

## UPTAKE OF $^{67}\text{Ga}$ IN SPACE-OCCUPYING LESIONS IN THE LIVER

We have read with interest the recent short article by Geslien, et al (1), in which attention is drawn to uptake of  $^{67}\text{Ga}$ -citrate in the periphery of defects seen in the radiocolloid liver scan. It was suggested that the  $^{67}\text{Ga}$ -citrate is taken up in the inflammatory zone surrounding the central necrotic amebic abscess cavity.

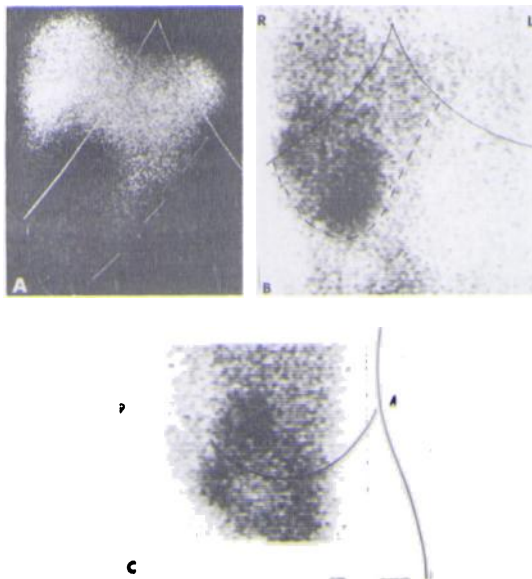
In a series of 110 patients (2,3) we have also noted this finding in both the pyogenic abscess (Fig. 1) and also, unusually, in a case of primary liver cell carcinoma complicating cirrhosis (Fig. 2). It is thought that since this finding is present in pathologic conditions other than acute hepatic amebic abscess,

the appearance of peripheral uptake of  $^{67}\text{Ga}$ -citrate is a nonspecific observation.

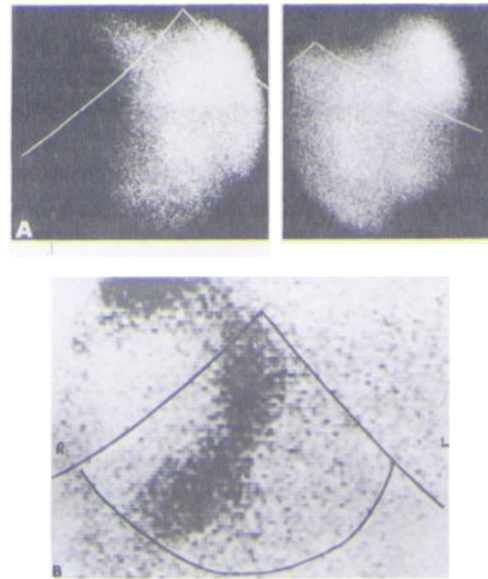
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**FIG. 1.** Anterior-posterior scintillation camera picture (A) of liver  $^{99\text{m}}\text{Tc}$ -colloid distribution showing filling defect in lower right lobe of an enlarged liver. Anterior-posterior (B) and (C) right lateral scintiscan projections of  $^{67}\text{Ga}$ -citrate uptake related to defect of colloid scan. In (C) note uptake in peripheral inflammatory area surrounding pyogenic abscess from which 400 ml pus was aspirated at laparotomy.



**FIG. 2.** Anterior-posterior scintillation camera pictures (right and left sides of abdomen) of liver  $^{99\text{m}}\text{Tc}$ -colloid distribution showing (A) filling defect in right lobe of enlarged liver. Marked "spillover" of colloid into spleen has occurred. Anterior-posterior  $^{67}\text{Ga}$ -citrate scintiscan (B) shows uptake in periphery of lesion. Diagnosis of primary liver cell carcinoma complicating cirrhosis was confirmed histologically.

### THE AUTHOR'S REPLY

Maze and Woods are directed to the experimental work of Blair, et al (1), Harvey, et al (2), and Burleson, et al (3) on the localization of  $^{67}\text{Ga}$ -citrate in pyogenic abscesses. They found concentration of the  $^{67}\text{Ga}$ -citrate to be greatest in the granulation tissue of the wall of the abscess and not in the central area of pus. The mechanisms whereby  $^{67}\text{Ga}$ -citrate is localized in the abscess wall are not entirely known. However, they postulate that the abscess wall is detected because of hyperemia-increased vasculariza-

tion which occurs in any inflammatory process. Gallium 67-citrate is known to bind transferrin and other plasma proteins and is carried to these areas of hyperemia and granulation tissue within the blood pool (4).

Blair's and Harvey's work, as mentioned in our article (5), is used to explain the greater concentration of  $^{67}\text{Ga}$ -citrate in the rim of the acute hepatic amebic abscesses when scanned with  $^{67}\text{Ga}$ -citrate.

Technetium 99m-sulfur colloid liver scans only

reflect the distribution of functioning Kupffer cells and we must therefore discard the concept that focal scan defects represent "space-occupying lesions." The point to be stressed is that abscesses have more than one phase (as do necrotic tumors) and the actual size of the cavities (necrotic centra) can probably be more accurately assessed with  $^{67}\text{Ga}$ -citrate than with  $^{99\text{m}}\text{Tc}$ -sulfur colloid. This was demonstrated in both our cases by the discrepancy in size of the abscesses as seen on the  $^{99\text{m}}\text{Tc}$ -sulfur colloid scans (larger) compared with the  $^{67}\text{Ga}$ -citrate scans (smaller). Moreover, the rim of increased activity on the  $^{67}\text{Ga}$ -citrate liver scans was essentially contained within the area of the  $^{99\text{m}}\text{Tc}$ -sulfur colloid scan defects. The rationale for the larger scan defect with  $^{99\text{m}}\text{Tc}$ -sulfur colloid is that the peripheral area of hyperemia about the acute abscess cavity inactivates the Kupffer cells in this region and, thus, the radio-colloid scan defect not only represents the abscess cavity but the inflammatory hyperemic zone. Delineation of the cavity is best done with  $^{67}\text{Ga}$ -citrate since it localizes in the hyperemic zone and to only a lesser extent in the necrotic center. Secondly, as the abscess becomes older, the zone of hyperemia disappears and the phagocytic activity in this zone returns. This results in a defect on the  $^{99\text{m}}\text{Tc}$ -sulfur colloid scan

which is smaller and represents the negative defect of the pus cavity. This rapid return of phagocytic activity when the zone of hyperemia disappears may account for the discrepancy in the literature on the rate of healing of amebic abscesses.

We thank Maze and Wood for allowing us to reaffirm our observations.

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#### TOXICITY OF $^{99\text{m}}\text{Tc}$ -Sn-EHDP

Tofe and Francis (1) representing Procter and Gamble state that the appropriate amount of  $^{99\text{m}}\text{Tc}$ -Sn-EHDP for human administration (1-2 mg EHDP) for the purpose of skeletal scintigraphy is very low. The same authors state that 10-20 mg/kg/day EHDP has been therapeutically administered orally to patients with disordered mineral metabolism with no adverse effect. These statements are misleading.

An oral dose of 10-20 mg/kg/day of EHDP of which an average of 2-3% is absorbed (range, 0.1-15%) represents an average total absorbed dose of 14-42 mg (range, 0.7-210 mg) in a man weighing 70 kg (2). This dosage produces therapeutic effects in certain bone diseases (2), and reproducibly causes hyperphosphatemia (2) and decreased ionized serum calcium associated with elevation of plasma PTH (3).

I would hesitate to claim that these effects may not be "adverse."

Obviously all EHDP injected intravenously is absorbed and the dose recommended by Tofe and Francis for bone scans, 1-2 mg, is definitely in the range of absorbed dose (> 0.7 mg) associated with the previously mentioned effects, at least in chronic administration of the drug to some patients.

Although the safety margin of the recommended bone scan dose of EHDP is acceptable, it most certainly is not a tracer dose and its having some pharmacologic action in at least a fraction of patients to whom it is administered can be predicted.

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#### THE AUTHORS' REPLY

We believe that the concern expressed about the possibility of a pharmacologic action related to the intravenous administration of  $^{99\text{m}}\text{Tc}$ -Sn-EHDP (1-2 mg EHDP) can be satisfied by the results of more

extensive urinary excretion studies (unpublished) and a review of the recent literature.

Urinary excretion data collected on both normal patients and those with Paget's disease show that the