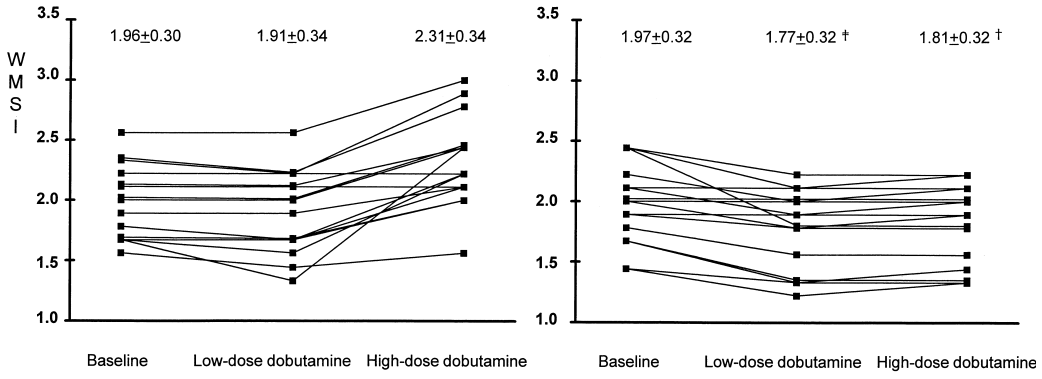


Figure 3. Infarct zones (n = 16) without contractile reserve: changes in WMSI on dobutamine stress echocardiography before (**left**) and after revascularization (**right**). Individual and mean values (± SD) are shown. †$p < 0.001$, ‡$p < 0.05$ versus value before revascularization.

fraction increased ≥5% in seven patients (58.3%) previously classified as positive for contractile reserve (four also showing improved NYHA class) and in two (22.2%) with previously negative findings (none showing improved NYHA class; p = 0.2 between the two groups).

Dobutamine stress echocardiography. *General data*. No complications occurred during follow-up dobutamine testing. No studies were interrupted because of either severe wall motion worsening in infarct zones or new remote dyssynergy. All patients received the maximal dose of dobutamine. Changes in heart rate and systolic blood pressure that occurred during dobutamine testing after revascularization are shown in Table 1.

Infarct zones. Recovery in basal contraction was observed in 8 (57.1%) of 14 infarct zones previously showing contractile reserve and in 1 (6.3%) of 16 without ($p < 0.01$). Baseline WMSI decreased in infarct zones previously showing contractile reserve (−11.0 ± 10.8%, $p < 0.01$), but it was unchanged in those without (−0.8 ± 9.9%, p = 0.79) (Fig. 2 and 3). In zones previously showing reserve, the decrease in baseline

Figure 4. Wall motion at baseline and during low and high dose dobutamine infusion in 126 LV segments of infarct zones showing contractile reserve before (**top**) and after revascularization (**bottom**). The **numbers in the boxes** show the number of normal, hypokinetic and akinetic (A)/dyskinetic segments. The **numbers beside the arrows** indicate the number of segments showing wall motion changes during dobutamine testing.

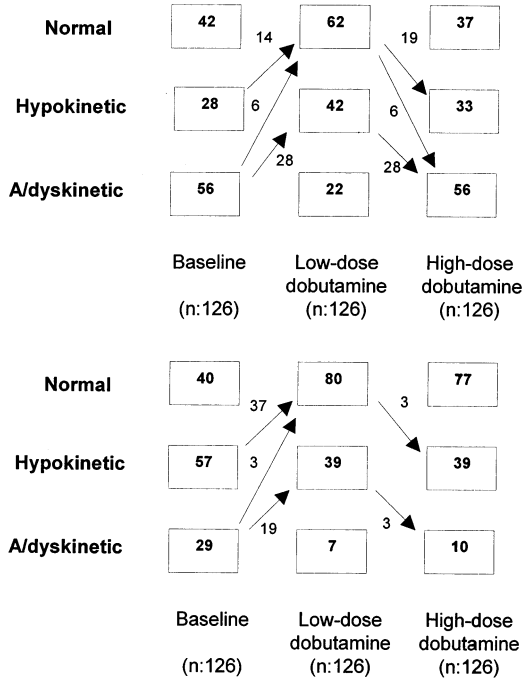
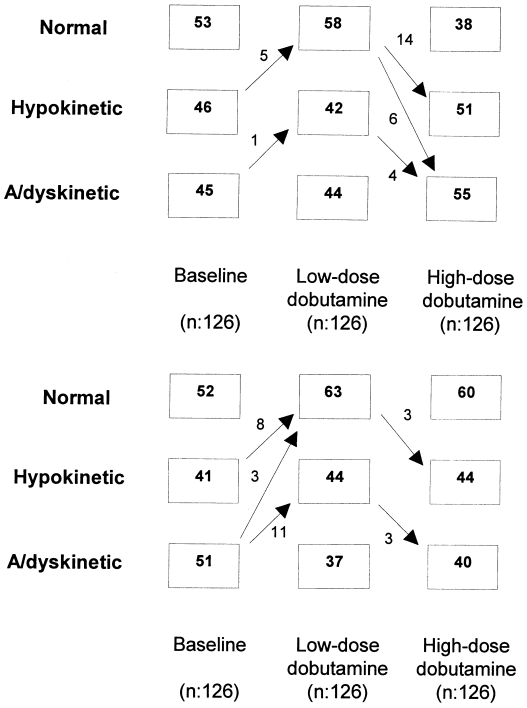


Figure 5. Wall motion at baseline and during low and high dose dobutamine infusion in 126 segments of infarct zones showing no contractile reserve before (**top**) and after revascularization (**bottom**). Format and abbreviation as in Figure 4.



WMSI achieved after revascularization (contractile recovery) was lower than the contractile reserve detected before revascularization ($p < 0.05$).

Moreover, after revascularization, contractile reserve was detected in 13 infarct zones in the group previously showing reserve (93%; 8 with and 5 without recovery in basal contraction) and in 6 in the group not showing reserve (38%; all without recovery in basal contraction). Compared with baseline values, WMSI during low dose dobutamine infusion decreased in infarct zones with and without contractile reserve before revascularization ($-25.6 \pm 10.2\%$ and $-10.2 \pm 8.1\%$, respectively; both $p < 0.001$). WMSI during low dose dobutamine was significantly lower than that before revascularization in both the group previously showing reserve ($1.43 + 0.22$ vs. $1.72 + 0.38$, $p < 0.01$) and the group not showing reserve ($1.77 + 0.32$ vs. $1.91 + 0.34$, $p < 0.05$). Compared with values during low dose dobutamine, WMSI during high dose dobutamine did not change significantly in infarct zones with or without contractile reserve before revascularization ($+3.4 \pm 4.1\%$, $p = 0.55$, and $+2.3 \pm 3.2\%$, $p = 0.36$, respectively). No infarct zone showed contractile worsening during high dose dobutamine.

In comparison with prerevascularization findings, the number of akinetic or dyskinetic segments decreased in infarct zones with contractile reserve ($p < 0.001$) and did not change significantly in those without (Fig. 4 and 5). During low dose dobutamine infusion, wall motion improved, respectively, in 59 (46.8%) and 22 (15.3%) segments of infarct zones with and without contractile reserve before revascularization ($p < 0.001$). Thus, after revascularization some segments not previously showing contractile reserve showed a positive response to dobutamine.

Compared with prerevascularization assessment, the mean Tl-201 uptake after revascularization increased in infarct zones with contractile reserve (from $56 + 8\%$ to $61 + 7\%$, $p < 0.05$), but it did not change significantly in zones without such reserve (from $53 + 8\%$ to $55 + 7\%$, $p = 0.22$), and thus was significantly greater in the former zones ($p < 0.01$). Specifically, the mean Tl-201 uptake increased in each infarct zone showing contractile reserve after revascularization.

Discussion

This study was performed in postinfarction male patients with various degrees of LV dysfunction who underwent revascularization of one or more infarct zones either with or without contractile reserve. Revascularization had the following effects on the wall motion of infarct zones: 1) Improvement in basal contraction was less than that expected on the basis of the previously documented contractile reserve; 2) in most infarct zones showing contractile reserve preoperatively, contractile reserve was maintained or increased and there was also a variable improvement in basal contraction; and 3) some amount of contractile reserve was also present in some infarct zones that showed no contractile reserve preoperatively. After revascularization, many patients had improved NYHA class. The gain in LV ejection fraction was modest in patients previously classified as positive for contractile reserve and was absent in those previously classified as negative. Such effects of revascularization on the contractile reserve of infarct zones may have contributed to the functional improvement of patients, the results being additive to the gain achieved in basal contraction.

Contractile recovery of infarct zones after revascularization. After revascularization, improvement in basal contraction in dysfunctional but viable myocardium may be less than that predicted by dobutamine stress echocardiography (3). The latter technique may result poorly in predicting the recovery of LV segments with severe dyssynergy than in predicting the recovery of less affected segments (4,5). In our study only 57% of infarct zones with contractile reserve showed an improvement in basal contraction after revascularization. In most zones, the amount of contractile recovery was less than that anticipated by contractile reserve (Fig. 2). These results can be explained by some inherent limitations of the dobutamine echocardiography test, such as tethering, which may lead to possible overestimation of myocardial viability. It is also possible that extensive fibrosis, commonly found in Q wave infarction, or even some combination of viable and fibrotic tissue in infarct zones, hinders the recovery in basal contraction but allows some contractile recruitment under catecholamine stimulation. It can be hypothesized that virtually all patients with chronic ischemic dysfunction have a different combination of areas of myocardial infarction, myocardial hibernation and normally functioning nonischemic myocardium. As a consequence, a variety of contractile responses (as well as of tracer uptake) may appear both during inotropic stimulation and at rest follow-up, as recently elucidated by Armstrong (13). The results of this study, demonstrating the preservation of systolic reserve in the absence of recovery of rest function after revascularization, support this hypothesis. Other factors such as insufficient distal runoff, abnormal microvasculature within the revascularized bed (14), no-reflow phenomenon (15), chronic hibernation (16) and myocardial "embalmment" (17) may also account for the unsatisfactory recovery of basal contraction in infarct zones and cannot be excluded. Varying delay in contractile recovery after revascularization should be also considered (16).

Contractile reserve of infarct zones after revascularization. Asynergic but viable myocardium usually thickens under catecholamine stimulation (1–9,11). However, this effect may be limited or even abolished in the presence of a flow-limiting coronary artery stenosis, because of the early development of ischemia (9). Thus, the amount of contractile reserve detectable by inotropic stimulation of asynergic myocardium may depend not only on a critical mass of functional myocytes (6), but also on local arterial supply (9). Our findings support this possibility. Indeed, after revascularization, a substantial amount of contractile reserve was elicited in infarct zones previously showing contractile reserve (decrease in WMSI during low dose dobutamine infusion vs. baseline $25.6 \pm 10.2\%$), in addition to the gain achieved in basal contraction

(decrease in baseline WMSI vs. value before revascularization 11.0 ± 10.8%). Moreover, in these infarct zones the WMSI during low dose dobutamine was significantly lower after than before revascularization.

In this study the infarct zones without contractile reserve (presumably being composed mostly of fibrotic tissue) were submitted to revascularization whenever the coronary artery anatomy allowed it. As predicted, they showed no improvement in basal contraction after revascularization, but a limited amount of contractile reserve could be elicited (decrease in WMSI during low dose dobutamine infusion vs. baseline 10.2% ± 8.1%). Thus, after effective restoration of flow, the myocardium previously judged to be scarred became partly viable.

The persistence or the development of contractile reserve in infarct zones after revascularization is a novel but not surprising finding. Tl-201 uptake increased after revascularization in most infarct zones, in particular in those with contractile reserve. This increase was small, but it was calculated as a mean value for an entire infarct zone, including segments that were well perfused before revascularization. Furthermore, contractile worsening was abolished during high dose dobutamine infusion following revascularization. It is conceivable that improved perfusion and the relief of ischemia may have revealed some viable tissue in infarct zones previously unable to respond to inotropic stimulation (18). Reperfusion of asynergic but viable myocardium may then result in increased contractile reserve over and above the varying degrees of recovery in contraction at rest.

Clinical implications. An increased contractile reserve in infarct zones after revascularization may contribute to the functional improvement in patients after revascularization as a result of the effects of catecholamine stimulation during exercise. Our findings support this hypothesis. Most of our patients had moderate to severe systolic LV dysfunction, and >50% were in NYHA class III or IV. After revascularization, LV ejection fraction increased modestly in patients classified as positive for contractile reserve and remained unchanged in those classified as negative. However, NYHA functional class improved in 58% and 22%, respectively, suggesting that increased contractile reserve of infarct zones may contribute to functional improvement in many patients. However, we assessed the functional status of patients before and after revascularization only subjectively.

Study limitations. The number of patients in our study was relatively small, and exercise testing was not performed to assess their functional state. Furthermore, our patients were a select group because of a high prevalence of ischemia. Inclusion of patients with depressed ventricular function and without ischemia would have corroborated our results. Follow-up coronary angiography was not performed. However, all infarct zones underwent adequate revascularization, as shown by improved perfusion at repeat myocardial scintigraphy and by the absence of ischemia during high dose dobutamine. The true extension of infarct zones could not be delineated exactly in each patient because of the variability of the arterial blood supply. Indeed, the artificial delimitation of infarct zones may have also influenced our data on mean Tl-201 uptake, which was not significantly different before revascularization in zones with and without contractile reserve, in contrast to findings in the majority of previous studies. However, for clinical purposes it is more convenient to analyze the contractile reserve of broad infarct zones (11), as we did, than that of individual segments, as frequently reported (2,4,5). In patients with more than one infarct zone, a few myocardial segments were counted twice. However, this artifice did not substantially influence our data. Differences in study group (i.e., differences relating to LV function, percent of postinfarction patients, prevalence of homozonal and remote ischemia, extent of infarcted myocardium and amount of fibrotic and viable tissue in infarct zones) and in the definition of tissue viability may influence the results of dobutamine stress echocardiography (13,19). Although our echocardiographic studies were examined without knowledge of the intervention, postsurgical abnormal septal wall motion could have biased the analysis. However, follow-up echocardiography was performed a mean of 5.2 months postoperatively, thereby limiting the confounding effect of paradoxic septal dyssynergy.

Conclusions. Assessment of the benefits obtained by revascularization procedures is an important clinical issue in patients with chronic ischemic heart disease and global LV dysfunction (16,19–22). The gain in basal contraction alone may underestimate the benefits obtained by revascularization of infarct zones, as an increase in contractile reserve may also be achieved. Further studies are warranted to determine whether this effect contributes to a functional improvement in postinfarction patients after revascularization.

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