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REPLY

Thank you for the opportunity to respond to the letter of Drs. Rosenberg and Gronert.

We agree that the diagnosis of malignant hyperthermia may be difficult and must be made with care. Not treating an ongoing potential attack just because it is not classical may lead to serious consequences.

Our first patient surely had an MH attack, possibly potentiated by the hypothermia and shivering. He had not been drinking for nine months. Besides he was quite lucid, alert and orientated and not tremulous in the recovery room just prior to the temperature elevation, making an alcohol withdrawal state quite unlikely. We agree that none of his laboratory values went severely awry. He was promptly treated with dantrolene, as well as supportive treatment. When we began treatment we already had the results of the muscle biopsy which was done at the same time as the carpal tunnel release. The caffeine contracture response, both in the presence and absence of halothane were well within the susceptible range as provided by Dr. Britt's laboratory. There was no contracture in the presence of one per cent halothane, and, as the writers are aware, three per cent halothane is not used in Dr. Britt's laboratory, perhaps leading to some difference in results. There is not general agreement that the response to halothane is the most specific test for the diagnosis of MH.^{1,2}

Case two is even less in doubt. He had a biopsy because of family history of MH in the mother, and was associated

with a more vigorous metabolic disturbance, as well as myoglobinuria.

Temperature and pulse rate fell to normal ranges within 20 minutes of the institution of dantrolene and supportive therapy, although through the next 24 hours further treatment was necessary to maintain him. The acidosis improved over the initial five hours of treatment, although again, over the initial course there was some fluctuation necessitating further treatment. Blood cultures done at the height of the fever were reported as showing no growth, so a bacteremia is quite unlikely, both on clinical grounds as well as on laboratory grounds. This man's biopsy showed spontaneous halothane contractures, as well as being sensitive to caffeine alone, and in the presence of halothane.

The third patient would not submit to muscle biopsy, so the diagnosis of MH could not be confirmed. There was no previous or subsequent hypertension or any other metabolic disturbance to suggest pheochromocytoma, although catecholamine levels were not drawn to confirm that possibility. Again, we submit that to a clinician, a reaction of this sort, in the presence of this family history, is very suggestive of malignant hyperthermia.

The defense of the caffeine contracture test itself we leave to Dr. Britt. Certainly other laboratories use the test successfully to differentiate MH patients.^{2,3} We must be guided by the wisdom of our colleagues in the laboratory to test and diagnose the muscle and tell us that that patient does have malignant hyperthermia. It seems to us that no harm was done to any of the patients, as the first two already had been labeled as MH by their attending physician and the laboratory, and patient three was advised to have a muscle biopsy.

We suggest the occasional failure to diagnose MH in a patient is a more grievous error than occasionally labelling a patient without MH as having the disease.

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