Left Ventricular Remodeling after Late Revascularization

Correlates with Baseline Viability

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The ideal management of stable patients who present late after acute ST-elevation myocardial infarction (STEMI) is still a matter of conjecture. We hypothesized that the extent of improvement in left ventricular function after successful revascularization in this subset was related to the magnitude of viability in the infarct-related artery territory. However, few studies correlate the improvement of left ventricular function with the magnitude of residual viability in patients who undergo percutaneous coronary intervention in this setting.

In 68 patients who presented later than 24 hours after a confirmed first STEMI, we performed resting, nitroglycerin-enhanced, technetium-99m sestamibi single-photon emission computed tomography–myocardial perfusion imaging (SPECT–MPI) before percutaneous coronary intervention, and again 6 months afterwards. Patients whose baseline viable myocardium in the infarct-related artery territory was more than 50%, 20% to 50%, and less than 20% were divided into Groups 1, 2, and 3 (mildly, moderately, and severely reduced viability, respectively). At follow-up, there was significant improvement in end-diastolic volume, end-systolic volume, and left ventricular ejection fraction in Groups 1 and 2, but not in Group 3.

We conclude that even late revascularization of the infarct-related artery yields significant improvement in left ventricular remodeling. In patients with more than 20% viable myocardium in the infarct-related artery territory, the extent of improvement in left ventricular function depends upon the amount of viable myocardium present. The SPECT–MPI can be used as a guide for choosing patients for revascularization. (Tex Heart Inst J 2014;41(4):381-8)

Late presentation of acute ST-elevation myocardial infarction (STEMI) is not uncommon on the Indian subcontinent, or indeed in much of the rest of the world. Late presenters contribute an important portion of acute myocardial infarction (MI) patients in the day-to-day practice of a great many physicians.¹⁻³ Yet studies of late revascularization and its effects on myocardial salvage and clinical outcomes have yielded conflicting results.^{4,5}

The largest study of the efficacy of late revascularization, the Occluded Artery Trial (OAT)⁶—together with a few small studies^{7,8}—concluded that late intervention in acute-STEMI patients affords no clinical benefit. This, however, defies the basic concept of the “open-artery” hypothesis⁹ and all the reported secondary benefits^{10,11} of revascularization—even when performed late. Indeed, several smaller studies¹²⁻¹⁴ have shown the recovery of left ventricular (LV) function after late revascularization.

At the outset of this prospective single-center study, our hypothesis was that recovery of LV function after revascularization would relate to the magnitude of myocardial viability within the territory of the infarct-related artery (IRA). The Occluded Artery Trial–Viability Ancillary Study (OAT–NUC)¹⁵ attempted to study this aspect by classifying viability into 2 broad categories on the basis of an arbitrary cutoff point of 40%: an average infarct zone uptake of less than 40% indicated severely reduced viability. Viability, however, lies on a continuum^{16,17} that includes grades not categorized on an all-or-none basis. In the setting of late presentation of STEMI, we critically examined the magnitude of baseline infarct-zone viability and attempted to relate that to the extent of LV remodeling and to the recovery of LV function at 6 months after successful revascularization.

Patients and Methods

Table I shows the baseline clinical characteristics of the study patients. Patients enrolled in the study were from 30 to 76 years of age (mean age, 57.04 ± 10.38 yr). These were patients with confirmed first-index STEMI (that is, ischemic symptoms, elevated cardiac markers, and typical MI electrocardiographic changes) more than 24 hours after the onset of pain (3rd calendar day and up to 28 d), in the absence of signs of continued or recurrent ischemia. None of the patients had any prior history of infarction. All patients entering the paired analysis showed either occlusion of the IRA or significant stenosis in the patent IRA, regardless of the baseline preprocedural Thrombolysis in Myocardial Infarction (TIMI) flow grade on coronary angiography.

From June 2011 through June 2012, 92 patients were initially enrolled in the study. All 92 patients underwent single-photon emission computed tomography–myocardial perfusion imaging (SPECT–MPI) before angiography. All patients were administered 0.4 mg of sublingual nitroglycerin followed 5 to 10 minutes later by 15 to 20 mCi of technetium-99m sestamibi; one hour later, they underwent imaging at rest, by use of standard algorithms optimized for each Infinia™ Hawkeye™ 4

camera and computer system (GE Healthcare; Waukesha, Wis).

Eight patients were excluded after angiography showed that they had recanalized IRAs due to thrombolysis, either spontaneous or pharmacologic. Follow-up scans could not be performed in 12 patients, 2 for reason of death during the follow-up period and 10 for various other reasons. In 4 patients, either baseline or follow-up data were unusable for analysis. The exclusion criteria were substantial persistent or recurrent ischemia, cardiogenic shock, New York Heart Association functional class III or IV heart failure, serum creatinine level >2.5 mg/dL, and angiographically significant left main or 3-vessel coronary artery disease. A detailed history was taken of each patient's risk factor profile, index event, time to presentation, TIMI score, Killip score, and record of receiving thrombolytic therapy. After the elimination of these 24 patients, paired analysis was performed for the remaining 68 patients (Fig. 1).

The characteristics of the precipitating MI are described in Table II. Myocardial infarction was localized to the anterior wall in 70.6% of the study patients. The time to presentation varied from 3 to 28 days, with a mean presentation delay of 11.59 ± 8.5 days and a median delay of 9 days. Fifty percent of the patients

TABLE I. Baseline Clinical Characteristics

Variable	Overall (N=68)	Group 1 (n=37)	Group 2 (n=19)	Group 3 (n=12)	P Value
Demographics					
Age (yr)	57.04 ± 10.38	58.76 ± 9.92	54.68 ± 11	55.5 ± 10.7	0.329
Male sex	54 (79.4)	28 (75.7)	16 (84.2)	10 (83.3)	0.706
Clinical history					
Diabetes mellitus	17 (25)	11 (29.7)	4 (21.1)	2 (16.7)	0.593
Hypertension	22 (32.4)	12 (32.4)	4 (21.1)	5 (41.6)	0.245
Family history of CAD	6 (8.8)	4 (10.8)	2 (10.5)	0	0.494
Tobacco use	21 (30.9)	7 (18.9)	7 (36.8)	7 (58.3)	0.03
Height (cm)	163.68 ± 6.4	163.46 ± 6.64	164.84 ± 6.33	162.67 ± 6.01	0.615
Weight (kg)	68.66 ± 10.55	67.35 ± 9.72	68.47 ± 11.61	73 ± 11.03	0.275
Body mass index (kg/m ²)	25.74 ± 4.46	25.35 ± 4.39	25.25 ± 4.27	27.73 ± 4.8	0.237
Laboratory findings (mg/dL)					
Fasting blood sugar	117.41 ± 63.65	119.84 ± 70	115.95 ± 54.73	112.25 ± 60.78	0.933
Triglycerides	129.15 ± 58.06	123.03 ± 54.68	135.53 ± 61.6	137.92 ± 65.23	0.64
Total cholesterol	131.74 ± 42.94	121.35 ± 42.25	143.11 ± 41.52	145.75 ± 42.04	0.09
LDL cholesterol	75.54 ± 31.64	67.16 ± 29.87	83.89 ± 33.07	88.17 ± 29.29	0.052
HDL cholesterol	30.68 ± 8.41	29.73 ± 8.09	32.79 ± 9.95	30.25 ± 6.64	0.434
VLDL cholesterol	25.22 ± 11.31	23.95 ± 10.69	26.16 ± 11.68	27.67 ± 12.99	0.507

CAD = coronary artery disease; HDL = high-density-lipoprotein; LDL = low-density-lipoprotein; VLDL = very-low-density-lipoprotein
Data are presented as mean $\pm$ SD or as number and percentage. $P < 0.05$ was considered statistically significant.

had received thrombolysis. The risk-factor profile was relatively high, with a mean TIMI risk score of 6.07 ± 2.72 . However, the patients were relatively stable, with a mean Killip score of 1.62 ± 0.65 .

Fig. 1 Study enrollment and patient flow

IRA = infarct-related artery; MI = myocardial infarction; SPECT-MPI = single-photon emission computed tomography-myocardial perfusion imaging

The qualifying angiogram was reviewed critically for preprocedural and postprocedural angiographic status and for TIMI flow. That baseline angiographic picture is summarized in Table III. The culprit IRA was most frequently the left anterior descending coronary artery.

In regard to SPECT-MPI, the polar plot method of quantification was used to estimate the area of nonviable myocardium, because it is precisely quantitative and easily reproducible and provides better insight into other vascular territories. In preparing the polar maps, we used a 50% peak-uptake value as the cutoff standard for viability.¹⁸ The estimated area of nonviable myocardium was recorded as a percentage of the IRA zone, as a percentage of the total LV, and as the estimated mass of nonviable myocardium. The Emory Cardiac Toolbox 3.1™ (Koninklijke Philips N.V.; Best, The Netherlands) was used to process the images, and validated commercially available software was used to perform quantitative analysis. The percentage of viable myocardium in the IRA territory was noted. The IRA territory was defined in advance on the basis of the standard anatomic territory supplied by each of the 3 coronary arteries, and we noted the percentage of viable myocardium remaining in those IRA territories. On the basis of this percentage of remaining viable myocardium, we divided patients into 3 groups: Group 1 (mildly reduced viability), in whom the amount of viable myocardium in the IRA territory was more than 50%; Group 2 (moderately reduced viability), in whom the amount of viable myocardium in the IRA territory ranged from 20% through 50%; and Group 3 (severely reduced viability), in whom the amount of viable myocardium in the IRA territory was less than 20%. The baseline characteristics of patients in these 3 groups were similar with respect to age, sex distribution, and prevalence of traditional cardiovascular risk factors—except for the prevalence of tobacco use, which was most common in

TABLE II. Baseline Characteristics of Index Myocardial Infarction

Variable	Overall (N=68)	Group 1 (n=37)	Group 2 (n=19)	Group 3 (n=12)	P Value
Infarction location					0.299
Anterior wall	47 (69.2)	29 (78.4)	11 (57.9)	7 (58.3)	—
Lateral wall	2 (2.9)	2 (5.4)	0	0	—
Inferior wall	19 (27.9)	6 (16.2)	8 (42.1)	5 (41.7)	—
Presentation details					
Time to presentation (d)	11.59 $\pm$ 8.5	12.43 $\pm$ 9.04	11.94 $\pm$ 8.59	8.75 $\pm$ 6.37	0.432
TIMI score	6.07 $\pm$ 2.72	6.27 $\pm$ 3.04	5.58 $\pm$ 2.43	6.25 $\pm$ 2.14	0.654
Killip class	1.62 $\pm$ 0.65	1.54 $\pm$ 0.61	1.53 $\pm$ 0.7	2 $\pm$ 0.6	0.077
Thrombolysis	34 (50)	19 (51.4)	9 (47.4)	6 (50)	0.961

TIMI = Thrombolysis in Myocardial Infarction

Data are presented as mean $\pm$ SD or as number and percentage. $P < 0.05$ was considered statistically significant.

TABLE III. Angiographic Characteristics of Index Myocardial Infarction

Variable	Overall (N=68)	Group 1 (n=37)	Group 2 (n=19)	Group 3 (n=12)	P Value
Culprit coronary artery					0.012
Left anterior descending	48 (70.6)	30 (81.1)	11 (57.9)	7 (58.3)	
Left circumflex	10 (14.7)	5 (13.5)	5 (26.3)	0	
Right	10 (14.7)	2 (5.4)	3 (15.8)	5 (41.7)	
Angiographic characteristics					
Percent stenosis in infarct-related artery					0.796
100	25 (36.8)	14 (37.8)	8 (42.1)	3 (25)	
99	10 (14.7)	5 (13.5)	4 (21.1)	1 (8.3)	
90–95	26 (38.2)	14 (37.8)	6 (31.6)	6 (50)	
<90	7 (10.3)	4 (10.8)	1 (5.3)	2 (16.7)	
Baseline TIMI flow					0.345
TIMI 0/1	41 (60.3)	24 (64.9)	12 (63.2)	5 (41.7)	
TIMI 2/3	27 (39.7)	13 (35.1)	7 (36.8)	7 (58.3)	
Final TIMI Flow					0.005
TIMI 2	7 (13.2)	2 (5.4)	2 (10.5)	5 (41.7)	
TIMI 3	59 (86.8)	35 (94.6)	17 (89.5)	7 (58.3)	

TIMI = Thrombolysis in Myocardial Infarction

Data are presented as number and percentage. $P < 0.05$ was considered statistically significant.

patients with severely reduced viability ($P=0.03$). Body mass index and laboratory values of blood sugar and lipids (samples taken while fasting) were similar in the 3 groups except for low-density-lipoprotein-cholesterol levels, which were highest in the group with severely reduced viability. Nearly 80% of the patients were male, which might be considered a selection bias.

Left ventricular ejection fraction (LVEF), LV end-diastolic volume (LVEDV), and LV end-systolic volume (LVESV) were calculated with the aid of an automated software program.¹⁹ The use of gated SPECT for the determination of LVEF and volumes has been extensively validated.^{20–22} Wall-motion scores (WMS) were quantified via the standard 17-segment model, which assigned scores for wall motion ranging from normal (4) to akinetic (0). All analyses were performed independently for both the baseline scan and the follow-up (done 6 months later).

Statistical Analysis

Patients' clinical and MI characteristics, both at baseline and at hospital discharge, were evaluated as proportions or as mean $\pm$ SD. Their distributions among the groups were compared by means of χ^2 or Fisher exact tests. For comparing continuous variables in the 3 groups, one-way analysis of variance and post hoc analysis (the Bonferroni method) were used to determine significance in different groups. The endpoints of LV remodeling (LVEDV, LVESV, LVEF, and WMS) were evaluated by looking at changes from baseline to 6 months. Two-sided 2-group t tests were performed in comparing those changes from baseline to 6 months. To evaluate the influence of the magnitude of viability on the extent of

LV remodeling, we compared the 6-month LV volumes and LVEFs with our original findings in regard to baseline viability. Bivariate correlation was performed by the Pearson method, with 2-tailed significance. A P value <0.05 was specified as significant. All analyses were performed with SPSS 16.0 software (IBM Corporation; Armonk, NY).

Results

Group 1 patients (mildly reduced viability) numbered 37; Group 2 patients (moderately reduced viability) numbered 19; and Group 3 patients (severely reduced viability) numbered 12. Of all the patients finally entering the paired analysis, nearly 60% had TIMI 0 or 1 flow in the IRA, while the remaining 40% had either TIMI 2 or 3 flow in the IRA and significant stenosis. At the end of 6 months, more than 85% of enrolled patients achieved TIMI 3 flow. The distribution of pre-procedural baseline TIMI flow was not significantly different among the 3 groups that we studied. In contrast, the percentage of patients who did not achieve final TIMI 3 flow was significantly higher in Group 3.

Relation of Baseline Viability to Left Ventricular Remodeling at 6 Months

At baseline, the percentage of viable myocardium in the IRA territory was $48.44\% \pm 24.84\%$ in the overall population, and $68\% \pm 10.66\%$, $34.63\% \pm 7.95\%$, and $10\% \pm 7.33\%$ in Groups 1, 2, and 3, respectively ($P < 0.001$). The baseline LVEFs were not significantly different among the 3 groups. Table IV shows the LV remodeling indices, which include the LV volume indices,

TABLE IV. Left Ventricular Volume Indices, Ejection Fractions, and Wall-Motion Scores in the Overall Cohort and 3 Groups

Variable	Overall (N=68)	Group 1 (n=37)	Group 2 (n=19)	Group 3 (n=12)	P Value (intergroup)
LVEDV (mL)					
Baseline	124 ± 29.8	123 ± 27.7	117.7 ± 27	138.5 ± 38	0.158
6 mo	118.3 ± 30.7	118.3 ± 28.7	110.6 ± 27.6	132.5 ± 38.7	0.155
Change	-5.6 ± 10.1	-4.7 ± 8.9	-7.1 ± 11.1	-6.0 ± 12.8	0.712
P value (of change)	<0.001	0.003	0.012	0.133	—
LVESV (mL)					
Baseline	67.7 ± 26.2	68.4 ± 20.5	59.8 ± 24	79.4 ± 40.2	0.128
6 mo	61.5 ± 24.5	62.8 ± 20.5	52.3 ± 22.5	73 ± 34.5	0.066
Change	-6.2 ± 7.1	-5.5 ± 4.3	-7.5 ± 8.3	-6.4 ± 11.1	0.625
P value (of change)	<0.001	<0.001	0.001	0.074	—
LVEF					
Baseline	0.47 ± 0.12	0.45 ± 0.09	0.50 ± 0.14	0.46 ± 0.16	0.293
6 mo	0.49 ± 0.13	0.48 ± 0.11	0.54 ± 0.14	0.48 ± 0.16	0.172
Change	0.03 ± 0.02	0.03 ± 0.03	0.04 ± 0.05	0.02 ± 0.05	0.412
P value (of change)	<0.001	<0.001	0.006	0.336	—
Wall-motion score					
Baseline	20.5 ± 1.8	20.1 ± 1.7	20.3 ± 1.6	21.8 ± 2	0.011
6 mo	19 ± 1.5	18.6 ± 1.5	18.6 ± 1.1	20.3 ± 1.5	0.001
Change	-1.5 ± 1	-1.5 ± 0.9	-1.7 ± 1.1	-1.5 ± 1.2	0.75
P value (of change)	<0.001	<0.001	<0.001	0.002	—

LVEDV = left ventricular end-diastolic volume; LVEF = left ventricular ejection fraction; LVESV = left ventricular end-systolic volume

Data are presented as mean ± SD. *P* < 0.05 was considered statistically significant.

the LVEFs, and the WMSs in the overall cohort and 3 study groups at baseline and at the 6-month follow-up. Group 3 had larger mean LVEDVs and LVESVs and lower LVEFs at baseline and at 6 months, in comparison with Groups 1 and 2, but this difference was not statistically significant. However, the WMS was significantly higher in Group 3.

At follow-up, there was significant reduction in the LVEDV and LVESV and improvement in the LVEF in the overall cohort. However, this improvement was in the groups with mildly reduced and moderately reduced viability, not in the group with severely reduced viability. Even the group with severely reduced viability had a trend toward improvement in LVESV, but not in LVEDV or LVEF. However, the WMS improved comprehensively, regardless of the extent in viability. In patients with either TIMI 0/1 or TIMI 2/3 flow in the IRA at baseline, there was reduction in the LVESV and LVEDV at follow-up, together with incremental improvement in LVEF. In other words, the extent of improvement in LV indices was not different between groups with preprocedural TIMI 0/1 and preprocedural TIMI 2/3 flow.

Influence of Clinical Variables on Remodeling

None of the baseline characteristics of age, TIMI score, presence or absence of conventional risk factors, early postinfarction LV volumes, or angiographic values was significantly associated with the degree of quantitative changes in ventricular volume at follow-up.

Discussion

The beneficial effects of early opening of an occluded IRA within the window period after MI are established, but the relevance of late opening of the IRA has been controversial.^{23,24} The OAT trial does not support the late-open-artery hypothesis.⁶ However, across the globe, even late presenters receive percutaneous coronary intervention (PCI) in real-life situations.^{9,25} This issue is of larger importance in India, where timely presentation with acute STEMI remains unlikely.³ Although the thrust of management no doubt will always be toward reduction of “total ischemic time,”²⁶ patients in the larger subgroup that presents late warrant a clearer evaluation of the effects on LV function of successful, albeit late, revascularization. The main findings of our study are twofold. First, revascularization of the IRA improves LV function, even if done late. Second, the extent of that improvement relates to the magnitude of viable myocardium in the IRA territory.

Several small studies have documented improvement in LV function after delayed PCI of the culprit vessel in acute MI—the delay ranging from 18 days²⁷ to 6 weeks.²⁸ A few studies, such as Thrombolysis and Angioplasty in Myocardial Infarction-6 (TAMI-6),²⁹ do not support this finding and suggest that any improvement in LV function seen at one month is eventually lost by 6 months. The credibility of this observation, however, is reduced by the considerable restenosis rate in the TAMI-6 PCI group. Although it has been postu-

lated that the improvement in LV function was due to the residual viable myocardium, very few studies before this one have documented the extent of myocardial viability present in the IRA territory.

It was earlier believed that if necrosis is extensive, preserved blood flow in the infarct zone cannot, independent of myocardial salvage, prevent remodeling.³⁰ Our study reiterates this concept, in that the extent of improvement in LV indices was not different between groups with preprocedural TIMI 0/1 and preprocedural TIMI 2/3 flow. However, the latest American College of Cardiology Foundation/American Heart Association guidelines³¹ recommend delayed PCI in the setting of late presentation of acute STEMI in stable patients whose patent IRAs show significant stenosis (class II indication); in contrast, delayed PCI is not recommended in late-presentation patients whose IRAs are occluded (class III indication).

Although our study concludes that a benefit of improvement in LV remodeling indices cannot be judged by baseline TIMI flow, we think that preprocedural evaluation of the extent of viability can help discern which patients will experience an improvement in LV function.^{32,33} It has been proposed that, when infarct size is comparable, a transmural infarct leads to infarct expansion,³⁴⁻³⁶ and the extent of improvement varies inversely with the thickness of the surviving myocardium.^{35,37} Various imaging techniques have been used in the attempt to quantify the amount of viable myocardium: dobutamine stress echocardiography,³⁸ changes in the redistribution of thallium at rest,³⁹ and dobutamine stress cardiac magnetic resonance imaging.⁴⁰⁻⁴² Using technetium-99m sestamibi SPECT-MPI, we ourselves quantify viable myocardium on the basis of its proportion in the IRA territory.

Table V outlines the important results of our study in comparison with results from the main clinical trials that evaluated the interaction between the magnitude of myocardial viability and the extent of ventricular re-

modeling in the setting of successful late revascularization of IRA after MI. Our results are consistent with the results of these studies, although neither dealt specifically with the issue of the amount of viable myocardium that might influence the remodeling response of the ventricle in this setting. Our method of quantifying the residual viable myocardium in the IRA territory as a *proportion* is both unique and easily reproducible. We concluded that patients who, upon quantitative analysis, have more than 20% viable myocardium in the IRA territory experience a remodeling benefit, even when successful revascularization is performed late.

Although regional wall motion also improves in a comprehensive manner, global function improves in patients who have moderately or mildly reduced viability. This benefit in remodeling could translate into better clinical outcomes and prognosis.⁴³

An important angiographic observation from our study was that no preprocedural angiographic characteristic, including occlusive or nonocclusive IRA or baseline TIMI flow, correlated with improvement in LV function. In contrast, failure to achieve postprocedural TIMI 3 flow was more prevalent in patients with severely reduced preprocedural viability, which implies that failure to achieve postprocedural TIMI 3 flow is possibly a surrogate marker of reduced viability and of poor outcome.

Limitations of the Study

The arbitrary assignment of vascular territory by means of a polar plot could be a limitation, but the use of the total size of the nonviable region mitigates that limitation, because none of the subjects had any prior infarction. Another potential limitation of our study is its lack of a medical control arm, whereby stable patients who presented late after MI could have been treated medically with guideline-directed therapy and their LV indices compared at follow-up with the indices of patients undergoing successful PCI. It is therefore dif-

TABLE V. Comparison with Earlier Viability Studies

Variable	TOAT-CMR ¹⁴ (n=26)	OAT-NUC ¹⁵ (n=61 PCI + 63 MED)	Our Study (n=68; PCI Arm Only)
LVEDV	With increasing viability, does not improve in PCI or MED arm.	With increasing viability, does not improve, regardless of PCI or MED.	With increasing baseline viability, improves with PCI.
LVESV	With increasing viability, improves in PCI arm but not in MED arm.	With increasing viability, shows a trend toward improvement, although nonsignificant, regardless of PCI or MED.	With increasing baseline viability, improves with PCI.
LVEF	With increasing viability, improves in PCI arm but not in MED arm.	With increasing viability, shows a trend toward significant improvement, regardless of PCI or MED.	With increasing baseline viability, improves significantly with PCI.

LVEDV = left ventricular end-diastolic volume; LVEF = left ventricular ejection fraction; LVESV = left ventricular end-systolic volume; MED = medical therapy; OAT-NUC = Occluded Artery Trial-Nuclear Viability Ancillary Study; PCI = percutaneous coronary intervention; TOAT-CMR = Total Open Artery Trial-Cardiac Magnetic Resonance

difficult to say whether medical therapy alone would have yielded similar improvement in LV function. Further, the improvement that we found in LV function might not result in improved clinical outcomes; this would need to be investigated in larger randomized trials that apply similar methods for the quantitative evaluation of viability. In addition, our number of patients in Group 3 is so small that our failure to achieve statistical significance in that group might be the result of sample size, rather than magnitude of effect.

Conclusion

The most important message from this study is that successful revascularization of the IRA in patients who present late after an initial MI results in significant improvement in LVEDV, LVESV, and LVEF, except among patients with severely reduced myocardial viability (less than 20% viability in the IRA territory). This study, for the first time, quantitatively illuminates the important interaction of the amount of viable myocardium that is associated with functional improvement after late successful revascularization of the IRA.

This highly reproducible technique lends considerable weight to our contention that viability plays a significant role in the open-artery hypothesis: unless the patient has evidence of severely reduced viability in the IRA territory, he or she can be offered revascularization with the intent of improving LV function. This is a more scientific approach to such patients than is the outright refusal of revascularization on the basis of clinical outcome data alone. In this context, SPECT-MPI can be used as an adjunctive guide in choosing patients for revascularization.

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