surgery despite the use of acetaminophen for pain control (which I assume they used) include the cessation of omeprazole and cisapride medication after successful Nissen fundoplication and the use of a much smaller dosage of acetaminophen after the eye surgery.

The case reported by Drs. Côté and Bouchard might be a case of desflurane-induced hepatotoxicity. However, based on the case report they described, it is a case of acetaminophen-induced hepatotoxicity.

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Two Cases with Past and Family History of Febrile Convulsion Developed Seizure-like Movements during Sevoflurane Anesthesia

To the Editor:—Febrile convulsion in children is considered to develop because, as their brain is still immature, they are hypersensitive to hyperthermia. The incidence of febrile convulsions is highest in patients aged 6 months to 3 yr and rare in patients older than 6 yr.[1] Although the precise risk of developing epilepsy after febrile convulsions is uncertain, febrile convulsions are known to be associated with epilepsy in childhood.[2] Epileptic subjects are particularly sensitive to the epileptogenic effect of sevoflurane; however, the degree to which history of febrile convulsions contributes to the epileptogenic effect of sevoflurane has not been determined.[3] Here, we report the cases of two pediatric patients with a personal and family history of febrile convulsion who experienced seizure-like movements during general anesthesia.

Case 1: A 10-yr-old boy was scheduled to undergo an appendectomy for a perforation caused by appendicitis. The patient had experienced febrile convulsions at the age of 3 yr, and his father had a history of them as well. At the time the patient entered the operating room, his axillary temperature was 38.9°C. General anesthesia was induced by 2–3% sevoflurane, 2 l/min oxygen, and 4 l/min nitrous oxide. Twenty minutes after the start of surgery, generalized tonic–clonic seizure-like movements were observed in the patient for 60 s. They disappeared after the administration of 2.5 mg midazolam and reappeared 25 min later. The patient's rectal temperature was 38.9°C. Sevoflurane was replaced by 6 $mg \cdot kg^{-1} \cdot h^{-1}$ propofol, and the seizure-like movements did not reoccur. The patient's body surface was cooled by air blasting. His rectal temperature was 38.2°C at the completion of surgery. After the patient awoke from anesthesia, a clinical examination by a pediatrician was conducted, and no significant abnormalities were observed.

Case 2: A 4-yr-old girl was scheduled to undergo an appendectomy for appendicitis. She had experienced febrile convulsions at the ages of 1 and 2 yr, and her father had a history of them as well. General anesthesia was induced by 2–3% sevoflurane, 2 l/min oxygen, and 4 l/min nitrous oxide. The patient's rectal temperature was 37.8°C. Immediately after surgery, while the patient was awakening from anesthesia, rolling of her eyes and generalized tonic–clonic seizure-like movements were observed for 5 s but resolved spontaneously. The seizure-like movements then reappeared, persisted for 30 s, and then disappeared after the administration of 1 mg midazolam. After the patient awoke from anesthesia, a clinical examination by a pediatrician revealed no significant abnormalities.

When seizure-like activities were observed during sevoflurane anesthesia, the rectal temperatures in cases 1 and 2 were 38.9° and 37.8°C, respectively. Although the direct cause of the seizure-like movements in both cases could not be determined, hyperthermia and sevoflurane were considered to be major contributing factors. An increase in the temperature of neuronal tissue could enhance the rate, magnitude, or synchrony of neuronal firing.[2] However, seizure-like movements during sevoflurane anesthesia are most often described during masked induction of anesthesia when sevoflurane at high concentrations (4–8%) is administered; epileptiform electroencephalographic activity has been recorded in healthy subjects at 1.5–2 minimum alveolar concentration levels (2.5–3.3%) of sevoflurane anesthesia.[3] In addition, because febrile seizures do not generally occur in the age group of the two patients, it is possible that the interaction of the increased temperature and sevoflurane elicited neuronal excitability in patients susceptible to febrile convulsions, particularly in those with a positive family history. Although malignant hyperthermia was considered, the rapid recovery of the patients and the absence of a family history for malignant hyperthermia made this unlikely. The incidence of serum creatine kinase increase was not observed in these patients.

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