has not fully been elucidated. It may be either the hyperdynamism secondary to the hypermetabolism of total body skeletal muscle or the direct involvement of myocardium. The direct involvement of cardiac muscle has been suspected with microscopic observation at necropsy (*3*) and thallium-201 myocardial scan (*4*). In contrast, absence of myocardial involvement has been reported with the histologic examination at endomyocardial biopsy (*5*).

The uptake of [^{99m}Tc]PYP employed in the present study is limited to the necrotic and severely injured cells (*7,8*) and a positive accumulation is demonstrated 12 hr to 6 days after the muscle injury (*6*). Kawamoto et al. (*9*), using [^{99m}Tc]PYP scintigraphy, demonstrated absence of myocardial involvement following recovery from MH crisis. Their report was, however, not conclusive to rule out the myocardial involvement, since their patient did not develop either cardiac arrest nor severe cardiac dysfunctions during the crisis. To our knowledge, our report is the first to demonstrate that the myocardium is not involved by the pathogenesis of MH. It is thus strongly suggested that the severe and sometimes fatal cardiac dysfunctions occurring during the hyperthermic crisis are secondary to either or both the cardiac hyperdynamism due to the elevated skeletal muscle metabolism and hyperkalemia secondary to the muscular destruction. The prompt restoration of sinus rhythm following intravenous administration of calcium gluconate observed in the present patient support this postulate.

References

1. Gronert GA. Malignant hyperthermia. *Anesthesiology* 1980; 53:395–423
2. Britt BA. Malignant hyperthermia. *Can Anaesth Soc J* 1985; 32:666–677
3. Fenoglio JJ Jr, Irey NS. Myocardial changes in malignant hyperthermia. *Am J Pathol* 1977; 89:51–58
4. Huckell VF, Staniloff HM, Britt BA, Waxman MB, Morch JE. Cardiac manifestations of malignant hyperthermia susceptibility. *Circulation* 1978; 58:916–925
5. Mambo NC, Silver MD, McLaughlin PR, et al. Malignant hyperthermia susceptibility. A light and electron microscopic study of endomyocardial biopsy specimens from nine patients. *Hum Pathol* 1980; 11:381–388
6. Willerson JT, Parkey RW, Bonte FJ, Meyer SL, Atkins JM, Stokely EM. Technetium stannous pyrophosphate myocardial scintigrams in patients with chest pain of varying etiology. *Circulation* 1975; 51:1046–1052
7. Dewanjee MK, Kahn PC. Mechanism of localization of Tc-99m-labeled pyrophosphate and tetracycline in infarcted myocardium. *J Nucl Med* 1976; 17:639–646
8. Buja LM, Tofe AJ, Kulkarni PV, et al. Sites and mechanisms of localization of technetium-99m phosphorus radiopharmaceuticals in acute myocardial infarcts and other tissues. *J Clin Invest* 1977; 60:724–740
9. Kawamoto M, Yuge O, Kikuchi H, Kodama K, Morio M. No cardiac involvement in nonrigid malignant hyperthermia. *Anesthesiology* 1986; 64:93–94.

Masahiro Murakawa
Yoshio Hatano
Kenjiro Mori
Tatsuo Torizuka
Nagara Tamaki
Renpei Iwasaki
Kyoto University
Kyoto, Japan

Dynamic Xenon-133 SPECT in Dementia

TO THE EDITOR: In the paper "Assessment of Demented Patients by Dynamic SPECT of Inhaled Xe-133" (*J Nucl Med* 1988; 29:1621–1626), the authors report their experience with the evaluation of regional cerebral blood flow (rCBF) employing single photon emission tomographic (SPECT) determination using inhaled xenon-133 (^{133}Xe) in a group of patients with Alzheimer's disease (AD), senile dementia of the Alzheimer type (SDAT), multi-infarct dementia, as well as in a group of elderly control subjects. This is an interesting work, but it contains two significant errors:

1. In our own experience with inhaled [^{133}Xe]SPECT (*1–4*) we have found that the pattern of flow reduction in the posterior temporal and parietal regions may be symmetrical in patients with early or moderately advanced AD or SDAT, but it is quite commonly asymmetrical, with significant differences in relative posterior flow reduction. Further, left or right frontal flow reduction may occur in a smaller number of patients. There is a tendency for flow reduction to match general clinical symptoms, with left posterior rCBF deficits accompanying short-term memory problems, right posterior deficits accompanying spatial orientation difficulties, left frontal flow deficits in patients with aphasia, etc. These asymmetries have also been identified by investigators using other SPECT techniques (*5*).

2. The authors are not the first to report experience with ^{133}Xe inhalation SPECT in the entities under study. We, and our colleagues, have described our own experience in a similar group of patients in three abstracts (*1–3*), and one peer-reviewed paper (*4*), all published in universally available journals.

References

1. Bonte FJ, Devous MD Sr, Chehabi HH, Ross ED. Spect study of regional cerebral blood flow in dementia. *J Nucl Med* 1986; 27:732–733.
2. Bonte FJ, Devous MD Sr, Ajmani AK, Weiner MF, Hom J, Tintner R. Testing vasoreactivity in patients with dementias. *J Nucl Med* 1987; 27:407–408.
3. Bonte FJ, Devous MD Sr, Ajmani AK, Weiner MF, Hom J, Tintner R. Testing vasoreactivity in patients with dementias or cerebrovascular disease. *J Cereb Blood Flow Metab* 1987; 7:S201.
4. Bonte FJ, Ross ED, Chehabi HH, Devous MD Sr. SPECT study of regional cerebral blood flow in Alzheimer's disease. *J Comput Assist Tomogr* 1986; 10:579–583.
5. Derouesne C, Rancurel G, Le Poncin Lafitte M, Rapin JR, Lassen NA. Variability of cerebral blood flow defects in Alzheimer's disease on 123-iodoisopropyl-amphetamine and single-photon emission tomography. *Lancet* 1985; 1:1282.

Frederick J. Bonte
Michael D. Devous
The University of Texas
Southwestern Medical Center
Dallas, Texas

REPLY: We address the points in the letter by Bonte and Devous below.

1. Patients with dementia employed in our study were early stage and could not receive an examination of Hasegawa's

Dementia scale. They did not reveal any symptom of aphasia, hemiparesis or other dyskinesia; only memory problem and/or disorientation were present. In such patients, we could not find any asymmetric reduction, and the frontal reduction in cerebral blood flow was not observed.

2. We were previously unaware of the abstracts and paper. However, the paper does not discuss the differentiation of the patterns of regional reduction in cerebral blood flow among Alzheimer's disease, senile dementia Alzheimer type and multi infarct dementia. Also, our report appears original in the correlation between reduction in regional cerebral blood flow and degree of dementia in each demented group.

Akio Komatani
Yamagata University
School of Medicine
Zuolida Yamagata, 990-23, Japan

Radiation Exposure by Technitium-99m MAG_3

TO THE EDITOR: According to Taylor and colleagues (*1*) the radiation exposure by technetium-99m (^{99m}Tc) MAG_3 for the kidneys of normal volunteers is 0.018 rad/mCi; the dosimetry calculations have been only briefly described (*2*). This value is 50% higher than the former given by this same team (0.012 rad/mCi) (*3*), that was not discussed in this paper.

Mallinckrodt Diagnostica (Netherlands) B.V., the commercial producer of [Tc]MAG_3 for Europe, indicates in the European Registration File an organ dose for the kidneys of 0.062 rad/mCi, that means the 3.4-fold (*1*) or 5.2-fold (*3*) value (all calculations followed standard MIRD methods). Surprisingly, there are no major differences between the analogous results for iodine-123 (^{123}I) and iodine-131 (^{131}I) orthoiodohippurate (OIH), respectively, by Taylor et al. and the European reference values (*4*). In comparison with [^{123}I]OIH (0.020 rad/mCi) (*4*) Taylor et al. found significantly lower while Mallinckrodt found distinctly higher values for [^{99m}Tc]MAG_3. These differences are considerable and should be proved by more related measurements with detailed information about technique and methods of calculation.

Our theoretic estimate showed the following results: the effective half-lives of the radiopharmaceuticals mentioned above at normal renal function are approximately equivalent (*1,5*) and the absorbed energy fraction of the gamma radiation is nearly the same for both nuclides. Furthermore, the part of nonpenetrating conversion electrons additionally generated during decay is comparable, too. But because of the essentially higher blood concentration of [^{99m}Tc]MAG_3 compared with [^{123}I]OIH (*1,5*), the activity level of ^{99m}Tc in the kidneys is higher than of ^{123}I resulting in a higher organ exposure by [^{99m}Tc]MAG_3 in normal functioning kidneys. In severely reduced renal function the radiation exposure may be less by [^{99m}Tc]MAG_3 than by [^{123}I]OIH because of the distinctly shorter physical half-life of ^{99m}Tc compared with ^{123}I.

References

1. Taylor A Jr, Eshima D, Christian PE, Wooten WW, Hansen L, McElvany K. Technetium-99m MAG_3 kit formulation: preliminary results in normal volunteers and patients with renal failure. *J Nucl Med* 1988; 29:616–622.
2. Wooten WW, Eshima D, Taylor A Jr. Radiation absorbed dose from I-131 OIH and Tc-99m MAG_3 [Abstract]. *J Nucl Med* 1986; 27:898.
3. Taylor A Jr, Eshima D, Fritzberg AR, Kasina S, Christian PE. Preliminary evaluation of Tc-99m mercaptoacetyltriglycine as a replacement for I-131 OIH. *Contr Nephrol* 1987; 56:38–42.
4. Roedler HD, Kaul A, Hine D. Internal radiation dose in diagnostic nuclear medicine. Berlin: Hoffmann, 1978.
5. Bubeck B, Brandau W, Eisenhut M, Weidenhammer K, Georgi P. The tubular extraction rate (TER) of Tc-99m MAG_3: a new quantitative parameter of renal function. *Nuc Compact* 1987; 18:260–267.

Bernd Bubeck
University of Heidelberg
Im Neuenheimer Feld 400
D-6900 Heidelberg, FRG

REPLY: We appreciate Dr. Bubeck's letter regarding radiation dosimetry and especially his request for "more related measurements with detailed information about technique and methods of calculation."

Dr. Bubeck's letter focuses on the estimated dose to the kidneys from [^{99m}Tc]MAG_3 and notes that our estimates (*1, 3*) are one-third to one-fifth the estimate reported by Mallinckrodt Diagnostica (Netherlands) B.V. in the "European Registration File." Our lower estimate was based on the kinetics of high performance liquid chromatography purified [^{99m}Tc]MAG_3 while the higher estimate was based on the kit formulation. The kinetics are slightly different (*2,4*). Probably more important than the kinetic differences are differences in the assumptions and the methods of calculation used in the two studies. These are briefly outlined below.

The MIRD method is quite universally adopted for dosimetry calculations and is probably applied correctly by all parties. The MIRD method requires, however, an estimate of the amount of activity in the source organ as a function of time and herein lies the challenge and potentially large discrepancies. When estimating the dose to the kidney, the radiation to the kidney from the bladder and other organs is practically insignificant compared to the radiation to the kidney from the kidney itself; for this reason we have to focus on the amount of activity in the kidney as a function of time. The estimates we reported (*1–4*) were based on Anger camera images acquired posteriorly over the kidneys and stored in a mini-computer at 20 sec per frame for 20 min. The activity in both kidneys as a function of time was estimated from regions of interest over the kidneys. The system was calibrated by imaging a kidney phantom (Alderson Research Laboratories) in a water bath with the center of the phantom at depths ranging from 5 to 8 cm. The calibration factor for each volunteer depended on his own kidney depth as estimated by the formulas of Tonnesen et al. (*5*). We note that the formulas of Tonnesen were supported by Tanasescu et al. (*6*) by comparison to ultrasound. We feel that this method of estimating the activity in the kidney is probably accurate to within 15%, especially when averaged over ten volunteers (as was done for our dose estimation). The weakness of this method is that Anger camera images were not extended beyond 20 min and the longer term activity in the kidney can be significant. In