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 uncomplicated anesthetics without malignant hyperthermia being a factor.* While it is true that mild increases after anesthesia and surgery certainly can occur,[1,2] 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[3,4] 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.

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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 recommended 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

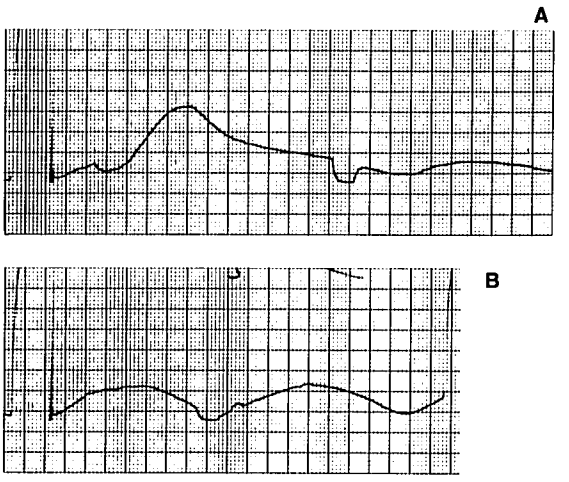

FIG. 1. Thermodilution curves from the same patient over a short sequence. American Edwards 9811 Strip Chart Recorder® sensitivity was 4 cm/V, paper speed 5 mm/s. A: thermal injection, output shown beside curve. B: mock injection during IPPV.

the thermal bolus. An additional hump was seen in the terminal decay phase, although this would not influence the calculated cardiac output. Other curves showed unusual fullness in the initial exponential decay phase. Suspecting an unstable baseline temperature, we observed the baseline during a mock injection (fig. 1B). The baseline variability occurred simultaneously with ventilator breaths. Inspired gas flowed at 3 l/min of unheated oxygen through a semiclosed circle system. The fluctuations we observed in temperature appear to exceed those reported by Woods and co-authors during open-chest, intermittent, positive pressure ventilation (IPPV) and baseline PA temperature averaging.[4]

Assuming that IPPV-induced baseline temperature fluctuations caused the large variability in cardiac output measurements, we placed a heat and moisture exchanger (Engstrom Edith Flexitube Humidifier®) between the Y-connector and the endotracheal tube. Insertion of this "artificial nose" greatly reduced baseline variation during mock injections, resulting in improved reproducibility of repeated thermodilution cardiac outputs (range 6.0–6.6 l/min). The heat and moisture exchanger provided a simple solution to this problem. We believe that occurrences such as this support routinely displaying the thermodilution curve generated by individual thermal injections, because the inscribed curve may assist in troubleshooting excessive cardiac output variability. Additionally, the need for thermodilution cardiac output measurements may represent an unusual indication for heating inspired gases or using low fresh gas flows.

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64:294–296, 1986

Ester or Amide Local Anesthetics in Malignant Hyperthermia—Who Knows?

To the Editor:—Adragna[1] 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.

* Adragna MG: The Communicator 3(4):1; 1985 published by Malignant Hyperthermia Association of the United States. Box 3231, Darien, CT 06820.

† American Society of Anesthesiologists: Technical bulletin for malignant hyperpyrexia. Newsletter 46:5, 1982