Assessment of Myocardial Perfusion Defect Size After Early and Delayed SPECT Imaging with Technetium-99m-Hexakis 2-Methoxyisobutyl Isonitrile After Stress

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It is thought that the distribution of ^{99m}Tc-sestamibi undergoes minimal change during the first 4 hr after injection. Thus [^{99m}Tc] sestamibi unlike ^{201}Tl should not demonstrate significant redistribution in ischemic myocardium. We tested this assumption by quantifying perfusion defect size in early and delayed clinical SPECT images obtained after exercise stress or after dipyridamole infusion using an automated algorithm. Twenty patients with coronary artery disease aged 55 ± 10 yr (13 male and 7 female) underwent stress imaging. Technetium-99m-sestamibi was injected at peak exercise stress in 12 patients and after an intravenous infusion of dipyridamole in 8 patients; SPECT images were obtained 1 and 4 hr later. All patients underwent rest imaging with a second dose of ^{99m}Tc-sestamibi 1–3 days after the stress studies. Total left ventricular mass and left ventricular defect mass were quantified with an automated algorithm previously validated in animal and patient studies. Estimates of total left ventricular mass from studies obtained 1 hr after stress (341 ± 90 g) were comparable to values obtained after 4 hr (336 ± 89 g), with a correlation of $r = 0.97$; $p < 0.0001$. The left ventricular defect size 1 hr after exercise or dipyridamole infusion (149 ± 74 g) was similar to that observed after 4 hr (151 ± 73 g). The two measurements of hypoperfusion with stress were highly correlated ($r = 0.91$; $p < 0.0001$) and were significantly larger than the defect size at rest (121 ± 70 g). These observations support the conclusion that ^{99m}Tc-sestamibi does not redistribute significantly in ischemic myocardium between 1 and 4 hr after injection.

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Thallium-201 has been used extensively to assess myocardial perfusion. Recently ^{99m}Tc-hexakis 2-methoxyisobutyl isonitrile tsestamibi) has been developed and tested as a substitute for ^{201}Tl in myocardial perfusion studies. Technetium-99m-sestamibi has radiation dosimetry and physical imaging characteristics that are advantageous when compared to ^{201}Tl (*1,2*). In addition, a simultaneous ventriculogram can be obtained with the same ^{99m}Tc-sestamibi injection used for a myocardial perfusion study (*3,4*).

It is thought that ^{99m}Tc-sestamibi is taken up by viable myocardium in proportion to coronary blood flow and undergoes little redistribution after its cellular uptake (*5*). Consequently, ^{99m}Tc-sestamibi injected during stress should accurately reflect peak stress perfusion whether imaging is performed soon after injection of the isotope or several hours later. However, we recently evaluated stress myocardial perfusion imaging with both ^{201}Tl and ^{99m}Tc-sestamibi in 24 patients with coronary artery disease (*6*). We noted that the stress ^{99m}Tc-sestamibi defect size was on average 19% smaller than that observed in the same patients undergoing stress ^{201}Tl imaging. In contrast, the defect size seen in the resting ^{99m}Tc-sestamibi images was similar to that seen in the ^{201}Tl studies obtained after 3–4 hr of redistribution. These discrepancies in stress-induced ^{201}Tl and ^{99m}Tc-sestamibi defect size have been confirmed by Maublant et al. in patients with coronary artery disease (*7*). In addition, Eisner and coworkers (*8*) have shown that the amount of experimentally induced ischemia in dogs is more accurately represented by ^{201}Tl defect size than by ^{99m}Tc-sestamibi.

Possible explanations for the discrepancy in stress-induced defect size we observed include differences in the uptake of the two isotopes by ischemic myocardium or the possibility that ^{99m}Tc-sestamibi undergoes some redistribution during ischemia. The manufacturer of ^{99m}Tc-sestamibi claims that the perfusion pattern obtained with the compound is stable in clinical work, and that the images obtained soon after isotope injection are similar to those seen after several hours have elapsed (*9*). However, some redistribution of ^{99m}Tc-sestamibi has been noted in

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reperfused canine myocardium (*10*), and two studies in cardiac patients have documented diminishing ischemic-to-normal wall ratios over 6 hr (*11,12*). Thus, the aim of our study was to determine the presence or absence of significant redistribution in clinical ^{99m}Tc-sestamibi SPECT images obtained 1 and 4 hr after pharmacologic or exercise stress.

METHODS

Patient Population

Twenty patients (mean age 55 ± 10 yr) with coronary artery disease who were referred to the nuclear medicine division for stress ^{99m}Tc-sestamibi imaging were enrolled into the study. There were 13 men and 7 women, all of whom gave informed consent for participation in this investigation. Eight patients had a prior myocardial infarction, two had coronary bypass surgery and four had percutaneous transluminal coronary angioplasty. Twelve patients were studied after maximal treadmill exercise stress and the remaining eight after an intravenous infusion of dipyridamole (0.56 mg/kg infused over 4 min). All patients were imaged 1 and 4 hr after the injection of 20 mCi (740 MBq) ^{99m}Tc-sestamibi at maximal stress or 1 min after completing dipyridamole infusion. A second injection of 20 mCi of ^{99m}Tc-sestamibi was given 24–72 hr later for resting imaging.

SPECT Imaging

Three SPECT studies were obtained in all patients. Early imaging was performed 1 hr after exercise stress or an intravenous dipyridamole infusion. Delayed imaging was performed 4 hr thereafter and rest imaging was performed 24–72 hr later. The acquisition protocol was identical for all three studies.

For SPECT acquisition, a rectangular head rotating gamma camera (SophyCamera DSX, Sopha Medical, Columbia, MD) equipped with a general-all-purpose, parallel-hole collimator was used. A 180° rotation from 45° left posterior oblique to 45° right anterior oblique projection was accomplished in 6° steps. At each step, a 40-sec acquisition was performed. The spectrometer was centered over the 140 keV emissions of ^{99m}Tc with a 20% window. To obtain maximal image counts, the camera rotation was allowed to follow the body contour of the patient using the manufacturer's software. Data processing was performed with a dedicated computer by means of a backprojection reconstruction algorithm with Butterworth (6/16) filtering and no attenuation correction. Transaxial slices were then reconstructed and realigned into frontal and sagittal sections. Only realigned slices cut perpendicular to the long-axis of the left ventricle (short-axis slices) were used for analysis in this study. No background subtraction was performed.

Estimation of Left Ventricular Mass and Defect Mass from SPECT

Estimated total left ventricular (LV) mass was determined using a method that we previously validated for ^{201}Tl in an animal model (*13*) and in patient studies (*14*). Left ventricular mass was estimated from each of the 6–9 short-axis slices in the SPECT studies. The only operator intervention was the definition of the approximate center of the left ventricle from one mid-ventricular short-axis slice. From this approximate center, 60 x-y count profiles every 6° were created for each short-axis slice. The maximal upslope and downslope in each radial profile was determined and was utilized to define the epicardial and endocardial edges of each short-axis slice. These points were then connected, smoothed and constrained to be circular. Myocardial mass was then estimated from the number of voxels within the epicardial and endocardial borders multiplied by the volume of each voxel which was then multiplied by 1.05 g/cm^3 (the weight of myocardial tissue). Areas of significant hypoperfusion were defined as those volume elements within the computer-defined myocardium in each short-axis slice that fell below 45% of the maximal counts in the ventricle. Details of the algorithm can be found in reference (*13*).

We have previously demonstrated that estimates of LV mass in canine hearts from a ^{201}Tl SPECT study (*13*) were highly correlated with autopsy heart weight ($r = 0.994$ over a range of 62–152 g). Estimates of LV mass in patient ^{201}Tl SPECT studies were also highly correlated with LV mass determinations made from contrast angiography ($r = 0.96$ over a range of 125–445 g) (*14*). Estimates of LV mass were reproducible with a 5.9% mean absolute difference between a single ^{201}Tl stress and redistribution study pair. An average absolute difference of 7.2% was seen when comparing the LV mass of two redistribution ^{201}Tl studies performed approximately 5 wk apart. Our previous work with ^{201}Tl SPECT has also demonstrated that the mass of infarcts determined histologically and biochemically correlates well with estimates of infarct weight defined with this technique ($r = 0.94$). Estimated LV mass obtained from ^{99m}Tc-sestamibi perfusion studies has correlated highly with data obtained in the same patient using ^{201}Tl imaging (*6*).

The images of individual short-axis slices with the computer-generated outlines of the total and defect mass of the left ventricle were also recorded as hard copy to ensure the validity of the automated edge detection algorithm. Total myocardial mass and defect mass were calculated by observers unaware of the patient's clinical findings. All values are expressed as mean ± 1 s.d. Regression analysis, correlation coefficients as well as paired and unpaired Student's t-tests were performed using standard techniques.

RESULTS

Image Characteristics

A visual inspection of the short-axis SPECT images showed some degree of ischemia in all patients studied. A typical patient study is shown in Figure 1. The defect size noted in the 1- and 4-hr post-stress SPECT images were visually similar with the exception of one patient who after a stress study appeared to have a larger defect at the time of the 1-hr images when compared with the 4-hr study. In general, the early images, obtained 1 hr after stress, had more liver and lung background than the 4-hr images. As a result, the contrast between the heart and the adjacent organs was higher in the 4-hr images. A relative disadvantage of delayed imaging was the lower count rate caused by physical decay of ^{99m}Tc, with 63% of the isotope activity remaining after 4 hr.

Estimated Left Ventricular Mass and Left Ventricular Defect Mass

The type of stress (exercise versus pharmacologic) utilized did not affect the outcome of this study (Table 1). The average estimated LV mass was 341 ± 90 g in the 1-

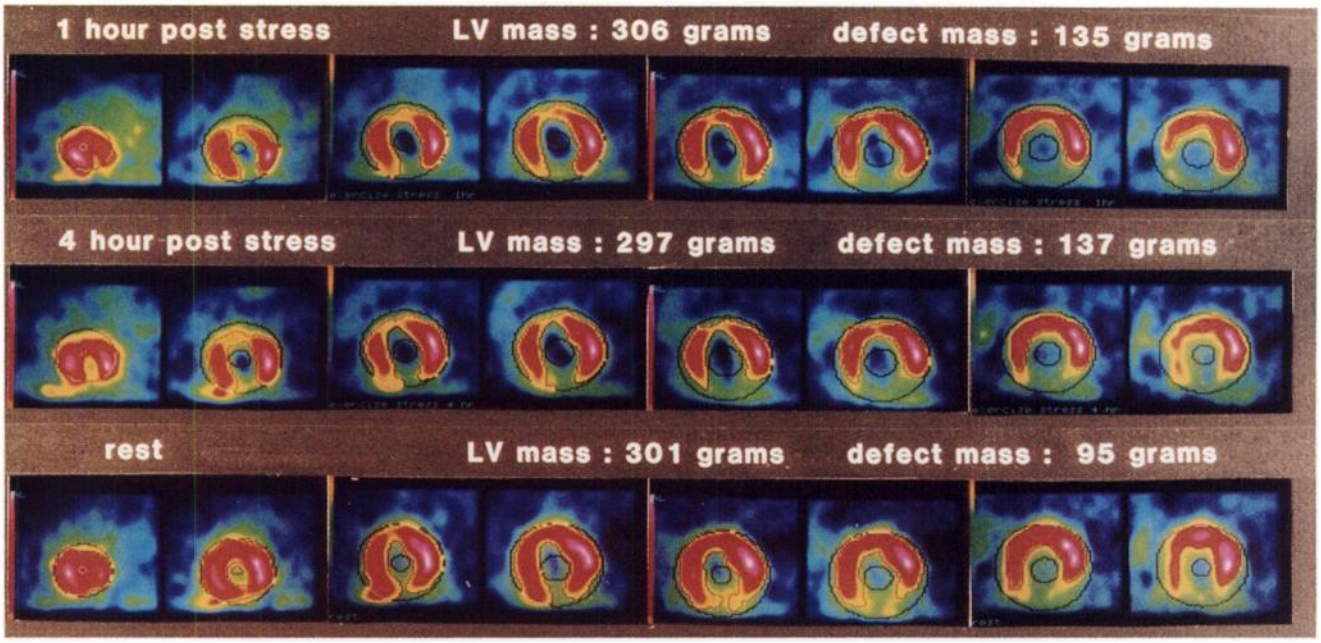

FIGURE 1. An example of a series of patient images from this study. Left ventricular mass was reproducibly estimated from the 1- and 4-hr post-stress images. There is minimal difference in the defect size seen at 1 hr (135 g) and 4 hr (137 g) after stress. Hypoperfusion is considered present at point 45% below the maximal counts in the image. This is depicted by the transition from red to yellow. Epicardial and endocardial outlines are in black.

hr images and was not significantly different from the LV mass estimated from the 4-hr studies (336 ± 89; $p = 0.89$) (Table 1). The LV mass estimates from the 1- and 4-hr post-stress images were highly correlated ($r = 0.97$; $p < 0.0001$) (Fig. 2). The estimated LV mass determined from resting SPECT images averaged 331 ± 79 g. This value was not significantly different from estimates of LV mass calculated from either the 1- or 4-hr post-stress images.

The mean estimated defect mass of the ^{99m}Tc-sestamibi studies was 149 ± 74 g in the 1-hr post-stress studies and was 151 ± 73 g in the 4-hr post-stress studies. The correlation between perfusion defect size determined 1 and 4 hr after stress yielded an r value of 0.92; $p < 0.0001$ (Fig. 3). The 1-hr and 4-hr post-stress estimates of defect size were significantly different from estimates of defect mass obtained from the resting SPECT images (121 ± 70 g; $p <$ 0.01). Defect size results are also presented as a percentage of the myocardium involved (Table 2).

TABLE 1
Left Ventricular Mass Estimates from SPECT Imaging Performed 1 and 4 Hours After Stress and at Rest

	LV mass estimates in grams (mean ± s.d.)		
	1 hr	4 hr	Rest
Total LV mass			
Exercise stress	368 ± 93	368 ± 91	367 ± 72
Dipyridamole stress	300 ± 73	289 ± 65	276 ± 58
Both groups	341 ± 90	336 ± 89	331 ± 79
Defect LV mass			
Exercise stress	163 ± 82	168 ± 82	140 ± 77
Dipyridamole stress	126 ± 58	126 ± 52	95 ± 51
Both groups	149 ± 74	151 ± 73	121 ± 70

DISCUSSION

Since redistribution of ^{201}Tl occurs soon after the injection of the isotope, it is important to begin imaging as soon as possible after a stress ^{201}Tl study. Flexibility in scheduling imaging studies is one of the advantages of ^{99m}Tc-sestamibi when compared to ^{201}Tl. Okada et al., using a fixed stenosis in an animal model, found that the clearance of ^{99m}Tc-sestamibi was similar in ischemic and normal myocardium with a range of 15–20 hr (*5*). Okada's findings are consistent with the current use of this imaging agent. Technetium-99m-sestamibi is administered during maximal stress to assess myocardial perfusion, and the actual imaging routinely occurs several hours after the injection of the isotope.

However, in an isolated rabbit heart model, Marshall et al. demonstrated a faster washout rate of ^{99m}Tc-sestamibi, with a mean half-time of 68–129 min (*15*). If this latter model is more representative of events in the human myocardium, the possibility of significant ^{99m}Tc-sestamibi redistribution may be a real concern when imaging patients with ischemic heart disease. For example, Li et al. demonstrated redistribution of ^{99m}Tc-sestamibi into reperfused myocardium, although this was slower and less complete than ^{201}Tl (*10*). Our previous observation that stress ^{201}Tl images have larger defects than those seen in images obtained from the same patients using ^{99m}Tc-sestamibi would be consistent with either redistribution or a

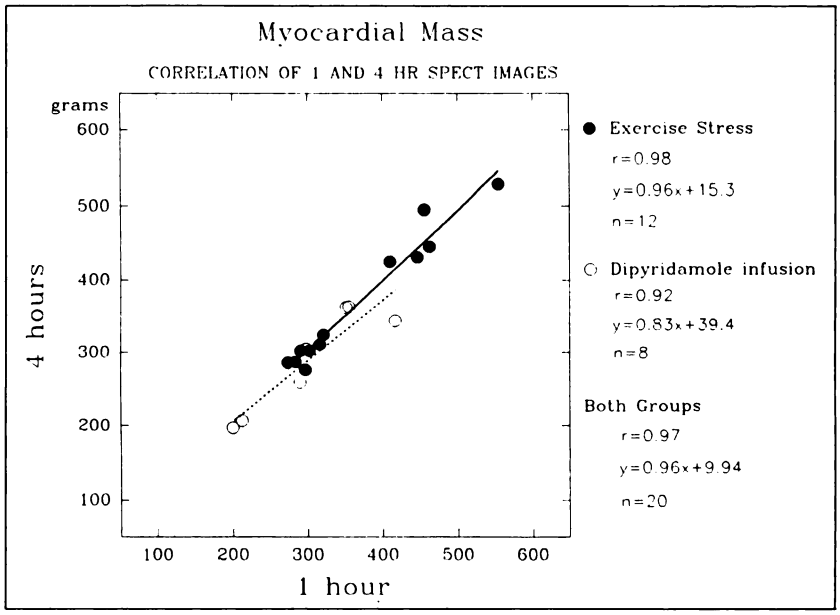

FIGURE 2. Estimated myocardial mass from images obtained 1 and 4 hr after exercise or pharmacologic stress are depicted. The solid line represents the regression equation for exercise stress studies; the dashed lines represent dipyridamole studies. There is no significant difference between the slopes of the two linear regressions.

difference in cellular uptake when ^{201}Tl is compared to the technetium imaging agent.

Franceschi (*11*) as well as Taillefer et al. (*12*) have examined the ischemic-to-normal wall ratios of ^{99m}Tc-sestamibi in patients with significant coronary artery disease. Both groups noted that washout from ischemic tissue occurred at a slower rate than from normal myocardium. This difference could tend to decrease visual differences between normal and ischemic myocardium with time. Taillefer et al. evaluated early and delayed post-stress imaging with ^{99m}Tc-sestamibi using planar imaging and a visual interpretation (*12*). They found that few ischemic segments (2 of 48) were missed with delayed imaging (3 hr) when compared to early imaging (1 hr). However, based on the data from ischemic-to-normal myocardial ratios, they concluded that early planar imaging was preferable for the diagnosis of ischemia.

We looked for clinically significant temporal changes in perfusion in ^{99m}Tc-sestamibi SPECT images using an automated determination of LV mass and hypoperfused myocardial mass. The technique is reproducible and is similar to a visual interpretation using precise, uniform definitions for defect size. We have used this technique previously to demonstrate differences in stress ^{201}Tl and ^{99m}Tc-sestamibi defect size.

However, in this investigation, we found no significant difference in defect size when routine clinical ^{99m}Tc-sestamibi SPECT images were obtained 1 and 4 hr after maxi-

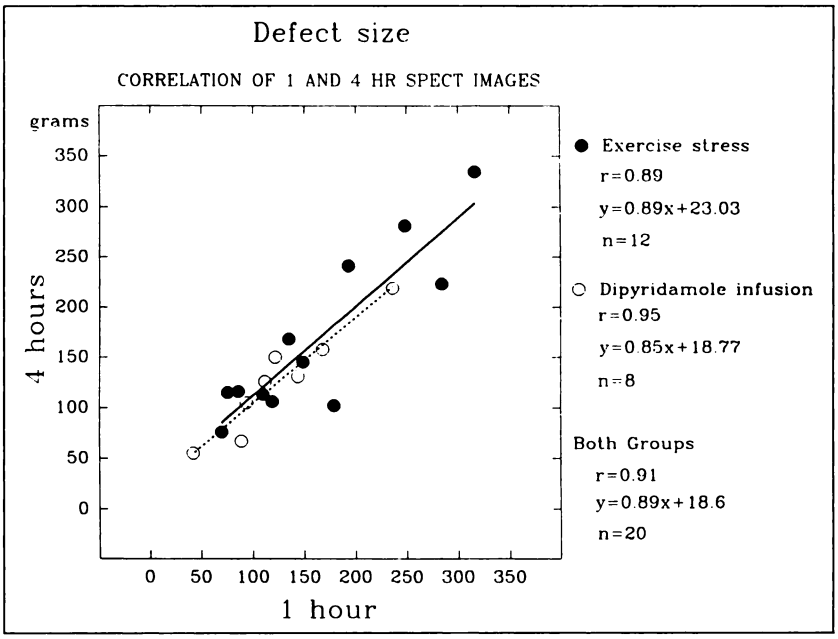

FIGURE 3. Estimates of defect size 1 and 4 hr after stress are shown. There is no significant difference between the slope of the exercise stress regression line (solid line) and that of dipyridamole (dashed line).

TABLE 2
Left Ventricular Defect Size as Percent of Total Mass
(mean values ± s.d.)

Defect	Defect size (%)		
	1 hr	4 hr	Rest
Exercise	40 ± 13	40 ± 10	31 ± 8
Dipyridamole	46 ± 15	50 ± 17	41 ± 18

mal exercise or pharmacologic stress. With only one exception, the estimated defect size obtained from the 1 and 4-hr post-stress images were within 10% of each other. There was no trend toward overestimation or underestimation of defect size when the 1- and 4-hr post-stress were analyzed. Our previous demonstration of differences in exercise defect size when ^{201}Tl images were compared to ^{99m}Tc-sestamibi (*6*) does not appear to be due to redistribution between 1 and 4 hr after stress. However, after this manuscript was prepared, evidence for redistribution of ^{99m}Tc-sestamibi in the initial 60 min after exercise stress has been presented (*16*). This possibility was not evaluated in our investigation.

In this study, data were obtained after maximal treadmill stress testing as well as after dipyridamole infusions. Both methods for creating hypoperfusion were used to broaden the clinical applicability of our results (*17*). Neither stress testing procedure was associated with significant differences in total LV mass or hypoperfused LV mass when 1- and 4-hr post-stress studies were examined (Figs. 2 and 3).

Alternative Explanations for Discrepancies in ^{201}Tl and ^{99m}Tc-Sestamibi Defect Size

Our previous evaluation of ^{201}Tl and ^{99m}Tc-sestamibi defect size utilized a SPECT reconstruction and a LV mass processing routine which were designed for ^{201}Tl SPECT studies. The substantial differences in imaging properties between ^{201}Tl and ^{99m}Tc-sestamibi might necessitate unique reconstruction techniques such as different filtering. However, results from redistribution ^{201}Tl imaging mirrored data obtained from the resting ^{99m}Tc-sestamibi studies. Thus, for rest/redistribution imaging, the methods we used for determining LV mass and LV defect size appeared to be appropriate for both isotopes.

The higher counting rates noted in exercise ^{99m}Tc-sestamibi studies might have allowed for more "shine through" or a "blooming effect" which could result in smaller image defects. However, the average maximum stress ^{99m}Tc-sestamibi count rate (903 counts/pixel) was only 22% higher than the maximal image counts found in the resting ^{99m}Tc-sestamibi studies that had defect sizes similar to the redistribution ^{201}Tl images. The use of different isocount values to define hypoperfusion did not alter the difference in stress defect size when comparing ^{99m}Tc-sestamibi with ^{201}Tl.

Another possible explanation for our previous results is the observation by Leppo and Meerdink (*18*) that net myocardial extraction of ^{201}Tl is significantly different from ^{99m}Tc-sestamibi in the isolated perfused rabbit heart. The notion that ^{201}Tl and ^{99m}Tc-sestamibi are handled differently during conditions of ischemia is also supported by a recent report by Maubant et al. (*19*). They noted that cyanide (an inhibitor of the respiratory chain) in combination with iodoacetate (an inhibitor of glycolysis) resulted in a significant reduction in ^{201}Tl uptake in cultured rat myocardial cells, whereas the two agents had no effect on ^{99m}Tc-sestamibi uptake either alone or administered together. Since ischemia alters intracellular metabolic events, the reduced uptake of ^{201}Tl noted experimentally could explain the presence of larger stress-induced ^{201}Tl defects than was seen with ^{99m}Tc-sestamibi. Thus, differences in tracer kinetics during or subsequent to stress-induced ischemia may cause differences in ^{201}Tl and ^{99m}Tc-sestamibi defects.

SUMMARY

We have previously demonstrated differences in exercise stress defect size when ^{201}Tl is compared with ^{99m}Tc-sestamibi. Our current data using a quantitative analysis performed 1 and 4 hr after a stress ^{99m}Tc-sestamibi study do not support a role for redistribution as a cause for discrepancies in defect size. Differences in isotope kinetics during or subsequent to stress-induced ischemia may account for disparate ^{201}Tl and ^{99m}Tc-sestamibi defect sizes. Further testing will be necessary to evaluate this possibility.

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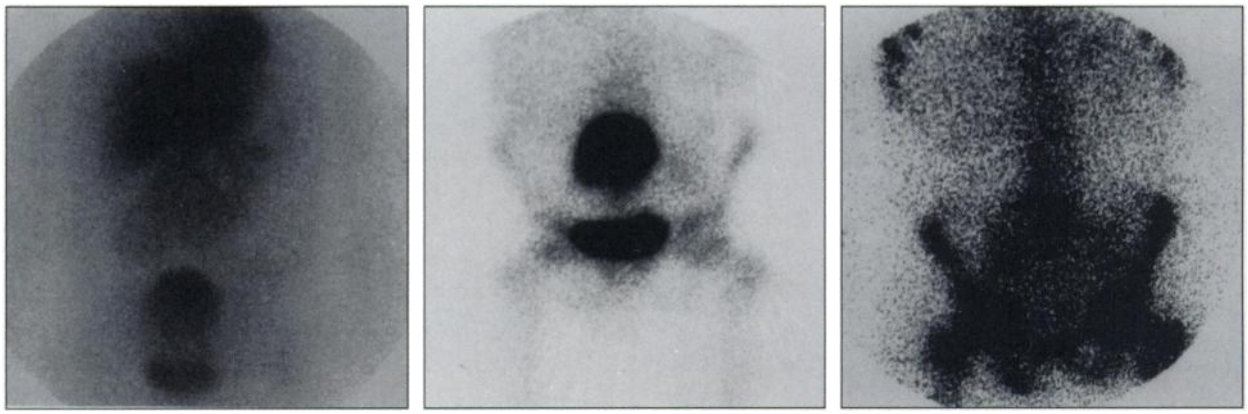
FIRST IMPRESSIONS



FIGURES 1-3. Iodine-123-MIBG, ^{99m}Tc-MDP and CT scans of an abdominal mass.

PURPOSE
A 12-yr-old male presented with an abdominal mass. Transmission computed tomography showed a retroperitoneal tumor extending from the mid-abdomen to the pelvis behind the urinary bladder. The 24-hour delayed [^{123}I]MIBG scan showed activity in the region of the tumor. The ^{99m}Tc-MDP bone scan showed intense activity in the same region. A neuroblastoma would have been highly suspected based on these images were it not for the 24-hour delayed postvoid pelvic bone image which showed no abnormality. At surgery, the tumor was found to be impinging on and constricting the urinary bladder below its dome. This explains the hour-glass appearance of the urinary bladder when distended with radioactive urine. The final histologic diagnosis of the tumor was synovial sarcoma.

TRACER
[^{123}I]MIBG and ^{99m}Tc-MDP.

ROUTE OF ADMINISTRATION
Orally and intravenous.

TIME AFTER INJECTION
24 hours and 4 and 24 hours.

INSTRUMENTATION
LFOV gamma camera and all-purpose collimator.

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