**99mTc-N-NOET Myocardial Uptake Reflects Myocardial Blood Flow and Not Viability in Dogs With Reperfused Acute Myocardial Infarction**

Gérald Vanzetto, MD, PhD; David K. Glover, ME; Mirta Ruiz, MD; Dennis A. Calnon, MD; Roberto Pasqualini, PhD; Denny D. Watson, PhD; George A. Beller, MD

**Background**—N-Ethoxy-N-ethyl-dithiocarbamato-nitrido-99mTc (99mTc-N-NOET) is a new neutral lipophilic 99mTc-labeled myocardial perfusion agent with a high first-pass extraction fraction and delayed redistribution kinetics after transient ischemia comparable to what is observed with 201Tl. It is unknown whether the uptake of this tracer reflects myocardial viability or just reperfusion flow in the setting of a reperfused myocardial infarction.

**Methods and Results**—In 13 anesthetized open-chest dogs, the left anterior descending coronary artery was occluded for 180 minutes, followed by 180 minutes of reperfusion. 201Tl and 99mTc-N-NOET were injected after either 60 (group 1, n = 9) or 175 (group 2, n = 4) minutes of reperfusion. Myocardial blood flow was measured by radioactive microspheres, and 201Tl and 99mTc-N-NOET tissue activities were determined by gamma-well counting. Normalized myocardial blood flow in the central infarct zone fell from 0.80 ± 0.03 (SEM) and 0.89 ± 0.01 at baseline to 0.18 ± 0.04 and 0.13 ± 0.02 during the occlusion in groups 1 and 2, respectively. Normalized 201Tl activity in these segments was 0.39 ± 0.04 and 0.43 ± 0.04 and reflected myocardial viability rather than reperfusion flow (P < 0.001). Normalized 99mTc-N-NOET activity in the same segments was 0.84 ± 0.08 and 0.64 ± 0.03, respectively (P < 0.01 versus 201Tl; P = NS versus reperfusion flow) and more accurately reflected reperfusion flow (0.99 ± 0.17 and 0.70 ± 0.04) than residual viability.

**Conclusions**—The myocardial uptake of 99mTc-N-NOET reflects reperfusion myocardial blood flow and not viability in a canine model of reperfused acute myocardial infarction. The clinical use of early 99mTc-N-NOET imaging to assess the success of coronary reperfusion in patients with acute myocardial infarction should be investigated. *(Circulation. 2000;101:2424-2430.)*

**Key Words:** radioisotopes ▪ myocardial infarction ▪ thallium ▪ technetium

**The aim of thrombolytic therapy in the acute phase of myocardial infarction is to achieve early reperfusion of the infarct-related artery achieving TIMI-3 flow.** Despite advances in this field, failure to achieve successful reflow still occurs in 30% to 40% of cases.1 Rescue PTCA may be useful in subsets of patients who fail to reperfuse with thrombolytic therapy.2 Clinical and ECG criteria for assessing the success or failure of thrombolysis are suboptimal,3 and deciding whether a patient is a candidate for rescue PTCA is often difficult. Myocardial contrast echocardiography has been shown to be of interest for such indication, but an intravenous myocardial contrast agent has not yet been validated for clinical applicability.4

Radionuclide perfusion imaging theoretically should be an excellent approach for assessing the efficacy of reperfusion early after thrombolytic therapy, but the myocardial uptake of tracers in clinical use is dependent on myocardial metabolic status and cellular viability and therefore will underestimate reperfusion flow in this setting.5-9 Results from studies using 99mTc-teboroxime, a neutral, highly lipophilic agent, in this setting are conflicting.10,11 Furthermore, rapid myocardial clearance of 99mTc-teboroxime limits its clinical usefulness for acute single photon emission CT (SPECT) imaging.12,13 N-Ethoxy-N-ethyl-dithiocarbamato-nitrido-99mTc (99mTc-N-NOET) is a new neutral, lipophilic myocardial perfusion imaging agent that has an uptake pattern that correlates well with myocardial blood flow in a canine model of myocardial ischemia14 and undergoes significant rest-redistribution over time in experimental models of transient coronary occlusion and sustained low coronary flow.14,15 Furthermore, cellular uptake of 99mTc-N-NOET has recently been shown to be independent of metabolic poisons that damage cell membranes and the cellular ATP content in cultured newborn rat cardiomyocytes.16

The aim of this study was to determine whether 99mTc-N-NOET uptake after coronary reperfusion, preceded by 180
minutes of coronary occlusion, will be more reflective of reperfusion flow than of cellular viability, because cellular uptake of $^{99m}$Tc-N-NOET is not energy-dependent and does not require intracellular uptake for myocardial sequestration.

Methods

Surgical Preparation

Thirteen fasted male adult mongrel dogs were anesthetized with sodium pentobarbital (30 mg/kg), intubated, and ventilated on a respirator (Harvard Apparatus) with 4 cm H$_2$O of positive end-expiratory pressure. Arterial blood gases were closely monitored, and appropriate ventilation adjustments were performed to maintain pH, PO$_2$, P$\text{CO}_2$, and HCO$_3$ within physiological ranges. Lead II of the ECG was continuously monitored during the experiments. The left femoral vein was cannulated with an 8F polyethylene catheter (Cordis) for fluid administration, additional anesthesia, and $^{99m}$Tc-N-NOET or $^{201}$TI injections. Both femoral arteries were cannulated with similar catheters for arterial blood samples for blood gas determinations and radiolabeled microsphere reference blood withdrawals. The left main carotid artery was cannulated with an 8F sheath for monitoring systemic arterial pressure. A Millar high-fidelity pressure-recording catheter was introduced through this sheath and advanced into the LV cavity for measurement of LV pressure and dP/dt.

The open-chest canine surgical preparation used in these experiments was described previously. Briefly, a left thoracotomy was performed at the level of the fifth intercostal space, and the heart was suspended in a pericardial cradle. A 1.5-cm section of the LAD was dissected free of the epicardium and loosely coapted. The surgical preparation of this radiopharmaceutical has been described in detail. Quality control was performed with thin-layer chromatography with silica gel plates (J.T. Baker) and dichloromethane. Radiochemical purity was >90% in each experiment.

In Vivo Image Acquisition and Defect Magnitude Quantification

In the group 1 dogs, planar images were acquired in the left lateral oblique projection with a standard gamma-camera (Technicare 420, Ohio Nuclear) equipped with an all-purpose, low-to-medium-energy collimator at 5, 15, 30, 60, 90, and 120 minutes after tracer injection. Background was subtracted from all images by use of a previously validated interpolative algorithm without thresholding or filtering. To quantify the $^{99m}$Tc-N-NOET activity on images, regions of interest (ROIs) were drawn on the defect area of the anteroseptal wall supplied by the LAD and on the posterior wall supplied by the LCx. The LAD/LCx defect count ratio was computed by dividing the average counts in the ischemic ROI by the average counts in the nonischemic ROI.

Microsphere-Derived Regional Myocardial Blood Flow and $^{201}$TI and $^{99m}$Tc-N-NOET Myocardial Activities

The technique used in our laboratory to quantify myocardial blood flow by radiolabeled microspheres was described previously. After the dogs were euthanized, the LV was divided into 4 slices ~1.5 cm thick, and each slice was divided into 6 transmural sections. Each of these transmural sections was then subdivided into epicardial, midwall, and endocardial segments, resulting in 24 LV segments. The myocardial segments were counted for $^{99m}$Tc activity in a gamma-well counter (MINAXI 5550, Packard Instruments) within 24 hours. The myocardial segments were recounted for $^{201}$TI activity 3 days later after $^{99m}$Tc had decayed. Finally, a third count was performed 3 weeks later for quantification of microsphere activities when $^{201}$TI activity was negligible. The gamma-counter window settings were $^{113}$Sn, 340 to 440 keV; $^{103}$Ru, 450 to 550 keV; $^{95}$Nb, 640 to 840 keV; and $^{46}$Sc, 842 to 1300 keV. Tissue counts were corrected for background, decay, and isotope spillover, and regional myocardial blood flow was calculated by computer software developed for this purpose (PCGERDA, Scientific Computing Solutions, LLC).

Postmortem Determination of Risk Area and Infarct Size

The endocardial and epicardial surfaces of each heart slice and the borders of the monastral blue dye–determined risk area were traced on acetate sheets. The heart slices were then incubated for 10 minutes at 37°C in a 2% solution of TTC to delineate infarct area. The infarct area was then traced onto the previous acetate sheets. Risk area (monastral blue dye-negative) and infarct areas (TTC-negative) were determined with a digital planimeter program as previously described.

Data Analysis

Myocardial blood flows and tracer activities in each sample were normalized to the average activity of 15 reference samples from the posterior LV wall (5 transmural segments × 3 layers) that exhibited normal absolute myocardial blood flow during occlusion. Each segment was then grouped according to the flow reduction observed during the occlusion period. Segments with flow <0.3, ≥0.3 and <0.5, and ≥0.5 mL · min$^{-1}$ · g$^{-1}$ were classified as infarct, border, and normal zones, respectively.
models of coronary occlusion and reperfusion with severe myocardial injury. In group 2 dogs, there was a statistically significant decrease in normal-zone flow during occlusion (0.89 mL/min), and it remained unchanged during occlusion and reperfusion. Ultrasonic left anterior descending coronary artery (LAD) flow fell from 21 ± 3 mL/min during baseline to 0 ± 0 mL/min after the occlusion was set (P < 0.001). Sixty minutes after reflow, at the time when 201 Tl and 99m Tc-N-NOET were injected in 9 of the 13 dogs, LAD flow had returned to baseline (24 ± 4 mL/min). However, as is frequently observed in experimental models of coronary occlusion and reperfusion with severe myocardial infarction, coronary flow gradually decreased in the reperfused LAD zone over 3 hours.

Regional systolic wall thickening in the central region supplied by the LAD distal to the occluder decreased from 21 ± 2% during baseline to −6 ± 1% after occlusion (P < 0.001) and remained constant throughout the rest of the experiment during occlusion and after reperfusion.

### Results

#### Hemodynamics

Hemodynamic data for the 13 dogs are summarized in Table 1. There was no significant difference in hemodynamic data between the 2 groups, and therefore, the hemodynamic data were pooled. Heart rate, left circumflex coronary artery (LCx) ultrasonic flow, and maximum positive first derivative of LV pressure (dP/dt) did not change throughout the experiments. Mean arterial pressure remained constant during baseline, occlusion, and reperfusion but decreased significantly at the end of the experiments. Mean left atrial pressure increased significantly after occlusion and then remained unchanged during occlusion and reperfusion. Ultrasonic left anterior descending coronary artery (LAD) flow fell from 21 ± 3 mL/min during baseline to 0 ± 0 mL/min after the occlusion was set (P < 0.001). Sixty minutes after reflow, at the time when 201 Tl and 99mTc-N-NOET were injected in 9 of the 13 dogs, LAD flow had returned to baseline (24 ± 4 mL/min). However, as is frequently observed in experimental models of coronary occlusion and reperfusion with severe myocardial infarction, coronary flow gradually decreased in the reperfused LAD zone over 3 hours.

Regional systolic wall thickening in the central region supplied by the LAD distal to the occluder decreased from 21 ± 2% during baseline to −6 ± 1% after occlusion (P < 0.001) and remained constant throughout the rest of the experiment during occlusion and after reperfusion.

#### Risk Area and Infarct Size

The risk area (monastral blue dye–negative area) was 29.9 ± 1.8% of the left ventricle (LV). By triphenyltetrazolium chloride (TTC) staining, 15.9 ± 2.1% of the LV was infarcted. The infarct size was 43.8 ± 4.4% of the risk area.

#### Microsphere Blood Flow

Table 2 summarizes the normalized myocardial blood flow and 201 Tl and 99mTc-N-NOET activities in normal, border-zone, and infarcted samples from the 2 groups of dogs. In group 1 dogs, regional myocardial blood flow ratios in the risk area and infarcted samples from the 2 groups were compared. However, in group 2 dogs, there was no significant difference in normal-zone flow between baseline and occlusion. Nevertheless, flow in these segments during occlusion (0.89 mL·min⁻¹·g⁻¹) remained within the normal flow range of a canine heart (0.8 to 1.2 mL·min⁻¹·g⁻¹). In border-zone and infarcted samples of both groups, there was a trend toward slightly higher myocardial blood flow at baseline in group 2 versus 1; however, this difference did not reach statistical significance.

### Table 2. Normalized Myocardial Blood Flow and 201 Tl and 99mTc-N-NOET Activities

<table>
<thead>
<tr>
<th>Group</th>
<th>Baseline MBF</th>
<th>Occlusion MBF</th>
<th>Injection MBF</th>
<th>2 hr Postinjection MBF</th>
<th>201 Tl Activity</th>
<th>NOET Activity</th>
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<tr>
<td>Group 1</td>
<td></td>
<td></td>
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<tr>
<td>Normal samples</td>
<td>0.97 ± 0.01</td>
<td>0.93 ± 0.02</td>
<td>0.96 ± 0.02</td>
<td>0.94 ± 0.02</td>
<td>0.95 ± 0.01</td>
<td>1.01 ± 0.01†</td>
</tr>
<tr>
<td>Border-zone samples</td>
<td>0.84 ± 0.03</td>
<td>0.48 ± 0.03*</td>
<td>1.15 ± 0.12</td>
<td>0.74 ± 0.06</td>
<td>0.65 ± 0.02</td>
<td>1.02 ± 0.05§</td>
</tr>
<tr>
<td>Infarct samples</td>
<td>0.80 ± 0.03</td>
<td>0.18 ± 0.04*</td>
<td>0.99 ± 0.17</td>
<td>0.50 ± 0.06*</td>
<td>0.39 ± 0.04</td>
<td>0.84 ± 0.08§</td>
</tr>
<tr>
<td>Group 2</td>
<td></td>
<td></td>
<td></td>
<td></td>
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<tr>
<td>Normal samples</td>
<td>0.98 ± 0.01</td>
<td>0.89 ± 0.01*</td>
<td>0.93 ± 0.02†</td>
<td>. . .</td>
<td>0.93 ± 0.01</td>
<td>0.94 ± 0.01†</td>
</tr>
<tr>
<td>Border-zone samples</td>
<td>0.90 ± 0.03</td>
<td>0.39 ± 0.04*</td>
<td>0.82 ± 0.03</td>
<td>. . .</td>
<td>0.64 ± 0.06</td>
<td>0.77 ± 0.02‡</td>
</tr>
<tr>
<td>Infarct samples</td>
<td>0.89 ± 0.01</td>
<td>0.13 ± 0.02*</td>
<td>0.70 ± 0.04†</td>
<td>. . .</td>
<td>0.43 ± 0.04</td>
<td>0.64 ± 0.03§</td>
</tr>
</tbody>
</table>

MBF indicates myocardial blood flow determined by radiolabeled microspheres; Injection, at the time of 201 Tl and 99mTc-N-NOET injection after either 1 hour (group 1) or 3 hours (group 2) of reperfusion. *P < 0.01 vs baseline; †P < 0.05 vs baseline; ‡P < 0.05 group 1 vs group 2; §P < 0.01 NOET vs 201 Tl.
significance. After the occlusion was set, myocardial blood flow in the border and infarct zones decreased significantly. There was no significant difference in the magnitude of the flow reduction in the central infarct zone during the occlusion between the 2 groups (0.18±0.04 versus 0.13±0.02 mL · min⁻¹ · g⁻¹). In group 1 dogs, there was complete reperfusion at 60 minutes after reflow at the time when ²⁰¹Tl and ⁹⁹mTc-N-NOET were injected, with no significant hyperemia. In contrast, group 2 dogs had significantly reduced flow at 3 hours after reflow when the tracers were injected.

**Comparison Between Myocardial ⁹⁹mTc-N-NOET and ²⁰¹Tl Activities**

**Group 1**
As shown in Figure 2, when ⁹⁹mTc-N-NOET was injected 1 hour after reperfusion followed by 2 additional hours of reperfusion flow, the final activity ratios in the border-zone and infarcted segments (1.02±0.05 and 0.84±0.08) were significantly higher than those of ²⁰¹Tl in the same segments (0.65±0.02 and 0.39±0.04). In addition, there was no significant difference between the final ⁹⁹mTc-N-NOET activity ratios and the myocardial blood flow ratios at the time of injection (1.15±0.02 and 0.99±0.17) in these same segments. There was no significant difference between ⁹⁹mTc-N-NOET and ²⁰¹Tl activities in the normal-zone segments.

**⁹⁹mTc-N-NOET Activity by In Vivo Imaging**
Figure 3 depicts mean ⁹⁹mTc-N-NOET defect count ratios (LAD/LCx) obtained from serial quantitative imaging in the group 1 dogs. Substantial myocardial ⁹⁹mTc-N-NOET uptake was observed initially, with a defect count ratio of 0.72±0.06 at 5 minutes after injection. In addition, significant redistribution resulted from differential myocardial clearance of ⁹⁹mTc-N-NOET over the next 2 hours, with defect count ratios of 0.71±0.08, 0.74±0.05, 0.77±0.04, 0.80±0.04, and 0.82±0.03 at 15, 30, 60, 90, and 120 minutes after injection, respectively (ANOVA P<0.001).

**Group 2**
In the 4 group 2 dogs that received ⁹⁹mTc-N-NOET and ²⁰¹Tl injections 3 hours after reperfusion, after which the dogs were immediately killed, ⁹⁹mTc-N-NOET activity was highly correlated with reperfusion flow at the time of injection (Figure 4). In contrast, there was poor correlation between ²⁰¹Tl activity and reperfusion flow. Note that in all 4 examples shown in Figure 4, ⁹⁹mTc-N-NOET activity tracked reperfusion flow, whereas ²⁰¹Tl activity in the same segments fell below the line of identity; ie, ²⁰¹Tl uptake underestimated reperfusion flow, even in segments with complete reperfusion (flow fraction ≥1.0). As expected, ⁹⁹mTc-N-NOET and ²⁰¹Tl activities were comparable in no-reflow regions (flow fraction <0.5), as well as in some segments with normal to high reperfusion flow that presumably came from either the normal or viable border zones. Figure 5 shows a photograph of a TTC-stained heart slice as well as ²⁰¹Tl and ⁹⁹mTc-N-NOET ex vivo images of the same heart slice from a representative dog. As can be seen in this figure, the large transmural infarct observed on the TTC photograph is reflected by the large perfusion defect observed on the ²⁰¹Tl image (defect count ratio=0.39). The defect on the ⁹⁹mTc-N-NOET image is less severe (defect count ratio=0.58) and reflects the degree of flow restoration in this region.

**Discussion**
The main finding of this study is that ⁹⁹mTc-N-NOET uptake in reperfused, acutely infarcted myocardium is significantly higher than that of simultaneously administered ²⁰¹Tl and reflects myocardial blood flow restoration at the time of tracer injection, rather than myocardial viability. This finding is unique when ⁹⁹mTc-N-NOET is compared with most of the other myocardial perfusion imaging agents administered in this setting.

Because 60% of ²⁰¹Tl uptake is related to Na⁺/K⁺-ATPase activity, necrotic cells with membrane disruption and loss in ATP content cannot retain ²⁰¹Tl. In cultured myocardial cells submitted to ischemia-like injury, as well as in isolated rat hearts under hypoxic and energy-depletion conditions, ²⁰¹Tl uptake is dramatically reduced compared with controls. This
decrease in $^{201}$Tl uptake is correlated to the amount of lactate dehydrogenase efflux and therefore to cell necrosis. Similarly, myocardial uptake of $^{99m}$Tc-sestamibi is depressed in infarcted myocardium. This decrease is thought to be related to the diminution of sarcolemmal and mitochondrial membrane potentials. In a similar canine model of reperfused, acutely infarcted myocardium, $^{99m}$Tc-sestamibi defect magnitude on planar images correlated with the extent of necrosis as assessed by histochemical analysis rather than reperfusion flow. Similarly, the ischemic-to-normal $^{99m}$Tc-sestamibi myocardial uptake ratio by gamma-well counting correlated with extent of necrosis and was considerably lower than reperfusion flow. Similar findings have been observed with $^{99m}$Tc-tetrofosmin.

The exact uptake mechanism of $^{99m}$Tc-teboroxime, a highly lipophilic perfusion agent, is unknown, but it is thought to bind nonspecifically to cell membranes. Like $^{99m}$Tc-N-NOET, $^{99m}$Tc-teboroxime uptake is not dependent on cellular metabolism and viability. However, results from studies examining the uptake of this agent in reperfused acute myocardial infarction are conflicting. Using a rabbit model, Heller et al found that $^{99m}$Tc-teboroxime uptake in this setting reflects myocardial blood flow and is independent of myocardial viability. Conversely, in a study in pigs by Abraham et al, the infarct-to-normal $^{99m}$Tc-teboroxime uptake ratio (0.85±0.32) is intermediate between myocardial blood flow ratios during occlusion (0.25±0.35) and after reperfusion (1.54±0.94) and thus could be dependent on both flow and viability.

Figure 4. Individual scatterplots of $^{201}$Tl (○) and $^{99m}$Tc-N-NOET (●) activities vs flow at time of tracer injection after reflow in 4 group 2 dogs. In all 4 dogs, $^{99m}$Tc-N-NOET activity was highly correlated with reperfusion flow, even though some segments were nonviable. In contrast, $^{201}$Tl activity fell below identity line, and there was poor correlation with flow in reperfused, nonviable segments. Tracer activities were comparable in nonreperfused (no reflow) and in reperfused, viable segments.

Figure 5. TTC-stained heart slice (left) and $^{201}$Thallium (middle) and $^{99m}$Tc-N-NOET (right) images of same heart slice. Prominent anteroseptal defect observed on $^{201}$Thallium image reflects large transmural infarct on TTC photograph. In contrast, there was substantial heterogeneous $^{99m}$Tc-N-NOET uptake in same anteroseptal region despite presence of severe infarction. Heterogeneous pattern of $^{99m}$Tc-N-NOET uptake resulted from a similar pattern of reperfusion flow.
viability. These divergent findings may result from species differences or from differing delays between tracer injection and image acquisition or sacrifice, a potentially major source of variation due to the rapid myocardial clearance of this agent.\textsuperscript{12}

Although the precise mechanism of \textsuperscript{99m}Tc-N-NOET myocardial uptake is unknown, this radiopharmaceutical has been demonstrated to associate with the lipophilic components of the myocytes,\textsuperscript{26} to have an extremely high myocardial retention in isolated perfused rabbit hearts,\textsuperscript{27} and to undergo increased clearance after severe cell membrane damage.\textsuperscript{27} In cultured newborn rat cardiomyocytes, \textsuperscript{99m}Tc-N-NOET uptake has also been demonstrated to be independent of cellular metabolic status or ATP content.\textsuperscript{16}

Thus, the data from the present study could be explained by the fact that in reperfused, acutely infarcted myocardium, \textsuperscript{99m}Tc-N-NOET may bind to cell membranes, even if the myocytes are significantly damaged by ischemia and reperfusion. Within the first hour after reperfusion after acute myocardial infarction, microvascular integrity may permit almost complete reflow, as demonstrated in the present study.\textsuperscript{28} This reflow allows \textsuperscript{99m}Tc-N-NOET to gain access to myocytes that may be in the phase of irreversible cell injury.\textsuperscript{4} Another potential explanation for high \textsuperscript{99m}Tc-N-NOET uptake in zones of ongoing necrosis is binding of the tracer to neutrophils that aggregate in reperfused myocardium, ultimately contributing to the no-reflow phenomenon.\textsuperscript{28} The experimental findings of the present study do not necessarily contradict the diminution in \textsuperscript{99m}Tc-N-NOET uptake observed in patients with chronic myocardial infarction.\textsuperscript{29} In this setting, no myocyte membranes available for \textsuperscript{99m}Tc-N-NOET binding exist, and fibrosis and scar prevent the uptake of \textsuperscript{99m}Tc-N-NOET.

**Study Limitations**

In the present study, \textsuperscript{99m}Tc-N-NOET and \textsuperscript{201}Tl were injected either 1 or 3 hours after reperfusion. It is possible that mild hyperemia was present at the time when the tracers were injected, which may have resulted in excess tracer uptake. However, it is important to point out that both tracers were injected under the same flow conditions, yet their uptake patterns were quite different.

**Clinical Implications**

Our experimental model of reperfused, acutely infarcted myocardium suggests that \textsuperscript{99m}Tc-N-NOET uptake reflects more the magnitude of flow restoration rather than the extent of myocardial salvage. Clinically, it remains to be determined whether \textsuperscript{99m}Tc-N-NOET imaging performed 15 to 20 minutes after injection in the setting of thrombolysis in acute myocardial infarction can prove useful in assessing vessel patency. Another clinical implication of this study is that \textsuperscript{99m}Tc-N-NOET will not be a valid viability agent in attempts to assess the degree of salvage very early after coronary reperfusion. Myocardial perfusion agents such as \textsuperscript{201}Tl, \textsuperscript{99m}Tc-sestamibi, and \textsuperscript{99m}Tc-tetrofosmin would appear to be preferable in this setting. Finally, studies comparing radionuclide imaging using \textsuperscript{99m}Tc-N-NOET with other noninvasive techniques, such as contrast echocardiography and contrast MRI imaging, for accurate assessment of the degree of reflow after reperfusion in the setting of acute myocardial infarction appear warranted.

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**References**


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