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# Demonstration of Viable, Stunned Myocardium with Technetium-99m-Sestamibi

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Delayed improvement of left ventricular contractile function in the setting of acute ischemia followed by reperfusion ("stunned myocardium") has been observed in a number of clinical scenarios, and may have important clinical implications. At present, there are no widely accepted techniques available to demonstrate its presence. We report a case in which a rest  $^{99m}\text{Tc}$ -sestamibi scan performed 12 hr after thrombolytic therapy in the setting of acute myocardial infarction demonstrated viable myocardium in a region that was akinetic by contrast ventriculography. After surgical revascularization, follow-up  $^{99m}\text{Tc}$ -sestamibi images showed normal perfusion and radionuclide ventriculography demonstrated normal left ventricular function. Demonstration of preserved  $^{99m}\text{Tc}$ -sestamibi myocardial uptake in the infarct zone despite an extensive region of akinesis by contrast ventriculography predicted the recovery of left ventricular function after revascularization in this case. This suggests that perfusion imaging with  $^{99m}\text{Tc}$ -sestamibi early after myocardial reperfusion can detect stunned myocardium and thus facilitate the decision-making process regarding management of such patients.

**Key Words:** stunned myocardium; technetium-99m-sestamibi

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The term "stunned myocardium" was introduced by Braunwald and Kloner in 1982 to describe reversible left ventricular contractile dysfunction in the setting of acute ischemia followed by reperfusion (1). The time course of postreperfusion improvement in left ventricular systolic function may span hours to weeks. This phenomenon has been demonstrated in a variety of clinical situations, including reperfusion with thrombolytic agents during acute myocardial infarction (2), in the recovery phase after exercise-induced ischemia (3), in the postoperative period after coronary artery bypass surgery and cardioplegia administration (4), in the setting of unstable angina (5) and after coronary angioplasty (6).

Despite recognition of this phenomenon, there are no widely accepted techniques for prediction of reversibility of ventricular dysfunction in the posts ischemic period. De-

tection of the presence of stunned myocardium after acute myocardial ischemia has important clinical implications, as such information may impact on decisions regarding myocardial revascularization. At present, the only reliable method for detecting its presence rests on demonstration of improvement of left ventricular function 7-10 days after the event.

We report a case in which viable, "stunned" myocardium was identified 12 hr after thrombolytic therapy by retained uptake of  $^{99m}\text{Tc}$ -sestamibi in a region that was akinetic by contrast ventriculography.

## CASE REPORT

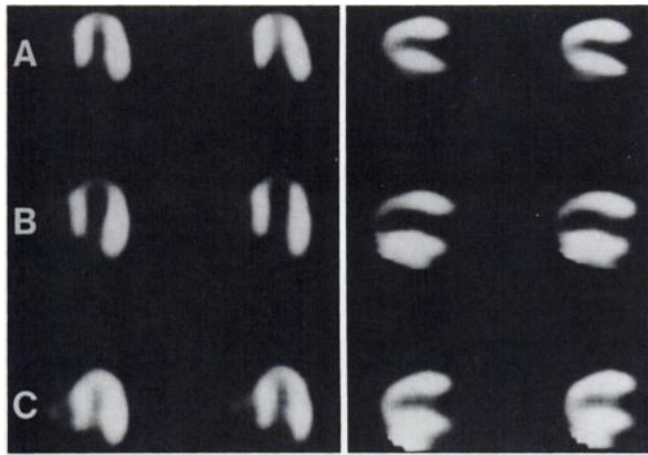
Our patient was a 52-yr-old male with a history of hypertension, hyperlipidemia and coronary artery disease (prior percutaneous transluminal coronary angioplasty of the left anterior descending artery), who developed recurrent angina pectoris 4 mo after the initial angioplasty. Coronary angiography demonstrated a 70% stenosis of the proximal left anterior descending artery at the site of prior angioplasty, and directional coronary atherectomy was performed with an excellent angiographic result and no residual luminal stenosis. Four days after the procedure, the patient experienced recurrence of exertional angina, culminating in re-admission to the hospital after an episode of chest pain at rest. A rest  $^{99m}\text{Tc}$ -sestamibi scan performed at this time (Fig. 1A) demonstrated a small apical perfusion defect.

Several hours later, the patient developed severe chest discomfort. A 12-lead electrocardiogram revealed anterior ST segment elevations (Fig. 2). Streptokinase (1.5 million units) was administered intravenously, with resolution of chest pain and ST elevations. Twelve hours later, a second rest  $^{99m}\text{Tc}$ -sestamibi scan was performed, which demonstrated an interval increase in left ventricular dimension and an increase in the extent of the initial perfusion defect;  $^{99m}\text{Tc}$ -sestamibi uptake was preserved in the majority of the left anterior descending territory (Fig. 1B). Coronary angiography performed that same day revealed a 99% luminal stenosis of the proximal left anterior descending artery with extensive intimal dissection beginning at the site of atherectomy and extending into the first septal perforator branch. Contrast ventriculography demonstrated anterior, septal and apical akinesis (Fig. 3).

The patient developed recurrent angina later that day and underwent emergency coronary artery bypass surgery (saphenous vein graft to the left anterior descending artery). Peak creatine kinase prior to surgery was 1227 IU/liter. Postoperative recovery was uneventful, and a rest  $^{99m}\text{Tc}$ -sestamibi study obtained 1 wk after surgery showed normal perfusion (Fig. 1C). A first-pass radionuclide ventriculogram obtained at this time demonstrated

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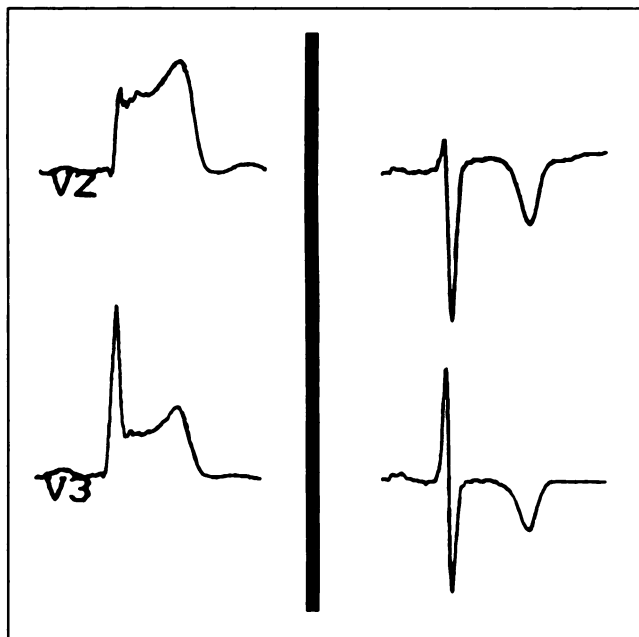


**FIGURE 1.** Rest  $^{99m}\text{Tc}$ -sestamibi images. Horizontal long-axis (left panel) and vertical long-axis (right panel) views. (A) Baseline images show a small apical perfusion defect. (B) After thrombolytic therapy for transmural anterior myocardial ischemia, cavity dilatation and a larger apical defect are seen; septal and anterior wall perfusion are preserved. (C) After bypass grafting of the left anterior descending artery, perfusion is normal.

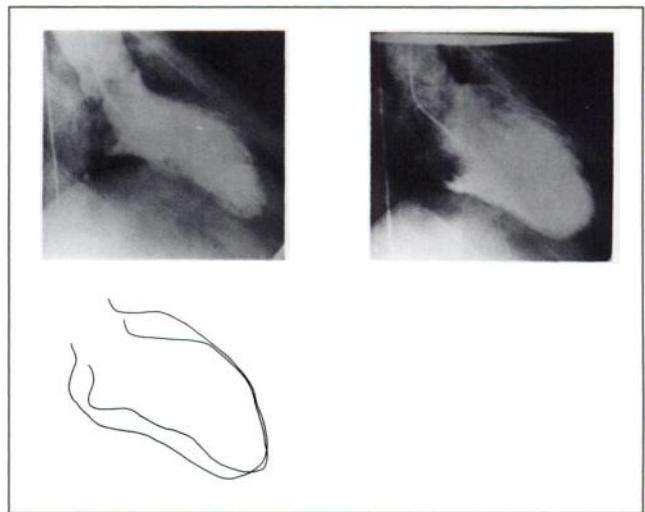
normal global and regional left ventricular size and function. The electrocardiogram after revascularization showed anterior T-wave inversions, but no Q-waves were present (Fig. 2).

## DISCUSSION

Identification of viable myocardium in the postinfarction period is of critical importance to clinical decision-making, yet information is lacking as to the optimal method for its



**FIGURE 2.** Electrocardiogram leads V2 and V3 demonstrate marked ST segment elevation (left panel), signifying transmural anterior myocardial ischemia. After thrombolytic therapy (right panel), ST segments have returned to baseline and T-waves are inverted; no Q-waves are detected.



**FIGURE 3.** End-systolic (left panel) and end-diastolic (right panel) frames from contrast ventriculogram. Diagram (bottom panel) displays end-systolic and end-diastolic ventricular silhouettes, demonstrating anteroapical and septal akinesis.

detection. Radionuclide imaging techniques have been used to demonstrate viable, reversible left ventricular dysfunction (“hibernating” myocardium) in patients with chronic coronary artery disease (7). The utility of these techniques in the early postinfarction period has not been well studied. Dobutamine echocardiography has been reported to identify viable myocardium in patients studied 1 wk after thrombolytic therapy for myocardial infarction (8), as evidenced by improvement in myocardial thickening and regional wall motion. Earlier detection is preferable, however, and catecholamine infusion in the early postinfarction period may potentially aggravate myocardial ischemia and precipitate arrhythmias.

Technetium-99m-sestamibi is a radiopharmaceutical which has favorable physical properties (a shorter half-life and higher photon energy) in comparison to thallium-201, resulting in better image quality. In addition,  $^{99m}\text{Tc}$ -sestamibi demonstrates minimal redistribution, allowing for uncoupling of the time of injection and the time of imaging. Experimental evidence suggests that  $^{99m}\text{Tc}$ -sestamibi uptake and retention reflect cell viability (9).

Perfusion imaging with  $^{99m}\text{Tc}$ -sestamibi has been used to demonstrate myocardial salvage with thrombolytic therapy (10,11). In a group of 32 patients, Christian et al. (11) measured perfusion defect size at hospital discharge (median 7 days postinfarction) and compared these values to measurements of ejection fraction and regional wall motion (by radionuclide ventriculography) at discharge and 6 wk later. A close correlation for the group as a whole between perfusion defect size and 6-wk ejection fraction was demonstrated. However, five of these patients had a significant increase in ejection fraction between discharge and 6 wk. In this subgroup, the ejection fraction at discharge was

significantly lower than that predicted on the basis of perfusion defect size, but the 6-wk ejection fraction correlated well with that predicted by perfusion imaging. Thus,  $^{99m}\text{Tc}$  scintigraphy at 1 wk accurately predicted the presence of viable myocardium in this subgroup.

In the patient being discussed, the perfusion scan performed 12 hr after myocardial infarction (Fig. 1B) demonstrated cavity dilatation and extension of the left ventricular apical perfusion defect in comparison to the baseline scan; perfusion was preserved to the majority of the left anterior descending artery territory. The contrast ventriculogram performed that same day provided discordant information, however, demonstrating akinesis of the anterior wall, septum and apex (Fig. 3). The accuracy of  $^{99m}\text{Tc}$ -sestamibi uptake with respect to preoperative viability assessment is corroborated by the recovery of normal left ventricular contractile function; of note, left ventricular size and perfusion were essentially normal on the postoperative scan.

It is interesting to note that the rest  $^{99m}\text{Tc}$ -sestamibi scan after thrombolysis displayed a perfusion defect that was no longer present after revascularization. The presence of this defect was likely secondary to inadequate tracer delivery to the distal left anterior descending artery territory, due to a high-grade coronary stenosis. With revascularization, distal flow was restored and tracer uptake was present. This illustrates the dependence of myocardial tracer uptake (and by inference, viability detection) on adequate tracer delivery. In addition, the extent of the defect may have appeared greater on the post-thrombolysis images due to cavity dilatation and resultant apical thinning. This abnormality may be accentuated by the effects of akinesis on count recovery. This latter phenomenon has been addressed by gating images with the cardiac cycle.

In the present case, preserved  $^{99m}\text{Tc}$ -sestamibi myocardial uptake predicted the improvement of left ventricular contractile function after revascularization despite an extensive region of akinesis on contrast ventriculography. This suggests that early postinfarction perfusion imaging

can detect stunned myocardium and thus facilitate the decision-making process regarding management of such patients. Thallium-201 may be useful in this setting as well, and further study of both  $^{99m}\text{Tc}$ -sestamibi and  $^{201}\text{Tl}$  for the clinical detection of viable myocardium in such settings is warranted.

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