tory, chosen because it is the closest of 11 centers in North America recognized by the Malignant Hyperthermia Association of the United States. Ellis and Halsall should be aware that, although this center and our hospital both happen to be located in Boston, we remain “fiercely independent” (like any good New Englanders) of any “group” labels. What remains undisputed, however, is the fact that masseter spasm correlates with MHS in a majority of patients who have in vitro muscle biopsy testing. This is true with our study using calcium uptake, as well as with other reports using halothane and caffeine contracture tests. Ellis and Halsall provide additional support with their own incidence of 64%.

Van Der Spek and his colleagues attempt to offer explanations other than MHS for the development of masseter spasm. These include:

1. The patients may not have been fully paralyzed. However, all received at least 1 mg/kg of succinylcholine intravenously before intubation, making this an unlikely factor. Transcutaneous nerve stimulation revealed complete relaxation in the two patients who were monitored at the time of their masseter spasm and, in all cases in which peripheral muscle tone was noted, the extremities were flaccid. Furthermore, 10 min of jaw rigidity can usually be differentiated from “light” anesthesia.

2. Serum creatine phosphokinase (CPK) and myoglobin levels can increase after uncompleted anesthetics without malignant hyperthermia being a factor. While it is true that mild increases after anesthesia and surgery certainly can occur, CPK values up to 40,000 IU are distinctly abnormal. Ignoring this in any patient, especially when associated with myoglobinuria, is dangerous.

3. In many cases, no other clinical or laboratory signs indicative of malignant hyperthermia were present other than masseter spasm. This is precisely why masseter spasm is such an important finding. It permits the early detection of a potential problem before other life-threatening events develop. The child who developed myoglobinuria and a CPK over 40,000 IU is a good example; he had no other laboratory abnormalities, including blood gases and temperature, and even the initial CPK at the time of the masseter spasm was unremarkable (280 IU). The prompt discontinuation of anesthetic agents probably explains why dantrolene is usually not necessary in these situations. If surgery is urgent, discontinuing the use of all potential triggering agents is prudent, not continuing the administration of halothane as Van Der Spek and colleagues suggest. The use of dantrolene should be considered strongly when surgery cannot be postponed.

In summary, our study showed that masseter spasm develops in approximately 1% (15/1460) of children who are anesthetized with halothane followed by intravenous succinylcholine. These patients are likely to be positive to in vitro muscle biopsy testing for MHS. These observations should not be casually disregarded because of a controversy surrounding current muscle biopsy assays. Recent reports suggest that some of these patients have a muscle response that may be distinct from normal or MHS. Unfortunately, these patients cannot be differentiated clinically from those with MHS. Therefore, until more is known about this condition, masseter spasm after induction of anesthesia should not be dismissed as a benign event.

LYNNAE SCHWARTZ, M.D.
MARK A. ROCKOFF, M.D.
BABU V. KOKA, M.D.
Department of Anesthesia
Children's Hospital and Harvard Medical School
Boston, MA 02115

REFERENCES


(Accepted for publication September 13, 1985.)

Effect of Ventilation on Baseline Pulmonary Artery Temperature

To the Editor.—A stable baseline temperature in the pulmonary artery contributes importantly to the accuracy of thermodilution cardiac output measurement with a pulmonary artery catheter.1,2 Recently, we observed an exaggerated variability of thermodilution cardiac outputs occurring in a patient undergoing coronary artery bypass grafting. We injected thermal boluses at various times in the respiratory cycle in duplicate or triplicate as recom-

mended by Snyder and Powner,3 using a closed system and an American Edwards* thermodilution pulmonary artery catheter and cardiac output computer. Injectate temperatures were 1–4° C. With otherwise stable hemodynamics, the cardiac outputs ranged from 4.1 to 6.8 l/min, and the thermodilution curve configurations appeared abnormal. Figure 1A shows an accessory hump preceding the ascent despite rapid consistent injection of
Ester or Amide Local Anesthetics in Malignant Hyperthermia—Who Knows?

To the Editor.—Adragna1 asks: "Is there any evidence that amide local anesthetics are contraindicated in malignant hyperthermia susceptible patients (MHSP), or is our habit of avoiding them just a habit?" As he recognizes, both the amides and esters have been used safely in malignant hyperthermia susceptible pigs. Furthermore, he is aware that the Malignant Hyperthermia Association* has stated that, based on limited clinical and laboratory experience, all local anesthetic drugs appear to be safe for the MH susceptible individuals. On the other hand, the ASA Technical Bulletin† advises that the amide type local anesthetics should be avoided in the MHSP.
