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

Wenzel D. Kovarik, M.D., Jeffrey P. Murray, M.D.*

ALTHOUGH hyperkalemia after succinyllcholine administration has been associated with burns, tetanus, paraplegia, encephalitis, crush injuries, and neuromuscular disease, 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 succinyllcholine 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 2.8 ± 2 U/l (upper limit of normal 215 U/l); electrolytes were 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. Murray: 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: Drugs; potassium; hyperkalemia. Neuromuscular relaxants: succinyllcholine. Purpura fulminans.

Anesthesiology, V 83, No 1, Jul 1995
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. 

Acetylcholine receptors can be both up- and down-regulated. 

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. 

The time course of the spread of receptors is variable; for disorders involving a less-than-complete loss of acetylcholine activity, the onset may be 1.7 to 10 days. Initially, the receptors increase in peri junctional areas but ultimately spread through 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. 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 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 to a normally functioning liver capable of rapid uptake of potassium and is able to attenuate the rise in serum concentration. 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. 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


Anesthesiology. V 83, No 1, Jul 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, enfurane, 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.
† 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; enfurane; isoflurane; Carbon dioxide; absorption; baralyme; barium; calcium hydroxide; soda lime; sodium hydroxide; Carbon monoxide; Monitoring; mass spectrometry.

Anesthesiology. V 83, No 1, Jul 1995