

## REFERENCES

1. Carter DM, O'Keefe EJ: Hereditary cutaneous disorders, Dermatology. Edited by Moschella SL, Hurley HJ. Philadelphia, WB Saunders Co., 1985, pp 1200-1206
2. James I, Wark H: Airway management during anesthesia in patients with epidermolysis bullosa dystrophica. ANESTHESIOLOGY 56:323-326, 1982
3. Reddy ARR, Wong DHW: Epidermolysis bullosa: A review of anesthetic problems and case reports. Can Anaesth Soc J 19:536-548, 1972
4. Marshall BE: A comment on epidermolysis bullosa and its anesthetic management for dental operations. Br J Anaesth 35:724-772, 1963
5. Petty WC, Gunther RC: Anesthesia for nonfacial surgery in polydysplastic epidermolysis bullosa (dystrophic). Anesth Analg 49:246-250, 1970
6. LoVerme SR, Oropollo AT: Ketamine anesthesia in dermolytic bullous dermatosis (epidermolysis bullosa). Anesth Analg 56:398-401, 1977
7. Mark LC, Marx GF, Arkins RE, Erlanger H, Joffe S, Kepes E, Ravin MB, Steen SN: Anesthesia in epidermolysis bullosa. Clinical Anesthesia Conference. NY State J Med 66:511-512, 1966
8. Berryhill RE, Benumof JL, Saidman LJ, Smith PC, Plumer MH: Anesthetic management of emergency cesarean section in a patient with epidermolysis bullosa dystrophica polydysplastica. Anesth Analg 57:281-283, 1978
9. Kubota Y, Norton ML, Goldenberg S, Robertazzi RW: Anesthetic management of patients with epidermolysis bullosa undergoing surgery. Anesth Analg 40:244-249, 1961
10. Lee C, Nagel EL: Anesthetic management of a patient with recessive epidermolysis bullosa dystrophica. ANESTHESIOLOGY 43:122-124, 1975
11. Milne B, Rosales JK: Anaesthesia for correction of oesophageal stricture in a patient with recessive epidermolysis bullosa dystrophica: Case report. Can Anaesth Soc J 27:169-171, 1980
12. Singler RC: Pediatric regional anesthesia, Pediatric Anesthesia. Edited by Gregory GA. New York, Churchill Livingstone, 1983, pp 481-518
13. Schulte-Steinberg O: Neural blockade for pediatric surgery, Neural Blockade in Clinical Anesthesia and Management of Pain. Edited by Cousins MJ, Bridenbaugh PO. Philadelphia, JB Lippincott, 1980, pp 503-523

Anesthesiology  
67:264-266, 1987

## Hyperkalemia after Succinylcholine Administration in a Patient with Closed Head Injury without Paresis

DAVID D. FRANKVILLE, M.D.,\* JOHN C. DRUMMOND, M.D.†

Succinylcholine-induced hyperkalemia can occur in many clinical conditions,<sup>1-8</sup> both with and without skeletal muscle paralysis. The report by Stevenson and Birch<sup>9</sup> describing hyperkalemia following succinylcholine administration to a patient recovering from a closed head injury is unique. As pointed out by Miller and Savarese,<sup>10</sup> one should be hesitant in concluding that succinylcholine should not be administered to patients with a closed head injury on the basis of a single case report. This report presents another case of hyperkalemia associated with succinylcholine administration in a patient recovering from a closed head injury, and further justifies concern when administering succinylcholine to this group of patients.

### REPORT OF A CASE

The patient was a previously healthy, 22-yr-old man who sustained an isolated closed head injury following a motorcycle accident. The patient was admitted with an initial Glasgow coma scale of 5 (eyes-1, verbal-1, motor-3). CT-scan showed "diffuse axonal injury with numerous small punctate hemorrhages" of the frontal lobes. There was no surgically correctable lesion. At 31 days post-injury, he was not able to adequately protect his airway and underwent an uncomplicated halothane-atracurium anesthetic for placement of a tracheostomy.

A follow-up CT scan revealed hydrocephalus, and the patient was brought to the operating room 71 days post-injury for placement of a ventriculo-peritoneal shunt. Physical examination revealed a young man, 60 kg, who was unresponsive to voice but would move all extremities spontaneously and in response to pain. He demonstrated good muscle strength and no spasticity, though the Babinski reflex was positive bilaterally. Ventilation was spontaneous *via* the tracheostomy. He was afebrile with no evidence of infection. Medications included tube feedings, trimethoprim/sulfamethoxazole, and phenobarbital 150 mg po BID. Preoperative laboratories included a hematocrit of 40%, white blood cell count of 8,600 cells/mm<sup>3</sup>, and a potassium of 3.6 meq/l.

Operative monitoring included a precordial stethoscope, automatic arterial blood pressure cuff, pulse oximeter, temperature, and continuous electrocardiogram (modified V-5 configuration). Anesthesia was induced with sodium thiamylal (350 mg) iv, followed by hyperventilation with O<sub>2</sub>, N<sub>2</sub>O, and isoflurane. Because of the presence of copious secretions, the patient was suctioned through his tracheostomy which prompted vigorous and persistent coughing. Succinylcholine (60 mg) was administered iv without evidence of fasciculations. Approximately

\* Resident in Anesthesiology.

† Associate Clinical Professor of Anesthesiology.

Received from the Departments of Anesthesia, University of California at San Diego, San Diego, California; and the Veteran's Administration Medical Center, La Jolla, California. Accepted for publication March 12, 1987.

Address reprint requests to Dr. Drummond: University of California at San Diego, M-029, La Jolla, California 92093.

Key words: Anesthesia; neurosurgical. Ions: potassium. Neuromuscular relaxants: succinylcholine.

1–2 min later, the ECG revealed widened QRS complexes with no ectopy or change in rate (fig 1). Arterial blood pressure and oxygen saturation remained stable. Before any specific therapy could be initiated, the ECG began to return to its baseline configuration. A series of serum potassium determinations were performed, with results as follows: preoperatively—3.6 meq/l; 2 min after succinylcholine—9.4 meq/l; 20 min after succinylcholine—4.5 meq/l; and 90 min after succinylcholine—3.4 meq/l.

The remainder of the anesthetic and operation proceeded uneventfully. There were no postoperative sequelae.

### DISCUSSION

Numerous conditions have been associated with succinylcholine-induced hyperkalemia, including: burns,<sup>2</sup> skeletal trauma,<sup>3</sup> severe and chronic infection,<sup>6,7</sup> prolonged immobilization,<sup>11,13</sup> paraplegia,<sup>14</sup> hemiplegia,<sup>5</sup> neuromuscular disorders,<sup>4,5</sup> encephalitis,<sup>12</sup> ruptured cerebral aneurysms,<sup>8</sup> and closed head injury.<sup>9</sup>

Gronert and Theye<sup>1</sup> have outlined a mechanism to account for the hyperkalemic response to succinylcholine which can occur in some of the clinical conditions listed above. This mechanism involves changes in the muscle membrane resulting in “supersensitivity” to acetylcholine and succinylcholine, which results in an exaggerated response to succinylcholine leading to muscle contracture and efflux of potassium. Membrane supersensitivity occurs as a result of both muscle denervation<sup>15</sup> and degeneration of muscle after trauma.<sup>16</sup> However, this mechanism alone cannot account for all hyperkalemic responses to succinylcholine. Thermal injury, disuse of innervated muscle, and upper motor lesions lead to muscle membrane changes, but to a much lesser degree than denervation.<sup>1,17,18</sup> While tetanus and massive potassium efflux can result from succinylcholine administration in these situations, the measured change in muscle membrane sensitivity is actually relatively small.

Of the conditions that have been associated with a hyperkalemic response to succinylcholine, only prolonged immobilization and closed head injury are potentially relevant to this case. In experimental studies, the administration of succinylcholine following prolonged immobilization results in minimal increases in serum potassium.<sup>11,13</sup> However, we are unable to exclude prolonged immobilization as a possible contributing factor.

Our patient was similar to the patient described by Stevenson and Birch.<sup>9</sup> Both experienced a closed head injury and had evidence of upper motor neuron damage (Babinski responses present in both), yet neither demonstrated appreciable skeletal muscle weakness. The lack of motor weakness in a patient with a history of neurologic injury may not be sufficient assurance that succinylcholine administration will not result in hyperkalemia. The observations of Iwatsuki *et al.*<sup>8</sup> are

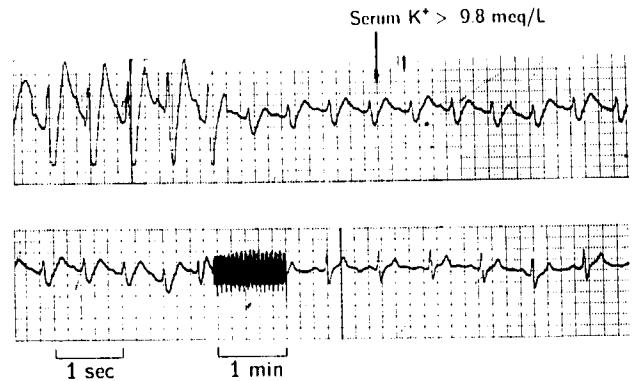


FIG. 1. A continuous EKG recording beginning approximately 90 s after succinylcholine administration. The polygraph was turned on when the change in QRST morphology was noted on the oscilloscope. The gain was reduced by 50% after the fifth QRST complex. The QRS intervals and T wave amplitudes were already decreasing at that time.

consistent with this suggestion. In his report, two of 18 neurologically intact patients experienced massive hyperkalemia (greater than 9.0 meq/l) following succinylcholine administration during induction of anesthesia for repair of ruptured cerebral aneurysms.

The precise significance of the phenomenon we have observed is unknown. Clinical studies have shown that hyperkalemic responses to succinylcholine occur with some regularity, but exhibit great variability in magnitude,<sup>1–4,6–8</sup> even with repeat administration to the same patient.<sup>4</sup> In addition, the response to succinylcholine in patients who have had a full recovery after closed head injury is unknown. It should be reiterated that this patient suffered no sequelae from this event, and that marked hyperkalemia associated with succinylcholine administration has been previously observed without associated cardiac arrest.<sup>2–8</sup> However, the case previously reported by Stevenson and Birch<sup>9</sup> can no longer be considered an isolated report. It would seem prudent to avoid the use of succinylcholine in patients recovering from closed head injury unless the perceived benefits outweigh the potential risks. This circumstance should arise infrequently, given the current availability of intermediate-acting, non-depolarizing muscle relaxants, such as vecuronium and atracurium.

### REFERENCES

1. Gronert GA, Theye RA: Pathophysiology of hyperkalemia induced by succinylcholine. *ANESTHESIOLOGY* 43:89–99, 1975
2. Schaner PJ, Brown RL, Kirksey TD, Gunther RC, Ritchey CR, Gronert GA: Succinylcholine induced hyperkalemia in burned patients. *Anesth Analg* 48:764–770, 1969
3. Birch AA, Mitchell GD, Playford GA, Lang CA: Changes in serum potassium response to succinylcholine following trauma. *JAMA* 210:490–493, 1960

4. Cooperman LH: Succinylcholine induced hyperkalemia in neuromuscular disease. *JAMA* 213:1867-1871, 1970
5. Cooperman LH, Strobel GE, Kennell EM: Massive hyperkalemia after administration of succinylcholine. *ANESTHESIOLOGY* 32:161-164, 1970
6. Kohlschutter B, Baur H, Roth F: Suxamethonium induced hyperkalemia in patients with severe intra-abdominal infections. *Br J Anaesth* 48:557-561, 1976
7. Khan TZ, Khan RM: Changes in serum potassium following succinylcholine in patients with infections. *Anesth Analg* 62:327-331, 1983
8. Iwatsuki N, Kuroda N, Amaha K, Iwatsuki K: Succinylcholine induced hyperkalemia in patients with ruptured cerebral aneurysms. *ANESTHESIOLOGY* 53:64-67, 1980
9. Stevenson PH, Birch AA: Succinylcholine induced hyperkalemia in a patient with a closed head injury. *ANESTHESIOLOGY* 51:89-90, 1979
10. Miller RD, Savarese JJ: Pharmacology of muscle relaxants and their antagonists, Anesthesia, 2nd edition. Edited by Miller RD. New York, Churchill Livingstone, 1986, pp 920
11. Gronert GA, Theye RA: Effect of succinylcholine on skeletal muscle with immobilization atrophy. *ANESTHESIOLOGY* 40:268-271, 1974
12. Cowgill DB, Lucille AM, Shapiro HM: Encephalitis and a hyperkalemic response to succinylcholine. *ANESTHESIOLOGY* 40:409-411, 1974
13. Solendt DY, Partridge RC, Hunter J: The effect of skeletal fixation on skeletal muscle. *J Neurophysiol* 6:17-22, 1943
14. Tobey RE: Paraplegia, succinylcholine and cardiac arrest. *ANESTHESIOLOGY* 32:359-364, 1970
15. Drachman DB, Witzke I: Trophic regulation of acetylcholine sensitivity of muscle: Effect of electrical stimulation. *Science* 176:514-516, 1972
16. Katz B: The development of acetylcholine sensitivity in nerve-free segments of skeletal muscle. *J Physiol (Lond)* 170:389-396, 1964
17. Stone WA, Beach TP, Hamelberg W: Succinylcholine induced hyperkalemia in dogs with transected sciatic nerves or spinal cords. *ANESTHESIOLOGY* 32:515-520, 1970
18. Solandt DT, Magladeny TW: A comparison of effects of upper and lower motor neurone lesions on skeletal muscle. *J Neurophysiol* 5:373-380, 1942

Anesthesiology  
67:266-268, 1987

## Hemodynamic Effects of Intermittent Pneumatic Compression of the Legs

RICHARD J. UNGER, M.D.,\* JOHN R. FEINER, A.B.†

Venous return to the heart is one of the major factors controlling stroke volume.<sup>1</sup> Various physical and pharmacological interventions alter venous return, and, therefore, may have noticeable effects on stroke volume. Increases in stroke volume increase pulse pressure and arterial blood pressure.<sup>2</sup> We report a case in which changes in central venous pressure, pulmonary artery pressure, and pulse pressure were noted as a result of intermittent augmentation of venous return by pneumatic compression stockings.

### REPORT OF A CASE

A 67-yr-old man was admitted to the intensive care unit (ICU) for placement of invasive monitors and determination of a Starling curve the night prior to resection of an abdominal aortic aneurysm. His history was significant for a left ventricular aneurysm following a myocardial infarction in 1982. Coronary angiography showed diffuse coronary artery disease. The left ventriculogram demonstrated areas of hypokinesis, akinesis, and dyskinesis. His radionuclide ejection fraction was 25%.

A 20-gauge catheter was inserted percutaneously into the right radial artery, and a pulmonary artery catheter was placed *via* the right internal jugular vein. Pressures were monitored continuously and car-

diac output was determined by thermodilution. A Starling curve was determined (stroke volume and cardiac output *versus* pulmonary capillary wedge pressure (PCWP)) by altering preload using hetastarch and nitroglycerin (fig. 1). During this process, phasic variations in the pressure tracings were noted at intervals of almost exactly 1 min, and, on inspection, were seen to correspond to the inflation of the pneumatic compression stockings which were placed on the lower extremities for prophylaxis against deep venous thrombosis (DVT) (fig. 2). The timing of the inflation of the stockings was later measured by attaching a T-piece to the air hose of the instrument and recording the pressure on the chart recorder. The variation in pressures ceased when the compression stockings were turned off. Upon review of the hemodynamic tracings, maximal changes apparently were caused by the inflation of the pneumatic compression stockings when the PCWP was 6 mmHg. Pulse pressure increased by 20 mmHg, with a diastolic pressure increase of 10 mmHg. At a PCWP of 15 mmHg, the plateau of the patient's Starling curve, the inflation of the pneumatic compression stockings had virtually no effect on the above variables (fig. 2).

### DISCUSSION

Intermittent pneumatic compression of the legs is an effective technique for decreasing the incidence of deep venous thrombosis by preventing stasis in the venous system of the lower extremities.<sup>3</sup> Peak blood flow in the femoral veins, as measured by Doppler studies, is increased during compression. Factors which maximize the flow have been studied, including the optimal inflation pressures and optimal decompression period. Nicolaides *et al.*<sup>3</sup> demonstrated higher peak blood flows and more rapid clearance of iv radiographic contrast with the sequential compression stockings, which sequentially inflates separate compartments from the

\* Assistant Clinical Professor of Anesthesiology.

† Senior Medical Student.

Received from the University of California, San Diego, UCSD Medical Center, Department of Anesthesia, H-770, 225 Dickinson Street, San Diego, California 92103. Accepted for publication March 16, 1987.

Address reprint requests to Dr. Unger.

Key words: Heart: myocardial function. Veins: pneumatic compression device; prevention; thrombosis.