

Fluorine-18 deoxyglucose PET for assessment of viable myocardium in perfusion defects in ^{99m}Tc -MIBI SPET: a comparative study in patients with coronary artery disease*

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Abstract. Extent and frequency of viable tissue in myocardial segments yielding a perfusion defect on technetium-99m methoxyisobutylisocyanide (^{99m}Tc -MIBI), single photon emission tomography (SPET) at rest was prospectively investigated with 2- ^{18}F -2-deoxyglucose (^{18}F FDG) positron emission tomography (PET) in 46 patients with chronic coronary artery disease (CAD). Of these, 43 had a history of old myocardial infarction. For comparative visual and quantitative evaluation of identical anatomical slices, PET image files were converted into the SPET file structure and into the same matrix size. SPET and PET images were documented and visually (9 segments/patient) or semiquantitatively evaluated by a target-like polar map. Relative perfusion was expressed in percentage of peak ^{99m}Tc -MIBI uptake. Sample ^{18}F FDG uptake was related to the ^{18}F FDG uptake in the area of such maximal perfusion (^{18}F FDG uptake was 100% at the 100% ^{99m}Tc -MIBI uptake area). Of 414 segments, 167 (40%) revealed a resting perfusion defect. ^{18}F FDG uptake was present in 38 (23%) of the defects, while another 40 (24%) segments yielded ^{18}F FDG uptake in the periphery of the defect. When grouped according to the degree of ^{99m}Tc -MIBI uptake-reduction (in percentage of peak activity), 80% of severe defects ($\leq 30\%$ of peak uptake), 48% of moderate (31%–50% of peak uptake) and 31% of mild ($> 50\%$ of peak uptake) defects were considered as non-viable on the basis of ^{18}F FDG uptake. Complete viability was found in none of the severe defects in contrast to 29% of moderate and 35% of mild perfusion defects. From these data we conclude that ^{99m}Tc -MIBI uptake as a

myocardial perfusion marker underestimates myocardial viability in patients with chronic CAD and after myocardial infarction. Nevertheless, only moderate reductions of ^{99m}Tc -MIBI uptake seem to imply a greater likelihood for viability. Comparative analysis of metabolism and flow is possible with different tomographic systems and is valuable for clinical evaluation of the cardiac patient.

Key words: Technetium-99m methoxyisobutylisocyanide single photon emission tomography (^{99m}Tc -MIBI SPET) – Fluorine-18 deoxyglucose positron emission tomography (^{18}F FDG PET) – Myocardial viability – Myocardial infarction – Coronary artery disease

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Introduction

The differentiation of myocardial scar from ischaemic or hibernating myocardium as a cause of left ventricular dysfunction has important clinical implications for patient management. Patients with ischaemic but viable myocardium often benefit from surgical revascularization (Gibson et al. 1983; Brundage et al. 1984; Braunwald and Rutherford 1986; Tillisch et al. 1986).

Measurement of fluorine-18 deoxyglucose (^{18}F FDG) uptake in the myocardium is now an accepted method to indicate myocardial viability in ischaemic heart disease (Schwaiger and Hicks 1991) and has been shown to have a predictive value in the outcome of bypass surgery (Tillisch et al. 1986; Tamaki et al. 1989).

In contrast to investigations with ^{18}F FDG, evaluation of myocardial perfusion with positron emission tomography (PET) requires a cyclotron or generator near the PET scanner due to the short half-life of the isotopes used (Gould 1991).

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Single photon emission tomography (SPET) with either thallium-201 or technetium-99m methoxyisobutylisonitrile (MIBI) may be used instead to investigate myocardial perfusion. Due to the more ideal physical characteristics of ^{99m}Tc compared with ^{201}Tl and its favourable kinetics (Okada et al. 1988; Stirner et al. 1988; Glover and Okada 1990), ^{99m}Tc -MIBI SPET has been employed increasingly in the evaluation of coronary artery disease (CAD) and has demonstrated a high sensitivity in the detection and localization of CAD (Kahn et al. 1989; Buell et al. 1990) and good agreement with TI (Stirner et al. 1988; Kiat et al. 1989).

The aim of this study was to establish a method for direct comparison of SPET (flow) and PET (metabolism) to ensure exact alignment of identical myocardial regions for comparable visual analysis and quantification. A previously described and established method for SPET quantification based on a target-like polar map was therefore adapted (Stirner et al. 1986; Buell et al. 1990). With this method, the extent and frequency of myocardial viability was then prospectively assessed in resting ^{99m}Tc -MIBI perfusion defects.

Materials and methods

Patients. A total of 46 patients (41 male, 5 female, mean age 56.6 ± 7.6 years) with angiographically proven CAD (three-vessel disease, $n=19$; two-vessel disease, $n=13$; one-vessel disease, $n=14$) and severe wall motion abnormalities (akinesis or severe hypokinesis) on cineventriculography was selected for this prospective study. The mean interval between coronary angiography and SPET/PET, which were performed on the same day, was 5.5 ± 7.5 weeks. Some 43 patients (40 with pathological Q-waves on ECG) had had a myocardial infarction 1 month to 7 years before PET (median 2 months). Patients with diabetes mellitus requiring oral antidiabetics or insulin therapy, left bundle branch block, unstable angina, angina during investigation or acute myocardial infarction were excluded from the study. Further clinical features of these patients were history of anterior wall infarction ($n=20$); posterior wall infarction ($n=20$); anterior and posterior infarction ($n=2$); lateral wall infarction ($n=1$); thrombolytic therapy ($n=14$); prior bypass surgery ($n=1$); prior coronary angioplasty ($n=3$).

MIBI SPET. ^{99m}Tc -MIBI SPET was performed on the same day as PET under resting conditions (Table 1). Anti-anginal drugs were withdrawn for 12 h to avoid potential effects on cardiac performance. Patients were fasted. ^{99m}Tc -MIBI was prepared according to the manufacturer's guidelines (Cardiolite, DuPont de Nemours). The labelling efficacy was more than 95% in all cases. After intravenous injection of 7–10 mCi (259–370 MBq) ^{99m}Tc -MIBI, patients were sent to have breakfast to increase biliary elimination. Myocardial SPET was performed between 1 and 2 h after administration. Arms were kept outside the field of view behind the patient's head. ^{99m}Tc -MIBI projections were obtained with a Siemens-Gamma-sonics ROTA-Dual double-head gamma-camera equipped with a leap (low-energy, all-purpose) collimator. The camera was rotated through 180° arc at 6° increments for 30 s each head in a matrix size of 64×64 . The transaxial slices (slice thickness of 6.25 mm) were reconstructed with a Butterworth filter of 5th order and a

Table 1. Imaging protocol

| | |
|----|--|
| 1. | Technetium-99m methoxy isobutyl isonitrile (^{99m}Tc -MIBI) injection at rest (7–10 mCi, patient fasted) |
| 2. | Patients sent to have breakfast |
| 3. | Marking of left ventricular borders (gamma-camera) |
| 4. | Transmission scan: 40 min acquisition time |
| 5. | Patients receive 50 g oral glucose (in tea) |
| 6. | Myocardial single photon emission tomography (SPET) (1–2 h after MIBI injection) |
| 7. | Fluorine-18 deoxyglucose (^{18}FDG) injection (6–8 mCi, 1 h after oral glucose) |
| 8. | Emission scan: 40 min acquisition time, starting 30–40 min after ^{18}FDG injection |

cut-off frequency of 0.5 on a Max Delta based on a $\mu\text{VAX II}$ as processing unit and a CDA image processor.

FDG PET. Attenuation corrected, ungated PET scans were obtained using a Siemens-CTI ECAT 953/15 tomograph with a field-of-view (fov) of 497 mm. Attenuation correction was performed by measuring a transmission scan with a germanium-68 ring source. Due to the one block ring configuration providing 15 transaxial image planes over a 5 cm axial width, two adjacent bed positions were needed to cover the entire heart. Each slice has a thickness of 3.375 mm. For patient positioning, the upper and lower left ventricular borders were determined by means of a radioactive surface marker (cobalt pen) after ^{99m}Tc -MIBI injection (arms beside the thorax, gamma-camera used for "fluoroscopy"). The exact position of the patient in the PET tomograph during the transmission scan was marked on the chest using a low-power laser light beam from the tomograph. Sixty minutes before injection of 6–8 mCi (222–296 MBq) ^{18}FDG , patients received 50 g of glucose orally over 30 min. After another 45 min emission scans were obtained after exact repositioning of the patient in the tomograph. During transmission and emission scan the patient's arms were within the field of view beside the thorax.

Emission PET scans were reconstructed with a Hanning filter (cut-off frequency 0.4) in a matrix size of 64×64 and a defined zoom factor of 1.2436 to obtain the same in-plane pixel size as the corresponding SPET studies (6.25×6.25 mm). Two PET slices were added to achieve a comparable axial pixel size for PET and SPET (6.75 mm and 6.25 mm, respectively).

To determine the spatial resolution in the final reconstructed image (equal acquisition and reconstruction parameters used in each study) the LSF (line spread function) was measured with a 2 mm steel line source (filled with ^{57}Co or ^{18}F) in the center of the FOV. The FWHM (full width at half-maximum) is 18.7 mm for SPET and 6.5 mm for PET.

The myocardial transaxial PET image file was converted into the file structure of the SPET files and transferred to the MAX Delta system for SPET using a LAN (local area network). After the arrangement of short and long axis cuts of the isotropic transaxial PET and SPET slices, corresponding tomograms in each direction were justified and documented. For a semiquantitative, three-dimensional region of interest (ROI)-based procedure of the short-axis cuts, the method of Stirner et al. (1986) was adapted (for details, see the Appendix).

Analysis of SPET and PET images. Myocardial ^{99m}Tc -MIBI distribution (documented on X-radiography film using a 10% lower

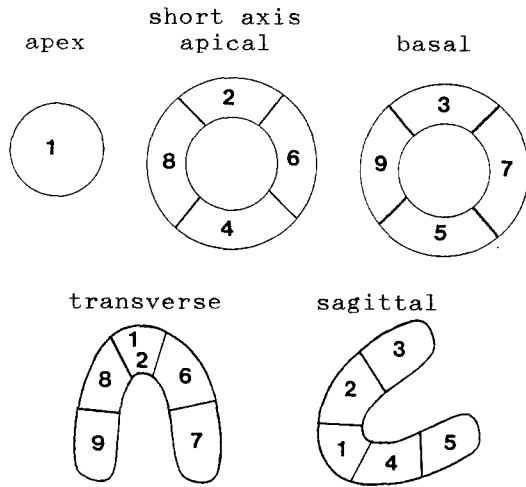


Fig. 1. The 9 left ventricular segments in the transverse slices, sagittal long-axis and short-axis cuts analysed in the study. 1, Apex; apical segments: 2, anterior, 4, inferior, 6, lateral, 8, septal; basal segments: 3, anterior, 5, inferior, 7, lateral, 9, septal

cut-off) was visually assessed by three independent and experienced clinicians. In 9 left ventricular segments (Fig. 1), ^{99m}Tc -MIBI uptake was differentiated into (a) normal or only slightly reduced or (b) definite perfusion defect (markedly reduced, no or only very faint uptake). A perfusion defect was defined if at least two observers agreed to this pattern, which had to be present in two different projections.

^{18}F FDG uptake in myocardial segments yielding a perfusion defect was assessed in the identical slices. A "match" was defined as a markedly reduced, very faint or lacking ^{18}F FDG uptake in the area of a perfusion defect (interpreted as myocardial scar). A "complete mismatch" refers to a normal, increased, or only very slightly reduced ^{18}F FDG uptake in the area of a perfusion defect (interpreted as myocardial ischaemia) and a "peripheral mismatch" to a preserved ^{18}F FDG uptake in the periphery of the defect only (considered to represent myocardial scar with adjacent ischaemia). If perfusion defects extended through a given segment and were also partly present in other segments, all of these were considered to be affected.

For quantitative evaluation ROIs were attached to myocardial segments according to Figs. 1 and 2. The outer (basal) circle of the target (Fig. 2) with 8 ROIs was omitted for quantification since, depending on the individual long-axis length and size of the membranous septum, this circle does not always contain myocardial tissue.

Relative ^{99m}Tc -MIBI uptake values (in percentage of peak activity) of all ROIs (according to Fig. 2) covering the identified perfusion defect in the given segment were added and divided by the number of ROIs, thus resulting in the average uptake value within the perfusion defect. In large perfusion defects extending more than one segment (according to Fig. 1), each affected segment was separately evaluated. Thus, if a perfusion defect for instance affected the apex, anterior wall and parts of the septum, for each of the affected segments ^{99m}Tc -MIBI uptake values were derived.

^{18}F FDG uptake values were derived from corresponding ROIs in the PET study. Again, average values were estimated and expressed as percentage of ^{18}F FDG uptake, which was set to 100% at the ROI presenting maximal ^{99m}Tc -MIBI uptake (100% ^{18}F FDG corresponds to 100% ^{99m}Tc -MIBI). Thus, segmental myocardial

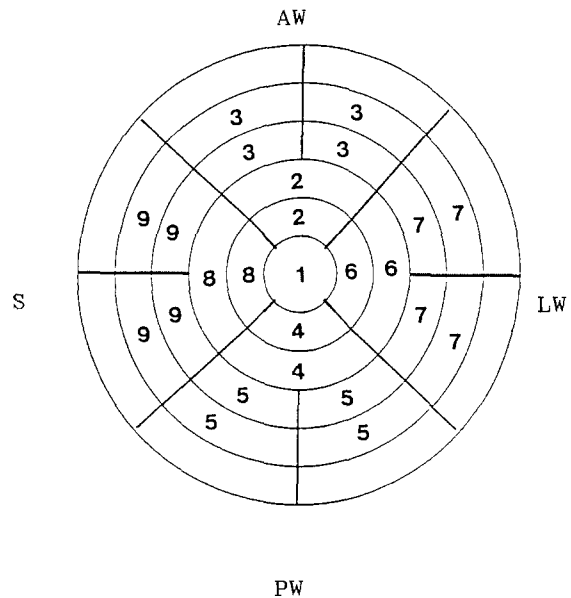


Fig. 2. Polar map of the left ventricle from apex (central region of interest, ROI) to base (outer circle) in form of a target disc, displaying anterior (AW), lateral (LW), posterior (PW) wall and septum (S). Numbers reflect segments (see Fig. 1), to which ROIs are related

^{18}F FDG uptake was related to the ^{18}F FDG uptake in the area revealing the "best" perfusion within the left ventricle.

Statistical analysis. Data are expressed as mean \pm standard deviation (SD). The paired Student's *t*-test was used for comparison of paired regional ^{99m}Tc -MIBI and ^{18}F FDG uptake values in perfusion defects. Uptake values between different groups were analysed with the non-paired Student's *t*-test. Comparison between flow/metabolism patterns with the degree of ^{99m}Tc -MIBI uptake was performed with the χ^2 analysis.

Results

Perfusion defects at rest were identified in 167 of 414 (40%) left ventricular segments in 46 patients (Table 2). Visual assessment of ^{18}F FDG uptake in the area of a perfusion defect demonstrated a "match" in 89/167 (53%) segments, while a "complete mismatch" was seen in 38/167 (23%) segments and a "peripheral mismatch" in 40/167 (24%) of segments (Table 3).

Quantitative assessment of ^{99m}Tc -MIBI and ^{18}F FDG uptake showed a good correlation with visual results. ^{99m}Tc -MIBI uptake values were below or equal to 30% of peak uptake in 41 (25%), between 31% and 50% in 100 (60%) and above 50% in 26 (15%) of the 167 segments with a perfusion defect (for location see Table 2).

Within segments presenting a "match" (Fig. 3), values for relative ^{99m}Tc -MIBI or ^{18}F FDG uptake were not significantly different [35.0% or 37.7%; $P > 0.05$; ^{18}F FDG range from 5% (minimal) to 46% (maximal)].

Table 2. Location and severity of perfusion defects assessed with ^{99m}Tc -MIBI SPET (in percentage of peak activity)

| Left ventricular segment | Severity of perfusion defect | | |
|--------------------------|------------------------------|---------|----------|
| | $\leq 30\%$ | 31%–50% | $> 50\%$ |
| 1 | 16 | 11 | 0 |
| 2 | 6 | 18 | 3 |
| 3 | 3 | 7 | 0 |
| 4 | 6 | 16 | 8 |
| 5 | 4 | 18 | 8 |
| 6 | 0 | 2 | 1 |
| 7 | 0 | 3 | 4 |
| 8 | 5 | 14 | 1 |
| 9 | 1 | 11 | 1 |
| Total | 41 | 100 | 26 |

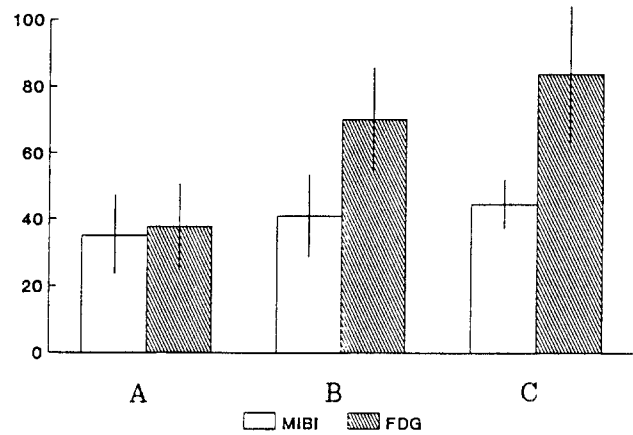
Values are numbers of defect

Table 3. Mean ^{18}F FDG uptake (values in percentage of ^{18}F FDG uptake in the ROI with maximal perfusion = 100% ^{99m}Tc -MIBI ROI) according to severity of perfusion defect (groups I–III)

| Visual classification | Severity of perfusion defect | | |
|--------------------------------|------------------------------|---------------------------|--------------------------|
| | Group I ($\leq 30\%$) | Group II (31%–50%) | Group III ($> 50\%$) |
| Match ($n=89$) | $n=33$ 29.8 ± 11.0 | $n=48$ 41.3 ± 11.2 | $n=8$ 49.0 ± 9.2 |
| Peripheral ($n=40$) mismatch | $n=8$ 61.9 ± 8.5 | $n=23$ 67.6 ± 15.0 | $n=9$ 85.0 ± 11.6 |
| Complete mismatch ($n=38$) | $n=0$ | $n=29$ 80.8 ± 21.0 | $n=9$ 94.6 ± 16.1 |
| Total ($n=167$) | $n=41$ | $n=100$ | $n=26$ |

In contrast, significant differences were found in areas with a “peripheral” (40.9% or 70.4%; $P < 0.00001$; ^{18}F FDG range 50%–110%) or “complete mismatch” (44.6% or 84.0%; $P < 0.00001$; ^{18}F FDG range 70%–180%). However, in these regions relative perfusion was significantly higher than in “match” regions (35.0% vs. 40.9%; $P < 0.05$, and 35.0% vs. 44.6%; $P < 0.0001$, respectively; Fig. 3).

If the severity of the perfusion defect (classified into three groups according to uptake values in percentage of peak activity, see Table 3) is taken into consideration, differences in the frequency of the derived categories became evident. None of the areas being perfused $\leq 30\%$ (group I) showed a “complete mismatch”, but 80% (33/41) represented a “match”, indicating a complete scar. The corresponding ^{18}F FDG uptake values differ significantly from those in groups II and III ($P < 0.01$). The relative frequency of a “match” decreases from 80% (33/41) in group I to 48% (48/100) in group II and 31% (8/26) in group III. On the other hand, a “complete

**Fig. 3.** Quantitative ^{99m}Tc -MIBI (in percentage of peak activity) and ^{18}F FDG (in percentage of ^{18}F FDG uptake in the ROI with maximal perfusion = 100% ^{99m}Tc -MIBI ROI) uptake in persisting perfusion defects according to visual SPET/PET findings. A, “match”; B, “peripheral mismatch”; C “complete mismatch” Bars represent means, I bars 1 SD

mismatch” was not seen in group I, and its occurrence increases from 29% (29/100) in group II to 35% (9/26) in group III. Thus, the frequency of a completely preserved or lacking ^{18}F FDG uptake in an area with a perfusion defect seems to be associated with the severity of perfusion reduction ($P < 0.0001$).

Of the 38 segments with a “complete mismatch”, 20 (53%) had a ^{99m}Tc -MIBI uptake $< 45\%$ of peak and 18 (47%), $\geq 45\%$ of peak.

Discussion

Several studies have investigated glucose metabolism in cardiac disease with ^{18}F FDG PET and have demonstrated preserved metabolism despite impaired contractile dysfunction (Brunken et al. 1986; Fudo et al. 1988; Tamaki et al. 1988), electrocardiographic Q-waves (Brunken et al. 1986) or perfusion defects on PET (Marshall et al. 1983) or ^{201}Tl SPET (Brunken et al. 1987,

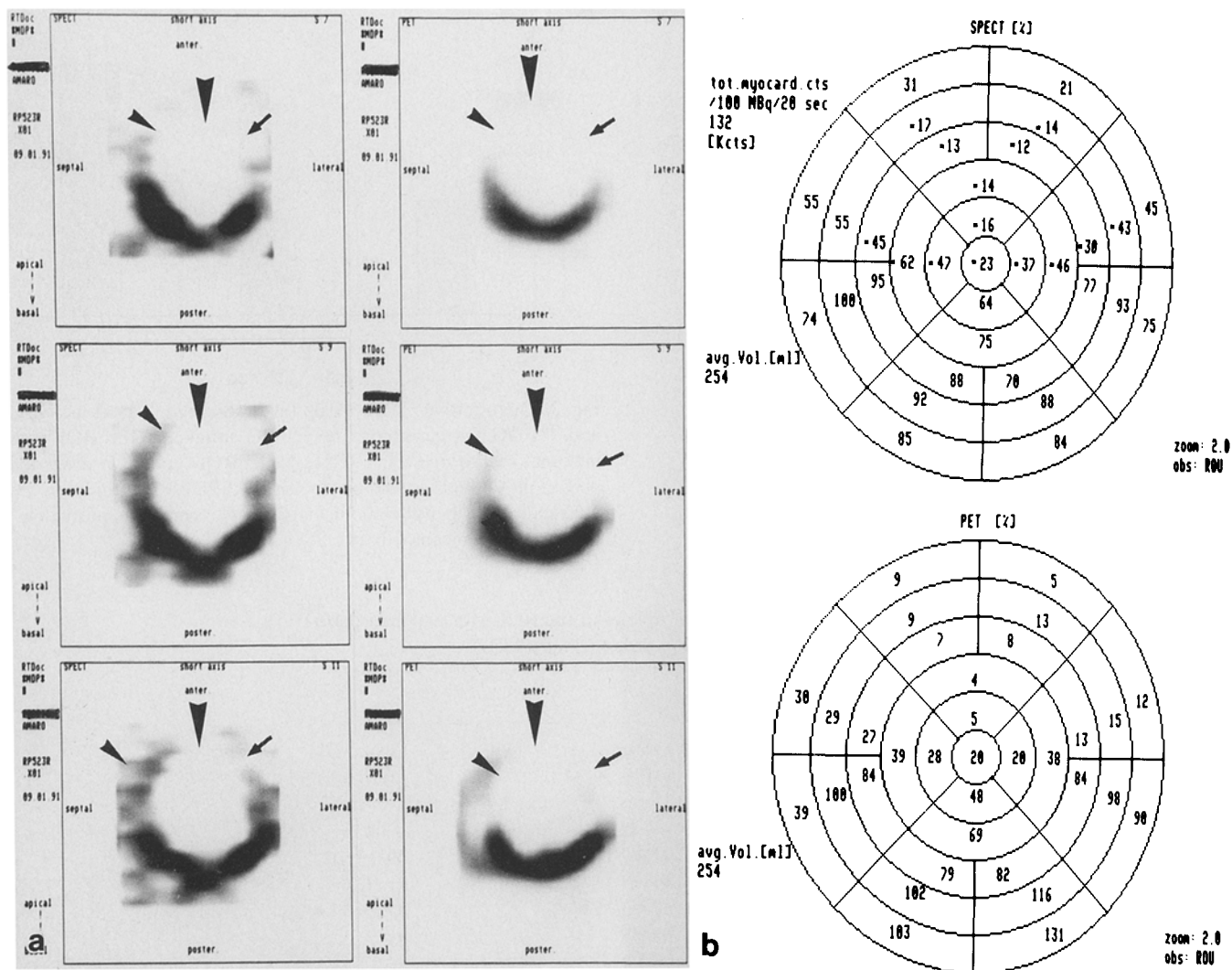


Fig. 4a, b. A 56-year-old male patient with a history of an old anterior myocardial infarction and Q-waves in leads aVL and V₃ on electrocardiogram (ECG). Coronary angiography revealed a 60% left anterior descending artery (LAD) stenosis, irregularities of left circumflex artery (LCX) and a large aneurysm of the apex and anterior wall. SPET/PET (a) shows a large “match” in the

LAD territory with decreased ^{99m}Tc-MIBI (a: left, arrows) and ¹⁸F₂DG (a: right, arrows) uptake. Relative ^{99m}Tc-MIBI uptake is markedly decreased in numerous ROIs (b: upper target, marked ROIs) with a corresponding ¹⁸F₂DG uptake (b: lower target) between 4% and 39%

1989). The predictive value of ¹⁸F₂DG PET for the recovery of contractile function after revascularisation in myocardium with impaired perfusion has been shown by Tillisch et al. (1986) and Tamaki et al. (1989) with a positive predictive value of 78%–85% and a negative predictive value of 78%–92%.

The ^{99m}Tc-labelled isonitrile MIBI, which has recently been introduced as a myocardial perfusion marker (Wackers et al. 1989a), correlates well with microsphere-determined blood flow (Li et al. 1988; Okada et al. 1988) and has been successfully used in the assessment of the area at risk (Gibbons et al. 1989) and the effect of thrombolytic therapy (Santoro et al. 1990; Wackers et al. 1989b). While ^{99m}Tc-MIBI SPET defect size demonstrated a good correlation with pathological infarct size in animal studies (Verani et al. 1988), little is known

about the significance of ^{99m}Tc-MIBI defects at rest in patients with CAD and after myocardial infarction for tissue viability and the outcome of revascularisation.

Thus, the aim of this prospective study was to determine whether perfusion defects seen with ^{99m}Tc-MIBI SPET at rest and thought to represent myocardial scar might exhibit preserved glucose metabolism as an indicator of viable myocardial tissue. A 1-day rest protocol was chosen to ensure equal physiological conditions for both SPET and PET investigations. Our data indicate that only 53% of myocardial regions with a perfusion defect lack glucose metabolism, whereas in 23% of these perfusional regions, a completely preserved and in 24%, a partially preserved glucose metabolism is demonstrable. ^{99m}Tc-MIBI uptake at rest therefore underestimates the extent of viable myocardium in patients with CAD.

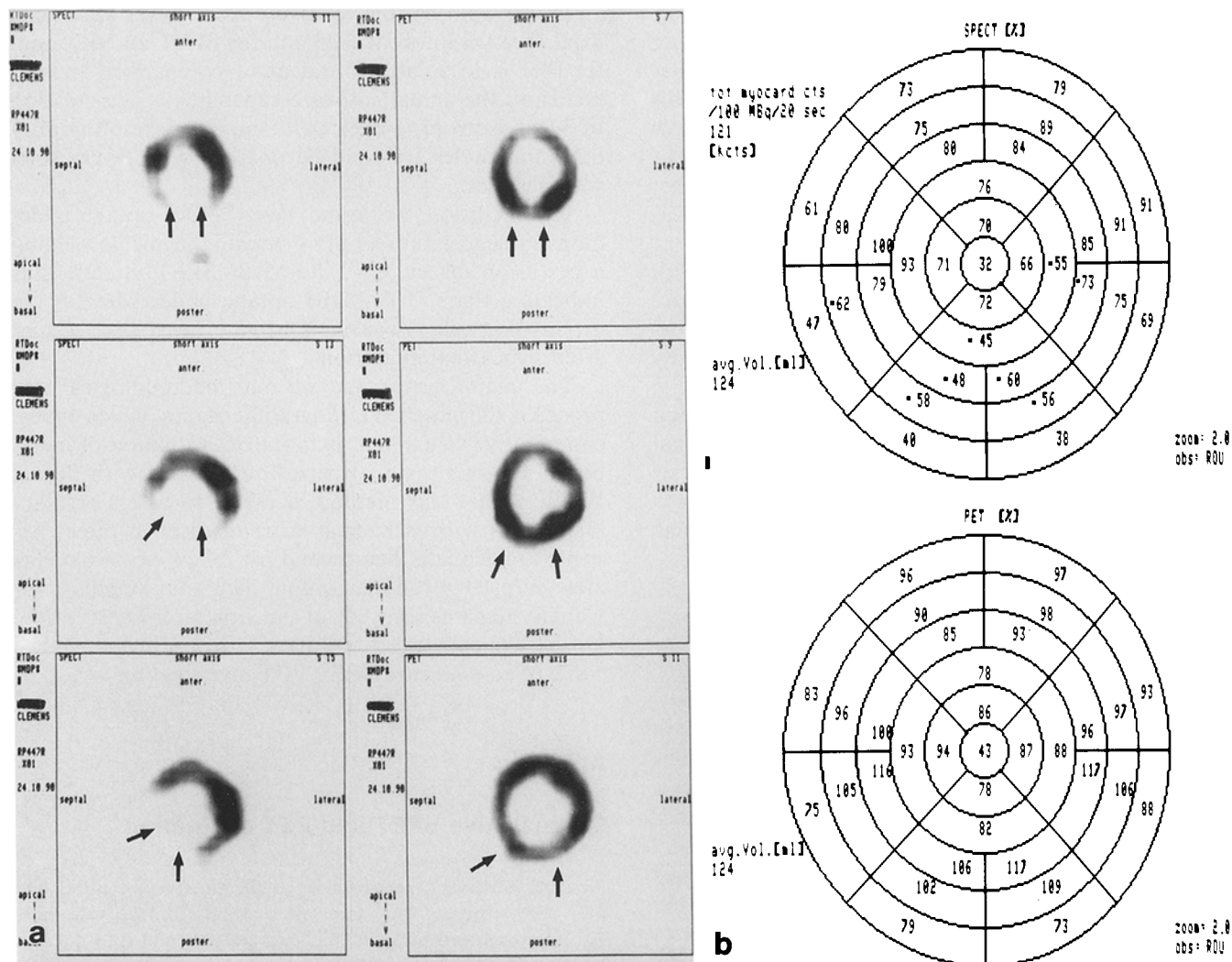


Fig. 5a, b. A 59-year-old male patient with previous myocardial infarction of the posterior wall (on ECG Q-waves in leads II, III and aVF). Coronary angiography revealed an occlusion of the right coronary artery (RCA), 90% LCX stenosis, irregularities of LAD and an akinetic posterior wall. SPET (left)/PET (right) (a) shows

a “complete mismatch” in the RCA territory (arrows) with a reduced regional ^{99m}Tc -MIBI uptake (b, marked ROIs in upper target) between 45% and 73% and a corresponding ^{18}F FDG uptake between 82% and 117% (b, lower target)

Perfusion defects in our study with a ^{99m}Tc -MIBI uptake below 30% of peak uptake always contained scar tissue according to PET criteria. On the other hand, 30% (38/126) of segments with a ^{99m}Tc -MIBI uptake of more than 30% of peak uptake or 35% (9/26) of segments with a ^{99m}Tc -MIBI uptake above 50% of peak are regarded as viable, while another 25% (32/126) of segments with a score of more than 30% uptake at least partly contain viable tissue. From these data we conclude that only moderate reduction of perfusion assessed with ^{99m}Tc -MIBI SPET implies a greater likelihood of potential viability than severe reduction.

Buell et al. (1990) reported a considerable correlation between regional minimal myocardial ^{99m}Tc -MIBI uptake of an exercise-induced defect and reversibility at rest in patients with CAD. They reported that exercise-induced defects with a minimum ^{99m}Tc -MIBI uptake

below 45% of peak activity proved irreversible in 94% of involved segments. In the present study 18/38 (47%) segments with a “complete mismatch” exceeded this uptake value at rest. Assuming partial reversibility of an exercise-induced defect as a criterion for myocardial viability, these segments are likely to be detected by a stress/rest study. However, the remaining 20 segments (53%) with a ^{99m}Tc -MIBI uptake below 45% of peak activity were found viable with ^{18}F FDG only.

The ^{18}F FDG uptake values in this study correlate well with visual results (Figs. 4, 5) and distinguish regions with a “match” from those with a “peripheral” or “complete mismatch”, which show a wide range of uptake values. In myocardial regions which are regarded as centrally infarcted and ischaemic at the periphery (“peripheral mismatch”), the degree of ^{18}F FDG uptake primarily depends on the extent of viable tissue in this

area. Thus, quantification of ^{18}F FDG uptake, which corresponds to exogenous glucose metabolism (Schwaiger and Hicks 1991), might give useful additional information for the indication of invasive procedures in this situation. In ischaemic myocardium, ^{18}F FDG uptake values were between 70% and 180% of ^{18}F FDG uptake in the area of "best perfusion" and probably reflect different metabolic situations in ischaemic regions (Camicci et al. 1989).

Rocco et al. (1989) demonstrated in 8 patients with $^{99\text{m}}\text{Tc}$ -isonitrile defects at rest after myocardial infarction that improvement of $^{99\text{m}}\text{Tc}$ -isonitrile uptake following coronary bypass surgery occurred more frequently in segments with a higher uptake value preoperatively. The predictive value of ^{18}F FDG uptake values in relation to $^{99\text{m}}\text{Tc}$ -MIBI uptake for the improvement of regional contractile function after percutaneous angioplasty or bypass surgery has to be ascertained by direct comparison of pre- and postintervention evaluation of cardiac function.

Limitations of the SPET/PET protocol

The main technical limitation of a direct comparison of SPET and PET lies in the different imaging modalities of these tomographic systems (Sorenson and Phelps 1987). While PET data are corrected for photon attenuation, this still remains a problem with SPET. In the present study perfusion defects were defined on the basis of a markedly reduced $^{99\text{m}}\text{Tc}$ -MIBI uptake, which could not be attributed to photon attenuation. Uptake values in these areas were always clearly below normal values of patients meeting the <5% likelihood of CAD (Buell et al. 1990). False-positive SPET results in identifying ischaemic or infarcted areas are therefore unlikely in this series. Although attenuation effects for $^{99\text{m}}\text{Tc}$ are much less than for ^{201}Tl due to its higher photon energy, attenuation from the inferior wall and septum influences $^{99\text{m}}\text{Tc}$ -MIBI uptake values. For comparable quantification of SPET and PET data, the lack of attenuation correction for SPET has to be taken into consideration for ^{18}F FDG/ $^{99\text{m}}\text{Tc}$ -MIBI uptake ratios to differentiate myocardial scar from ischaemia. In this respect, uptake ratios will vary between different anatomical areas. Two approaches are directed towards this problem. First, ^{18}F FDG and $^{99\text{m}}\text{Tc}$ -MIBI uptake values can be investigated in patients without evidence of cardiac disease to derive normal ratios. Second, the predictive value of these ratios can be retrospectively determined from pre- and postintervention studies for all myocardial regions.

In addition, uptake ratios will be influenced by the partial volume effect. The underestimation of true tracer concentration increases as wall thickness (e.g. after myocardial infarction) declines and depends on the spatial resolution (Sorenson and Phelps 1987), which is different for SPET and PET.

In our opinion, the relatively small deviation of the axial slice thickness of 6.25 mm for SPET and 6.75 mm for PET is acceptable for adequate comparison and depends on the actual software capabilities. The creation of ideal isotropic voxels with an axial resolution of 6.25 mm for PET should be possible with future software releases.

In conclusion, we found that ^{18}F FDG uptake is demonstrable in a number of myocardial segments yielding a perfusion defect on $^{99\text{m}}\text{Tc}$ -MIBI SPET at rest, thus indicating that $^{99\text{m}}\text{Tc}$ -MIBI uptake underestimates the extent of viable tissue in ischaemic heart disease and after myocardial infarction.

The major advantage of our methodological approach is the matched tomographic planes, which enable comparative visual and quantitated evaluation of metabolic PET and regional perfusion assessed with SPET. We find, that this method is useful to assess viability in patients with ischaemic heart disease, to direct patients to adequate therapy and for follow-up of patients after surgical revascularization. The gold standard for viability remains recovery of function after intervention. Larger clinical studies will have to confirm the clinical value of comparative SPET/PET investigations.

Appendix

Comparative SPET and PET evaluation

First, the observer determined 16 consecutive oblique SPET and PET slices covering the entire left ventricle from apex to base. For improved statistical analysis, one slice was added to the next iteratively, yielding 15 overlapping double slices of approximately 12.5 mm in SPET (13.5 mm in PET). Correspondence of SPET and PET slices was then achieved by adapting the long-axis outlines rebuilt from summed profiles of the 15 oblique slices in each study.

In the next step, the observer fitted circular (at the apex) or ring-shaped ROIs to the myocardial pattern to obtain a total of 33 ROIs over 6 oblique double slices from apex to base (1-4-4-8-8-8). Calculated numerical arrays of extracted regional parameters (Table 4) were finally displayed in a target-like, two-dimensional, polar fashion (Figs. 4b, 5b). The first target with regional parameters (1 of 33 ROIs) represents the regional uptake, Upt. (SPET), of $^{99\text{m}}\text{Tc}$ -MIBI SPET derived from the relation of the ROI-density,

Table 4. Definitions of calculated parameters

| | |
|---|--------|
| Upt. (SPET) _i = DE(SPET) _i / max {DE(SPET) _j } | × 100% |
| Upt. (PET) _i = Co (PET) _i / Co(PET) _{mx} | × 100% |

DE(SPET)_i, density of region index i on SPET means counts of ROI_i/voxel of ROI_i; max = mx, index of region with the maximum

density DE(SPET)_i on SPET

Co(PET)_i, concentration of ^{18}F FDG in nCi/ml of region i in PET; i, number of ROI

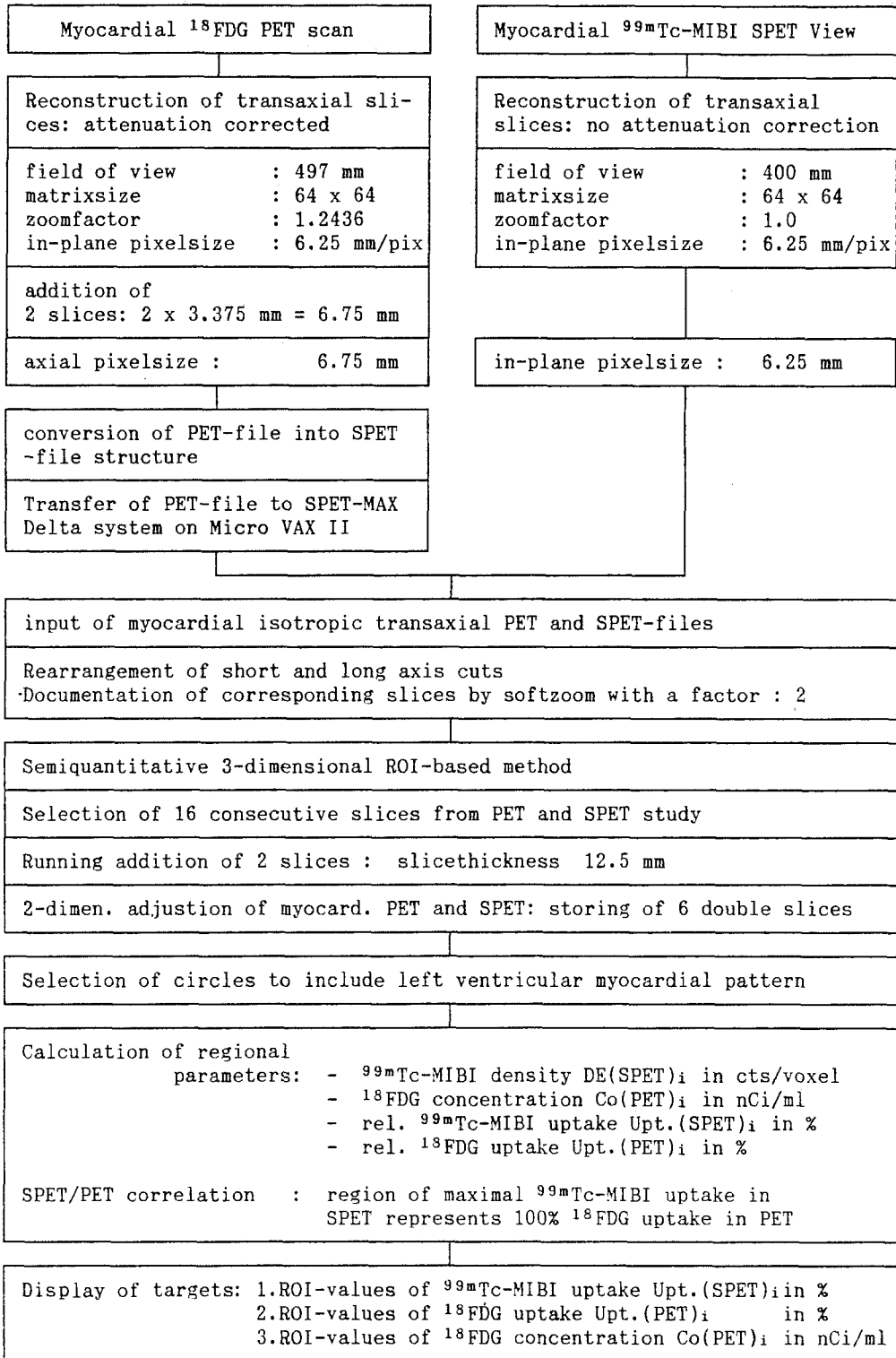


Fig. 6. Flow chart for correlation and comparative evaluation of SPET and PET scans

$DE (SPET)_i$, and the maximum density, $\max_i \{DE(SPET)_i\}$, in percentage of peak activity (Table 4), normalized to an applied activity of 100 MBq and an acquisition time of 20 s/ projection view.

The second target represents the regional ^{18}F FDG uptake, $Upt. (PET)_i$, obtained by the relation of the concentration, $Co (PET)_i$,

in nCi/ml and $Co(PET)_{mx}$ where index mx means the region with the maximum density on SPET, normalized to an applied activity of 100 MBq.

The third target shows additionally the concentration of ^{18}F FDG $Co(PET)_i$ itself.

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