## Error in Mo-99 Breakthrough Check

We have two methods with which to check Mo-99 breakthrough in our Mo-99 → Tc-99m generator eluates. One requires the use of a dose calibrator* with a special lead pig that can be placed inside the dose calibrator. The other method uses a scintillation well counter in which a known source is compared with our eluate. The values we obtained for each method were always different by about 30% or more. Something was wrong. We thought that possibly the dose calibrator was not functioning properly, so we had it checked; no problem was found. The well counter was also reported to have no problems. That left only one item: the known source. The label on the source says that it is Cs-137 that is calibrated to equal 92 $\mu$Ci of Mo-99. A closer examination of the label revealed that there was no date of calibration on it. Another pharmacist said that the Cs-137 probably did not decay by a significant amount since the well-counter readings were the same each day and its half-life is 30 yr. One point the pharmacist failed to realize is that the well counter is calibrated with another Cs-137 source. New England Nuclear was called to find out the date of calibration. The information on the label, which included, "Cs-137 = 92 $\mu$Ci Mo-99" and the lot number AZ-82, was given to them. After ample time (one month) to research this problem, N.E.N. was called again, but they told us they could not determine a date of calibration. Their records indicated that these sources are sent out as Mo-99 breakthrough check sources with the purchase of your first generator; however, neither we nor they could determine when our first generator was shipped to us. Since the source could be measured in our dose calibrator, we could determine its present Mo-99 activity only if we had the conversion factor used by N.E.N. to specify Mo-99 in terms of Cs-137. Finally, they were able to supply us with this information; their factor is 4.4. The amount of Cs-137 measured on our dose calibrator was 15.4 $\mu$Ci, and 15.4 $\mu$Ci × 4.4 = 67.7 $\mu$Ci. Thus the source was faulty and, therefore, our values were always off when compared with the dose calibrator readings. By using the decay equation $C = C_0e^{-\lambda t}$, we were able to determine that this source was calibrated 13.3 yr ago.

We suggest, accordingly, that others may also be using a nonvalid source for the Mo-99 breakthrough test, and to encourage manufacturers to put a calibration date on all products supplied. If a new source is not to be purchased periodically, it is also recommended that the Cs-137 sources be refigured annually.

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FOOTNOTE

* Capintec.

## Obscure Hepatic Process with Radiogallium Accumulation

Hepatic areas with decreased reticuloendothelial function and avid radiogallium accumulation are usually associated with hepatoma. We report a case in which this combination of findings was noted, but with no malignancy.

An 81-year-old man had been followed for several years with known ethanol abuse, cirrhosis, and more recently ascites. Because of an elevated alpha-fetoprotein concentration (21,500 ng/ml; normal <20 ng/ml), he was presumed to have a hepatoma. On this admission, the liver was found to be two fingerbreadths below the right costal margin, quite firm, but with no distinct masses. A Tc-99m sulfur colloid scan (Fig. 1, left) showed a liver ~16 cm in length. In the lower portion of the left lobe was a region (~10 cm diameter) with reduced radiocolloid uptake. Radiotracer was also noted in the sternum, ribs, and vertebral bone marrow. The spleen was 13 cm in length. Thirteen days later, the patient was injected intravenously with Ga-67 citrate. Images at 6, 48, and 72 hr (Fig. 1, right) showed greater concentration in the region previously noted to have reduced radiocolloid uptake. At this time the total bilirubin was 11.0 mg/dl and the indirect was 6.5. Serum albumin was 1.9 g/dl. An abdominal tap was performed and ascitic fluid removed. No malignant cells were found on microscopic examination of the fluid. An hepatic angiogram was carried out. No tumor-related vessels could be identified (the films were subsequently reviewed by several radiologists, who concurred with the interpretation). At autopsy 2½ mo later, the liver weighed 2,000 g. There was a yellow-tan nodular surface. On section the liver had a macronodular appearance. In the lower portion of the left lobe, the tissue looked similar to that elsewhere except for some increased vascularity. The spleen weighed 250 g and had an intact smooth capsule. Multiple sections through the right and left hepatic lobes, including the "lesion" in the left lobe, were examined microscopically. No evidence of hepatoma was found. There was no unusual inflammatory tissue to distinguish this region from the remainder of the liver. The microscopic sections were reviewed by several pathologists who agreed with the conclusions. The hepatic area was "benign" by several criteria. (a) No malignant cells were identified in the ascitic fluid. (b) Angiography did not reveal tumor-associated changes. (c) At autopsy, both gross and microscopic examination failed to reveal tumor cells. However, the region was certainly unusual by at least three biological measurements. (d) The tissue did not accumulate Tc-99m sulfur colloid. (e) There was avid uptake of Ga-67. (f) Circulating levels of alpha-fetoprotein were enormously elevated; this may have been due to the hepatic lesion, but proof is lacking.

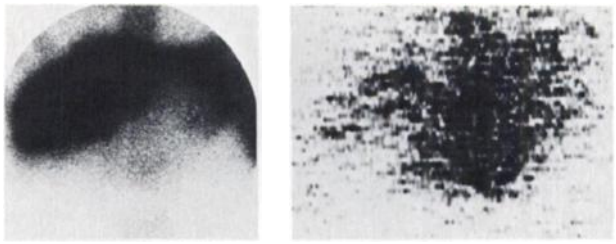

**FIG. 1.** Anterior images of hepatic region. Left: overexposed scintiphoto (Tc-99m sulfur colloid) showing left-lobe lesion, ~10 cm in largest diameter, with decreased radiocolloid accumulation. Right: rectilinear scan of same area (slightly greater magnification) obtained 72 hr after i.v. Ga-67 citrate.

This case raises the interesting question of the degree of "coupling" of hepatic functions. In two studies reported in the literature (*1*,*2*), primary hepatocellular carcinoma selectively concentrated Ga-67 in 11/12 and 14/16 patients. In our case the decreased Tc-99m sulfur colloid uptake and presence of circulating alpha-fetoprotein were also consistent with a hepatoma, abscess, or other abnormality. Yet no hepatoma was found at autopsy. It is possible that the radiogallium-concentrating mechanism involves a cellular marker not readily associated with structures identifiable by light microscopy.

James and co-workers (*2*) commented on the low rate of false-positive radiogallium studies during a search for hepatomas in patients with cirrhosis. Suzuki and associates (*3*) noted that the grade of malignancy of tumors was not proportional to their degree of uptake of Ga-67. The present case showed intense concentration of radiogallium by a "lesion" that did not differ significantly from the remainder of the liver on microscopic examinations. We must hypothesize that there are "preneoplastic" or other conditions in the liver associated with decreased reticuloendothelial function,

elevated radiogallium uptake, and possibly with increased alpha-fetoprotein in the blood stream.

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