Title: EFFECT OF SUCCINYLCHOLINE ON PLASMA POTASSIUM IN CHILDREN WITH CEREBRAL Palsy

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Introduction. Succinylcholine induced hyperkalemia occurs after burn injury, massive trauma, and a variety of neurologic disorders. Hyperkalemia after succinylcholine has been well documented in patients after spinal cord injury. Succinylcholine induced hyperkalemia has also been reported in patients with encephalitis, cerebral vascular accidents, and closed head injury. It has been suggested, although never reported, that succinylcholine induced hyperkalemia may occur in children with cerebral palsy. Our extensive clinical experience with cerebral palsy did not support this suggestion.

Methods. Following approval by the Committee on Protection of Human Subjects and informed parental consent, we studied 36 normal patients (control) and 36 patients with cerebral palsy. An intravenous catheter was inserted after local anesthesia. Blood samples for zero time potassium levels (K+) (control) were drawn from the intravenous catheter. Intravenous fluid administered during the study was normal saline. The anesthetic induction sequence consisted of atropine 10 μg/kg, thiopental 6 mg/kg, and succinylcholine 2 mg/kg. After tracheal intubation end-tidal CO2 was maintained between 4.5 and 5.5. Blood samples for potassium were drawn from the intravenous catheter with minimal limb compression at 1, 3, 5 and 10 minutes after succinylcholine. In each group of 36 patients, 18 received halothane-nitrous oxide for maintenance anesthesia and 18 received isoflurane-nitrous oxide. In each group of 18 there were six patients aged 1-5 years, six aged 6-10 years, and six aged 11 years or greater. This grouping permitted comparisons between different anesthetics and different ages in addition to the comparison between normal children and cerebral palsy patients.

A three way analysis of variance (ANOVA) was used to analyze for differences in zero time potassium levels, halothane versus isoflurane, and age groups. Differences between the cerebral palsy and control patients were analyzed with a one way ANOVA followed by a least significant difference (LSD) test. The effect of succinylcholine on potassium was analyzed with a four way ANOVA followed by Dunnnett's test.

Results. There were no statistically significant differences between control and cerebral palsy patients at any sampling times. There were no differences in zero time potassium levels between any of the groups. Within the control group there was no significant change in potassium after succinylcholine. In the cerebral palsy group there was a small but statistically significant decrease in potassium at 5 and 10 minutes after succinylcholine.

<table>
<thead>
<tr>
<th>Time</th>
<th>0min</th>
<th>1min</th>
<th>3min</th>
<th>5min</th>
<th>10min</th>
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<tbody>
<tr>
<td>K+ (mEq/L)</td>
<td>4.06</td>
<td>4.03</td>
<td>3.93</td>
<td>3.92</td>
<td>3.98</td>
</tr>
<tr>
<td>NS</td>
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When potassium levels were expressed as per cent of control, there were no statistically significant differences in potassium between any of the groups with respect to type of anesthesia, age, or control versus cerebral palsy patients.

Discussion. Succinylcholine produces a massive K+ efflux from denervated muscle(1). Succinylcholine induced hyperkalemia has been well documented in some patients with upper motor neuron lesions (e.g. spinal cord injury) and sporadically reported in other types of upper motor neuron lesions. It has been shown that upper motor neuron lesions can adversely affect lower motor neuron function and may produce muscle demervation(2). This may explain why succinylcholine produces hyperkalemia in a variety of upper motor neuron lesions. In our study the cerebral palsy patients did not demonstrate a rise in potassium after succinylcholine. It could be speculated that cerebral palsy does not produce muscle demervation. Time from injury may also affect succinylcholine sensitivity. A temporary period of increased muscle sensitivity may exist after which sensitivity return to normal. Most of our patients developed cerebral palsy after birth asphyxia. A minimum of 12 months had elapsed between injury and succinylcholine. The control patients did not have an increase in potassium after succinylcholine. This is similar to a previous study which measured potassium after thiopental and succinylcholine in children(3).

References.