Failure of Succinylcholine to Alter Plasma Potassium in Children with Myelomeningocele

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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 myelomeningocele 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 myelomeningocele.

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 myelomeningocele presenting for elective surgery. The myelomeningocele 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 myelomeningocele 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

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procedure. 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 myelomeningocele 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. 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. 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. A prospective study in children with cerebral palsy showed no increase in potassium levels after thiopental and succinylcholine.

Children with myelomeningocele have both upper and lower motor neuron dysfunction. Electromyographic and histologic studies of muscle from children with myelomeningocele have shown evidence of denervation and abnormal muscle development. Malignant hyperthermia also has been reported in children with myelomeningocele. Children with myelomeningocele 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 myelomeningocele.

**REFERENCES**


**TABLE 1. Changes in Plasma Potassium after Succinylcholine**

<table>
<thead>
<tr>
<th>K (± SD)</th>
<th>0 Min</th>
<th>1 Min</th>
<th>5 Min</th>
<th>5 Min</th>
<th>10 Min</th>
</tr>
</thead>
<tbody>
<tr>
<td>Control</td>
<td>4.1 ± 0.3</td>
<td>4.1 ± 0.3</td>
<td>4.0 ± 0.3</td>
<td>4.0 ± 0.3</td>
<td>4.0 ± 0.3</td>
</tr>
<tr>
<td>(n = 24)</td>
<td></td>
<td>NS*</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Myelomeningocele</td>
<td>4.1 ± 0.4</td>
<td>4.1 ± 0.4</td>
<td>4.0 ± 0.4</td>
<td>4.0 ± 0.4</td>
<td>4.2 ± 0.4</td>
</tr>
<tr>
<td>(n = 24)</td>
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</table>

* NS = not significant.