The Clinical Estimation of Acute Myocardial Infarct Size with $^{99m}$Tc Technetium Pyrophosphate Scintigraphy

D. Norman Sharpe, M.B., Elias H. Botvinick, M.D., David M. Shames, M.D., Anne Norman, Kanu Chatterjee, M.B., and William W. Parmley, M.D.

SUMMARY We evaluated scintigraphic techniques in estimating infarct size. In 26 patients with acute transmural myocardial infarction, $^{99m}$Tc Technetium pyrophosphate (TcPYP) infarct scintigraphy, gated cardiac blood pool scintigraphy and 201-Thallium (201-Tl) perfusion scintigraphy were performed. Invasive hemodynamic measurements were obtained and serial venous blood specimens taken for measurement of total and MB creatine phosphokinase (CPK). Infarct size was estimated from the area of abnormal TcPYP uptake, the extent of reduced 201-Tl uptake, the percentage of abnormally contracting segments, and serial enzyme measurements. Left ventricular ejection fraction (LVEF) and stroke work index (LVSWI) were calculated.

IN RECENT YEARS, considerable interest has centered on methods aimed at determining the extent of acute myocardial infarction. Until such methods are reliably established, it will remain difficult to assess the effects of therapeutic interventions designed to reduce or minimize myocardial damage and thus improve prognosis after infarction.

$^{99m}$Tc Technetium pyrophosphate (TcPYP) scintigraphy is a sensitive and relatively specific indicator of the presence of acute infarction.1, 2 Experimental studies have shown good correlation between acute infarct size as judged from the area of abnormal myocardial uptake of radionuclide on the TcPYP scintigram and the extent of histologic infarction or gross infarct weight.3, 4 We sought to assess the value of TcPYP scintigraphy in determining infarct size clinically, relating it to other scintigraphic techniques, enzymatic estimates of infarct size, and indices of left ventricular function.

Materials and Methods

We studied 26 patients admitted to the Coronary Care Unit with acute transmural myocardial infarction (MI). The diagnosis of MI was based on the clinical history, serial electrocardiographic abnormalities with the development of Q waves > 0.04 sec, and serum MB creatine phosphokinase (CPK) elevation. Historical, electrocardiographic (ECG) and enzymatic evidence of prior infarction, as well as ECG evidence of left ventricular hypertrophy5 were sought.

Informed consent was obtained and the initial scintigrams and invasive hemodynamic measurements were carried out in the CCU within 72 hours of admission.

Myocardial infarct scintigraphy was performed 24–48 hours after admission, following the intravenous administration of 15 mCi of TcPYP manufactured according to the method of Huberty and co-workers.7 Anterior, 45° left anterior oblique and left lateral projections, each taken to 300,000 counts, were obtained on Polaroid prints at least two hours after TcPYP administration using a portable Ohio Nuclear Series 120 or Searle Pho-Gamma IV scintillation camera with a high resolution collimator. TcPYP scintigrams were evaluated by two independent observers who had no knowledge of the clinical history or other laboratory findings. Scintigrams were graded on a scale of 0–4 based on the level of radioactivity in the cardiac region: zero represented background activity; 1+ represented slight, indefinite increase in activity beyond background; 2+ represented definite increase in activity less intense than bone; 3+ represented activity equal to bone; and 4+ represented activity greater than bone. Scintigrams graded 2–4+ were called abnormal and the region of radionuclide localization was determined after evaluation of all projections. The observers agreed totally on the TcPYP area of abnormality in 19 of 26 patients with small regions of disagreement amounting to less than 10% of measured infarct size in the remaining seven patients. The area of involvement in the seven controversial cases was always clarified by discussion after review of the scintigrams in multiple projections. One observer reread all images two months after the initial reading and showed only minor discrepancies in three cases when compared to his original evaluation. The area of infarction was calculated by planimetry of the area of abnormal TcPYP activity for anterior infarcts, using the projection which demonstrated the largest area of involvement. For inferior infarcts, the product of linear measurements obtained from orthogonal projections (anterior and left lateral) was used. A radiative grid source of known dimensions was used to provide a magnification correction factor for the scintigrams. The TcPYP infarct area thus obtained was expressed in absolute terms (cm²).

Gated cardiac blood pool scintigraphy using $^{99m}$Tc Technetium albumin (TcAlb)6 was performed with the same cameras 24 hours after TcPYP scintigraphy. TcAlb was prepared according to the method of Dworkin and

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Gutkowski. An ECG and phonocardiograph were interfaced to an ECG gate (Riverside Bioengineering). Counts were accumulated at 50 msec intervals at end diastole, defined during each cardiac cycle by the electrocardiographic R wave peak, and end systole, defined by the onset of the second heart sound, to a total of 800,000 counts. Thirty degree right anterior oblique (RAO) and 60° left anterior oblique (LAO) projections were completed within 30 minutes and displayed on Polaroid prints and microdot transparency. End-diastolic and end-systolic outlines of the left ventricle in both projections were superimposed using stationary reference points according to an adaptation of the method of Rigo et al. and the circumferential length of abnormally contracting segments measured. Less than 20% inward motion toward the diastolic major or minor axis of the ventricle was defined as abnormal. This measurement was chosen from the current literature as the value most sensitive to and specific for significant contraction abnormalities. The extent of abnormally contracting segments was expressed as a percentage of the total circumference of the ventricle in either projection excluding aortic and mitral valve planes. The mean percentage of abnormally contracting segments from RAO and LAO projections was taken as an index of infarct size. Left ventricular ejection fraction (LVEF) was calculated from the RAO view using the single plane area-length formula. In a prior study, we have documented the accuracy of this method and its reproducibility.

Hemodynamic measurements were obtained on the same day as the gated cardiac blood pool scan via a triple lumen balloon-tipped Swan-Ganz catheter with thermistor introduced through an antecubital vein and floated into the pulmonary artery. Left ventricular filling pressure (LVFP) was determined from the occluded pulmonary artery pressure using Statham P23Db transducers positioned at mid-chest level and recorded on a Gould Brush 440 pressure ink recorder. Arterial blood pressure was measured by cuff, and cardiac output determined in triplicate by the thermodilution technique. Cardiac index was calculated as CI = cardiac output/body surface area in L/min/m². Left ventricular stroke work index was calculated as

\[ LVSWI = SI \ (\text{MSP-LVFP}) \ 0.0136 \ \text{g-m/m}^2, \text{ where SI (stroke index)} = \frac{CI}{\text{heart rate}}, \text{ and MSP (mean systolic arterial pressure)} = \frac{2}{3} (\text{systolic-diastolic}) + \text{diastolic pressure}. \]

201-Thallium (201-Tl) myocardial perfusion scintigraphy was performed between days 4 and 7. Two mCi of 201-Tl was injected intravenously and scintigraphy performed using an Ohio Nuclear Series 120 or Searle Pho-Gamma IV scintillation camera and a converging collimator with a 20% window centered at 75 keV. Imaging to 200,000 counts was performed in the anterior, 45° left anterior oblique and left lateral projections. Evaluation of perfusion scintigrams was made by an independent observer. Each projection was divided into seven equal circumferential zones, each zone being assessed for reduced 201-Tl uptake. The total number of zones showing reduced 201-Tl uptake was obtained by summation from all projections and taken as another index of infarct size. In a previous study we documented the accuracy and reproducibility of 201-Tl image interpretation.

Serial venous blood specimens were taken from the time of admission for measurement of serum total creatine phosphokinase (CPK) by a fluorimetric method. MB-CPK was measured by an electrophoretic method. Specimens were taken every two hours for 24 hours, and then every six hours for 48 hours. Infarct size was estimated from the serial enzyme measurements using an individualized disappearance rate constant (kd) and other constants for total CPK and MB-CPK as recently proposed. A further estimate of infarct size was obtained by integration of the completed total CPK-time curve.

**Results**

Of the 26 patients studied, 11 had ECG evidence of anterior infarction and 15 inferior infarction. Four patients had a documented prior MI and six patients had ECG evidence of left ventricular hypertrophy. The data for all patients is presented in tables 1 and 2. TcPYP scintigrams were abnormal in all 26 patients. Discrete uptake of radionuclide was localized in all cases to regions which corresponded anatomically with the electrocardiographic location of the infarct.

**Table 1. Scintigraphic and Hemodynamic Data**

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<th>Body weight (kg)</th>
<th>Infarct location</th>
<th>TcPYP infarct area (cm²)</th>
<th>201-Ti scintigraphy zones with reduced perfusion</th>
<th>Gated cardiac blood pool scintigram (mean RAO + LAO)</th>
<th>LVSWI (gcm/m²)</th>
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*LVH, MI.*

**Abbreviations:** A = anterior; I = inferior; Ant = anterior; LAO = left anterior oblique; LLAT = left lateral.
limited availability of 201-Tl allowed us to image only 17 of the 26 patients by this technique. A perfusion defect was seen on the 201-Tl scintigram in each of the 17 patients studied. In each case, the site of the 201-Tl deficit corresponded with that of abnormal TcPYP uptake seen on the TcPYP scintigram. Comparison of corresponding projections in individual patients with anterior infarcts showed that whereas the TcPYP abnormality appeared largest when seen on film in the anterior projection, the 201-Tl perfusion defect was most apparent when the infarction was seen in profile in the LAO or left lateral projection (fig. 1). TcPYP infarct and 201-Tl perfusion scintigrams in patients with inferior infarction correlated better in corresponding projections as the infarction was profiled by both scintographic techniques (fig. 2). TcPYP infarct area correlated with the total number of zones in all projections of the 201-Tl scintigram which showed reduced perfusion ($r = 0.66$) (fig. 3). This correlation was not improved when anterior or inferior infarctions were considered separately or when patients with prior infarcts were excluded.

TcPYP infarct area correlated weakly with the percentage of abnormally contracting segments on the gated cardiac blood pool scintigram ($r = 0.64$) (fig. 4).

A negative correlation was found between TcPYP infarct area and both LVSWI ($r = -0.73$) (fig. 5) and LVEF ($r = -0.58$) (fig. 6).

TcPYP infarct area did not correlate with infarct size estimated from either cumulative total CPK release or MB-CPK release, or from the integrated total CPK-time curve. Cumulative CPK release did not correlate with the percentage of abnormally contracting segments or with LVSWI or LVEF.

**Discussion**

Our study demonstrated that in an optimal population with well defined acute transmural infarction, infarct size as

![Figure 1: Anterior infarction. Shown are the TcPYP (top row) and 201-Tl (bottom row) scintigrams from patient 14 with anterior MI. The TcPYP scintigram shows a doughnut pattern of abnormal myocardial uptake of radionuclide in the anterior projection. The 201-Tl scintgram shows reduced perfusion in the region of the anterior wall, apex and septum most evident with the infarct profiled in the LAO and left lateral projections.](image1)

![Figure 2: Inferior infarction. Shown are the TcPYP (top) and 201-Tl (bottoms) scintigrams from patient 21 with inferior MI. The infarct is profiled by both imaging techniques in the left lateral projection.](image2)
judged by the area of myocardial uptake of radionuclide on the TcPYP scintigram correlated with other scintigraphic estimates of the size of infarction and with indices of LV function but not with enzymatic estimates of infarct size.

Discrete myocardial uptake of TcPYP is a sensitive and relatively specific indicator of the presence of infarction and correlates well with the electrocardiographic localization of infarction. Previous animal studies have shown a good correlation between TcPYP infarct area and the extent of histologic infarction or gross infarct weight. Experimental TcPYP infarct image radioactivity in vivo and at post-mortem examination has also been positively correlated with gross infarct weight in animals with hemorrhagic infarctions of small to moderate size. A further experimental study reported a significant direct relationship between the maximum precordial TcPYP image radioactivity and the histologic extent of infarction following transient coronary occlusion. However, TcPYP radioactivity varied inversely with histologic infarct size following permanent coronary occlusion. Morphologic studies have shown that in dogs subjected to proximal left anterior descending coronary occlusion resulting in large anterior infarcts, uptake of TcPYP was concentrated peripherally in a doughnut pattern in association with selective peripheral accumulation of myocardial calcium. Further experimental studies utilizing 43-K, 201-T1 and radioactive microspheres have demonstrated that myocardial perfusion is important in determining TcPYP distribution in regions of myocardial necrosis. It appears then that the intensity of the TcPYP infarct image is related in part to the degree to which the infarcted myocardial cells are perfused and not entirely to the size or density of infarction. Large infarctions concentrate TcPYP radioactivity peripherally as a result of maintained native or collateral flow while central poorly perfused regions fail to accumulate such large amounts of TcPYP. Thus, a small infarct which is relatively well perfused may be demonstrated on the TcPYP scintigram as a small region of intense radioactivity, while a large infarct with a poorly perfused central region may demonstrate a doughnut pattern. Indeed, several of our patients with large anterior infarcts had this characteristic pattern with central infarct radioactivity less than that seen peripherally (figs. 1 and 7A).

The explanation relating TcPYP image radioactivity to histologic infarct size also appears to be relevant to the relationship of serum enzyme activity and infarct size. Roe et al. found it impossible to distinguish small from large histologic infarcts by serum CPK enzyme measurements in
dogs following permanent circumflex coronary artery occlusion. A very poor correlation between enzymatic estimates and histologic measurements was found, although there was significant improvement in the correlation when animals with larger infarcts were excluded. The loss of correlation produced by the larger infarcts resulted from relatively less total enzyme release in the larger compared to the smaller infarcts. Again, as with reduced TcPYP accumulation in the center of large infarcts, this is possibly related to diminished central perfusion which affects the amount of enzyme released into the peripheral circulation. The application of various methods in enzymatic estimates of infarct size, all of which produced an overestimate of histologic infarction, did not improve the correlation, nor was the correlation improved by the use of isoenzyme data. The lack of correlation between TcPYP infarct area and enzymatic estimates of infarct size is probably related in part to variation in blood flow to infarcts of different sizes. In figure 7, TcPYP scintigrams and CPK data from two patients with anterior infarcts are demonstrated. Patient 7 had a large doughnut type infarct on the TcPYP scintigram, considerably larger in area than that of patient 20. However, enzymatic estimates of infarct size in patient 20 are approximately three times those in patient 7.
since no better correlation was obtained through exclusion of patients with large infarct areas (20 cm² or more). Another important factor is that serial enzyme measurements provide an index of myocardial mass infarcted, rather than infarct area as estimated from the TcPYP scintigram, although exclusion of patients in our series with ECG evidence of left ventricular hypertrophy did not improve the correlation. None of our patients in whom complete CPK curves were obtained were subject to electrical defibrillation, and intramuscular injections were avoided in all patients.

Henning et al., 27 in a clinical study on patients with anterior infarction, reported a good correlation between TcPYP infarct area and infarct size estimated from serial serum CPK values and the integrated complete CPK curve. When we divided our patient population into those with anterior and those with inferior infarction, no new correlations between scintigraphic or enzymatic infarct size or parameters of left ventricular function could be established for the separate groups. A further clinical study 28 demonstrated a correlation between infarct size calculated from cumulative total CPK release and postmortem measurement of infarct size in 15 patients. This finding, although supported by the initial experimental data of Shell et al., 19 disagrees with the experimental findings of Roe et al. 29 reviewed above. The apparent discrepancies between studies should not be unexpected. The factors governing TcPYP accumulation are quite different from those of CPK release and these factors likely explain differences between infarct size measured scintigraphically and that measured enzymatically. While TcPYP infarct size, as obtained here, is primarily a measure of infarct area, enzymatic infarct size is influenced significantly by infarct mass and volume and is strongly determined by infarct density. Any attempt to relate scintigraphic infarct size to infarct density in patients would have to relate to prognosis as illustrated by Bleifeld et al. 28 as well as by Willerson and co-workers 29 who have shown that TcPYP infarct size correlates with the presence or absence of congestive heart failure and Holman et al. 31 who have shown that TcPYP infarct size helps predict subsequent mortality.

Thallium-201 and TcPYP imaging in the dog 32 has shown that both techniques correlate with histologic estimates of infarct size. However, TcPYP scintigraphic abnormalities in corresponding projections were consistently larger than 201-T1 perfusion defects. We found that for anterior infarcts the TcPYP abnormality was usually larger in the anterior projection where it was seen en face, whereas the 201-T1 perfusion defect was largest in profile in the LAO or left lateral projection. Inferior infarcts were generally more comparable in corresponding projections of TcPYP and 201-T1 scintigrams, being seen in profile with both techniques. An alternate explanation for the larger size of the TcPYP abnormality may relate to the presence of a kind of border zone with scattered necrosis and TcPYP uptake but also possessing a significant amount of viable, perfused myocardium. Superimposition of infarct and perfusion scintigrams did not demonstrate a classic underperfused, noninfarcted peri-infarction border zone as defined experimentally. 35 However, in a dual isotope study, Parke et al. 34 did demonstrate some cases with 201-T1 defects greater than TcPYP abnormalities, suggesting a marginal, potentially reversible ischemic border zone. Nevertheless, it seems unlikely that demonstration of the border zone defined experimentally 35 could be achieved by in vivo imaging using these techniques. The borders of the abnormal scintigraphic region are more difficult to delineate in the cold spot perfusion image than in the hot spot infarct image. Depending on the size and location of the perfusion defect and the projection used, surrounding normally perfused myocardium may make definition of the limits of the perfusion defect difficult or mask it altogether. On the other hand, some normal cardiac regions at the base and apex may show relatively diminished 201-T1 uptake. For these reasons, we did not attempt quantitative assessment of the 201-T1 infarct image, but evaluated the scintigrams grossly and visually as generally performed and reported in the literature. 36, 37, 38 Additionally, visualization of perfusion defects following infarction appear to be time-dependent. 39 Had we performed 201-T1 perfusion scintigraphy earlier, we might have seen larger defects. Nonetheless, the correlation of TcPYP infarct area with the overall extent of a 201-T1 perfusion defect as summed from all projections is supportive evidence that TcPYP infarct area is a valid estimate of infarct size. The gated cardiac blood pool scintigram and hemodynamic findings provided further anatomic and functional evidence for the validity of TcPYP infarct area as such an estimate.

We sought to evaluate the ability of the TcPYP infarct scintigram to size acute myocardial infarction in man and chose a study population optimal for that technique. The study has a high sensitivity and generally reveals intense, discrete abnormalities in patients with acute transmural infarction. 1, 2 The scintigraphic findings and our results might differ in patients with subendocardial infarction or in a group of patients with a less clear clinical event.

We did not employ computer techniques in the analysis of
any scintigraphic study as we did in our initial attempt to size infarction with TcPYP in animals.4 We felt that a visual, manual method would be more desirable and more widely applicable than a complex mechanized analysis. In fact, the method for evaluating TcPYP infarct size seems valid and showed good inter and intra-observer agreement. There is no good objective, quantitative method for determining areas of 201-Tl perfusion abnormality. Computer applications to the 201-Tl scintigram have largely been for display purposes and an accepted objective method of quantitating the area of 201-Tl scintigram abnormality is needed. Clearly, our technique has the inherent geometric error arising from determining the extent of a three-dimensional infarct from a two-dimensional image. Threedimensional reconstruction8 may allow a more accurate definition of infarct size by scintigraphic methods bringing an element of volume to what is presently an area measurement.

The relatively long half-life of 99m Technetium and the fact that positive scans are not seen until 12 to 24 hours after the onset of symptoms limits the application of this technique for serial imaging of patients and judging the effect of therapeutic intervention. Nevertheless, the diagnostic value of TcPYP infarct scintigraphy is established and the present data suggest it has some value in estimating clinical infarct size.

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