# *Serum Potassium Changes after Succinylcholine in Patients with Acute Massive Muscle Trauma*

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The effect of intravenous succinylcholine on serum potassium was studied in 21 combat casualties receiving their first anesthesia within three hours of sustaining massive muscle trauma. In 20 of these patients, there were no statistically or physiologically significant increases in serum potassium. In one patient, an increase in serum potassium was apparent ten minutes after injection of succinylcholine, progressing to a peak level of 6.2 mEq/l, 20 minutes after injection. It is uncertain whether succinylcholine caused the hyperkalemia in this patient. (Key words: Succinylcholine; Hyperkalemia; Muscle injury.)

MASSIVE HYPERKALEMIA after administration of succinylcholine to a variety of patients, including burned patients,[1] patients with neurologic lesions,[2-4] and patients with sepsis in large soft-tissue wounds,[5] has been reported. In each of these groups, hyperkalemic cardiac arrest has occurred soon after various doses of succinylcholine. In a study of massively traumatized, septic, cachectic battle casualties, Mazze *et al.*[5] found increases of potassium to 9.8 mEq/l, with concomitant cardiac arrests, after single doses of succinylcholine. Patients were studied as early as ten days after wounding, and several developed sequentially higher elevations in potassium as the elapsed time from wounding to administration of succinylcholine increased. On the basis of this evidence and the lack of reports of cardiac arrest in patients receiving succinylcholine during the first anesthesia after massive wounding, they postulated a period of maximum vulnerability beginning in the third week after wounding. However, we are aware of no objective data concerning the effects on serum potassium of succinylcholine given as part of the first anesthesia after acute, massive muscle trauma. The present study was done to establish whether succinylcholine-induced hyperkalemia is a hazard in the first few hours after massive muscle trauma.

## Clinical Material and Methods

Twenty-one battle casualities, each with acute massive muscle injury involving at least three extremities, were chosen for the study. Each patient had multiple wounds extending deep into muscle, and in many patients large areas of muscle had been destroyed. All patients had been wounded less than two hours before arrival at the hospital. The average age was 21 years. All were patients admitted to the Naval Support Activity Hospital, Da Nang, Vietnam, after sustaining multiple fragment wounds from booby-trap explosions. Patients with intra-abdominal, intrathoracic, and intracranial injuries in addition to massive muscle trauma were also included in the study group. Upon arrival at the hospital, each patient was given Ringer's lactate solution, followed by type-specific crossmatched whole blood, until systolic blood pressure was at least 90 mm Hg. In some cases, it was necessary to place tourniquets on extremities to control hemorrhaging from the wounds. However, not more than one tourniquet was in place at

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TABLE 1. Arterial Potassium Values (mEq/l) of Control Patients

| Patient | Pre-succinyl-choline | Minutes after Injection of Succinylcholine | | | | | | | | | Highest $K^+$ | Maximum Change (mEq/l) |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | 1 | 2 | 3 | 4 | 5 | 7½ | 10 | 15 | 20 | | |
| 1 | 3.6 | 3.6 | 4.0 | 3.7 | 3.4 | 3.4 | 3.5 | 3.6 | 3.7 | 4.0 | 4.0 | +0.4 |
| 2 | 3.5 | 3.7 | 3.7 | 3.6 | 3.7 | 3.7 | 3.5 | 3.4 | 3.7 | 3.9 | 3.9 | +0.4 |
| 3 | 3.3 | 3.5 | — | 3.4 | 3.2 | 3.2 | 3.1 | 3.1 | 3.7 | 3.4 | 3.7 | +0.4 |
| 4 | 3.4 | 3.3 | 3.5 | 3.3 | 3.4 | 3.3 | 3.4 | 3.5 | 3.8 | 3.4 | 3.8 | +0.4 |
| 5 | 4.1 | 4.0 | 4.1 | 4.2 | 4.1 | 4.3 | 4.1 | 4.1 | 4.4 | 4.4 | 4.4 | +0.3 |
| 6 | 3.3 | 3.5 | 3.4 | 3.4 | 3.3 | 3.2 | 3.2 | 3.2 | 3.3 | 3.5 | 3.5 | +0.2 |
| 7 | 4.3 | 4.2 | 4.3 | 4.1 | 3.8 | 3.6 | 3.5 | 3.7 | 3.9 | 4.2 | 4.2 | −0.6 |
| 8 | 3.5 | 3.6 | 3.6 | 3.5 | 3.5 | 3.3 | 3.2 | 3.4 | 3.5 | 3.4 | 3.6 | +0.1 |
| 9 | 3.9 | 3.9 | 3.6 | 3.4 | 3.3 | 3.4 | 3.4 | 3.5 | 3.5 | 3.6 | 3.9 | −0.5 |
| 10 | 4.1 | 4.2 | 4.3 | 4.1 | 4.0 | 4.2 | 4.0 | 3.9 | 4.1 | 4.2 | 4.3 | +0.2 |
| 11 | 3.8 | 3.8 | 3.7 | 3.7 | 3.7 | 3.7 | 3.8 | 3.8 | 4.0 | 3.8 | 4.0 | +0.2 |
| 12 | 3.7 | 3.7 | 3.8 | 3.7 | 3.7 | 3.7 | 3.6 | 3.7 | 3.8 | 3.8 | 3.8 | +0.1 |
| 13 | 4.0 | 4.1 | 3.9 | 4.0 | 3.9 | 3.8 | 3.8 | 3.8 | 3.9 | 4.2 | 4.2 | +0.2 |
| 14 | 2.8 | 2.8 | 2.7 | 2.8 | 2.7 | 2.6 | 2.6 | 2.6 | 2.7 | 3.1 | 3.1 | +0.3 |
| Mean | 3.7 | 3.7 | 3.7 | 3.6 | 3.6 | 3.5 | 3.5 | 3.5 | 3.7 | 3.8 | | |
| SD | 0.40 | 0.38 | 0.42 | 0.38 | 0.37 | 0.43 | 0.39 | 0.38 | 0.40 | 0.39 | | |
| *P* | — | 1 | 1 | >0.5 | >0.3 | >0.2 | >0.1 | >0.1 | >0.1 | >0.5 | | |

the time of induction of anesthesia or during the sampling period. A no. 18 Argyle plastic cannula was placed percutaneously in a radial or brachial artery. Baseline samples were drawn immediately before induction for determination of serum potassium, sodium, *p*H, $P_{CO_2}$, and $P_{O_2}$. Anesthesia was induced in all patients within three hours of injury. Atro-

TABLE 2. Arterial Potassium Values (mEq/l) of Trauma Patients

| Patient | Pre-succinyl-choline | Minutes after Injection of Succinylcholine | | | | | | | | | Highest $K^+$ | Maximum Change (mEq/l) |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | 1 | 2 | 3 | 4 | 5 | 7½ | 10 | 15 | 20 | | |
| 1 | 5.3 | 5.0 | 4.7 | 4.6 | 4.4 | 4.5 | 4.9 | 4.5 | 4.8 | 5.0 | 5.0 | −0.3 |
| 2 | 4.4 | 4.0 | 4.3 | 3.9 | 4.0 | 3.8 | 3.9 | 4.0 | 4.0 | 4.2 | 4.3 | −0.5 |
| 3 | 3.5 | 3.4 | 3.3 | 3.2 | 3.3 | 3.1 | 3.5 | 3.5 | 3.4 | 3.4 | 3.5 | −0.4 |
| 4 | 3.2 | 3.0 | 2.8 | 2.8 | 2.8 | 2.8 | 2.7 | 2.9 | 2.8 | 3.2 | 2.8 | −0.4 |
| 5 | 4.0 | 4.0 | 4.0 | 4.0 | 4.0 | 3.6 | 3.5 | 3.5 | 3.9 | 3.8 | 4.0 | −0.5 |
| 6 | 3.6 | 3.8 | 3.7 | 3.7 | 3.6 | 3.7 | 3.6 | 3.3 | 4.4 | 4.3 | 4.4 | −0.8 |
| 7 | 4.2 | 4.2 | 4.3 | 4.3 | 4.1 | 4.2 | 4.2 | 4.3 | 4.5 | 4.8 | 4.8 | +0.6 |
| 8 | 3.4 | 3.0 | 3.0 | 2.9 | 2.9 | 2.8 | 2.8 | 2.7 | 2.9 | 3.2 | 3.2 | −0.7 |
| 9 | 3.5 | 3.8 | 3.7 | 3.6 | 3.2 | 3.3 | 3.2 | 3.4 | 3.7 | 4.2 | 4.2 | +0.7 |
| 10 | 5.0 | 4.9 | 5.2 | 5.1 | 4.9 | 5.0 | 5.2 | 5.7 | 5.8 | 6.2 | 6.2 | +1.2 |
| 11 | 4.4 | 4.0 | 4.3 | 3.9 | 4.0 | 3.8 | 3.9 | 4.0 | 4.0 | 4.2 | 4.3 | −0.5 |
| 12 | 3.5 | 3.5 | 3.3 | 3.2 | 3.1 | 3.0 | 3.0 | 2.9 | 3.1 | 3.3 | 3.5 | −0.6 |
| 13 | 3.8 | 3.9 | 4.0 | 3.9 | 3.9 | 3.9 | 4.0 | 3.7 | 4.0 | 4.2 | 4.2 | +0.4 |
| 14 | 3.8 | 3.9 | 3.8 | 3.7 | 3.7 | 3.7 | 3.9 | 4.0 | 3.8 | 4.3 | 4.3 | +0.5 |
| 15 | 3.2 | 3.1 | 2.9 | 2.9 | 2.8 | 2.8 | 2.8 | 2.9 | 3.1 | 3.4 | 3.4 | +0.2 |
| 16 | 3.1 | 3.2 | 3.2 | 3.1 | 3.0 | 3.0 | 3.0 | 3.1 | 3.1 | 3.1 | 3.2 | +0.1 |
| 17 | 3.6 | 3.6 | 3.7 | 3.4 | 3.3 | 3.2 | 3.2 | 3.3 | 3.8 | 3.7 | 3.8 | +0.2 |
| 18 | 3.3 | 3.5 | 3.6 | 3.4 | 3.6 | 4.0 | 4.0 | 3.6 | 4.0 | 3.7 | 4.0 | +0.7 |
| 19 | 3.2 | 3.1 | 3.1 | 3.1 | 3.1 | 3.2 | 3.3 | 3.3 | 3.6 | 3.8 | 3.8 | +0.6 |
| 20 | 3.9 | 3.9 | 3.8 | 3.7 | 3.9 | 4.2 | 4.2 | 4.2 | 4.2 | 4.4 | 4.4 | +0.5 |
| 21 | 3.2 | 3.2 | 3.2 | 3.5 | 3.5 | 3.4 | 3.5 | 3.5 | 3.7 | 4.0 | 4.0 | +0.8 |
| Mean | 3.7 | 3.7 | 3.7 | 3.6 | 3.5 | 3.6 | 3.6 | 3.6 | 3.9 | 4.0 | | |
| SD | 0.59 | 0.55 | 0.61 | 0.58 | 0.55 | 0.60 | 0.66 | 0.68 | 0.70 | 0.73 | | |
| *P* | — | 1 | 1 | >0.5 | >0.2 | >0.5 | >0.6 | >0.6 | >0.3 | >0.1 | | |

TABLE 3. Statistical Analysis of Differences between the Mean Serum Potassium Concentrations after Succinylcholine in the Control and Massively Traumatized Groups*

| Minutes after Succinylcholine | $t$ | $P$ |
|---|---|---|
| 1 | 0.0000 | 1 |
| 2 | 0.0000 | 1 |
| 3 | 0.0000 | 1 |
| 4 | 0.0000 | 1 |
| 5 | 0.5385 | 0.5 |
| 7½ | 0.5109 | 0.6 |
| 10 | 0.5000 | 0.6 |
| 15 | 0.9694 | 0.3 |
| 20 | 0.9367 | 0.3 |

* A $t$ test weighted for the difference between sample sizes of the control and massively traumatized groups was used in the comparison. The large P values indicate that there was no statistically significant difference between the mean post-succinylcholine serum potassium in the control group and the mean post-succinylcholine serum potassium in the massively-traumatized group.

pine, 0.4–0.6 mg, was given intravenously. Administration of 100 per cent oxygen for two to three minutes preceded induction with thiopental (Pentothal) (average dose 225 mg, range 125–250 mg), which was followed by 80 mg succinylcholine (Anectine) for intubation. Sellick's maneuver was used when the patient was likely to have a full stomach. The trachea was intubated and an esophageal stethoscope placed. Ventilation was controlled manually. Serial arterial blood samples drawn 1, 2, 3, 4, 5, 7½, 10, 15, and 20 minutes after injection of succinylcholine were analyzed for serum potassium and sodium on an Instrumentation Laboratories flame photometer, and for

TABLE 4. Maximum Postinduction *p*H Changes and Corresponding Serum Potassium Changes*

| Direction of Maximum *p*H Change | Potassium Increased | Potassium Decreased | No Change |
|---|---|---|---|
| *p*H decreased (15 patients) | 6 | 7 | 2 |
| *p*H increased (7 patients) | 3 | 4 | 0 |

* In one patient, the maximum increase in *p*H equalled the maximum decrease. Therefore, 2 maximum *p*H changes are recorded for this patient, making a total of 22 changes for the 21 study patients.

*p*H, $P_{CO_2}$, and $P_{O_2}$ using an Instrumentation Laboratories blood-gas analyzer. Care was taken to avoid hemolysis of samples. Blood, Ringer's lactate solution, and sodium ampicillin were administered intravenously during the induction and sampling period, but potassium supplements and potassium penicillin were excluded from the solutions infused. Neither *d*-tubocurarine nor gallamine was given before injection of succinylcholine, in order to avoid possible masking of succinylcholine-induced potassium changes.[7] Anesthesia was maintained with either nitrous oxide–oxygen–halothane (Fluothane) or nitrous oxide–oxygen–methoxyflurane (Penthrane). Blood pressures were taken by the Riva-Rocci method. The average preinduction blood pressure was 126/76 mm Hg. Average systolic pressures after induction were: 5 minutes, 115 mm Hg; 10 minutes, 102 mm Hg; 15 minutes, 95 mm Hg; 20 minutes, 85 mm Hg. Diastolic pressures were not always recorded during induction. Surgical preparation with povidone-iodine (Betadine) was carried out during the sampling period.

A control group of 14 patients with small wounds not involving significant muscle masses was treated identically. The average dose of thiopental in this group was 250 mg (range 125–500 mg). The average preinduction blood pressure was 125/80 mm Hg. There was no clinically-significant change in blood pressure during the 20-minute study period.

In the traumatized group, *p*H changed significantly after induction, due to respiratory and metabolic variations. The range of all post-induction *p*H values was 7.10–7.59. Moore[6] has stated that serum potassium changes follow changes in *p*H. It seemed that *p*H changes thus might have masked or accentuated potassium changes resulting from succinylcholine. Therefore, before attempting to correlate potassium changes with succinylcholine administration, we felt it was necessary to determine whether changes in *p*H predictably influenced serum potassium. The largest change from preinduction *p*H which occurred in each patient at any time during the 20-minute sampling period was calculated. The serum potassium value for the period of greatest *p*H change was then compared with the preinduction potassium value. Table 4 shows this comparison. We concluded that minute-to-minute

changes in *p*H do not correlate with predictable changes in serum potassium. Because of the absence of any predictable change in potassium with change in *p*H, we believe that *p*H changes did not mask or accentuate any changes in serum potassium due to succinylcholine.

## Results

Serum potassium values for the control group are shown in table 1. The mean value before succinylcholine was 3.7 mEq/l. The highest single value after succinylcholine was 4.4 mEq/l, and the largest increase from preinduction values was only 0.4 mEq/l. The mean value at any of the intervals after succinylcholine was not significantly different from the preinduction mean, as determined by *t* test. We were unable to detect the small but significant increases in normal patients reported by Mazze and Houston.[5]

Table 2 shows the potassium values of the massively-traumatized patients. The mean preinduction potassium concentration in this group was 3.7 mEq/l (SD ± 0.60). The means at all intervals after administration of succinylcholine were compared with the preinduction mean. There was no statistical difference. The mean potassium value at each interval in the control group was compared with the mean potassium value for the corresponding interval in the traumatized group. Differences were not significant (table 3).

Only two of the traumatized patients had serum potassium values above 5 mEq/l at any time during the study (table 2). Patient 1 had a high preinduction level of 5.3 mEq/l. His serum potassium decreased after succinylcholine, reaching a low of 4.4 mEq/l four minutes after injection, and remained below the preinduction level throughout the entire sampling period. Patient 10 had a preinduction level of 5 mEq/l. For the first 7.5 minutes after succinylcholine was given, serum potassium stayed essentially unchanged. At ten minutes, however, it had reached 5.7 mEq/l. After this sample was drawn, brisk bleeding from the right leg occurred, and the tourniquet on this extremity was inflated. There was no artificial interruption of circulation to the macerated left arm or to the injured left leg. At 15 minutes, serum potassium was 5.8 mEq/l. After the 15-minute sample was drawn, the tourniquet on the right leg was released. The potassium concentration in the 20-minute sample was 6.2 mEq/l. An irregular pulse was noted during induction, but became regular as anesthesia progressed. No antiarrhythmic drugs were employed.

## Discussion

The results of this study suggest that clinically-significant hyperkalemia may occur occasionally after succinylcholine is administered to patients with acute, massive muscle trauma. Although the hyperkalemia in Patient 10 could have resulted from rapid transfusion of bank blood or from an unsuspected hemolytic transfusion reaction, it is impossible to rule out succinylcholine as the cause. Further studies are necessary. Meanwhile, it seems wise to consider the possible role of hyperkalemia in massively-traumatized patients who develop serious arrhythmias or cardiac arrest after receiving succinylcholine in the first few hours after injury.

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