The Application of Radionuclide Infarct Scintigraphy to Diagnose Perioperative Myocardial Infarction following Revascularization


SUMMARY To evaluate the application of radionuclide infarct scintigraphy to diagnose myocardial infarction after revascularization, we obtained postoperative technetium 99m pyrophosphate myocardial scintigrams, serial electrocardiograms and CPK-MB isoenzymes in ten control and 51 revascularized patients. All control patients had negative electrocardiograms and scintigrams, but eight had positive isoenzymes. Eight revascularized patients had positive electrocardiograms, images and enzymes and two had positive scintigrams and enzymes with negative electrocardiograms. Thirty-four patients with negative electrocardiograms and scintigrams had positive isoenzymes; in only seven patients were all tests negative. Our data suggest radionuclide infarct scintigraphy is a useful adjunct to the electrocardiogram in diagnosing perioperative infarction. The frequent presence of CPK-MB in postoperative patients without other evidence of infarction suggests that further studies are required to identify all factors responsible for its release.

IN ADDITION TO THE RELIEF OF ANGINA, the salvage of functionally depressed but viable myocardium and the prolongation of life by prevention of myocardial infarction are now being considered as possible benefits of coronary artery bypass graft surgery. Although the acute mortality of such surgery has been acceptably low, the reported incidence of electrocardiographically determined perioperative myocardial infarction has varied from 2 to 35%. The mortality of perioperative infarction is probably less than that of infarction occurring in the population at large. However, the loss of functional myocardium associated with such an event, and its subsequent morbidity, must be weighed against the expected benefits of surgery if the clinician is to decide whether surgery is appropriate in an individual patient.

In the absence of prior infarction the appearance of new Q waves on the electrocardiogram has been confirmed as a relatively specific indication of perioperative infarction. However, in patients with previous infarction, or those demonstrating nonspecific ST-T changes, it is difficult to document the presence and extent of perioperative myocardial damage because of changes in the electrocardiogram and elevations in serum enzymes which may occur secondary to surgery itself. Recent studies of radionuclide scintigraphy with technetium 99m pyrophosphate have demonstrated that its accumulation in the myocardium is a sensitive and specific indicator of acute myocardial damage in the nonsurgical setting. Therefore, we evaluated this new scintigraphic technique for the detection of perioperative myocardial infarction, and compared the scintigraphic findings with the analysis of changes in serial electrocardiograms and serum CPK-MB.

Methods

Patient Population

We studied 61 patients referred to our medical center for evaluation and surgical treatment of cardiac disease. Ten patients who had normal coronary arteriograms, or who were premenopausal females without clinical or laboratory evidence of myocardial ischemia, served as a control population. They were placed on cardiopulmonary bypass for repair of calcific aortic stenosis (3 patients), chronic aortic insufficiency, mixed mitral valve disease (2 patients), chronic mitral insufficiency, aortic and mitral insufficiency, mitral stenosis with partial anomalous pulmonary venous return and atri al septal defect. Fifty-one patients with documented coronary artery disease had revascularization procedures. In this latter group, two patients also had valve replacement, one aortic and one mitral.

Protocol

Within twenty-four hours before surgery we obtained a standard 12-lead electrocardiogram and blood samples for determination of CPK-MB in all patients. One control and 15 revascularization patients were examined with 99m technetium pyrophosphate scintigrams preoperatively. During surgery cardiopulmonary bypass was initiated by cannulation of the superior vena cava through a right atrial incision with return via the ascending aorta or femoral artery. Perfusion was maintained using an Olson pump, with either a Harvey or Bently bubble oxygenator and a PALL filter placed in the arterial line. Cardioplegia was accomplished using either iced potassium coronary washout or electrical fibrillation and surgery was performed in the presence of patient hypothermia to approximately 28°C. Intermittent aortic cross-clamping was employed to perform the distal anastomoses during all the revascularization procedures. Left ventricular venting was performed via the pulmonary veins and left atrium or by direct left ventricular vent. Following the operation electrical rhythm was restored spon-
Table 1. Criteria for Interpretation of Technetium 99m Sestamibi Myocardial Scintigrams

<table>
<thead>
<tr>
<th>Intensity grade</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>No accumulation of radionuclide in the myocardium.</td>
</tr>
<tr>
<td>1+</td>
<td>Slight, indefinite, accumulation of radionuclide in the cardiac region.</td>
</tr>
<tr>
<td>2+</td>
<td>Definite accumulation of radionuclide in the region of the myocardium with activity less than that of the ribs.</td>
</tr>
<tr>
<td>3+</td>
<td>Definite accumulation of radionuclide in the region of the myocardium with activity equal to that of the ribs.</td>
</tr>
<tr>
<td>4+</td>
<td>Definite accumulation of radionuclide in the region of the myocardium with activity greater than that of the ribs.</td>
</tr>
</tbody>
</table>

**Distribution**
- Diffuse: Generalized radionuclide accumulation in the cardiac region apparently involving all aspects of the heart - without evidence of the ventricular cavity.
- Discrete: Radionuclide accumulation in a specific region of the myocardium.

**Final interpretation**
- NEGATIVE: Intensity grade 0-1+ regardless of distribution.
- DIFFUSE: Diffuse distribution regardless of intensity grade.
- POSITIVE: Intensity grade 2-4+ with discrete radionuclide distribution.

Taneously or by application of 20–60 watt-sec shocks to the epicardium. The patients were returned to the Intensive Care Unit where a standard 12-lead electrocardiogram was obtained and serial electrocardiograms were performed daily for the next three days. Blood samples for CPK-MB isoenzymes were obtained 6 to 8 hours following the induction of anesthesia, and then every six hours for the next two days. Two to five days after surgery a technetium 99m pyrophosphate myocardial scintigram was obtained in all patients.

The preoperative and serial postoperative electrocardiograms were analyzed by one blinded observer. The diagnosis of perioperative infarction by electrocardiogram (+ECG) was made on the basis of the appearance of Q waves greater than or equal to 0.04 sec which were not present on the preoperative tracing. Patients who did not develop Q waves (−ECG) were subdivided into those with ST depression greater than 2 mm and/or symmetrical T wave inversion lasting longer than 24 hours (−ECG (+ST)) and those with nonspecific or no ST-T abnormalities (−ECG(−ST)). Total serum CPK was determined by the method of Rosalki. The percent CPK-MB was obtained by agar gel electrophoresis, converted to serum concentration, and called +MB if present, and −MB if absent.

All myocardial scintigrams were obtained using a Searle Pho-Gamma IV camera with a low energy all purpose collimator two to three hours following the peripheral intravenous injection of 15 mCi of technetium 99m stannous pyrophosphate. Images were taken to 300,000 counts using a 20% window in the anterior, left anterior oblique, and left lateral projections. The scintigrams were independently analyzed by two observers, neither of whom had knowledge of the electrocardiographic or enzyme data. The scintigrams were categorized according to the criteria listed in Table 1. Scintigrams were graded as negative (−PYP) if there was no (0), or slight indefinite (+1+) activity in the cardiac region, and positive (+PYP) if there was definite, discrete myocardial isotope localization. The diffuse pattern of radioisotope uptake was considered separately due to its non-specific nature. Between the two observers there was no disagreement in the interpretation of the scintigraphic images.

The results for the electrocardiograms, enzymes and scintigrams were compared and differences between subsets of patients were evaluated by unpaired t-tests or analysis of variance.

**Results**

**Preoperative Studies**

Prior to surgery none of the ten control patients had electrocardiographic evidence of prior myocardial infarction, ST-T wave changes suggestive of acute ischemia, or CPK-MB present in their serum. One patient had a preoperative myocardial scintigram which was negative.

Twenty-five of the 51 patients who had coronary artery revascularization demonstrated electrocardiographic evidence of a prior myocardial infarction. None of the revascularization patients had CPK-MB present in the preoperative blood sample. Nine preoperative myocardial scintigrams were negative, while six displayed a diffuse pattern of uptake in the cardiac region. Of the six patients displaying the diffuse pattern, two had symptoms consistent with a diagnosis of crescendo angina. One of these sustained a documented subendocardial infarction two months prior to surgery, and one

Table 2. Demographic, Preoperative and Postoperative Data for Control Patients

<table>
<thead>
<tr>
<th>Patient no.</th>
<th>Age</th>
<th>Sex</th>
<th>Preoperative diagnosis</th>
<th>Coronary arteryogram</th>
<th>Operation</th>
<th>ECG preop/postop</th>
<th>Scintigram preop/postop (day postop)</th>
<th>Peak postop CPK-MB (IU/ml)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>71</td>
<td>M</td>
<td>AS</td>
<td>normal</td>
<td>AVR</td>
<td>−</td>
<td>ND (−3)</td>
<td>45</td>
</tr>
<tr>
<td>2</td>
<td>64</td>
<td>M</td>
<td>AS</td>
<td>normal</td>
<td>AVR</td>
<td>−</td>
<td>ND (−3)</td>
<td>24</td>
</tr>
<tr>
<td>3</td>
<td>70</td>
<td>M</td>
<td>AS</td>
<td>normal</td>
<td>AVR</td>
<td>−</td>
<td>ND (−3)</td>
<td>36</td>
</tr>
<tr>
<td>4</td>
<td>54</td>
<td>M</td>
<td>AI</td>
<td>normal</td>
<td>AVR</td>
<td>−(+ST)</td>
<td>ND (−3)</td>
<td>0</td>
</tr>
<tr>
<td>5</td>
<td>45</td>
<td>F</td>
<td>MS,MR</td>
<td>normal</td>
<td>MVR</td>
<td>−(+ST)</td>
<td>ND (−5)</td>
<td>25</td>
</tr>
<tr>
<td>6</td>
<td>48</td>
<td>F</td>
<td>MS,Anom.</td>
<td>no</td>
<td>MVR, PVD</td>
<td>−(+ST)</td>
<td>ND (−4)</td>
<td>50</td>
</tr>
<tr>
<td>7</td>
<td>38</td>
<td>F</td>
<td>MR</td>
<td>no</td>
<td>MVR</td>
<td>−(+ST)</td>
<td>ND (−3)</td>
<td>100</td>
</tr>
<tr>
<td>8</td>
<td>27</td>
<td>F</td>
<td>AI, MR</td>
<td>no</td>
<td>MVR, MVR</td>
<td>−(+ST)</td>
<td>ND (−5)</td>
<td>17</td>
</tr>
<tr>
<td>9</td>
<td>20</td>
<td>F</td>
<td>ASD</td>
<td>no</td>
<td>Repair</td>
<td>−(+ST)</td>
<td>ND (−3)</td>
<td>0</td>
</tr>
<tr>
<td>10</td>
<td>46</td>
<td>F</td>
<td>MS,MR</td>
<td>no</td>
<td>MVR</td>
<td>−</td>
<td>ND (−3)</td>
<td>16</td>
</tr>
</tbody>
</table>

Abbreviations: AS = aortic stenosis, AI = aortic insufficiency; MS = mitral stenosis, MR = mitral regurgitation, Anom. PVD = anomalous pulmonary venous drainage, ASD = atrial septal defect; ND = not done, − = negative, −(+) = new Q wave but ST depression greater than 2 mm and/or symmetrical T wave inversion lasting longer than 24 hours.
had a history of three documented infarctions, the last occurring seven years before his operation. A third patient had a one month history of gradually increasing angina without a documented infarction and two other patients had stable angina persisting two months and four months after documented infarctions. The last such patient was asymptomatic, but had severe, greater than 95%, narrowing of the left main coronary artery.

Postoperative Studies

The mean cardiopulmonary bypass time for the revascularization patients was longer than for the control group (117 ± 6 min vs 74 ± 11 min, P < 0.02). However, the mean total aortic cross-clamp time was shorter for revascularization patients (43 ± 3 min vs 69 ± 5 min, P < 0.02), reflecting the technique of intermittent cross-clamping.

In tables 2 and 3, the pre and postoperative ECG and scintigram interpretations and peak postoperative CPK-MB values are listed together with the pertinent clinical and laboratory data for control and revascularization patients, respectively. Note that no control patient had a +ECG or +PYP, but eight of ten, or 80%, had +MB. In the revascularization group eight patients had +ECG, 10 had +PYP and 44 (or 86%) had +MB.

Table 4 compares the postoperative CPK-MB data with that of the electrocardiogram. In the control patients the mean peak CPK-MB was 31 units, with a range of 0 to 100. The revascularization patients first were divided into those with a −ECG and those with a +ECG. Among the former the mean peak CPK-MB was 53 units with a range of 0 to 378, while among the latter the mean peak CPK-MB was 115 units, with a range of 12 to 297. When tested by analysis of variance, the mean peak CPK-MB level of the revascularization patients with a +ECG was not significantly different from that of the other the revascularization patients with a −ECG or the control patients. Also, there was no significant difference between the means for the control patients and the revascularization patients who had a −ECG. To further analyze the results for the revascularization patients, the data for the −ECG patients were divided into those with (+ST) and those without (−ST) ST depression greater than 2 mm and/or symmetrical T wave inversion lasting more than 24 hours.
values for these groups (+ECG: mean 115, range 12-297; -ECG (+ST): mean 53, range 0-245; -ECG (-ST): mean 53, range 0-378). Table 5 presents the means of the hours post induction of anesthesia of the blood samples containing the peak CPK-MB and of the last samples containing CPK-MB for the revascularization patients. Although there was a tendency for the peak value to occur later and for the isoenzyme to persist longer in the +ECG patients, there were no significant differences between these groups in the timing of either the peak CPK-MB or the last blood sample containing the isoenzyme.

Figure 1 demonstrates the preoperative and negative postoperative myocardial scintigrams obtained in a patient (table 3, patient 26) who underwent coronary artery revascularization. Note the relative increase in uptake of radioisotope in the sternal region compared to that of the ribs on the postoperative images. Presumably, this is due to bone reparative processes following the sternal splitting incision. Note also the absence of radionuclide in the region of the myocardium.

Figure 2 illustrates a typical +PYP image in patient 17, who sustained an inferopapal infarction. The discrete accumulation of the radionuclide is evident and corresponds to the appearance of new Q waves shown in the inferior leads of the electrocardiogram. Note also that in this patient the peak CPK-MB was only 34 units.

Figure 3 left is a positive scintigram showing an inferior wall infarction in revascularization patient 18. In figure 3 right the preoperative electrocardiogram of this patient is shown together with the postoperative electrocardiogram obtained on the same day the scintigram was performed, postoperative day three. Although there has been a right axis shift, inferior T wave changes, and loss of precordial R wave voltage, the tracing is not diagnostic of an acute inferior infarction. A tracing obtained on the fifth postoperative day did demonstrate Q waves consistent with the damage documented on the scintigrams up to two days earlier. This patient had continued chest pain after surgery and developed progressive heart failure. Repeat catheterization was performed sixteen days after his operation, which demonstrated akinesis of the inferior wall and occlusions of grafts to the posterior descending, left anterior descending and diagonal coronary arteries. Peak CPK-MB was 12 units.

Two patients had a -ECG with a +PYP after surgery. The postoperative scintigram of one such patient (#19) is shown in figure 4 left. The arrows point to the discrete apical radioactivity absent in the preoperative image. Her postoperative electrocardiogram, illustrated in figure 4 right,
Figure 2. Apical perioperative infarction. Shown are the postoperative myocardial scintigrams (with arrows pointing to the discrete accumulation of technetium pyrophosphate) and representative pre and postoperative electrocardiograms of a patient who sustained an inferoapical perioperative infarction. The peak CPK-MB obtained during the first two postoperative days was only 34 units.

Figure 3. Inferior perioperative infarction. Left) Shown are the positive postoperative myocardial scintigrams of patient 18 who sustained an inferior infarction. Note the arrows pointing to the discrete accumulation of isotope in the inferior region of the myocardium. These scintigrams, performed on postoperative day three, were abnormal before the ECG diagnosis was possible. Right) Shown are representative preoperative and postoperative electrocardiograms of patient 18 obtained on the same day as the myocardial scintigrams. While the postoperative tracing was not diagnostic of infarction, a tracing obtained two days later revealed diagnostic Q waves in leads II, III and aVF.
mented myocardial injury without development of Q waves. Unfortunately, a preoperative scintigram was not obtained.

Figure 5 presents the results of these tests in a combined fashion for the revascularization patients. In all eight patients with a +ECG and +PYP, the infarct location determined from the scintigram corresponded to that documented by the electrocardiogram. All patients with −MB also had −PYP, but 34 of 41 patients (83%) with both a −ECG and −PYP, had +MB after surgery.

Discussion

Because conduction defects and ST-T changes are frequently seen in postoperative patients without other clinical or laboratory evidence to suggest perioperative infarction, the appearance of new Q waves has served as the basic criterion for making such a diagnosis.6−7 While studies demonstrating corresponding deterioration of wall motion at postoperative ventriculography appeared to confirm the infarct diagnosis by Q wave criteria,8 the recent report of Bassan et al. suggests that not all perioperative Q waves may represent myocardial damage occurring at surgery.9 These investigators describe findings in four patients with old inferior infarcts who demonstrated new Q waves following surgery, but without deterioration of inferior wall motion seen at postoperative ventriculography. On the other hand, patients with prior infarction or left bundle branch block may not demonstrate changes in QRS morphology despite the occurrence of significant myocardial damage. Among the revascularization patients we studied, seven of the eight with new postoperative Q waves developed them in the inferior leads, raising the possibility of unmasking an old infarction. Twenty-five of the 43 patients who did not develop new Q waves had Q waves prior to surgery. Thus, the electrocardiographic findings of the patients we studied serve to emphasize the need to confirm the presence of new myocardial damage suggested by the postoperative electrocardiogram, as well as to develop techniques to document damage which may not be detected because of the strict criteria required for interpretation of postoperative tracings.

Several investigators have reported general agreement

| ECGs | 8+ | 43− |
| Scintigrams | 8+ | 2+ | 41− |
| Enzymes | 8+ | 34+ | 7− |

Figure 5. Shown are the electrocardiographic (ECG), scintigraphic, and enzymatic results in our 51 revascularization patients. All those with new perioperative electrocardiographic infarcts had abnormal scintigrams. Two patients showed new perioperative scintigraphic infarctions in the presence of nondiagnostic electrocardiograms.
among total CPK, SGOT or LDH and electrocardiographic evidence of infarction, but in each study there were considerable numbers of false positive and false negative findings. Further studies demonstrated that serum levels of total CPK were elevated in patients undergoing noncardiac thoracic or abdominal surgery, presumably because of skeletal muscle trauma. Hemolysis during cardiopulmonary bypass has been shown to cause an elevation of LDH unrelated to myocardial damage. Therefore, more recent studies have investigated postsurgical changes in levels of CPK-MB, the release of which is considered to be a more sensitive and specific indicator of myocardial cell necrosis. These investigations have consistently demonstrated the postoperative appearance of CPK-MB in many patients undergoing cardiac surgery without electrocardiographic evidence of infarction. In our study we were not able to demonstrate a statistical difference between the mean peak CPK-MB values of the revascularization patients who developed new Q waves or persistent ST-T changes and those who did not. Furthermore, the overlap in the range of individual values suggests that our using either the appearance of this isoenzyme or a given peak level to confirm the diagnosis in an individual patient would not be valid, even if statistical differences were found between the mean values for patient groups. Other investigators have documented statistical differences between the mean values of CPK-MB for groups of patients with new Q waves, persistent ST-T changes and "negative" electrocardiograms. However, they have also called attention to a significant number of patients (12-15% in their reported series) with negative electrocardiograms, negative technetium pyrophosphate scintigrams, and levels of serum CPK-MB exceeding an empirically defined "upper limit of normal." Differences in surgical technique, variability of underlying disease, duration of cardiopulmonary bypass and aortic cross-clamping, and type of anesthesia may all be factors which influence the magnitude and time course of isoenzyme release. Such factors may lead to localized or nonlocalized regions of myocardial cell necrosis which may not be extensive enough to produce new Q waves or to be imaged by technetium pyrophosphate, but which can be detected by the presence of CPK-MB. In addition, CPK-MB values measured in individual patients may be influenced by sampling time and frequency. Pending outcome of further studies, the application of isoenzyme analysis remains undefined for both establishing the diagnosis and quantifying the extent of perioperative infarction in individual patients undergoing coronary artery bypass surgery.

In the Coronary Care Unit setting, the use of 99m technetium stannous pyrophosphate myocardial scintigraphy has been shown to be a relatively sensitive and specific means to establish the diagnosis of acute infarction. Although there have been isolated reports of false positive scintigrams thought to be secondary to left ventricular aneurysms, studies at our institution, as well as others, have confirmed the original observation that a discrete localized accumulation of the isotope within the myocardium is a highly specific indicator of recent myocardial damage. In addition to its reliability in demonstrating both the presence and location of an acute infarction, other studies have demonstrated the potential use of this technique to quantify the extent of infarcted myocardium. While the application of infarct scintigraphy to the diagnosis of subendocardial infarction remains a subject of considerable investigation and controversy, some studies hold promise of defining the limits of the application of the imaging technique to the establishment of this diagnosis as well.

Prior to the interpretation of the results in patients undergoing revascularization, we felt that it was important to establish whether factors associated with surgery itself, such as cardiopulmonary bypass, dissection required for valve replacement or repair of congenital atrial abnormalities, or placement of a left ventricular vent, could result in an abnormal image without other evidence to suggest infarction. The postoperative results for our control group suggest that the surgical factors we considered, particularly venting the left ventricle through its apex, usually will not cause a false positive scintigram, but increased activity over the sternum was consistently present postoperatively (fig. 1). In this respect, our findings agree with those recently reported by others.

Among the patients undergoing revascularization, those who had a +ECG also had a +PPY. As noted in one of these patients, illustrated in figure 3, the electrocardiogram obtained on the day of the radionuclide examination was not diagnostic of the inferior myocardial damage documented by the scintigram and confirmed by subsequent electrocardiograms and postoperative catheterization. It is feasible to propose that increasing ischemia of this patient's anterior wall delayed the development of the electrocardiographic manifestations of the inferior infarction, while the myocardial scintigram was capable of documenting its presence.

Of the 43 patients who underwent revascularization with −ECG after surgery, two had a +PPY. One of these patients (#19) had clearly documented pyrophosphate image changes with corresponding postoperative abnormalities of wall motion and relative myocardial perfusion in the absence of diagnostic electrocardiographic changes (fig. 4). The other patient (#20) had sustained an enzyme documented myocardial infarction seventeen days prior to her postoperative scintigram. It is possible that the abnormality we saw was related to this prior event; however, a preoperative scintigram was not performed and the elapsed time between that infarction and her postoperative study is beyond the usual period for which a discrete scintigraphic abnormality has been reported to remain positive. A preoperative scintigram might have provided us with useful interpretive data in this patient. Because of the wide range in individual values of peak CPK-MB seen in all patients, this patient's peak value of CPK-MB of 70 units cannot be used to confirm or reject the diagnosis of perioperative infarction. Thus, we may only speculate whether this case represents a false positive image or a false negative electrocardiogram.

Eighteen of the 34 patients with a −ECG, −PPY and +MB had Q waves on their preoperative electrocardiograms. It is possible that some of these patients sustained significant myocardial damage which was not reflected by changes in QRS morphology and also was not detected by scintigraphy (false −PPY). Since we obtained all scintigrams within 2-5 days of surgery, we are not inclined to invoke delayed imaging as a factor. In studies like ours, other investigators have noted patients with similar findings. Most recently, Righetti et al. concluded that in the absence of new Q waves, both an elevated CPK-MB con-
centration time integral and a +PYP were required to diagnose perioperative myocardial damage. Our findings for patient 19 illustrate that infarct scintigraphy can detect new perioperative damage in the presence of prior infarction without changes on the postoperative electrocardiogram. However, the limitations of applying isoenzyme analysis alone to diagnose perioperative infarction in the absence of Q waves prevents our use of the data from this study to define the incidence of false --PYP.

It is interesting to note that the incidence of preoperative scintigrams with the diffuse pattern of uptake was higher among the small number of studies we performed (40%) than that which we previously reported in a large number of patients with ischemic heart disease (16%).

The surgical patients who demonstrated this finding all had severe coronary artery disease and two had crescendo angina. None had calcific valvular disease, or left ventricular aneurysms, which have been reported as causes for positive preoperative scintigrams.

In the absence of a large number of studies to determine the true incidence of diffuse scintigrams in patients who are candidates for coronary artery revascularization, we suspect that in recommending surgical therapy we may have preselected a subset of patients with severe disease who might have a higher incidence of such image abnormalities than the overall population of patients with ischemic heart disease.

Our data demonstrate results comparable to those found in the nonsurgical setting. Although some workers have also reported a good correlation between electrocardiographic and scintigraphic findings in postoperative patients, some workers have noted an increased incidence of abnormal scintigrams compared to electrocardiograms.

However, these studies include cases demonstrating diffuse, rather than discrete, radionuclide accumulation as evidence of perioperative infarction. We consider this to be a tenuous conclusion because a diffuse pattern of radionuclide accumulation may be relatively nonspecific. We saw no images with diffuse uptake postoperatively. The reason for this, as well as the exact cause of the diffuse pattern, is unclear.

If, in some cases, diffuse uptake indeed represents myocardial damage, we speculate that in some patients a discrete accumulation may have obscured a diffuse pattern which was also present. It is also possible that in our patients all ongoing perioperative ischemic events were completed by the time of imaging, resulting in either transmural infarction, resolution, or damage below the threshold of detection by imaging techniques which was not clarified by electrocardiographic or enzymatic findings.

The results of our study indicate that radionuclide infarct scintigraphy with technetium 99m pyrophosphate is a useful adjunct to the electrocardiogram for the diagnosis of perioperative infarction in patients undergoing coronary artery revascularization. In some cases, it may provide conclusive evidence of myocardial damage before it can be established on the basis of the electrocardiographic findings and occasionally may be the only method yielding reliable diagnostic information. At the present time the role of preoperative myocardial scintigraphy remains unclear, but we feel it is reasonable to obtain such examinations in patients with crescendo angina or persons undergoing surgery for angina persisting after a recently documented infarction.

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Follow-up Technetium-99m Stannous Pyrophosphate Myocardial Scintigrams after Acute Myocardial Infarction

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SUMMARY Technetium-99m stannous pyrophosphate (99mTc-PYP) myocardial scintigrams were obtained in 68 patients during acute myocardial infarction (AMI) and at follow-up 15.9 ± 8.8 weeks later. All patients with AMI had a positive scintigram (2+ or greater); only one of 46 control patients (2%) had a positive (2+) scintigram. At follow-up scintigraphy 6 to 37 weeks following AMI, 57% of patients had a persistently positive scintigram even though recurrent AMI was suspected in only one of these patients. Patients with persistently positive scintigrams tended to have more severe disease as evidenced by compensated congestive heart failure (41%), persistent angina (77%), and ECG evidence of ventricular dyssnergy (51%). We conclude that 1) in patients with prior AMI, a 2+ abnormality on 99mTc-PYP scintigrams may not represent new AMI; 2) a persistently positive 99mTc-PYP scintigram may have prognostic implications since it occurs predominantly in patients with severe symptomatic coronary disease.

THE USEFULNESS OF MYOCARDIAL IMAGING by various radionuclides in the diagnosis of acute myocardial infarction (AMI) in man has been demonstrated.1-9 It has been suggested that "hot spot" imaging agents such as technetium99m stannous pyrophosphate (99mTc-PYP) may be helpful in separating acute from remote myocardial infarction.9,10 This hypothesis is based on observations of myocardial scintigrams in patients and animals studied during the first two weeks following AMI.5, 8, 11, 12

Recently, however, several groups have reported positive 99mTc-PYP scintigrams in patients with left ventricular aneurysms or segmental dysfunction who did not have evidence of recent myocardial infarction, thus challenging the concept that 99mTc-PYP scintigrams can always discriminate between chronic and acutely infarcted myocardium.11-14 To further investigate this question, the present study was undertaken to assess the value and limitations of

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