$P_{\tau R}$. Division of these two relations yields

$$ERPF_L/ERPF_R = (U_L/U_R)(\beta_L/\beta_R)(\tau_R/\tau_L)$$

This equation may be compared with the assumptions suggested by Dr. Reese when it is noted that $ERPF_T/ERPF_T = 1$, that

$$ERPF_L/ERPF_R = (U_L/U_R)(\beta_L\, ERPF_T/\beta_R\, ERPF_T)(\tau_R/\tau_L)$$

Since

$$\beta_L\, ERPF_T = ERPF_L$$

and

$$\beta_R\, ERPF_T = ERPF_R$$

$$1 = (U_L/U_R)(\tau_R/\tau_L)$$

or

$$U_L/U_R = \tau_L/\tau_R$$

as we stated. This result also precludes the necessity of concluding that $3.33 \neq 2$.

We regret that Dr. Reese was misled by our figures. There was, in fact, no data from normal subjects in Figs. 3, 4, or 5. Over half of the patients with both split-function test and the RP/ED had unilateral disease. In the majority of our data we do not have $ERPF_L/ERPF_R = 1$. As shown in Fig. 3, only a minority of the flow fractions were in the range of 0.4–0.6. This could happen only when most of the flow ratios were less than 0.67 or greater than 1.5. In addition, Fig. 4 shows that the relation $U_L/U_R = \tau_L/\tau_R$ holds even for flow fractions on the order of 0.2 or 0.8, or ERPF ratios of about 0.25 and 0.12, our lowest value.

The tubular transport times reported are *not* comparable to the conventional "transit" times of compartmental analysis [as a glance at Eqs. 6 or 7 on p. 112 of (*1*) will show], since they estimate transit within tubular lumina only. "Cortical transit times" referred to by Dr. Reese are believed to be a composite measurement of OIH flow through the proximal tubular cells and the transport of the OIH in the lumen of the nephrons (*3,4*). The intracellular flow and content of OIH has been considered in detail in the excellent work of Wedeen (*2*).

Dr. Reese has stated three possible ways to estimate the distribution volume (and hence ERPF) in his letter. Strictly speaking, we do not use any of these methods since our initial conditions begin with the assumptions that OIH has already entered all of the body compartments. Analysis is based upon the conditions assumed to exist after mixing is completed. The solutions of Eqs. 1, 5, and 11 along with the adjoined initial conditions, accounts for the initial distribution. Detailed analysis of the solution shows that the initial distribution of isotope must be included in the analysis.

Finally, Dr. Reese asserts that "A creatinine clearance of 26 ml/min 'goes' with an ERPF of about 130 ml/min." This relationship does not necessarily pertain in the sick kidney (*5*). One must be prepared to separate the effects of disease on a glomerulus with relatively little reserve capacity from that on tubular cells which have an extremely efficient mechanism for OIH transport (*6*) and a sizable (1000-fold) reserve capacity. It does not surprise us that a patient with arteriolarnephrosclerosis, glomerular nephritis, or acute transplant rejection will have a significant dissociation between ERPF and GFR. Indeed this is a partial justification for this work and PAH studies. Dr. Reese is quite right to observe this separation; indeed review of our paper will show dissociation is also present in other cases (Figs. 7–9). The creatinine clearance fraction was particularly useful in our initial evaluation of MC (Fig. 7) who suffered renal vein thrombosis.

We would like to thank Dr. Reese for his interest in our work.

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REFERENCES

1. DeGrazia JA, Scheibe PO, Jackson PE, et al: Clinical applications of a kinetic model of hippurate distribution and renal clearance. *J Nucl Med* 15: 102–114, 1974

2. Wedeen RP: Autoradiography of hippuran-I^{125} in rat kidney: The intrarenal basis of the radioisotope renogram. In *Radioisotopes in the Diagnosis of Diseases of the Kidneys and the Urinary Tract*, Timmermans L, Merchie G, eds, Amsterdam, Excerpta Medica, 1969, pp 461–466

3. Farmelant MH, Sachs CE, Genna S, et al: A physiological model for the renal excretion of labeled compounds. *J Nucl Med* 10: 664–671, 1969

4. Farmelant MH, Bakos K, Burrows BA: Physiological determinants of renal tubular passage times. *J Nucl Med* 10: 641–645, 1969

5. Smith HW: *The Kidney, Structure and Function in Health and Disease*. New York, Oxford University Press, 1951, pp 594, 843–847

6. Pitts RF: Physiology of the kidney and body fluids. Chicago, Year Book Medical Publishers, 1963, pp 132–133

INTRAPERITONEAL ^{99m}Tc-SULFUR COLLOID DISTRIBUTION

The March issue of the *Journal of Nuclear Medicine* contains a concise communication by Drs. Tully, Goldberg, and Loken (*J. Nucl. Med.* 15: 190–191, 1974). We have used ^{99m}Tc-sulfur colloid prior to intraperitoneal installation of therapeutic radiocolloids for a number of years. A preliminary report of

this application was noted in a Letter to the Editor of the *British Journal of Radiology* (*1*). The technique that we use is similar to that reported by Tully, et al except that we instill the sulfur colloid and take gamma camera images prior to instilling the therapeutic radiocolloids. One can then evaluate the adequacy of distribution before the therapeutic dose is instilled, allowing one to avoid an unsatisfactory or potentially hazardous installation.

The first example demonstrates what we feel is an unsatisfactory distribution. Figure 1A is a posterior view of the patient's lower abdomen with the patient in the knee–chest position. This indicates that the activity is primarily located in the lower abdomen and pelvis. Figure 1B is an anterior view of the same region again demonstrating the confinement of activity to the lower abdomen. The lower marker indicates the level of the symphysis pubis. Figure 2 is a right lateral view of the lower abdomen of another patient indicating loculation of the sulfur colloid in the anterior abdominal wall or loculation in the peritoneal space. The linear activity is from an anterior abdominal wall marker.

In the first situation, we felt that we should limit the therapeutic radiocolloid to less than the usual amount due to the uneven distribution in the abdomen. In the second situation, the therapeutic colloid was not instilled because this would result in a high local radiation dose to the anterior abdominal wall. A catheter was later surgically inserted in the abdomen and many adhesions were evident. The distribution was still not satisfactory and the therapeutic colloid was not instilled.

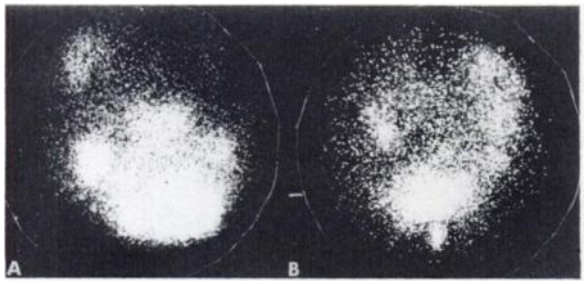

FIG. 1. Posterior view (A) and anterior view (B) of lower abdomen with patient in knee–chest position.

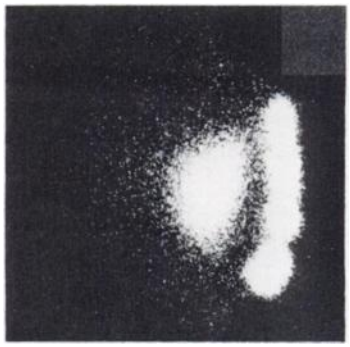

FIG. 2. Right lateral view of lower abdomen.

We agree with Tully, et al that ^{99m}Tc-sulfur colloid is a good agent for demonstrating the distribution of therapeutic colloids in the abdomen. We would recommend performing the diagnostic study prior to instilling the therapeutic colloid in what may be a potentially dangerous distribution.

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REFERENCE

1. VIGARIO GD, WETZEL RA, CONSTABLE WC: Use of ^{99m}technetium sulfur colloid scan prior to intraperitoneal installation of radioisotopes for therapy purposes. *Br J Radiol* 43: 582–583, 1970

THE AUTHORS' REPLY

We would like to thank Charles Teates and William Constable for their comments. We were unaware of Dr. Constable's Letter to the Editor in the *British Journal of Radiology*.

We quite agree that the injection of ^{99m}Tc-sulfur colloid prior to the installation of the ^{32}P-chromic phosphate suspension should accurately predict the distribution of the chromic phosphate in the body cavity being treated if one assumes that the two agents will occupy the same space and are distributed in a similar fashion.

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