

PRELIMINARY REPORTS

Lack of a Hyperkalemic Response in Emergency Department Patients Receiving Succinylcholine

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ABSTRACT

Objective: To determine whether serum potassium (K) levels increase significantly following succinylcholine (SCh)-assisted intubation in ED patients.

Methods: A prospective, noncontrolled, consecutive case series design was used to evaluate the change in serum K levels in ED patients who received SCh for emergency intubation. The study was performed at an academic medical center staffed by board-certified emergency physicians. The subjects were 100 consecutive prescreened ED patients with various diagnoses who received SCh for intubation. The eligible subjects had serum K levels determined prior to and 5 minutes after administration of a 1.0–1.5-mg/kg IV dose of SCh. Serum K levels were measured by the ion-selective electrode assay method.

Results: The mean change in serum K levels was -0.04 mmol/L (95% CI -0.14 to 0.06). The maximum increase was 1.10 mmol/L. The serum K level rose in 46 cases, decreased in 46 cases, and was unchanged in eight cases. No instance of SCh-induced cardiac arrest was identified.

Conclusion: Changes in serum K levels following SCh administration in prescreened ED patients were minimal. A hyperkalemic response is uncommon in ED patients who undergo SCh-assisted intubation.

Key words: succinylcholine; hyperkalemia; paralytic agent; endotracheal intubation; emergency department.

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Succinylcholine (SCh)-assisted endotracheal intubation (ETI) has become increasingly more common in ED airway management over the past decade.^{1,2} However, SCh administration may lead to hyperkalemia and cardiovascular collapse in certain clinical situations. Case reports of SCh-induced hyperkalemia in multiple trauma and burn patients were first published in the late

1960s.^{3–6} Since that time it has been recognized that other subsets of patients may be susceptible to SCh-induced hyperkalemia, including patients with neuromuscular diseases, near drowning, and serious infections.^{3,7–13} However, in these cases, SCh was administered weeks to months after the initial acute disease process.

Our English-language MEDLINE literature review found one “acute” case of SCh-induced hyperkalemic arrest in a patient who had severe postpartum hemorrhage and metabolic acidosis.¹⁴ We found no reported case of SCh-induced hyperkalemia in the ED care of patients with multiple trauma, burns, or neurologic or medical diseases. When conducting educational programs on neuromuscular blockers and rapid-sequence intubation at a number of medical centers and conferences, the authors have found that some emergency physicians (EPs) are reluctant to use SCh to assist intubation because of concern over hyperkalemia and cardiovascular collapse. We therefore undertook a study of the change in serum potassium (K) levels in ED

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patients who received SCh for emergency intubation. The null hypothesis for this study was that SCh does not cause a significant elevation of serum K when administered in the acute care of selected ED patients.

METHODS

Study Design

A prospective, noncontrolled, consecutive case series design was used to study serial serum K levels in prescreened patients who received SCh for ED ETI.

Population and Study Site

The setting for the study was a university hospital ED with an annual census of approximately 50,000 patient visits. All patients requiring ED ETI for whom the treating physician elected to use SCh to facilitate the intubation were eligible for the study. Those patients who had multiple trauma, head injury, burns, and neuromuscular diseases whose disease processes were >24 hours old were ineligible for the use of SCh and study enrollment. Additional exclusion criteria are noted in Table 1. The protocol was reviewed by the institutional review board, and informed consent was waived. The premise for this decision was that it was necessary and reasonable for a limited period of time to determine post-SCh K levels in all ED patients receiving SCh to ensure that post-SCh hyperkalemia was not occurring.

Experimental Protocol

A standard dose of 1.0–1.5 mg/kg SCh was given at the discretion of the attending EP. A second dose was administered if needed, but blood sampling was done only after the first dose. Premedication with vecuronium, a nondepolarizing neuromuscular blocker, to prevent SCh-induced fasciculations was given at the discretion of the attending EP. Some subjects also received atropine or lidocaine as premedication. All attending EPs had completed an educational program on the use of neuromuscular blockers prior to this study, and were aware of the study exclusion criteria.

Measurements

Serum was drawn for determination of K level within 10 minutes of SCh administration and at 5 minutes post-dose by separate venous or arterial punctures. Five minutes was selected as the time for post-SCh K measurement based on previous studies that found the maximal increase in K to occur 3 to 5 minutes after SCh administration.^{3,8,11,12}

All patients had cardiac monitoring and continuous pulse oximetry before, during, and for at least one hour

TABLE 1 Patient Exclusion Criteria

- Age <2 years
- Presentation with severe trauma, burns, closed head injury, ruptured cerebral aneurysm, neuromuscular disease, intra-abdominal infection, or near drowning where the disease process is >24 hours in duration
- Presentation with upper-airway obstruction or acute epiglottitis, or a condition for which cricothyrotomy would be difficult or impossible
- Penetrating eye injuries or glaucoma
- History of malignant hyperthermia or pseudocholinesterase deficiency
- Known hyperkalemia
- Hemolyzed blood samples

after SCh administration. A nurse or physician member of the ED team was responsible for continually watching for and recording dysrhythmias from the time of SCh administration until the 5-minute post-SCh blood sample was obtained.

Blood samples were analyzed in the hospital clinical laboratory by the ion-selective electrode assay method (Dupont Dimension AR). Hemolysis was noted and recorded by the laboratory technician. Subjects who had more than slight hemolysis on either pre- or post-SCh samples were excluded from the study.

Data Analysis

Descriptive statistics were used to analyze the data. Means \pm SDs and 95% CIs were calculated. Student's two-tailed t-test was used to compare the subjects who received vecuronium premedication and those who did not. The sample size of 100 subjects, assuming an SD of 0.5 mmol/L, with α set at 0.05, provides a power of 0.95 to detect a ± 0.25 -mmol/L change in serum K levels.

RESULTS

One hundred seven subjects were enrolled in the study from June 1991 to January 1993. Six subjects were excluded due to hemolyzed pre- or post-SCh blood samples. One subject was excluded because the pre-SCh blood sample was obtained hours before SCh administration. The remaining 100 subjects constitute the study group. Violation of the clinical exclusion criteria was discovered in one case. A 71-year-old man who had suffered a head injury and traumatic aortic dissection in a motor vehicle crash was enrolled in the study. Following SCh-assisted intubation it was learned that the patient was a wheelchair-bound paraplegic. The pre-SCh K level was 4.6 mmol/L, and the post-SCh K level was 4.1 mmol/L in this subject. Data for this subject were included in the study.

The primary diagnosis and indication for SCh-assisted intubation were grouped into five major categories: trauma (including burn and traumatic brain injury), respiratory failure, neurologic disease, drug overdose, and medical/cardiac disease. The distribution and specific diagnoses of the subjects in these groups are shown in Table 2. The overall mean age was 43 ± 21 years. Five patients were <13 years old, and nine were >70 years old.

Estimated weight was documented for 90 of the 100 patients. The mean initial dosage of SCh was 1.1 mg/kg (range 0.8 to 1.85 mg/kg). Three subjects received a second dose of SCh. For these patients, blood for determination of the post-SCh K level was drawn after the first SCh dose. Overall, the mean pre-SCh K level was 3.9 ± 0.5 mmol/L, and the mean post-SCh K level was 3.9 ± 0.6 mmol/L. The mean change in serum K levels was -0.04 ± 0.49 mmol/L (95% CI -0.14 to 0.06) (Fig. 1).

The serum K level rose in 46 patients following SCh administration, decreased in 46 patients, and remained the same in eight patients. The largest increase in serum K level was 1.10 mmol/L. This occurred in a patient who had facial and head trauma; the change was from 3.4 mmol/L to 4.5 mmol/L. The post-SCh K level was >5 mmol/L in only two patients. In the first case, a patient with status epilepticus had an increase in serum K level from 4.6 to 5.6 mmol/L following SCh administration. Slight hemolysis was noted on the post-SCh specimen. In the second case, an asthmatic patient with respiratory arrest had an increase in serum K level from 4.4 to 5.1 mmol/L following SCh administration. Slight hemolysis was noted in a pre-SCh sample in one case (the pre-SCh K level was 4.2 mmol/L and the post-SCh K level was 3.6 mmol/L). No instance of post-SCh asystole, ventricular tachycardia, ventricular fibrillation, or cardiac arrest was identified for these 100 subjects.

Documentation of the use of vecuronium prior to SCh administration could be determined for 85 subjects. Only 25 of these 85 subjects had received a standard defasciculating dose of vecuronium prior to SCh administration. The distribution of diagnoses for these subjects was similar to that for the subjects who had not received vecuronium. The change in K level in the vecuronium premedication group was -0.036 ± 0.24 mmol/L, compared with -0.051 ± 0.24 mmol/L for the 60 subjects who were documented as not receiving vecuronium ($p = 0.79$).

DISCUSSION

Succinylcholine is routinely used in many EDs for rapid-sequence intubation. Our study is the first to assess the change in serum K level that occurs when ED patients receive SCh for a wide variety of indications. The results

■ **TABLE 2** Diagnosis and/or Indication for Succinylcholine-assisted Intubation ($n = 100$)

| Diagnosis | No. Subjects |
|---|--------------|
| Trauma ($n = 48$) | |
| Head injury | 24 |
| Multiple trauma | 13 |
| Facial fractures | 4 |
| Burn/smoke inhalation | 2 |
| Aortic dissection | 1 |
| Gunshot wound, face | 1 |
| Stab wound, chest | 1 |
| Cervical spine fracture | 1 |
| Neurologic ($n = 18$) | |
| Intracerebral or subarachnoid hemorrhage | 8 |
| Seizure | 5 |
| Cerebrovascular accident | 4 |
| Posterior fossa abscess | 1 |
| Overdose ($n = 15$) | |
| Unknown drug(s) | 9 |
| Benzodiazepine | 2 |
| Cyclic antidepressant | 1 |
| Cyclic antidepressant/lithium | 1 |
| Caffeine | 1 |
| Carbamazepine | 1 |
| Medical/Cardiovascular ($n = 13$) | |
| Congestive heart failure | 5 |
| Gastrointestinal hemorrhage | 5 |
| Acute myocardial infarction | 1 |
| Aortic dissection | 1 |
| Abdominal aortic aneurysm | 1 |
| Respiratory ($n = 6$) | |
| Pneumonia | 2 |
| Asthma | 2 |
| Respiratory arrest | 2 |

of this study suggest that SCh-assisted intubation in selected ED patients with acute illnesses does not produce significant changes in serum K level. The average change in serum K level was negligible, and no case of clinically important hyperkalemia was detected. It should be emphasized that the patients in this study were pre-screened. The attending EPs received an educational program on the use of SCh prior to the study. This educational program stressed the potential danger of using SCh for patients with multiple trauma, head injury, burns, or neuromuscular diseases when the disease process was >24 hours old.

Numerous cases of SCh-induced hyperkalemic cardiac arrest have been reported in the medical literature. Most of these cases were reported in the late 1960s and 1970s.³⁻¹² Since then, fewer cases have been reported, presumably because of measures to prevent SCh-induced hyperkalemia in high-risk patients. Recently, a

number of cases of SCh-induced hyperkalemic arrest have been reported during induction of anesthesia in children with previously undiagnosed myopathies or clinically occult muscular dystrophy.¹⁵⁻¹⁸ This has led some anesthesiologists to question the use of SCh for elective pediatric anesthesia.^{19,20} To date, similar cases have not been reported among ED patients.

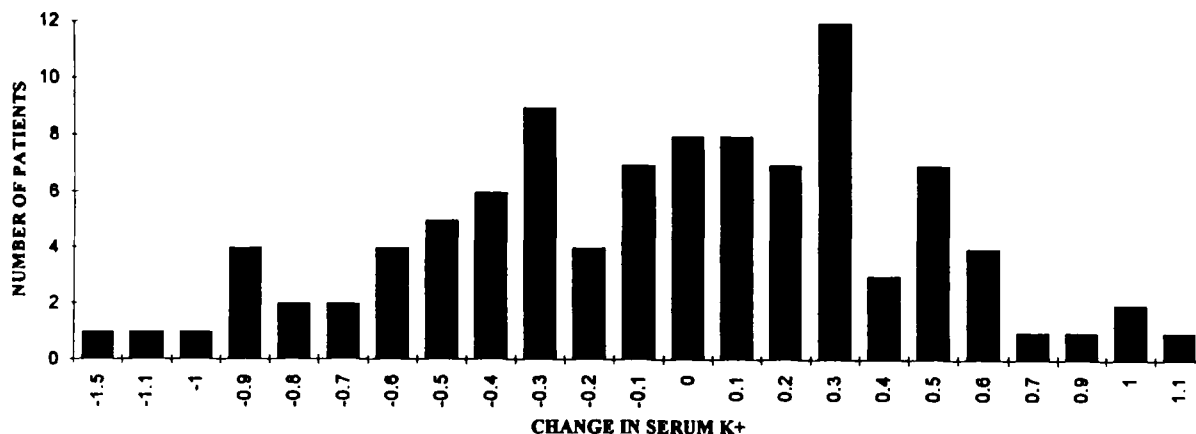
Some data are available describing the "normal" changes in serum K level in patients who receive SCh. Schaner et al.⁵ and Khan and Khan¹¹ found the mean increase in serum K level following SCh administration in patients undergoing elective surgery to be <0.4 mmol/L. Bourke found that while serum K levels, for which blood was drawn 2 minutes after SCh administration in 70 American Society of Anesthesiologists (ASA) class 1 or 2 patients, changed significantly from baseline, the magnitude was not clinically important (i.e., from 3.97 ± 0.07 to 4.18 ± 0.10 mmol/L).²¹ Dronen et al. found that 5-minute post-SCh serum K levels in 12 overdose patients were unchanged in 50% of cases, higher in 33%, and lower in 17%, with an average rise in serum K level of 0.6 mmol/L.²²

It is not clear whether SCh use is less safe for patients with severe shock or acidosis. Our study did not investigate shock and acidosis, although it is likely, on reviewing diagnoses, that several of our subjects had shock and/or acidosis. Succinylcholine is routinely used in the operating room for intubation in acute multiple trauma patients who have shock and acidosis, and we could find no case report of SCh-induced hyperkalemia in that setting. The only case report of SCh-induced hyperkalemic arrest in an acutely ill patient was in a 42-year-old woman who had circulatory collapse three hours after an elective cesarean section.¹⁴ The patient had severe hemorrhagic shock from a uterine laceration, with a hematocrit of 19% and a serum bicarbonate concentration of 5 mmol/L. Antognini and Gronert, using

a rabbit model, found that hemorrhagic shock with profound metabolic acidosis led to hyperkalemia (mean K level 7.0 ± 1.8 mmol/L) prior to administration of SCh, with a further increase following SCh administration.²³ However, the largest case series in humans with "acute massive muscle trauma" from battlefield injuries that were less than two hours old found only one case of hyperkalemia (without cardiac arrest) among 21 patients. In this patient, the K level slowly rose to a maximum of 6.2 mmol/L 20 minutes after SCh administration, and the authors were not certain whether SCh caused the hyperkalemia.²⁴ The presence of severe hemorrhagic shock or acidosis was not documented in this study. Further human data for the subset of patients who are in shock or acidotic who receive SCh are needed to determine whether SCh is contraindicated in this group.

LIMITATIONS AND FUTURE QUESTIONS

Some limitations are present in our study. It is possible that by obtaining serum K samples at 5 minutes post-SCh administration, transient elevation of serum K that occurred between 1 and 5 minutes could have been missed. Most previous studies that have examined the timing of hyperkalemia following SCh administration have found the maximal increase to occur at 4 to 5 minutes post-SCh administration.^{8,11,12} The exception to this is a study by Iwatsuki et al. of patients who had ruptured cerebral aneurysms and underwent surgery (days to weeks later) for repair. In two of the 22 patients, the serum K level rose markedly (to 10.0 and 9.2 mmol/L) following SCh administration. In both of these patients, serum K levels were highest at 1 minute post-SCh administration and had decreased to 5.4 mmol/L and 6.3 mmol/L by 5 minutes post-SCh administra-



■ FIGURE 1. Change in serum potassium (K⁺) level (mmol/L) in 100 patients following succinylcholine administration.

tion.²⁵ In our study, no electrocardiographic evidence of hyperkalemia was observed in the first 5 minutes following SCh administration, making it unlikely that clinically significant hyperkalemia occurred during this period.

A confounding factor in this study was the failure to control resuscitation medications prior to SCh use. Nondepolarizing neuromuscular blockers were used for approximately a third of the subjects, with no significant effect on the change in K level, when compared with the subjects who did not receive this premedication. The attending EPs elected to use vecuronium to prevent fasciculations on a case-by-case basis. It is possible that a hyperkalemic response from SCh was attenuated or prevented in some subjects by prior use of a nondepolarizing agent. Atropine was routinely used for children, and lidocaine was used for some patients who had traumatic brain injury. Both of these agents, theoretically, could affect hyperkalemic dysrhythmias.

Our study had sufficient power to detect a significant change in serum K levels following SCh administration. To date this is the largest study of ED patients that documents K levels following SCh administration. However, given the low incidence of hyperkalemia among ED patients who receive SCh, a sample size of 100 is not sufficient for determining the true risk of hyperkalemia or subgroups at higher risk. Further, while it may be reasonable to assume that other ED patient populations would have similar K responses to SCh, it must be emphasized that the subjects in our study were prescreened. Hyperkalemia induced by SCh may be seen more frequently when a patient selection process is not used.

CONCLUSION

Succinylcholine is widely used for rapid-sequence intubation in ED patients. Average changes in serum K levels following SCh administration in prescreened ED patients were minimal. No instance of hyperkalemic cardiac arrest occurred. These findings suggest that a hyperkalemic response is uncommon in emergency patients who require SCh-assisted intubation and when proper patient selection is used.

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