

Succinylcholine—Danger in the Spinal-cord-injured Patient

WILLIAM A. STONE, M.D.,* THOMAS P. BEACH, M.D.,†
WILLIAM HAMELBERG, M.D.‡

Patients with burns,^{1,2} massive trauma,³ and tetanus⁴ have increased myocardial sensitivity to the intravenous injection of succinylcholine. A rapid increase in the extracellular potassium concentration has been postulated as the cause.¹⁻⁴ In unreported data, Stone and Tobey observed similarly increased myocardial sensitivity to intravenous succinylcholine in patients with recent injuries to the spinal cord.⁵ Tobey demonstrated consistent elevation of serum potassium in these patients. Of the three patients studied (paraplegic of 44 days, quadriplegic of 24 days, and hemiplegic of 21 days), one had "cardiac arrest" during the observation period.⁶

Two case reports are presented to illustrate our clinical experience with this problem.

REPORT OF TWO CASES

Patient 1. A 31-year-old man was in an auto accident and sustained a transection of the spinal cord at the level of T8-9, with resulting paraplegia. Decompression laminectomy and spinal-column stabilization were performed the day of injury with the patient under halothane-nitrous oxide-oxygen anesthesia after induction with sodium thiopental. Succinylcholine, 120 mg, was given intravenously to facilitate intubation. No difficulty was encountered.

The patient was returned to the operating room on the 23rd hospital day for re-exploration of the laminectomy site. Premedication consisted of meperidine, 100 mg, and atropine, 0.4 mg, intramuscularly, two hours prior to surgical operation. Cardiac activity was monitored by an ORM-1 cardiograph. A catheter was placed in the superior vena cava through the cephalic vein. Oxygen was given by mask for ten minutes.

After induction with sodium thiopental, 260 mg, a control blood sample was drawn for serum sodium and potassium determinations. Succinylcholine, 60 mg, was given intravenously and the patient intubated without difficulty. Oxygen (100 per cent) was given and the chest auscultated to confirm the position of the endotracheal tube. Blood samples for serum sodium and potassium

determinations were drawn at one-minute intervals. No changes in the electrocardiogram were noted. Serum potassium rose from a control value of 4.3 mEq/l to 5.6 mEq/l at two minutes (table 1).

Patient 2. A 17-year-old boy sustained a neck injury in a diving accident, as a result of which he was quadriplegic except for minimal proximal function at the shoulders. He was areflexic. Radiography showed a linear fracture of the sixth cervical vertebra. Myelography was not remarkable. He was treated with cervical traction and stabilized at a C6 sensory level. On the 12th hospital day he was transferred to our hospital.

Physical examination on admission showed normal vital signs except for a temperature of 101 F. Radiography showed acute angulation at C4-5. Roentgenogram of the chest and laboratory data were unremarkable.

The hospital course was complicated by continuous fever due to infiltration of the right middle lobe, acute gastrointestinal hemorrhage, and pulmonary emboli. On the 26th hospital day, with lung-scan evidence of pulmonary emboli, an inferior vena cava ligation was done with the patient under halothane-nitrous oxide-oxygen anesthesia. No succinylcholine was given.

Following this procedure, the patient showed continuous improvement, and on the 46th hospital day he was brought to the operating room for anterior cervical laminectomy to stabilize the fracture site. Premedication consisted of atropine sulfate, 0.3 mg, intramuscularly, and Innovar, 2 ml, intravenously, in divided doses. After oxygenation for ten minutes, anesthesia was induced with sodium thiopental, 100 mg, intravenously, in divided doses. Respirations were vigorously assisted and succinylcholine, 60 mg, was rapidly injected intravenously. Ventilation was controlled and the trachea was intubated without difficulty one minute after cessation of mild fasciculations. The chest was noted to rise symmetrically.

Three minutes after injection of succinylcholine, the electrocardiogram showed S-T segment depression and the rhythm rapidly deteriorated to ventricular fibrillation. No pulse or blood pressure could be detected. Resuscitative measures were instituted immediately. After no improvement was noted with external cardiac massage, sodium bicarbonate, and direct-current counter-shock, open cardiac massage was begun. When the chest was opened the heart was found to be flaccid. Isoproterenol, 64 µg, was given intravenously, leading to a sinus or nodal rhythm with frequent premature contractions. After lidocaine, 30 mg, intravenously, normal sinus rhythm was reported. The blood pressure was 160/80 mm

* Instructor.

† Second-year Resident.

‡ Professor and Chairman.

Received from the Department of Anesthesiology, College of Medicine, The Ohio State University, Columbus, Ohio 43210.

Hg, heart rate 180 beats/min. With no further medication, the blood pressure dropped and stabilized at 120/80 mm Hg; the heart rate stabilized at 110 beats/min. Since the patient began to move and breathe spontaneously, administration of *d*-tubocurarine, 12 mg, in divided doses, and halothane was begun. The remainder of the chest closure was uneventful.

On the patient's return to the recovery room, tidal volume was 300 ml and vital capacity 1,100 ml (Wright respirometer); the trachea was extubated. After 30 minutes of spontaneous respiration, with nasal oxygen at 12 l/min, P_{aO_2} was 68 mm Hg; pH , 7.45; P_{CO_2} , 44 mm Hg. With the patient under topical lidocaine anesthesia, nasotracheal intubation was performed under direct vision. Thirty minutes later, with spontaneous ventilation and oxygen at 12 l/min, P_{aO_2} was 135 mm Hg; pH , 7.49; P_{CO_2} , 42.

During the period of anesthesia blood samples were drawn from the catheter in the superior vena cava. Following withdrawal a control specimen, blood samples were drawn every minute for seven minutes for sodium and potassium determinations. A rapid rise from the control value of 4.5 mEq/l to 11.6 mEq/l at two minutes occurred (table 1).

DISCUSSION

Of the patients studied (Tobey⁶ and the two patients in the present report), three had lesions which had been present for only 21–24 days. Although there were increases in serum potassium on the injection of succinylcholine the patients experienced no difficulty during induction of anesthesia. Two patients with lesions which had been present 44–46 days had greater potassium changes and cardiovascular collapse.

It is known that increased serum potassium leads to cardiac arrhythmia. List⁷ and Evers *et al.*⁸ showed a rise in serum potassium on administration of succinylcholine to normal patients, and Tolmie² documented an augmented rise of serum potassium on the injection of succinylcholine to a burned patient. Roth⁴ showed similar results in patients with tetanus.

After denervation of skeletal muscle, the muscle fibers, in contrast to the motor endplate, become sensitive to acetylcholine in their whole length, and therefore the acetylcholine sensitivity of the muscle increases considerably.⁹ By inference, succinylcholine may have the same effect. Central nervous system injury with paralysis of an extremity is followed by atrophy of the muscles. However,

TABLE 1. Potassium Values (mEq/l)

	Patient 1	Patient 2
Control	4.3	4.5
1 min	5.1	9.5
2 min	5.6	11.6
3 min	5.1	9.8
4 min	4.8	11.5
5 min	4.8	11.5
6 min	—	10.4
7 min	—	10.5

Thesleff has reported that disuse does not produce hypersensitivity of the muscle membrane. We know of no evidence that the sensitivity of muscle to succinylcholine is increased in subjects with spinal cord injury.

Laboratory investigation to determine the extent of the lesions necessary to produce susceptibility to succinylcholine in spinal-cord-injured animals and the time sequence is now in progress. It is suggested that succinylcholine be used with caution in patients with recent spinal-cord injuries.

REFERENCES

- Allan, C. M., Cullen, W. G., and Gillies, D. M.: Ventricular fibrillation in a burned boy, *Canad. Med. Assoc. J.* 85: 432, 1961.
- Tolmie, J. D., Joyce, T. H., and Mitchell, G. D.: Succinylcholine danger in the burned patient, *ANESTHESIOLOGY* 28: 467, 1967.
- Houston, J. B.: Serum potassium changes following succinylcholine (to be published). Quoted in Evers, W., Racz, G. B., and Dobkin, A. B.: A study of plasma potassium and electrocardiographic changes after a single dose of succinylcholine, *Canad. Anaesth. Soc. J.* 16: 273, 1969.
- Roth, F., and Wuthrich, H.: The clinical importance of hyperkalaemia following suxamethonium administration, *Brit. J. Anaesth.* 41: 311, 1969.
- Stone, W. A., and Tobey, R. E.: Personal observation.
- Tobey, R. E.: Personal communication.
- List, W. F.: Serum potassium changes during induction of anaesthesia, *Brit. J. Anaesth.* 39: 480, 1967.
- Evers, W., Racz, G. B., and Dobkin, A. B.: A study of plasma potassium and electrocardiographic changes after a single dose of succinylcholine, *Canad. Anaesth. Soc. J.* 16: 273, 1969.
- Thesleff, S.: Nervous control of chemosensitivity in muscle, *Ann. N. Y. Acad. Sci.* 94: 535, 1961.