Anesthesiology
83:211–213, 1995

Hyperkalemic Cardiac Arrest after Succinylcholine Administration in a Child with Purpura Fulminans

Wenzel D. Kovarik, M.D., Jeffrey P. Morray, M.D.†*

ALTHOUGH hyperkalemia after succinylcholine administration has been associated with burns, tetanus, paraplegia, encephalitis, crush injuries, and neuromuscular disease,[1–4] it has not been reported in the setting of purpura fulminans. The following case illustrates this association.

Case Report

A 15-yr-old, 75-kg girl underwent orthotopic liver transplantation for treatment of fulminant Epstein-Barr viral hepatitis and hepatic failure. The intraoperative course was complicated by splenic rupture requiring splenectomy. During the first postoperative week, acute renal failure developed requiring hemodialysis. Her extremities remained cool and poorly perfused despite vasodilator therapy. Areas of purpura developed on both lower extremities and the right upper extremity, and purpura fulminans was diagnosed. She developed evidence of rhabdomyolysis with creatine phosphokinase (CPK) levels of approximately 160,000 U/l. Two weeks after surgery, a liver biopsy was performed that showed evidence of mild rejection. Of note is the fact that she received succinylcholine for the liver biopsy without incident.

Three weeks after surgery, she was transferred to our institution for treatment of purpura fulminans. Her right lower extremity was cold and without sensation below mid-calf. The toes on her right foot were immobile and gangrenous, and she had no palpable right dorsalis pedis or posterior tibial pulses. She was scheduled for an amputation of her right leg below the knee for the following day.

After dialysis 18 h before surgery, her CPK was 2,842 U/l (upper limit of normal 215 U/l); electrolytes were within normal limits, including a serum potassium of 4.8 mEq/l; hematocrit was 33.4; and leukocyte count was 22,900/mm^3, with 92% polymorphonuclear leukocytes. Her liver function tests were abnormal, including an SGPT of 158 U/l (upper limit of normal 30 U/l), SGOT of 72 U/l (upper limit of normal 41 U/l), a GGT of 349 U/l (upper limit of normal 55 U/l), and a conjugated bilirubin of 5.3 mg/dl (upper limit of normal 0.3 mg/dl). Her blood urea nitrogen was 91 mg/dl (upper limit of normal 20 mg/dl), and her creatinine was 4.4 mg/dl (upper limit of normal 1.0 mg/dl).

On the day of surgery, gastric feedings were stopped 12 h before induction of anesthesia; however, because of concerns about delayed gastric emptying and the risk of aspiration, a rapid-sequence induction with cricoid pressure was performed. After oxygenation *via* mask, 500 mg sodium thiopental and 110 mg succinylcholine were given intravenously. Fasciculations were noted. The trachea was intubated without difficulty; breath sounds were auscultated bilaterally, and carbon dioxide was detected in the expired gas. While the endotracheal tube was being secured, ventricular tachycardia was noted on the oscilloscope; pulses were undetectable, and closed-chest massage was begun. Lidocaine was administered intravenously, and cardioversion was attempted unsuccessfully. Arterial blood gas analysis included *p*H 7.36, Pa_{CO_2} 17 mmHg, Pa_{O_2} 175 mmHg, and base deficit − 15.7 mEq/l. Serum potassium was 7.8 mEq/l; a repeat value was 9.0 mEq/l. Multiple doses of sodium bicarbonate, calcium chloride, glucose, and insulin were given. The cardiac rhythm altered between wide complex tachycardia and severe bradycardia, and pulses remained nonpalpable. Intravenous epinephrine and bretylium failed to restore cardiac function, and resuscitative efforts were discontinued after 1 h.

Pertinent findings at autopsy included changes in the liver consistent with graft rejection. Interstitial nephritis and fibrosis were seen in the kidneys. Multiple areas of recent (less than 1 week) infarction were present in the liver and left kidney. The gangrenous areas of the leg showed extensive necrosis of skeletal muscle. Many degenerating muscle fibers were found in the psoas muscle and sections of the heart. As all cultures remained negative, the etiology of the purpura fulminans was not determined. Death was ascribed to cardiac arrest associated with hyperkalemia.

Discussion

Hyperkalemia and the associated cardiac arrest probably resulted from an excess release of potassium from damaged muscle after succinylcholine administration. The serum potassium value was within normal limits

* Assistant Professor, Department of Anesthesia, Oregon Health Sciences.

† Professor, Department of Anesthesiology, University of Washington and Children's Hospital and Medical Center.

Received from Oregon Health Sciences, Portland, Oregon, and the University of Washington and Children's Hospital and Medical Center, Seattle, Washington. Submitted for publication December 12, 1994. Accepted for publication March 13, 1995.

Address reprint requests to Dr. Morray: Department of Anesthesiology, University of Washington and Children's Hospital and Medical Center, 4800 Sand Point Way NE, P.O. Box 5371, Seattle, Washington 98105.

Key words: Ions, potassium: hyperkalemia. Neuromuscular relaxants: succinylcholine. Purpura fulminans.

before surgery, and no exogenous potassium was given before or during the procedure.

Depolarizing agents, such as acetylcholine and succinylcholine, act at the end-plate receptor to increase ionic permeability; depolarization occurs as a secondary effect. The associated ionic fluxes include influx of sodium and efflux of potassium. Some of the increased extracellular potassium is taken up by the venous circulation, accounting for the small increase in serum potassium (0.5 mEq/l) found after succinylcholine administration to subjects with normal muscle.[5]

Acetylcholine receptors can be both up- and down-regulated.[6] With up-regulation, receptors develop in extrajunctional areas; as a result, the area of chemosensitivity, depolarization, and chemical transmission expands, and more ion channels become available to release potassium during depolarization with succinylcholine.

Conditions associated with up-regulation may include interruption of nerve impulses, as with upper or lower motor neuron injuries. The risk of hyperkalemia is greater with lower motor neuron injuries, suggesting that loss of a neurotropic effect from muscle denervation is important in up-regulation. Degeneration of muscle after a burn or traumatic injury can result in up-regulation. Even disuse of muscle may increase sensitivity to depolarizing agents, though the amount of potassium release is less than that associated with denervation or muscle degeneration.[5] The time course of the spread of receptors is variable; for disorders involving a less-than-complete loss of acetylcholine activity, the onset has been estimated at 7–10 days.[6] Initially, the receptors increase in perijunctional areas but ultimately spread throughout the muscle membrane. Once this has occurred, succinylcholine can result in life-threatening hyperkalemia. It is noteworthy that our patient did not have an adverse clinical response to succinylcholine 1 week before her cardiac arrest and death.

Ours is the first report of hyperkalemic cardiac arrest with succinylcholine in association with purpura fulminans. Purpura fulminans may result in up-regulation of receptors through denervation injury and muscle injury. Severe rhabdomyolysis has been reported as an occasional association with purpura fulminans.[7] This was the case with our patient. Muscle injury was evident from her increased CPK levels, though the value was less (2842 U/l) when she had a fatal reaction to succinylcholine than it had been a week previously (160,00 U/l), when she did not. This emphasizes the importance of the spread of acetylcholine receptors over a short period. The injured muscle in her legs represented a depot of acetylcholine receptors. The autopsy suggests that clinically unsuspected areas of skeletal muscle necrosis were present.

Denervation injury may have been present in our patient. Sensation was absent distal to her calves bilaterally, and she was unable to move the toes of her right foot, findings that suggest both sensory and motor deficits. The neuropathy that results from uremia has been associated with succinylcholine-induced hyperkalemia[8] and could have played a role in our patient.

The multiorgan system failure from which our patient suffered probably decreased her ability to tolerate the hyperkalemic insult. A normally functioning liver is capable of rapid uptake of potassium and is able to attenuate the rise in serum concentration.[9] Our patient's liver dysfunction may have limited this attenuation. In addition, the scattered areas of myocardial degeneration found at autopsy may have decreased our patient's ability to tolerate a period of ischemia, thereby precluding successful resuscitation.

In summary, we have presented a girl with purpura fulminans in whom fatal hyperkalemia developed after administration of succinylcholine. Several strategies for prevention should be considered. Small doses of nondepolarizing muscle relaxants can attenuate the hyperkalemic response to succinylcholine.[10] However, a more effective preventive strategy would be to recognize conditions that predispose to hyperkalemia after succinylcholine and to use nondepolarizing muscle relaxants exclusively, if relaxation is required.

References

1. Tolmie JD, Joyce TH, Mitchell GD: Succinylcholine danger in the burned patient. ANESTHESIOLOGY 28:467–470, 1967

2. Cooperman LH: Succinylcholine-induced hyperkalemia in neuro-muscular disease. JAMA 213:1867–1871, 1970

3. Smith RB, Grenvik A: Cardiac arrest following succinylcholine in patients with central nervous system injuries. ANESTHESIOLOGY 33: 558–560, 1970

4. Mazze RI, Escue HM, Houston JB: Hyperkalemia and cardiovascular collapse following administration of succinylcholine to the traumatized patient. ANESTHESIOLOGY 31:540–547, 1969

5. Gronert GA, Theye RA: Pathophysiology of hyperkalemia induced by succinylcholine. ANESTHESIOLOGY 43:89–99, 1975

6. Martyn JAJ, White DA, Gronert GA, Jaffe RS, Ward JM: Up-and-down regulation of skeletal muscle acetylcholine receptors. ANESTHESIOLOGY 822–843, 1992

7. Marcus EL, Hersch M, Sonnenblick M: A rare association of rhabdomyolysis and purpura fulminans in Strep. pneumoniae septicemia. Intensive Care Med 16:521, 1990

8. Walton TD, Farman JV: Suxamethonium-induced hyperkalemia in uraemic neuropathy. Anaesthesia 28:666–668, 1973

9. Fenn WO: The role of potassium in physiological processes. Physiol Rev 20:377–415, 1940

10. Weintraub HD, Heisterkamp DV, Cooperman LH: Changes in plasma potassium concentration after depolarizing blockers in anaesthetized man. Br J Anaesth 41:1048–1052, 1969

Anesthesiology
83:213–217, 1995

Indirect Detection of Intraoperative Carbon Monoxide Exposure by Mass Spectrometry during Isoflurane Anesthesia

Harvey J. Woehlck, M.D., Marshall Dunning III, Ph.D.,† Shantilal Gandhi, M.D.,‡ David Chang, M.D.,§ Douglas Milosavljevic, M.D.§*

INTRAOPERATIVE exposure of a patient to carbon monoxide appears to be uncommon but is a potentially lethal complication of inhalation anesthesia. The passage of difluoromethyl-ethyl ethers, such as isoflurane, enflurane, and desflurane, through dry carbon dioxide absorbents has been shown to result in carbon monoxide production and anesthetic destruction.[1] The true incidence of carbon monoxide exposure during clinical anesthesia is unknown, and no adequate means to detect intraoperative exposure exists at this time. We present two cases in which abnormalities evident *via* mass spectrometry led to the identification of increased carboxyhemoglobin concentrations in patients receiving isoflurane anesthesia without other risk factors for carbon monoxide exposure.

* Assistant Professor of Anesthesiology.

† Assistant Professor of Pulmonary Medicine and Critical Care.

‡ Professor of Anesthesiology.

§ Resident in Anesthesiology.

Received from the Medical College of Wisconsin, Milwaukee, Wisconsin. Submitted for publication December 12, 1994. Accepted for publication March 14, 1995.

Address correspondence to Dr. Woehlck: Froedtert Memorial Lutheran Hospital, 9200 West Wisconsin Avenue, Milwaukee, Wisconsin 53226.

Key words: Anesthetics, volatile: enflurane; isoflurane. Carbon dioxide, absorption: baralyme; barium; calcium hydroxide; soda lime; sodium hydroxide. Carbon monoxide. Monitoring: mass spectrometry.

Case Reports

Case 1

On a Monday morning, a 26-yr-old, 65-kg man underwent bifrontal and bitemporal electroencephalographic grid placement for cortical mapping of intractable seizures. The patient's only medication was 900 mg gabapentin twice daily. The patient had no other medical problems, was a nonsmoker, and had no known preoperative exposure to carbon monoxide. Induction of general anesthesia was performed with thiopental, fentanyl, and pancuronium. Ventilation *via* face mask with isoflurane in oxygen was performed before intubation. After tracheal intubation, anesthesia was maintained with isoflurane in oxygen at 1 l/min and nitrous oxide at 2 l/min. A circle system was used with Baralyme (Chemtron Medical Division, Allied Healthcare Products, St. Louis, MO), the standard carbon dioxide absorbent at our hospital. Gas monitoring was performed with a properly maintained and calibrated MGA 1100 Marquette Gas Analysis system (Milwaukee, WI).

During the surgical procedure, the mass spectrometer indicated that a mixture of isoflurane and enflurane was present, although isoflurane was the only halogenated agent administered to the patient. Anesthetic gas concentrations at this time are shown in table 1. Initially, the possibility was raised that the isoflurane vaporizer was contaminated with enflurane, although this was unlikely because enflurane had not been stocked in our hospital for nearly 6 months. The vaporizer was drained intraoperatively and refilled from a new, unopened bottle of isoflurane, while temporarily maintaining anesthesia with boluses of propofol. To quickly reestablish the previous end-tidal anesthetic concentrations, the fresh gas flows were increased to 4 l/min of oxygen and 4 l/min of nitrous oxide. Only isoflurane was indicated on the mass spectrometer during this period of increased gas flows. The fresh gas flow rates were reduced to 2 l/min of nitrous oxide and 1 l/min of oxygen after the desired end-tidal concentration (6.0–7.5 mmHg) of isoflurane was obtained. Shortly thereafter, the mass spectrometer again indicated that a mixture of isoflurane and enflurane was present. A small portion of isoflurane