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Masseter Muscle Rigidity after Rapid-sequence Induction of Anesthesia

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Masseter muscle rigidity (MMR) has been reported to occur with a frequency of 1% or higher after halothane induction and subsequent succinylcholine administration.^{1,2} Recently Lazzell *et al.* have reported that administration of succinylcholine without concurrent administration of a halogenated anesthetic is not associated with MMR.³ Although references to MMR after intravenous inductions have been alluded to in the anesthesia literature, no such cases have been described in detail.⁴ We report such a case.

CASE REPORT

An 8-yr-old, 22-kg child was brought to the operating room for emergency repair of a laceration of his right upper eyelid under general anesthesia.

His medical history, physical examination, and laboratory data were unremarkable except for an increased white blood cell count of 17,800 white blood cells · dl⁻¹. Prior surgical history consisted of a tonsillectomy/adenoidectomy at the age of 5 yr without anesthetic complications, and his mother denied any family history of anesthetic complications. He had been fasting since 8 PM the prior night.

One milligram of midazolam was given intravenously as premedication. In the operating room, monitoring included noninvasive blood pressure, electrocardiogram, temperature, and hemoglobin O₂ saturation (SpO₂). After preoxygenation, anesthesia was induced with thio-pental 100 mg followed by succinylcholine 40 mg intravenously. Upon injection of the succinylcholine, the heart rate increased from 100 to 140 beats/min. Intubation was attempted after 45 s but the mouth could not be opened. Because it was believed that the dose of succinylcholine was adequate (1.8 mg · kg⁻¹), an additional 45 s was allowed to elapse, but again the jaw was found to be too tightly closed to open, even though peripheral stimulation of the ulnar nerve demonstrated complete paralysis. While cricoid pressure was maintained, the lungs

were easily ventilated and the patient did not exhibit peripheral rigidity. The jaw remained rigidly closed for approximately 15 min, during which time 100% O₂ and 5 μg · kg⁻¹ fentanyl was administered. During this time, the SpO₂ remained 100%; the end-tidal CO₂ was 34–44 mmHg; the heart rate returned to 120 beats/min; blood pressure was 100/55–110/60 mmHg; and the skin temperature measured by liquid crystal probe was 37.2° C. Because of the stability of the vital signs, end-tidal CO₂, and SpO₂, and because of the severity of the injury, we elected to proceed with the anesthesia and surgery. Atracurium was used to facilitate the tracheal intubation, and anesthesia was maintained with fentanyl, 70:30 N₂O:O₂, and atracurium.

An arterial blood gas drawn immediately after intubation (about 20 min after succinylcholine administration) showed a metabolic acidosis: pH 7.19, PO₂ 136 mmHg, PCO₂ 44 mmHg, base excess -10.2 mM, and SpO₂ 98%. A second arterial blood gas drawn 15 min later (35 min after induction), after a bolus of lactated Ringer's solution had been administered, was normal except for a base excess -2.6. Urine analysis showed a specific gravity of 1.028, ketones of 160 units (normal = 0), 0-1 red blood cells, and negative myoglobin. Other abnormal laboratory values included a serum sodium of 134 mEq · l⁻¹ (normal 136–143 mEq · l⁻¹), glucose of 164 mg · dl⁻¹ (70–100 mg · dl⁻¹), calcium of 8.4 mg · dl⁻¹ (9.1–10.6 mg · dl⁻¹), a serum glutamic-oxaloacetic transaminase of 58 IU (10–50 IU), and a creatine kinase (CK) of 3184 IU (57–374 IU). Lactated Ringer's solution was administered to maintain the urinary output at greater than 2 ml · kg⁻¹ · h⁻¹.

The case proceeded without event. The esophageal temperature remained stable at 37.2–37.9° C. The patient's blood pressure remained stable, and the heart rate returned to baseline. A serum myoglobin drawn immediately after intubation was returned at 628 ng · dl⁻¹ (normal = 0–55). The muscle relaxation was antagonized with glycopyrrolate and neostigmine at the termination of the case, and the trachea was extubated without difficulty. The patient was taken to the recovery room in stable condition, where he was monitored for approximately 90 min, during which time his vital signs remained stable; his oral temperature was 37.7° C; and SpO₂ while breathing 40% O₂ via face shield was 99%. He was transferred to the pediatric ward in good condition and with no intraoperative recall.

Admission tympanic membrane temperature on the ward was 37.5° C. The patient's temperature was measured hourly for the first 24 h, and at 7 h after induction, his temperature had slowly increased to 39.1° C, despite the administration of acetaminophen elixir. An arterial blood gas drawn at that time, while he was breathing room air, showed a pH 7.4, PO₂ 77 mmHg, PCO₂ 36 mmHg, base excess -0.8 mM, and SpO₂ 96%. The lungs were clear, and the chest x-ray was normal. The patient's temperature peaked 10 h after induction at 39.2° C and then decreased through the night to 37.2° C at 4 AM.

Urine myoglobin remained negative. The CK was 17,740 IU on the first postoperative day and 1,675 IU on discharge the second postoperative day.

The day after surgery, the patient's mother offered additional family history obtained from the child's maternal grandmother. Two of the child's maternal uncles had had life-threatening events as children (believed to be respiratory in nature) associated with anesthesia and requiring termination of surgery in both cases. Further details were not available from the grandmother. The child's mother herself had had a high fever after general anesthesia for cesarean section, which at the

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time was attributed to a medication effect. There were no family deaths associated with anesthesia.

The patient continued to do well and was discharged on the second postoperative day. Arrangements were made for him and his family to be evaluated at our MH Referral Center at the Uniformed Services University of the Health Sciences. The recommendation of the referral facility at the time of this occurrence was to defer muscle biopsy until the child was 10 yr old.

DISCUSSION

We present this case because of the unexpected occurrence of MMR after a rapid-sequence induction. Recent reports in the anesthesia literature indicate that MMR is extremely rare after induction with thiopental followed by succinylcholine.^{3,5} The presence of thiopental has been believed to exert a protective effect,⁶ and the report by Lazzell *et al.*³ supports this concept.

Criteria for defining masseter spasm has been the subject of much discussion. Van der Spek *et al.* have demonstrated that animals as well as children will exhibit a normal decrease in mouth opening and an increase in jaw stiffness when given succinylcholine after exposure to halogenated anesthetics,⁷⁻⁹ and Leary and Ellis have demonstrated this effect in adults.¹⁰ Inadequate dosage of succinylcholine has also been attributed to false interpretations of MMR.¹¹ Goudsouzian has suggested that because children have a larger volume of distribution than adults, the dosage should be at least $2.0 \text{ mg}^{-1} \cdot \text{kg}^{-1}$ in infants and $1.0 \text{ mg}^{-1} \cdot \text{kg}^{-1}$ in older children.¹² More recently, Meakin has suggested that these dosages are not sufficient and that up to $2.0 \text{ mg}^{-1} \cdot \text{kg}^{-1}$ in children and $3-4 \text{ mg}^{-1} \cdot \text{kg}^{-1}$ in infants may be required to achieve adequate relaxation.¹¹ We felt certain that our patient was experiencing MMR and not simply a normal phenomenon or an inadequate succinylcholine dosage because the dose of succinylcholine administered was $1.8 \text{ mg}^{-1} \cdot \text{kg}^{-1}$, and still his jaw remained tightly closed for at least 15 min, during which time stimulation of the ulnar nerve demonstrated complete paralysis.

Some authors have advocated continuing the anesthetic after occurrence of MMR during emergency surgery with nontriggering agents,¹³ and more recently some authors have reported continuing halogenated anesthetics after presumed MMR without generalized rigidity and saw no adverse outcome.^{14,15} Because of the association of MMR with malignant hyperthermia¹⁶ and the potential difficulty distinguishing the normal jaw tightness from abnormal masseter muscle spasm, the decision to proceed with the anesthetic (and which anesthetic) is not always obvious. Given the stability of our patient's vital signs and our ability to monitor end-tidal CO_2 levels, we elected to continue the emergency surgery but did so with agents not associated with triggering malignant hyperthermia. Our patient's initial metabolic acidosis was troublesome. How-

ever, coupled with the high urine ketones and his *non per os* status and with the rapid resolution of the acidosis with hydration, it was believed that this was not an indication that a malignant hyperthermia episode was occurring.

Postoperatively, electrolyte abnormalities, elevated CK values, myoglobinuria, and fever can be seen after an episode of MMR. Our patient experienced a fever, and because it is well known that malignant hyperthermia may not occur immediately after MMR, we were concerned that a delayed episode of malignant hyperthermia might be occurring. Vital signs, chest x-ray, and laboratory studies, including an arterial blood gas analysis, were normal except for an elevated CK value and except for a decreased PaO_2 , which could be accounted for by his febrile state and increased metabolic demands as well as postoperative ventilation-perfusion abnormalities.

The postoperative CK values greater than 17,000 IU suggest that this child may well be malignant hyperthermia-susceptible. The series by Rosenberg and Fletcher showed that, in the absence of muscle disease, when the CK value after MMR was greater than 20,000 IU, these children were found to be malignant hyperthermia-susceptible by muscle biopsy subjected to halothane-caffeine contracture testing.¹⁶ Furthermore, a large percentage of the patients with CK values greater than 15,000 IU were also found to be malignant hyperthermia-susceptible in response to contracture testing.¹⁶

In summary, we present a case of MMR after intravenous induction of anesthesia. We believe that although recent data seem to indicate that the incidence of MMR following induction of anesthesia with thiopental and succinylcholine is extremely small, the potential for development of MMR after rapid-sequence induction with these agents still exists. Furthermore, because intravenous induction in children is used in many institutions primarily for emergency conditions, an episode of MMR may occur when it would be least optimal to cancel surgery.

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Intraoperative Awareness with Propofol-Oxygen Total Intravenous Anesthesia for Microlaryngeal Surgery

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Microlaryngeal surgery represents a dynamic clinical challenge for both the otolaryngologist and the anesthesiologist. The need to manage the airway cooperatively has led to the development of innovative anesthetic techniques using jet venturi ventilation and total intravenous (iv) anesthesia (TIVA).¹ Propofol (2,6-diisopropylphenol) is an iv anesthetic with well-described characteristics that make it particularly amenable for use in such cases.^{2,‡} Recently, however, we encountered a case of intraoperative awareness during TIVA with jet venturi ventilation for laser laryngoscopy using propofol as the sole anesthetic agent.

CASE REPORT

A 46-yr-old, 72-kg man in otherwise good health presented for outpatient carbon dioxide laser treatment of recurrent vocal cord papillomas. He had no drug allergies, took no medication, used alcohol socially, and had previously undergone many identical procedures using a TIVA (methohexital and succinylcholine infusions)-jet venturi ventilation technique without complications. As was his habit, the patient

requested an anesthetic compatible with rapid recovery and early discharge. He also refused preanesthetic medication.

In the operating room, iv access and routine anesthetic monitoring were established. Initial blood pressure was 137/80 mmHg, and heart rate was 82 beats/min. After administration of glycopyrrolate 0.2 mg iv, induction proceeded with 150 mg ($2.1 \text{ mg} \cdot \text{kg}^{-1}$) propofol and 120 mg succinylcholine followed immediately by a propofol infusion *via* infusion pump at $200 \mu\text{g} \cdot \text{kg}^{-1} \cdot \text{min}^{-1}$. Hemodynamics after induction remained stable at preinduction levels. Suspension of the larynx and initiation of oxygen-driven proximal jet venturi ventilation as described by Koufman *et al.*¹ was accompanied by hypertension (200/105 mmHg) and tachycardia (122 beats/min), which quickly returned to baseline after a single 5-mg dose of labetalol iv. Anesthetic maintenance consisted of propofol infusion continued at $200 \mu\text{g} \cdot \text{kg}^{-1} \cdot \text{min}^{-1}$ combined with neuromuscular paralysis using a succinylcholine infusion and titrated to 0/4 twitches by train-of-four stimulation of the ulnar nerve. The remainder of the intraoperative course was uneventful. Case duration was 25 min, during which the patient received a cumulative (induction plus maintenance) propofol dose of 450 mg. Emergence from anesthesia was rapid and smooth without hemodynamic perturbations or respiratory compromise.

Upon entering the postanesthesia care unit, the patient spontaneously exclaimed, "I remember all of this one." He described detailed accounts of intraoperative events, such as the surgeon's request for more neuromuscular paralysis because of vocal cord movement and a later request from the surgeon for a spatula to remove burn tissue from the larynx. The patients denied any intraoperative discomfort, including pain or shortness of breath, and remarked that the experience had been quite interesting because "now I know what you guys do to me every 6 weeks."

Close follow-up over the subsequent 18 months revealed the patient to be doing well. He continues to be meaningfully employed and has no psychological problems or sleep disturbances related to this incident.

DISCUSSION

The abolition of memory for intraoperative events (*i.e.*, amnesia) is an integral and desirable component of prop-

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