

BIPHASIC CHANGE IN SERUM POTASSIUM CONCENTRATION FOLLOWING A SINGLE DOSE OF SUCCINYLCHOLINE CHLORIDE

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Following the introduction of depolarizing muscle relaxants into clinical practice, data indicating that these substances increase blood plasma potassium levels began to appear in the medical literature (^{1, 2}).

Articles in anesthesiological journals repeatedly confirmed this finding (^{3, 7}), which has been incorporated into many standard textbooks (^{8, 10}), and is now generally accepted. However, there also exists the contrary opinion that there is no connection between serum potassium and succinylcholine induced muscle depolarization (¹¹).

In their recent thorough studies Bali *et al.* (¹²⁻¹⁵), demonstrated consistent small increases in plasma potassium following succinylcholine, although the timing and degree of the potassium increase varied with the induction agent. In these as well as the majority of other studies, the timing of blood sampling was according to the clock rather than to clinical observation. Since both the rate of injection and the timing of patient response are variable, and potassium ions are of fundamental importance in electrolyte balance, we undertook a study of the changes in serum potassium concentration at different clinical stages of the induction of endotracheal anesthesia with thiopentone sodium and succinylcholine chloride.

SUMMARY

Serum potassium concentration during the thiopentone-succinylcholine induction of endotracheal anesthesia was examined in 53 patients undergoing elective surgery. There was no change in serum potassium after thiopentone. During muscle fasciculation serum potassium fell by 0.37 meq/L and returned to the initial level by the end of fasciculation. During muscle relaxation there was a further rise of 0.41 meq/L. The expected increase in serum potassium following the administration of succinylcholine is shown to be preceded by a transient decrease.

MATERIAL AND METHODS

The study included 53 subjects ranging in age from 17 to 73 years, 34 males and 19 females, scheduled for elective surgery such as herniorrhaphy, prostatectomy, ovarian cystectomy and orthopedic procedures. All were in good general health with no abnormal findings on clinical examination or laboratory investigation, including normal renal functions and electrolyte balance. Premedication consisted of oral diazepam, 10 mg, the previous evening and atropine 0.5 mg, meperidine 50 mg, and promethazine 25 mg, by intramuscular injection in the ward 30-60 minutes before anesthesia.

Table 1. — *Mean serum potassium concentrations during five stages of thiopentone - succinylcholine induction.*

Sample	Mean serum potassium (meq/l)	Mean change from control value (meq/l)	Limits	P
Before induction (control group)	4.20	—	—	—
After Thiopentone	4.24	+ 0.04		Statistically not significant
During fasciculation	3.83	— 0.369	± 0.0283	P < 0.05
After fasciculation	4.34	+ 0.14		Statistically not significant
During relaxation	4.61	+ 0.411	± 0.0323	P < 0.05

Anesthesia was induced with sodium pentothal*. The dose was 2.5-3.0 mg per kg body weight. Succinylcholine chloride** was injected in a dose of 1.0-1.5 mg per kg body weight. The lungs were ventilated with 100 % oxygen until the cessation of muscle fasciculations when endotracheal intubation was performed and ventilation continued with nitrous oxide, 60 %, and oxygen 40 %. Blood samples (4-5 ml) in a dry syringe were withdrawn from the antecubital vein of the arm opposite to that of the injection. No other injections or infusions were given, nor was surgery commenced, until blood sampling was complete. The first sample was taken from all patients before induction of anesthesia. Further samples were withdrawn at the following stages of induction: *a*) after thiopentone (10 patients); *b*) during the peak of muscle fasciculation (17 patients); *c*) Immediately after termination of muscle fasciculation (10 patients); *d*) during maximal muscle relaxation (10 patients). In 6 other patients sampling was performed at all of the above stages and the results for each stage were incorporated into the statistical analysis of the results.

RESULTS

The results (table 1) are as follows: after thiopentone there was no statistically significant change in serum potassium. During fasciculation there was an

average decrease in serum potassium level of $0.37 \text{ meq/l} \pm 0.0283$, $P < 0.05$. Thus the administration of succinylcholine chloride after thiopentone produces a biphasic change in serum potassium concentration. The phases may be designated as: *a*) Transient hypokalemia during the period of muscle fasciculation and *b*) Hyperkalemia which begins during the period of muscle relaxation. However, absolute hypo or hyperkalemia were not encountered. Serum potassium levels remained within normal limits despite the observed changes.

DISCUSSION

It is difficult to postulate that the transient hypokalemia, which occurred immediately after the administration of succinylcholine and passed within two minutes, is due to increased renal secretion of potassium. We suggest that during muscle fasciculation there is a shift of potassium ions towards the muscle cells and that this shift is reversed during the following phase of muscle relaxation.

It is generally accepted that depolarizing muscle relaxants should be used with caution in patients with hypokalemia, but our results indicate that induction with thiopentone and succinylcholine may also be dangerous for the patient with hypokalemia.

* Sodium thiopentone for injection B. P., Abbott Laboratoires Ltd., Queensborough, Kent, England.

** Succinyl = Taro for Injection U. S. P., Taro Pharmaceutical Industries Ltd., Haifa, Israel.

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