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Positron Emission Tomography Detects Evidence of Viability in Rest Technetium-99m Sestamibi Defects

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Objectives. **The purpose of this study was to determine the relative value of single-photon emission computed tomographic (SPECT) imaging at rest using technetium-99m methoxyisobutyl isonitrile (technetium-99m sestamibi) with positron emission tomography for detection of viable myocardium.**

Background. **Recent studies comparing positron emission tomography and thallium-201 reinjection with rest technetium-99m sestamibi imaging have suggested that the latter technique underestimates myocardial viability.**

Methods. **Twenty patients with a previous myocardial infarction underwent rest technetium-99m sestamibi imaging and positron emission tomography using fluorine (F)-18 deoxyglucose and nitrogen (N)-13 ammonia. In each patient, circumferential profile analysis was used to determine technetium-99m sestamibi, F-18 deoxyglucose and N-13 ammonia activity (expressed as percent of peak activity) in nine cardiac segments and in the perfusion defect defined by the area having technetium-99m sestamibi activity <60%. Technetium-99m sestamibi defects were graded as moderate (50% to 59% of peak activity) and severe (<50% of peak activity). Estimates of perfusion defect size were compared between technetium-99m sestamibi and N-13 ammonia.**

Results. **Sixteen (53%) of 30 segments with moderate defects and 16 (47%) of 34 segments with severe defects had ≥60% F-18 deoxyglucose activity considered indicative of viability. Fluorine-18 deoxyglucose evidence of viability was still present in 50% of segments with technetium-99m sestamibi activity <40%. There was no significant difference in the mean (± SD) technetium-99m sestamibi activity in segments with viable (40 ± 7%) and nonviable segments (49 ± 7%, p = 0.84). Of the 18 patients who had adequate F-18 deoxyglucose studies, the area of the technetium-99m sestamibi defect was viable in 5 (28%). In 16 patients (80%), perfusion defect size determined by technetium-99m sestamibi exceeded that measured by N-13 ammonia. The difference in defect size between technetium-99m sestamibi and N-13 ammonia was significantly greater in patients with viable (21 ± 9%) versus nonviable segments (7 ± 9%, p = 0.007).**

Conclusions. **Moderate and severe rest technetium-99m sestamibi defects frequently have metabolic evidence of viability. Technetium-99m sestamibi SPECT yields larger perfusion defects than does N-13 ammonia positron emission tomography when the same threshold values are used.**

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Positron emission tomography, using perfusion and metabolic imaging, is currently considered the most accurate noninvasive method for differentiating ischemic but viable myocardium from infarcted tissue. Its high cost and limited availability have led to studies comparing its role with that of conventional imaging techniques for assessing myocardial viability. Recent studies have shown that imaging with thallium-201 and reinjection after 3-h delayed imaging can improve the diagnostic accuracy of this agent for detection of viable myocardium (1,2). Technetium-99m methoxyisobutyl isonitrile (technetium-99m sestamibi) is a perfusion imaging agent that has been shown to have similar accuracy to thallium-201 for the detection of coronary artery disease (3,4). Less is known with regard to its value in the detection of viable myocardium (5,6). Reliable estimates of the extent of salvaged myocardium can be obtained using rest technetium-99m sestamibi imaging before and after thrombolytic therapy for acute myocardial infarction (7). However, in more chronic coronary artery disease states the accuracy of technetium-99m sestamibi for assessing the presence and extent of viable myocardium has not been defined. Several recent studies have reported that rest technetium-99m sestamibi imaging underestimates the number of left ventricular segments that are identified as viable by positron emission tomography or thallium-201 reinjection (8–10). However, this has not been a universal finding, and to date most of the studies have relied on qualitative image

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analysis (9–12). In this investigation, quantitative analysis techniques were applied to determine the value of rest technetium-99m sestamibi imaging for detection of viable myocardium using positron emission tomography as the reference standard.

Methods

Study patients. Twenty patients with a documented myocardial infarction were enrolled in this study. The mean age was 61 ± 8 years (range 44 to 71); 18 patients were men. Fifteen patients had electrocardiographic (ECG) Q waves. Six patients had previous coronary artery bypass grafting, and three others had previous coronary angioplasty. Coronary angiography was not performed as part of the investigative protocol; however, it was performed before recruitment in 12 patients and during the study period in 1 patient. Rest technetium-99m sestamibi imaging was performed 5 days to 3 weeks after infarction in 7 patients, and >6 months after infarction in the remaining 13. The mean interval between technetium-99m sestamibi imaging and positron emission tomography was 18 ± 15 days. No subject underwent revascularization or had a change in clinical status or medical therapy between the two imaging studies.

Rest technetium-99m sestamibi imaging. Seventeen patients underwent rest technetium-99m sestamibi imaging as part of a 1-day rest-stress protocol, and rest imaging alone was performed in three subjects. After an overnight fast, each patient was injected with 8 to 10 mCi of technetium-99m sestamibi. One hour after injection, imaging was performed using a rotating gamma camera (GE 400 AC) equipped with a high resolution collimator. The spatial resolution at 100 mm was 10.2 mm full-width half-maximum. The photopeak was set at 140 keV with a 20% window. Sixty-four images over a 180° arc were acquired in a 64 × 64-pixel matrix for 25 s each.

Positron emission tomographic imaging. Informed consent was obtained from each patient before positron emission tomographic imaging. The images were acquired using a whole-body tomograph (ECAT 931/Siemens) with an in-plane resolution of 8.5 mm. A 2-min scout transmission scan was used to ensure appropriate positioning of the detectors over the heart. A 15-min transmission scan was then acquired for subsequent attenuation correction of emission data. Each patient then received an intravenous injection of 20 mCi of N-13 ammonia. Three minutes after injection, static images were acquired for a 10-min period. After decay of the N-13 ammonia dose, 10 mCi of F-18 deoxyglucose was administered intravenously. Forty minutes later, static images were obtained for a 10- to 20-min period. In subjects without diabetes mellitus, F-18 deoxyglucose injection was conducted after an oral glucose load. Short-acting insulin was administered in five subjects with hyperglycemia according to a protocol established in our laboratory to enhance myocardial uptake of F-18 deoxyglucose.

The transaxial data were reconstructed in a 128 × 128-pixel matrix using a Hanning filter with a cutoff frequency of 0.3 cycles/pixel. The transaxial images were reoriented along the major axis of the heart and resliced to yield horizontal, vertical and short-axis images using a computer workstation (Sun Microsystems, Inc.).

Qualitative analysis. The location of the infarct territory was identified by clinical history and ECG information. Twelve patients had an inferior infarction; four had an anterior infarction; and four had both an inferior and an anterior infarction. In each patient, the severity of reduction in technetium-99m sestamibi uptake in the infarct territory was scored using a four-point grading system (4 = absent or severe reduction; 3 = moderate reduction; 2 = mild reduction; 1 = normal) by a nuclear medicine physician who was unaware of the clinical and quantitative image data. The positron emission tomographic studies were visually assessed. Moderate or severe reduction in both N-13 ammonia and F-18 deoxyglucose activity was defined as a matched pattern. Reduction in N-13 ammonia activity with normal or enhanced F-18 deoxyglucose activity was defined as flow-metabolism mismatch.

Quantitative analysis of segmental tracer activity. A semiautomated regional analysis program, developed at our institution, was applied to the short-axis rest technetium-99m sestamibi, N-13 ammonia and F-18 deoxyglucose images (13). For each image, 8 to 12 short-axis planes encompassing the heart from the apex to the mitral valve plane were used for analysis. The endocardial and epicardial edges of the myocardium for each plane were defined by ellipses chosen by an operator. The posterior point of intersection of the right and left ventricles was also defined, and a radius was automatically drawn between the center of the ellipse and this point. A circumferential profile was then generated for each plane using the radius as the starting point. The program searches for the maximal average activity in a 3 × 3-pixel area in each of 60 sectors for every plane. These data were then displayed in a polar coordinate map. This map was divided into five segments (septal, anterior, lateral, inferior and apical). Excluding the apex, the remaining segments were further subdivided into basal and distal planes, yielding a total of nine segments. The regional data were expressed as the mean percent of the peak myocardial activity in the polar map.

For the purposes of this study, regional F-18 deoxyglucose uptake ≥60% peak activity was defined as metabolic evidence for tissue viability. Regions with partial reduction in F-18 deoxyglucose uptake (≥50% of activity of normal reference regions) have preserved wall thickening and may represent a mixture of viable and infarcted myocardium (1,14). In this study, F-18 deoxyglucose uptake was 71 ± 11% in segments distant from the infarct territory that had normal perfusion at rest and no stress-induced defects. Thus, the threshold value chosen for viability was within 1 SD of normal.

The reduction in technetium-99m sestamibi activity was graded as moderate (50% to 59% of peak activity) and severe

(<50% of peak activity). Technetium-99m sestamibi and N-13 ammonia activity were compared in patients with viable (F-18 deoxyglucose ≥60%) and nonviable (F-18 deoxyglucose <60%) infarction zone segments.

Analysis of tracer activity in the perfusion defect. Analysis of tracer activity within an individual segment may overestimate tracer activity within an ischemically injured territory if the perfusion defect subscribes only a portion of any given segment. For this reason, tracer activity was determined within each perfusion defect. The area of the perfusion defect was defined on the technetium-99m sestamibi polar maps for each patient by choosing a threshold value of 60% of peak counts (7,15). The territory defined by values below this threshold was copied onto each patient's N-13 ammonia and F-18 deoxyglucose images to derive perfusion defect values for each of these tracers.

Estimation of perfusion defect size. Finally, perfusion defect size was compared between technetium-99m sestamibi and N-13 ammonia. A threshold value <50% of peak activity was chosen to define the most severely hypoperfused area of the ischemically injured territory for both technetium-99m sestamibi and N-13 ammonia. The area of the perfusion defect was expressed as a percent of the total area of the polar map.

Statistical analysis. The data are reported as mean value ± 1 SD. Comparisons of tracer activity were made with unpaired *t* tests. A statistically significant p value was defined as < 0.05.

Results

Coronary angiography. Twelve of the 13 patients who had coronary angiography before or during the study period had significant disease (>50% diameter stenosis) of at least one epicardial coronary artery or bypass graft. Four patients had three-vessel disease; six had two-vessel involvement; and two had one-vessel disease. The remaining patient had bypass grafts without significant obstruction. Nine (75%) of the 12 patients who had significant coronary artery disease had at least one totally occluded vessel.

Quantitative analysis. Quantitative analysis of technetium-99m sestamibi and N-13 ammonia activity was conducted in all patients. Two patients had F-18 deoxyglucose studies of limited quality that could not be analyzed. Both of these patients had hyperglycemia at the time of positron emission tomographic imaging, and administration of insulin failed to improve image quality. In the remaining 18 subjects, four segments were excluded from analysis because of displacement from the field of view or technical factors that prevented accurate quantitation. Five additional segments were excluded in two patients who had enhanced uptake of technetium-99m sestamibi activity in lateral segments that resulted in falsely reduced technetium-99m sestamibi activity in noninfarct segments. A total of 153 segments were included in the comparison of segmental technetium-99m sestamibi, N-13 ammonia and F-18 deoxyglucose activity.

Table 1. Technetium-99m Sestamibi Defect Severity and F-18 Deoxyglucose Activity in 153 Segments

Technetium-99m Sestamibi Activity (% of peak activity)	FDG ≥60%	FDG <60%
≥60	71	18
50–59	16	14
<50	16	18

FDG = F-18 deoxyglucose.

Segment analysis. The reduction in technetium-99m sestamibi activity within the infarction territory was *qualitatively* graded as severe in 15 patients and moderate in 5. Visual analysis revealed flow-metabolism mismatch in 9 of 18 subjects who had adequate F-18 deoxyglucose studies.

Using *quantitative* criteria, 19 patients had at least one segment with severe reduction in technetium-99m sestamibi activity, and the remaining patient had a moderate perfusion defect. There were 34 segments with technetium-99m sestamibi activity <50%. The technetium-99m sestamibi activity in these segments was compared with the values from segments in normal subjects with rest technetium-99m sestamibi scans to determine whether activity <50% of peak activity identified segments with a significant reduction in tracer uptake. Thirty-two (94%) of the 34 segments classified as having a severe defect had a reduction in tracer activity ≥2 SD below normal values.

The results of F-18 deoxyglucose imaging and the severity of reduction in technetium-99m sestamibi uptake are correlated in 153 segments in Table 1. Severely reduced technetium-99m sestamibi uptake did not exclude the presence of significant F-18 deoxyglucose uptake. Sixteen (47%) of 34 segments with severely reduced technetium-99m sestamibi activity had evidence of viability. These 16 segments were distributed among 10 (56%) of 18 patients who had adequate F-18 deoxyglucose studies. Five patients had one viable segment; four had two viable segments; and one had three viable segments. Flow-metabolism mismatch was observed in 5 of the 10 patients who had viable segments with technetium-99m sestamibi activity <50%. There were still equal numbers of viable (9) and nonviable (9) segments with technetium-99m sestamibi activity <40%. The lowest values of technetium-99m sestamibi activity in viable and nonviable segments were 29% and 26%, respectively. Thus, no lower limit of technetium-99m sestamibi activity was found that excluded significant F-18 deoxyglucose uptake.

The predictive value of technetium-99m sestamibi activity ≥60% of peak activity for segment viability was 80%. The predictive value of activity <60% for nonviable segments was 50%. The positive and negative predictive values using a threshold of 50% of peak activity were 73% and 53%, respectively.

The mean activity of F-18 deoxyglucose in viable segments (69 ± 7%) was within the 95% confidence limits of F-18 deoxyglucose uptake in noninfarct segments with normal perfusion (71 ± 11%). All segments exhibiting flow-

Table 2. Segment and Perfusion Defect Activity of Technetium-99m Sestamibi, N-13 Ammonia and F-18 Deoxyglucose

	Technetium-99m Sestamibi <50%		p Value
	FDG ≥60%	FDG <60%	
Segment			
Tc-99m sestamibi	40 ± 7%	49 ± 7%	0.843
N-13 ammonia	64 ± 9%	57 ± 8%	0.0318
FDG	69 ± 7%	51 ± 8%	0.0001
Perfusion defect			
Tc-99m sestamibi	46 ± 3%	40 ± 6%	0.0216
N-13 ammonia	60 ± 3%	55 ± 7%	0.16
FDG	67 ± 5%	50 ± 6%	0.0001

Values presented are mean value ± 1 SD. FDG = F-18 deoxyglucose; Tc = technetium.

metabolism mismatch had F-18 deoxyglucose activity ≥60%. The mean value of technetium-99m sestamibi in segments with a severe defect did not distinguish between those segments with and without viability (Table 2). However, the mean value of N-13 ammonia activity was significantly higher in viable compared with nonviable segments (Table 2).

Analysis of perfusion defect tracer activity. The perfusion defect had F-18 deoxyglucose evidence of viability in 5 (28%) of 18 patients (Fig. 1). The mean values of technetium-99m sestamibi, N-13 ammonia and F-18 deoxyglucose activity in the perfusion defect are shown in Table 2. In contrast to the segmental values of technetium-99m sestamibi activity, tracer uptake was significantly higher in defects with viable myocardium compared with those without viability.

Perfusion defect size. The estimates of defect size by the two blood flow tracers are shown in Table 3. In 16 patients the size determined by technetium-99m sestamibi exceeded that measured by N-13 ammonia (Fig. 1). The N-13 ammonia defects were marginally larger than the technetium-99m sestamibi defects in the remaining patients. Larger N-13 ammonia defects were confined to patients with nonviable segments and perfusion defects. The mean difference in perfusion defect size was compared between patients with viable and nonviable defects and between those with and without viable segments. The disparity in defect size was not significantly different in those with and without viability in the area of the perfusion defect (20 ± 9% vs. 13 ± 12%, respectively, p = 0.31). However, the difference in defect size was three times greater in patients with viable segments (21 ± 9%) versus those without viable segments (7 ± 9%, p = 0.0068).

Table 3. Perfusion Defect Size Determined by Tc-99m Sestamibi and N-13 Ammonia

Pt No.	Location of MI	Segment Viability	Perfusion Defect Viability	Defect Size (% LV)		
				Tc-99m Sestamibi	N-13 Ammonia	Tc-99m Sestamibi–N-13 Ammonia
1	Inf	Yes	Yes	22.5	1.7	20.8
2	Inf, Ant	Yes	Yes	36.0	26.9	9.1
3	Inf	Yes	Yes	28.3	4.2	24.1
4	Inf	Yes	Yes	14.4	1.9	12.5
5	Inf, Ant	Yes	Yes	72.6	41.7	30.9
6	Inf, Ant	Yes	No	26.1	18.8	7.3
7	Inf	Yes	No	26.7	6.8	19.9
8	Inf, Ant	Yes	No	54.0	21.5	32.5
9	Inf	Yes	No	37.4	13.8	23.6
10	Inf	Yes	No	32.6	3.5	29.1
11	Ant	No	No	17.1	9.6	7.5
12	Inf	No	No	37.6	20.6	17.0
13	Inf	No	No	23.6	4.0	19.6
14	Inf	No	No	18.8	9.0	9.8
15	Ant	U	U	35.0	31.8	4.2
16	Inf	No	No	23.3	8.7	14.6
17	Ant	No	No	7.6	12.3	−4.7
18	Ant	No	No	17.5	22.1	−4.6
19	Inf	No	No	20.4	20.6	−0.2
20	Inf	U	U	11.5	12.0	−0.5
Mean ± 1 SD				28.2 ±15*	14.6 ±11*	

*p = 0.0022. Ant = anterior; Inf = inferior; LV = left ventricle; MI = myocardial infarction; N = nitrogen; Pt = patient; Tc = technetium; U = unknown.

Discussion

Assessment of viability with technetium-99m sestamibi. In the evaluation of patients with coronary artery disease, the role of perfusion imaging has been expanded to include the assessment of myocardial viability. Single-photon emission computed tomographic (SPECT) thallium-201 scintigraphy,

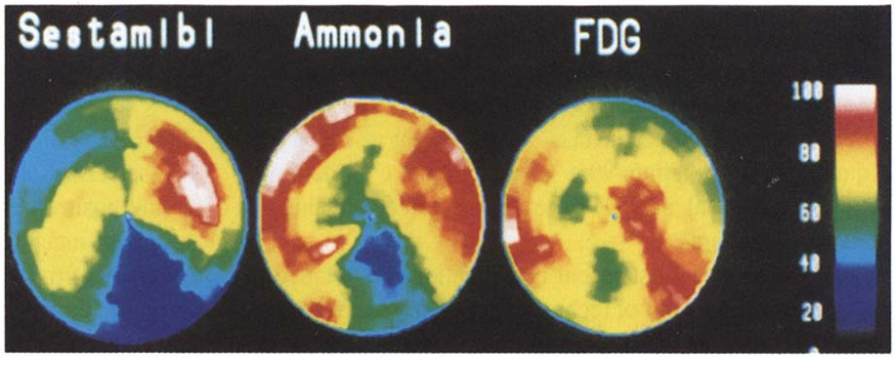

Figure 1. Technetium-99m sestamibi (Sestamibi), nitrogen (N)-13 ammonia (Ammonia) and F-18 deoxyglucose (FDG) polar maps in a 56-year old patient with a severe sestamibi defect (blue) with a mean activity of 43 ± 14% and F-18 deoxyglucose evidence of viability (activity 70 ± 11%). The infarction area was measured at 36% of the left ventricle by technetium-99m sestamibi and 27% by N-13 ammonia.

using imaging after reinjection, has demonstrated a sensitivity approaching that of positron emission tomography for detection of viable myocardium (1,2).

Technetium-99m sestamibi is a new perfusion tracer with properties that suggest its potential for use in assessing tissue viability, but this has yet to be established. Myocardial cell viability is essential for technetium-99m sestamibi uptake and retention (16,17). Intracellular accumulation and retention of technetium-99m sestamibi appear to be dependent on the negative charge gradient generated by the mitochondria (18). In the setting of acute myocardial infarction, technetium-99m sestamibi imaging before and after successful reperfusion can be used to document the presence and extent of myocardial salvage (7,19). However, technetium-99m sestamibi may be less useful for assessment of viability when flow remains severely compromised, as in patients with chronic coronary artery disease (6). In this situation, the close relation of flow and viability is uncoupled, so that delivery and accumulation of the tracer may underestimate viability.

Comparison of technetium-99m sestamibi and positron emission tomographic metabolic imaging. The results of this study demonstrate that technetium-99m sestamibi underestimates viability in this setting. The clinical significance of this finding depends in part on the frequency of its occurrence. In a study of 11 subjects, Soufer et al. (20) reported underestimation of viability in only two segments. In our investigation, metabolic evidence of viability was found in ~50% of segments with a moderate and severe reduction in technetium-99m sestamibi uptake. The results of our study are more in agreement with an investigation by Altehoefer et al. (10), who demonstrated that 52% of segments with 31% to 50% of peak technetium-99m sestamibi activity were viable. These investigators utilized qualitative analysis of F-18 deoxyglucose scans for determination of myocardial viability by identifying segments with matched and mismatched patterns of technetium-99m sestamibi and F-18 deoxyglucose activity. More recently, Vom Dahl et al. (21) reported that 37% of segments with 40% to 49% of peak technetium-99m sestamibi activity were viable when F-18 deoxyglucose uptake ≥70% of the reference region was used as the threshold for preserved viability.

Altehoefer et al. (10) and Vom Dahl et al. (21) found weak correlations between the degree of technetium-99m sestamibi activity and the presence of viability when segments with activity <30% were separately categorized. We found that ≥50% of peak activity had a modest predictive value for segment viability but that <50% of peak activity was poorly predictive of nonviable myocardium. The studies of Altehoefer et al. (10) and Vom Dahl et al. (21) also demonstrated that reductions in technetium-99m sestamibi uptake in the range 30% to 49% of peak activity have a poor predictive value for nonviable segments.

All of the three most recent investigations have failed to determine a lower limit of technetium-99m sestamibi activity that reliably excluded myocardial viability. Technetium-99m sestamibi overestimates blood flow when perfusion is severely reduced to levels resulting in myocardial injury (17,22). Technetium-99m sestamibi activity in acutely infarcted tissue may exceed 30% of the activity detected in nonischemic regions (17). Tissue viability may be preserved with reductions in flow to 30% of normal rest values (23). Thus, it may not be possible to exclude the presence of viable myocardium on the basis of the severity of reduction in technetium-99m sestamibi activity.

Technetium-99m sestamibi imaging 1 h after injection leads to underestimation of viability in a substantial proportion of patients. Our study and that of Dilsizian et al. (8) found that viability was underestimated in approximately 50% of those evaluated. However, in the majority of patients, viability was underestimated in only one or two segments. Failure to detect small areas of viable myocardium may have only a minor impact on the outcome of patients with coronary artery disease. Revascularization in patients who have limited amounts of viable myocardium does not result in improvement in global systolic function or exercise capacity, two of the most important prognostic indicators in coronary artery disease (24,25).

In our study, the analysis of F-18 deoxyglucose activity in the area of the perfusion defect provided additional information on the extent of underestimation of viability by technetium-99m sestamibi. The area encompassed by the perfusion defect demonstrated viability in a minority of patients, but in a few subjects (Table 3) the size of the defect occupied substantially more myocardium than one segment. Thus, in a few patients large areas of viable myocardium may be missed.

Comparison of perfusion defect size. Perfusion defect size was greater for technetium-99m sestamibi SPECT than for N-13 ammonia positron emission tomography when the *same* threshold value for defining defect area was used for both tracers. Previous investigators (26) have documented the accuracy of N-13 ammonia for estimation of infarction size. Overestimation of defect size was most pronounced in those patients who had viable tissue. None of those with comparatively larger N-13 ammonia defects had viable tissue. These findings are consistent with those of Baudhuin et al. (27), who demonstrated that the size and severity of the perfusion abnormality were worse when assessed by technetium-99m sestamibi. Our results indicate that the degree of mismatch between perfusion defect size estimated by the two blood flow tracers provides clues to the presence or absence of viable myocardium.

Study limitations. The ultimate goal of methods used for detection of myocardial viability is identification of myocardium that will have improvement in contractile function with revascularization. Our study and other investigations comparing positron emission tomographic and technetium-99m sestamibi imaging have provided no information as to the accuracy of this tracer for prediction of functional recovery in revascularized myocardium. The results of these studies and investigations demonstrating the high accuracy of

positron emission tomography for prediction of functional recovery suggest that rest technetium-99m sestamibi may be less useful for this purpose. This speculation is supported by a recent study indicating that the degree of technetium-99m sestamibi uptake is a poor indicator of functional recovery after revascularization (28).

In this and previous investigations, segments with decreased technetium-99m sestamibi activity were categorized on the basis of tracer concentrations that were normalized to the maximal values for each subject. This approach does not take into account regional heterogeneity in tracer distribution that may particularly affect SPECT images lacking attenuation correction. However, in our study nearly all defects classified as severe by this approach had technetium-99m sestamibi activity ≥2 SD below segmental values derived from normal subjects. Additionally, there is modest regional heterogeneity (13% to 19%) in myocardial glucose utilization in normal subjects evaluated after glucose loading (29,30). Glucose utilization and F-18 deoxyglucose concentrations are lowest in the septum. Selection of a single threshold value for viability could lead to underestimation of viability in septal segments. However, in our study all "nonviable" septal segments had a reduction in F-18 deoxyglucose activity that was >19% below the threshold value for viability. Additionally, all segments with flow-metabolism mismatch had F-18 deoxyglucose activity ≥60%.

The disparity in perfusion defect size may be partially attributed to the differences in positron emission tomographic and SPECT methods. Both the lack of attenuation correction and the higher resolution of positron emission tomography may have contributed to the observation of larger technetium-99m sestamibi defects. Additionally, differences in retention of the two perfusion tracers may necessitate use of different thresholds for each.

Finally, a technetium-99m sestamibi protocol utilizing imaging 1 h after tracer injection may not be optimal for detection of viable myocardium. Technetium-99m sestamibi is known to redistribute to a small degree, so that an additional delay in imaging may have reduced the frequency of viable myocardium in severe defects and the size of those defects (31).

Conclusions. The results of this and previous investigations indicate that SPECT imaging 1 h after injection of technetium-99m sestamibi at rest has limited accuracy for identification of myocardial viability on the basis of direct comparison with metabolic positron emission tomographic imaging. Thus, clinical decisions with regard to tissue viability should not rely on assessment of the extent or severity of technetium-99m sestamibi perfusion defects.

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