REPLY: We agree that our use of the word "dose" might be confusing. We were using it in the common clinical sense, as in Solomon's statement that "Despite the many complicated schemes for determining the dose of radioactive iodine to administer . . . variability in uptake and ultimate response foils the most elegant methods to standardize the delivery of radiation to the thyroid gland" (*1*). From a pure radiobiologic point of view, the word "dose" might be reserved for the energy deposited in the thyroid gland; for practical purposes, "administered activity" or "administered dose" might be used for the amount given orally.

We hope, however, that this use of words will not obscure our fundamental aim, which was not "to make the patient hypothyroid without total ablation," but to provide choices for both physicians and patients when the desired endpoint is cure of hyperthyroidism.

Solomon (*1*) defines two major schools of thought regarding the treatment of hyperthyroidism with ^{131}I that imply different values and goals. In the first approach, "one administers a dose of ^{131}I calculated to produce hypothyroidism in most recipients," with rapid cure of hyperthyroidism as the central goal. It assumes that hypothyroidism will occur in the long run for most patients regardless of administered dose, and therefore its development is of less concern. When about 15 mCi is administered, the percent cure is about 95% (*2*). In the second approach, "one administers a dose calculated to have the best chance of resulting in a cure, with the lowest combined incidence of hypothyroidism and persisting hyperthyroidism." Using a dose calculated to meet this set of goals, the mean percent cure has remained at about 70% for the past 40 years (1950–1990) (*3*).

Each of these methods has its proponents, and each may be used for individual patients under different circumstances. Our efforts were to de-emphasize the dichotomy by providing a better understanding of the direct relationship between administered dose and both cure of hyperthyroidism and early (but not delayed) development of hypothyroidism. This information provides patients and physicians alike with a clearer choice between administered dose and the likelihood of persisting hyperthyroidism with its increased disability and cost.

REFERENCES

1. Solomon DH. Treatment of Graves' hyperthyroidism. In: Ingbar SH, Braverman LE, eds. *Werner's the thyroid, fifth edition.* Philadelphia: Lippincott; 1986:1001–1005.
2. Nordyke RA, Gilbert FI Jr. Optimal iodine-131 dose for eliminating hyperthryoidism in Graves' disease. *J Nucl Med* 1991;32:411–416.
3. Nordyke RA, Gilbert FI Jr. Unpublished literature compilation of 10,213 hyperthyroid patients from 48 studies.

Robert A. Nordyke
Fred I. Gilbert, Jr.
Straub Clinic & Hospital
Honolulu, Hawaii

Assessment of Myocardial Infarction with Three Radiotracers

TO THE EDITOR: Henrich et al. (*1*) compared myocardial accumulation of ^{201}Tl, fluorodeoxyglucose (FDG) and a type of ^{123}I-pentadecanoic acid (oPPA) in 32 patients with recent myocardial infarction in the July issue of the *Journal*. The authors are correct in that they measured uptakes rather than metabolic rates of FDG.

There are, however, some areas in this paper that need clarification.

1. One of the problems with this work is that the authors did not perform reinjection of ^{201}Tl to uncover viable myocardium. As Bonow et al. have demonstrated, reinjection induces ^{201}Tl uptake in many segments that show fixed defects on conventional ^{201}Tl scintigraphy (*2*). The power of the reinjection method is probably based on reversion of the unavoidable fall in ^{201}Tl concentration in the blood (*3*). Of course, this problem does not exist when imaging with hexamibi or teboroxime, since two separate injections of radiopharmaceutical are required. Why were these data submitted without reinjection results?
2. Equally bothersome is the absence of a gold standard for definition of infarct size, a parameter which ultimately is critical for prognostication in these patients. Incidentally, and for clinical research purposes, wall thickening assessment (preferably using the three-dimensional volume element approach) is superior to conventional wall motion analyses in distinguishing ischemic from nonischemic zones and in mapping regional function (*4*).
3. Interestingly, of the 128 myocardial quadrants in the 32 patients with fixed ^{201}Tl defects, only 13 were akinetic (Table 1). Of these 13, only 7 had normal FDG uptake, i.e., 7/128 of quadrants (=5%) were akinetic and had normal FDG uptake. Of the 408 myocardial segments with fixed ^{201}Tl defects, 160 (=39%) had normal FDG uptake (Table 2). No wall function data are given for segments.
   Subsequently, we have no idea about infarct size, and there are disparate results for quadrants and segments.
4. The scattergram shown in Figure 3 is not particularly revealing. Theoretically, there is an inverse relationship between uptake of glucose and of free-fatty acids (*5*). In this work, the metabolic conditions at the time of the FDG study and at the time of oPPA study were vastly different (i.e., oral glucose versus overnight fast). While the authors see a correlation between FDG and oPPA uptakes in Figure 3, many would disagree with this assertion.
5. The authors conclude by saying that a minor number of segments showed FDG uptake only, indicating a tendency toward glycolysis and inhibition of fatty acid uptake. Many investigators have now confirmed the data of Liedtke et al. (*6*) that in the reperfused heart fatty acids, not glucose, are the preferred substrate for myocardial oxidation leading to generation of ATP. FDG, in any case, is a poor tracer of the glycolytic pathway (*7*).

REFERENCES

1. Henrich MM, Vester E, von der Lohe E, et al. The comparison of 2-^{18}F-2-deoxyglucose and 15-(ortho-^{123}I-phenyl)-pentadecanoic acid uptake in persisting defects on thallium-201 tomography in myocardial infarction. *J Nucl Med* 1991;32:1353–1357.
2. Bonow RO, Dilsizian V, Cuocolo A, et al. Identification of viable myocardium in patients with chronic coronary artery disease and left ventricular dysfunction. *Circulation* 1991;83:26–37.
3. Budinger TF, Knittel BL. Cardiac thallium redistribution and model. *J Nucl Med* 1987;28:588.
4. Azhari H, Sideman S, Weiss JL, et al. Three-dimensional mapping of acute ischemic regions using MRI: wall thickening versus motion analysis. *Am J Physiol* 1990:H1492–H1503.