never have been exposed to induction of anesthesia in children without parents. Moreover, since our institution's policy is to allow parents to be present during anesthesia induction, the residents are not in an atmosphere in which they objectively can evaluate whether having the parents present represents a disadvantage with faculty, who have practiced in this manner for several years, present.

We conclude that under the conditions of the study, resident anesthesiologists came to accept the concept of parent's presence during anesthesia induction. The moderate degree of anxiety expressed decreased significantly with experience. Although there was an extra measure of inconvenience, the residents indicated that such an arrangement did not interfere with their training experience.

REFERENCES


Malignant Hyperthermia and Glucose-6-phosphate Dehydrogenase Deficiency

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The diagnosis of malignant hyperthermia (MH) may be suspected prior to anesthesia only if the patient has had a previous episode, is a member of a family identified as being susceptible to MH,1–4 or has known associated neuromyopathy.5–8 Consequently, both sporadic cases as well as unrecognized MH susceptible patients who have survived previous anesthesia without any manifestations of MH may escape notice.9–12 We therefore report the following case as an alert to its broad spectrum of presentation.

REPORT OF A CASE

A 22-month-old, 12-kg male child with known glucose-6-phosphate dehydrogenase (G6PD) deficiency was admitted for a staged hypoplastic repair. His red blood cell enzyme deficiency was diagnosed at birth by the maleimide-NAPD screening test. His growth and developmental milestones had been normal, and he had suffered no hemolytic crises. Physical examination was entirely normal except for the presence of his known urologic defect. Laboratory studies also were unremarkable, and there was no evidence of active hemolysis. He had had no prior surgeries. Moreover, his mother, father, a male sibling, and maternal grandmother had all received general anesthesia without recognized complications.

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After administration of meperidine 14 mg, promethazine 14 mg, and scopolamine 0.15 mg im, arterial blood pressure was 90/50 mmHg, respiratory rate 20·min⁻¹, heart rate 110 beats/min and skin temperature 35.9°C. An inhalation induction was initiated by means of a Bain circuit delivering a mixture of N₂O, O₂, and halothane at a fresh gas flow of 2.4·l·min⁻¹. Upper airway obstruction resulted, which was not relieved by insertion of an orotracheal endotracheal. After an adequate level of anesthesia had been achieved, an iv infusion was instituted, the vocal cords exposed and subsequently sprayed with 0.5 ml 4% lidocaine. Immediate laryngospasm resulted, which did not respond to gentle continuous positive pressure with a F₁O₂ of 1.0. Succinylcholine 25 mg was given iv and the laryngospasm resolved. However, severe trismus then developed. The diagnosis of MH susceptibility was considered likely. Anesthesia was discontinued, and the Bain circuit and machine were exchanged for uncontaminated equipment. Ventilation was controlled with an F₁O₂ of 1.0 until the patient was fully awake. Rectal temperature did not increase. Analysis of peripheral venous and arterial blood gases and blood creatine phosphokinase (CPK) concentrations were determined (table 1). The patient was monitored in the recovery room for 6 h without changes in vital signs and then transferred to an intensive care unit for overnight observation. Ten hours after induction of anesthesia, while resting in his mother's arms, he had a sudden increase in heart rate, respiratory rate, and a slight increase in rectal temperature (table 1). While in transport to the recovery room, dantrolene sodium was given, 3 mg/kg, iv. The trachea then was intubated and the patient sedated. Both an arterial line as well as Foley catheter were inserted. Urine output was maintained at 3 ml·kg⁻¹·hr⁻¹ with administration of crystalloids iv. Dantrolene sodium, 1 mg/kg, was given iv every 4 h.

Twice during the next 24 h, resting heart rate and respiratory rate increased prior to the next scheduled dose of dantrolene. When this occurred, additional dantrolene, 2 mg/kg iv was given. After 12 h, the CPK values decreased slowly, the metabolic acidosis corrected, and vital signs remained stable. No free hemoglobin or myoglobin was found in the serum or urine. The trachea was extubated the next morning. He received a tapering oral dantrolene sodium regimen for 48 h. His reticulocyte count remained normal, and repeated peripheral blood smears showed no evidence of Heinz body formation. His subsequent recovery was rapid and uneventful. He and his family then
Table 1. Laboratory Data during and Following the Malignant Hyperthermia Episode

<table>
<thead>
<tr>
<th>Day</th>
<th>1</th>
<th>1</th>
<th>1</th>
<th>1</th>
<th>1</th>
<th>2</th>
<th>2</th>
<th>3</th>
</tr>
</thead>
<tbody>
<tr>
<td>Time</td>
<td>06:00</td>
<td>10:00</td>
<td>11:00</td>
<td>19:00</td>
<td>22:00</td>
<td>02:15</td>
<td>08:30</td>
<td>04:55</td>
</tr>
<tr>
<td>Blood gas</td>
<td>Venous</td>
<td>Arterial</td>
<td>Arterial</td>
<td>Arterial</td>
<td>Arterial</td>
<td>Arterial</td>
<td>Arterial</td>
<td>Arterial</td>
</tr>
<tr>
<td>Fio₂</td>
<td>0.21</td>
<td>1.0</td>
<td>0.40</td>
<td>0.21</td>
<td>0.40</td>
<td>0.40</td>
<td>0.21</td>
<td>0.21</td>
</tr>
<tr>
<td>Pao₂ (mmHg)</td>
<td>75</td>
<td>458</td>
<td>122</td>
<td>99</td>
<td>188</td>
<td>165</td>
<td>84.9</td>
<td>95.9</td>
</tr>
<tr>
<td>%sat</td>
<td>90.9</td>
<td>98.5</td>
<td>98.5</td>
<td>98.5</td>
<td>97.9</td>
<td>97.7</td>
<td>95.9</td>
<td>95.9</td>
</tr>
<tr>
<td>pH</td>
<td>7.24</td>
<td>7.30</td>
<td>7.30</td>
<td>7.51</td>
<td>7.47</td>
<td>7.27</td>
<td>7.35</td>
<td>7.35</td>
</tr>
<tr>
<td>Pco₂ (mmHg)</td>
<td>54.9</td>
<td>41.8</td>
<td>48.9</td>
<td>25.8</td>
<td>32.0</td>
<td>55.1</td>
<td>49.8</td>
<td>49.8</td>
</tr>
<tr>
<td>HCO₃ (meq/l)</td>
<td>22.6</td>
<td>19.7</td>
<td>23.3</td>
<td>19.9</td>
<td>23.0</td>
<td>24.6</td>
<td>26.8</td>
<td>26.8</td>
</tr>
<tr>
<td>Be (meq/l)</td>
<td>-5.1</td>
<td>-5.9</td>
<td>-3.2</td>
<td>-6.0</td>
<td>+1.0</td>
<td>-2.9</td>
<td>+1.1</td>
<td>+1.1</td>
</tr>
</tbody>
</table>

were referred to the section of Pediatric Neurology for further investigation of the pedigree.

**DISCUSSION**

G6PD deficiency is a well-studied cause of nonspherocytic hemolytic anemia in humans. This enzyme is central to the proper function of the pentose–phosphate pathway in the erythrocyte. One of the important products of this pathway is reduced nicotinamide adenine dinucleotide phosphate (NADPH). This, in turn, is utilized by glutathione reductase, a potent antioxidant enzyme, to maintain erythrocyte glutathione (GSSG) in the reduced state (GSH). Erythrocyte GSH is a major defense mechanism in free radical detoxification and protects the globin from oxidative damage.

Similarly, the erythrocytes of MH susceptible swine appear deficient in glutathione peroxidase activity, an antioxidant enzyme also dependent upon the pentose phosphate pathway for production of NADPH. Various discrete enzymes of both the glutathione peroxidase system and the pentose phosphate pathway are known to be deficient in MH susceptible humans as well. Because of trismus following succinylcholine, elevated CPK levels and a slight increase in temperature, we believe the diagnosis of MH is justified, which is the first description of G6PD deficiency in a MH susceptible patient. This finding lends credence to the hypothesis that a facet of MH susceptibility may involve depressed activity of a major antioxidant enzyme system.

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Treatment of a Subarachnoid–Cutaneous Fistula with an Epidural Blood Patch

JEFFREY KATZ, M.B., CH.B.*

Lumbar cerebrospinal fluid (CSF) drainage is used in neurosurgical procedures to drain excess CSF away from the surgical wound in order to provide better surgical exposure. Further, the incidence of CSF leaks following transphenoidal surgery can be decreased by continuing this drainage through the early postoperative period. A patient is described who had lumbar CSF drainage and developed a subarachnoid cutaneous CSF fistula that was treated with an epidural blood patch.

REPORT OF A CASE

A 33-year-old woman who had infertility and galactorrhea underwent a transphenoidal excision of a prolactin-secreting pituitary microadenoma. Anesthesia was induced with thiopental iv and maintained with isoflurane and nitrous oxide in oxygen. Endotracheal intubation was facilitated with pancuronium, and additional doses of fentanyl and pancuronium were administered iv as required. Then the patient was turned onto her left side, placed in a flexed position, and a Cordis® lumbar drainage catheter was inserted at L3–4. A 14-gauge Touhy needle and a nonkinkable silastic catheter that passes through the needle and is left in the subarachnoid space were used. The catheter then can be connected to a closed drainage bag or drained as needed by aspiration with a syringe.

During the surgery, which took about 4 h, 60 ml CSF was aspirated through the catheter. The surgery was uneventful, and the trachea was extubated at the end of the procedure. The lumbar drainage catheter was left in the subarachnoid space to help prevent a CSF leak postoperatively. Following an uncomplicated postoperative course, the catheter was removed 5 days after surgery. At this stage she was taking cortisol acetate 37.5 mg/day po and variable amounts of desmopresin (DDAVP) according to urine output.

After removal of the catheter, a CSF leak persisted that exited at the skin through the original lumbar puncture hole. Over the next 3 days, the CSF leak continued and was especially troublesome during straining in the bathroom or with everyday activity. Cerebrospinal fluid specimens were taken daily and examined for any signs of infection. These remained negative until the leak was controlled.

By the fourth day after removal of the catheter, the rate of CSF leak had decreased, but the leak did not stop completely. It was suspected that the patient had formed a subarachnoid cutaneous fistula. Apart from the discomfort of the constant wetting from the leak, the patient also complained of an uncomfortable feeling in her head, which was not severe enough to limit her activity on the floor while in the hospital. On the 5th day after pulling the catheter out, an attempt was made to seal the leak with an epidural blood patch. The leak was such that when the patient sat up for performance of the blood patch, the stream of CSF flow from the puncture site spurted 25 cm away from her back. Eighteen milliliters of autologous blood was injected into the epidural space at L2–3. Apart from pain during injection of the blood, which was thought to be typical root pain, no problems were encountered. The leak was immediately and permanently controlled.

DISCUSSION

Despite the ease with which this leak was controlled, a subarachnoid cutaneous fistula represents a major source of discomfort to the patient and a continuous portal of entry for bacterial infection.