ACCURACY OF LIVER SCINTISCANNING

In the recent paper by Lunia, et al (*1*), it was concluded that the accuracy of liver scintiscanning varied in relation to the site of the primary malignant lesion and that carcinoma of the urinary bladder and of the thyroid yielded particularly unsatisfactory results.

This conclusion is unsatisfactory in two ways. First, the authors completely ignore the possibility of sampling error in interpreting their results. It is obvious that more confidence can be placed in the "Ca colon" data based on 118 patients than in the "Ca thyroid" data based on only 5 cases. A proper interpretation of Table 6 in this paper requires the calculation of 95% confidence limits (i.e., ±2 s.e.) for each percentage of correct diagnoses. When this is done, it is seen that all the confidence intervals overlap except that for carcinoma of the urinary bladder, where the difference may be as little as 1%. For example, the 95% confidence limits for the accuracy of scintiscanning in the colon are 84.7 ± 6.6%, or 78.1–91.3%, and for the thyroid the limits are 40.0 ± 43.8%, or 0–83.8%!

The second way in which the data were misinterpreted is even more serious. It is clear from Table 3 that the accuracy of liver scintiscanning depends on whether the patient actually has a liver secondary. The success rate for the 253 patients who had a liver secondary was 60.1% while for the 328 who did not the accuracy was 90.5%. It is obvious, therefore, that the accuracy obtained in any sample of patients depends on the proportion who actually have liver secondaries. If healthy individuals are studied, a 90.5% accuracy could be expected. Increasing the proportion of patients with liver metastases reduced the accuracy. Clearly, the accuracy found in any particular sample of patients depends on the number who actually have liver metastases, and no account is taken of this by these authors.

In their main conclusion, the authors claim that the level of diagnostic accuracy achieved by liver scintiscanning has not greatly changed since 1963. This conclusion, based on the results quoted, is also invalid since it does not take account of this differential accuracy in patients with and without liver metastases.

PETER J. GILLESPIE
Northern Ireland Radiotherapy Centre
Purdysburn, Belfast
KENNETH D. MacRAE
Department of Medical Statistics
Queen's University, Belfast

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CELLULAR SITE OF SECRETION OF $^{99m}TcO_4^-$ IN THE STOMACH—A CONTROVERSIAL POINT

It is believed that pertechnetate, like the halogens, is secreted by the parietal cells of the human stomach (*1,2*). This concept is based on experiments carried out by Meier-Ruge and Fridrich (*3*), who showed by histologic studies of the cat's stomach that technetium is selectively secreted by the gastric parietal cells. On the other hand, the gastric antrum is devoid of parietal cells (*4*) and therefore does not secrete hydrochloric acid (*5,6*). One would expect that the gastric antrum should not concentrate pertechnetate. Contrary to this inference, however, Chaudhuri, et al (*7*) showed in dogs that an isolated gastric antrum does concentrate $^{99m}TcO_4^-$ even though it is devoid of parietal cells. Thus, the secretion of ^{99m}Tc may be a property of both fundic and antral gastric mucosa and is probably not mediated by parietal cells—at least not in dogs. At the organ level this observation agrees with the histologic findings of Marsden, et al (*8*).

No histologic study is available as yet in man, however, and a species difference cannot be ruled out from the available data. Nevertheless, while scanning the abdomens of pernicious-anemia patients with histamine-fast achlorhydria, I have found that TcO_4^- is secreted in significant amounts by the stomachs of these patients (*9*). This observation would indicate that in man also, technetium is not secreted by the parietal cells alone, and perhaps not by them at all. Since mucus-secreting cells are present throughout the normal stomach including the antrum, and also in the stomachs of patients with pernicious anemia, it may be that ^{99m}Tc is secreted by

the mucous cells. Since none of the human evidence permits quantitative comparison of total and regional gastric secretion, it is premature to say whether gastric concentration and secretion of ^{99m}Tc is accomplished by a single mechanism or by two separate mechanisms. Until further evidence is available, the hypothesis that the parietal cells alone are involved seems to be debatable, at least in dogs and man, and the possible role of mucus-secreting cells in handling ^{99m}Tc should also be considered. Further studies are needed to pinpoint the exact cellular location of ^{99m}Tc in the stomachs of different species.

TAPAN K. CHAUDHURI
Veterans Administration Center
and Eastern Virginia Medical School
Hampton, Virginia

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SPLENIC UPTAKE OF ^{111}In

The statement by Merrick, et al (*1*) concerning ^{111}In uptake by the normal spleen deserves further comment. We were motivated by their assertion that "indium is always taken up by the normal spleen," which at first glance would appear to be at variance with our experience and that of other authors.

After intravenous injection of $^{111}InCl_3$ at acid pH, whole-body imaging at 24–72 hr normally displays the bone marrow and liver. The normal spleen is, in our experience, not visualized. Staub (*2*) reported similar experience; his additional data supported splenic localization of ^{111}In as evidence of extramedullary erythropoiesis.

Touya (*3*) has suggested that metabolically indium behaves like iron from the nonhemoglobin-oriented binding site. It would then be expected to follow the metabolic fate of such iron and be taken up by the liver and spleen. Organ distribution studies revealed 29% in the liver, 20% in bones, 14% in the skin, 14% in muscles, 5% in kidneys, and 3% in the spleen. Farrer (*4*) noted splenic uptake of indium to be significantly less than with technetium–sulfur colloid.

The observation by these authors of a low but definite uptake of ^{111}In by the spleen, in our judg-

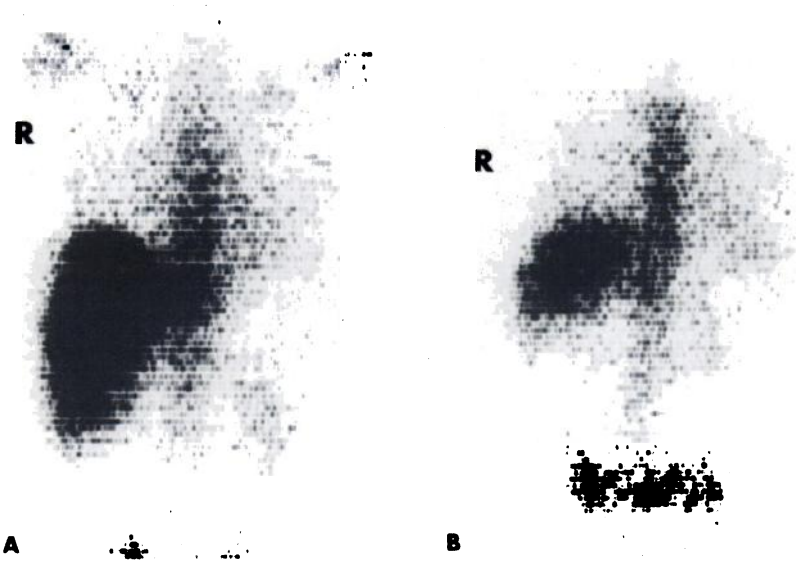

FIG. 1. Indium-111 posterior whole-body images. Imaging performed 24 hr after intravenous injection of 2 mCi of $^{111}InCl_3$ at acid pH. (A) Man (57 years old) with lymphosarcoma, 5 years postsplenectomy. Study felt to be normal except for hepatomegaly. (B) Woman (52 years old), control.