One patient vomited during induction. Ten patients had maximal rectal temperatures of 37.6° C, a normal value. Of the two pyrexic patients, one had fulminant epiglottitis and the other had pulmonary disease. One patient had nothing but one temperature taken. Arterial blood gases and serum potassium levels were all normal. Myoglobin levels were not measured or were missing. Simultaneous administration of halothane and succinylcholine is a potent stimulator of myoglobin and creatinine phosphokinase release,[2] as are intramuscular injections and trauma (four patients underwent fracture reduction).

"Aborted" malignant hyperthermia has been proposed to explain "isolated" symptoms, such as masseter spasm.[3] A positive calcium uptake assay in patients with masseter spasm is indicative of susceptibility to malignant hyperthermia and warrants a Medic Alert bracelet.* The validity, however, of this assay is unknown; the method has not received peer review.† The test's reproducibility, verifiability, sensitivity, and specificity are undetermined.[4] Still, none of the 12 patients with an abnormal assay developed a hypermetabolic muscle syndrome consistent with malignant hyperthermia, making inference of malignant hyperthermia susceptibility contentious. If, however, Schwartz *et al.* consider masseter spasm a harbinger of malignant hyperthermia, advising not to institute dantrolene treatment seems contradictory.

The authors' estimation of the incidence of masseter spasm is unclear. Did the 15 cases of masseter spasm occur in the total population's subgroup of 2920 cases to which halothane and succinylcholine were administered? This would give an incidence of 0.51% or 1/200. Accepting for a minute that masseter spasm is the harbinger of malignant hyperthermia, then either malignant hyperthermia has been poorly defined and its prognosis has been too gloomy, or most calamities attributable to malignant hyperthermia must have gone unnoticed.

Difficulty in opening the mouth after induction is clinically judged; its interpretation may vary from tightness to trismus. Retrospective differentiation between normal and abnormal responses is difficult. We have observed jaw stiffness in children without a history of prior disease during general anesthesia, without subsequent development of malignant hyperthermia. While continuing general anesthesia with halothane, we waited until the jaw relaxed, intubated the patient, and proceeded with anesthesia using appropriate monitoring for periods exceeding 1 h. Perhaps the muscles of mastication show a response to succinylcholine (and halothane), which is akin to the response of human extraocular muscles or of tonic muscles;[5] this contractile property may be modified during maturation.

This paper does not support the notion that "masseter spasm" indicates the presence of malignant hyperthermia. A prospective trial is needed to establish the incidence of masseter spasm and its relation to malignant hyperthermia. We are concerned that patients with an unexpected response to succinylcholine will be labeled malignant hyperthermia susceptible. The implications of this article and its suggestion, to monitor patients with masseter spasm in the intensive care unit postoperatively, have far reaching medical and legal consequences.

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In Reply:—The purpose of our report was not to advocate one particular muscle biopsy methodology for the determination of malignant hyperthermia susceptibility (MHS). On the contrary, we clearly noted that controversy exists regarding a laboratory "gold standard." Our muscle biopsy tests were performed by an independent labora-

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