TABLE 1. DISTRIBUTION OF VARIOUS RADIOPHARMACEUTICALS IN TURPENTINE-INDUCED ABSCESSES*

Pharmaceuticals	% dose (×10³)/gram (mean)			Ratio (mean)	
	Muscle	Abscess	Blood	Abscess/muscle	Abscess/blood
Ga-67 citrate (3)†	1.2	7.2	20.0	6.0	0.4
Tc-99m DTPA (3)	0.2	1.5	0.9	7.5	2.1
Tc-99m pertechnetate (3)	1.1	7.4	8.6	7.1	1.0
In-111 (pre-incubated with serum) (3)	0.4	11.5	22.2	27.3	0.5
Tc-99m minimicrosphere (4)	0.4	2.0	3.7	5.5	0.5

* Abscesses were 2 days old. Results were obtained 4 hr after i.v. injection of radioactive tracers and were expressed as mean.

† Numbers in parentheses indicate numbers of experiments.

Gallium-67 Accumulation in Inflammatory Lesions

In 1969, Edwards and Hayes (*1*) noted that gallium-67 accumulated in the involved lymph nodes of a patient with Hodgkin's disease. Since then, Ga-67 has been shown to localize in a variety of tumors as well as inflammatory lesions. Earlier studies (*2,3*) have suggested that the localization of Ga-67 in inflammatory lesions is due mainly to concentration of Ga-67 by polymorphonuclear leukocytes (PMN) at the sites of inflammatory reaction. In vitro studies of Ga-67 uptake, however, have consistently revealed that PMN do not significantly accumulate Ga-67, unless the plasma membrane permeability barrier is disrupted (*4,6*). Analysis of the abscess contents also reveal that the majority of Ga-67 is in the noncellular fraction (2,500 g supernate) (*4,7*). Furthermore, Ga-67 accumulates in inflammatory lesions of agranulocytotic patients, in which no PMN are found in the circulation or the sites of infection (*7,8*). After i.v. injection, Ga-67 binds to plasma proteins, notably transferrin (*8,9*), and less than 1% is associated with cellular elements (*10*). Based on the above observations, we have proposed (*10*) that accumulation of Ga-67 in inflammatory lesions is primarily due to leakage of protein-bound Ga-67 through capillaries with increased permeability as the result of inflammation. Once in the inflammatory site, Ga-67 is preferentially bound by nonviable PMN, with lesser amounts in bacteria and viable PMN. The remainder stays in the noncellular fraction (*10*).

If the above nonspecific mechanisms account for the accumulation of Ga-67 in areas of inflammation, one would expect that other radiopharmaceuticals should concentrate similarly and, indeed, this is the case (Table 1). In this study, abscesses were induced with turpentine in the inner aspect of one thigh of rabbits. Forty-eight hours later, the radiopharmaceuticals were injected intravenously through a marginal ear vein. The results shown in Table 1 were obtained 4 hr after injection of the radioactive tracers. In all instances, high abscess to normal muscle ratios were obtained.

Why is Ga-67 used almost exclusively for the localization of inflammatory lesions? Several factors make Ga-67 suitable, though not ideal, for this purpose: a) gallium-67 remains in the circulation long enough to allow sufficient amounts of it to be delivered to the sites of inflammation; b) the concentration of Ga-67 in the blood reaches low levels after 24 hr (*10*), so that the background activity is relatively low; and c) the 78-hr physical half-life of Ga-67 makes imaging feasible 2-3 days after injection.

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Missed Testicular Torsion Demonstrated by Scintigraphy

Missed testicular torsion is the postinfarction state caused by torsion of the testicle and a subsequent delay in diagnosis. Three cases of missed testicular torsion appear in a review by Holder et al. (*1*). In all of these cases the testicular radionuclide angiogram was described as normal. The static images showed a cold round area replacing the testicle, as seen in acute testicular torsion, with a variable degree of hyperemia surrounding the testicle.

Recently we imaged a patient with missed testicular torsion,