

Prospective Identification of Myocardial Stunning Using Technetium-99m Sestamibi-Based Measurements of Infarct Size

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Objectives. We sought to prospectively identify patients with stunning and hyperkinesia at hospital discharge on the basis of mismatches between left ventricular (LV) function and infarct size as assessed by technetium-99m (Tc-99m) sestamibi perfusion tomographic imaging.

Background. Mechanical indexes of LV function may not accurately reflect myocardial damage after acute myocardial infarction (MI) because of myocardial stunning and compensatory hyperkinesia in noninfarct-related territories. Myocardial perfusion techniques are unaffected by these variables.

Methods. Eighty-four patients with acute MI underwent hospital admission and discharge Tc-99m-sestamibi tomographic imaging. Global LV ejection fraction (LVEF) was measured at hospital discharge and 6 weeks later. The perfusion defect size was quantified and expressed as a percentage of the LV. The discharge perfusion defect, which is a measure of infarct size, was used to predict the 6-week LVEF for each patient based on a previously reported regression equation. Patients were classified into one of three groups depending on whether their LVEF at hospital discharge fell within, above or below one standard error (6.8 LVEF points) of the predicted 6-week LVEF.

Results. There were 48 patients classified as having a "match" between function and infarct size; these patients demonstrated no

significant change in LVEF at 6 weeks. There were 21 patients (25%) classified as "mismatch stunned" who had discharge LVEFs lower than those predicted by infarct size. These patients demonstrated a significant improvement in mean LVEF at 6 weeks (mean [\pm SD] discharge LVEF 0.41 ± 0.08 , 6-week LVEF 0.47 ± 0.10 ; $p = 0.003$). Fifteen patients (18%) were classified as "mismatch-hyperkinetic." The mean LVEF for these patients significantly declined at 6 weeks (discharge LVEF 0.64 ± 0.06 , 6-week LVEF 0.58 ± 0.09 ; $p = 0.002$). There was a marked increase in LVEF within the infarct zone (8 ± 15 LVEF points; $p = 0.03$) for patients predicted to have stunning and a marked decline in LVEF outside the infarct zone (9 ± 15 LVEF points; $p = 0.06$) in patients predicted to have hyperkinesia. Both discharge LVEF ($p < 0.0001$) and group classification ($p = 0.005$) were independent predictors of LVEF 6 weeks later.

Conclusions. Perfusion imaging with Tc-99m-sestamibi can identify post-MI patients at hospital discharge in whom LV function is discordant with the measured infarct size. Patients with stunning have late increases in LVEF; patients with hyperkinesia have late decreases. This methodology, performed at discharge, is predictive of late changes in LV function.

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Left ventricular (LV) function is an important predictor of patients' prognosis after myocardial infarction (MI) (1,2) and is linearly related to infarct size (3). It is now well established that impaired LV function, especially in the immediate post-reperfusion setting, may not represent an irreversible state (4). Multiple reports have described a delayed improvement in left ventricular ejection fraction (LVEF) and regional wall motion in some patients who received acute reperfusion therapy during MI, presumably secondary to recovery of stunned myocardium (5-9). Consequently, early indices of mechanical function may be inaccurate in up to one-third of post-MI patients who receive early reperfusion therapy (6).

Technetium-99m (Tc-99m) sestamibi uptake and retention are generally not affected by myocardial stunning (10). Owing to favorable imaging qualities, infarct size with Tc-99m sestamibi can be accurately quantified (11-14). A previous study demonstrated that infarct size measured by Tc-99m sestamibi was linearly related to LV function 6 weeks after the index event in patients receiving early reperfusion therapy. It was evident that these measures could potentially be used to prospectively identify patients with myocardial stunning (6). The purpose of this study was to prospectively identify patients with stunning and hyperkinesia at hospital discharge on the basis of identifying mismatches between LV function and infarct size measured by Tc-99m sestamibi tomographic imaging in a separate patient cohort.

Methods

Study group. The study group consisted of a consecutive series of patients enrolled in a prospective trial (15) of Tc-99m

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Abbreviations and Acronyms

ANCOVA	= analysis of covariance
ANOVA	= analysis of variance
ECG	= electrocardiogram, electrocardiographic
LV	= left ventricle, left ventricular
LVEF	= left ventricular ejection fraction
MI	= myocardial infarction
Tc-99m	= technetium-99m

sestamibi between 1988 and 1992 who met the following inclusion criteria: 1) chest pain ≥ 30 min in duration; 2) electrocardiographic (ECG) ST segment elevation at least 0.1 mV in two or more leads in the same vascular territory; 3) significant elevation in creatine kinase, MB isoenzyme within 24 h of chest pain; and 4) rest equilibrium gated radionuclide ventriculography performed at hospital discharge and at 6 weeks after the infarction. Exclusion criteria included 1) recurrent acute MI ($n = 1$) or a coronary revascularization procedure between hospital discharge and 6 weeks ($n = 5$); 2) left bundle branch block, ventricularly paced rhythm or ventricular pre-excitation syndrome; 3) atrial fibrillation during radionuclide ventriculography; and 4) inclusion in a previous study on myocardial stunning (6). Eighty-four patients met these criteria and formed the study group.

Radionuclide studies. Technetium-99m sestamibi tomographic acquisitions were performed on hospital admission and at discharge 1 to 6 h after injection of 20 to 30 mCi of Tc-99m-sestamibi using a previously described technique (14-16). For acute imaging, Tc-99m-sestamibi was injected before any reperfusion therapy, and acquisition was delayed until completion of the therapy (16-19). Quantification of the extent of LV severe hypoperfusion was performed from five short-axis slices using a 60% of maximum counts threshold method, which has been previously described (14-16). The extent of the acute and discharge defects is a measure of myocardium at risk and infarct size, respectively, with the difference (acute-discharge defect extent) reflecting myocardial salvage (10,17). Measures of myocardium at risk by Tc-99m-sestamibi in animal models of permanent coronary occlusion as well as after reperfusion have been validated (12,18). Quantified infarct size with Tc-99m sestamibi has been extensively studied. This measure has been carefully validated in animal models of coronary occlusion and reperfusion (11,18). It has correlated closely with other clinical markers of infarct size, including cardiac enzyme release (20), global and regional systolic function (6,13,21), thallium defect size (22) and human pathology (23).

Gated equilibrium radionuclide ventriculography with Tc-99m labeled red blood cells was performed at hospital discharge (1 day after Tc-99m-sestamibi imaging) and at 6 weeks using the modified in vivo method of Callahan et al. (24). Gated images were acquired and processed using previously described techniques (25). Global LVEF was calculated from the background-corrected LV counts versus the time curve by

use of a commercially available operator-interactive program (25). The standard deviation (SD) of the difference between repeat measurements obtained by this technique is 0.04 (26). The 95% confidence limit for a definite change in LV ejection fraction (LVEF) was therefore considered to be ≥ 0.08 and a probable improvement (67% confidence limits) was a change of >0.04 .

Regional LVEF was obtained by dividing the LV region into four quadrants in the 45° left anterior oblique view from a fixed center derived from the end-diastolic images (27). These quadrants defined the anterior, septal, inferior and lateral territories. Regional changes in LVEF were classified as within the infarct zone and outside the infarct zone. The anterior quadrant was excluded, as it represents primarily valve plane motion. For patients with an anterior infarction, the infarct zone consisted of the septal quadrant. For patients with an inferior or lateral infarction, the corresponding quadrant was defined as the infarct zone. The two quadrants not in the infarct zone were averaged to obtain representative values outside the infarct zone. The SD of reproducibility of this methodology is 3.5 LVEF points for intraobserver measurements and 4.2 LVEF points for interobserver measurements (27). Six patients did not have regional LVEF values calculated because their data could not be retrieved from the archive.

Predicted global late LVEF. A predicted global LVEF was calculated for each patient based on the previously reported relation between infarct size by Tc-99m sestamibi and LVEF 6 weeks later in a separate patient cohort (6). The equation of this regression line was:

$$\text{Predicted LVEF} = (-0.47) (\text{Infarct size } [\%LV]) + 58$$

The correlation (r) and standard error of the estimate (SEE) were -0.81 and 0.067 , respectively (Fig. 1).

By design, none of the patients used to derive this regression equation of predicted LVEF are included in this report. For the present study, those patients whose discharge LVEF fell within 1 standard error (SE) of their predicted LVEF 6 weeks later were assigned to the "matched" group; no significant change in 6-week LVEF was predicted for this group (Fig. 1). Patients whose discharge LVEF was >1 SE above the predicted late LVEF were classified as "mismatched-hyperkinetic." These patients were expected to have a decrease in their global LVEF between hospital discharge and 6 weeks. Patients whose global LVEF at discharge was >1 SD below the predicted LVEF were classified as "mismatched-stunned." These patients were expected to have a significant increase in their global LVEF between discharge and 6 weeks.

Statistical analysis. Data are presented as mean values \pm SD. Simple linear regression analysis was used to compare Tc-99m sestamibi defect size with LVEF at hospital discharge. Repeated measures analysis of variance (ANOVA) was used to compare LVEF at discharge and 6 weeks later. One-factor ANOVA was used to compare changes in global and regional LVEF by group classification. Analysis of covariance (ANCOVA) was performed to make certain that changes in

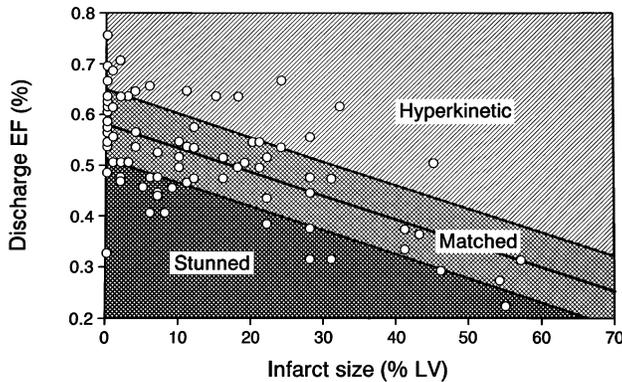


Figure 1. Previously derived relation between infarct size by Tc-99m sestamibi and LVEF 6 weeks later in a separate group of 32 patients undergoing successful reperfusion therapy. **Central line** = line of regression, **upper and lower lines** = boundaries of 1 SEE from the regression line. The three classification groups are represented by the differently shaded regions. Data points from the 84 patients in the present study are superimposed over the previously defined relation between discharge infarct size and 6-week LVEF. These values reflect discharge LVEF. Most patients fell within the previously derived range, but there were outliers in both directions. These patients were predicted to move into the predicted range (matched zone) 6 weeks later.

LVEF between hospital discharge and 6 weeks were not solely a function of the discharge LVEF (regression to mean). In this analysis the dependent variable was 6-week LVEF and the independent variables were discharge LVEF and group assignment. A value <0.05 was considered significant. All analyses were done using commercially available statistical software (Statview 4.1 and Superanova 1.0, Abacus concepts).

Results

The clinical characteristics of the study group are detailed in Table 1. The relation between global LVEF and predicted LVEF, based on the perfusion defect size for each patient, is shown in Figure 1. Patients were classified on the basis of this relation into three groups. There were 48 patients (57%) whose discharge LVEF was within 1 SE of the previously reported regression line relating discharge infarct size by Tc-99m sestamibi with 6-week LVEF. Twenty-one

patients (25%) had a LVEF >1 SE below the regression line (mismatch-stunned). Clinical characteristics were similar by group classification, with the exception of infarct location. There was a greater percentage of patients with an anterior infarction in the mismatch-stunned group (67%) and a lower percentage of patients with an anterior infarction in the other two groups. Acute reperfusion therapy was given to 79 of 84 patients; 38 underwent direct percutaneous transluminal coronary angioplasty and 41 received intravenous thrombolytic therapy. There were no differences in reperfusion modality or time to reperfusion therapy between the groups.

Perfusion imaging variables (Table 2). There was a trend ($p = 0.08$) toward a difference in myocardium at risk between the groups. Myocardium at risk tended to be greater for patients classified as “mismatch-stunned” ($39 \pm 21\%$ of the LV) compared with those classified as matched ($30 \pm 19\%$ of the LV) or mismatched-hyperkinetic ($25 \pm 20\%$ of the LV). There was no difference in infarct size between the groups, however. Consequently, the mean predicted global LVEF for all three groups was similar. Myocardial salvage was greatest for patients classified as “mismatch-stunned”, and there was a significant ($p = 0.03$) difference in myocardial salvage between the three groups.

Global LV function (Fig. 1 to 3, Table 2). LVEF at hospital discharge was well preserved for the study group (0.51 ± 0.11), and there was no significant overall change at 6 weeks (0.52 ± 0.10). The mean predicted LVEF for the study group was not significantly different from either of the actual acquisitions (0.52 ± 0.07).

However, there were significant differences in LVEF between the groups. The mismatch-stunned group had an initially low mean LVEF of 0.41 ± 0.08 , which improved significantly by 6 weeks (6-week LVEF 0.47 ± 0.10 , $p = 0.003$ vs. discharge LVEF). However, these values were still significantly lower than predicted (0.52 ± 0.07 , $p = 0.004$ vs. 6-week LVEF). On an individual basis, 13 (62%) of 21 patients in the mismatch-stunned group had a probable improvement in LVEF (>0.04) and 7 (33%) of 21 had a definite improvement (>0.08).

The mismatch-hyperkinetic group had a high LVEF of 0.64 ± 0.06 . At 6 weeks these values had declined to 0.58 ± 0.09 ($p = 0.002$ vs. discharge LVEF), but were still significantly

Table 1. Clinical Characteristics by Classification

	Overall (n = 84)	Match (n = 48)	Mismatch- Stunned (n = 21)	Mismatch- Hyperkinetic (n = 15)	p Value*
Age (yr)	60 ± 11	59 ± 12	60 ± 12	66 ± 10	0.13
Male	74%	79%	62%	73%	NS
Anterior location	37%	29%	67%	20%	0.01
Acute reperfusion therapy (PTCA/thrombolysis)	45%/49%	46%/50%	48%/48%	40%/47%	NS
Time to reperfusion therapy (h)	4.5 ± 3.6	4.5 ± 3.3	4.5 ± 3.9	4.5 ± 4.2	NS

*Analysis of variance between the three group classifications. Data are presented as mean values ± SD or percentages. PTCA = percutaneous transluminal coronary angioplasty.

Table 2. Radionuclide Variables by Classification (expressed as percent of left ventricle)

	Overall (n = 84)	Match (n = 48)	Mismatch- Stunned (n = 21)	Mismatch- Hyperkinetic (n = 15)	p Value*
Perfusion					
Myocardium at risk	31 ± 20	30 ± 19	39 ± 21	25 ± 20	0.08
Infarct size	13 ± 15	13 ± 15	13 ± 16	14 ± 14	NS
Salvage	18 ± 17	17 ± 16	26 ± 18	12 ± 16	0.03
Left Ventricular Function					
Predicted LVEF	0.52 ± 0.07	0.52 ± 0.07	0.52 ± 0.07	0.52 ± 0.06	NS
Discharge LVEF	0.52 ± 0.11	0.52 ± 0.08	0.41 ± 0.08	0.64 ± 0.06	< 0.0001
6-week LVEF	0.52 ± 0.10	0.52 ± 0.10	0.47 ± 0.10	0.58 ± 0.09	0.007
6 week vs. discharge LVEF	0 ± 0.08	0 ± 0.06	0.06 ± 0.08	-0.06 ± 0.06	< 0.0001

*Between-group analysis of variance. Data are presented as mean values ± SD. LVEF = left ventricular ejection fraction.

higher than predicted (0.52 ± 0.06 , $p = 0.008$ vs. 6-week LVEF). Nine (60%) of 15 patients had a probable decline in LVEF (≥ 0.04) and 7 (47%) of 15 had a definite decline (≥ 0.08). The matched group showed no significant group change in LVEF (0.52 ± 0.08 at discharge vs. 0.52 ± 0.10 at 6 weeks, $p = \text{NS}$), and neither value was different from the predicted LVEF based on perfusion defect size at discharge (0.52 ± 0.07). There were fairly equal percentages of patients demonstrating a probable increase (25%) as well as a probable decrease (29%) in LVEF and for those demonstrating a definite increase (12%) and decrease (10%).

Regional changes in LV function (Fig. 4, Table 3). *Within the infarct zone.* There was a significant difference ($p = 0.002$) in regional LVEF within the infarct zone between the three groups (Table 3), with the mismatch-stunned group showing the lowest and the mismatch-hyperkinetic group showing the highest values. This difference resolved at 6 weeks. An overall significant improvement in regional LVEF from hospital discharge to 6 weeks occurred within the infarct zone (0.52 ± 0.17 at discharge to 0.55 ± 0.16 at 6 weeks, $p = 0.05$). This change was primarily due to an improvement in the regional LVEF within the infarct zone for patients classified as mismatch-

stunned (0.42 ± 0.17 to 0.50 ± 0.15 , $p = 0.03$). The change in regional LVEF within the infarct zone (6 week–discharge) is shown in Figure 4A. There was a strong trend toward a difference in the change in LVEF within the infarct zone by group classification.

Outside the infarct zone. There was no overall change in LVEF outside the infarct zone for the study group as a whole (Table 3). There were significant differences in regional LVEF values at hospital discharge by group classification, with the mismatch-hyperkinetic group having the highest and the mismatch-stunned group having the lowest values ($p < 0.0001$). Consistent with the analysis within the infarct zone, this difference was no longer significant at 6 weeks. The largest magnitude of change (a decrease in LVEF) was found in the mismatch-hyperkinetic group ($p = 0.06$, Table 3). The overall differences in the change in regional LVEF outside the infarct zone was significant by classification group (Fig. 4B).

Multivariate analysis. Because of the differences in discharge global LVEF between the groups, it is conceivable that the differences in the change in LVEF from discharge to 6 weeks between the groups might merely represent regression to the mean. However, by ANCOVA, both discharge EF ($p <$

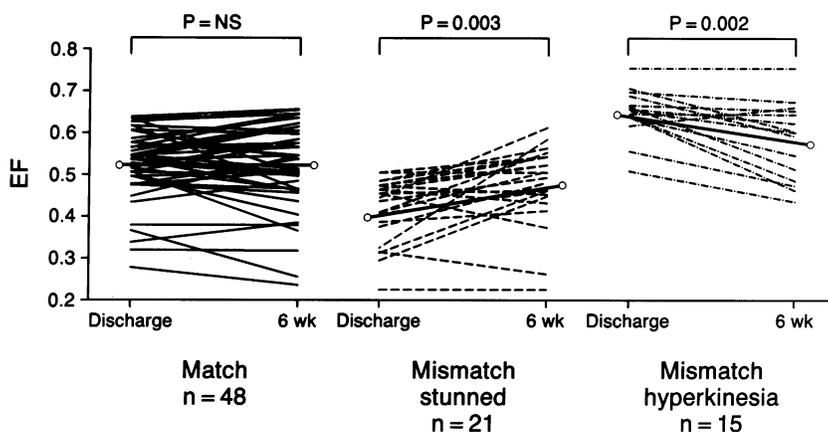


Figure 2. Discharge and 6-week LVEF values for each patient according to the three prospective classifications. There was a significant increase in LVEF in the “mismatch-stunned” group and a significant decrease in LVEF in the “mismatch-hyperkinetic” group. Mean values are shown by solid lines with open circles. EF = ejection fraction.

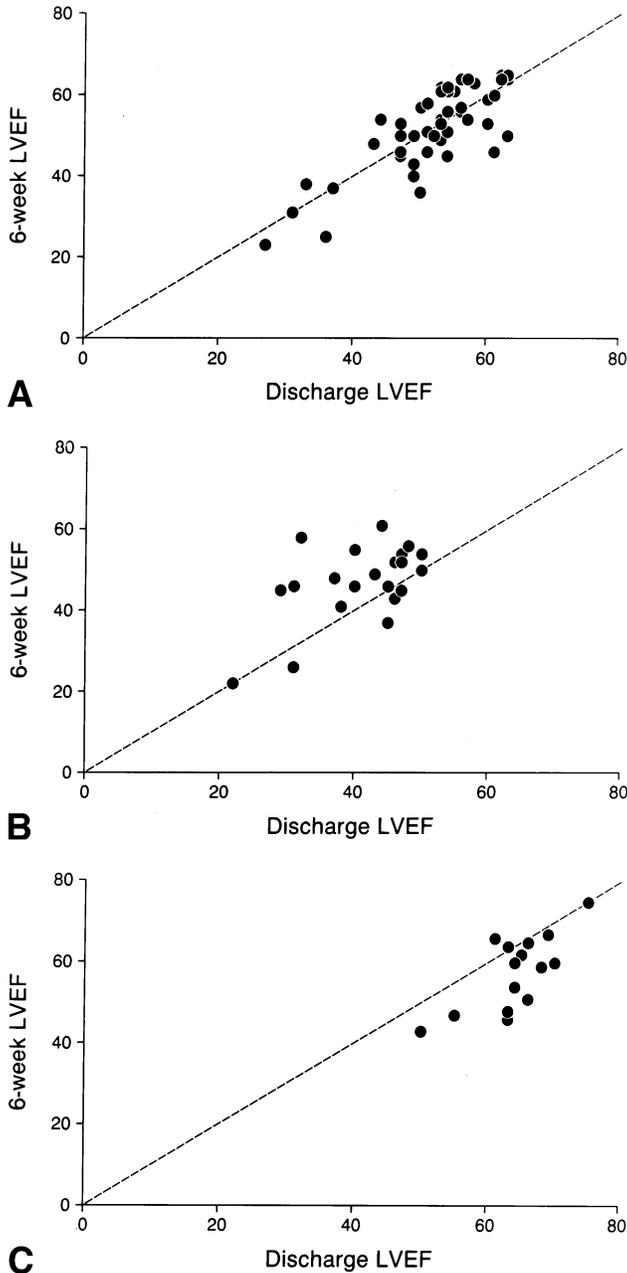


Figure 3. Scatterplots by prospective classification group by initial and late LVEF. **A**, Match group; **B** (stunning) and **C** (hyperkinetic), mismatch groups. **Dashed line** = line of identity. The majority of patients in the two mismatch groups (**B** and **C**) are on one side of the line of identity. The distribution is more equal in the match group (**A**). The relation between initial and late LVEF was significantly different ($p = 0.005$) between the three groups.

0.0001) and group classification ($p = 0.005$) were independent determinants of 6-week LVEF.

Discussion

Myocardial stunning is characterized by a mismatch of myocardial perfusion (normal) and function (depressed)

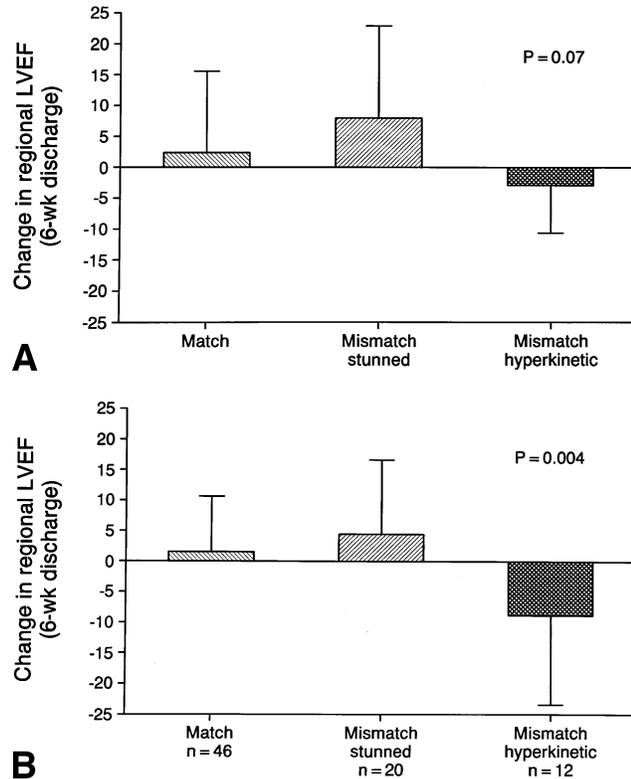


Figure 4. Change in regional LVEF values (6 weeks vs. discharge) by group classification. **A**, Within the infarct zone. **B**, Outside the infarct zone. Data are presented as mean values \pm SD.

within viable myocardium, which improves spontaneously over time. There are relatively few clinical studies that directly examine both perfusion and function to identify myocardial stunning. Most studies have deduced the presence of myocardial stunning by a temporal resolution in myocardial dysfunction (5,7-9). However, to accurately diagnose the presence of

Table 3. Regional Left Ventricular Ejection Fraction Values

	No. of Pts	Discharge	6 Weeks	p Value (discharge vs. 6 weeks)
Within infarct zone				
Overall	78	0.52 \pm 0.17	0.55 \pm 0.16	0.05
Match	46	0.53 \pm 0.14	0.55 \pm 0.15	>0.20
Mismatch-stunned	20	0.42 \pm 0.17	0.50 \pm 0.15	0.03
Mismatch-hyperkinesia	12	0.63 \pm 0.19	0.60 \pm 0.20	>0.20
		$p = 0.002^*$	$p > 0.20^\dagger$	
Outside infarct zone				
Overall	78	0.51 \pm 0.13	0.52 \pm 0.13	>0.20
Match	46	0.50 \pm 0.11	0.52 \pm 0.11	>0.20
Mismatch-stunned	20	0.44 \pm 0.14	0.49 \pm 0.14	0.12
Mismatch-hyperkinesia	12	0.65 \pm 0.10	0.56 \pm 0.15	0.06
		$p < 0.0001^*$	$p > 0.20^\dagger$	

*Analysis of variance between-group classification for regional left ventricular ejection fraction at discharge. \dagger Analysis of variance between-group classification for regional left ventricular ejection fraction at 6 weeks. Pts = patients.

uncoupling of flow and contractility, both flow and function must be simultaneously evaluated.

The present study was designed to simultaneously measure indices of perfusion and LV systolic performance at the time of discharge in postinfarction patients to identify discordance between these two variables. Technetium-99m sestamibi is uniquely suited to assess perfusion and infarct size because of its linear uptake with myocardial blood flow (28) and the optimal physical characteristics that allow for accurate quantification of infarct size (14). Uptake and retention of Tc-99m-sestamibi is dependent on cellular viability (10,29). Consequently, myocardial perfusion can be assessed within the LV and the extent of infarction can be defined. The uptake and retention of the tracer to assess perfusion do not appear to be affected by myocardial stunning (6,10).

The mechanisms that produce stunning have not been unequivocally defined; actually, a combination of factors may be involved (30). Oxygen free radicals indiscriminately disrupt metabolic pathways. It has been postulated that the generation of oxygen free radicals from reperfusion flow may interfere with the function of sarcoplasmic reticulum, producing calcium overload in the cytosol (31). Technetium-99m sestamibi is largely sequestered within mitochondria owing to its large cationic charge (32). Consequently, there is no physiologic reason to suspect its uptake and retention are affected by the presence of stunning so long as blood flow is restored. Sinusas et al. (10), in a carefully designed series of experiments, have demonstrated that sestamibi kinetics within myocytes are unaffected by the presence of stunning produced in an animal model. Based on these observations and other published data demonstrating the lack of uptake in regions of infarcted myocardium despite reperfusion flow (12,18), Tc-99m-sestamibi is an attractive agent used to help study myocardial stunning.

A previously derived relation between infarct size with Tc-99m sestamibi and LVEF 6 weeks later (when stunning is likely to have resolved) was used to generate a predicted late LVEF in a separate patient group. Discordance between the actual LVEF at discharge and the predicted late LVEF was used to identify patients likely to demonstrate significant changes in global LV function (6). These classifications were reasonably accurate. For patients classified as having myocardial stunning, there was a significant increase in mean global LVEF over time, and 60% of patients had an improvement in LVEF. Mean regional LVEF values showed a significant improvement in contractility within the infarct zone in these prospectively classified patients. It is of interest that patients in this group predominantly had an anterior infarction and, consequently, a greater amount of myocardium at risk and subsequent myocardial salvage (17). The larger territory exposed to reperfusion flow may have predisposed these patients to more clinically significant depression in LV function. For patients whose LVEF was inappropriately high for the extent of infarction, there was a significant decline for the group as a whole, and the majority of patients (60%) showed a probable decline in LVEF. Regional LVEF values showed a marked decline outside the zone of infarction for this subgroup of

patients, which was not apparent in the other two subgroups. This phenomenon in post-MI patients has been previously described and may be related to fluctuations in catecholamine release (33).

The temporal changes in LV function found in this study are in agreement with several previous reports that suggest that stunning at the time of hospital dismissal is present in only a portion of patients receiving reperfusion therapy (6,34). Overestimation of LV function from compensatory hyperkinesia is as likely to be present as stunning after reperfusion therapy. Bestetti et al. (34) found that 30% of patients treated with thrombolysis had a significant improvement in LV function from 3 weeks to 6 months after the index infarction (34). The percentage of patients showing late improvement in LVEF in this study (25%) is similar to that in the pilot data (6) on which this study was based (15%). The percentage of patients showing a late decline in LVEF was identical in this study and the pilot study (18% for both) (6). As in the pilot study (6) and other reports (35), those patients showing improvement had a lower LVEF initially; those showing a decline had higher LVEF values initially. To our knowledge, this is one of the first studies to propose a methodology to predict future changes in LV function at a time point where clinical decisions regarding therapy are often required.

Study limitations. Follow-up imaging of the study group only extended to 6 weeks after the index infarction and no later. This prevents any exploration of the more prolonged condition of myocardial hibernation, which has been reported by others using Tc-99m sestamibi imaging after infarction (36,37).

It is conceivable that all the directional changes in LVEF are merely due to regression to the mean; patients with a "low" LVEF at hospital discharge will tend to increase on retesting. To adjust for this effect, we used a multivariate analysis with group classification and discharge LVEF as factors in determining the association with 6-week LVEF. If all the results could be explained by this phenomenon, then the factor of group classification should not be an independent predictor of 6-week LVEF once the discharge LVEF was known. Because group classification was significant and independent of the discharge LVEF, the results cannot be solely attributed to regression to the mean, but this phenomenon could still be partially responsible for the directional changes seen within the groups.

The directional changes seen on the regional LVEF analysis were similar both inside and outside the infarct zone. Although the LVEF in the stunned group improved significantly within the infarct zone, it also tended to improve outside the zone. A similar pattern of declining LVEF was seen in the hyperkinetic group. This may reflect extensions of these physiologic effects beyond the boundaries defined by the segments on the radio-nuclide angiogram, which are only approximations of the expected extent of an infarct by location. It should be noted that the reproducibility of regional LVEF values may not be as precise compared with the global determination of LVEF. Consequently, the regional findings should be interpreted with

more caution. The measurement of LVEF may be less accurate in the presence of mitral regurgitation and apical aneurysms.

Conclusions. This study provides a unique method to prospectively identify patients with stunning or hyperkinesia fairly early (~1 week) after the index infarction, using rest tomographic imaging with Tc-99m sestamibi. The simultaneous assessment of global LV function from first-pass Tc-99m sestamibi acquisitions (38) and gating of the perfusion images (39) are an attractive method to assess both perfusion and function in a single study. Other techniques may hold promise in this regard (40,41). The goal should be to assess both perfusion and function early to identify patients likely to demonstrate late changes in systolic performance. Mismatches between function and perfusion at ~1 week after reperfusion therapy for acute MI are present in a little over one-third of patients.

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