Metabolic Activity in the Areas of New Fill-in After Thallium-201 Reinjection: Comparison with Positron Emission Tomography Using Fluorine-18-Deoxyglucose

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Reinjection of thallium-201 after recording the 3-hr delayed scan often demonstrates improvement in areas of persistent abnormalities. To determine the metabolic activity of these areas, the changes seen on stress/redistribution/reinjection thallium SPECT were compared with PET using fluorine-18-fluorodeoxyglucose (FDG) in 18 patients with coronary artery disease. Of 48 segments showing no redistribution on the delayed scan, the reinjection scan identified new fill-in in 20 segments (42%), all of which demonstrated FDG uptake. In contrast, only 7 of the 28 segments (25%) showing no fill-in after reinjection were PET viable ($p < 0.01$). Eleven patients had coronary bypass graft surgery after the radionuclide study. The majority of the segments showing redistribution (87%) and new fill-in after reinjection (65%) improved in wall motion, whereas only eight segments (25%) without new fill-in improved after surgery. Of those without new fill-in, two segments showing PET ischemia improved in wall motion, whereas the remaining six segments showing PET scar did not improve after surgery. Thus, the segments showing new fill-in after reinjection are PET viable myocardium. However, reinjection thallium imaging still underestimates the extent of tissue viability compared to PET imaging.

J Nucl Med 1991; 32:673–678

Stress-thallium-201 (^{201})Tl myocardial imaging has been widely used for detection and characterization of ischemic heart disease (*1–5*). Although it is valuable to differentiate reversible ischemic myocardium from irreversible myocardial scar (*6–8*), it has limited value when the initially hypoperfused areas fail to show definite redistribution on the delayed scan (*9,10*). Persistent glucose utilization has been observed in some of these areas in a comparative study of positron emission tomography (PET) using fluorine-18-fluorodeoxyglucose (FDG) single-photon imaging with ^{201}Tl (*11–13*). To improve detection of reversible ischemia, 24-hr delayed imaging (*14–16*) or reinjection ^{201}Tl imaging (*17–20*) has been suggested. However, little is known about the metabolic activity of these segments. This study is undertaken to compare ^{201}Tl findings after reinjection with FDG-NH_3 PET findings.

Received Aug. 13, 1990; revision accepted Nov. 16, 1990.

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METHODS

Patient Populations

The reinjection ^{201}Tl study was performed in 75 consecutive patients who showed initial perfusion abnormalities on stress-^{201}Tl imaging. Of these, 18 patients were randomly selected for the PET study. Therefore, this study includes 18 patients who underwent both reinjection ^{201}Tl imaging and PET imaging. Their ages ranged from 50 to 65 yr with a mean value of 56.6. Fourteen patients had prior myocardial infarction, including 12 Q-wave and 2 non-Q-wave lesions. The interval from the onset of infarction ranged from 2 mo to 8 yr. Coronary angiograms showed single-vessel disease in eight, two-vessel disease in five, and three-vessel disease in five patients. Nine vessels showed 100% occlusion of the coronary artery. Eleven patients had coronary artery bypass surgery after the radionuclide study.

Thallium-201 Imaging

The procedure for performing stress redistribution and reinjection SPECT ^{201}Tl scans has been fully described elsewhere (*19–22*). Briefly, all patients underwent graded bicycle exercise starting at a 25-watt workload with 25 watt increments every 3 min. Approximately 100 MBq (2.7 mCi) of ^{201}Tl were injected at peak exercise and the exercise continued for another minute. Stress-^{201}Tl imaging began within 10 min after the tracer injection. The patients were asked to remain sedentary and refrain from eating any carbohydrate meals for the 3-hr interval between their initial and delayed thallium scans. Immediately after the delayed scan, 40 MBq (1.1 mCi) of ^{201}Tl were reinjected at rest. Ten minutes later, reinjection ^{201}Tl images were recorded.

Single-photon emission computed tomography (SPECT) was recorded using a General Electric 400AC/T model camera equipped with a low-energy general-purpose collimator, collecting 32 projection images for 30 sec each over 180° (*19–22*). A series

of transaxial slices were reconstructed with filtered backprojection without attenuation correction. Oblique tomograms parallel to the long- and short-axes of the left ventricle were also reconstructed.

Positron Emission Tomography

PET was performed with a whole-body, multi-slice positron camera (Positologica III, Hitachi Medical Co.) (*23*). Each patient was studied in a fasting condition for at least 5 hr to maintain steady-state during the study. The patients were positioned under the PET camera using ultrasound to locate important cardiac landmarks; a transmission scan was performed for accurate correction of photon attenuation, followed by administration of 80–300 MBq (2.2–8.1 mCi) of FDG. Approximately 60 min later, glucose images were recorded for 8–10 min. Immediately after the first scan, a second scan was carried out in a position 8 mm caudal to the first scan. These two scans provided a total of 14 contiguous transverse slices of the myocardium with an 8-mm interval (*24*).

A PET perfusion study was performed within 1 week of the FDG study. Approximately 400–600 MBq (10.8–16.2 mCi) of nitrogen-13-ammonia (^{13}N-) ammonia was injected at rest and the rest perfusion scan was started 3 min later. Two emission scans were obtained, each for 5–8 min.

From a series of transverse slices, oblique tomograms perpendicular to the long- and short-axis of the left ventricular myocardium were also reconstructed to compare the segments with ^{201}Tl imaging if necessary (*24*).

Image Analysis

The left ventricular myocardium was divided into nine segments to assess ^{201}Tl uptake of each SPECT study (Fig. 1). Two experienced physicians scored the uptake using a five-point grading system (0 = normal, 1 = equivocal, 2 = mild, 3 = moderate, and 4 = severe reductions) without knowledge of clinical, angiographic or PET data.

Initial perfusion was considered normal when the postexercise score was 0 or 1; when a myocardial segment showed a postexercise score of 2 or more, the initial perfusion was abnormal. When the score decreased one or more on the delayed scan, the segment was considered to be redistributed. When the score was unchanged on the delayed scan but decreased on the reinjection scan, the segment was considered to be new fill-in after reinjection. When the score did not decrease even on the reinjection scan, the segment was considered to be a persistent defect (*19*, *20*).

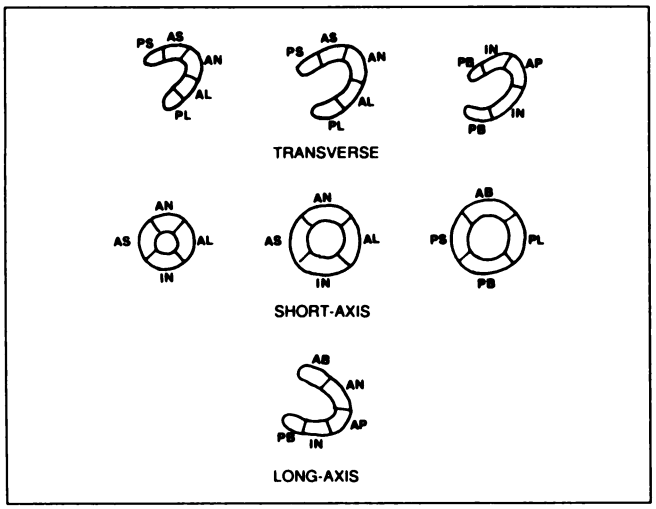

FIGURE 1. Schematic presentation of nine myocardial segments in transaxial, short-axis, and vertical long-axis slices. (AB = anterobasal; AN = anterior; AP = apical; IN = inferior; PB = posterobasal; AS = anteroseptal; PS = posteroseptal; AL = anterolateral; and PL = posterolateral segment)

The ^{13}N-ammonia perfusion and FDG glucose metabolic images were compared in the corresponding segments. The segments with perfusion above the lower limit of the normal values were considered to be normal. The lower limit was determined as the mean minus 2 s.d. of the perfusion profiles at each segment from 12 normal subjects who had <5% likelihood of coronary artery disease (*25*,*26*). The segments with perfusion below the lower limits were defined as hypoperfused segments. Those hypoperfused segments were divided into two groups based on FDG uptake. FDG uptake was quantitatively measured as %ID/100g tissue in each segment. The hypoperfused segments with an increase in FDG uptake above the normal range were defined as PET ischemia, while those with no increase in FDG uptake were defined as PET scar (*27–29*). The normal range of FDG uptake was defined as FDG uptake of mean ± 2 s.d. of eight normal subjects. The upper limit was approximately 0.7%ID/100 g in the septal, anterior, and apical regions and 0.85%/ID/100 g in the lateral and inferior regions (*30*).

Wall Motion Analysis

Eleven patients had coronary bypass grafting after the ^{201}Tl scan. Each patient underwent radionuclide ventriculography in the anterior and left anterior oblique projections after intravenous injection of 740 MBq (20mCi) of technetium-99m-red blood cells before and after the interventions. The left ventricle was divided into anterior, apical, inferior, septal, and lateral segments. The left ventricle was divided into anterior, apical, inferior, septal, and lateral segments. The left lateral view acquisition was also added in the study for assessment of inferior wall motion if necessary. Wall motion was visually assessed by two experienced observers using a five-point grading system (normal, mild hypokinesis, severe hypokineses, akinesis, and dyskinesis) (*19*). When the wall motion score improved by 1 or greater after intervention, the segments were considered to be improved wall motion (*19*).

Statistical Analysis

Comparisons of proportions were performed by way of chi-square analysis or Fisher's exact test. Probability values <0.05 were considered significant.

RESULTS

All of the 18 patients showed perfusion abnormalities on the initial ^{201}Tl scan. Twelve patients showed redistribution in at least one myocardial segment, while the remaining six patients did not have any segment showing redistribution. The reinjection ^{201}Tl scan demonstrated new fill-in in two out of six patients (33%). The FDG-PET study showed an increase in FDG uptake in these patients. On the other hand, the reinjection scan did not show new fill-in in the remaining four patients. The PET study showed FDG uptake in only one of these patients.

Of the total 162 myocardial segments, 95 segments showed perfusion abnormalities on the initial ^{201}Tl scan

and forty-seven showed redistribution on the delayed scan. The reinjection ^{201}Tl scan showed new fill-in in 20 of the 48 segments (42%) without redistribution (Table 1). Thus, new fill-in was demonstrated after reinjection in approximately one-half of the persistent defects on the delayed scan.

The FDG-NH3 PET scan was normal in 80 segments and showed PET ischemia in 61 segments and PET scar in 21 segments (Table 1). Segments showing redistribution on the delayed scan were either PET normal (16 segments) or PET ischemia (31 segments), indicating PET viable myocardium. In the segments showing new fill-in after reinjection, one segment was PET normal and the remaining 19 segments were PET ischemia (Fig. 2). Thus, PET ischemia was more often observed in the segments showing new fill-in (95%) than those having redistribution (66%) ($p < 0.05$), but these segments were all PET viable myocardium. On the other hand, 21 of the 28 segments (75%) showing no fill-in after reinjection were PET scar ($p < 0.01$). However, the remaining seven segments showing no fill-in even after reinjection had persistent metabolic activity on FDG-PET (Fig. 3).

Table 2 shows the relationship of ^{201}Tl and FDG-PET findings with postoperative improvement in regional wall motion. Of 31 segments showing redistribution, 27 (87%) improved in wall motion after surgery. Similarly, 11 of 17 segments (65%) showing new fill-in after reinjection also improved in wall motion (ns). On the other hand, only two of eight segments (25%) showing no fill-in after reinjection improved in wall motion ($p < 0.01$ versus redistribution). The two segments showing PET ischemia improved in wall motion (Fig. 4), whereas the remaining six segments showing PET scar did not improve after surgery.

DISCUSSION

These data indicate that the reinjection ^{201}Tl scan is helpful for identifying ischemic myocardium, which is often missed on routine stress and delayed scans. Particularly, the persistent defects without redistribution, which showed new fill-in after reinjection, may be considered as PET viable myocardium. However, this elegant technique may still occasionally underestimate the extent of ischemic myocardium.

TABLE 1
Number of Segments on ^{201}Tl Imaging in Relation with PET Findings

Thallium findings	PET normal	PET ischemia	PET scar	Total
Normal	63	4	0	67
Redistribution	16	31	0	47
New fill-in	1	19	0	20
Persistent defect	0	7	21	28
Total	80	61	21	162

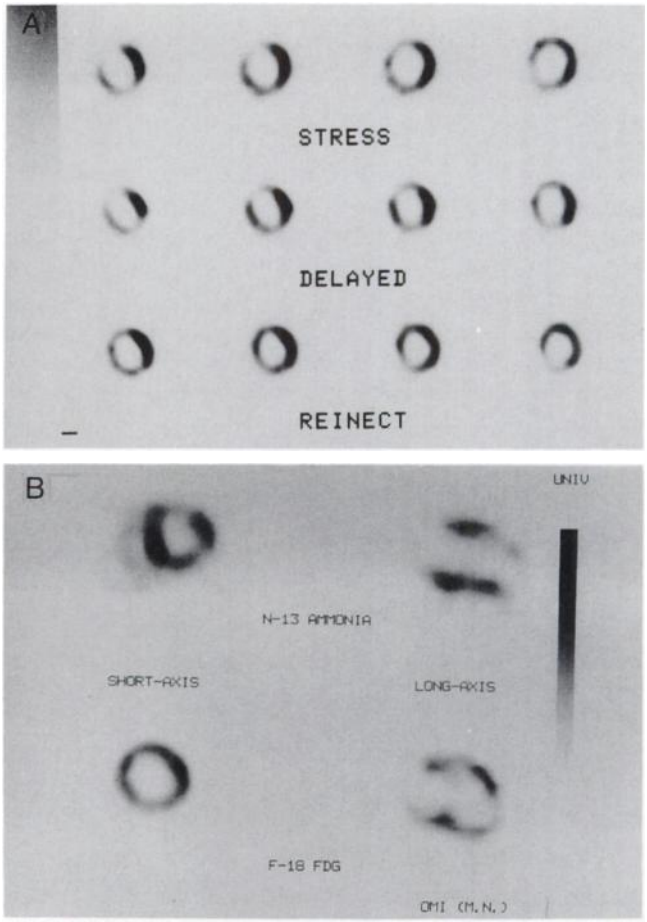

FIGURE 2. (A) A series of short-axis slices of stress (top), 3-hr delayed (middle), and reinjection (bottom) ^{201}Tl SPECT images of a patient with anterior wall myocardial infarction. Hypoperfusion in anterior, septal, and inferior regions is noted without definite redistribution on the delayed images, except slight redistribution in septal region. The reinjection images, however, show new fill-in in anterior and inferior regions. (B) Representative short-axis (left) and long-axis (right) slices of ^{13}N-ammonia perfusion (top) and FDG (bottom) images. Note hypoperfusion with increased uptake of FDG in anterior and inferior regions.

Value of ^{201}Tl Reinjection

Although the redistribution analysis on stress-^{201}Tl scans is valuable for assessing tissue viability in patients with coronary artery disease, the persistent defects without redistribution often contain ischemic myocardium that is likely to improve in regional function after restoration of blood flow (*8–10*).

Rocco and Dilsizian (*17,18*) proposed that reinjection of ^{201}Tl after delayed scanning may enhance detection of new fill-in in the areas of persistent defect on the delayed scan. We also have demonstrated that reinjection identified new fill-in in approximately 40% of the segments without redistribution on the delayed scan (*19,20*); findings similar to the current study. The segments showing new fill-in after reinjection are expected to improve regional function after revascularization while those without fill-in are less likely to do so (*18,19*). Reinjection ^{201}Tl

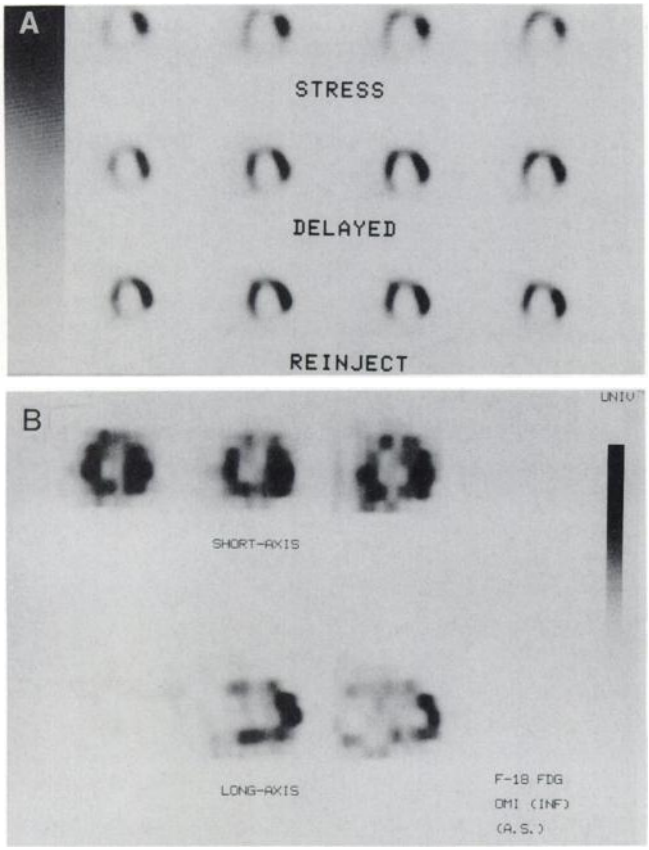

FIGURE 3. (A) A series of short-axis slices of stress (top), delayed (middle), and reinjection (bottom) ^{201}Tl SPECT images of a patient with inferior wall myocardial infarction. Note initial perfusion defects in anterior, septal and inferior regions with redistribution in septal and anterior regions. However, persistent defect is observed in inferior wall even after reinjection. (B) A series of short-axis and long-axis slices of FDG images show diffuse persistent uptake of FDG, including in inferior wall.

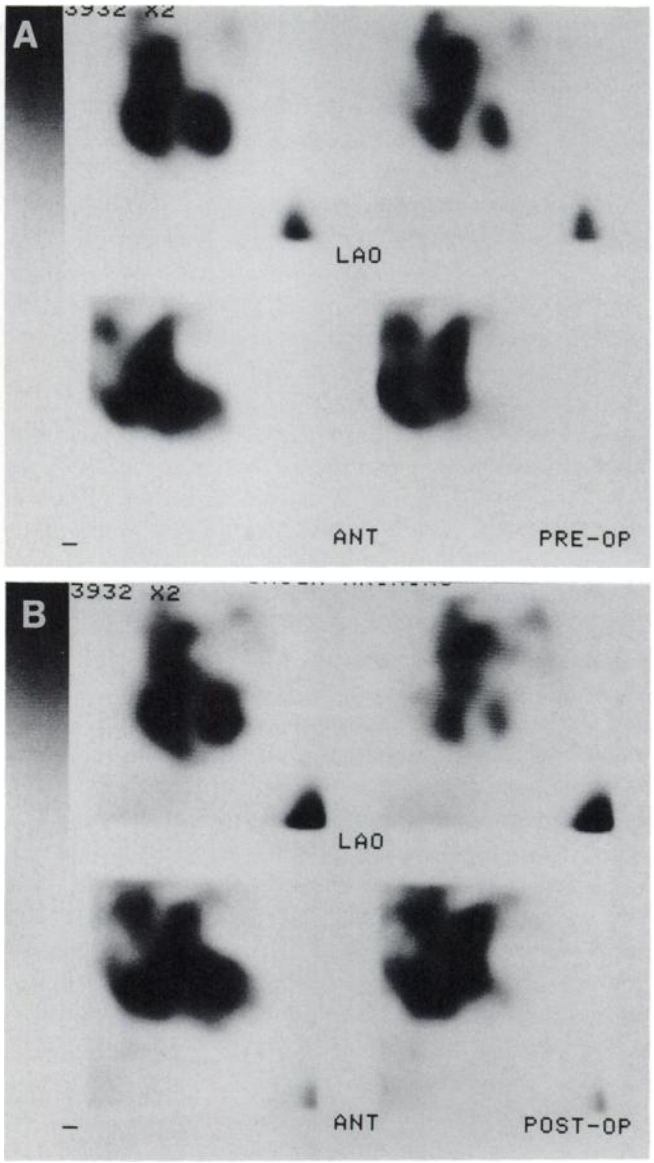

FIGURE 4. Preoperative (A) and postoperative (B) radionuclide ventriculography of the same patient as in Figure 3. Note wall motion abnormalities in apical, anterior, and inferior regions preoperatively. Postoperative study shows significant improvement in wall motion in these regions.

studies provide high quality images with acceptable counts that may be helpful for identifying even a slight change in ^{201}Tl distribution. In addition, the whole study can be completed within 4–5 hr, a particularly useful application for out-patients, as compared with 24-hr delayed imaging.

TABLE 2
Number of Segments on ^{201}Tl Imaging in Relation to Improvement in Wall Motion After Coronary Bypass Grafting

Thallium findings	n	Wall motion after surgery	
		Improved	Not improved
Redistribution	31	27	4
New fill-in	17	11	6
Persistent defect	8	2*	6†
Total	56	40	16

* PET ischemia.
† PET scar.

Mechanisms of New Fill-in After Reinjection

Two separate injections of perfusion tracers at rest and during exercise showed reversible ischemia more often than the stress and delayed imaging with single-tracer injection (*13,31–34*). Since reinjection ^{201}Tl images represent partly delayed distribution and resting perfusion, the reinjection images may resolve some perfusion abnormalities better than the delayed images.

The ischemic segments that fail to show redistribution are mainly supplied with severely stenotic vessels. Our previous study (*20*) also indicated that the new fill-in after reinjection was observed more often in the segments supplied by vessels with severe stenosis with very impaired wall motion.

In cases with severe coronary stenosis, the tracer delivery to the ischemic tissue may be severely prolonged due to loss of post-stress hyperemia or resting hypoperfusion. Therefore, the routinely performed 3–4-hr delayed scan may not be long enough to reach equilibrium of the tracer in the potassium pool, and thus, it may not show redistribution in the severely ischemic myocardium. Actually, the inverse relation of the rate of ^{201}Tl redistribution was observed with the severity of coronary stenosis (*14*). In this respect, the reinjection of ^{201}Tl or the 24-hr delayed scan (*15,16*) may help reach this equilibrium state and thus identify new fill-in of tracer in the severely ischemic myocardium (*20*).

Redistribution depends on plasma concentration of the tracer as well. Low plasma ^{201}Tl concentration after injection at exercise often lacks redistribution in the ischemic myocardium (*35,36*). Thus, reinjection of a small amount of ^{201}Tl after delayed scanning is considered to be reasonable for enhanced detection of new fill-in in the areas of ischemic myocardium by an increase in plasma ^{201}Tl concentration.

Comparison with Metabolic Activity

The experimental and clinical studies showed that preservation of FDG uptake correlated well with the presence of viable myocardium (*28,29,37,38*). In his canine model, Sochor et al. (*37*) demonstrated the preservation of metabolic activity in association with histologic presence of a significant amount of residual viable myocardium. Tillisch et al. (*28*) and our previous report (*29*) both indicated that segments with metabolic activity are likely to improve regional function after coronary bypass surgery, and thus, are mainly reversible ischemic myocardium. Therefore, we consider it important to compare reinjection ^{201}Tl findings with FDG-PET findings to see whether the segments showing new fill-in after reinjection really represent preserved metabolic activity.

In this study, the segments showing new fill-in after reinjection were all metabolically active as assessed by FDG-PET. In addition, the majority of the segments showing new fill-in after reinjection had improved wall motion after surgery (*19*). Therefore, segments with new fill-in may be considered as reversible ischemic myocardium. In his preliminary data, Bonow et al. also indicated close correlation of reinjection ^{201}Tl findings with FDG accumulation (*39*).

There were some segments that showed no fill-in after reinjection but did have persistent metabolic activity on FDG-PET. Our comparative study of reinjection ^{201}Tl findings with improvement in regional function after coronary bypass grafting showed that segments showing new fill-in are all reversible ischemic myocardium and that some segments without fill-in after reinjection also showed improvement as well (*19*). These segments showed an increase in FDG uptake. Thus, the reinjection method seems to be useful for assessing tissue viability but still has the potential for underestimation of the extent of tissue viability.

In the areas with severe ischemia where coronary flow was severely reduced, the perfusion tracer might not go to the ischemic tissue. As a result, it might be difficult to detect change in tracer distribution. On the other hand, FDG, as a tracer of exogenous glucose utilization, accumulates in excess of blood flow (*37,38*). In this sense, a metabolic marker rather than the flow tracer may be a better marker for assessing tissue viability. Further work in a larger patient population is needed to support this concept.

Limitations

FDG myocardial uptake is known to be altered with changes in substrate levels. In the postprandial condition, increased utilization of glucose by normal myocardium results in an increase in FDG uptake in the infarcted tissue with relative decrease in its uptake in ischemic myocardium. As a result, the scan may underestimate the extent of tissue viability (*40*). In contrast, in the fasting condition, as tested in the current study the myocardium preferential utilizes fatty acids. As a result, even small areas of ischemic tissue may show intense uptake of FDG compared to the normal myocardium. Thus, an FDG study under fasting conditions may overestimate tissue viability. In addition Gropler et al. suggested that fasting causes heterogeneous uptake of FDG in normal myocardium (*41*). To overcome these limitations, we quantitatively measured FDG uptake to identify the segments with uptake above the normal range in each segment considered to be PET ischemia.

Clinical Implications

The reinjection of ^{201}Tl after delayed imaging may enhance detection of new fill-in in areas of no redistribution on routine ^{201}Tl scans. Since all of these segments had metabolic activity on FDG-PET, they were considered to be ischemic but viable myocardium. However, the segments without new fill-in even after reinjection occasionally showed PET ischemia, indicating that this technique may still underestimate presence of PET ischemia. We conclude that reinjection ^{201}Tl scans should be performed when the routine stress and delayed scans showed persistent lesions without redistribution.

ACKNOWLEDGMENTS

We greatly acknowledge the valuable comments of Makoto Shindo, Norio Takahashi, Takehiko Yamada, and Shinji Ono. We also thank Emiko Komori, Toru Fujita, and the Cyclotron staff for technical assistance.

REFERENCES

1. Bailey IK, Griffith LSC, Rouleau J, Strauss HW, Pitt B. Thallium-201 myocardial perfusion imaging at rest and during exercise: comparative sensitivity to electrocardiography in coronary artery disease. *Circulation* 1977;55:79–87.

2. Ritchie LJ, Trobaugh GB, Hamilton GW, et al. Myocardial imaging with thallium-201 at rest and during exercise: comparison with coronary angiography and resting and stress electrocardiography. *Circulation* 1977;56:66–71.
3. Rigo P, Bailey IK, Griffith LSC, et al. Value and limitations of segmental analysis of stress thallium myocardial imaging for localization of coronary artery disease. *Circulation* 1980;61:973–981.
4. Maddahi J, Garcia EV, Hamilton GW, et al. Improved noninvasive assessment of coronary artery disease by quantitative analysis of regional stress myocardial distribution and washout of thallium-201. *Circulation* 1981;64:924–935.
5. Verani MS, Jhingran S, Attar M, Rizk A, Quinones MA, Miller RR. Post-stress redistribution of thallium-201 in patients with coronary artery disease, with and without prior myocardial infarction. *Am J Cardiol* 1979;43:1114–1122.
6. Pohost GM, Zir LM, Moore RH, McKusick KA, Guiney TE, Beller GA. Differentiation of transiently ischemic from infarcted myocardium by serial imaging after single dose of thallium-201. *Circulation* 1977;55:294–302.
7. Rozanski A, Berman DS, Gray R, et al. Use of thallium-201 redistribution scintigraphy in the preoperative differentiation of reversible and nonreversible myocardial asynergy. *Circulation* 1986;64:936–944.
8. Berger BC, Watson DD, Burwell LR, et al. Redistribution of thallium at rest in patients with stable and unstable angina and the effect of coronary artery bypass surgery. *Circulation* 1979;60:1114–1125.
9. Gibson RS, Watson DD, Taylor GJ, et al. Prospective assessment of regional myocardial perfusion before and after coronary revascularization surgery by quantitative thallium-201 scintigraphy. *J Am Coll Cardiol* 1983;1:804–815.
10. Liu P, Kiess MC, Okada RD, et al. The persistent defect on exercise thallium imaging and its fate after myocardial revascularization: does it represent scar or ischemia? *Am Heart J* 1985;110:996–1001.
11. Brunken R, Schwaiger M, Grover-McKay M, Phelps ME, Tillisch J, Schelbert HR. Positron emission tomography defects tissue metabolic activity in myocradial segments with persistent thallium perfusion defects. *J Am Coll Cardiol* 1987;10:557–567.
12. Tamaki N, Yonekura Y, Yamashita K, et al. Relation of left ventricular perfusion and wall motion with metabolic activity in persistent defects on thallium-201 tomography in healed myocardial infarction. *Am J Cardiol* 1988;62:202–208.
13. Tamaki N, Yonekura Y, Yamashita K, et al. SPECT thallium-201 tomography and positron tomography using N-13-ammonia and F-18-fluorodeoxyglucose in coronary heart disease. *Am J Cardiac Imaging* 1989;3:3–9.
14. Gutman J, Berman DS, Freeman M, et al. Time to complete redistribution of thallium-201 in exercise myocardial scintigraphy: relationship to the degree of coronary artery disease. *Am Heart J* 1983;106:989–995.
15. Cloninger KG, DePuey EG, Garcia EV, et al. Incomplete redistribution in delayed thallium-201 single-photon emission tomographic (SPECT) images: an overestimation of myocardial scarring. *J Am Coll Cardiol* 1988;12:955–963.
16. Kiat H, Berman DS, Maddahi J, et al. Late reversibility of tomographic myocardial thallium-201 defects: an accurate marker of myocardial viability. *J Am Coll Cardiol* 1988;12:1456–1463.
17. Rocco T, Dilsizian V, Maltais F, Strauss HW, Boucher CA, McKusick KA. Thallium-201 reinjection after delayed imaging demonstrates fill-in to regions with "fixed" defects [Abstract]. *J Nucl Med* 1988;29:769.
18. Dilsizian V, Rocco TP, Freedman NMT, Leon MB, Bonow RO. Enhanced detection of ischemic but viable myocardium by the reinjection of thallium after stress-redistribution imaging. *N Engl J Med* 1990;323:141–146.
19. Ohtani H, Tamaki N, Yonekura Y, et al. Value of thallium-201 reinjection after delayed SPECT imaging for predicting reversible ischemia after coronary artery bypass grafting. *Am J Cardiol* 1990;66:394–399.
20. Tamaki N, Ohtani H, Yonekura Y, et al. Significance of fill-in after thallium-201 reinjection following delayed imaging: comparison with regional wall motion and angiographic findings. *J Nucl Med* 1990;31:1617–1622.
21. Tamaki N, Yonekura Y, Mukai T, et al. Segmental analysis of stress thallium myocardial emission tomography for localization of coronary artery disease. *Eur J Nucl Med* 1984;9:99–105.
22. Tamaki N, Yonekura Y, Mukai T, et al. Stress thallium-201 transaxial emission computed tomography: quantitative versus qualitative analysis of coronary artery disease. *J Am Coll Cardiol* 1984;4:1213–1221.
23. Senda M, Tamaki N, Yonekura Y, et al. Performance characteristics of Positologica III, a whole-body positron emission tomography. *J Comput Assist Tomogr* 1985;9:940–946.
24. Senda M, Yonekura Y, Tamaki N, et al. Interpolating scan and oblique-angle tomograms in myocardial PET using nitrogen-13-ammonia. *J Nucl Med* 1986;27:1180–1836.
25. Yonekura Y, Tamaki N, Senda M, et al. Detection of coronary artery disease with ^{13}N-ammonia and high-resolution positron emission computed tomography. *Am Heart J* 1987;113:645–654.
26. Tamaki N, Yonekura Y, Yamashita K, et al. Value of rest-stress myocardial positron tomography using N-13-ammonia for the preoperative prediction of reversible asynergy. *J Nucl Med* 1989;30:1302–1310.
27. Marshall RC, Tillisch JH, Phelps ME, et al. Identification and differentiation of resting myocardial ischemia and infarction in man with positron computed tomography, ^{18}F-labeled fluorodeoxyglucose and N-13-ammonia. *Circulation* 1983;67:766–778.
28. Tillisch J, Brunken R, Marshall R, et al. Reversibility of cardiac wall-motion abnormalities predicted by positron tomography. *N Engl J Med* 1986;314:884–888.
29. Tamaki N, Yonekura Y, Yamashita K, et al. Positron emission tomography using fluorine-18-deoxyglucose in evaluation of coronary artery bypass grafting. *Am J Cardiol* 1989;64:860–865.
30. Tamaki N, Yonekura Y, Ohtani H, et al. Change in glucose utilization after coronary bypass surgery: assessment with positron tomography using F-18-deoxyglucose [Abstract]. *Circulation* 1989;80:II-377.
31. Tamaki N, Yonekura Y, Senda M, et al. Value and limitation of stress thallium-201 tomography: comparison with N-13-ammonia perfusion positron tomography. *J Nucl Med* 1988;29:1181–1188.
32. Kahn JK, McGhie I, Akers MS, et al. Quantitative rotational tomography with ^{201}Tl and ^{99m}Tc-2-methoxy-isobutyl-isonitrile. *Circulation* 1989;79:1282–1293.
33. Blood DK, McCarthy DM, Scicca RR, Cannon PJ. Comparison of single-dose and double-dose thallium-201 myocardial perfusion scintigraphy for the detection of coronary artery disease and prior myocardial infarction. *Circulation* 1981;58:777–788.
34. Ritchie JL, Albro PC, Caldwell JH, Trobaugh GB, Hamilton GW. Thallium-201 myocardial imaging: a comparison of the redistribution and rest images. *J Nucl Med* 1979;20:477–483.
35. Budinger TF, Knittel BL. Cardiac thallium redistribution and model [Abstract]. *J Nucl Med* 1987;28:588.
36. Nelson CW, Willson RA, Angello DA, Palac RT. Effect of thallium-201 blood levels on reversible myocardial defects. *J Nucl Med* 1989;30:1172–1175.
37. Sochor H, Schwaiger M, Schelbert HR, et al. Relationship between Tl-201, Tc-99m(Sn)pyorphosphate and F-18-2-deoxyglucose uptake in ischemically injured dog myocardium. *Am Heart J* 1987;114:1066–1077.
38. Schwaiger M, Schelbert HR, Ellison D, et al. Sustained regional abnormalities in cardiac metabolism after transient ischemia in the chronic dog model. *J Am Coll Cardiol* 1985;6:336–347.
39. Bonow RO, Dilszian V, Cuocolo A, Bacharach SL. Identification of viable myocardium in patients with chronic coronary disease and left ventricular dysfunction: comparison of thallium scintigraphy with reinjection and PET imaging with ^{18}F-fluorodeoxyglucose. *Circulation* 1991;83:26-37.
40. Brunken R, Tillisch J, Schwaiger M, et al. Regional perfusion, glucose metabolism, and wall motion in patients with chronic electrocardiographic Q-wave infarctions: evidence for persistence of viable tissue in some infarct regions by positron emission tomography. *Circulation* 1986;73:951–963.
41. Gropler RJ, Siegel BA, Lee KJ, et al. Nonuniformity in myocardial accumulation of fluorine-18-fluorodeoxyglucose in normal fasted humans. *J Nucl Med* 1990;31:1749-1756