2. Fairley HB: Prolonged intercostal paralysis due to a relaxant. Brit Med J, Oct. 28, 1950, p 986
3. Feldman SA, Levi JA: Prolonged paresis following gallamine. Brit J Anaesth 35:804, 1963
4. Churchill-Davidson HC, Way WL, deJong RH: The muscle relaxants and renal excretion. ANESTHESIOLOGY 28:540, 1967
5. Jenkinson DH: The antagonism between tubocurarine and substances which depolarize the motor end-plate. J Physiol 152:309, 1960
6. Paton WDM, Waud DR: The margin of safety of neuromuscular transmission. J Physiol 191: 59, 1967
7. Blackman JG: Stimulus frequency and neuromuscular block. Brit J Pharmacol 20:5, 1963

Cardiac Arrest Following Succinylcholine in Patients with Central Nervous System Injuries

R. BRIAN SMITH, M.B., B.S., AND AKE GRENVIK, M.D.

The danger of cardiac arrest following administration of succinylcholine to patients with burns,[1, 2] massive trauma,[3] tetanus,[4] and spinal-cord injuries[5-7] has been described. This paper reports this complication in a patient who developed spastic paralysis following severe brain injury and a patient who was quadriplegic following a posterior fossa craniotomy for a medulloblastoma of the brain stem.

REPORT OF TWO CASES

Patient 1: A 20-year-old white man was involved in an auto accident in which he sustained a head injury which resulted in fixed, dilated pupils, deviated eyes, and incoordinated movements of all extremities. On admission to the hospital, the blood pressure was 60 mm Hg systolic and the pulse rate 140 beats/min. Roentgenograms of the chest disclosed no abnormalities. Serum potassium was 3.8 mEq/l. After improvement of the patient's condition with intravenous fluids, a tracheostomy was performed.

Upon transfer to the Intensive Care Unit, the patient remained unconscious, but the vital signs were stable and breathing was spontaneous. Neurologic evaluation revealed generalized, intermittent spasms which resembled grand mal seizures. Brain-stem injury was diagnosed and the patient was treated with steroids and anticonvulsants. Three days later, he developed a left pneumothorax, which was treated with a tube thoracostomy.

Neurologically, the patient improved slowly, but remained spastic. Fourteen days after admission the spasticity was markedly reduced, his eyes opened on command, and he responded to painful stimuli by moving. Twenty-one days after admission, his arms moved with coordination, but some spasticity remained. He had recovered from intermittent respiratory infections and the tracheostomy tube was removed.

On the 26th day after accident, the patient was scheduled for an open reduction of the left humerus. Preoperative medication consisted of meperidine, 35 mg, and atropine, 0.4 mg. After preoxygenation, anesthesia was induced with 125 mg sodium thiamylal. Intravenous infusion of 0.2 per cent succinylcholine was started and intubation of the trachea was accomplished after about 125 mg had been administered. Following this, the patient suddenly became cyanotic and pulseless. An EKG monitor showed ventricular fibrillation. Ventilation with oxygen and external cardiac compression were started immediately. After six minutes of external cardiac compression and administration of intravenous bicarbonate, the heart was defibrillated successfully with the first countershock. Five minutes later, the patient moved and breathed spontaneously, and the trachea was extubated. Serum potassium was 3.8 mEq/l three and a half hours after the cardiac arrest. In the Intensive Care Unit, the patient had mild spasticity, but could move all limbs. He responded to painful stimuli by movement, but did not speak.

Thirty-five days after the accident, the patient was scheduled again for surgery of his left humerus. Premedication consisted of atropine, 0.4 mg, given 45 minutes preoperatively. Anesthesia was induced uneventfully with methoxyflurane and nitrous oxide, and intravenous 0.2 per cent succinylcholine was started. After about 50 mg of succinylcholine, cardiac arrest recurred and the EKG again showed ventricular fibrillation. After intubation of the trachea, ventilation with oxygen and external cardiac compression were instituted. Following administration of sodium bicarbonate intravenously, satisfactory cardiac function with sinus rhythm returned spontaneously within four minutes. Recovery was uneventful and the patient rapidly returned to his preoperative condition.

At this point, the patient was transferred to the

Received from the Department of Anesthesiology, University of Pittsburgh, School of Medicine, Pittsburgh, Pennsylvania 15213.

Presbyterian–University Hospital for the operation on his left humerus. Induction of anesthesia and endotracheal intubation were accomplished uneventfully with Innovar and nitrous oxide; succinylcholine was avoided.

Patient 2: A 20-year-old white man was admitted in November 1969, with a history of progressive headache, dizziness and vomiting. He had no motor or sensory deficits, but there was bilateral papilledema. He was scheduled for a craniotomy. Preoperative medication consisted of pentobarbital, 75 mg, and atropine, 0.4 mg, given an hour prior to surgery. Anesthesia was induced with 300 mg sodium thiamylal, preceded by 6 mg *d*-tubocurarine. To facilitate intubation, 100 mg of succinylcholine were given, followed by nitrous oxide and halothane for maintenance. No EKG abnormalities were present during intubation or maintenance. A posterior fossa craniotomy was performed with subtotal removal of a medulloblastoma of the fourth ventricle. Postoperatively, the patient developed quadriplegia, with a sensory level of approximately C4.

In May 1970, when the patient was readmitted for radiation therapy, he developed pain in a decayed bicuspid tooth. He was scheduled for dental extraction under general anesthesia. He was premedicated with 75 mg meperidine and 0.4 mg of atropine, an hour prior to surgery. Anesthesia was induced with 100 mg sodium thiamylal after 6 mg *d*-tubocurarine and oxygenation for three minutes. Immediately after intubation of the trachea after administration of 60 mg succinylcholine, cardiac arrest occurred and the EKG showed ventricular fibrillation. Ventilation with oxygen and external cardiac compression were instituted. After intravenous administration of bicarbonate, calcium chloride, and norepinephrine, the patient was defibrillated successfully on the third attempt. The duration of external cardiac compression was six minutes. The patient regained consciousness rapidly and the trachea was extubated uneventfully. The patient regained his preoperative physical status. Fifteen minutes after the administration of succinylcholine, serum potassium was not significantly elevated.

Discussion

Tobey studied the effects of infusion of 0.1 per cent succinylcholine in four paraplegic patients who had sustained injuries 44–85 days previously.[7] Their peak levels of serum potassium, 7.3 to 13.6 mEq./l, returned to normal within 10–15 minutes after cessation of succinylcholine infusion. All had EKG evidence of hyperkalemia. In one patient, circulatory arrest occurred after the administration of 20 mg succinylcholine, but he was resuscitated successfully. Stone, Beach and Hamelberg reported potassium elevation from 4.3 to 5.6 mEq/l in a patient following administration of succinylcholine 23 days after a T8–9 cord transection.[5] They also reported cardiac arrest following succinylcholine in a quadriplegic patient 46 days after injury. His potassium level rose from 4.5 to 11.2 mEq/l two minutes after the administration of succinylcholine. Stone, Beach and Hamelberg studied the effect of the administration of succinylcholine in dogs with bilaterally sectioned sciatic nerves or transected spinal cords.[5] The hyperkalemia was significant beginning after the fourteenth day, becoming highly significant at 28 days.

In our two cases, serum potassium levels were not measured during the administration of succinylcholine. A normal serum potassium 15 minutes after the administration of succinylcholine does not preclude the possibility of a rapid rise and fall in the serum potassium level, since Tobey demonstrated return to normal levels within this period of time.[7] There is convincing evidence that hyperkalemia following administration of succinylcholine is the cause of cardiac arrest. High serum potassium has been shown in patients with spinal-cord injuries by Tobey[7] and by Stone *et al.*[5] It has also been demonstrated in a severely burned patient by Tolmie,[2] and in three of 19 severely traumatized patients by Escue.[9]

Our first case is unusual because cardiac arrest following succinylcholine occurred in a patient with spastic paralysis on the 26th and 35th days following a head injury. The arrests reported by Tobey and by Stone *et al.* occurred in patients with flaccid paralysis secondary to spinal-cord injuries. Our second patient developed flaccid quadriplegia following a posterior fossa craniotomy for a medulloblastoma. Cardiac arrest occurred six months after the patient became quadriplegic. Previously, the longest reported interval between injury and arrest following administration of succinylcholine was 85 days.[7] Although our patient received 6 mg of *d*-tubocurarine prior to succinylcholine, this apparently offered no protection against the effect of succinylcholine. On the other hand, it has been shown in dogs that prior administration of gallamine may prevent the elevation of serum potassium by succinylcholine.[10]

The experience obtained with these two

cases demonstrates that cardiac arrest after administration of succinylcholine may occur in patients who have a wide variety of neurologic problems. Care should be taken not only in the cases of patients with burns, massive trauma and paraplegia from spinal-cord injury, but also following brain injury and other central nervous system diseases, particularly those involving muscular paralysis. Cardiac arrest may occur after the use of succinylcholine in immobilized patients with injuries other than central nervous system injuries.

References

1. Allan CM, Cullen WG, Gillies DM: Ventricular fibrillation in a burned boy. Canad Med Ass J 85:432, 1961
2. Tolmie JD, Joyce TH, Mitchell GD: Succinylcholine danger in the burned patient. Anesthesiology 28:467, 1967
3. Houston JB: Serum potassium changes following succinylcholine (to be published), quoted in Evers W, Racz GB, Dobkin AB: A study of plasma potassium and electrocardiographic changes after a single dose of succinylcholine. Canad Anaesth Soc J 16:273, 1969
4. Roth F, Wuthrich H: The clinical importance of hyperkalaemia following suxamethonium administration. Brit J Anaesth 41:311, 1969
5. Stone WA, Beach TP, Hamelberg W: Succinylcholine—danger in the spinal-cord-injured patient. Anesthesiology 32:168, 1970
6. Thomas ET: Circulatory collapse following succinylcholine: Report of a case. Anesth Analg 48:333, 1969
7. Tobey RE: Paraplegia, succinylcholine and cardiac arrest. Anesthesiology 32:359, 1970
8. Stone WA, Beach TP, Hamelberg W: Succinylcholine-induced hyperkalemia in dogs with transected sciatic nerves or spinal cords. Anesthesiology 32:515, 1970
9. Escue HM, Houston JB, Rudman, H.L, *et al.*: Succinylcholine induced hyperkalemia: A potential hazard following severe trauma or burns. Scientific exhibit, American Society of Anesthesiologists' Meeting, October 1968
10. Stevenson DE: Changes in the blood electrolytes of anaesthetised dogs caused by suxamethonium. Brit J Anaesth 32:364, 1960

Pediatric Bronchial Blocking

Charles E. Hogg, M.D.,* and Paul H. Lorhan, M.D.†

Pulmonary resection in pediatric patients is seldom complicated by copious secretions. Preoperative preparation with intermittent positive-pressure breathing, postural drainage, and antibiotics have minimized the incidence of life-threatening secretions. The rare pediatric patient who has localized copious secretions presents a difficult and dangerous anesthetic situation.

Patients undergoing pulmonary resection for bronchiectasis, pulmonary abscess, hemorrhage, bronchopleural fistula, or hydatid cyst may be managed in one or a combination of three ways to prevent inundation of the good lung with secretions from the diseased lung. The patient may be positioned in a manner which facilitates drainage to the exterior, rather than into the good lung. This generally involves a prone, head-down position. Although this method is reasonably effective in controlling secretions, it has disadvantages from the surgical viewpoint. The exposure afforded by a posterior thoracotomy is less than optimal, and surgeons are reluctant to attempt pulmonary resection with the posterior approach. Double-lumen tubes of the Carlens type [1] offer a satisfactory means of controlling secretions, and allow the use of a standard surgical approach, but they are not satisfactory for pediatric patients because of the size limitations imposed by the airway. A double-lumen tube in pediatric sizes would create excessive resistance to respiration and provide insufficient space for suctioning. Bronchial blockers, which offer a satisfactory means of controlling localized secretions without compromising the surgical approach or unduly restricting the airway,[2] have also been considered unsatisfactory for small pediatric patients because of the limitations imposed by the available methods and mate-

* Assistant Professor of Anesthesiology, U.C.L.A. School of Medicine, Los Angeles, California.
† Professor of Anesthesiology, U.C.L.A. School of Medicine, Los Angeles, California.
Received from the Department of Anesthesiology, Harbor General Hospital, Torrance, California.