References

1. Editorial: Are postoperative pulmonary complications preventable? Lancet 2:1079–1080, 1984
2. Dundee JW, Assem ESK, Gaston JM, Keilty SR, Sutton JA, Clarke RSJ, Grainger D: Sensitivity to intravenous anaesthetics: a report of three cases. Br Med J 1:63–65, 1974
3. Evans JM, Keough JAM: Adverse reactions to intravenous anaesthetic induction agents. Br Med J 2:735–736, 1977
4. Adams AP, Pybus DA: Delayed respiratory depression after use of fentanyl during anaesthesia. Br Med J 1:278–279, 1978
5. Sebel PS, Lalor JM, Flynn PJ, Simpson BA: Respiratory depression after alfentanil infusion. Br Med J 289:1581–1582, 1984
6. Becker LD, Paulson BA, Miller RD, Severinghaus JW, Eger EI: Biphasic respiratory depression after fentanyl—droperidol or fentanyl alone used to supplement nitrous oxide anesthesia. ANESTHESIOLOGY 44:291–296, 1976
7. Danielson DA, Porter JB, Dinan BJ, O'Connor PC, Lawson DH, Kellaway GSM, Jick H: Drug monitoring of surgical patients. JAMA 248:1482–1485, 1982
8. Jick H, Miettinen OS, Shapiro S, Lewis GP, Siskind J, Slone D: Comprehensive drug surveillance. JAMA 213:1455–1460, 1970
9. Rothman KJ, Boice JD: Epidemiologic analysis with a programmable calculator. Washington D. C., U. S. Government Printing Office, NIH Publication No. 79-1649, June 1979, p 36
10. Miettinen OS: Estimability and estimation in case-referent studies. Am J Epidemiol 103:226–235, 1976
11. Armitage P: Tests for linear trends in proportions and frequencies. Biometrics 11:375–386, 1955
12. Wightman JAK: A prospective survey of the incidence of postoperative pulmonary complications. Br J Surg 55:85–91, 1968

Anesthesiology
64:272–273, 1986

Failure of Succinylcholine to Alter Plasma Potassium in Children with Myelomeningocoele

STEPHEN F. DIERDORF, M.D.,* WILLIAM L. MCNIECE, M.D.,† CHALAPATHI C. RAO, M.D.,† THOMAS M. WOLFE, M.D.,† LYNDA J. MEANS, M.D.†

Succinylcholine-induced hyperkalemia often occurs in patients after spinal cord injury.[1] Hyperkalemia after succinylcholine occurs inconsistently in patients with encephalitis, cerebral vascular accidents, Parkinson's disease, and closed head injury.[2–4] There are few prospective studies of the response of potassium to succinylcholine in patients with neurologic disease.[5] Because children with myelomeningocoele have both upper motor neuron and lower motor neuron dysfunction and might be susceptible to succinylcholine-induced hyperkalemia, we prospectively studied the effect of succinylcholine on venous plasma potassium levels in children with myelomeningocoele.

Methods

Following approval by our Committee on Protection of Human Subjects and informed parental consent, we studied 24 normal patients (control) and 24 patients with myelomeningocoele presenting for elective surgery. The myelomeningocoele patients had high lumbar or thoracic lesions that produced significant neuromuscular dysfunction. Ages in both groups ranged from 1 to 16 yr (mean of 8.6 yr for control patients and mean of 6.4 yr for myelomeningocoele patients). The children received no preoperative medication. An iv catheter was inserted after local anesthesia. A zero time potassium level was measured after insertion of the iv catheter and prior to the induction of anesthesia. Anesthesia was induced with 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 the administration of nitrous oxide and isoflurane initiated (60 to 90 s after succinylcholine). End-tidal carbon dioxide was maintained between 4.5 and 5.5%. Blood samples for potassium were drawn from the iv catheter using minimal limb compression at 1, 3, 5, and 10 min after succinylcholine. Normal saline was intermittently flushed through the iv catheter during the study. Potassium levels were measured with an IL501® (Instrumentation Laboratory) ion selective potassium analyzer. This is a direct potentiometric technique that avoids the volume displacement error of flame photometry.[6]

The data in each group were analyzed with a one-way analysis of variance (ANOVA) followed by Dunnett's

* Associate Professor of Anesthesia.
† Assistant Professor of Anesthesia.
Received from the Department of Anesthesia, Indiana University School of Medicine, 1120 South Drive, Fesler Hall 204, Indianapolis, IN 46223. Accepted for publication September 11, 1985.
Address reprint requests to Dr. Dierdorf.
Key words: Neuromuscular relaxants: succinylcholine. Ions: potassium. Muscle, skeletal: myelomeningocoele.

TABLE 1. Changes in Plasma Potassium after Succinylcholine

K (x ± SD)	0 Min	1 Min	3 Min	5 Min	10 Min
Control (n = 24)	4.1 ± 0.3	4.1 ± 0.3 NS*	4.0 ± 0.3 NS	4.0 ± 0.3 $P < 0.05$	4.0 ± 0.3 NS
Myelomeningocoele (n = 24)	4.1 ± 0.4	4.1 ± 0.4 NS	4.0 ± 0.4 NS	4.0 ± 0.4 NS	4.2 ± 0.4 NS

* NS = not significant.

procedure.[7] Zero time potassium levels were compared between the two groups with a one-way ANOVA. $P < 0.05$ was considered significant.

RESULTS

There was no statistically significant difference in plasma potassium concentration at zero time between the two groups. The myelomeningocoele patients had no significant change in potassium after succinylcholine. In the control group, there was a small but statistically significant decrease in plasma potassium at 5 min after succinylcholine (table 1). The maximum individual potassium increase was 0.6 mEq/l in both groups.

DISCUSSION

Succinylcholine produces a potassium efflux by increasing potassium permeability of the muscle membrane.[1] In normal muscle, the chemosensitive receptor area is confined to the end-plate region. In contrast, denervated muscle develops an increased number of receptor sites.[8] Denervated muscle consequently exhibits a greater potassium efflux after succinylcholine.

Succinylcholine-induced hyperkalemia occurs in patients with some types of upper motor neuron lesions, such as high spinal cord transection. The mechanism by which upper motor neuron lesions increase muscle membrane permeability has not been determined. Electromyographic evidence suggests that upper motor neuron lesions affect lower motor neuron function and produce denervation.[9] A prospective study in children with cerebral palsy showed no increase in potassium levels after thiopental and succinylcholine.[5]

Children with myelomeningocoele have both upper and lower motor neuron dysfunction. Electromyographic and histologic studies of muscle from children with myelomeningocoele have shown evidence of denervation and abnormal muscle development.[10] Malignant hyperthermia also has been reported in children with myelomeningocoele.[11] Children with myelomeningocoele do not develop hyperkalemia after succinylcholine, despite evidence of neuromuscular dysfunction. This study and our previous study of children with cerebral palsy indicate that children with congenital neurologic lesions do not exhibit the marked hyperkalemic response to succinylcholine observed in patients with acquired neurologic lesions. We speculate that congenital neurologic disorders retard the maturation process of muscle, which may result in the lack of potassium release after succinylcholine. Early fetal denervation might interfere with development of cholinergic muscle receptor sites. The muscle membrane would not, consequently, exhibit an increased sensitivity to succinylcholine.

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

REFERENCES

1. Gronert GA, Theye RA: Pathophysiology of hyperkalemia induced by succinylcholine. ANESTHESIOLOGY 43:89–99, 1975
2. Cowgill DB, Mostello LA, Shapiro HM: Encephalitis and a hyperkalemic response to succinylcholine. ANESTHESIOLOGY 40: 409–411, 1974
3. Gravlee GP: Succinylcholine-induced hyperkalemia in a patient with Parkinson's disease. Anesth Analg 59:444–446, 1980
4. Stevenson PH, Birch AA: Succinylcholine-induced hyperkalemia in a patient with a closed head injury. ANESTHESIOLOGY 51: 89–90, 1979
5. Dierdorf SF, McNiece WL, Rao CC, Wolfe TM, Krishna G, Means LJ, Haselby KA: Effect of succinylcholine on plasma potassium in children with cerebral palsy. ANESTHESIOLOGY 62:88–90, 1985
6. Ladenson JH: Direct potentiometric analysis of sodium and potassium in human plasma: evidence for electrolyte interaction with a nonprotein, protein-associated substance(s). J Lab Clin Med 90:654–665, 1977
7. Steel RGD, Torrie JH: Principles and Procedures of Statistics. New York, McGraw-Hill, 1960
8. Axelsson J, Thesleff S: A study of supersensitivity in denervated mammalian skeletal muscle. J Physiol (Lond) 147:178–193, 1959
9. Krueger KC, Waylonis GW: Hemiplegia: lower motor neuron electromyographic findings. Arch Phys Med Rehabil 54:360–364, 1973
10. Mortier W, VonBernuth H: The neural influence on muscle development in myelomeningocoele: histochemical and electrodiagnostic studies. Dev Med Child Neurol 13(suppl. 25):82–89, 1971
11. Anderson TE, Drummond DS, Breed AL, Taylor CA: Malignant hyperthermia in myelomeningocoele: a previously unreported association. J Pediatr Orthop 1:401–403, 1981