

## CASE REPORTS

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## Delayed Recovery from Muscle Weakness Due to Malignant Hyperthermia during Sevoflurane Anesthesia

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MALIGNANT hyperthermia (MH) is a disorder of skeletal muscle metabolism precipitated by volatile anesthetic "trigger" agents, resulting in a various degree of destruction of skeletal muscle tissue.<sup>1</sup> It is not surprising that subsequent muscle weakness might be one of the postoperative manifestations of MH; however, to our knowledge, no report about it has been available. In a patient recovering from MH related to anesthesia with sevoflurane, one of the newer volatile anesthetics, we document post-event muscle weakness.

### Case Report

A 27-yr-old, 85-kg man was scheduled for tonsillectomy because of recurrent tonsillitis. Neither the patient nor his family had any history of neuromuscular disease, and he had not received general anesthesia previously. Preoperative laboratory examinations revealed a slight

Table 1. Time Course of Manual Muscle Test\*

Muscles Innervated by:	1 mo		2 mo		3 mo	
	R	L	R	L	R	L
Humerus N.	4	4	5	5	5	5
Axillar N.	4	4	5	5	5	5
Musculocutaneous N.	4	4	5	5	5	5
Radial N.	4	4	5	5	5	5
Median N.	4	3	4	4	5	5
Ulnar N.	3	3	4	4	5	5
Femoral N.	4	4	5	5	5	5
Obturator N.	4	4	5	5	5	5
Gluteus N.	4	4	5	5	5	5
Sciatic N.	4	4	5	5	5	5
Fibular N.	4	4	4	4	5	5
Tibial N.	3	3	4	4	5	5

\* Grading system: Complete range of motion against gravity, 5 = with full resistance at end of range; 4 = with some resistance at end of range. Complete range of motion, 3 = against gravity; 2 = with gravity eliminated. No range of motion, 1 = slight contraction; 0 = no contraction.

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increase in concentration of serum creatine phosphokinase (CK, 221 U/L; normal range, 20-100 U/L).

Before induction of anesthesia, body temperature was 36.8°C and his heart rate was 78 beats/min. General anesthesia was induced with thiopental (350 mg). Endotracheal intubation was facilitated using vecuronium (10 mg). Anesthesia was maintained with oxygen-nitrous oxide (66%) and sevoflurane (2.5-5.0%). Twenty-five minutes after



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induction, an increase in heart rate from 78 to 120 beats/min and an elevation of  $P_{ET}CO_2$  from 35 to 60 mmHg were noted. Arterial blood gas measurements at this point showed a severe combined metabolic and respiratory acidemia (pH, 7.006;  $Pa_{O_2}$ , 291.6 mmHg;  $Pa_{CO_2}$ , 97.3 mmHg; BE, -11.7 mEq/l). Rectal temperature increased to 39.3°C 30 min after induction, and marked rigidity of the upper and lower limbs was noted. With a likely diagnosis of malignant hyperthermia, the patient was treated with active surface and core cooling and with intravenous administration of 100 mg of dantrolene. The immediate postoperative course was essentially uneventful.

Beginning on the day after the event, the patient complained of severe muscle weakness of the extremities. Muscle weakness did not return to normal even by 1 month after the episode, despite muscle rehabilitation. Hence, manual muscle tests (MMT) of the upper and lower limbs, commonly used for evaluation of muscle strength, were performed. Muscle strength of all muscles in the extremities was reduced to grade 3 or 4 (normal value, 5). Two months after the episode, the distal part of the limbs, such as the muscles of the forearms and those of the crus (those innervated by fibular and tibial nerves), remained unimproved, and a further month was necessary for all the muscles to improve completely. Muscle weakness remained in the areas where severe muscle rigidity had occurred at the time of the episode (table 1). A hand dynamometer test showed marked

improvement of grasping power from 20 kg on the right and 19.5 kg on the left at 1 month to 44 kg and 42.5 kg, respectively, at 2 months.

## Discussion

Pathohistologic studies have revealed various degrees of skeletal muscle destruction in muscle specimens obtained from patients recovering from MH.<sup>2</sup> The degree of muscle weakness resulting from such muscle destruction may vary, but from the present case, it is suggested that at least 2 or 3 months is required for complete recovery from muscle destruction.

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# Fatal Acute Myocardial Infarction during General Anesthesia in a 7-yr-old Boy Associated with Total Intramural Coronary Arteries

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INTRAMURAL coronary artery, mural coronary artery, tunneled coronary artery, myocardial coronary bridging, and myocardial loop are all terms used to describe

an anatomic variation in which an epicardial coronary artery, especially the left anterior descending (LAD) artery, becomes surrounded by myocardial fibers for some distance and depth but returns to the epicardial surface distally. We describe the case of a child in whom all epicardial coronary arteries had an intramyocardial course, probably causing fatal myocardial infarction during general anesthesia.

## Case Report

A 7-yr-old boy with shortening of the right Achilles tendon from right hemiparesis as a result of congenital cerebral palsy was scheduled for a lengthening of the Achilles tendon. He had been born at 35 weeks of gestation and had weighed 1780 g. A ventricular septal defect (VSD) was diagnosed immediately after birth. He was treated with digoxin, and at age 3 yr, right ventricular angiography and catheterization revealed little shunt flow through the VSD, a mean pulmonary pressure of 20 mmHg, a cardiac index of  $6.45 \text{ l} \cdot \text{min}^{-1} \cdot \text{m}^{-2}$ , and a left ventricular ejection fraction

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