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