

■ CASE REPORTS

Anesthesiology
1999; 91:1165-6
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Cardiogenic Failure after Isolated Neurologic Injury: A Report of Two Cases

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CARDIAC sequelae after neurologic injury are a well-described phenomenon.^{1,2} However, most reports focus on electrocardiographic findings as well as pulmonary edema.^{3,4} We present two cases of overt cardiac failure after isolated neurologic injury in two previously healthy patients.

Case Reports

Case 1

A 26-yr-old healthy woman fell from a moving golf cart, striking her occiput. She lost consciousness and experienced seizures at the scene. Initial Glasgow coma scale was 6. She was intubated at the scene and transferred to Hermann Hospital. Intravenous mannitol (70 g) was administered in flight. Upon arrival at the hospital, blood pressure BP was 110-150/75-110 mmHg, and heart rate was 120-150 beats/min. Intravenous lorazepam and phenytoin were administered to treat seizure activity. Computed tomography scan showed occipital fracture with diffuse cerebral edema, obliteration of basal cisterns, subarachnoid hemorrhage, and contusions in the frontal and temporal areas. In the intensive care unit, BP was 98/57 mmHg, mean arterial pressure was 67 mmHg, heart rate was 140 beats/min, and bladder temperature was 38°C. An intracranial pressure (ICP) monitor was placed, showing an initial ICP of 33-38 mmHg.

Efforts to control elevated ICP were immediately instituted. Phenylephrine was started to support mean arterial pressure, and hypothermia to 33°C was instituted for aid in controlling ICP. Status epilepticus was diagnosed on electroencephalogram, for which appropriate anti-convulsant therapy was initiated with phenytoin and lorazepam.

A pulmonary artery catheter was inserted. Central venous pressure was 19 mmHg, pulmonary capillary wedge pressure was 22 mmHg, cardiac index was $1.3 \text{ l} \cdot \text{min}^{-1} \cdot \text{m}^{-2}$, systemic vascular resistance index was $2,733 \text{ dyne} \cdot \text{s}^{-1} \cdot \text{cm}^{-5}$, and mixed venous oxygen satu-

ration was 66%. Phenylephrine was discontinued, and multiple vasoactive agents (dopamine, dobutamine, norepinephrine, milrinone) were sequentially initiated to treat cardiogenic failure. Electrocardiogram showed sinus tachycardia along with poor R-wave progression anteriorly, associated with elevation of cardiac isoenzymes. A two-dimensional transthoracic echocardiogram showed severe depression of systolic function, dilated left ventricle, no valvular abnormalities, and an estimated ejection fraction of 30%.

Other complications included rhabdomyolysis with acute renal failure, as well as hepatic dysfunction from hypoperfusion. By hospital day 4, the patient's hemodynamic profile improved, allowing reduction of pharmacologic support to single-agent therapy with dopamine, and thereafter removal of pulmonary artery catheter.

Despite a complicated course, metabolic derangements eventually corrected, and she was transferred to a subacute facility approximately 4 weeks after injury. Subsequent functional recovery was good, with no evident lasting cardiac dysfunction.

Case 2

A 29-yr-old healthy female presented to the Emergency Center with a chief complaint of the worst headache of her life. Her mental status declined quickly to deep coma, with a Glasgow coma scale of 4. She was intubated and transferred to our facility *via* helicopter. Empirically, 60 g mannitol was administered intravenously. Initial vital signs were BP 80/50 mmHg and heart rate 80 beats/min. A computed tomography scan showed massive intraventricular hemorrhage with associated hydrocephalus, and a ventriculostomy was placed; initial ICP was 18 mmHg. Arteriogram showed a retro-splenic arteriovenous malformation.

In the intensive care unit, BP was 100/65 mmHg, and heart rate was 87 beats/min, with phenylephrine infusion in use to support mean arterial pressure. A pulmonary artery catheter was inserted. Cardiac index was $1.2 \text{ l} \cdot \text{min}^{-1} \cdot \text{m}^{-2}$, pulmonary capillary wedge pressure was 18 mmHg, mixed venous oxygen saturation was 54-67%, and systemic vascular resistance index was $3,720 \text{ dyne} \cdot \text{s}^{-1} \cdot \text{cm}^{-5}$. Profound metabolic acidemia ensued; serum lactate level was 10.7 mmol/L, and phenylephrine was discontinued. Dopamine then dobutamine were begun. A two-dimensional transthoracic echocardiogram demonstrated severely depressed biventricular function with a qualitative ejection fraction of 20-24%. Afterload reduction was attempted with enalaprilat, as well as additional inotropic support with milrinone. Cardiac index eventually improved to $3.7 \text{ l} \cdot \text{min}^{-1} \cdot \text{m}^{-2}$, and pharmacologic support was reduced to single-agent therapy.

She, too, developed hepatic dysfunction and acute renal failure. Malignant ICP developed; barbiturate coma was induced, and ICP gradually improved. Inotropic support was eventually converted to digoxin, which was continued beyond her stay in the intensive care unit. The patient made a full neurologic recovery and wished to

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Received from the Department of Anesthesiology and Critical Care, University of Texas Medical School at Houston, Houston, Texas. Submitted for publication February 1, 1999. Accepted for publication May 11, 1999. Support was provided solely from institutional and/or departmental sources.

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Key words: Complications; hypotension; neurogenic.

proceed with her originally scheduled arteriovenous malformation resection. Preoperative cardiologic workup showed moderately depressed left ventricular function with mildly dilated left atrium and mild mitral regurgitation with a qualitative ejection fraction of 35–39%. An adenosine stress test was performed with normal hemodynamic response, and a myocardial perfusion single-photon emission computed tomography imaging study showed normal results. The patient underwent resection of her arteriovenous malformation 42 days after admission. Her postoperative course was not characterized by cardiovascular complication, although she did develop malignant intracranial hypertension and cerebral venous thrombosis. Computed tomography scan showed diffuse edema, and magnetic resonance imaging identified midbrain infarction. Life support was withdrawn and the patient died.

Discussion

Avoiding secondary brain injury, primarily by avoiding hypotension, is paramount to the long-term outcome of the neurologically injured patient.^{5,6} Classic teaching states that hypotension in the face of trauma is not attributable to brain injury alone, and hypovolemia as a source of hypotension should be investigated. Recently, there has been interest in the clinical existence of neurogenic hypotension. Chesnut *et al.*⁷ reviewed the data in the Traumatic Coma Data Bank to find hypotension associated in the absence of severe extracranial injury in a number of patients; however, the cause was believed to be related to liberal use of diuretics in the early injury period. Direct cardiogenic effects of brain injury have been described in animal models; reductions in heart rate, mean arterial pressure, and cardiac output are presumably attributable to excessive vagal tone.⁸

We report two cases of overt cardiogenic failure in previously healthy patients after isolated neurologic injury. In our experience, the unique aspects of these cases are the marked cardiac dysfunction in the absence

of pulmonary edema. Myocardial necrosis from effects of catecholamine surge has been described, as has an increase in sympathetic nervous system activity with exaggerated catecholamine release after brain injury.⁹ Conceivably, this catecholamine outpouring and tremendous stimulation of cardiac β -receptors result in metabolic activity beyond anerobic threshold, and reduction in cardiac output is similar to that observed in stunned myocardium.

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CASE REPORTS

Anesthesiology
 1999; 91:1167-8
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Use of the Laryngeal Mask for Exchange of Orotracheal Tubes

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ENDOTRACHEAL tubes can be exchanged by passing a tube exchanger through an old endotracheal tube into the trachea, removing the old tube, and then passing a new tube over the exchanger into the trachea.¹⁻⁵ One major problem associated with this technique is that it often may be difficult to advance an endotracheal tube over an exchanger because the tube's progress may be impeded by the epiglottis, the arytenoids, or the pyriform fossae.^{6,7} For example, when a fiberoptic bronchoscope is used as an exchanger, there may be difficulties in tracheal intubation in 50-90% of patients.^{6,8} I report the use of the laryngeal mask, which has a potential role in treatment of patients with difficult airways,^{9,10} for exchange of endotracheal tubes in a patient in whom tracheal intubation and ventilation through a face mask were difficult.

Case Report

A 45-yr-old man, (height, 172 cm; weight, 68 kg) who attempted suicide by taking diazepam and by stabbing himself in the abdomen, was scheduled for emergency laparotomy. Preoperatively, he was drowsy and responsive to simple verbal command only. Chest radiography showed opacity in the right lower lobe. Analysis of arterial blood gases showed arterial oxygen tension (P_{aO_2}) of 70 mmHg, arterial carbon dioxide tension (P_{aCO_2}) of 30 mmHg, and pH of 7.45 (fractional inspired oxygen tension [FiO_2] = 0.21). A gastric tube was inserted and gastric lavage was performed; the tube was left in place.

There was no difficulty in opening the mouth or in extending or flexing the neck; the jaw was not small, nor was the neck short or

"bull-necked"; the thyromental distance was greater than three-fingers width. It was not possible to assess other features of his airway after factors caused by insufficient patient cooperation.

In the operating room, routine monitors were attached while the patient was breathing oxygen. Cricoid pressure was applied and anesthesia was induced using thiopental and succinylcholine. Tracheal intubation using a size 4 Macintosh-type laryngoscope was attempted; however, only the tip of the epiglottis was seen, and it was impossible to insert an 8.0-mm ID endotracheal tube into the trachea. Ventilation via a face mask was attempted, but it was inadequate. Another two attempts at tracheal intubation, using the same laryngoscope but optimizing the patient's head and neck position and using a gum elastic bougie, failed and oxygen saturation by pulse oximeter (Sp_{O_2}) decreased to 88%. While a laryngeal mask and a percutaneous transtracheal airway were being prepared, the fourth attempt at tracheal intubation was made, and Sp_{O_2} further decreased to 65%. This time, a 7-mm ID tube was successfully inserted into the trachea and Sp_{O_2} rapidly increased to 99%. A suction catheter was passed through the endotracheal tube and a considerable amount of viscous fluid was aspirated. Anesthesia was maintained with fentanyl, nitrous oxide, and sevoflurane in oxygen, and muscle relaxation was produced with vecuronium. Excision of a perforated segment of the small intestine proceeded uneventfully.

After surgery, it was decided to transfer the patient to the intensive care unit before tracheal extubation. Nitrous oxide was discontinued, and 4 mg midazolam, 200 μ g fentanyl, and 3 mg vecuronium were injected. Because the pilot balloon of the endotracheal tube was overexpanded, it was planned to adjust the cuff volume. An assistant attempted to remove air from the cuff, but it was difficult. An attempt at adding a small amount of air also was difficult, but suddenly obstruction was relieved and approximately 5 ml air was inadvertently infused. This caused a marked gas leak around the endotracheal tube. It was apparent that the cuff had ruptured, and therefore it became necessary to replace the tube.

Sevoflurane in oxygen was given. The existing endotracheal tube was affixed to the jaw using tape, and a size 5 laryngeal mask was placed without difficulty while the endotracheal tube was still in place. The cuff of the laryngeal mask was inflated with 20 ml air. A fiberoptic bronchoscope (4 mm in diameter) was passed through a 7.0-mm ID endotracheal tube, and the combination was passed through the laryngeal mask so that the tip was just beyond the grill at the aperture of the laryngeal mask. The old endotracheal tube could be seen passing through the vocal cords. The fiberscope was then passed through the vocal cords into the trachea, alongside the old tube. After the carina was identified, the old tube was removed while the laryngeal mask was held in position. The lungs were ventilated through the new tube, which was in the laryngeal mask. The new tube was then advanced over the fiberscope into the trachea without difficulty, and correct reintubation was confirmed by fiberoptic, capnography, and auscultation. The length of the new endotracheal tube was extended by using

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Received from the Department of Anesthesiology, Kansai Medical University, Fumizono-cho, Moriguchi City, Osaka, Japan. Submitted for publication February 9, 1999. Accepted for publication May 11, 1999. Support was provided solely from institutional and/or departmental sources.

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Key words: Difficult airway; endotracheal intubation; fiberoptic bronchoscope.

a second endotracheal tube, and the laryngeal mask was removed over these tubes. The entire exchange procedure took only a few minutes, and time from removal of the old tube to reintubation took less than 10 s. Sp_O₂ remained 100% throughout the exchange procedure.

Discussion

The presence of an orotracheal tube does not prevent the placement of the laryngeal mask because the tube passes through the larynx, whereas the distal part of the laryngeal mask is inserted into the hypopharynx. When the endotracheal tube is affixed to the jaw and the laryngeal mask is inserted, using the index finger to slide the mask against the hard palate, it is possible to place the mask while barely touching the endotracheal tube and the tongue. The endotracheal tube can also be removed without dislodging the laryngeal mask. In our previous descriptions of 30 patients, it was always possible to place the laryngeal mask while the endotracheal tube was still in place, to remove the tracheal tube, and to ventilate through the laryngeal mask.^{6,11}

The laryngeal mask often can be placed successfully in patients in whom tracheal intubation, ventilation through a face mask, or both, has been difficult,^{9,10} although there are circumstances in which placement of the laryngeal mask is also difficult.¹² Even if correct placement of the laryngeal mask has failed, a patent airway will not be lost, because an endotracheal tube is still in place. Attempt at placement of the mask may be repeated (possibly using a different size), or the technique of the use of the laryngeal mask may be abandoned.

There are three possibly useful features of the use of the laryngeal mask for tube exchange. First, by connecting the breathing system to a new endotracheal tube that is inserted in the laryngeal mask, it is possible to ventilate the lungs after removal of the old endotracheal tube during insertion of the fiberoptic and during reintubation. Even if ventilation through the new endotracheal tube is inadequate during exchange procedure, jet ventilation through the suction port of the fiberoptic can be applied.¹³ Although not used in this case, by inserting a tube exchanger through the existing tracheal tube before extubation, it is also possible to apply jet ventilation through the exchanger.¹⁴ Second, the laryngeal mask may facilitate location of the vocal cords using a fiberoptic.¹⁵ Third, advancement of a new endotracheal tube over the fiberoptic into the trachea is less likely to be difficult when the tube is passed through the laryngeal mask than when it is not passed through the laryngeal mask.⁶

The limitation of the use of the laryngeal mask for tube

exchange is that this method can be used only for the exchange of oral endotracheal tubes: it is not possible to place the laryngeal mask while a nasotracheal tube is in place; nor is it possible to insert a nasotracheal tube while the laryngeal mask is in place. Another limitation is that a 6.0-mm ID endotracheal tube is the largest that can be passed through the size 3 or size 4 laryngeal mask, although a 7.0-mm ID tube can be passed through the size 5, as used in this patient.

Possible pitfalls of this technique include dislodgment of the fiberoptic, laryngeal mask, or both, during removal of the old endotracheal tube and failure in advancing a new tube into the trachea. To reduce such problems, the old endotracheal tube should be removed gently while the laryngeal mask is stabilized and while confirming that the fiberoptic is in the trachea. In addition, the old tube should be removed only after the position of the laryngeal mask is confirmed to be optimal.

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CASE REPORTS

Anesthesiology
 1999; 91:1169-70
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Delayed-onset Epinephrine-induced Pulmonary Edema

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EPINEPHRINE overdose is not an uncommon event and is reported in the literature to be caused by inadvertent injection,^{1,2} injection of an incorrect concentration,³ or rapid absorption *via* mucosal surfaces and incisional wounds.⁴⁻⁶ There is, however, no report of epinephrine overdose caused by large debridement and wound surface absorption.

Case Report

A 25-yr-old, 70-kg woman was scheduled for wound debridement. Seventeen days previously she was injured, resulting in a left femoral segmental open fracture and a tibia-fibular fracture. Before this operation, she received general anesthesia three times for open reduction and internal fixation, debridement, and sequestrectomy. She was scheduled for debridement of three areas (a 10 × 5-cm open wound, a 20 × 4-cm open wound, and a 3-cm laceration wound) of her left leg.

Preanesthetic blood pressure (BP) was 131/77 mmHg and heart rate was 115 beats/min. One day before surgery, laboratory data showed the following: hemoglobin, 9.2 g/dl; hematocrit, 27.6%; blood urea nitrogen, 18 mg/dl; creatinine, 1.8 mg/dl; potassium, 4.2 mmol/l. A central venous pressure line was inserted *via* the right femoral vein, and 500 ml packed erythrocytes were transfused for correction of the anemia.

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Received from the Department of Anesthesia, Chang Gung Memorial Hospital, Kweishan, Taoyuan, Taiwan, Republic of China. Submitted for publication September 29, 1998. Accepted for publication May 19, 1999. Support was provided solely from departmental sources of the Chang Gung Memorial Hospital.

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Key words: Asystole; overdose absorption; succinylcholine-induced hyperkalemia.

Standard noninvasive monitors were placed. Anesthesia was induced with 100 µg fentanyl and 300 mg thiopental, with 35 mg atracurium given to facilitate tracheal intubation. Anesthesia was maintained with isoflurane, nitrous oxide, and oxygen. The anesthesia course was uneventful for approximately 3 h. Total blood loss was 500 ml; the amount of intraoperative fluid administration (D51/4NS plus Ringer's Lactate) was 1,600 ml, and urine output was 1,200 ml. However, at the end of surgery, several pieces of gauze dressing containing diluted epinephrine (epinephrine solution, 0.1 w/v%, 100 ml per bottle; DAI-CHI Pharmaceutical Co., Ltd., Tokyo, Japan) were placed over the wound area. The gauze dressings were said to contain a total amount of 100 ml epinephrine solution, 1:100,000. Hypertension (BP near 180/100 mmHg) and tachycardia (120 beats/min) with occasional ventricular premature contractions were noted immediately after the gauze placement. The patient was awakened and extubated in the operation room and was sent to the recovery room.

At arrival in the recovery room, the patient was conscious. BP was 165/115 mmHg; heart rate was 110 beats/min, and respiratory rate was 20 breaths/min. Oxygen saturation by pulse oximetry (Sp_{O₂}) was 99%. One hour later, BP decreased to 106/74 mmHg and tachycardia (134-140 beats/min) was noted. Arterial blood showed the following: pH, 7.35; arterial oxygen tension (Pa_{O₂}), 64 mmHg; arterial carbon dioxide tension (Pa_{CO₂}), 34 mmHg; bicarbonate (HCO₃⁻), 19 mmol/l; base excess, -5.4; hemoglobin, 8.9 g/dl, arterial oxygen saturation (Sa_{O₂}), 91.6%; sodium, 131 mmol/l; and potassium, 2.26 mmol/l. Five hundred milliliters packed erythrocytes and 20 mEq KCl in 800 ml Ringer's lactate were infused slowly. Heart rate increased rapidly to 145 beats/min despite treatment, chest auscultation revealed bilateral moist rales, and central venous pressure was 27 cm H₂O with poor urine output. Twenty milligrams furosemide was given. Chest radiography, performed 50 min later, confirmed the diagnosis of pulmonary edema. One hour later, hypotension (82/59 mmHg) developed. The patient became hypoxemic (Sp_{O₂} < 85%) and began coughing up frothy pink sputum. Tracheal intubation was immediately performed after 10 mg diazepam and 80 mg succinylcholine was given intravenously.

Polymorphic ventricular tachycardia with no palpable cardiac output was noted after intubation. Cardiopulmonary resuscitation (CPR) was immediately started. An arterial blood sample, drawn 20 min after intubation, revealed the following: pH, 7.09; Pa_{O₂}, 45 mmHg; Pa_{CO₂}, 52 mmHg; hemoglobin, 12.5 g/dl; BE, -14.7; Sa_{O₂}, 63.7%; sodium, 131 mmol/l; potassium, 8.0 mmol/l; and bicarbonate, 15.6 mmol/l. The patient was treated with epinephrine, direct current defibrillation, lidocaine, sodium bicarbonate, calcium chloride, and regular insulin. Potassium concentrations obtained at 45, 60, 70, and 120 min after intubation were 13.3, 6.3, 7.2, and 5.8 mmol/l, respectively. After 2 h 40 min of CPR, sinus rhythm reappeared with a heart rate of 130 beats/min and a BP of 112/58 mmHg. The patient regained consciousness later.

The patient was transferred to the intensive care unit and was

treated with blood transfusion, intravenous furosemide, and hemodialysis because of post-CPR acute renal failure. A retrospective questioning of the scrub nurse revealed that 100 ml epinephrine, 1:1000 (100 mg), instead of 1:100,000 epinephrine had been inadvertently added into the pieces of gauze that were laid onto the surgical wound at the end of the surgery. Additional information from the ward showed that the patient had not been moving much in the previous 17 days because of nervousness and the fear of movement-associated pain.

During her stay in the intensive care unit there was no evidence of rhabdomyolysis. The patient was discharged to home without any neurologic sequelae after a 3-month hospital stay.

Discussion

The use of epinephrine-soaked gauze is commonly used to stop bleeding.^{5,6} It typically results in some hypertension and tachycardia, but few other problems.^{3,4,6} In this case, however, a nursing error resulted in the use of perhaps a 10-fold increase in the dose of epinephrine to which the patient was exposed. Surprisingly, however, this did not result in a sudden deterioration in cardiovascular function, but rather led to the gradual development of pulmonary edema during a period of several hours after surgery. Typically pulmonary edema induced by epinephrine overdose occurs within 20 min of administration.⁷ However, in this patient, more than 1 h elapsed after placement of epinephrine-soaked gauze before the onset of pulmonary edema. The reason for this delay is not clear, but is presumably related to slow absorption of the applied drug through the damaged skin and a change that may have been related to the drug's local vasoconstrictive effect. Physicians should be aware that delayed complications from the topical administration of epinephrine may occur.

Unfortunately, our efforts to treat this patient's pulmonary edema and hypoxia resulted in another serious complication; *i.e.*, a hyperkalemic cardiac arrest, which was probably secondary to the administration of succinylcholine. Hyperkalemia after succinylcholine administration is well-described and can occur in patients with extensive trauma and prolonged immobilization.^{8,9} The use of succinylcholine was not considered a risk because the patient should have been able to move around after trauma despite her injuries. It was not until after resuscitation we knew that, because of nervousness and pain, she had not been moving much for the previous 17 days. In retrospect, severe hyperkalemia was perhaps predictable, and the use of succinylcholine in this patient was probably inappropriate.¹⁰

There have been articles that reported outcomes in prolonged resuscitation.^{11,12} This patient required almost 3 h of continuous closed chest CPR, and yet survived without neurologic sequelae. This supports the belief that continued resuscitative efforts in young persons may be worthwhile, particularly in the face of a potentially "reversible" or treatable cause, such as occurred in this patient.

In conclusion, we present a case of long-term immobilization that was complicated with inadvertent administration of high-dose epinephrine and succinylcholine-induced hyperkalemia cardiac arrest. Because absorption of epinephrine through the wound may be slow, late responses should be watched for carefully. Succinylcholine must be used with caution in patients with long-term disabilities, metabolic acidosis, and hypoxemia.

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CASE REPORTS

Anesthesiology
1999; 91:1171-3
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Vagotonia and Cardiac Arrest during Spinal Anesthesia

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SUDDEN cardiac arrest during spinal anesthesia can occur in otherwise healthy patients.¹⁻⁶ Although respiratory depression secondary to excessive sedation is responsible in some cases, sudden cardiac arrest can occur without overt signs of respiratory depression in hemodynamically stable patients.¹

Vasovagal reactions have been reported to cause cardiac arrest during spinal and epidural anesthesia.^{5,6} Increased vagal tone, or vagotonia, is present in approximately 7% of the population.⁷ Such individuals frequently have a history of recurrent reactions precipitated by emotional or physical stress. Vagotonic manifestations can include nausea, sweating, pallor, bradycardia, hypotension, and syncope.^{7,8} If a patient who is prone to have vasovagal reactions is exposed to emotional stress during spinal anesthesia, what might otherwise be a benign or transient reaction may progress to cardiac arrest.^{5,6} Two incidents of syncope and asystole, during two separate spinal anesthetics, are described in a patient with a history of recurrent vasovagal reactions.

Case Report

A 37-yr-old, 170-cm, 67-kg, 14-weeks-pregnant patient was scheduled to undergo cerclage for cervical incompetence. Medications included prenatal vitamins, and no allergies were reported. An electrocardiogram was not available. The remainder of the history and physical examination were unremarkable, except for a history of "pass-

ing out" if exposed to needles. Three years earlier, the patient fainted during a spinal anesthetic for a similar procedure. Medical records from the first cerclage were received only after discharge from this admission. Then, the patient was 12 weeks pregnant and scheduled for her first cervical cerclage. She was given 50 mg of 5% lidocaine without epinephrine in the sitting position. Soon thereafter, she fainted, but she recovered spontaneously after 30-60 s. Blood pressure (130/80 mmHg) and heart rate (85 beats/min) were reported as stable during the syncopal episode. The anesthesiologist did not record or recall the sensory level achieved by the spinal anesthetic, except that the level was not high and sensory and motor function of the upper extremities was intact. No sedation was administered. Despite the patient's fear of needles, she consented to a spinal anesthetic for the second procedure.

Upon arrival in the operating room, standard monitors were applied, including a pulse oximeter and a nasal cannula with an end-tidal carbon dioxide sampling port. Midazolam, 2 mg intravenously, was given to reduce her anxiety during placement of the spinal anesthetic. Before the intrathecal injection, 1,500 ml of Ringer's lactated solution was administered intravenously. An intrathecal injection of 80 mg of 5% lidocaine in dextrose was performed using a 25-gauge needle, with the patient in the left lateral decubitus position. Immediately after the injection of the anesthetic, the patient was moved to the supine position. The operating table was tilted so that the patient's head was elevated slightly. Blood pressure and heart rate decreased slightly from 120/70 to 105/65 mmHg and 80 to 70 beats/min, respectively, after injection of the anesthetic. A stable T4 level was achieved 10 min after the lidocaine injection. The patient was awake and breathing comfortably. Twenty minutes after administration of the spinal anesthetic, the patient complained of left hand and arm pain as an antibiotic was given intravenously. Immediately after verbalizing these complaints, bradycardia was noted, which progressed to asystole with loss of consciousness and no palpable blood pressure. The patient was given 100% oxygen by mask, chest compressions were begun, and 1 mg of atropine was administered intravenously. Approximately 30 s later, a sinus rhythm and palpable pulse were noted, and chest compressions were discontinued. Blood pressure and heart rate were 115/70 mmHg and 90 beats/min, respectively; the patient was awake, alert, and breathing without difficulty. Hand strength was not diminished, and the sensory level remained at T4. The patient stated, "I guess that I passed out again."

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Received from the Department of Anesthesiology, University of South Florida College of Medicine, Tampa, Florida. Submitted for publication January 29, 1999. Accepted for publication June 4, 1999. Support was provided solely from institutional and/or departmental sources.

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Key words: Asystole; bradycardia; spinal anesthesia; vagotonia; vasovagal.

Discussion

The balance of parasympathetic and sympathetic activity is not equal in approximately 7% of the population who have increased vagal tone, or vagotonia.⁷ Although

vagotonia can occur at any age, it frequently is observed in young, athletic individuals and typically is precipitated by an emotionally or physically stressful event. Pallor, weakness, sweating, nausea, and retching frequently precede unconsciousness. Physiologic studies have demonstrated that vagal stimulation decreases heart rate, cardiac output, blood pressure, and systemic vascular resistance.⁸ Electrophysiologic studies have demonstrated that conduction across the atrioventricular node is prolonged or dissociated, and resting electrocardiograms frequently reveal first- or second-degree atrioventricular block.⁷ Significant bradycardia, hypotension, and syncope for up to 20 min can occur, even in the absence of anesthesia. Patients with severe or frequent syncope may be treated long-term with atropine, a sympathomimetic agent, or a permanent pacemaker.⁷ In most other cases, symptoms usually are transient and resolve spontaneously, as the balance between sympathetic and parasympathetic activity is restored.

The primary physiologic mechanism responsible for the syncope and cardiac arrest in this patient could be (1) uniquely related to the underlying vagotonia, (2) caused by a combination of the underlying vagotonia and the autonomic disturbances caused by the spinal anesthesia, (3) caused solely by the autonomic perturbations of the spinal anesthesia, or (4) related to either the underlying vagotonia or the autonomic disturbances but unable to make a distinction.

We believe that the syncopal incident during the first cerclage was uniquely related to the underlying vagotonia. Our patient was young and athletic and had a history of "passing out" if exposed to needles. Considering her underlying vagotonia it is not surprising that a reaction occurred in a hospital setting with ample exposure to needles and other stressful circumstances. The sensation of the spinal needle as it entered or exited the skin, or exposure to a sensory-level testing needle, was probably the precipitating event.

The cardiac arrest during the second cerclage resulted from a combination of the underlying vagotonia and the autonomic changes accompanying the spinal anesthesia. If the sympathetic system is blocked by a spinal anesthetic, and a vasovagal reaction occurs, then the balance between the parasympathetic and sympathetic systems may be disturbed even further. The additional vagal stimulation may lead to bradycardia and cardiac arrest.^{3,5,6} Blood pressure and heart rate were stable for over 20 min before asystole suddenly occurred after a painful intravenous injection. This stress event produced additional vagal stimulation, which in the presence of a

sympathetic blockade led to cardiac arrest. In the absence of either contributing factor, the autonomic disturbances of the spinal anesthesia or the vagotonia, symptoms may be mild or unappreciated.

The autonomic changes that accompany spinal anesthesia can cause significant bradycardia and hypotension and could have been the sole cause of either the syncope or cardiac arrest. However, the syncope during the first episode occurred almost immediately after the intrathecal injection, before the local anesthetic had time to have an effect, and blood pressure and pulse were stable, and motor and sensory function in the upper extremity were intact before and after the cardiac arrest. For these reasons, it seems unlikely that the described events were solely related to the spinal anesthesia.

Although reported,¹ respiratory depression is an unlikely explanation for either described incident. The patient received no sedation during the first anesthetic and received only 2 mg intravenous midazolam before cardiac arrest and was responsive, without change in pulse oximetry or end-tidal carbon dioxide readings.

Treatment of sinus arrest during spinal anesthesia depends upon the mechanisms responsible. Because most vagally mediated syncopal episodes resolve rapidly and spontaneously, treatment may be unnecessary. However, if the hypotension, bradycardia, or sinus arrest do not resolve quickly, then a vagolytic agent, such as atropine, or a sympathomimetic agent may be appropriate. Because the bradycardia and sinus arrest were felt to be vagally mediated during the second cerclage, atropine was administered intravenously and proved to be effective. If no response was observed with atropine, epinephrine would have been the next best choice. Administering a potent sympathomimetic agent as the first line of treatment, particularly if there is any doubt as to the cause of the cardiac arrest, has its advantages. Alpha-adrenergic stimulation raises systemic vascular resistance essential for coronary and cerebral blood flow during cardiopulmonary resuscitation, and β -stimulation opposes the negative chronotropic and the inotropic effects of vagal stimulation.

Prompt treatment is important. In a closed-claim study, Caplan *et al.*¹ attributed significant neurologic injury or death to delayed treatment of cardiac arrest during spinal anesthesia. Prophylactic treatment also may be warranted. A history of syncope after exercise or noxious stimuli and first- or second-degree atrioventricular block indicate a state of vagotonia. Prophylactic administration of atropine should be considered, and an external pacemaker readily available.

CASE REPORTS

Otherwise healthy patients can experience sudden cardiac arrest during spinal anesthesia. Vasovagal reactions can occur during spinal anesthesia, may exacerbate the adverse hemodynamic consequences of spinal anesthesia, and led to bradycardia, syncope, and cardiac arrest. Our report suggests that a history of vasovagal reactions may indicate an increased risk of cardiac arrest during spinal anesthesia. In such patients, the clinician should consider administration of prophylactic atropine and be prepared to provide prompt and appropriate resuscitative measures.

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Anesthesiology

1999; 91:1173-5

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Photosensitivity and Perioperative Polyneuropathy Complicating Orthotopic Liver Transplantation in a Patient with Erythropoietic Protoporphyrin

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ERYTHROPOIETIC protoporphyria (EPP) is an inherited disorder of porphyrin metabolism.¹ Common manifestations of EPP include photosensitivity and mild hepatic dysfunction. Occasionally, in patients with EPP, end-stage hepatic failure and, very rarely, neurologic dysfunction develop.²⁻⁷ We describe the perioperative treat-

ment of a patient with EPP and end-stage liver disease, necessitating liver transplantation, whose care was complicated by photosensitivity and severe polyneuropathy.

Case Report

A 54-yr-old man with a medical history of EPP associated with hepatic dysfunction and photosensitivity manifesting as painful swelling and skin discoloration during exposure to artificial or natural light was admitted to the hospital with increasing abdominal pain, low-grade fever, and increasing fatigue. Neurologic examination at admission revealed normal sensory motor, deep-tendon reflex and cranial nerve function and the absence of asterixis. During the patient's first 4 weeks of hospital admission, his liver function deteriorated and generalized weakness developed, with which he involuntarily dropped objects, such as cigarettes and cups, and complained that "his mouth and body felt paralyzed." He was treated with four cycles of plasmapheresis, which reduced his total serum porphyrin level from an initial value of 2,792 $\mu\text{g}/\text{dl}$ to a minimum of 448 $\mu\text{g}/\text{dl}$ (normal range, 16-60 $\mu\text{g}/\text{dl}$), but his hepatic and neurologic function continued to worsen. By the fifth week, the patient was unable to raise his legs, his speech was unclear, he had difficulty swallowing, and he was intubated for

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Received from the Department of Anaesthesia and Critical Care, and the Department of Clinical Engineering, Massachusetts General Hospital, Boston, Massachusetts. Submitted for publication March 5, 1999. Accepted for publication May 19, 1999. Support was provided solely from institutional and/or departmental sources.

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Key words: Hepatic failure; phototoxicity; porphyria; protoporphyria.

respiratory failure. During his sixth week of hospital admission, a suitable donor liver became available.

In preparation for taking this photosensitive patient to the operating room, the intensities of the lights in the operating room were assessed using a detector and photometer (model numbers XRD140A and 1700, respectively; International Light, Newburyport, MA). The detector, which had a detection range of 350 nm to 500 nm, was placed on a table in the center of the operating room directly under the focused lights. Covering the overhead incandescent operating room lights with an amber TA-81 filter (Madico Inc., Woburn, MA) reduced their intensities in the 350–500 nm range by 97%. The addition of a clear filter (CLS-200-X; Madico Inc., Woburn, MA) further reduced the intensity to immeasurable levels. Similarly, the TA-81 and CLS-200-X filters together reduced the light intensity from a halogen head lamp to immeasurably low levels in the aforementioned wavelength range. The light intensity of the operating room's three fluorescent ceiling lights was only 5% of that produced by even a single overhead incandescent operating room light, and therefore we concluded that it was not necessary to filter this light during surgery.

The patient was transported to the operating room while intubated but awake. Anesthesia was induced with fentanyl and was maintained with nitrous oxide and isoflurane and small doses of muscle relaxant. The procedure proceeded smoothly, with an estimated blood loss of 4,000 ml, and the patient was transported in stable condition to a cubicle that was equipped with light filters in the intensive care unit. The patient was awake by the next morning and his new liver functioned well. Although still weak, the patient was oxygenated and was ventilating well with low levels of pressure support in the morning of postoperative day 3 and a trial extubation was performed. However, he was reintubated later that day secondary to tachypnea and respiratory fatigue with an arterial carbon dioxide tension of 99 mmHg. A neurologic evaluation showed profound, diffuse proximal muscle weakness with some distal sparing. Deep-tendon and gag reflexes were absent. Electromyography confirmed the presence of a profound axonal neuropathy, showing attenuated compound muscle action potentials and sensory nerve action potentials in the upper extremities, relatively preserved compound muscle action potentials and sensory nerve action potentials in the lower extremities, and active proximal denervation. A lumbar puncture revealed a normal level of protein in the cerebrospinal fluid. The patient was extubated again on postoperative day 9, but was reintubated 3 days later for increasing respiratory distress. He returned to the operating room on postoperative day 14 for placement of a tracheostomy and jejunostomy under filtered light. One month after the transplantation surgery, the patient was completely awake and alert but remained areflexic and completely unable to raise his arms or legs. He required mechanical ventilation until the ninth postoperative week. At that time, he recovered sufficient strength to lift his right arm several inches from his bed. After nearly a year of physical rehabilitation, he has recovered most of his strength and has resumed work.

Discussion

Porphyrias are diseases resulting from either inherited or acquired enzymatic abnormalities in heme synthesis.¹ First described by Magnus in 1961, EPP results from an inherited error of porphyrin metabolism caused by a partial deficiency of ferrochelatase, the terminal enzyme

of the heme biosynthetic pathway that catalyzes the insertion of iron into protoporphyrin IX.⁸ The clinical manifestations of EPP result from the accumulation of free protoporphyrin in the skin, erythrocytes, plasma, bile, and feces.⁹ Although mild abnormalities in liver function test results are common, progression to hepatic failure may occur. It is believed that crystalline protoporphyrin deposits lead to hepatocyte injury. Transplantation is an effective treatment option for protoporphyrin-induced liver failure, but recurrent hepatic graft injury in patients with EPP has been reported.¹⁰

Neurologic abnormalities are not uncommon in patients with hepatic porphyrias, such as acute intermittent porphyria, hereditary coproporphyrin, and variegated porphyria, but have rarely been reported in patients with EPP. In our review of the English literature, we found reports of only 12 other cases of neurologic complications arising in patients with EPP.^{2–6} In all but one patient, neurologic dysfunction occurred in the setting of end-stage liver disease and high levels of protoporphyrins in erythrocytes or plasma, or both. In the one exception, the neuropathy was attributed to Guillain-Barré syndrome, suggesting that the polyneuropathy seen in patients with EPP may result from an accumulation of protoporphyrin within nerve tissue.⁴ Postoperatively, our patient exhibited profound, diffuse weakness and a normal cerebrospinal fluid protein, and electromyographic studies were indicative of active denervation without demyelination. These findings have been reported in the small number of other patients with EPP-associated neuropathy and are not consistent with the demyelinating syndrome of Guillain-Barré syndrome.¹¹ Cyclosporin and tacrolimus have also been associated with neurologic dysfunction.^{12,13} Although these drugs may have contributed to the postoperative weakness exhibited by our patient, significant dysfunction was noted before the administration of immunosuppressive drugs. Finally, profound muscle weakness may be a manifestation of critical illness polyneuropathy. In our patient, the time of onset, the greater proximal involvement, the rapid progression of weakness, and the electromyography findings tend to argue against this diagnosis, although we cannot exclude the possibility that it contributed to his neuropathy, particularly in the immediate perioperative period.

In addition to polyneuropathy, our patient also experienced photosensitivity. This is not an uncommon complaint of patients with EPP who have high levels of protoporphyrin, and it presents a practical problem for medical providers in the operating room.¹⁴ Protoporphyrin

CASE REPORTS

rin absorbs light in the violet region, with a maximal absorption between 400–410 nm. This may lead to the formation of free radicals that can induce tissue damage, ultimately leading to the formation of biliary fistulas, intestinal perforation, and even death.^{2,3,15}

Two preventive measures were used in this patient to decrease the risk of light-induced injury. First, plasmapheresis was used preoperatively to decrease protoporphyrin levels.¹⁶ Exchange transfusion has also been suggested as a treatment before surgery, although it was not performed in this case.¹⁷ Second, the operating room lights and halogen head lamps were fitted with filters to minimize light output at the wavelengths absorbed by protoporphyrin.¹⁸ Filters were not placed over the fluorescent ceiling lights because we thought that the light output was not sufficient to cause tissue damage. In retrospect, it may have been prudent to filter these lights as well; after sitting on a table in the operating room for 2 hours following resection, the native liver developed a dark discoloration on fluorescent light-exposed surface, indicative of a phototoxic reaction.

The authors thank Saad Shafqat, M.D., Ph.D., for helpful comments during the preparation of this report.

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