merous diagnostic approaches have been developed to evaluate specific aspects of these processes, such as the one proposed by Gould et al. (*2*), which was based on the experimental study by Goldstein (*3*).

It must be remembered however, that the goal of such diagnostic approaches is to identify patients likely to benefit from interventions such as coronary artery bypass grafting or balloon angioplasty. Although improved electrical stability, better infarct remodeling, and the reduction in anginal symptoms are all desirable outcomes, the primary benefit sought by restoring nutritive perfusion to dysfunctional myocardium is improvement in contractile performance. Consequently, criteria for myocardial viability based on detection of specific biochemical processes are going to be most useful clinically when the presence of a specific biochemical trait identifies the capacity for functional recovery. We feel that neither enhanced uptake of ^{18}F-fluorodeoxyglucose with respect to flow nor the demonstration of increased myocardial washout of ^{82}Rb have been shown to unambiguously identify the potential for improved contractile function after revascularization.

In the experimental study of Dr. Goldstein (*3*) which clearly delineated alterations in ^{82}Rb kinetics with the time of ischemia, triphenyltetrazolium chloride (TTC) was used to identify "viable" from irreversibly injured myocardium. The tetrazolium salts are converted to insoluble colored diformazan salts in the presence of reduced coenzymes, dehydrogenases and diaphorases (*4*). The macroscopic observation of "TTC positive" areas within a risk region identifies the presence of cells with these specific biochemical components but does not, a priori, indicate that these cells are functionally intact or would recover active shortening after recanalization. Some investigators have reported that "TTC negative" areas can recover functionally with appropriate interventions (*5*), and conversely, enhanced deoxyglucose uptake has been observed in areas of histologically documented necrosis (*6*, *7*) emphasizing the mutability of such metabolic "fingerprints."

While recovery of contractile function can be delayed temporally after therapeutic interventions and can be difficult to assess clinically, it is the primary goal of therapies for dysfunctional myocardium. Accordingly, we stand by our contention that the accuracy of radionuclide imaging approaches purported to identify viable myocardium can only be ascertained by direct comparisons with changes in regional function.

REFERENCES

1. Gropler RJ, Bergmann SR. Myocardial viability—What is the definition? [Editorial]. *J Nucl Med* 1991;32:10–12.
2. Gould KL, Yoshida K, Hess MJ, et al. Myocardial metabolism of fluorodeoxyglucose compared to cell membrane integrity for the potassium analogue rubidium-82 for assessing infarct size in man by PET. *J Nucl Med* 1991;32:1–9.
3. Goldstein RA. Kinetics of rubidium-82 after coronary occlusion and reperfusion—assessment of patency and viability in open-chested dogs. *J Clin Invest* 1985;75:1131–1137.
4. Klein HH, Puschmann S, Schaper J, et al. The mechanism of the tetrazolium reaction in identifying experimental myocardial infarction. *Virchows Arch (Pathol. Anat)* 1981;393:287–297.
5. Barnard RJ, Okamoto F, Buckberg GD, et al. Studies of controlled reperfusion after ischemia. III. Histochemical studies: inability of triphenyltetrazolium chloride nonstaining to define tissue necrosis. *J Thorac Cardiovasc Surg* 1986;92:502–512.
6. Bianco JA, Sebree L, Subramanian R, et al. C-14-deoxyglucose accumulation in myocardial infarction [Abstract]. *J Nucl Med* 1990;31:835.
7. Sebree L, Bianco JA, Subramanian R, et al. Discordance between accumulation of C-14-deoxyglucose and T1-201 in reperfused myocardium. *J Mol Cell Cardiol* 1991;23:in press.

Robert J. Gropler
Mallinckrodt Institute of Radiology
Washington University School of Medicine
St. Louis, Missouri

Steven R. Bergmann
Washington University School of Medicine
St. Louis, Missouri

In Vivo Quality Control of Technetium-99m-HMPAO

TO THE EDITOR: Technetium-99m-HMPAO is a useful radiopharmaceutical to determine brain death noninvasively. Scanning can be performed even at an intensive care unit using a mobile gamma camera. In a recent report, Laurin et al. published their results (*1*). In a letter to the editor, Brandau et al. (*2*) pointed at the possibility of a labeling failure since this pharmaceutical is susceptible to inaccuracies occuring during the labeling procedure. Actually, a false-positive result obtained in the scintigraphy can be hazardous. Brandau et al. (*2*) suggest performing an in vitro quality control (chromatography) as well as an in vivo quality control by scanning the thyroid gland, lung, and liver of the patient.

We also use ^{99m}Tc-HMPAO for the determination of brain death. We perform an in vivo quality control by preparing a larger amount of tracer (30 mCi) and injecting one-half of the vial into the patient, in whom brain death is suspected and the other half to a patient, who requires ^{99m}Tc-HMPAO SPECT imaging during the clinical routine for different reasons (e.g., Alzheimer's disease).

Cerebral tracer uptake within a normal range in the latter patient proves the integrity of the prepared radiopharmaceutical. In most cases, brain death does not have to be determined immediately, so there are no logistical problems for injecting both patients at the same time. In our opinion, this procedure represents an elegant alternative of "in vivo quality control" to the one suggested by Brandau et al. (*2*), since only one scan has to be performed in the patient with suspected brain death. Nonvisualization of the thyroid gland, which was taken as a proof for the absence of free pertechnetate (*2*), can be caused by a thyroid blockade (e.g., contrast media).

REFERENCES

1. Laurin NR, Driedger AA, Hurwitz GA, et al. Cerebral perfusion imaging with technetium-99m-HMPAO in brain death and severe central nervous system injury. *J Nucl Med* 1989;30:1627–1635.
2. Brandau W, Schober O, Knapp WH. Determination of brain death with technetium-99m-HMPAO (Letter). *J Nucl Med* 1990;31:2075–2076.

F. Grünwald
H. J. Biersack
University of Bonn
Bonn, Germany