transferrin is saturated, it will no longer bind indium (*4*). Furthermore, renal scans have been produced in animals by first giving parenteral iron and then injecting indium intravenously (*5*).

The practical importance of these observations is that if a patient with a fully saturated transferrin is given ^{113m}In for placental scanning, the indium may be excreted promptly by the kidneys and no adequate view of the placenta may be obtained. The problem may then be further compounded by radioactivity which appears in the bladder urine.

Transferrin saturation is only likely to occur commonly in patients who have been given blood transfusions or parenteral iron or who have liver disease. In a series of 200 placental scans with ^{113m}In, Wright (*6*) noted that a "pyelogram" was obtained in three patients and speculated that oral or intravenous iron may have been responsible.

Lin and his colleagues do not mention whether the iron-binding capacity and serum iron were measured in any of their patients or whether any of them had received blood transfusions or parenteral iron or had liver disease or any of the less common conditions which might have led to saturation of the plasma transferrin. It may be of interest to review their data with these points in mind.

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AUTHORS' REPLY

Binding to plasma proteins is frequently an important consideration in discussing plasma disappearance and urinary excretion of small molecular substances. Specifically, we realized the potential implication of indium binding to transferrin and alpha globulins (*1–3*) in evaluating urinary excretion of indium. Wochner, et al (*2*) reported ^{113m}In spaces grossly greater than albumin spaces in three patients with severe hepatitis and iron-saturated transferrin. We thought that plasma disappearance and urinary excretion of indium could be similarly affected by altered indium binding to plasma proteins, transferrin or otherwise.

Thus in 20 of our 25 patients studied (*4*), we determined early plasma disappearance as well as urinary excretion of the indium. As shown in Table 1, we found no correlation between the early plasma disappearance and the early urinary excretion of the indium.

TABLE 1. RELATIONSHIP BETWEEN EARLY PLASMA DISAPPEARANCE AND EARLY URINARY EXCRETION OF ^{113m}In FOLLOWING ITS INTRAVENOUS ADMINISTRATION*

Group	No. of patients	Mean (μ) ± s.d. and range	30-min urinary excretion (% dose)	Plasma level after 27–30 min (%)
I	6	μ ± s.d.	0.58 ± 0.28	90 ± 5
		range	0.33 — 1.09	82 — 95
II	8	μ ± s.d.	0.12 ± 0.05	88 ± 12
		range	0.09 — 0.24	57 — 98
III	6	μ ± s.d.	0.05 ± 0.02	88 ± 7
		range	0.01 — 0.07	74 — 93

* A total of 20 patients were divided into three groups according to their measured ^{113m}In excretion. For each group, the plasma ^{113m}In level 20 min after 7–10 min is shown as percent of the 7–10-min value.

We agree with Dr. Richards in that the degree of transferrin saturation could have been an important factor in the urinary excretion of indium in our patients. If it had been so however, we would not have been able to demonstrate it, apparently because other unknown factors were more important in the excretion.

Serum iron and iron-binding capacity were not determined in the 25 patients. None of them had received parenteral iron or blood transfusions in the period of months just preceding the study. None was under clinical evaluation for liver diseases except for one with acute serum hepatitis. He received the indium primarily for the urinary excretion study and had serum SGOT of 2,000 U (Karmen) at the time of the study. His cumulative 30- and 90-min excretion were 0.05 and 0.12% dose, respectively. These values were about ¼ of corresponding average values for the 25 patients.

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SUPERIOR VENA CAVAL OBSTRUCTION AND INCREASED RADIOCOLLOID ACTIVITY ON LIVER SCINTIPHOTOS

Recent case reports in the *Journal of Nuclear Medicine* have recorded the unusual occurrence of focal increases in radiocolloid activity on liver scintiphotos (*1–3*). We have recently observed two additional such cases. Both patients presented with superior vena caval obstruction secondary to metastatic carcinoma. In each case static and dynamic scintiphotos demonstrated focal areas of increased colloid at the junction of the right and left hepatic lobes (Fig. 1).

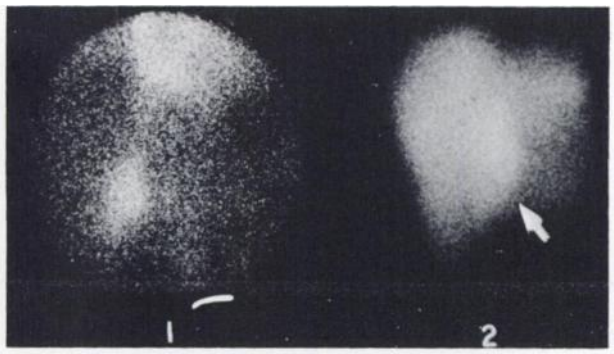

FIG. 1. Dynamic (1) and static (2) scintiphotos of radiocolloid hot spot. Patient had superior vena caval obstruction secondary to metastatic lung carcinoma.

Four of five previously reported cases of radiocolloid "hot spots" have also been associated with metastatic carcinoma and superior vena caval obstruction. The fifth case was due to a hemangioma (*4*). Caval portal shunting due to vena caval obstruction would appear a likely mechanism for this phenomenon (*2*). Metastatic tumors do not contain Kupffer's cells and "colloid-concentrating" metastatic tumors have yet to be documented.

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ERRATA

In the Works in Progress abstract entitled "^{111}Indium-Bleomycin: A New Radiopharmaceutical for Tumor Scintigraphy" (*J Nucl Med* 14: 641, 1973) Melvin J. Silverstein should be added to the list of authors. The correct sequence of authors is: Ramesh C. Verma, Leslie R. Bennett, Juan J. Touya, Melvin J. Silverstein, Donald L. Morton, and Ewa Witt.

The Letter to the Editor by N. Adiseshan (*J Nucl Med* 14: 722, 1973) should be entitled "Radiostrontium Localization in Normal Lungs?" and the Author's Reply (*J Nucl Med* 14: 723, 1973) by T. K. Chaudhuri should be entitled "Radiostrontium Deposition in Lungs: "Apparently Normal" Lungs versus Occult Aspergillosis.'