Succinylcholine—Danger in the Spinal-cord-injured Patient

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Patients with burns,1,2 massive trauma,2 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. 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 cardioscope. 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 aedetic. Radiography showed a linear fracture of the sixth cervical vertebra. Myelography was not remarkable. He was treated with cervical traction and stabilized at a C8 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 inmigration of the right middle lobe, acute gastrointestinal hemorrhage, and pulmonary emboli. On the 36th 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 48th 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 countershock, open cardiac massage was begun. When the chest was opened the heart was found to be flaccid. Isoproterenol, 64 mg, 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

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Table 1. Potassium Values (mEq/l)

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<thead>
<tr>
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<th>Patient 1</th>
<th>Patient 2</th>
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<tbody>
<tr>
<td>Control</td>
<td>4.3</td>
<td>4.5</td>
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<tr>
<td>1 min</td>
<td>5.1</td>
<td>9.5</td>
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<td>2 min</td>
<td>5.6</td>
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<td>4 min</td>
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<tr>
<td>5 min</td>
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<td>6 min</td>
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<td>10.4</td>
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<td>7 min</td>
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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


6. Tobey, R. E.: Personal communication.

