space. The air may have passed through the same ball-valve mechanism that later allowed CSF to exit the subarachnoid space. This sequence of events is substantiated by the presence of an anatomic communication, by a sufficient driving pressure for air flow, and by a time sequence that is compatible with previous experimental findings. Active movement of air into the cranium seems to have occurred postoperatively in this case. Pneumocephalus, therefore, must be included in the differential diagnosis of postoperative neurologic deterioration.

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Anesthetic Management of a Child with Asthma and Presumed Susceptibility to Malignant Hyperthermia

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The anesthetic management of a patient with asthma or malignant hyperthermia (MH) presents certain well-documented problems.1,2 When these two diseases coexist, potentially conflicting anesthetic requirements are present. We describe the anesthetics for two separate surgeries in a child with well-documented asthma and presumed MH.

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REPORT OF A CASE

A 6-year-old, 16-kg boy with multiple congenital abnormalities, including strabismus, was scheduled for closure of a patent ductus arteriosus. Past medical history included at least six hospitalizations for pneumonia and asthma in the first two years of life. Treatment was with antibiotics, subcutaneous and inhaled epinephrine, and oral theophylline. He had one previous anesthetic for diagnostic bronchoscopy at 12 months of age. A family history of MH was not elicited prior to the procedure. A 25-min anesthetic with halothane and paralysis using succinylcholine was performed with no adverse sequelae, apart from bronchospasm of mild degree persisting postoperatively. Physical examination disclosed a mildly retarded boy with facial abnormalities. Chest auscultation revealed no wheezes. Laboratory values were normal.

On the morning of this surgery, the patient’s grandmother gave a history of an asthma-associated death in a 16-year-old cousin, with a rise in temperature to 42° C. A creatine phosphokinase (CPK) estimation in our patient gave a value of 508 units (normal value, 45 to 215); surgery was postponed. We elected to reschedule surgery and treat the child as susceptible to MH. Dantrolene, 1.5 mg/kg, was given orally qid for two days. This is within the dose range recommended for prophylaxis of MH.2
Premedication included 3 mg diazepam, po, 3 mg morphine sulfate, im, and 4 mg dexamethasone, im. The dexamethasone was given as prophylaxis against intraoperative bronchospasm. The premedication left the patient drowsy and cooperative. The patient was placed on a cooling blanket. Intravenous dantrolene, ice, refrigerated fluids, and a protocol for management of MH were immediately available. Induction of anesthesia by inhalation of 70% nitrous oxide allowed for insertion of an intravenous cannula. Rectal and axillary temperature probes were inserted. A latex blood pressure cuff, doppler pulse monitor, and ECG monitor were attached. Thiopental, 60 mg, and 1 mg pancuronium were administered iv to facilitate endotracheal intubation. With FiO₂ at 1.0, 25 μg fentanyl and 1.5 mg/kg procaine were injected iv. Following endotracheal intubation, moderate end-expiratory wheezing with increased tracheobronchial secretions were noted. Adequate ventilation was easily maintained, as judged by manual estimation of bag pressure and emptying rate and confirmed by blood-gas analysis. Inhalation of metaproterenol (via a "medihaler" into a T piece of the endotracheal tube) resulted in little improvement in the wheezing.

During the 150-min surgery, the rectal temperature ranged from 36.2°C to 36.5°C. Heart rate and arterial blood pressure did not change. Arterial blood-gas analysis disclosed no abnormalities (PaO₂, 119 mmHg; PaCO₂, 42 mmHg; pH, 7.56 on FiO₂ of 0.4). Auscultatory findings of increased secretions did not respond to endotracheal suction or repeated inhalation of metaproterenol. Anesthesia was maintained with 50% nitrous oxide, and intermittent increments of thiopental, fentanyl, and procaine. An additional 0.5 mg pancuronium was administered iv.

The patient was transferred to the pediatric intensive care unit with the trachea still intubated because of reluctance to reverse the neuromuscular blockade. The trachea was extubated four hours following surgery. The child continued to produce large quantities of tracheobronchial secretions. Eighteen hours after surgery, bronchospasm worsened, requiring intravenous infusion of aminophylline, and terbutalene and epinephrine by inhalation for the next 24 hours. Temperature remained normal. Dantrolene was discontinued 24 hours postoperatively. The patient recovered with no further significant problems and was discharged home on the seventh postoperative day on no medical therapy.

Four months later, the child was scheduled for a tympanoplasty. A different anesthesiologist was in attendance. Oral dantrolene, 1.5 mg/kg, qid for 24 hours was started. The evening prior to surgery, 5 mg/kg aminophylline were administered iv followed by a maintenance infusion of 1 mg·kg⁻¹·h, which was continued throughout surgery. Premedication included 3 mg diazepam, po, 3 mg morphine sulfate, im, and 4 mg dexamethasone, im. Rectal and axillary temperature probes were inserted. A blood pressure cuff, doppler pulse probe, and ECG were attached. The patient received 6 mg diazepam and 50 μg fentanyl iv, for induction of anesthesia. Pancuronium, 2 mg, was administered to facilitate endotracheal intubation. Anesthesia was maintained with 60% nitrous oxide, intermittent doses of fentanyl, and 1 mg/kg procaine, iv. Following endotracheal intubation, no wheezing was audible, but increased secretions were noted. During the two-hour case, temperature ranged from 36.4°C to 36.6°C. Heart rate, blood pressure, and arterial blood gases remained normal.

At the end of surgery, paralysis was reversed with atropine and neostigmine. The trachea was extubated and the patient was taken to the recovery room where respiratory rhonchi were first noted along with copious secretions. These did not clear, and in addition to aminophylline, an epinephrine aerosol was administered. Temperature remained normal. Dantrolene was discontinued 24 hours postoperatively. Aminophylline was discontinued 48 hours postoperatively and the patient was discharged with no medical therapy.

**Discussion**

Although there is no definite evidence that this child was indeed susceptible to the syndrome of malignant hyperthermia, the family history and an abnormal CPK value made it mandatory to treat the child as susceptible. The fact that the previous anesthetic at one year of age did not trigger the hyperthermia syndrome does not rule out later susceptibility, since many episodes of MH have been reported in patients who have had uneventful anesthetics with triggering agents. This may represent an inadequate triggering stimulus, or possibly a variation in susceptibility with time.²,³ The well-documented asthma also required appropriate management. The two conditions have anesthetic requirements that are frequently in direct conflict with each other.

In the asthmatic child, optimal management may require deep anesthesia with a potent inhaled anesthetic.¹ This is unacceptable in malignant hyperthermia-susceptible patients.⁴ Adjunctive drugs, which may be used freely in the asthmatic patient, such as ketamine,⁵ catecholamines,⁶ aminophylline,⁷ atropine,⁵ succinylcholine,⁶ and lidocaine⁸ are possibly contraindicated in the MH susceptible patient. The anesthetic technique recommended for the MH susceptible patient, based on nitrous oxide, narcotics, and intravenous sedatives, is not the ideal choice for the patient with airway hyperreactivity because this combination does not reliably block airway reflexes, relax airway smooth muscle, or inhibit mediator release.¹

Furthermore, in the asthmatic patient, avoidance of endotracheal intubation, or at least timely removal of the tube, is recommended to avoid irritation of the trachea.¹ However, reversal of the residual effects of muscle relaxants involves the use of neostigmine or other cholinergic agonists. Since these agents increase acetylcholine concentration at the postsynaptic membrane, they may help to trigger MH in a manner similar to succinylcholine. No clearly documented case of initiation of MH by reversal of muscle relaxants has been reported, although the suspicion has been raised.⁵

Adequate pretreatment with dantrolene sodium clearly greatly raises the threshold for initiation of the MH syndrome.² However, it is not clear whether this would allow free use of those agents which may initiate the syndrome, such as halothane, or even those which may be suspected to move the patient towards the threshold of initiation. Also, preoperative use of dantrolene may not be totally protective even in the absence of known initiating agents, as an instance of triggering of MH has been reported in a patient pretreated with dantrolene.⁶
A recent review states that aminophylline is contraindicated in MH. This conclusion is not supported by experience in human patients or animal models of the condition. However, aminophylline is a methylxanthine similar to caffeine. It seems prudent to avoid its use, unless there are overriding considerations. In our patient, the episode of postoperative bronchospasm after the first procedure, possibly related to mediator release during the period of endotracheal intubation, led us to infuse aminophylline, and use neostigmine to allow earlier extubation of the trachea, in the second procedure. However bronchospasm ensued postoperatively in spite of these precautions.

In view of the serious nature of both asthma and MH, the anesthetic management of this patient required careful balancing of the conflicting requirements of these two conditions.

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