Increased Incidence of Masseter Spasm in Children with Strabismus
Anesthetized with Halothane and Succinylcholine

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Muscle rigidity is known to be an early sign of malignant hyperthermia, a life-threatening metabolic response triggered by certain anesthetics.1 Masseter spasm is rigidity limited to the muscle responsible for clenching the jaws. Masseter spasm may herald a life-threatening episode of malignant hyperthermia (MH), or it may occur in isolation, without signs of rhabdomyolysis (such as elevated creatine phosphokinase (CPK) and myoglobinemia) and without progression to the full metabolic derangement seen in malignant hyperthermia.2

In 1984, Schwartz et al.3 published a review of 6500 anesthetics from a children’s hospital. The overall incidence of masseter spasm was reported to be 1 in 800. However, in the subgroup of cases in which anesthesia was induced with halothane followed by succinylcholine (a very common and convenient induction technique for the pediatric age group), the incidence was about eight times higher, 1 in 100. None of the cases of masseter spasm in that report occurred in children undergoing eye muscle surgery, and none developed into MH. However, these authors stopped surgery when masseter spasm occurred.

Strabismus may be an early sign of an underlying myopathic disorder that could predispose to malignant hyperthermia.4 We sought to determine, retrospectively, the incidence of masseter spasm in children undergoing eye muscle surgery for correction of strabismus. All of these patients were anesthetized with halothane and given succinylcholine intravenously to facilitate tracheal intubation, a combination of drugs frequently used in patients undergoing surgery at our hospital. All the episodes of masseter spasm in Schwartz et al.’s study5 occurred with this combination of drugs.

MATERIALS AND METHODS

We reviewed the anesthesia records of all children anesthetized at the Children’s Hospital of Pittsburgh during the 18-month period from October 1, 1983 to March 31, 1985. We included all cases in which halothane was used for induction and intravenous succinylcholine was given subsequently to facilitate tracheal intubation or to diminish laryngospasm. No patient received intramuscular succinylcholine. We excluded patients given other inhalational or intravenous agents.

Masseter spasm was defined as jaw tightness interfering with intubation that occurred despite adequate doses of succinylcholine. Its occurrence was the clinical judgment of the supervising anesthesiologist. There was no consistent recording of muscle twitch, CPK, blood gas tensions, or other laboratory studies in the records. The data were statistically analyzed by Fisher’s exact test, which is used to compare two groups of very different size; we accepted a minimum level of significance of P < 0.05.

RESULTS

A total of 1,468 children received halothane followed by succinylcholine during the study period. Of these, 211 underwent surgery to correct strabismus. Fifteen episodes of masseter spasm were reported in the records of the 1,468 halothane anesthetics. Six of the 15 episodes of masseter spasm occurred in the 211 strabismus patients (table 1, P < 0.05 by Fisher’s exact test).

The overall incidence of masseter spasm in patients receiving halothane-succinylcholine for induction was 1.02%. The incidence was 2.8% among the 211 strabismus patients compared with 0.72% in patients without strabismus, a fourfold difference.

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None of the 15 patients with masseter spasm had any family history of adverse reaction to anesthesia. The majority, including all the strabismus patients, were unpremedicated; the rest had various combinations of intramuscular atropine, barbiturates, morphine, and scopolamine. Three patients had had prior anesthetics, and 12 had not. Of the former group, two had had uneventful anesthesia with halothane without succinylcholine, and details were not available for the third.

One case was stopped after masseter spasm occurred, mainly because of concern that the patient had also aspirated gastric contents. All other cases were continued when end-tidal CO₂ concentration and arterial O₂ saturation remained normal. In three of the 15 cases of masseter spasm halothane was discontinued, and anesthesia was continued with fentanyl and a nondepolarizing muscle relaxant (usually atracurium). Most cases in which halothane was continued lasted less than 1 h.

One patient, who was thought to have MH, had metabolic acidosis on intraoperative arterial blood gas analysis. This patient had asthma and experienced increased wheezing after aspirating a peanut. Blood gas analysis had not been performed preoperatively. After induction of anesthesia, masseter spasm was noted along with cyanosis and hypercarbia, but without rigidity of other skeletal muscles. The patient was undergoing bronchoscopy to remove the peanut from the airway, and the procedure was not abandoned, but halothane was discontinued. The patient was treated with dantrolene because of the concurrent cyanosis and metabolic acidosis, although these could have been due to airway obstruction from the peanut or from the underlying asthma. No arrhythmias were noted, and the temperature, which was elevated before induction, decreased during the procedure. The child required postoperative care in the intensive care unit for wheezing. A second dose of dantrolene was not needed. This patient (who had received no intramuscular premedication) had a postoperative CPK level of 2508. The parents refused to consider muscle biopsy.

Arterial or venous blood gas tensions were measured in five of the patients with masseter spasm and found to be abnormal in only the patient reported above. CPK was measured in only three patients and was elevated (2508, 4558, and 707) in all of these. These CPK measurements were all obtained within the first 6 h after masseter spasm occurred. Except for the possible case of MH described above, the patients with increased CPK showed no other sign of MH—no arrhythmia, hypercapnia, fever, or tachycardia. Urine myoglobin was not measured in any patient.

Arrhythmias were noted in the charts of only two patients with masseter spasm. One patient had sinus tachycardia with a rate of 170, and another had bigeminy. In both of these cases, halothane was continued when acidosis, hypoxia, and hypercapnia were found to be absent.

Although no patient had a muscle biopsy, two patients returned to our hospital for anesthesia after the episode of masseter spasm. One was given thiopental followed by succinylcholine and halothane; the other had halothane and atracurium. We think that unavailability of old records was the reason the patients received these anesthetic agents. Both subsequent anesthetics were uneventful.

### DISCUSSION

In the total group of patients anesthetized with halothane and succinylcholine, the incidence of masseter spasm, 1.02%, is the same as that reported by Schwartz, et al. 5 In the subset of patients with strabismus, the incidence of masseter spasm was nearly four times higher than in other patients who also received halothane-succinylcholine. By contrast, no case of masseter spasm occurred in patients with strabismus in Schwartz’s study. However, in their sample, the only neuromuscular blocking drug given to patients with strabismus was pancuronium (personal communication from MA Rockoff), which may explain why no case of masseter spasm occurred in their patients with strabismus.

Rosenberg and Fletcher5 have reported that 50% of patients who had masseter spasm and, subsequently, had muscle biopsies and contracture testing proved to be MH susceptible. However, their patients were referred from various hospitals, and do not represent all patients from a given location who experienced an episode of masseter spasm. If Rosenberg and Fletcher’s 50% incidence of MH susceptibility among patients with masseter spasm is applied to our patients, 1 in 200 may be MH susceptible, and possibly 3–4 in 200 patients with strabismus may be MH susceptible. This differs from the widely quoted figure of 1 in 15,000 incidence of MH in children. Perhaps patients with masseter spasm who are referred for biopsy represent a population biased toward MH susceptibility.

When masseter spasm occurs after induction of anesthesia with halothane-succinylcholine, as long as there is no biochemical evidence of MH, we do not ask that...
Heart Rate and Rhythm Following an Edrophonium/atropine Mixture for Antagonism of Neuromuscular Blockade during Fentanyl/N₂O/O₂ or Isoflurane/N₂O/O₂ Anesthesia

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The efficacy and sustained duration of antagonism of neuromuscular blockade by edrophonium has become widely accepted since the initial reports of its use by Kopman1 and Bevan.2 Its rapid onset of action and reduced atropine requirement may make edrophonium preferable to the use of neostigmine or pyridostigmine,3–6 and the drug of choice for antagonism of atracurium and vecuronium.6–8

Azar et al. demonstrated that administration of an edrophonium-atropine mixture produced superior heart rate stability when compared to administration of an edrophonium-glycopyrrolate mixture.9 Cronnelly et al. demonstrated that the simultaneous administration of 0.5 mg/kg of edrophonium and 7 μg/kg of atropine produced fewer changes in heart rate than either neostigmine and atropine or edrophonium, and higher doses (15 μg/kg or 30 μg/kg) of atropine.4 Both studies reported the occurrence of dysrhythmias after the administration of an edrophonium-atropine mixture. Miller and Cronnelly3 suggested, in their 1983 editorial, that further study of the action of edrophonium was required, including a critical look at the assumption...