coded, inversion of the bottle collars may lead to confusing and potentially hazardous bottle adaptor mismatch.

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Use of Spinal Anesthesia in Patients with Idiopathic Hypertrophic Subaortic Stenosis

To the Editor:—Without question, the patient with undiagnosed idiopathic hypertrophic subaortic stenosis (IHSS) who was given a spinal anesthetic tolerated it poorly.[1] However, without knowing the level of the spinal block, which was not stated in the report, and from this one must conclude that it was not measured, it is impossible to know the mechanism(s) for the untoward events that occurred following completion of the block. Furthermore, by publishing this report, Drs. Loubser, Suh, and Cohen and the Editorial Board of ANESTHESIOLOGY imply that spinal anesthesia may be hazardous in patients with IHSS. Such an implication is unjustified. The presence of IHSS may have had nothing to do with the untoward events that occurred.

The symptoms of chest pain and nausea; the signs of diaphoresis, vomiting, hypotension, tachycardia; and ECG changes denoting ischemia can occur in elderly patients who do not have IHSS if the level of spinal block is sufficient to produce a near total chemical sympathectomy. Even the rapid onset of the symptoms and signs after the block does not preclude this possibility. Supposing the patient had not been found to have IHSS when evaluated following the spinal anesthetic, which is altogether plausible and, in fact, a much more common event than spinal plus IHSS, then the diagnosis would have been an adverse response to high spinal anesthesia, and the case would not have been reportable.

The point of raising this issue is twofold. First, it is imprudent to conclude that because something unexpected is found after an untoward event, that that caused or even contributed to the event, particularly when the event is well known to occur in the absence of the unexpected finding. Such is the situation in this case report. Second, this gives me the opportunity to reemphasize the value of continuous spinal anesthesia in situations such as this one. The insertion of a catheter into the subarachnoid space makes the technique highly controllable and quite appropriate in elderly patients with all varieties of heart disease requiring anesthesia for lower extremity surgery.

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Concerning the Antiemetic Efficacy of Metoclopramide

To the Editor:—The results reported by Cohen *et al.*[1] confirm a finding from this Department concerning the brevity of action of metoclopramide.[2,3] In patients premedicated with morphine or meperidine with metoclopramide and having minor gynecologic operations with a standard anesthetic technique, we found a minimal reduction in postoperative vomiting unless a second dose was given at the end of operation. Our doses were 10–20 mg, and these had a short therapeutic but also minimal toxic effects. We would suggest caution with higher doses of metoclopramide, as these can produce extrapyramidal side effects.

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Masseter Spasm Heralds Malignant Hyperthermia—Current Dilemma or Merely Academia Gone Mad

To the Editor:—While the incidence of malignant hyperthermia (MH) encountered during anesthesia is quite low, the probability of mortality occurring in a patient who develops MH is quite high. Hence, any sign that a patient may be susceptible to MH, based either on medical history or on response to anesthetics and anesthetic adjuncts is likely to alarm the attending anesthesiologist. Among the responses to anesthetics or anesthetic adjuncts that are considered to signal patient susceptibility to MH is masseter spasm.[1] Recently, masseter spasm was diagnosed in a patient at our institution. Subsequent discussion among the faculty indicated uncertainty about the definition of masseter spasm, the question of whether or not masseter spasm can be graded by intensity, and, if the spasm can be graded, whether or not any correlation exists between intensity of spasm and susceptibility to malignant hyperthermia.

REPORT OF A CASE

A 2-year-old, 10-kg, male patient was scheduled for orchiopexy. Medical history (part of which was taken retrospectively) revealed no history of unexplained fever, no history of muscle cramps, no extraocular muscle abnormalities, no caffeine intolerance, and no family history pertinent to MH.

After application of EKG and blood pressure cuff, anesthesia was induced smoothly with halothane and N_2O/O_2. At an inspired concentration of 2.5% halothane, the patient was anesthetized deeply, required assisted ventilation, and tolerated iv insertion and laryngoscopy without movement or muscle rigidity. The jaw was quite lax until iv succinylcholine was administered to facilitate endotracheal intubation. Within 30 seconds, the masseter tone had stiffened considerably, making it difficult, but not impossible, to open the mouth. Endotracheal intubation was accomplished quickly, despite the tightened jaw. Rectal temperature was 37° C, heart rate 120 beats/min, blood pressure 100/60 mmHg. A decision to awaken the patient was made by the staff anesthesiologist. The jaw relaxed and the patient was awakened with 100% O_2. He was extubated in the operating room and had an uneventful recovery in the postanesthetic room, except for a temperature elevation to 38.3° C. Serum CPK levels were 380 mU/ml in the operating room, 10,600 mU/ml at 12 h, and 430 mU/ml at 24 h.

DISCUSSION

We feel that the CPK evaluation adds support to the diagnosis of possible susceptibility of this patient to MH.[2] Other staff members argue that the tightness of the jaw observed was nothing more than that—and clearly was not a spasm.

The dilemma concerning definition of masseter spasm and what to do following masseter spasm probably is not unique to our staff. Doubtlessly, there are many experienced anesthesiologists who, based upon troublefree past experiences, would and do continue anesthesia, despite jaw tightness following succinylcholine administration. In our opinion, to do so is to take a risk of unknown magnitude. On the one hand, the reported incidence of MH associated with anesthesia is low. On the other, the incidence of masseter spasm and associated MH is not established firmly. Schwartz *et al.* reported that muscle biopsy tests followed by calcium uptake and actomyosin ATPase studies all were positive for MH susceptibility in 12 patients who had masseter spasm after receiving halothane for induction of anesthesia followed by succinylcholine to facilitate intubation.[3] None of these patients developed classic MH. However, various preventative measures were taken when masseter spasm was diagnosed. Discussion by malignant hyperthermia experts about definition of masseter spasm and grading of "jaw tightness" in terms of predicting susceptibility to malignant hyper-