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Changes in Total Serum Ca^{++}, Na^{+}, and K^{+} with Administration of Succinylcholine

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In the 1950's, succinylcholine was shown to increase serum potassium. The cause is mechanical damage to muscle cells secondary to asynchronous depolarizations.[1–3] Although the changes are usually small, they can be greater in disease states such as burns, trauma, uremia, and neuromuscular disorders. We have considered the possibility that clinical doses of succinylcholine may cause changes in other serum electrolytes.

MATERIALS AND METHODS

Seventy randomly selected, informed surgical patients, ranging in age from 21 to 50 years, all of ASA physical status I or II, entered the operating room fasting and premedicated one hour previously with either morphine, 10 mg, or pentobarbital, 100 mg, as well as atropine 0.4 mg, im. In addition to routine monitoring and peripheral iv cannulation, for venous blood sampling a 19-gauge scalp vein needle was placed in the arm opposite the peripheral iv cannula.

Subjects were divided into seven groups of ten subjects each. In each group, five subjects had morphine premedication and five, pentobarbital premedication. A control sample of venous blood, Sample 1, was withdrawn from each patient immediately prior to induction of anesthesia. The induction techniques are indicated in table 1. Pretreatment with nondepolarizing muscle relaxants was administered 4 minutes before induction of anesthesia. Inhalational anesthetic inductions, Groups I–III, were effected with enflurane and oxygen:nitrous oxide 1:1. Succinylcholine, 100 mg, iv, was administered after loss of lid reflex. Thiopental inductions, Groups III–VI, were accomplished with 6 mg/kg thiopental and succinylcholine, 100 mg, iv, in rapid sequence. Group VII received only thiopental. A second sample, Sample 2, was withdrawn 2 minutes after thiopental and/or succinylcholine administration.

During the study period, only the above-mentioned drugs were administered,[4] and ventilation was controlled as necessary to maintain near-normal minute ventilation.

Blood samples (10 ml each) were inspected for hemolysis, then centrifuged at 3,000 rpm for 10 minutes. Duplicate serum sodium (Na^{+}), potassium (K^{+}) and calcium (Ca^{++}) determinations were obtained for all samples. Na^{+} and K^{+} were measured with an IL Flame photometer, model 143, (accurate to ±0.3 per cent) and Ca^{++} was measured by the cresol–complexone method (accurate to ±.5 per cent) using a dual AutoAnalyzer.

RESULTS

Concentrations of K^{+}, Na^{+}, and Ca^{++} in Samples 1 and 2, Groups I–VII, are shown in tables 2, 3, and 4, respectively. All Sample 1 values fell within the normal range. Analysis of variance on control samples did not demonstrate any difference among the groups for the ions under consideration. Calcium ion concentration decreased in each group, I–VI (table 4). Analysis of variance showed no effect of either pretreatment (*i.e.*, *d*-tubocurarine, pancuronium, or nothing) or

TABLE 1. Induction Techniques

Induction	Pretreatment		
	None	*d*Tc, 3 mg	Pancuronium, 1 mg
Enflurane + succinylcholine	I	II	III
Thiopental + succinylcholine	IV	V	VI

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TABLE 2. Changes in Serum Potassium (K^+) (Mean ± SEM)

Group	Potassium (mEq/l)			Significance
	Sample 1 (Control)	Sample 2	Δ	
I	3.97 ± 0.07	4.18 ± 0.10	0.21 ± 0.07	$P < 0.05$
II	4.04 ± 0.13	4.08 ± 0.11	0.04 ± 0.07	N.S.
III	3.91 ± 0.13	4.08 ± 0.29	0.17 ± 0.14	N.S.
IV	3.99 ± 0.07	4.05 ± 0.09	0.06 ± 0.04	N.S.
V	3.94 ± 0.10	3.97 ± 0.10	0.03 ± 0.10	N.S.
VI	4.03 ± 0.05	4.04 ± 0.08	0.01 ± 0.06	N.S.
VII	3.89 ± 0.10	3.80 ± 0.15	−0.09 ± 0.07	N.S.

Significance determined by Student's t test for paired variables, n = 10.

TABLE 3. Changes in Serum Sodium (Na^+) (Mean ± SEM)

Group	Sodium (mEq/l)			Significance
	Sample 1 (Control)	Sample 2	Δ	
I	139.4 ± 1.2	138.3 ± 1.4	−1.1 ± 0.4	$P < 0.05$
II	139.2 ± 1.6	137.6 ± 1.4	−1.6 ± 0.6	$P < 0.05$
III	139.1 ± 1.0	137.6 ± 1.2	−1.5 ± 0.6	$P < 0.05$
IV	136.7 ± 1.3	135.1 ± 0.9	−1.6 ± 0.9	N.S.
V	137.9 ± 0.9	135.6 ± 0.9	−2.3 ± 0.4	$P < 0.01$
VI	136.9 ± 1.3	136.6 ± 1.6	−0.3 ± 0.5	N.S.
VII	138.7 ± 1.0	135.9 ± 1.6	−2.8 ± 1.2	N.S.

Significance determined by Student's t test for paired variables, n = 10.

type of induction (thiopental *vs.* inhalational anesthetics), nor was there an interaction effect. There was no change in Ca^{++} in Group VII.

Sodium decreased in Groups I, II, III, and V. Despite decreases in all of the inhalational-induction groups, there was no effect due to type of induction. Likewise, there was no effect of pretreatment, nor was there an interaction effect.

The only group to show a significant K^+ increase was Group I. Considered together, Groups I, II, and

TABLE 4. Changes in Serum Calcium (Ca^{++}) (Mean ± SEM)

Group	Calcium (mg/100 ml)			Significance
	Sample 1 (Control)	Sample 2	Δ	
I	9.60 ± 0.17	9.22 ± 0.19	−0.38 ± 0.11	$P < 0.05$
II	9.22 ± 0.12	8.76 ± 0.15	−0.47 ± 0.06	$P < 0.01$
III	9.16 ± 0.20	8.78 ± 0.23	−0.38 ± 0.11	$P < 0.01$
IV	9.80 ± 0.14	9.29 ± 0.16	−0.51 ± 0.20	$P < 0.05$
V	10.0 ± 0.30	9.33 ± 0.20	−0.65 ± 0.10	$P < 0.01$
VI	9.27 ± 0.19	8.99 ± 0.23	−0.29 ± 0.07	$P < 0.01$
VII	9.27 ± 0.19	9.08 ± 0.13	−0.19 ± 0.13	N.S.

Significance determined by Student's t test for paired variables, n = 10.

III (*i.e.*, inhalational anesthetic inductions) showed a significant increase in K^+, as did the combined Groups I and IV (no pretreatment). Analysis of variance demonstrated a significant effect of pretreatment for both *d*-tubocurarine and pancuronium, as well as an effect of thiopental induction versus inhalational anesthetic induction. There was no interaction effect.

There was no correlation between the changes in K^+, Na^+, and Ca^{++} concentrations. No correlation was found between electrolyte changes and physical status, type of premedication, age, or sex. No correlation was found between electrolyte changes and intensity or duration of fasciculations.

DISCUSSION

Our results for serum K^+ are similar to those previously reported. Clinical doses of succinylcholine cause increases in serum K^+, usually less than 0.5 mEq/l. The results confirm that the effect is reduced by administration of thiopental, or pretreatment with *d*-tubocurarine or pancuronium. Effects of succinylcholine on serum Na^+ were minor, with no obvious pattern.

All groups, I–VI, demonstrated clearly significant decreases in serum Ca^{++} following administration of 100 mg succinylcholine, iv. The range of group mean differences was 0.19–0.65. Neither pretreatment with a nondepolarizing muscle relaxant nor type of anesthetic induction had any effect on this response. Based on this evidence, it is reasonable to assume a cause–effect relationship between the iv administra-

tion of clinical doses of succinylcholine and the observed decreases in serum Ca^{++}.

The increase in serum K^+ after succinylcholine is believed to be the result of leakage through traumatized skeletal muscle cell membranes. This mechanism could explain our results as well. Since very little intracellular Ca^{++} is rapidly exchangeable,[5] there is a concentration gradient to force Ca^{++} intracellularly following the disruptive depolarizations due to succinylcholine.

Another explanation for the decrease in serum Ca^{++} involves active intracellular transport during depolarization. There is considerable evidence that most excitable cells (neurons, smooth muscle cells, and myocardial cells[5–8] have a net uptake of calcium ions associated with excitation. Several papers[6–11] offer convincing data that skeletal muscle cells utilize extracellular Ca^{++} during depolarization. Calcium, concentrated in the transverse tubules, which are in free communication with the extracellular space, is transported intracellularly as depolarization proceeds past the transverse tubule. Intracellularly, it acts to initiate the events of contraction. Loss of extracellular Ca^{++} to the intracellular space may account for the decreases we observed.

The finding of significant decreases in serum Ca^{++} calcium following administration of succinylcholine may not be clinically significant. At present we know of no disease or other circumstance that would make these changes reason to modify current anesthetic techniques.

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References

1. Klupp J, Kraupp O, Honetz N, et al: Uber die freisetzung von kalium aus der muskulatur unter der einwirkung einiger muskelrelazentien. Arch Int Pharmacodyn 98: 340–344, 1954
2. Paton WDM: The effects of muscle relaxants other than muscular relaxation. Anesthesiology 30:453–463, 1959
3. Paton WDM: Mode of action of neuromuscular blocking agents. Br J Anaesth 28:470–480, 1956
4. Austin WH: The relation of IV fluid administration to laboratory values. Am J Clin Pathol 53:288, 1970
5. Ebashi S, Endo M: Calcium ion and muscle contraction. Progr Biophys Mol Biol 18:123–183, 1968
6. Baker PF: Transport and metabolism of calcium ion in nerve. Progr Biophys Mol Biol 24:177–223, 1972
7. Reuter H: Divalent cations as charge carriers in excitable membranes. Progr Biophys Mol Biol 26:1–43, 1973
8. Harris P, Opie L (editors): Calcim and the Heart. New York, Academic Press, 1971, p 198
9. Bianchi CP: Pharmacological actions on excitation–contraction coupling in striated muscles. Fed Proc 27:126–131, 1968
10. Bianchi CP, Bolton TC: Action of local anesthetics on coupling systems in muscles. J Pharmacol Exp Ther 157:388–405, 1967
11. Ashley CC, Ridgway EB: On the relationships between membrane potential calcium transient and tension in single barnacle muscle fibres. J Physiol 209:105–130, 1970

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Barotrauma, a Potential Hazard of Manual Resuscitators

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Hand resuscitators are used in intensive care units and recovery areas for resuscitation and for transport of patients who need ventilatory support. This paper reports two cases in which inadvertent modification of hand resuscitators resulted in morbidity.

Report of Two Cases

Patient 1. A 65-year-old man was admitted to the intensive care unit following elective repair of an abdominal aortic aneurysm. The patient's trachea remained intubated immediately postoperatively and he was ventilated with a constant-volume ventilator. Prior to suctioning the endotracheal tube, an attempt was made to ventilate the patient with a hand resuscitator (Hope Resuscitator, Ohio Medical Products, Madison, Wis.) connected to a source of oxygen. Following the initial manual squeeze on the resuscitator bag, the resuscitator rapidly became distended, the patient was unable to exhale, and he suddenly became agitated. Removing the resuscitator from the endotracheal tube relieved the problem, and the patient was readily ventilated with the constant-volume ventilator without incident.

Patient 2. An 81-year-old woman was admitted to the recovery room after an eight-hour operation on the biliary tract. During transport from the operating theater, the patient, whose trachea was still intubated, was ventilated with a hand resuscitator (Hope Resuscitator, Ohio Medical Products, Madison, Wis.), without supplemental oxygen and without incident. When the resuscitator was connected to a flowmeter-controlled source of oxygen in the recovery room, the resuscitator bag rapidly became tense, and the house officer was unable to ventilate the patient. The patient promptly sustained obvious subcutaneous emphysema and suf-

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