Characterization of Nontransmural Myocardial Infarction by Positron-emission Tomography

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SUMMARY The present study was performed to determine whether positron emission tomography (PET) performed after i.v. $^{11}$C-palmitate permits detection and characterization of nontransmural myocardial infarction. PET was performed after the i.v. injection of $^{11}$C-palmitate in 10 normal subjects, 24 patients with initial nontransmural myocardial infarction (defined electrocardiographically), and 22 patients with transmural infarction. Depressed accumulation of $^{11}$C-palmitate was detected with sagittal, coronal and transverse reconstructions, and quantified based on 14 contiguous transaxial reconstructions. Defects with homogeneously intense depression of accumulation of tracer were detected in all 22 patients with transmural infarction (100%). Abnormalities of the distribution of $^{11}$C-palmitate in the myocardium were detected in 23 patients with nontransmural infarction (96%). Thallium scintigrams were abnormal in only 11 of 18 patients with nontransmural infarction (61%). Tomographically estimated infarct size was greater among patients with transmural infarction (50.4 ± 7.8 PET-g-Eq/m² [± SEM]) compared with those with nontransmural infarction (19 ± 4 PET-g-Eq, p < 0.01). Residual accumulation of $^{11}$C-palmitate within regions of infarction was more intensely depressed among patients with transmural compared to nontransmural infarction (33 ± 1 vs 39 ± 1% maximal myocardial radioactivity, p < 0.01). Thus, PET and metabolic imaging with $^{11}$C-palmitate is a sensitive means of detecting, quantifying and characterizing nontransmural and transmural myocardial infarction.

Materials and Methods

Patients

All 56 subjects gave informed written consent. The group included 10 normal subjects (six males and four females, average age 39 years, range 23–68 years) and 46 patients who had survived acute myocardial infarction for at least 72 hours. Control subjects were seven normal volunteers without a history of chest pain or myocardial infarction and with normal physical examinations and three patients admitted to the coronary care unit for the evaluation of atypical chest pain in whom myocardial infarction was excluded based on serial electrocardiographic and enzymatic criteria. None of the controls had experienced previous myocardial infarction or exhibited Q waves on the ECG.

The 46 patients (31 male, 15 female, mean age 57 years, range of 29–84 years) experienced myocardial infarction documented by a history of precordial chest pain of at least 20 minutes duration and serial elevations of total and MB plasma CK. Demographic data are summarized in table 1.

Patients with myocardial infarction preceding the index episode were excluded. Transmural myocardial infarction was considered to be present if Q waves of 30 msec duration were seen in recordings from at least two concordant leads. In the absence of these criteria, nontransmural myocardial infarction was considered to be present. Infarction was transmural in 22 and nontransmural in 24 patients.

Radiochemicals and Tomographic Procedures

The $^{11}$C-palmitate was prepared with a synthesis developed at Washington University that involves the addition of $^{11}$CO₂ produced in the medical center cyclotron to a Grignard reagent (1-MgBr-pentade-
TABLE 1. Characteristics of Subjects

<table>
<thead>
<tr>
<th></th>
<th>Transmural infarction</th>
<th>Non-transmural infarction</th>
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<tr>
<td>n</td>
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<td>Mean age (years)</td>
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<td>Sex (M/F)</td>
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<td>Infarct location (ECG)</td>
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<tr>
<td>Anterior</td>
<td>—</td>
<td>15</td>
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<tr>
<td>Inferior</td>
<td>—</td>
<td>7</td>
</tr>
<tr>
<td>Undetermined</td>
<td>—</td>
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</tr>
</tbody>
</table>

Blood pool tomographic imaging was performed with red blood cells labeled in vivo with $^{14}$CO administered by inhalation. The total body radiation absorbed from combined myocardial and blood pool tomography using 20 mCi of $^{11}$C-palmitate and 30 mCi of $^{14}$C-carbon monoxide was $\leq$ 450 mrem, with a maximum of 2600 mrem delivered to the liver and 1740 mrem delivered to the blood.

Tomography was performed with a positron-emission transaxial tomograph (PETT-IV), which provides for the simultaneous acquisition of data needed to reconstruct seven parallel transaxial cross sections (slices) of the heart. Midpoints of the sections are separated by approximately 1 cm. After moving the patient 1 cm and repeating the tomographic imaging procedure, 14 interlacing transaxial slices can be reconstructed, spanning a height of 12.35 cm. Initially, a ring containing a positron-emitting radionuclide, gallium-68 ($^{68}$Ga), was placed around the patient to provide data for measuring attenuation coefficients. Subsequently, patients were studied after i.v. injection of 15-20 mCi of $^{11}$C-palmitate. After a delay of approximately 2 hours to permit decay of the $^{11}$C-palmitate (half-life of 20.4 minutes), patients were restudied after inhaling 20-30 mCi of $^{14}$CO to delineate the approximate endocardial borders in each transaxial slice by comparing the $^{14}$CO reconstruction superimposed on the $^{11}$C-palmitate reconstruction. All studies were performed without gating of the cardiac cycle, because the limitations of the spatial averaging used were not considered critical to the primary purpose of detecting and localizing subendocardial injury. The resolution of the tomographic system was 13.5 mm (full-width, half maximum) in the plane of the section with a section thickness of 16 mm.

To assess the potential dependence of the sensitivity of PET on the time after onset of myocardial infarction, we performed studies at selected intervals after the apparent onset of infarction. Patients with non-transmural infarction were studied an average of 31 days (range 3-46 days) after its onset, when hemodynamics were stable and the patients were not experiencing chest pain. Patients with transmural infarction were studied an average of 42 days after onset (range 2-175 days). During all tomographic studies, patients were attended by a physician. The ECG and vital signs were monitored continuously. No complications of the procedure were encountered.

Assessment of the Extent of Myocardial Injury

Tomographic reconstructions of the distribution of $^{11}$C-palmitate accumulation in normal subjects generally exhibited homogeneous accumulation of tracer throughout a horseshoe-shaped region with a nearly constant ventricular wall thickness. The most posterior aspect of the ventricle generally exhibited a discontinuity of $^{11}$C-palmitate accumulation in the region of the mitral valve apparatus, because the apparatus and atrial myocardium are so thin and metabolically inactive. However, among patients with infarction, regions of depressed accumulation of palmitate appeared as either abnormal discontinuities of accumulation of palmitate or as apparent variation of the thickness of the horseshoe-shaped region of ventricular uptake and inhomogeneity in the magnitude of accumulation of palmitate within this region (figs. 1-3). Data from each transaxial, sagittal, and coronal section were displayed on an oscilloscope in a 100 × 100 element grid with a 256-level gray scale. A printout was generated with a Versatec printer/plotter of the region encompassing the heart in which a numerical value from 0-255 was assigned to each pixel, indicating the relative radioactivity detected within each corresponding region of tissue. The contour of the ventricle was constructed and the region of infarction defined as a zone within the contour that contained less than 50% of the maximal myocardial radioactivity as previously described

\[
\text{Infarct size} = \sum_{i=1}^{n} V_i \left[ \frac{100-a_i}{100} \right] \times 1 \left[ \frac{g}{cm^2} \right]
\]

where \( n \) is the number of transverse reconstructions with a defect, \( V \) is the volume of the infarction in a reconstruction (calculated from the thickness of the section and multiplied by the total area of the defect), and \( a \) is the average radioactivity within the region of the defect in the slice. This calculation reflects both the distribution and the integrated magnitude of the depression of accumulation of $^{11}$C-palmitate. The calculated volume of the lesion is converted to gram-equivalents (g-Eq), based on the assumption that the specific density of myocardium is approximately 1 g/cm$^3$.

Both maximal radioactivity within normal myocardium and mean activity within regions of infarction were corrected for background radioactivity by measuring mean radioactivity within a background region within the atrial blood pool. Background was not based on the ventricular blood pool because cardiac motion may artifactually increase apparent activity in the ventricular blood pool region due to partial volume effects. A separate background value was obtained for each tomographic reconstruction. In the caudal sections that contained no atrial blood pool re-
FIGURE 1. Color superimpositions of images obtained after i.v. 11C-palmitate (green) and subsequent inhalation of 11C-carbon monoxide (red) from a normal subject and from patients with anterior nontransmural and anterior transmural myocardial infarction.

region, background was determined from the descending aortic blood pool region.

Among patients with inferior and inferoapical myocardial infarction (either transmural or nontransmural), sagittal and coronal reconstructions were required to estimate the extent of myocardial injury reflected by diminished accumulation of 11C-palmitate because reductions of uptake were often confined to the one or two most caudal transaxial sections in which the delineation of the endocardial borders is difficult. However, inferoapical and inferoposterior regions of injury were easily visualized in coronal and sagittal reconstructions. These reconstructions were performed after mathematical stacking of interdigitated transaxial reconstructions from initial and repeat data collections. Since sagittal and coronal reconstructions contained data that were collected in a nonsimultaneous fashion, with data collection intervals of different durations (in order to ensure adequate counting statistics), data from either the first or second set of transverse tomographic reconstructions were corrected with a scaling factor to normalize the count density in the alternating sections. In normal subjects, the accumulation of isotope was homogeneous, reflecting a relatively uniform apparent myocardial wall thickness in sagittal and coronal reconstructions with variations of only two to four pixels (fig. 2).

Sagittal and coronal reconstructions were displayed oscilloscopically and the data within the region encompassing the heart were plotted with a 16-level gray scale. Regions with less than 40% of the maximal myocardial radioactivity were white. A 40% threshold (as opposed to a 50% threshold used for transaxial sections) was used to minimize a banding effect related to the time-dependent acquisition of differing counts in alternating transaxial sections. Myocardial outlines were constructed (assuming a uniform wall thickness for the normal ventricle), and regions within the outline containing less than 40% of maximal myocardial radioactivity were designated as zones of infarction (fig. 2). Data from the sagittal and coronal reconstructions were used only to delineate the approximate coordinates of the region of infarction rather than for calculations. The information defining boundaries obtained from sagittal and coronal reconstructions was transposed to the transverse reconstruction as shown in figure 2B, and both normal and abnormal regions were outlined. Infarct size was then calculated with the same algorithm used for direct analysis of transaxial data.

Thallium Imaging

Myocardial imaging was performed with 201Tl to compare its sensitivity to that of PET. Imaging was performed with the patient at rest, 10–15 minutes after administration of 1–1.5 mCi of 201TlCl

venously. Data were collected with a standard-field (25.4-cm-diameter, 0.64-cm-thick NaI crystal) scintillation camera (Searle LEM) fitted with a low-energy, medium-resolution, parallel-hole collimator interfaced to a dedicated minicomputer (Technicare VIP 450). Images were obtained in the anterior, 35° and 65° left anterior oblique and left lateral projec-
tions. Each image consisted of at least 250,000 counts. Thallium imaging was performed within a mean of 2 ± 3 (SD) days of the corresponding PET study. Studies were interpreted from both analog images obtained at the time of data collection displayed on transparency film and from digital data displayed on the minicomputer after selected levels of background subtraction and contrast enhancement. We previously reported a close agreement between independent observers using this technique. Thallium and PET studies were interpreted independently by separate observers without knowledge of the results of the other procedure or the clinical diagnosis.

**Statistical Methods**

Results are expressed as mean ± SD or SEM, as indicated. Mean data were compared using the *t* test for unpaired samples, and proportions were compared by chi-square analysis or McNemar's test, where appropriate.

**Results**

**Tomography in Control Subjects**

The normal left ventricular contour delineated by PET at the midventricular level conformed to a horseshoe-shaped region with a uniform apparent wall thickness averaging 7.0 ± 0.3 (SEM) pixels in the septum, 8.0 ± 0.3 in the anterior wall and 7.9 ± 0.2 in the lateral wall (fig. 1). The regions corresponding to the mitral valve and atria did not accumulate enough radiopharmaceutical to be visualized due to the thinness of these structures and their relatively low metabolic rate. The right ventricle was visualized as a thin crescent, anterior and to the right of the left ventricle. The extent of accumulation of palmitate in the right ventricle was less than that in the left ventricle (often only 20–40% of maximal myocardial palmitate activity). This relatively slight accumulation reflects the low metabolic demands and the thin structure of the right ventricle. When 11C-carboxyhemoglobin blood pool and 11C-palmitate reconstructions obtained in normal patients were superimposed, the blood pool completely filled the horseshoe-shaped ventricular region of palmitate accumulation and extended posteriorly, representing atrial blood pools as well. The borders of the region of 11C-carbon monoxide activity and 11C-palmitate accumulation were in close approximation (fig. 1).

Serial sagittal and coronal reconstructions in normal subjects demonstrated comparable apparent thickness of the regions of palmitate accumulation in the anterior, anteroapical and inferior walls with homogeneous accumulation of palmitate (fig. 2A). In posterior coronal reconstructions, a zone of absent accumulation was generally observed in the superior portion of the septal region due to the thin, membranous portion of the ventricular septum. In sagittal

**Figure 2.** (A) Representative midventricular transverse reconstruction from a normal subject obtained with positron-emission transaxial tomography (PET IV) after 11C-palmitate is displayed (center) along with sagittal (top) and coronal (right) reconstructions. The planes of sagittal and coronal reconstructions are indicated by the solid lines. The white area to the left of the coronal reconstruction represents the liver, which has been partially blocked out for photographic purposes. S = superior; I = inferior; A = anterior; P = posterior; L = left; R = right. (B) Schematic representations of transverse (center), sagittal (top) and coronal (right) reconstructions from a patient with an inferior-posterior infarction. The horizontal bars on the sagittal and coronal views indicate the plane of the transverse reconstruction. The solid lines connecting the sagittal, coronal and transverse views indicate the planes of section. The discontinuities indicated by the interrupted chamber outlines indicate regions of infarction.
TOPIC

In patient transaxial reconstructions corresponding criteria, enzymatic transverse reconstructions.

The mitral valve and the coronary activity diminished region of confluent in whom to demonstrated this images medical center.

Homogeneously decreased accumulation of tracer, and normal zones demonstrated homogeneous uptake throughout.

A close concordance was observed between electrocardiographic and the tomographic loci of infarction. Thus, infarction associated with Q waves in $V_1-V_6$ was associated with depression of accumulation of palmitate in regions corresponding to the septum and midanterior wall. Infarction associated with Q waves in leads $V_4-V_6$ was associated with depressed accumulation of palmitate in regions corresponding to anterolateral regions of the ventricle. Electrocardiographically typical apical or inferior infarctions were usually associated with no tomographic defect in the midventricular transverse reconstructions. However, defects were evident consistently in sagittal and coronal reconstructions and were localized to the apical and inferior walls. Decreased accumulation of palmitate was detected in all 11 patients with transmural infarction, with the estimated mass of infarction averaging $50.4 \pm 7.8 \text{ (SEM)}$ (table 2).

**Nontransmural Infarction**

Nontransmural myocardial infarction was evident as a region of diminished myocardial accumulation of palmitate. However, the regions of depressed accumulation of isotope were often not transmural. Frequently, they were manifest by apparent thinning of the zone of accumulation of palmitate (fig. 1). In the reconstruction shown, the septum and posterior wall exhibited normal accumulation of palmitate. The

| Table 2. Tomographic Characteristics of Infarcts |
|----------------|----------------|----------------|
| Infarct size (PET-g-Eq) | Transmural infarction | Nontransmural infarction |
| Residual radioactivity in zone of infarction (% max. myocardial radioactivity) | $50.4 \pm 7.8$ | $19 \pm 4$ | $p < 0.01$ |
| Proportion of patients with abnormal PET images | $100\% (22/22)$ | $96\% (23/24)$ | NS |

Values are mean ± SEM.

Abbreviation: PET = positron-emission tomography.
of transmural infarction, but only heterogeneously and less severely depressed in regions of nontransmural infarction (fig. 3). Residual radioactivity within the region of infarction (expressed as a percentage of the maximal myocardial radioactivity) averaged 33 ± 1% (SEM) among patients with transmural infarction in contrast to 39 ± 1% among patients with nontransmural infarction (p < 0.01) (table 2).

Nontransmural infarction in tomographic reconstructions was characterized by apparent thinning of the region of accumulation of palmitate within left ventricular myocardium and by heterogeneity of depression of accumulation as well. Figure 4 shows transverse PET reconstructions from a normal subject and from subjects with nontransmural myocardial infarction. Only one of the 24 patients with nontransmural myocardial infarction exhibited normal and homogeneous patterns of accumulation of 11C-palmitate in transverse, sagittal and coronal reconstructions. The remaining 23 (96%) had striking abnormalities; examples are shown in figure 4. Patients 109 and 94 had regions of accumulation of palmitate, with only shell-like intramural regions of apparently normal accumulation of palmitate remaining within the regions of apparent infarction. Palmitate accumulation is heterogeneous within the

Figure 4. Positron-emission transaxial tomographic (PETT IV) images obtained after i.v. 11C-palmitate at approximately the midventricular level in a normal subject and from patients with nontransmural myocardial infarction at the loci indicated.
apparently normal regions as well as within the zones of infarction. Patient 104 demonstrated a similar heterogeneous pattern of depression of palmitate within the anterolateral and posterolateral walls of the ventricle. Patients 105 and 112 showed small-to-moderate, apparently transmural defects, seen in only a minority of the patients with nontransmural infarction (30%) and comparable in overall extent to many examples of nontransmural infarction (based on enzymatic as well as tomographic criteria). Infarct size in patients with nontransmural myocardial infarction ranged from 0–66 PET-g-Eq (average SEM 19 ± 4 PET-g-Eq). Overall, these values were significantly less than values of average infarct size in patients with transmural myocardial infarction (p < 0.01; table 2).

The electrocardiographic locus of infarction did not correspond closely to the locus of depressed accumulation of palmitate among patients with nontransmural infarction (table 2). The ECG failed to identify any specific locus in two. Nevertheless, anteroseptal and anterolateral electrocardiographic abnormalities corresponded well to the localization of metabolic defects visualized by PET (table 3).

**Thallium Imaging**

Thallium-201 imaging was performed in 18 of the 24 patients with nontransmural infarction; the six others were reluctant to undergo a second radionuclide procedure. There were no obvious or statistically significant differences in gender, age, infarct location or estimated infarct size among patients who were compared to those who were not studied with $^{201}$TI. Among the 18 patients studied with thallium, 11 (61%) had abnormal $^{201}$TI images. The location of the abnormalities in the $^{201}$TI images corresponded to the location of the discontinuities of the accumulation of palmitate in PET reconstructions in these patients. In contrast, PET reconstructions exhibited regions of depressed regional palmitate accumulation in 17 of the 18 patients (94%). Overall, abnormalities were detected in 23 of 24 patients with nontransmural infarction studied with PET, compared to 11 of 18 patients studied with $^{201}$TI (p < 0.05) (table 4).

No significant differences in mean estimated infarct size distinguished patients with normal compared to those with abnormal thallium images. Among the patients with normal $^{201}$TI images, one exhibited a large region of diminished accumulation of palmitate (patient 104, fig. 4). Another exhibited relatively small, nontransmural diminutions of accumulation of palmitate (patient 94, fig. 4). Several exhibited primarily apical diminution of accumulation of $^{11}$C-palmitate, evident only in sagittal and coronal reconstructions. In general, among patients who exhibited an abnormality on the $^{201}$TI scan, the locus corresponded to regions of

**Table 3. Correlation Between Infarct Localization by Electrocardiography, Positron-emission Tomography and Thallium-201 Imaging: Patients with Nontransmural Myocardial Infarction**

<table>
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<th>Pt</th>
<th>ECG location</th>
<th>PET location</th>
<th>Thallium location</th>
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<td>72</td>
<td>Anteroinferior</td>
<td>Anteroapical-inferior</td>
<td>Apicoanterior</td>
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<tr>
<td>90</td>
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<td>Inferior</td>
<td>Inferior</td>
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<td>91</td>
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<td>Apical</td>
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<td>93</td>
<td>Anterolateral/inferior</td>
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<td>Normal</td>
</tr>
<tr>
<td>94</td>
<td>Anterior</td>
<td>Anterior</td>
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<td>Normal</td>
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<tr>
<td>101</td>
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<td>Posterior</td>
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<td>120</td>
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<td>122</td>
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<tr>
<td>124</td>
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<tr>
<td>127</td>
<td>Anteroseptal</td>
<td>Anteroseptal</td>
<td>Septal-apical</td>
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</table>
diminished accumulation of $^{11}$C-palmitate seen with PET (table 3).

**Discussion**

Results of this study indicate that PET is a sensitive and specific technique for detecting nontransmural and transmural myocardial infarction. Abnormalities were detected by PET in all 22 patients with transmural infarction and 23 of 24 patients with nontransmural infarction. In contrast to the apparent sensitivity of PET for nontransmural infarction of 96%, only 61% of patients with nontransmural infarction had abnormal $^{201}$Tl scintigrams. Small regions of depressed accumulations of palmitate were noted in septal regions of two of 10 control subjects. However, one of these subjects may have had antecedent intramural infarction.

Substantial differences distinguished the tomographic appearance of transmural from nontransmural infarction. Transmural infarction (defined electrocardiographically) exhibited homogeneous depression of accumulation of $^{11}$C-palmitate extending from the endocardial to the epicardial surface. In contrast, PET reconstructions from patients with nontransmural infarction exhibited marked spatial heterogeneity of myocardial accumulation of palmitate. In some patients, only marked heterogeneity was observed; in others, regions of heterogeneous accumulation of isotope were interspersed with regions of apparent thinning of the palmitate uptake zone. In a few patients, homogeneous depression was seen similar to that more typical of transmural infarction. The extent of the regions with depressed accumulation of tracer appeared to be more modest and more heterogeneous than that of the regions typical of transmural infarction. Overall, the extent of myocardial infarction, assessed by PET, was less among patients with nontransmural compared to those with transmural infarction. In addition, the distribution of palmitate was more heterogeneous in grossly normal zones.

Previous studies have shown substantial variability in the sensitivity of thallium imaging for detecting myocardial infarction, depending on the time of the imaging after infarction, the location, intramural extent, and the size of the infarct. A sensitivity of almost 100% has been achieved among patients with transmural infarction imaged within the first 6-12 hours after onset of infarction. However, when imaging is delayed by 24 hours, sensitivity is dramatically decreased. Unfortunately, the high early sensitivity may reflect scintigraphic changes due to ischemia rather than infarction per se. In the present study, $^{201}$Tl imaging was performed late. However, tomographic studies and $^{201}$Tl scintigraphy were performed at approximately the same interval after infarction. Three of the seven patients with normal $^{201}$Tl scintigrams were imaged before a PET study that was positive. The greatest decrease in sensitivity of $^{201}$Tl scintigraphy occurs within the first 24-36 hours after infarction. Further delays do not appear to change sensitivity markedly. Thus, only slight differences in sensitivity would be expected among patients studied by $^{201}$Tl scintigraphy on the fifth compared to the tenth day after infarction.

Autopsy data indicate a wide spectrum of anatomic abnormalities in hearts of patients who die with nontransmural infarction, ranging from relatively small scars that are limited to the inner one-third or one-half of the myocardium to large circumferential zones of necrosis involving 50-70% of the inner layer of the left ventricle. Some subjects who exhibit only ST-T abnormalities on the ECG exhibit typical transmural lesions at autopsy. It is not surprising that the spectrum of abnormalities detectable by PET in patients with electrocardiographically identified nontransmural infarction is wide.

Heterogeneous accumulation of palmitate in patients with nontransmural infarction reflects interspersed regions of normal and abnormal accumulation. In addition, the almost continuous variation of activity even within small zones may well reflect closely admixed normal and abnormal cells. The gross, spatial heterogeneity is probably a reflection of the consequences of the multivessel coronary artery disease so common among patients with nontransmural infarction.

Tomographic reconstructions are affected by cardiac motion during data collection. Wall motion tends to blur borders between metabolically normal and abnormal regions. Movement within the plane of section and movement in and out of the plane of the section influences results. The consequent blurring results in spatial averaging, and tends to "smooth" the data, making it appear more homogeneous. The consistently high accumulation of palmitate in normal regions and relatively uniform depression of accumulation of palmitate within abnormal regions of transmural infarcts are consistent with completed, homogeneous infarction adjacent to myocardium remaining viable with normal metabolism when the patient is at rest. Patients with nontransmural infarction often have a clinically stuttering syndrome, with small extensions of infarction after the initial insult. Thus, in hearts of patients with nontransmural infarction, zones of completed infarction may be interspersed with regions of evolving or very recent infarction and with regions remaining at high risk for subsequent infarction. This heterogeneous distribution appears to be reflected by corresponding heterogeneity in tomographic images.

Our data indicate significant differences in the appearance of PET reconstructions from patients with

**Table 4. Relative Sensitivity of Positron-emission Tomography and Thallium-201 Imaging**

<table>
<thead>
<tr>
<th>PET</th>
<th>Thallium</th>
<th>$p &lt; 0.05$</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sensitivity (abnormal/total)</td>
<td>96% (23/24)</td>
<td>61% (11/18)</td>
</tr>
<tr>
<td>Specificity (abnormal/total)</td>
<td>80% (2/10)</td>
<td>90%* (1/10)</td>
</tr>
</tbody>
</table>

*Previously published from this institution.

Abbreviation: PET = positron-emission tomography.
transmural compared with nontransmural myocardial infarction, but they do not permit an assessment of the value of PET for separating patients with unstable angina from those with nontransmural infarction. A recent study performed at our institution indicates that the initial accumulation of palmitate is comparable for regions of canine myocardium supplied by normal and partially obstructed coronary arteries. The region supplied by the partially obstructed coronary artery cannot be distinguished from normally perfused myocardium with the first transaxial tomographic reconstruction. However, when time-activity curves are constructed for normally perfused and underperfused regions, clearance of palmitate is depressed in ischemic but still viable regions supplied by partially occluded coronary arteries. Thus, ischemic, infarcted and normal myocardium are distinguished by dynamic tomographic studies that should be applicable to patients as rapid scanning instrumentation becomes available.

Our findings indicate that PET, performed after the i.v. injection of 11C-palmitate, is a sensitive and specific means of detecting and characterizing nontransmural and transmural myocardial infarction. Transmural and nontransmural myocardial infarctions are distinctly different. Transmural infarction exhibits homogeneous, intense depression of the accumulation of palmitate. Nontransmural infarction exhibits marked heterogeneity in the accumulation of palmitate. The spectrum of tomographic manifestations of nontransmural infarction reflects the spectrum of morphologic abnormalities. The present findings suggest that PET with 11C-palmitate will be useful not only in detecting nontransmural infarction and its definitive diagnosis, but also in characterizing its natural history and response to interventions designed to salvage jeopardized regions within the overall zone at risk.

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