TABLE 1. COMPARATIVE DATA ON FIVE COMMERCIAL SOURCES OF ^{99m}Tc ELUATE

Dates	Vendor	Average μCi ^{99}Mo per mCi ^{99m}Tc	Percent standard deviation	Description (concentration at 9:00 am)	Container shielding
One week trials:					
10/23–11/3	Chromatographic generator (about 150 mCi in tandem)	0.009	±49%	Generator milked (5.3–9.0 mCi/ml)	fair
11/6–10	Instant A	0.067	±93%	instant (21.5 mCi/ml)	poor
11/13–17	Instant B	0.055	±21%	instant (14.4 mCi/ml)	good
11/20–24	Instant C	0.048	±42%	instant (40.6 mCi/ml)	poor
11/27–12/1	Instant D	0.014	±35%	instant (24.3 mCi/ml)	good
Single events:					
12/27/72	Instant D	3.23		^{99}Mo breakthrough	
1/25/73	Instant B	0.25		marginal ^{99}Mo breakthrough	

netium is a fact that is not widely appreciated and demonstrates the necessity for checking ^{99}Mo activity daily when instant technetium is being used. The discrepancy between our observations and those of Robinson (*1*) may be explained in part by the fact that instant technetium may have been allowed to stand for long periods before distribution with a consequent building up of the relative ^{99}Mo concentration.

The concentration of ^{99}Mo also varied from day to day by a considerable amount as is evident by the column in Table 1 showing percent standard deviation of ^{99}Mo concentration per mCi of ^{99m}Tc. The variation is shown in the extreme case in the single events indicated in Table 1. Instant D had been previously selected for use in a longer term trial because it had the lowest ^{99}Mo concentration of all instant ^{99m}Tc supplied during the initial trials. On a single day, however, significant ^{99}Mo breakthrough above the AEC limits was found. The supplier claimed that a procedural error in distribution was at fault and was being corrected. After switching to Instant B for longer term trials, significant ^{99}Mo breakthrough was again found on a single day which would not have allowed usage of 15 mCi on patients during the afternoon of that day.

The utility of supplied concentration of ^{99m}Tc although somewhat subjective was uniform for all instant ^{99m}Tc supplied and did not vary during the week as did ^{99m}Tc eluate milked from supplied generators. Concentrations of 15–20 mCi/ml as of 9 a.m. were found most convenient to work with. The generator-eluted ^{99m}Tc would have too low a concentration toward the end of the day; the generator used was a tandem with an average of about 150 mCi of ^{99}Mo and was not fractionally eluted. The Instant C was judged too concentrated and difficult to handle in the morning.

The adequacy of the lead shielding on the container plays some role in reducing the radiation dose to the technician. All suppliers had adequate shielding from the side and bottom; however, those judged good had the vial top recessed with a small opening on the container top in which fit a removable lead cover. This type of container provided the most protection for the technician drawing up the dose.

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TRANSFERRED BINDING OF INDIUM

Lin, Burke, and Lee (*1*) have presented data to show that there is considerable variation in the amount of ^{113m}In excreted in the urine after it is given intravenously for placental, cardiac, or brain scanning. They were unable to offer any explanation for their findings.

My observations (*2*) suggest that when a patient's plama transferrin is saturated, intravenously administered ^{113m}In may be excreted by the kidneys, thus producing an unintentional renal scan. It has been shown (*3*) that indium becomes bound to transferrin after intravenous injection and also that, once the

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.