Contrast Enhancement of Myocardial Infarction: Dependence on Necrosis and Residual Blood Flow and the Relationship to Distribution of Scintigraphic Imaging Agents

CHARLES B. HIGGINS, M.D., PHILLIP L. HAGEN, M.S., JOHN D. NEWELL, M.D., WALTER S. SCHMIDT, AND FRANK H. HAIGLER

SUMMARY All or part of a myocardial infarction (MI) can be preferentially enhanced on computerized transmission tomography after i.v. administration of iodinated contrast material. To examine the relationship of this phenomenon to the presence of myocardial necrosis, the blood flow profile of the MIs and the distribution of radionuclide infarct imaging agents, collateralized infarcts were produced in six dogs (group A) and noncollateralized infarcts were produced in nine dogs (group B). After 48 hours of coronary occlusion, each dog received technetium-99m pyrophosphate ($^{99}$mTc-PYP), thallium-201 ($^{201}$TI), indium-111-labeled microspheres and meglumine sodium diatrizoate (Renografin-76) before sacrifice.

Residual blood flow in the center of the MI was 3–27% in group A and less than 3% in group B. In group A, the iodine concentration in the center (1.33 mg/g myocardium), periphery (1.48 mg/g), and margin (1.09 mg/g) were several times higher than normal myocardium (0.45 mg/g). The distribution of $^{99}$mTc PYP was similar to that of iodine; the distribution of $^{201}$TI was roughly the inverse of that of iodine.

In group B, the average iodine and $^{99}$mTc concentrations in the margin and periphery of the MI were several times higher than that in normal myocardium, but in the center neither concentration was significantly higher than normal. However, there was no consistent relationship between iodine and $^{201}$TI, as concentrations of iodine and $^{201}$TI were low in the center of the infarct and were inversely related at other sites.

The results show that contrast material has a distribution in MI similar to that of $^{99}$mTc PYP and both are markers of ischemic myocardial necrosis. Distribution of both agents in the MI depends on a threshold level of residual myocardial blood flow.

COMPUTERIZED transmission tomography (CTT) has been used to image myocardial infarctions in experimental animals1–4 and, in a few instances, in man.4 After i.v. or intracoronary administration of iodinated contrast material to dogs with myocardial infarctions, CTT scans have shown delayed and differential enhancement of the area of ischemic damage compared with normal myocardium. Measurement of myocardial tissue concentration of iodine have confirmed this preferential accumulation of contrast material in infarcted myocardium and have shown peak concentrations of iodine in the infarct at 10 minutes after i.v. administration of the contrast material.5

Contrast material therefore can provide a positive image of the infarct on the CTT scan, similar to the image produced by technetium-99m pyrophosphate ($^{99}$mTc-PYP) on scintigraphic scans of acute myocardial infarction.4 Moreover, like the $^{99}$mTc-PYP scan, the pattern of contrast enhancement has been homogeneous in some infarcts and only partial or peripheral in others1–2 (fig. 1). For the most part, $^{99}$mTc-PYP accumulates in irreversibly damaged myocardial cells and signifies the presence of myocardial necrosis.6–8 The significance of differential accumulation of iodinated contrast material in areas of ischemic myocardial damage has not been determined.

The purpose of the current study was to determine the significance of contrast enhancement of myocardial infarctions by studying the relationship of accumulation of contrast material (iodine) to myocardial necrosis and residual myocardial blood flow. We also compared the myocardial distribution of contrast material with the distribution of the important hot spot ($^{99}$mTc-PYP) and cold spot ($^{201}$TI) radiopharmaceuticals. Two types of acute myocardial infarctions were produced. One group of dogs had well-collateralized infarcts and the other group had poorly collateralized dense myocardial infarctions.

Methods

Fifteen mongrel dogs underwent operative ligation of the largest obtuse marginal branch of the circumflex coronary artery during general anesthesia (pentobarbital sodium, 25 mg/kg). In six dogs (group A), all potential collateral vessels were left intact; in nine dogs (group B), the distal ends of epicardial vessels that coursed toward the area of distribution of the ligated obtuse marginal artery were also ligated. After 48 hours, the dogs were reanesthetized, the thoracotomy was reopened and a catheter was placed in the left atrium. Before sacrifice, the following substances were administered intravenously: 5–6 mCi of $^{99}$mTc-PYP at 45 minutes, 530–590 mCi of $^{201}$TI at 15 minutes and 2 ml/kg of Renografin at 10 minutes before sacrifice. Five minutes before sacrifice, 1

From the Department of Radiology, University of California San Diego Medical Center, San Diego, California.

Dr. Higgins is the recipient of USPHS Career Development Award 1KO4 HL-2001 from the NHLBI.

Address for correspondence: Charles B. Higgins, M.D., University Hospital, Department of Radiology, 225 Dickinson Street, San Diego, California 92103.

Received March 9, 1981; revision accepted July 21, 1981.

Circulation 65, No. 4, 1982.

739
millions $^{113}$In-labeled (420–460 mCi) albumin microspheres (3M Co.) were injected through the atrial catheter. The microspheres were administered within 1 hour after labeling. Before injection, the microspheres were agitated for 5 minutes. The diameter of the microspheres was 10–35 μ (mean 20 μ). In two other dogs, 1 million $^{85}$Sr-labeled (500 mCi) carbonized microspheres and 1 million $^{113}$In-labeled (500 mCi) albumin microspheres were injected through the atrial catheter 5 minutes before sacrifice. The carbonized microspheres had a diameter of 15 μ. In one dog, the $^{85}$Sr-labeled microspheres were injected first, followed by the $^{113}$In labeled microspheres; in the other dog the order was reversed.

After sacrifice, each heart was removed and sectioned at 1-cm intervals from apex to base along the major axis of the left ventricle and incubated in nitroblue tetrazolium dye as described previously. The endocardial and epicardial borders of the LV wall were traced onto clear film overlays. In the septal area, the right ventricular side of the septum was substituted for the epicardial border. The area of grossly visible myocardial infarction was also traced onto the same overlays. Two myocardial slices were selected where the infarcted area was largest; five tissue samples were cut from each of the two transverse slices (fig. 2). The tissue sample included the subendocardium and extended through approximately half the thickness of the myocardial wall. Some samples extended through less than half of the myocardial wall when the infarct was confined to a small subendocardial site. Each sample from infarcted tissues was taken from within the grossly visible border of the infarct. Samples were obtained from the center of the infarct, the periphery of the infarct, and from just inside the visible margin between infarcted (unstained) and normal (stained) myocardium. Tissue samples were also obtained from grossly normal myocardium adjacent to the infarct and from normal myocardium on the ventricular wall opposite the infarct. Each sample was divided into two equal parts. A thin slice from each sample was taken for histologic examination after hematoxylin and eosin staining. The volume of tissue samples varied from 0.21–1.06 ml. The volume of samples was predicated upon the volume of the infarct. In each dog, approximately similar volumes were obtained from each site. The volume of each tissue sample was determined by placing the sample in a graduated 1.5-ml cylinder and measuring from a calibrated burette the volume of fluid needed to fill the cylinder to the 1.5-ml mark.

The percentage of the left ventricular mass involved by myocardial infarction was measured by planimetry.

**Figure 1.** Examples of computerized transaxial tomographic scans from two dogs with 48-hour-old myocardial infarctions. The dogs were sacrificed 10 minutes after i.v. administration of Renografin-76, 2 ml/kg, and the excised hearts were scanned in a cranial scanner. The scan on the left shows partial or peripheral enhancement of the infarct; the central area of the infarct is not enhanced. The scan on the right shows enhancement of the entire infarct. These scans were obtained as described previously.

**Figure 2.** The sites in the infarct and normal myocardium from which samples were obtained to measure iodine and radionuclide concentrations. MI = myocardial infarction; RV = right ventricle; LV = left ventricle; normal = normal myocardium near MI; normal = normal myocardium far from MI.
metry of the infarcted area and the area of the left ventricular wall previously traced on the overlays. This was accomplished with an ultrasonic digitizer interfaced to a Hewlett-Packard Model 9825T desktop microprocessor and printer-plotter. The size of the infarct in each instance was expressed as a percentage of the total left ventricular wall.

The iodine content in each tissue sample was determined by fluorescent excitation analysis and the concentration was calculated as the quotient of the iodine content and the volume of the tissue sample. Fluorescent excitation analysis and its use for assessing iodine concentration in the myocardium have been described.9

The tissue samples were also assayed for radioactivity in a well-type automatic gamma scintillation counter (Searle model 1185R). Counts were obtained from the photon energy peaks of $^{99m}$Tc (140 keV), $^{201}$TI (80 keV) and $^{111}$In (243 keV). Background counts in each of these channels were also obtained and subtracted from total counts. For each isotope, counts were obtained for 1 minute. Isotope activities were determined within 3 hours after sacrifice and repeated again 24 hours after sacrifice. The raw counts in the specific channel for each isotope were corrected for cross-contribution activity from the channels of the other two isotopes using simultaneous equations for the three isotopes.10 The concentration for each element from each site was computed as the average concentration of four samples. These four samples resulted from the samples obtained from the same site on the two adjacent myocardial transverse slices and each of these samples was divided into two samples.

Iodine was expressed as mg/g of tissue and radionuclides as counts/g of tissue. To compare the distribution of iodine and various radionuclides, the concentrations were expressed as a percentage of the concentration in normal myocardium. There was no difference in the concentrations of elements between the two sampling sites in normal myocardium.

In the two dogs given $^{85}$Sr-labeled carbonized microspheres and $^{111}$In-labeled albumin microspheres, the distributions of the two sets of microspheres were almost identical. The distributions of the isotopes showed a very close linear relationship in the two dogs ($r = 0.99$ and 0.98). Although there are apparent differences in the distribution of microspheres between the subendocardial and subepicardial myocardial layers based upon the size of the spheres,11 the distribution of the 15-μ carbonized microspheres and the larger albumin microspheres in various regions of the infarct and normal myocardium were nearly identical.

The relationship between the distribution of iodine to the distribution of $^{99m}$Tc-PYP, $^{201}$TI and $^{111}$In-labeled microspheres was analyzed by multiple regression analyses to determine the best-fit formula. The concentrations of the various elements at sites in the infarct were compared with that in normal myocardium using repeated-measures analysis of variance. The residual blood flow at various sites in the infarcts of group A were compared with those in group B by analysis of variance. The volumes of the infarcts in group A were compared with those in group B by group t test.

**Results**

**Characteristics of Myocardial Infarctions**

**Collateralized Infarcts (Group A)**

The collateralized infarcts were almost uniformly pink, in contrast to the reddish color of normal myocardium. Hemorrhagic areas were interspersed throughout the infarct, and there was always at least a thin rim of grossly normal myocardium between the edge of the infarct and the epicardial surface. Histologic characteristics were neutrophilic infiltration, coagulation necrosis, prominent vascular congestion and hemorrhage, and microcalcification. Sections from the center and periphery of these infarcts had similar features. Sections from the margin revealed interspersed areas of necrotic and normal myocardial cells.

The volume of the collateralized infarcts averaged $13.1 \pm 5.3\%$ (± sd) of left ventricular volume, which was not significantly different from that in group B (table 1). Residual blood flow was $14.0 \pm 11.6\%$ of

**Table 1. Individual Values for Residual Blood Flow in Regions of Myocardial Infarctions and Percentage of Left Ventricle Involved by Myocardial Infarction**

<table>
<thead>
<tr>
<th>Dog</th>
<th>Blood flow in MI (% normal)</th>
<th>Volume of MI (% LV)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Center</td>
<td>Periphery</td>
</tr>
<tr>
<td>2</td>
<td>8.01</td>
<td>19.12</td>
</tr>
<tr>
<td>3</td>
<td>13.24</td>
<td>32.42</td>
</tr>
<tr>
<td>5</td>
<td>3.00</td>
<td>5.12</td>
</tr>
<tr>
<td>6</td>
<td>4.12</td>
<td>4.23</td>
</tr>
<tr>
<td>14</td>
<td>32.34</td>
<td>66.21</td>
</tr>
<tr>
<td>17</td>
<td>23.30</td>
<td>35.41</td>
</tr>
<tr>
<td>Mean</td>
<td>14.00</td>
<td>27.09</td>
</tr>
<tr>
<td></td>
<td>± 11.64</td>
<td>± 23.22</td>
</tr>
</tbody>
</table>

**Dense infarcts (group B)**

<table>
<thead>
<tr>
<th>Dog</th>
<th>Blood flow in MI (% normal)</th>
<th>Volume of MI (% LV)</th>
</tr>
</thead>
<tbody>
<tr>
<td>7</td>
<td>1.90</td>
<td>6.11</td>
</tr>
<tr>
<td>8</td>
<td>2.18</td>
<td>7.40</td>
</tr>
<tr>
<td>9</td>
<td>0.77</td>
<td>3.39</td>
</tr>
<tr>
<td>10</td>
<td>0.10</td>
<td>0.01</td>
</tr>
<tr>
<td>11</td>
<td>0.01</td>
<td>14.10</td>
</tr>
<tr>
<td>12</td>
<td>0.05</td>
<td>3.22</td>
</tr>
<tr>
<td>13</td>
<td>0.01</td>
<td>0.04</td>
</tr>
<tr>
<td>15</td>
<td>1.26</td>
<td>6.01</td>
</tr>
<tr>
<td>16</td>
<td>0.01</td>
<td>0.01</td>
</tr>
<tr>
<td></td>
<td>0.69</td>
<td>3.63</td>
</tr>
<tr>
<td></td>
<td>± 0.88</td>
<td>± 5.56</td>
</tr>
</tbody>
</table>

*p* < 0.01 NS NS NS

*Group A vs group B.

Abbreviations: MI = myocardial infarction; LV = left ventricle.
normal in the center of the infarct, 27.0 ± 23.2% in the periphery, and 35.6 ± 18.9% at the margin of the infarct. The lowest value for residual blood flow in the center of the infarct in group A was 3% of normal. The residual blood flow in the center of the infarct was significantly higher than that in group B (p < 0.01).

**Dense Infarcts (Group B)**

The dense infarcts consisted grossly of a central core of pale gray tissue surrounded by a peripheral zone of variable size. The peripheral region was pink and contained hemorrhagic areas. Histologic appearance in the central core was characterized by coagulation necrosis and the absence of neutrophilic infiltration, vascular congestion and calcification. The periphery was characterized by coagulation necrosis, vascular congestion and hemorrhage, neutrophilic infiltration and microcalcification. Sections from the margin revealed interspersed areas of necrotic and normal myocardial cells.

The volume of the infarcts averaged 19.9 ± 9.9% of left ventricular volume (table 1). Residual blood flow was 0.7 ± 0.9% of normal in the center, 6.4 ± 5.6% in the periphery, and 46.3 ± 17.2% at the margin of the infarct. In all infarcts in group B, residual flow in the center was less than 3% of normal blood flow.

**Distribution of Iodine and Radionuclides in Collateralized Infarcts**

Iodine and **99m**Tc-PYP showed a similar distribution profile in collateralized infarcts (fig. 3). There was a high correlation coefficient for the direct linear relationship between iodine and **99m**Tc-PYP concentrations in all six dogs in this group (table 2). The concentration of **99m**Tc-PYP was substantially higher in the margin (p < 0.05), periphery (p < 0.05) and center (p < 0.05) of the infarct than in normal myocardium (fig. 4). The distribution of iodine was also higher in the margin (p < 0.05), periphery (p < 0.01) and center (p < 0.05) of the infarct than in normal myocardium.

The distributions of **201**TI and **111**In-labeled microspheres showed an inverse nonlinear relationship to that of iodine (fig. 5, table 2). The concentration of **201**TI and **111**In microspheres decreased progressively from the margin to the center of the infarct (fig. 4). The concentrations of **201**TI and **111**In at each of the three sites in the infarct were significantly lower than in normal myocardium (p < 0.01).

**Distribution of Iodine and Radionuclides in Dense Infarcts**

Iodine and **99m**Tc-PYP showed a similar distribution profile in the dense infarcts (figs. 6 and 7). There was a high correlation coefficient for the direct linear relationship between iodine and **99m**Tc-PYP distribution in all nine dogs in this group (fig. 6, table 2). In contrast to the collateralized infarcts, the concentrations of iodine and **99m**Tc-PYP were not increased in the center of the dense infarcts (fig. 7). The concentra-
The residual blood flow in the center of dense infarcts averaged 0.7 ± 0.9% of normal, and iodine and $^{99m}$Tc were not present in higher concentrations than in normal myocardium. In each dog in this group, residual blood flow in the center of the infarct was less than 3% of normal; in six dogs, blood flow was less than 1% in the center of the infarct. Thus, the distribution patterns of both iodine and $^{99m}$Tc-PYP depended upon a threshold level of residual myocardial blood flow to the center of the myocardial infarction.

**Discussion**

The results show that the distribution of iodinated contrast material in infarcted myocardium similar to that of the infarct-avid scintigraphic imaging agent, $^{99m}$Tc-PYP. Both agents were present in high concentrations in infarcted myocardium and were markers of myocardial necrosis. However, the distribution of both agents was also dependent on a threshold level of residual perfusion of the infarct. In well-collateralized infarcts, iodine and $^{99m}$Tc-PYP were present in high concentration throughout the infarct, whereas in the center of dense infarcts, there was almost no residual blood flow, neither agent was present in increased concentration. Additionally, iodine had a distribution profile that was inversely related to $^{201}$TI. This relationship did not hold for the center of the dense infarcts, however, where both iodine and $^{201}$TI were present in low concentrations.
higher in accumulated by ischemically damaged myocardial perfusion. This finding is similar to the distribution of iodine and technetium-99m pyrophosphate (99mTc-PYP) in normal myocardium, whereas the concentration was many times higher in the outer central and peripheral zones of the infarct; the average value for residual myocardial perfusion in the central zone was 4% of normal. The latter value correlates well with the threshold level of myocardial perfusion (3%) found in the current study for accumulation of 99mTc-PYP and iodine in acute myocardial infarctions. Comparison of the distributions of iodine and 99mTc-PYP in two types of infarction used in the current study are consistent with the notion that iodine, like 99mTc-PYP, is a marker of ischemic myocardial necrosis but its accumulation in regions of ischemically damaged myocardium depends upon a threshold level of residual myocardial perfusion.

Technetium-99m-labeled phosphate compounds are accumulated by ischemically damaged myocardial cells, but do not accumulate in normal myocardial cells. Although both experimental and clinical studies have raised the possibility that 99mTc-PYP can accumulate to a slight degree in reversibly damaged myocardial cells, accumulation of 99mTc-PYP generally indicates irreversible myocardial damage. A recent study from our laboratory using scanning electron microscopy with elemental x-ray dispersive microanalysis indicated that substantial levels of iodine were measured in myocardial cells within the infarcted region, but no iodine was detected in normal myocardium. The damaged myocardial cells that accumulated iodine showed drastic alteration of intracellular sodium-potassium ratio, suggesting the loss of cellular membrane integrity and probably irreversible cellular damage. Whether iodine accumulation occurs to any degree in reversible damaged myocardial cells has not been established.

In contrast to 99mTc-PYP and iodinated contrast material, 201TI, like other potassium analogs, is taken up by normal myocardial cells. Concentration of 201TI in the myocardium is related to the level of myocardial perfusion and to the integrity of active mem-

**FIGURE 6.** Direct linear relationship between iodine (I) concentration and technetium-99m pyrophosphate (99mTc-PYPh) in a dog with a dense infarct (group B).

The distribution of radiopharmaceuticals for hot spot (99mTc-PYP) and cold spot (201TI) imaging of myocardial infarctions and the dependence of this distribution on the level of residual myocardial perfusion have been studied. The distribution profiles found in the current study and in previous studies are qualitatively similar.

Buja et al demonstrated that in the maximally ischemic central zone of the infarct, the concentration of 99mTc-PYP was similar to that of normal myocardium, whereas the concentration was many times higher in the outer central and peripheral zones of the infarct; the average value for residual myocardial perfusion in the central zone was 4% of normal. The latter value correlates well with the threshold level of myocardial perfusion (3%) found in the current study for accumulation of 99mTc-PYP and iodine in acute myocardial infarctions. Comparison of the distributions of iodine and 99mTc-PYP in two types of infarction used in the current study are consistent with the notion that iodine, like 99mTc-PYP, is a marker of ischemic myocardial necrosis but its accumulation in regions of ischemically damaged myocardium depends upon a threshold level of residual myocardial perfusion.

Technetium-99m-labeled phosphate compounds are accumulated by ischemically damaged myocardial cells, but do not accumulate in normal myocardial cells. Although both experimental and clinical studies have raised the possibility that 99mTc-PYP can accumulate to a slight degree in reversibly damaged myocardial cells, accumulation of 99mTc-PYP generally indicates irreversible myocardial damage. A recent study from our laboratory using scanning electron microscopy with elemental x-ray dispersive microanalysis indicated that substantial levels of iodine were measured in myocardial cells within the infarcted region, but no iodine was detected in normal myocardium. The damaged myocardial cells that accumulated iodine showed drastic alteration of intracellular sodium-potassium ratio, suggesting the loss of cellular membrane integrity and probably irreversible cellular damage. Whether iodine accumulation occurs to any degree in reversible damaged myocardial cells has not been established.

In contrast to 99mTc-PYP and iodinated contrast material, 201TI, like other potassium analogs, is taken up by normal myocardial cells. Concentration of 201TI in the myocardium is related to the level of myocardial perfusion and to the integrity of active mem-

**FIGURE 7.** The mean concentrations of iodine (I), technetium-99m pyrophosphate (99mTc) and thallium-201 (201TI) at various sites in the group of dogs with dense infarcts (group B).
brane transport by myocardial cells. In the current study and in a previous study, there was substantial, but reduced, 201Tl activity at the margin of the infarct where 99mTc-PYP activity was most intense. This is apparently a zone where residual blood flow is substantial, but insufficient to maintain viability of many myocardial cells.

The blood flow profile in both the dense and collateralized infarcts can be compared with the blood flow observed in the necrotic and ischemic zones defined by Vokonas et al. From their precise comparison of histopathology with infarction, the transition from viable to necrotic myocardial cells in the ischemic zone was associated with residual blood flow averaging 55% of normal myocardial blood flow. In some animals in the study of Vokonas et al., the residual blood flow at the edge of the necrotic zone was 60–70% of normal blood flow. In the current study, the residual blood flow at the margin of the infarcts was in the range characteristic of the onset of myocardial necrosis in the study of Vokonas et al. Thus, uptake of 99mTc-PYP and iodinated contrast materials is increased in the ischemic zone in which myocardial blood flow has been reduced enough to result in myocardial necrosis.

Scintigraphic scans of 99mTc-PYP uptake in acute myocardial infarctions in experimental animals and in man have shown two patterns: global or homogeneous uptake and peripheral uptake producing a doughnut pattern. The former pattern tends to be associated with subendocardial infarctions and the latter pattern with large anterior myocardial infarctions. Likewise, CTT scans of experimental myocardial infarctions after i.v. administration of contrast material have shown both global and partial enhancement. Partial enhancement has generally, but not always, been peripheral. The distinctly different profiles of distribution of iodine and 99mTc-PYP in the collateralized and dense infarcts in the current study are consistent with the notion that the patterns of the scans with infarct-avid agents is critically influenced by the level of residual myocardial blood flow or reperfusion of the area of infarction. In the current study, the volume of the myocardial infarction was not a consistent determinant of the pattern of accumulation of contrast material and 99mTc-PYP.

Recently, Doherty et al. assessed the distribution of radiolabeled contrast material, infarct imaging radionuclides, and radiolabeled microspheres in relatively large transmural infarctions in dogs. The distribution of contrast material and Tc-PYP were roughly similar to those found in the current study; however, they believed that the pyrophosphate extended into the periphery of the infarction while the contrast material was confined to a border zone between the normal and infarcted myocardium. In the current study, direct comparison of tissue concentrations of iodine and 99mTc-PYP revealed a direct linear relationship between the distribution of the two elements in both collateralized and dense myocardial infarctions. The dispute between our interpretation, that contrast material is differentially accumulated in the periphery of poorly collateralized myocardial infarctions, and the conclusion by Doherty et al., that it marked a noninfarcted border zone, has implications for estimating the volume of infarcted myocardium. Our results suggest that the outer margin of the contrast-enhanced area of CTT scans should be used to measure area or volume of the infarct, whereas Doherty et al. used the inner margin of the enhanced myocardium to measure the size of the infarct.

CTT scans of myocardial infarctions obtained 10 minutes after i.v. administration of contrast material have shown differential enhancement of the infarcted area in experimental animals and in man. Contrast enhancement of the ischemically damaged region has been observed as early as 8 hours after coronary arterial occlusion and has also been observed in evolved myocardial infarctions 2 months after coronary arterial ligation. The highest concentration of iodine (contrast material) in infarcted myocardium and the widest absolute difference in iodine (contrast material) concentration between normal and infarcted myocardium occurs 10 minutes after i.v. administration of contrast material. Because discrimination between structures on CTT scans depends largely on absolute differences in x-ray density, detection and sizing of the infarct are best accomplished at this time.
This differential concentration of iodine in infarcted and normal myocardium can be detected for at least 3 hours using sensitive physiochemical analysis of tissue samples.

The ideal method for sizing infarctions on CTT scans has not been determined. It is not clear whether measurements should be made during the early perfusion phase scans after i.v. administration of contrast material when normal myocardium is differentially enhanced, or 10 minutes after cessation of contrast material administration when the margin of the infarct is enhanced. When delayed contrast-enhanced scans are used to size the infarct, it is not clear whether the inner or outer margin of the contrast-enhanced area should be used for volumetric measurements.

References
Contrast enhancement of myocardial infarction: dependence on necrosis and residual blood flow and the relationship to distribution of scintigraphic imaging agents.
C B Higgins, P L Hagen, J D Newell, W S Schmidt and F H Haigler

Circulation. 1982;65:739-746
doi: 10.1161/01.CIR.65.4.739

Circulation is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
Copyright © 1982 American Heart Association, Inc. All rights reserved.
Print ISSN: 0009-7322. Online ISSN: 1524-4539

The online version of this article, along with updated information and services, is located on the World Wide Web at:
http://circ.ahajournals.org/content/65/4/739.citation

Permissions: Requests for permissions to reproduce figures, tables, or portions of articles originally published in Circulation can be obtained via RightsLink, a service of the Copyright Clearance Center, not the Editorial Office. Once the online version of the published article for which permission is being requested is located, click Request Permissions in the middle column of the Web page under Services. Further information about this process is available in the Permissions and Rights Question and Answer document.

Reprints: Information about reprints can be found online at:
http://www.lww.com/reprints

Subscriptions: Information about subscribing to Circulation is online at:
http://circ.ahajournals.org//subscriptions/