On May 19, 1978 CNS symptoms recurred. The CSF was positive for leukemia, but it cleared after intrathecal cytosine arabinoside therapy. Nevertheless, CNS symptoms increased.

A radionuclide brain scan was performed using 8.3 mCi/m² of Tc-99m glucoheptonate (Fig. 1). The scintiphotos showed multiple focal cerebral lesions. A radionuclide cisternogram (2.0 mCi Tc-99m DTPA) demonstrated focal abnormalities of CSF flow over the cerebral convexities, with the left side more severely affected (Figs. 2A and 2B). The patient died the following month.

Price, et al. (1) described the pathology of CNS leukemia. The leukemic cells first appear in the veins of the arachnoid, then proliferate into the CSF. The process may eventually spread directly into the brain surface. It is the direct growth through the pia-glial membrane that is responsible for the rare intracerebral leukemic masses.

Cisternography has been used effectively to evaluate CSF flow dynamics in trauma, subarachnoid hemorrhage, and meningitis (2). In addition to the bulk-flow characteristics demonstrated with cisternography, areas of subarachnoid obliteration may be defined. Figures 2A and 2B demonstrate regions of obliterated subarachnoid space secondary to leptomeningeal leukemia. The radionuclide brain scan demonstrated multiple parenchymal brain lesions (Fig. 1) that corresponded to the areas of arachnoid obliteration demonstrated with cisternography. The combination of cisternographic and brain-image abnormalities correlates with the pathophysiologic process of CNS leukemia described by Price, et al. (1). For this reason, the findings on the radionuclide studies were interpreted as leptomeningeal leukemia with intracerebral leukemic masses. This was verified pathologically.

Recently, intracerebral leukemic masses have been evaluated with cranial computed tomography (3). The authors suggest that other CNS lesions may mimic intracerebral leukemic masses. Currently histopathology is necessary for definitive diagnosis, although both CCT and radionuclide studies suggest the disease with high probability in the proper clinical situation. Either examination may be used for screening. However, as with other intracerebral masses, the structure of the lesion is better characterized with CCT than with radionuclide imaging.

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Accumulation of Tc-99m Diphosphonate in Pericardial Effusion

Although Tc-99m pyrophosphate has been widely used in imaging for acute myocardial infarcts, pericardial uptake of the tracer has rarely been described. In a clinical and experimental study, it was suggested that the Tc-99m pyrophosphate heart image is normal in acute pericarditis in the absence of ischemic heart disease (1). Pericardial uptake of Tc-99m methylene diphosphonate, however, has been reported (2). We have incidentally observed an uptake of Tc-99m methylene diphosphonate in the pericardial region. It moved with change of the patient’s position, indicating the presence of the tracer in the pericardial effusion. The mechanism of this finding is unknown.

A 51-year-old white woman was admitted with chest pain. She had had a left mastectomy for breast carcinoma 5 yr before the admission. Three years later, a metastatic mass in the left pectoralis minor was found, for which radiotherapy was given. Four months before this admission, a metastasis to the right supracoelvular region was found. A mediastinal tomogram showed a right superior mediastinal mass. Again radiotherapy was given to the internal mammary and right infra- and supracavicular areas. The patient had no history of heart disease.

Physical examination revealed a pulse rate of 116. Blood pressure was 124/78 mm Hg, without paradoxical pulse. A PPD skin test was negative. The point of maximal impulse was diffuse in the 5th and 6th intercostal spaces in the left midclavicular line. Friction rubs and an S4 gallop were heard at the apex. Chest radiograph showed mild cardiomegaly, right perihilar and left upper radiodensities. An echocardiogram revealed pericardial effusion.

Bone imaging was performed with 15 mCi of Tc-99m methylene diphosphonate using a large-field scintillation camera. There were abnormal areas of activity indicating bone metastases in the skull, lumbar spine, and pelvis. An incidental finding was a right floating kidney (see Fig. 1C). With the patient in a supine position, increased uptake of tracer is seen in the pericardial region (Fig. 1A). Figure 1B shows the left lateral image with the patient supine. A curvilinear uptake is seen superimposed on the spine, indicating posterior pericardial uptake. When the patient stood up, the pericardial activity moved in the dependent direction (Fig. 1C),

FIG. 2. (A) Tc-99m DTPA cisternogram (6-hr images). Note asymmetric flow over cerebral convexities. (B) Images at 24 hr. Note photon-deficient areas, which are secondary to arachnoid obliteration. Cerebral masses are in same regions.
cardiac effusion may suggest the possibility of a malignant effusion.

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Elevation of a Hemidiaphragm Simulating Posterior Myocardial Fibrosis

Several causes of false-positive results from myocardial infarction scans obtained with bone-scanning agents have been reported (1-4), but when thallium is used, the false positives are limited to myocardial fibrosis (5,6), generally produced by old infarcts or myocardialopathy. The normal thallium distribution in the left-ventricular myocardium has been well described (7,8), and more recent warnings about the variations in posterior left-ventricular wall activity, especially in the left lateral projection (9). Although caution in interpreting posterior-wall abnormalities when seen in left lateral views alone has been emphasized, these apparent abnormalities are generally not very extensive, and their cause is ascribed to normal cardiac variation. No reports of extracardiac causes of decreased posterior myocardial-wall activity have been found in the literature. We have encountered a case of apparent extensive defect of thallium distribution in the posterior myocardial wall, identified in both exercise and redistribution studies of the left lateral projection, the cause being elevation of the left hemidiaphragm.

A 74-year-old white male was admitted to the hospital with a history of angina pectoris and an acute onset of substernal pressure not associated with dyspnea, diaphoresis, nausea, or vomiting. There was no relief of symptoms with nitroglycerin. The patient had no history of previous myocardial infarction or myocardialopathy. Electrocardiograms revealed the known sinus bradycardia and left axis deviation, and additionally a transient first-degree AV block. Cardiac enzymes revealed no evidence of myocardial necrosis. Blood gases revealed mild hypoxemia on supplemental oxygen but normal acid-base balance and alveolar ventilation as determined by PCO2.

Two days after hospital admission, a myocardial perfusion stress test was performed. The patient was exercised to 69% of predicted tolerance, terminating due to fatigue. The treadmill test did not elicit any chest symptoms or ST segment abnormalities attribut-