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Effect of Succinylcholine on Plasma Potassium in Children with Cerebral Palsy

STEPHEN F. DIERDORF, M.D.,* WILLIAM L. MCNIECE, M.D.,* CHALAPATHI C. RAO, M.D.,* THOMAS M. WOLFE, M.D.,* GOPAL KRISHNA, M.D.,† LYN J. MEANS, M.D.,* KENNETH A. HASELBY, M.D.†

Succinylcholine-induced hyperkalemia occurs in patients after burn injury, massive trauma, and a variety of neurologic disorders. Hyperkalemia after succinylcholine occurs in patients with spinal cord injury and sporadically in patients with encephalitis, cerebral vascular accidents, Parkinson's disease, and closed head injury.[1–4] Perhaps patients with other types of upper motor neuron lesions, such as cerebral palsy, develop hyperkalemia after succinylcholine. We, therefore, prospectively studied the effect of succinylcholine on venous plasma potassium levels in children with cerebral palsy.

METHODS

Following approval by our Committee on Protection of Human Subjects and informed parental consent, we studied 36 normal patients (control) and 36 patients with cerebral palsy presenting for elective surgery. The cerebral palsy patients had sustained ischemic or hypoxic brain injury and demonstrated either spastic diplegia or spastic quadriplegia. A minimum of 12 months had elapsed from brain injury until the subjects were studied. Each group of 36 patients was divided by age into three groups of 12 patients, each by age: 1 to 5 years, 6 to 10 years, and 11 to 17 years.

Within each age group of 12 patients, six received halothane and nitrous oxide to maintain anesthesia and six received isoflurane and nitrous oxide. Zero time (control) potassium levels were measured just prior to induction of anesthesia. Anesthesia was induced by atropine 10 μg/kg, thiopental 6.0 mg/kg, and succinylcholine 2.0 mg/kg iv. Following controlled ventilation via a mask, the trachea was intubated and administration of the volatile anesthetic initiated (60–90 s after succinylcholine). End-tidal carbon dioxide was maintained between 4.5 and 5.5%. Blood samples for potassium were drawn from an iv catheter using minimal limb compression at 1, 3, 5, and 10 min after succinylcholine. Normal saline was infused during the study period. Potassium levels were measured with a Space-Stat 30® potassium analyzer (Orion Biomedical). The Space-Stat 30® employs an ion-selective electrode for potassium determination. This is a direct potentiometric technique that eliminates the volume displacement error of flame photometry. There may be discrepancies between ion-selective techniques and flame photometry. Comparisons of results from ion-selective electrodes and flame photometry must be done with caution.[5]

Zero time potassium levels, the effects of halothane *versus* isoflurane, and the differences between age groups were analyzed with a three-way analysis of variance (ANOVA). The potassium levels of the cerebral palsy patients were compared with the potassium levels of the control patients at each sampling time with a one-way ANOVA, followed by a least significant difference (LSD) test. The effect of succinylcholine on potassium levels at 1, 3, 5, and 10 min was analyzed with a four-way ANOVA, followed by Dunnett's procedure.[6] A $P < 0.05$ was considered significant.

RESULTS

We observed no difference in potassium levels between the halothane and the isoflurane patients. Therefore, the halothane and isoflurane data were pooled for both the control and cerebral palsy groups. No significant differences in serum potassium between the control and the cerebral palsy patients occurred at time zero or at any other sampling time. The control patients had no significant change in plasma potassium after succinylcholine. In the cerebral palsy group, there was a small significant decrease in plasma potassium at 5 and 10 min after succinylcholine (table 1). The maximum individual potassium increase was 0.6 mEq/l in the controls and 0.4 mEq/l in the cerebral palsy group.

DISCUSSION

Succinylcholine produces a potassium efflux by increasing potassium permeability of the muscle mem-

* Assistant Professor of Anesthesia.
† Associate Professor of Anesthesia.
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Address reprint requests to Dr. Dierdorf.
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TABLE 1. Changes in Plasma Potassium after Succinylcholine

	0 Min	1 Min	3 Min	5 Min	10 Min
Control patients (median age 9 yrs; range 1–15 yrs)					
Postassium (mEq/l)	4.06 ± 0.30	4.03 ± 0.30 NS	3.93 ± 0.23 NS	3.93 ± 0.25 NS	3.98 ± 0.27 NS
Cerebral palsy patients (median age 8 yrs; range 1–20 yrs)					
Potassium (mEq/l)	4.19 ± 0.28	4.17 ± 0.25 NS	4.06 ± 0.17 NS	4.02 ± 0.19 $P < 0.01$	4.04 ± 0.22 $P < 0.05$

Potassium values are mean ± SD. NS is no statistical difference relative to the zero time potassium value.

brane. In normal muscle, the chemosensitive receptor area is confined to the end-plate region. In contrast, denervated muscle has a membrane that is supersensitive to chemical depolarizers (*e.g.*, succinylcholine). Consequently, denervated muscle exhibits an exaggerated potassium efflux after succinylcholine.[7]

Succinylcholine-induced hyperkalemia occurs in patients with some types of upper motor neuron lesions such as high spinal cord transections. There have been sporadic case reports of hyperkalemia after succinylcholine in many other types of upper motor neuron lesions. Prospective studies of the effect of succinylcholine on potassium in patients with upper motor neuron lesions have not been performed previously. Obviously, lower motor neuron lesions would produce denervated muscle. The mechanism by which upper motor neuron lesions increase muscle membrane permeability to potassium has not been elucidated. There is electromyographic evidence that some upper motor neuron lesions also may affect the lower motor neurons and produce a denervation pattern.[8] This could explain why succinylcholine produces hyperkalemia in some patients with upper motor neuron lesions and not others. In our study, the cerebral palsy patients did not demonstrate an increase in plasma potassium after succinylcholine. Perhaps the upper motor neuron lesion of cerebral palsy does not affect the lower motor neurons and does not produce muscle denervation. The time interval between neurologic injury and succinylcholine exposure also may be important. Carter *et al.*[9] demonstrated the risk of hyperkalemia to be greatest at 10–30 days after spinal cord transection. By 60 days after cord transection, hyperkalemia after succinylcholine did not occur. A temporary period of increased muscle sensitivity to succinylcholine may exist, after which sensitivity returns to normal. Most of the patients in this study developed cerebral palsy after birth asphyxia. Therefore, a minimum of 12 months had elapsed between injury and anesthesia.

Consistent with the findings of Keneally and Bush,[10] our control patients did not have an increase in plasma potassium levels after succinylcholine. Yet, in a study by Henning and Bush,[11] potassium levels increased by a mean of 0.4 to 0.45 mEq/l after induction of anesthesia with halothane and succinylcholine. The cerebral palsy patients demonstrated a small but significant decrease in plasma potassium levels at 5 and 10 min after succinylcholine. The control patients showed a similar but not significant trend. Potential causes of the potassium decrease include the effect of thiopental and changes in arterial carbon dioxide tension. Thiopental has been shown to decrease potassium.[12] Although we maintained end-tidal carbon dioxide at 4.5–5.5%, perhaps a decrease in carbon dioxide tension within this range could produce small changes in plasma potassium of the magnitude we observed.[13]

We conclude that succinylcholine does not produce an increase in plasma potassium after a thiopental induction in children with cerebral palsy.

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Tension Subcutaneous Emphysema

ALAN S. TONNESEN, M.D.,* WILLIAM WAGNER, M.D.,† JUDY MACKEY-HARGADINE, M.D.‡

The incidence of barotrauma during controlled ventilation of critically ill patients ranges from less than 1% to more than 40%.[1–3] Pulmonary barotrauma may present as pulmonary interstitial emphysema, pneumothorax, pneumomediastinum, pneumopericardium, retroperitoneal air dissection, pneumoperitoneum, systemic air embolism, and subcutaneous emphysema. While tension pneumothorax, tension pneumomediastinum, and tension pericardium are recognized causes of hemodynamic and pulmonary deterioration, the presence of subcutaneous emphysema in amounts sufficient to cause cardiopulmonary embarrassment is not commonly recognized. We report a patient who suffered severe restriction of ventilation by tense subcutaneous emphysema.

REPORT OF A CASE

A 25-year-old woman suffered a severe closed head injury (Glasgow coma score-4), pneumothorax, and subcutaneous emphysema in a motor vehicle accident. Her trachea had been intubated at the scene of the accident. She was mechanically hyperventilated to decrease intracranial pressure (ICP).

The initial radiologic examination of the chest revealed fractured ribs, a left pneumothorax, and subcutaneous emphysema of the left chest wall. Several hours after admission, a repeat chest roentgenogram showed the development of pneumomediastinum and a decrease in the amount of subcutaneous emphysema, after which the chest tube was repositioned on the left side. Consolidation developed at both lung bases. The left chest tube was removed on the second day, and two subsequent roentgenograms demonstrated the absence of pneumothorax and no change in pneumomediastinum, subcutaneous emphysema, or infiltrates. On the third to sixth day, pneumothorax of varying degrees recurred, despite repeated chest tube insertions. Suction at 25 cmH_2O was applied to all chest tubes at all times.

On the sixth day, tension pneumothorax again became evident. Despite replacement and repeated manipulation of both chest tubes on the left side, the tension pneumothorax persisted. Since air was leaking through chest tube insertion sites, purse string sutures were placed, occluding the leak. The pneumomediastinum began to increase. Subcutaneous emphysema on the right side was noted for the first time and became progressively more severe.

The patient was ventilated with a volume preset ventilator (Emerson) at 12 breaths/min, positive end-expiratory pressure of 7.5 cmH_2O, and a peak inspiratory pressure (PIP), 60 ± 2 cmH_2O. Exhaled tidal volume (Vt), measured at the connection between the endotracheal tube and ventilator circuit, remained constant at 750 ml/breath.

ICP increased from less than 10 to greater than 20 mmHg. Concurrently, jugular venous pressure rose from 20 to 36 mmHg. Pa_{CO_2} increased from 36 mmHg to 48 mmHg; Pa_{O_2} fell from 111 mmHg on an FI_{O_2} 0.80, to 60 mmHg on FI_{O_2} 1.00; ICP rose further to 48 mmHg. The heart rate increased from 105 to 140 bpm, but mean systemic arterial pressure remained stable at 77–83 mmHg.

At this time, the subcutaneous emphysema was found to be extremely tense, with tympany and crepitus extending from the patient's ankles to the mandible and extending down both arms to the wrists. The skin of the chest wall, breasts, and abdominal wall was extremely tense, wrinkle-free, and shiny. The sutures around the chest tubes were removed in order to express some of the subcutaneous air in the hope that chest wall compliance would be improved sufficiently so that ventilation would improve. Removal of the sutures was accompanied by the release of air under obvious pressure with an immediate fall in ICP and jugular venous pressure. PIP fell from 60 to 50 cmH_2O. Pa_{CO_2} decreased from 48 to 40 mmHg. A chest roentgenogram taken immediately after clinical improvement revealed that the pneumothorax had, in fact, increased in size, and a lateral chest film showed that one of the two chest tubes was not within the pleural space. Fiberoptic bronchoscopy revealed copious, thick, white secretions without evidence of tracheobronchial trauma.

* Associate Professor.
† Resident.
‡ Assistant Professor.

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Address reprint requests to Dr. Tonnesen: Department of Anesthesiology, University of Texas Medical School, 6431 Fannin, Room 5.186, Houston, Texas 77030.

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