JACC Vol. 25, No. 3
March 1, 1995:590–6

Technetium-99m Sestamibi Tomographic Evaluation of Residual Ischemia After Anterior Myocardial Infarction

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Objectives. **This study investigated the value of sestamibi scintigraphy in assessing residual ischemia after anterior myocardial infarction.**

Background. **Serial imaging with sestamibi, the uptake and retention of which correlate with regional myocardial blood flow and viability, has been used to estimate salvaged myocardium and risk area after acute infarction. We recently documented that recovery of perfusion and contraction in the infarcted area may continue well after the subacute phase, suggesting myocardial hibernation. Some underestimation of viability in the setting of hibernating myocardium by sestamibi imaging has been reported.**

Methods. **We studied 58 patients in stable condition after Q wave anterior infarction. Regional perfusion and function were quantitatively assessed by sestamibi tomography and two-dimensional echocardiography at 4 to 6 weeks and at 7 months after infarction. In sestamibi polar maps, abnormal areas with tracer uptake >2.5 SD below our reference values were computed at rest and after symptom-limited exercise. On two-dimensional echocardiography the ejection fraction and extent of rest wall motion abnormalities were assessed by a computerized system. All patients had coronary angiography between the two studies.**

Results. **At 7 months the extent of rest sestamibi defect was significantly reduced in 40 patients (69%, group 1) and unchanged in 18 (31%, group 2). Rest wall motion abnormalities and ventricular ejection fraction significantly improved in group 1 but not in group 2. Underlying coronary disease, patency of the infarct-related vessel and rest sestamibi defect extent at 5 weeks were comparable between the two groups. At 7 months, an increase in the reversible (stress–rest defect) tracer defect was observed in group 1 ($p < 0.05$) despite a smaller stress-induced hypoperfusion ($p < 0.05$). Reversible sestamibi defects and stress hypoperfusion were unchanged in group 2. In 38 (95%) of 40 group 1 patients, the area showing reversible sestamibi defects at 7 months matched the area showing fixed hypoperfusion at 5 weeks.**

Conclusions. **The reduction in the rest tracer uptake defect that can occur late after infarction may affect the assessment of ischemic burden by sestamibi imaging early after anterior myocardial infarction.**

(J Am Coll Cardiol 1995;25:590–6)

In the management of patients with ischemic heart disease and abnormal rest electrocardiographic (ECG) results, stress imaging techniques can be preferable to exercise electrocardiography alone (1). Patients with anterior Q wave infarction are frequently referred for radionuclide study because ST segment shifts on exercise stress testing cannot reliably identify the presence of residual ischemia (2,3).

Technetium-99m methoxyisobutylisonitrile (sestamibi) is a myocardial perfusion agent whose uptake and retention correlate closely with regional myocardial blood flow and tissue viability (4–6). Recent clinical studies have demonstrated its usefulness in assessing the risk area during the acute phase of myocardial infarction (7,8) and in determining the efficacy of various reperfusion strategies (9,10). The changes in the size and severity of the perfusion defect assessed by serial sestamibi imaging after thrombolytic therapy have been used to estimate salvaged myocardium and, thereby, patency of the infarct-related vessel (11,12).

However, the time course of the recovery of myocardial perfusion and function after myocardial infarction and its effect on the detection of residual ischemia remain unclear. Indeed, we recently documented (13) that recovery of perfusion and contraction in the infarcted area may continue well after the subacute phase in a sizable number of patients with recent anterior infarction, suggesting myocardial hibernation. Moreover, underestimation of myocardial viability in the setting of hibernating myocardium by sestamibi imaging has recently been acknowledged (14–17).

The aim of this study was to investigate the possible effects of the evolving tomographic estimate of myocardial "infarct area" by sestamibi on the assessment of residual ischemia in a

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Manuscript received January 6, 1994; revised manuscript received July 15, 1994, accepted October 3, 1994.

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0735-1097/95/$9.50
0735-1097(94)00451-U

selected group of patients with a first Q wave anterior myocardial infarction.

Methods

Study cohort. We prospectively studied 58 male patients (49 ± 8 years) with recent anterior myocardial infarction who met the following inclusion criteria: 1) history of a recent (4 to 6 weeks) first Q wave anterior myocardial infarction; 2) New York Heart Association functional class I or II; 3) no angina at rest; 4) sinus rhythm and no conduction disturbances; and 5) two-dimensional echocardiographic images of adequate quality for quantitative analysis. The diagnosis of acute myocardial infarction was supported by typical history of chest pain (>30 min), abnormal Q waves in at least two adjacent precordial leads and in lead I or aVL, or both, and a typical pattern of elevated serum myocardial enzymes. Patients with unstable angina, low threshold ischemia or angina uncontrolled by medical therapy, as well as those with severe left ventricular dysfunction (rest ejection fraction <25%), were excluded. Rest scintigraphic and echocardiographic data from 52 of these patients were included in a previous report (13).

Study protocol. Regional myocardial perfusion was evaluated with sestamibi single-photon emission tomography at rest and after exercise. Left ventricular function was assessed at rest by two-dimensional echocardiography. Resting sestamibi and echocardiographic studies were performed on the same day; exercise scintigraphy was performed within 24 h. All patients underwent the scintigraphic and echocardiographic studies 4 to 6 weeks after the necrotic episode (study 1) and then 6 months later (study 2). Patients had to be in stable condition during the interval between the two studies. Cardioactive drugs were discontinued at least 48 h before each evaluation. The study protocol was approved by the local Ethical Committee on Human Research, and written informed consent was obtained from all patients.

Sestamibi imaging and analysis. For both study 1 and study 2, a 2-day imaging protocol was used for rest and exercise scans (in a random order). A multistage symptom-limited exercise was performed; a 12-lead ECG was recorded every minute and blood pressure every 3 min. An incremental load of 25 W was given every 3 min; at peak exercise, the tracer was injected intravenously, after which patients were encouraged to continue exercising for 60 to 90 s.

The method of sestamibi scan acquisition and processing has recently been described (13). Briefly, both the rest and exercise scintigraphic image acquisitions were performed within 90 to 120 min from the tracer injection (925 MBq/70 kg). The camera head (Apex 409, Elscint, Israel) was rotated in a 180° arc in a circular orbit with 3° increments of 25 s in a step-and-shoot mode. Data were collected in a 64 × 64 array with a pixel size of 4.5 mm. Particular care was taken to avoid major artifacts, such as patient motion during acquisition. Transaxial slices were reconstructed using a filtered back-projection algorithm with a modified Wiener filter without attenuation or scatter correction; flood correction was applied during reconstruction. The spatial resolution in the transaxial plane was 8 mm. Short- and long-axis (horizontal and vertical) tomograms were reconstructed from the transaxial slices. Slices in the short-axis view were reconstructed, and polar maps of regional sestamibi distribution were displayed, both at rest and after exercise. Each polar map was normalized for peak myocardial activity and compared with our normal limits: Pixels with tracer uptake <2.5 SD below mean normal values were considered abnormal. The abnormal area on each short-axis slice was first multiplied by a correction factor (18) that corrects the spatial distortion and allows for differences in myocardial slice mass from apex to base. Corrected abnormal areas were then summed to obtain the total size of left ventricular defect at rest and after exercise, expressed as a percent of the left ventricular surface. Changes in the perfusion defect size from study 1 to study 2 and from rest to exercise studies were considered significant when they exceeded the 95% confidence limits of the method variability (13).

Echocardiographic data acquisition and analysis. Our methods of data acquisition and image digitalization and the computerized system for the automatic detection and quantification of regional wall motion and left ventricular function have been previously described (19,20). Briefly, the three apical views (four and two chambers and apical long axis) were analyzed. The endocardial contour of each view was automatically divided into 23 segments of equal length, so that the entire ventricular wall was represented by a total of 69 segments. By comparison with our normal data base, the presence of abnormal wall motion was automatically detected when the fractional shortening area from end-diastole to end-systole of each segment decreased 2 SD of the mean values in normal subjects. The extent of wall motion abnormalities was expressed as a percent of total endocardial length. Left ventricular ejection fraction was calculated using the biplane area–length method. All measurements were derived in blinded manner by a single experienced operator from three consecutive cardiac cycles, and the mean values were considered. Intraobserver variability values in endocardial contouring and in the evaluation of end-systolic and end-diastolic endocardial surface area by our quantitative analysis in normal subjects and in patients with different degrees of left ventricular dysfunction have been reported elsewhere (21).

Coronary angiography. All patients underwent cardiac catheterization by the Judkins technique during the 6-month interval between the two studies (on average 95 ± 12 days from study 1). After intracoronary administration of nitroglycerin, selective angiograms were obtained in at least two projections for the right coronary artery and in at least four for the left coronary artery. The presence of a significant (>50% coronary diameter reduction) stenosis was assessed using caliper measurements in two orthogonal projections by one experienced investigator who had no other information. The Thrombolysis in Myocardial Infarction (TIMI) grade of the infarct-related vessel patency was determined (0 to 1 = occluded artery; 2 to 3 = patent artery). The presence of collateral circulation was also assessed.

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Table 1. Clinical Characteristics of the 58 Study Patients

Age (yr)	49 ± 8
History of	
Smoking	39 (67%)
Hypertension	14 (24%)
Diabetes	5 (9%)
Acute phase of infarction	
Systemic thrombolysis	42 (72%)
Peak serum CK (IU/ml)	3,472 ± 2,181
Killip class I	51 (82%)
Killip class II	7 (12%)
Medications	
Beta-blockers	42 (72%)
Nitrates	16 (27%)
ACE inhibitors	5 (9%)
Calcium channel blockers	5 (9%)

Data presented are mean value ± SD or number (%) of patients. ACE = angiotensin-converting enzyme; CK = creatinine kinase.

The vascular attribution of scintigraphic and echocardiographic regions to the conventional anatomic distribution of the major coronary arteries was performed according to the division proposed by the Cedars-Sinai Laboratory (22) and the American Society of Echocardiography (23), respectively.

Statistical analysis. Data are reported as mean value ± SD. Normality of the data distribution was verified. The extent of the sestamibi perfusion defects at rest and after exercise, left ventricular volumes, ejection fraction and percent wall motion abnormality at studies 1 and 2 were compared using the Student *t* test for paired data. Baseline characteristics between groups were compared using the unpaired Student *t* test for continuous variables and the chi-square test to determine the significance of differences in rates of occurrence. For intergroup comparisons of scintigraphic and echocardiographic variables at studies 1 and 2, a one-way analysis of variance combined with the Scheffé test was used; $p < 0.05$ (two-tailed) was considered significant.

Results

Of the 66 patients who underwent rest and stress sestamibi imaging 5 weeks after infarction, 2 died in the subsequent months (1 of sudden cardiac death, 1 of cancer); 6 others showed a high risk exertional scintigraphic pattern (as defined by multiple perfusion defects) and soon underwent coronary artery bypass graft surgery or coronary angioplasty because of clinical worsening or extensive coronary disease. The remaining 58 medically treated patients underwent coronary angiography and completed the serial protocol 6 months later while in stable condition. Their clinical characteristics are reported in Table 1.

Coronary anatomy. Significant stenoses involving one and two vessels were found in 39 (67%) and 14 (24%) patients, respectively. In five patients (9%), no significant residual stenosis was documented. The infarct-related vessel stenosis affected the proximal left anterior descending coronary artery in 25 patients (47%) and its mid to distal portion in 28 (53%); angiographically visible collateral vessels toward the infarct-related vessel were observed in 20 patients (38%).

Serial rest sestamibi and echocardiographic results. At study 1, all patients showed sestamibi uptake defects and wall motion abnormalities in the region supplied by the left anterior descending coronary artery. The extent of hypoperfused left ventricle ranged from 2% to 55%; the percent wall motion abnormality ranged from 4% to 61%. At study 2, the entire study cohort had a significant reduction in the extent of perfusion defect (from 33 ± 13% to 26 ± 16%, $p < 0.001$) and percent wall motion abnormality (from 35 ± 15% to 31 ± 19%, $p < 0.01$) as well as a significant improvement in left ventricular ejection fraction values (from 48 ± 13% to 50 ± 15%, $p < 0.01$).

According to the changes in the rest perfusion defect from study 1 to study 2, sestamibi defect was smaller in 40 patients (69%, group 1) and unchanged in 18 (31%, group 2). There were no significant differences in perfusion defect extent between the two groups at study 1; however, group 2 patients had a significantly lower ejection fraction and higher percent wall motion abnormality (Table 2). At study 2, there was a significant improvement in the percent wall motion abnormality and ejection fraction in group 1 but not in group 2 patients. Other clinical and angiographic characteristics of the two groups are reported in Table 3. Age, thrombolysis in the acute phase, extent of underlying coronary disease, TIMI grade of the infarct vessel patency and collateral vessels to the infarct-related vessel were comparable between the two groups.

Serial exercise sestamibi results. Significant ECG changes (ST segment depression >1 mm), chest pain and reversible

Table 2. Scintigraphic and Echocardiographic Data at 4 to 6 Weeks (study 1) and at 7 Months (study 2) After Infarction in Patients With (group 1) and Without (group 2) Improvement in Rest Sestamibi Uptake

	Group 1 (n = 40)		Group 2 (n = 18)	
	Study 1	Study 2	Study 1	Study 2
Sestamibi defect extent at rest (%)	33 ± 14	23 ± 15*	33 ± 13	36 ± 19†
Left ventricular ejection fraction (%)	50 ± 13	53 ± 14*	42 ± 10†	43 ± 12
Wall motion abnormalities (%)	33 ± 16	28 ± 20‡	39 ± 12†	38 ± 14

*$p < 0.001$ and ‡$p < 0.01$ versus study 1. †$p < 0.05$ versus group 1 at corresponding time. Data presented are mean value ± SD.

Table 3. Clinical and Angiographic Findings in Patients With (group 1) and Without (group 2) Improvement in Rest Sestamibi Uptake Defect After 6 Months

	Group 1 (n = 40)	Group 2 (n = 18)	p Value
Age (yr)	49 ± 7	47 ± 10	NS
Acute phase of infarction			
Peak serum CK (IU/ml)	2,666 ± 1,978	4,276 ± 2,385	<0.05
Thrombolysis	29 (72%)	13 (72%)	NS
Coronary angiography			
No CAD	3 (8%)	2 (11%)	
Single LAD disease	27 (66%)	12 (67%)	NS
Double-vessel disease	10 (25%)	4 (22%)	
Infarct vessel			
TIMI grade	1.8 ± 1.3	1.6 ± 1.5	NS
Patency	21 (54%)	9 (50%)	NS
Collateral vessels	13 (33%)	7 (39%)	NS

Data presented are mean value ± SD or number (%) of patients. CAD = coronary artery disease; CK = creatine kinase; LAD = left anterior descending coronary artery; TIMI = Thrombolysis in Myocardial Infarction.

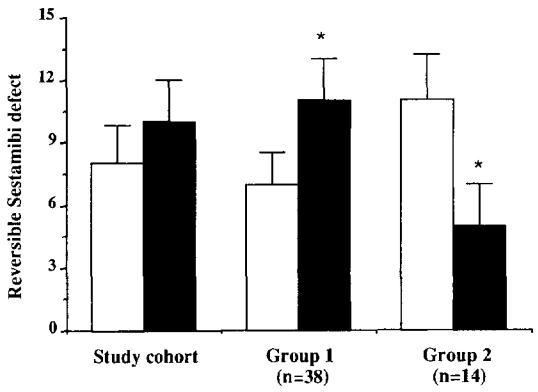

Figure 1. Extent of exercise-inducible "reversible" sestamibi defect (i.e., stress–rest percent of the total abnormal left ventricular surface) at 4- to 6-week imaging (study 1, **open bars**) and 6 months later (study 2, **solid bars**) in the general study cohort and in the two groups of patients. Data are reported as mean percent units ± SE. *p < 0.05 versus study 1.

(exercise–rest) perfusion defects were observed in study 1 in 13 (22%), 3 (5%) and 52 (90%) patients, respectively. In study 2, significant ST segment changes, chest pain and reversible tracer defects were observed in 15 (26%), 2 (3%) and 43 (74%) patients, respectively (p = NS vs. study 1). Overall, the extent of the reversible sestamibi defect was unchanged at study 2 (10 ± 9% vs. 8 ± 8% at study 1, p = NS) despite the significant reduction in the stress hypoperfusion extent (from 41 ± 11% to 36 ± 14% at study 2, p < 0.001).

Ergometric results and exercise scintigraphic data at studies 1 and 2 in groups 1 and 2 are summarized in Table 4. In group 1, a significantly greater extent of reversible tracer uptake defect was evident at study 2 (Fig. 1), although the actual stress hypoperfusion was smaller than in study 1. In 38 (95%) of 40 group 1 patients, the area showing reversible sestamibi defect at study 2 matched the area showing a fixed perfusion defect at study 1 (Fig. 2). Four of five patients who had fixed perfusion defect only at study 1 showed reversible sestamibi defects in myocardial areas previously classified as scarred.

In group 2, by contrast, the extent of the reversible sestamibi defect was significantly reduced at study 2 (p < 0.05, Fig. 1) as a result of a small and nonsignificant (p = 0.07) increase in the rest defect and without significant changes in the stress-induced hypoperfusion.

Discussion

The determination of the final infarct size and the assessment of residual ischemia have important implications for the risk stratification of patients with recent myocardial infarction. Because of the presence of both myocardial stunning and hyperdynamic ventricular function, predischarge left ventricular ejection fraction and wall motion analysis may be misleading. To assess both residual viability and ischemia in jeopardized areas, stress–rest quantitative myocardial perfusion imaging is frequently used. Our results indicate that at serial sestamibi imaging, an apparent increase in the ischemic area frequently occurs late after acute infarction in patients in stable condition because areas that appear to be scarred at 5 weeks appear viable and ischemic 6 months later.

Sestamibi estimate of infarct size. Sestamibi uptake and retention are sensitive and proportional to regional myocardial

Table 4. Ergometric Results at 4 to 6 Weeks (study 1) and at 7 Months (study 2) After Infarction

	Group 1 (n = 40)		Group 2 (n = 18)	
	Study 1	Study 2	Study 1	Study 2
Exercise duration (min)	11.7 ± 1.7	12.2 ± 1.6	11.5 ± 1.6	12.6 ± 2.3*
Rate–pressure product	24,855 ± 4,277	24,050 ± 4,488	24,673 ± 4,778	24,284 ± 5,517
Chest pain	3 (8%)	2 (5%)	0	0
ST segment depression >1 mm	10 (25%)	11 (28%)	3 (17%)	4 (22%)
Reversible sestamibi defects	35 (87%)	32 (84%)	17 (94%)	11 (78%)
Exercise sestamibi defect extent (%)	40 ± 11	35 ± 14†	42 ± 9	42 ± 12‡
Reversible sestamibi defect extent (exercise–rest defect) (%)	7 ± 7	11 ± 9*	10 ± 8	5 ± 6*

*p < 0.05, †p < 0.001 versus study 1. ‡p < 0.05 versus group 1 at corresponding time. Data presented are mean value ± SD or number (%) of patients.

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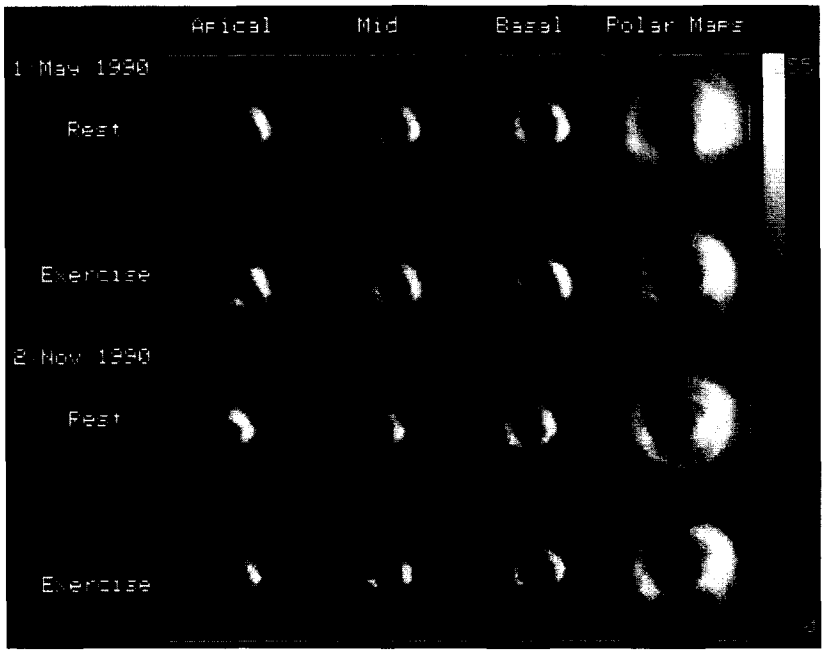

Figure 2. Rest and stress tomographic sestamibi images 5 weeks after acute infarction and 6 months later in a group 1 patient with a suboccluded mid–anterior descending coronary artery (Thrombolysis in Myocardial Infarction flow grade 0; no collateral vessels were seen at angiography). The rest tracer uptake defect, which involves the anteroseptal wall and the apex on 5-week images, is definitively reduced 6 months later. Although the 5-week stress images failed to show further hypoperfusion after exercise, an evident stress-induced perfusion defect is shown 6 months later in the area previously interpreted as "scarred."

blood flow and tissue viability (3–6). The chemical and physical properties of Tc-99m sestamibi make it an ideal agent for the assessment of myocardium at risk (5,7). In animal models of coronary occlusion and reperfusion, the tracer injection during occlusion yielded accurate estimates of the myocardium at risk as well as reliable measurements of the final infarct size when injected after reperfusion. In the setting of acute myocardial infarction in humans, serial changes in myocardial perfusion assessed by sestamibi imaging have been used as a measure of myocardial salvage in evaluating the efficacy of thrombolytic therapy (8–10), for the comparison of various reperfusion strategies (10,11) and for assessing the patency of the infarct-related artery (12). However, Pellikka et al. (24) reported a progressive reduction in the amount of hypoperfused myocardium from the tomographic study at 18 to 48 h to a later study at 6 to 14 days in patients with acute myocardial infarction who received thrombolytic therapy. We recently documented (13) a significant reduction in the rest sestamibi defect size in the infarcted area in a consistent percent of patients with stable coronary artery disease over a 6-month period after myocardial infarction. These findings of improved tracer distribution in the infarct area late after acute infarction suggest some delayed improvement of the blood flow supply to the infarcted area.

In the present study, half of the patients who showed improved sestamibi distribution in the infarcted area at study 2 had severe (>90%) infarct-related vessel stenosis. In the presence of such severe coronary obstruction, the preservation of rest blood flow in the jeopardized area has been related to a maintained coronary reserve (25). In patients with stable coronary artery disease, several clinical studies documented reduced levels of rest myocardial perfusion in the absence of either acute myocardial ischemia or necrosis (26–28). However, even in the absence of severe residual stenosis of the infarct-related vessel, an impaired coronary reserve could still affect blood flow to the salvaged myocardium and, consequently, sestamibi delivery. Brief periods of ischemia have been shown to cause prolonged impairment of coronary vasodilation (microvascular stunning) in addition to myocardial stunning (29,30).

Hibernating myocardium and residual ischemia. In our study, the reduction in the rest hypoperfused area was associated with an improvement in regional percent wall motion abnormality and left ventricular ejection fraction, suggesting that the observed reperfusion affected the dysfunctioning but salvaged myocardium that would improve thereafter (hibernating myocardium). In the setting of hibernating myocardium, underestimation of residual myocardial viability through the use of rest sestamibi imaging compared with rest or 24-h thallium-201 redistribution images (16,17) or metabolic fluorine-18 deoxyglucose imaging (14,15) has been reported. The assessment of residual viability may be flawed by the use of a tracer whose distribution is largely perfusion dependent and that redistributes minimally (4,31,32). Indeed, changes in blood flow supply can influence the resting tracer distribution. A decreased sestamibi uptake is often reversible after nitroglycerin administration (33) or coronary revascularization (34). However, different results have also been reported regarding the accuracy of sestamibi imaging to determine myocardial viability. Rest sestamibi distribution was found to compare favorably with delayed thallium-201 redistribution in an experimental model of sustained flow reduction, minimal necrosis and profound ischemic dysfunction (35). Furthermore, in a scintigraphic–pathologic study in patients undergoing cardiac transplantation, the size of the rest sestamibi defect correlated closely with the amount of histologic fibrosis (36).

In the present study, the late recovery of perfusion and contraction in the infarcted area was unrelated to the extent of underlying coronary disease, thrombolysis in the acute phase, TIMI grade of the infarct-related vessel or presence of collateral vessels on angiography. In interpreting our finding of some late "reperfusion" of the infarcted area, another explanation deserves mention. It has recently been documented (37,38) that "apparent" perfusion defects on ungated scintigraphic images can occur as a result of abnormal segmental contraction despite preserved regional myocardial blood flow. Even in the presence of preserved tracer distribution and retention, regional dissynergies can produce false tracer perfusion defects through the "partial volume effect." However, in the present study the explanation of a "false" perfusion defect from abnormal segmental contraction seems less likely. Different from group 2 patients who showed no delayed improvement in perfusion and contraction at study 2, group 1 patients showed better regional and ventricular function at study 1 despite the presence of a comparable perfusion defect size between the two groups of patients at study 1.

The salvaged myocardium, which is represented by the difference between the jeopardized territory and the residual damaged area, has been demonstrated (39,40) to be the main determinant of postinfarction ischemia. It is important to be aware that an imaging procedure that overestimates the size of the scar could lead to the underestimation of the extent of the still viable perinecrotic tissue at risk. In our serial evaluation, some underestimation of the extent of reversible hypoperfusion occurred at study 1 as a result of overestimating the scarred area. In 28 (70%) of 40 group 1 patients, the extent of the reversible tracer uptake defect was significantly greater 6 months later. However, the greater ischemic burden was mainly associated with the viable perinecrotic area, which showed "fixed" sestamibi uptake reduction (and was erroneously interpreted as scarred) at study 1. The speculation of a delayed improvement of the blood flow to the perinecrotic area in group 1 patients could also explain why their stress-induced hypoperfusion was significantly reduced at study 2. By contrast, rest and stress hypoperfusion defects remained substantially unchanged in group 2. In fact, we were unable to find significant differences in terms of TIMI grade of the infarct-related vessel flow and the presence of collateral circulation between the two groups. However, the limitations of coronary angiography in the assessment of collateral vessels are well known (41,42).

Limitations of the study. Only male patients with anterior Q wave myocardial infarction and rest ejection fraction values >25% were selected. Furthermore, patients with unstable coronary artery disease or with low-threshold ischemia were not considered, and patients with extensive coronary artery disease did not complete the serial evaluation. Thus, the observed results do not necessarily apply to all patients with recent myocardial infarction. Another limitation could stem from the fact that coronary angiography was carried out at an average of 3 months from the first scintigraphic evaluation. Although angiography was performed after a long interval from the acute phase and in patients in clinically stable condition, it is possible that the status of the infarct-related and collateral vessels at the time of the angiogram may have been different from that at the time of the study 1 evaluations, contributing to the lack of relation observed between sestamibi defect reduction and vessel patency or collateral vessels.

Conclusions. In patients with stable coronary artery disease evaluated by serial sestamibi imaging after myocardial infarction, a late reduction of the rest tracer defect may occur. Perinecrotic areas that appear to be scarred 5 weeks after acute infarction can appear viable 6 months later, causing an apparent increase in the stress-induced reversible perfusion defect. This should be taken into account when an even earlier stress sestamibi perfusion scintigraphy (i.e. at predischarge) is used to assess the residual ischemic burden.

We are grateful to Giacomina Romolo, RNT for technical advice; Fabio Comazzi for contributions to the data analysis; and Rosemary Allpress and Gillian Jarvis for their invaluable editorial assistance.

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