mittent injection of air and aspiration of fluid. The blood would have been drawn into the intravenous tubing from the patient by the application of negative pressure and would have been injected into the PCA syringe during retrograde injection of air.

Our findings indicate that medication can be aspirated from a PCA syringe *in situ* in a PCA machine simply by the use of negative pressure. Access can be from an injection port, with a needle and syringe used for negative pressure and also for positive pressure to fill the PCA syringe with air in retrograde fashion. Therefore, routine PCA set-up as commonly done may *not* be as secure from violation as was previously believed.

Unauthorized access to PCA opioids, such as we describe, requires the use of a syringe for aspiration of fluid and for retrograde injection of air. According to the clinical signs described above, we suspect that our patient was responsible. Since the meperidine concentration used was 10 mg/ml, and a maximum of 30 ml (300 mg) was replaced by air, if our patient did self-administer the missing meperidine, his 8-h use would have been 390 mg. This amount is close to the 400 mg that he used by PCA during each 8 h of the preceding day.

However, such tampering also could be done by *any* individual with a syringe and needle, including any physician, nurse, relative, or other visitor. Acquisition of syringes and needles by unauthorized personnel should be prevented by routine hospital policies but still may occur. Authorized medical personnel also must have access to syringes and needles and therefore would be able to obtain opioids from a PCA machine in this fashion. Therefore, even if certain patients who may be likely to tamper with PCA machines (such as those with a history of drug-seeking behavior) are excluded from PCA use, it is recommended that methods still be used to minimize PCA machine tampering by other individuals.

Our findings indicate that placement of a one-way valve between the PCA syringe and the first intravenous access port will prevent the type of unauthorized access that we have described. As long as air cannot enter the PCA syringe, no more than 4 ml of opioid-containing liquid can be obtained. This one-way valve should also be inside the locked portion of the PCA, so that it cannot be bypassed. Since this incident occurred, we have learned that C. R. Bard also manufactures an extension set with a one-way valve immediately next to the PCA syringe swivel connection (Bard Anti-Siphon Extension Set, C. R. Bard) Use of this type of extension set will prevent retrograde injection into the PCA syringe.

In an alternative solution without an additional valve, the PCA syringe would be mounted with its outlet upward, so that if air is introduced in the manner described, it would not displace liquid. One risk with doing so is that if such tampering does occur, air injection into the patient could result.

In conclusion, it appears that additional safeguards may be required in current PCA equipment to prevent unauthorized access to the opioid in the PCA syringe. Use of an additional one-way valve or of an upward-facing PCA syringe to prevent retrograde filling of the syringe with air may help to accomplish this.

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Malignant Hyperthermia during Sevoflurane Anesthesia in a Child with Central Core Disease

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Malignant hyperthermia (MH) is a catastrophic, hypermetabolic syndrome that arises in susceptible individuals when they are exposed to certain inhalational anes-

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thetics or muscle relaxants.[1] We recently encountered a case of fulminant MH during sevoflurane anesthesia that was successfully treated by the intravenous administration of dantrolene sodium.

Case Report

A 4-yr-old, 14.2-kg girl with congenital ptosis was scheduled to undergo a Berke operation (contracture of bilateral levator palpebrae superior muscles). Preoperative physical examination and laboratory data were unremarkable except for the presence of ptosis and scoliosis. Although there were no associated neuromuscular problems among family members, her mother also had congenital ptosis and had undergone uneventful regional anesthesia for the same operation. The patient had no known allergies and was receiving no medication.

She received a bromazepam suppository 3 mg and came to surgery well sedated. A precordial stethoscope, a blood pressure cuff, electrocardiogram leads, and a pulse oximeter sensor were applied before induction of anesthesia. Her blood pressure was 124/65 mmHg and her heart rate 142 beats per min. Anesthesia was induced with nitrous oxide (67%) and oxygen, and the inspired sevoflurane (MAC 1.71%) concentration was increased gradually to 3% over 5 min. An intravenous catheter was inserted, and vecuronium bromide 2 mg was administered to facilitate tracheal intubation using a 5.5-mm oral endotracheal tube. Ventilation was controlled, and anesthesia was maintained with 2–3.5% sevoflurane and 50% nitrous oxide in oxygen using a semiclosed anesthesia circuit. Because the patient was scheduled for surgery of 4 h duration, an intraarterial catheter, a rectal thermometer, and a bladder catheter were placed. Blood gas analysis before surgery revealed a *p*H of 7.45, arterial carbon dioxide tension of 32 mmHg, oxygen tension of 278 mmHg, and base excess of 0.1 mM. Her body temperature was 37.7° C.

Surgery proceeded uneventfully for 20 min, at which time the patient's heart rate increased to 180 beats per min and her blood pressure increased to 142/74 mmHg. Peak inspiratory pressure also increased to 30 mmHg, but hemoglobin saturation by pulse oximetry was maintained at 98%. During the next 10 min rectal temperature increased to 38.5° C, and her legs became rigid. The changes in body temperature, heart rate, blood pressure along with blood gas analysis data are summarized in figure 1. Arterial blood gas analysis at this point showed a severe, mixed acidosis. Because of the hyperthermia, rigidity, and severe acidosis, MH was strongly suspected, and body surface cooling was immediately initiated with ice packs and a cooling mattress. Surgery was stopped immediately. Sevoflurane and nitrous oxide were discontinued, and 100% oxygen was administered through a nonrebreathing circuit.

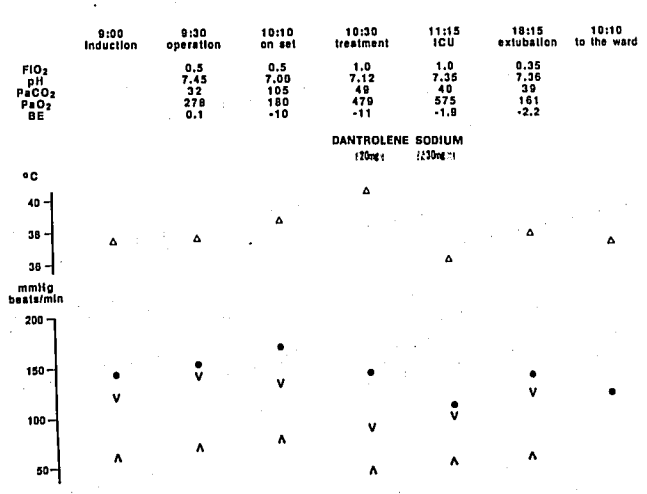

FIG. 1. Time course of analyses of blood gases, body temperature, heart rate, and blood pressure.

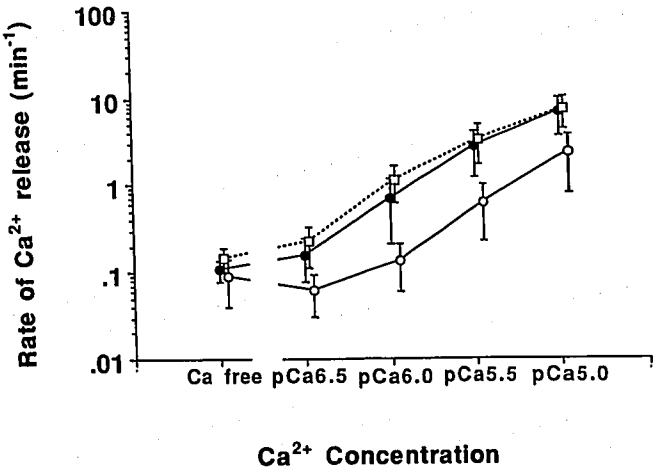

FIG. 2. Results of Ca^{2+}-induced Ca^{2+} release (CICR) rates from the sarcoplasmic reticulum in the skinned fibers at various Ca^{2+} concentrations. Normal subjects (open circles, n = 194) and CICR-enhanced subjects (closed circles, n = 44) are the cumulative data of the biopsied muscles, which were examined for MH susceptibility at Hiroshima University. The presented patient (open squares, n = 3) is included in CICR-enhanced subjects. Bars indicate ± standard deviation of the mean.

The patient then received fentanyl 0.2 mg, dantrolene sodium 20 mg, sodium bicarbonate 1400 mg (16.7 mEq), and furosemide 10 mg. A bundle branch block and slurring of the R waves were noted on the electrocardiogram. Her rectal temperature continued to increase to 40.5° C during the next 15–20 min despite aggressive cooling and improved arterial blood gas values. An additional dose of dantrolene sodium 10 mg was administered, followed by a continuous infusion at 14 $mg \cdot h^{-1}$. Muscle rigidity subsided slightly and rectal temperature began to decrease during the next 10 min. The patient became alert 30 min after discontinuation of anesthesia. At this time, arterial blood gas values were *p*H 7.35, arterial carbon dioxide tension 40 mmHg, arterial oxygen tension 575 mmHg, and base excess −1.9 mM. Urine output was 150 ml over 30 min, and no gross myoglobinuria was noted.

The patient received 245 ml of 2.5% dextrose/0.45% saline solution over 2 h perioperatively. When her rectal temperature decreased to 36.0° C, she was transferred to the intensive care unit. Creatine phosphokinase values were 526 IU/l (normal = 32–180 IU/l) 1 h after surgery and 5312 IU/l the next morning. The urine myoglobin concentration remained less than 10 ng/ml. Dantrolene sodium was discontinued after 2.5 h (total dose 50 mg), and the trachea was extubated 8 h later. The patient experienced no further episodes of muscle rigidity or hyperthermia and returned to the ward on the second morning.

Five days later, a quadriceps muscle biopsy was performed under local anesthesia with procaine and intravenous sedatives for a Ca^{2+}-release test and histologic evaluation. Skinned fibers were examined for the rate of Ca^{2+}-induced Ca^{2+} release from the sarcoplasmic reticulum with various concentrations of Ca^{2+} ion.[2] The results revealed a significant increase in the rate of Ca^{2+}-induced Ca^{2+} release (fig. 2), compatible with a diagnosis of MH susceptibility. Histologic examination disclosed changes typical of central core disease.[3]

The patient was uneventfully discharged from the hospital on the 8th postoperative day.

Discussion

Although Shulman *et al.* have reported that sevoflurane triggers MH in MH-susceptible swine,[4] there has been no report of MH during sevoflurane anesthesia in humans. It has been shown that the release of myoglobin and creatine phosphokinase from the muscle cells during sevoflurane anesthesia is less than that during halothane anesthesia,[5] and in MH-susceptible pigs halothane provokes more severe MH than does sevoflurane.[4,5] Vecuronium, which was used in this patient, is not a triggering agent for MH in susceptible pigs,[6] and thiopental and pancuronium have been shown to delay the onset of MH.[7] We used no anesthetics considered to be triggering agents for MH except sevoflurane.

During the preoperative evaluation, our attention was drawn to the patient's congenital ptosis—a clinical sign, like scoliosis, that has been observed in MH susceptible patients. However, neither the family history nor the preoperative laboratory studies suggested MH susceptibility. Therefore, a diagnostic contracture test was not performed, and dantrolene was not administered prior to anesthesia and surgery.

This case report demonstrates both that sevoflurane can trigger MH in susceptible patients and also that MH triggered by sevoflurane can be successfully treated with intravenous dantrolene.

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Intraoperative Use of Transesophageal Echocardiography with Pulsed-wave Doppler Evaluation of Ventricular Filling Dynamics during Pericardiotomy

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Transthoracic echocardiography is the most frequently used method for the diagnosis of pericardial effusions. In addition to detecting the presence of pericardial fluid, M-mode and two-dimensional echocardiography can establish the presence of tamponade.[1–5] Previous investigators have examined transthoracic M-mode, two-dimensional, and pulsed-wave Doppler echocardiography in nonanesthetized patients undergoing pericardiocentesis.[4–6] We report a case in which transesophageal echocardiography proved clinically useful during pericardiotomy in a patient receiving general anesthesia.

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Case Report

A 59-yr-old man with a history of controlled essential hypertension presented with a recent history of a flulike illness and a pericardial effusion. Two weeks before admission the patient had had an episode of paroxysmal atrial fibrillation associated with severe shortness of breath and fatigue. The patient was treated with metoprolol, digoxin, and quinidine with successful conversion of the atrial fibrillation to sinus rhythm. Diagnostic evaluation included a transthoracic echocardiogram that showed a large pericardial effusion without tamponade. Because of increasing shortness of breath at rest, a pericardiotomy (pericardial window) was scheduled. Preoperative examination revealed an arterial blood pressure of 118/60 mmHg and no pulsus paradoxus or jugular venous distention, and the electrocardiogram revealed sinus rhythm at a rate of 90 beats per min.