MASSETER SPASM INDUCED BY SUCCINYLCHOLINE IN CHILDREN: CONTRACTURE TESTING FOR MALIGNANT HYPERTHERMIA: REPORT OF SIX CASES

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ABSTRACT

We evaluated six boys who had developed isolated masseter muscle spasm following intravenous succinylcholine. All were receiving halothane by inhalation. *In vitro* muscle contracture tests utilizing halothane and caffeine were performed. Four of the six boys had contracture responses similar to those of malignant hyperthermia susceptible patients. Rigidity following succinylcholine should prompt the clinician to consider malignant hyperthermia but has been associated with other myopathic conditions as discussed.

KEY WORDS: Malignant hyperthermia; Succinylcholine, Masseter spasm.

MASSETER SPASM after administration of succinylcholine is an atypical response, but is not uncommon, especially among younger patients. This response may be followed by an acute episode of malignant hyperthermia (MH).[1–5] Among many of the reported patients with succinylcholine-induced masseter spasm the identification of MH susceptibility (MHS) has been equivocal.[4,6–13] Muscle contracture testing *in vitro* remains the best proven test for MHS.[14] In only two of these reports did patients have contracture testing done after the atypical response to succinylcholine.[7,13] Interpretation of this atypical response is important, not only for the immediate diagnosis of an impending MH episode, but also for the future implications of the diagnosis to the patient and his family.

In the present report we have done muscle contracture tests on six male children who developed atypical masseter spasm following the administration of succinylcholine with halothane anesthesia. These six children represent 11 per cent of all patients referred to our research center for evaluation of MH susceptibility.

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CASE REPORTS

Patient 1.

A three year old Latin American male weighing 13.5 kg was scheduled for diagnostic laryngoscopy because of persistent hoarseness. Premedication included intramuscular secobarbitone 40 mg and atropine 0.2 mg. Anaesthesia was induced by inhalation of halothane and nitrous oxide. Succinylcholine 15 mg was given intravenously to facilitate orotracheal intubation. Apnoea rapidly ensued and fasciculations were reported to be normal. His mouth could not be opened but the limbs were flaccid. A drip infusion of succinylcholine 0.2 per cent failed to alleviate the masseter spasm, while peripheral ulnar nerve stimulation revealed absence of evoked twitch. Heart rate was initially 105 bpm and increased to 130 bpm during attempts to open the mouth. No arrhythmia was detected on electrocardiographic monitoring. Rectal temperature was initially 37.3° C and fell to 37° C. The proposed procedure was aborted. Analysis of arterial blood obtained in the recovery room revealed $[H^+]a$ 39.81 nmol/l (pH 7.40); Pa_{CO_2} 36 torr (4.8 kPa); Pa_{O_2} 135 torr (18 kPa), BE-2 (face tent oxygen) and potassium 4.0 mmol/l. Venous creatine phosphokinase (CPK) drawn in the Recovery Room was 625 IU/l and increased to 1938 IU/l (normal 36–188) the following day. Urinalysis yielded no myoglobin. The patient complained of leg soreness for several days afterwards.

Patient 2.

A 10 year old black male weighing 29 kg was scheduled for repair of ptosis of the left eyelid. At the age of 8 years the child had repair of lacerated tendons under halothane-succinylcholine anaesthesia without apparent difficulty. Premedication included intravenous meperidine 30 mg, and intramuscular promethazine 15 mg and atropine 0.2 mg. Anaesthesia was induced by inhalation of halothane and nitrous oxide. Succinylcholine 30 mg and thiopentone 100 mg were given intravenously to facilitate intubation, but the

mouth could not be opened. A repeat intravenous injection of succinylcholine 20 mg and thiopentone 100 mg did not alleviate the masseter spasm. The limbs were flaccid. Initial heart rate was 100 bpm and blood pressure was 120 torr (16 kPa) systolic which increased to 130 bpm and 140 torr (18.6 kPa) respectively during intubation attempts. Rectal temperature remained 37° C. The proposed procedure was aborted. Analysis of arterial blood obtained in the operating room revealed: $[H^+]$a 39.81 nmol/l (pH 7.40); Pa_{CO_2} 42 torr (5.6 kPa); Pa_{O_2}, 519 torr (69 kPa) and potassium 4.2 mmol/l. Venous blood CPK was 1072 (normal 25–125 IU/l) which increased to 8400 the following day. The patient had an uneventful recovery.

Patient 3.

A six year old white male weighing 20.4 kg was scheduled for repair of esotropia. The patient was taking methylphenidate for hyperactivity. Premedication included intramuscular pentobarbitone 50 mg and glycopyrolate 15 mg. Anaesthesia was induced by inhalation of halothane and the trachea was intubated without difficulty. A large gas leak was noted around the orotracheal tube and it was removed so that a larger one could be inserted. Laryngospasm developed and succinylcholine 40 mg was administered intravenously. His mouth could not be opened. Limbs were flaccid. Initial heart rate was 110 bpm and blood pressure was 100 torr (13.3 kPa) systolic and these increased to 180 bpm and 130 torr (17.3 kPa) during a second intubation attempt. Rectal temperature was 36.9° C. The procedure was then aborted. Analysis of arterial blood revealed $[H^+]$a 33.88 nmol/l (pH 7.47); Pa_{CO_2} 26 torr (3.5 kPa); Pa_{O_2} 129 torr (17.2 kPa) (face tent oxygen) in the recovery room). Serum CPK was measured at 9702 (normal 50–180 IU/l) the next day and 3624 the second post-anaesthetic day. The patient recovered without difficulty.

Patient 4.

A two year old black male weighing 13.4 kg was scheduled for dental restorations. The patient had a congenital facial nerve palsy. Premedication included meperidine 10 mg, secobarbitone 35 mg, and atropine 0.2 mg, given intramuscularly. Anaesthesia was induced by inhalation of halothane and nitrous oxide. Succinylcholine 20 mg was then administered intravenously. Orotracheal intubation was attempted but the mouth could not be opened. Limbs were flaccid. Heart rate was initially 150 bpm and increased to 175 bpm. Intravenous succinylcholine 10 mg did not abolish the masseter spasm. Halothane and nitrous oxide were discontinued and the patient was hyperventilated with 100 per cent oxygen. After his heart rate decreased to 130 bpm, three per cent halothane was again administered. During a second attempt at orotracheal intubation, stiff jaw muscles and poorly relaxed vocal cords were observed and the heart rate increased to 160 bpm. Rectal temperature was 36.1° C. The procedure was then aborted. The patient recovered without complication.

Patient 5.

A five year old white male weighing 19.5 kg was scheduled for repair of esotropia. The patient had previously undergone halothane-succinylcholine anaesthesia without apparent difficulty. Premedication included intramuscular morphine 2 mg, pentobarbitone 30 mg and atropine 0.2 mg. Anaesthesia was induced by inhalation of halothane and nitrous oxide. Orotracheal intubation was accomplished with difficulty but a large gas leak was detected around the tube. He was extubated in preparation for placement of a larger tracheal tube. Laryngospasm developed and intravenous succinylcholine 30 mg and atropine 0.2 mg were administered. The mouth could not be opened. After a repeat dose of succinylcholine 30 mg jaw rigidity persisted and ventricular arrhythmia was noted. Limbs were flaccid. Cyanosis was observed in the head and neck. Rectal temperature varied between 37.2° and 37.3° C. The procedure was aborted. Serum CPK was 380 IU/l that day and 2 IU/l the following day (normal 4–43 IU/l). The child recovered without difficulty. No myoglobin was detected in the urine on the day of anaesthesia or the day after.

Patient 6.

A four year old white male weighing 17.3 kg was scheduled for tonsillectomy. Premedication included intramuscular meperidine 20 mg and atropine 0.2 mg. Anaesthesia was induced by thiopentone 75 mg intravenously and inhalation of halothane and nitrous oxide. Succinylcholine 30 mg and atropine 0.2 mg were administered intravenously. Limb fasciculations were noted to be normal. The mouth could not be opened and additional succinylcholine 40 mg was administered. The masseter muscle remained rigid. Limbs were flaccid. Heart rate and blood pressure remained unchanged and no arrhythmia was detected. Rectal temperature was 37.1° C. The procedure was aborted. The child recovered without difficulty. Clinical myoglobinuria and post-anaesthesia muscle pain were absent.

Methods

All patients were admitted to the Clinical Research Center of the University of Texas Medical Branch one day before operation for muscle biopsy. Detailed family and personal history was obtained and a complete physical examination was done. Blood samples were obtained for CPK estimations before and one day after muscle biopsy. Each patient had general anaesthesia for biopsy of the vastus lateralis muscle for MH contracture testing *in vitro* as previously described.[4] Premedication consisted of intramuscular diazepam and morphine. Anaesthesia was induced by the intravenous administration of diazepam, fentanyl and thiopentone over a 5–10 minute period and then inhalation of nitrous oxide 65 per cent in oxygen was begun by face mask.

Two specimens of vastus laterialis were dissected *in situ* and clamped at resting length. Each specimen was dissected to yield six to eight fascicles for contracture testing. Specimens

were kept in Krebs-Ringer solution at 37°C while the bath solution was being bubbled with carbogen. Individual fascicles were stimulated directly with supramaximal voltage, duration 1 msec, at a frequency of 0.2 Hz. Optimal length-twitch tension was achieved before testing. Each fascicle was subjected to one of three treatments: three per cent halothane in carbogen; cumulative additions of caffeine, 0.5 to 16 mM; or cumulative addition of caffeine 0.25 to 8 mM in one per cent halothane in carbogen. A Brush recorder and Statham FT 0.03 force transducer were used to record changes in twitch tension and contracture responses.

Contracture response to three per cent halothane was measured in at least two fascicles as the increase in grams of resting tension. The caffeine concentration (mM) required to produce a 1 g isometric contracture in the absence of halothane (CSC-1 g), and in the presence of halothane one per cent (HCSC-1 g) was determined on a minimum of two fascicles from each subject. Contracture testing was also done on the mother and brother of Patient 1, the mothers of Patients 2 and 3, and the father of Patient 5. The diagnostic protocol was approved by the Human Investigation Committee of The University of Texas Medical Branch and informed consent was obtained for each patient.

Results

Clinical findings

All six families reported members who had undergone general anaesthesia without apparent difficulty. Three of the boys (Patients 1, 2, and 3, each subsequently diagnosed as MH susceptible) had experienced cramping in the calf muscles, which occurred typically only at night after the child had fallen asleep (Table I). These episodes were of 5 to 20 minutes in duration and were relieved by massage or a warm bath. They occurred variously from once a week to once a month and tended to occur less often as the child grew older. Episodes could not be well correlated with increased muscular activity that day. The mother of Patient 3 reported similar calf pain beginning at the age of 20 (at this time she was 26).

All patients were normal in appearance except those with abnormality of the eye muscle. None exhibited skeletal abnormality. Findings on neurological examinations were all normal, as measured by muscular power and coordination, balance, cranial nerve function (except that Patient 4 had VII nerve palsy), sensation and tendon reflexes. All had normal muscle bulk. No electromyographic tests were done.

Only Patient 2 had an elevated CPK determination before muscle biopsy (Table I). Only Patient 3 did not have an elevated CPK determination the day after biopsy (Table I).

Contracture testing

Patient 1 was determined to be MH susceptible, based on abnormal contracture responses to three per cent halothane, to caffeine, and to caffeine in the presence of halothane one per cent (Table II). The brother of Patient 1 also had abnormal responses to all three tests and is considered MH susceptible. The mother of Patient 1 had normal responses for all tests. Patient 1 had red-tinged serum in the blood sample taken postoperatively for CPK determination and this was shown to be positive for myoglobin.

Patients 2, 3, and 4 had normal muscle contracture responses to three per cent halothane and caffeine (Table II). The contracture responses of their muscles to caffeine in the presence of halothane one per cent were abnormal, however, and fell within the range of responses for patients who have been established as MH susceptible (Table II). The mother of Patient 3 had an abnormal combined caffeine-halothane response and is considered MH susceptible. The mother of Patient 2 had normal contracture responses.

Patients 5 and 6 had normal responses to all three tests (Table II) and are considered MH resistant (MHR) (Table II). The father of Patient 5 was determined to be MH resistant by contracture testing.

Discussion

We consider four of these six children to be MH susceptible based on the responses to contracture testing *in vitro*. All were receiving halothane by inhalation when intravenous administration of succinylcholine produced isolated masseter spasm. Anaesthesia was then discontinued and no morbidity resulted.

The contracture responses of skeletal muscle to caffeine and/or halothane has become the best diagnostic test for MH susceptibility in man. This method has proven accurate and appears to be specific for MH susceptibility.[14,15] Three contracture tests are performed *in vitro* in which strips of skeletal muscle are exposed to halo-

TABLE I

DATA FOR SIX MALE CHILDREN WHO EXHIBITED SUCCINYLCHOLINE-INDUCED MASSETER SPASM

Patient No.	Age when spasm occurred (yr)	Age at contracture testing (yr)	Prebiopsy CPK (IU/litre)	Postbiopsy CPK (IU/litre)	Normal CPK (IU/litre)	Leg cramps	Previous halothane/ succinylcholine
1 (MHS)*	3	3	105	1036	36–188	yes	no
2 (MHS)	10	11	119	197	0–75	yes	yes
3 (MHS)	6	7	63	184	36–188	yes	no
4 (MHS)	2	3	79	579	36–188	no	no
5 (MHR)†	5	10	34	147	0–75	no	yes
6 (MHR)	4	5	54	418	36–188	no	no

*MHS = Malignant hyperthermia susceptible by contracture testing.
†MHR = Malignant hyperthermia resistant by contracture testing.

TABLE II

CONTRACTURE RESPONSE TO HALOTHANE AND CAFFEINE IN SIX MALE CHILDREN WHO EXHIBITED SUCCINYLCHOLINE-INDUCED MASSETER SPASM

Patient No.	Considered MHS	Contracture on Halothane 3% (g)	CSC-1g*	HSCS-1g†
1	yes	2.2 4.7	2.6	0.30
2	yes	0.3 0.3	4.3	0.64
3	yes	0.2 0.0	3.6	0.59
4	yes	0.0 0.0	3.0	0.44
5	no	0.1 0.2	4.6	1.00
6	no	0.0 0.0 0.3	4.8	1.04
TYPE H‡ n = 15	yes	3.2 ± 1.7 (1.1 − 5.8)	1.7 ± 0.9 (0.4 − 3.3)	0.34 ± 0.2 (0.17 − 0.65)
CONTROL‡ n = 14	no	0.1 ± 0.2 (0.0 − 0.8)	4.1 ± 1.9 (2.1 − 9.7)	0.93 ± 0.6 (0.38 − 2.3)

*CSC-1g = caffeine specific concentration, mM, required to produce 1g of contracture.

†HCSC-1g = caffeine specific concentration, mM, required to produce 1g of contracture in the presence of halothane, 1 per cent.

‡Type H = abnormal response to each of three contracture tests (see text).

For TYPE H and CONTROL, values are mean ± SD and range is in parenthesis.

thane three per cent; caffeine dose response; or caffeine dose response in the presence of halothane one per cent. Our experience is that muscle which has abnormal contracture response to three per cent halothane will have an abnormal caffeine response, with and without halothane. These individuals we call Type H, and an abnormal contracture response to halothane is considered pathognomonic for MH susceptibility. The response of Type H muscle to caffeine plus halothane is then compared to the control population and to MH diagnostic patients. A second MH diagnostic classification has emerged — Type K. The Type K patients have normal response to halothane alone or to caffeine alone, but have a response to combined halothane and caffeine that is similar to Type H patients. We have observed a Type H patient producing a Type K child, and two Type K parents producing a Type H child.

We are confident about the diagnosis of MH susceptibility in Patient 1 (Type H response). The diagnosis of MH susceptibility in Patients 2, 3, and 4 is more equivocal (Type K). Each of these boys had a HCSC-1 g which was within the range of previously diagnosed Type H individuals (Table II). We have observed overlap in HCSC-1 g responses between the Type H and control population (Table II). To date we have not observed a Type K response in a survivor of a clinical MH episode; all survivors have been Type H. Britt has observed a Type K contracture response in patients surviving clinical MH episodes.* Rosenberg, *et al.* did contracture tests on ten patients who exhibited masseter spasm without limb rigidity following succinylcholine.[13] Six had abnormal contracture response to halothane or caffeine alone (Type H response). The Type K response was not determined. Thus, patients 2, 3, and 4 are likely MH susceptible, but this diagnosis is less conclusive than in Patient 1. We consider them to be MH susceptible, however, until further diagnostic experience is achieved. Labeling a person as MH susceptible is not to be done lightly, as this diagnosis has profound implications for the future medical care of the patients and their

*Personal Communication: B.A. Britt, Dept. of Anaesthesia, University of Toronto, Toronto, Ontario, Canada.

relatives. The potential morbidity and mortality from MH, however, obliges conservatism. Other evidence supporting a conservative approach to the diagnosis of MH susceptibility is an elevation of serum CPK in Patient 2 and a history of leg muscle cramping in Patients 2 and 3. We have observed elevated CPK determinations in one third of Type H and seven per cent of type K patients examined in our laboratory. Leg muscle cramping is a common complaint of both Type H and K patients in our experience. The pain is often experienced only at night and awakens the patient. The cramping occurs intermittently, from several episodes a month to one episode every few months. Episodes occur most frequently in early childhood, decrease with age, and disappear by the age of fifty.

Patients 5 and 6 are not considered to be MH susceptible. The masseter spasm that occurred is unexplained. We counsel such patients to avoid succinylcholine. Cody[17] and Relton, *et al.*[18] have reported anaesthetized children in whom intravenous administration of succinylcholine has induced masseter spasm and limb rigidity, without the development of clinical MH. Barnes[12] has reported a similar case in a woman with chronic elevation of serum CPK.

The observation that masseter spasm following intravenous administration of succinylcholine, with or without associated limb muscle rigidity, can precede a clinical episode of MH has been well documented.[1–5,7] Britt and Kalow reported that 31 of 75 MH episodes associated with muscle rigidity occurred after administration of succinylcholine had caused a difficult tracheal intubation or an unsuccessful attempt at intubation.[1] The clinician should be aware that muscle rigidity may never be observed during an episode of MH and that this finding is not necessary to a diagnosis of MH.[19]

In addition to MH, succinylcholine-induced masseter spasm or skeletal muscle rigidity has been associated with several other skeletal muscle abnormalities: denervation, myotonia, polymyositis, and rhabdomyolysis. Brimm[20] and Orndahl[21] have reported the development of arm rigidity following intravenous administration of succinylcholine in patients with denervating brachial plexus injury or amyotrophic lateral sclerosis. We are unaware of any reports of MH in man associated with chronic denervation of traumatic origin.

Muscle rigidity following administration of succinylcholine in patients with underlying myotonia congenita (MC) or myotonia dystrophica has been documented.[22–24] We are unaware of myotinia dystrophica associated with MH in man. An association of myotonia congenita with human MH has been claimed,[26–28] but we feel this may be unwarranted. Saidman, *et al.*[26] reported a case but no substantiating evidence for the diagnosis of myotonia congenita was given. Morley, *et al.*,[26] Moulds and Denborough[15] and King, *et al.*[27] have reported on the same male patient with myotonia congenita. The patient was referred to Moulds who did halothane-caffeine contracture testing, which was negative for MH, while Morley reported that these tests were positive for MH. In view of the inadequate documentation in Saidman's report and the inconsistency of the interpretation of the contracture tests in Morley's patient, we conclude that the association of myotonia congenita with MH is equivocal.

Davies[28] reported the case of a woman with a history of generalized weakness and tenderness that was diagnosed as polymyositis. Administration of succinylcholine induced generalized contraction of skeletal muscle. Halothane was administered for an additional 40 minutes, during which tachycardia and hot flushed skin were observed. The patient recovered without complication but died suddenly four days later.

Massive rhabdomyolysis following intravenous administration of succinylcholine during halothane, enflurance, or methoxyflurane anaesthesia has been documented in 11 patients.[8–11,29–33] Muscle disruption produced myoglobinuria in all patients. None developed hyperthermia. Ten of the eleven patients were children under the age of 15 years. Nine of the children exhibited masseter spasm; five were isolated and four associated with a generalized increase in muscle tone. Auerback, *et al.*,[10] and Bernhardt and Hoerder[11] contended that rhabdomyolysis and MH are associated. Rhabdomyolysis and myoglobinuria commonly result from an episode of clinical MH in man.

In conclusion, masseter spasm as an atypical response to administration of succinylcholine may be associated with clinical MH, yet it is not always indicative of MH. Certain recognized myopathic processes may predispose a patient to develop muscle rigidity following administration of succinyl-choline. The observation of rigidity following succinylcholine should prompt the clinician to consider MH as a distinct possibility. Hazardous anaesthetic agents should be discontinued immediately and efforts undertaken to determine an underlying myopathy.

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RÉSUMÉ

Six jeunes garçons qui avaient présenté un spasme n'impliquant que le masséter à la suite d'une injection de succinylcholine par la voie veineuse ont été étudiés. Tous ces malades recevaient de l'halothane. Des tests de contracture à l'halothane et à la caféine in vitro ont été effectués et ont démontré dans quatre cas sur six des contractures identiques à celles qu'on rencontre chez le sujet susceptible à l'hyperthermie maligne. La rigidité qui suit l'injection de succinylcholine devrait toujours faire suspecter l'hyperthermie maligne mais ne peut éliminer d'emblée d'autres myopathies.