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Masseter Spasm with Anesthesia: Incidence and Implications

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Masseter spasm after succinylcholine administration is a well-documented event. In many cases it has been the harbinger of an acute malignant hyperthermia (MH) episode.¹⁻⁷ Despite this, the incidence of masseter spasm after succinylcholine administration is unknown. In addition, it is not clear whether masseter spasm can develop with induction techniques not utilizing succinylcholine. We therefore examined all cases of masseter spasm that occurred in our operating rooms over a 14-month period and evaluated patients for potential susceptibility to malignant hyperthermia.

METHODS

The medical records were reviewed on all patients from the Children's Hospital in Boston who, from January 1, 1982, through February 28, 1983, were admitted to the recovery room after developing masseter spasm in the operating room. The diagnosis of masseter spasm was established if the attending anesthesiologist found it extremely difficult, if not impossible, to open the patient's mouth after induction of anesthesia. Normal jaw mobility was documented before and after the event.

If succinylcholine was used to facilitate endotracheal intubation, a dose of at least 1 mg/kg given intravenously was considered necessary to assure adequate muscle relaxation.

We reviewed a random sample of 6,500 anesthetic records, or 53% of the 12,169 anesthetics delivered at our institution during that same time period, in order to determine the incidence of masseter spasm overall, and with different anesthetic techniques. The age distribution of patients who experienced masseter spasm was correlated with the age distribution of all patients who were anesthetized during that time; these data were analyzed by the Poisson probability equation.

All patients were seen and evaluated by the anesthesia service preoperatively. All received standard intraoperative care, including monitoring of the pulse, respirations, blood pressure, electrocardiogram, and temperature. By previously established protocol, patients who developed masseter spasm had a blood sample drawn promptly in the operating room for arterial blood gases, serum electrolytes, and creatine phosphokinase (CPK) levels. Postanesthetic care included careful observation, measurement of serial arterial blood gases, and serum CPK levels. Urinalyses were considered positive for myoglobin if they were dipstick positive for hemoglobin/myoglobin in the absence of red blood cells or evidence of intravascular hemolysis.

Whenever possible and after informed consent, muscle tissue was obtained during a subsequent anesthetic that included nitrous oxide, barbiturates, narcotics, nondepolarizing muscle relaxants, droperidol, or diazepam. Dantrolene pretreatment was not given in order to avoid any possible interference with the assay. The specimen immediately was placed in liquid nitrogen and sent with a summary of the clinical history to the nearest

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regional malignant hyperthermia center§ and assayed for MH susceptibility by the technique of Allen, Mabuchi, Sreter, and Ryan.⁸⁻¹⁰ Calcium uptake in thin sections was performed.

RESULTS

Fifteen cases of masseter spasm were identified out of a total of 12,169 anesthetics administered over a 14-month period. Therefore, the incidence of masseter spasm in our operating rooms during that time was 1/800 or 0.12%. However, all incidents of masseter spasm occurred in patients who received halothane for induction of anesthesia followed by intravenously administered succinylcholine. One thousand four-hundred sixty patients (12%) were anesthetized with this specific technique. In this subgroup of patients, the incidence of masseter spasm was 1.03%.

Figure 1 gives the age distribution of patients anesthetized at our institution during the study period. The age distribution of patients who developed masseter spasm ranged from 1.5 to 10.9 years, with a mean 7.1 years. Three were 10 years old, and seven were between

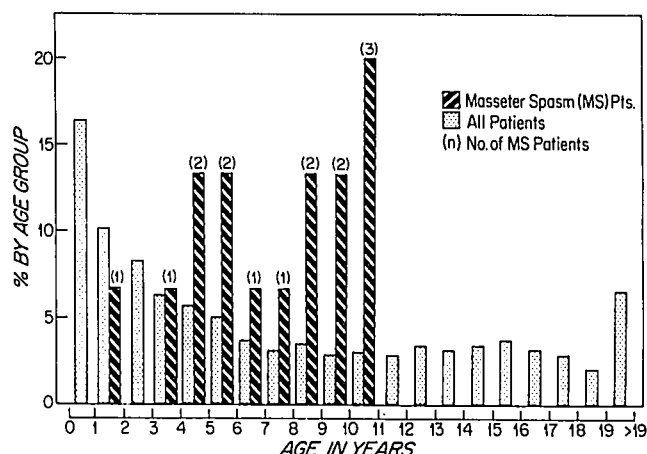


FIG. 1. Age distribution of anesthetized patients (1/82-2/83).

8 and 10 years old. No case of masseter spasm occurred in children under 1 year of age, although 17% (2,069) of all patients anesthetized were in this age group and 263 of them received halothane followed by intravenously administered succinylcholine.

The patients who developed masseter spasm are described in the table 1. None was suspected of being MH susceptible on the basis of the preoperative evaluation, and none had any evidence of neuromuscular disease.

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TABLE 1. Cases of Masseter Spasm

Patient Number	Age (Years)	Operation	Intraop Problems	Intraop Potassium (meq/l)	Intraop pH	Intraop PaCO ₂ (mm Hg)	Intraop Base Excess (mEq/l)	Myoglobinuria	T _{max} (Rectal)	CPK (Within 2 h) (IU)	Highest CPK (Within 24 h) (IU)	Ca ²⁺ Uptake (Muscle Biopsy) (μmol · g ⁻¹ · min ⁻¹)
1	4.4	Dental rehabilitation	Vomiting	4.0	7.31	35	-7	Positive	38.6	86	114	6.1
2	10.9	Caldwell-LUC	None	4.1	7.37	41	-2	NA	37.4	38	NA	2.3
3	1.5	Hypopadiis repair	None	4.5	7.36	44	0	Negative	37.2	433	1217	2.9
4	10.1	Bronchoscopy bronchogram	Bigeminy	4.4	7.29	49	-1	Negative	38.0	492	NA	5.3
5	6.9	Adenoidectomy	None	NA	NA	NA	NA	NA	37.4	NA	NA	6.2
6	9.1	Fracture reduction	None	4.1	7.47	29	-1	Negative	37.8	2970	3500	8.0
7	8.5	Adenoidectomy	Ventricular arrhythmia	3.5	7.31	40	-5	Negative	37.6	85	2673	3.6
8	8.5	Fracture reduction	None	3.5	7.36	39	-3	NA	36.8	135	2000	2.1
9	9.6	Central line placement	Bigeminy	4.9	7.39	39	0	NA	37.6	13	350	4.5
10	5.6	Cholesteatoma excision	None	3.9	7.35	39	-2	Positive	37.6	280	41,900	3.8
11	10.7	Fracture reduction	None	4.0	7.44	34	0	Negative	39.2	496	1490	5.1
12	5.5	Fracture reduction	Ventricular arrhythmia	4.7	7.43	33	-1	Positive	37.2	80	10,500	NA
13	3.1	Endotracheal intubation	Epiglottitis sepsis	3.7	7.30	39	-5	NA	39.8	34	NA	NA (Father positive)
14	7.1	Colostomy closure	None	3.7	7.30	43	-4	NA	37.6	521	1,496	NA
15	4.9	Cholesteatoma excision	None	3.8	7.30	52	0	Negative	37.6	240	9420	4.6

NA = Not available.

Six of the 15 patients had undergone previous anesthetics, four at our institution. All four of these patients had received halothane; two had also received succinylcholine, one before and one after halothane. The anesthetic records did not describe any untoward events, including masseter spasm.

When masseter spasm occurred, it was usually dramatic in presentation but subsided spontaneously within 10–20 min after onset. Two patients were monitored with a transcutaneous nerve stimulator applied to the ulnar nerve and were noted to have full neuromuscular blockade at the time of jaw rigidity.

Four of the children developed ventricular arrhythmias at the time of masseter spasm, and one additional patient vomited while the teeth were clenched shut. All five patients recovered uneventfully as the anesthetic was discontinued. Thirteen cases were aborted, and these patients were given oxygen by face mask until awake. Two brief cases were continued after halothane was withdrawn. Dantrolene was given to only one patient at the discretion of the attending anesthesiologist; that patient did not differ from the others in presentation or outcome. All patients were observed at least 1 day in the intensive care unit.

One child died 8 h postoperatively. She presented to the operating room with upper airway obstruction secondary to epiglottitis requiring endotracheal intubation. Intubation was performed uneventfully following administration of halothane and oxygen, but masseter spasm developed after succinylcholine was given to facilitate replacing the oral tube with a more stable nasal endotracheal tube. Her postanesthetic course did not suggest malignant hyperthermia, and autopsy revealed fulminant *Hemophilus influenzae* sepsis, epiglottitis, and meningitis. All other children recovered without difficulty, although three who had been afebrile preoperatively developed at least one postanesthetic rectal temperature greater than or equal to 38° C within 24 h of their masseter spasm.

Fourteen patients had CPK levels drawn. Postanesthetic CPK levels exceeded normal values (5–50 IU) in 12 of those 14 patients. Although three patients had mild increases (greater than 50 IU), nine had marked elevations in serum levels (greater than 1,000 IU). Two children had levels exceeding 10,000 IU. In all patients in whom multiple CPK values were obtained, the lowest value was always the sample taken in the operating room immediately after the development of masseter spasm. The CPK values obtained immediately after the development of masseter spasm ranged from 13 to 2,970 IU (mean 422 IU). After 12 h, the CPK range was 114–41,900 IU (mean 6,787 IU).

Postoperative urine specimens were positive for myoglobin in three of nine patients tested. Arterial blood gases and serum potassium levels were normal in all

cases. Mild metabolic acidosis was noted in five patients (base excess not exceeding –7 meQ/l) from operating room samples but resolved spontaneously.

Muscle biopsies were obtained from 12 of the 15 patients at the time the previously canceled surgery was performed. No patient experienced perioperative difficulties. Calcium uptake ranged from 2.1–8.0 $\mu\text{mol} \cdot \text{g}^{-1} \cdot \text{min}^{-1}$ (mean = 4.5). Normal values for the testing laboratory are 8.5–16.0 $\mu\text{mol} \cdot \text{g}^{-1} \cdot \text{min}^{-1}$.⁹

DISCUSSION

Masseter spasm was a much more common occurrence in our patient population than previously suspected, developing during approximately 1 of 800 anesthetics administered. Despite the wide variety of anesthetic techniques used, masseter spasm was noted to occur only in patients whose anesthesia was induced with halothane followed by intravenously administered succinylcholine. Twelve per cent of our total patient population was anesthetized in this manner, giving an incidence of masseter spasm with that technique of 1%. Inhalation inductions were performed so infrequently with enflurane or isoflurane that a comparison with halothane cannot be made.

Forty-six per cent of the patients with masseter spasm were between 8 and 10 years of age, despite the fact that only 11% of all patients were in this age group. No cases of masseter spasm were identified in patients less than 1 year of age, despite the fact that 263 infants were anesthetized with halothane followed by succinylcholine. However, because our numbers are small, we had only an 8% chance of identifying even one case of masseter spasm among these youngest patients.

In the operating room, patients with masseter spasm clearly were identified. By accepting into this series only patients who had received at least 1 mg/kg of succinylcholine intravenously, it is unlikely that difficulty in opening the patients mouth was due to administration of an inadequate amount of muscle relaxant. If anything, this study underestimates the incidence of masseter spasm, since additional cases may have occurred and were not apparent on our review of medical records.

Although myoglobinuria and elevated CPK levels have been observed after uneventful anesthetics with halothane and succinylcholine,^{11,12} the values seen in some of our patients were distinctly abnormal. Certainly, CPK values in the tens of thousands rarely occur in the absence of significant muscle pathology. Those patients who developed only mild elevations (<1,000) in the postanesthetic period were clinically indistinguishable from the two with dramatic elevations (>10,000). The initial CPK value obtained in the operating room immediately after the occurrence of masseter spasm was the lowest recorded for any individual patient and was most often unremarkable. Only subsequent sampling at

least 4 h after anesthesia revealed the marked increases that often occurred.

By the calcium uptake assay, all 12 (100%) of our patients tested were found to be MH susceptible. Other investigators also have documented the association of masseter spasm with susceptibility to malignant hyperthermia by using caffeine and halothane contracture testing. Flewellen *et al.*⁴ found four of six patients who had developed masseter spasm after receiving halothane followed by succinylcholine to be MH susceptible. Rosenberg *et al.*¹³ reported nine of 13 patients with a similar history to also have positive contracture results. Larach and Rosenberg¹⁴ reviewed the indications for muscle biopsy and the results of caffeine and halothane contracture testing in 39 children who has signs of MH susceptibility. Of the 23 whose history included masseter muscle rigidity, 50% were MH positive.

The precision of the available tests for MH susceptibility remains controversial. In particular, the calcium uptake assay has been questioned.¹⁵ Regardless of the varying proportions of MH positivity from separate centers using different assays, the fact remains that by all published reports, a majority of patients with masseter spasm have muscle biopsies consistent with the diagnosis of susceptibility to malignant hyperthermia.

If these patients have what generally is considered to be malignant hyperthermia, the incidence of susceptibility in the general population is approximately 100 times previous estimates (1/100 not 1/10,000).¹⁶ More likely, these children have a muscle abnormality similar or related to MH that is brought out by the administration of succinylcholine in the presence of halothane. Certainly, further studies need to be done to more closely examine this group of individuals.

It is not known whether a full-blown MH crisis would have developed in these patients if surgery and anesthesia have been continued. The results of arterial blood gases, serum CPK levels, or urinalysis obtained in the operating room at the time of masseter spasm do not seem to offer a reliable basis for distinguishing which patients might manifest other signs of a MH episode with further anesthesia. It does appear prudent to abort any elective procedures when masseter spasm occurs.¹⁷ If emergency surgery is necessary, discontinuing the use of all potential triggering agents is mandatory. The administration of dantrolene should be considered strongly when surgery cannot be postponed. Although our numbers are small, dantrolene does not appear necessary if anesthesia can be promptly terminated. It also appears that pretreatment with dantrolene during subsequent anesthesia is unnecessary for this group of patients, provided that potential MH triggering agents are carefully avoided.

In summary, masseter spasm during anesthesia is a common event occurring in 1/100 children who are anesthetized with halothane followed by intravenous

succinylcholine. Some of these patients are prone to ventricular arrhythmias, myoglobinuria, and impressive increases in CPK levels. A skeletal muscle abnormality can be detected in the majority by appropriate assays performed on muscle biopsy specimens, and this appears to be related to that found in patients with malignant hyperthermia. These children seem to do well if anesthesia is terminated promptly. Until more is known about this condition, masseter spasm after the induction of anesthesia cannot be dismissed as a benign event.

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