

Clinical Workshop

S. G. HERSHEY, M.D., Editor

Paraplegia, Succinylcholine and Cardiac Arrest

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Over a five-month period four cardiac arrests occurred within approximately four minutes of induction of anesthesia at our hospital. Each patient had a sleep dose of thiopental (Pentothal) followed by 60 to 80 mg of succinylcholine, tracheal intubation, then halothane (Fluothane) 1 to 2 per cent, N₂O 50 per cent, and O₂ 50 per cent. In no case was

hypoxia, hypercarbia or overdose of anesthetic a factor. All the patients were young Marines who had been wounded in Viet Nam between 44 and 85 days previously. One was febrile secondary to a subphrenic abscess and two had extensive wounds, but all were in positive nitrogen balance, gaining weight and in good states of hydration. The lesion common to all was spinal-cord injury between T8 and L3. All had paraplegia from the time of wounding.

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The opinions expressed in this paper are those of the author and do not necessarily reflect the views of the Bureau of Medicine and Surgery, U. S. Navy, or the Naval Service at large.

Venous blood obtained after each of the four patients was resuscitated to the point of normal sinus rhythm and normal blood pres-

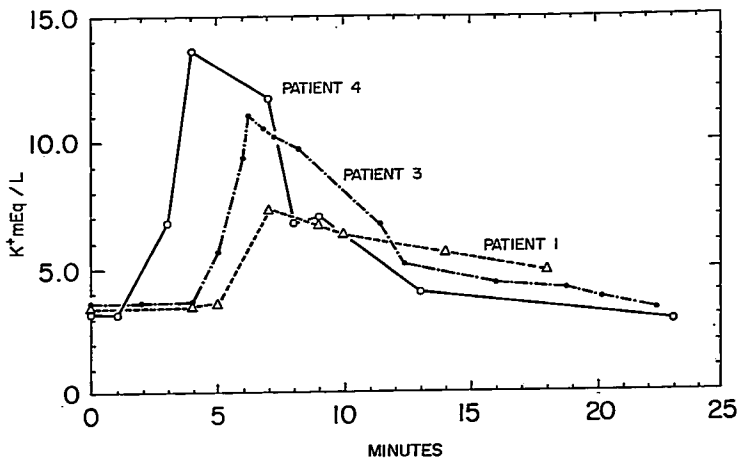


FIG. 1. Changes in serum potassium with infusion of 0.1 per cent succinylcholine. Total dose varies between 20 and 80 mg.

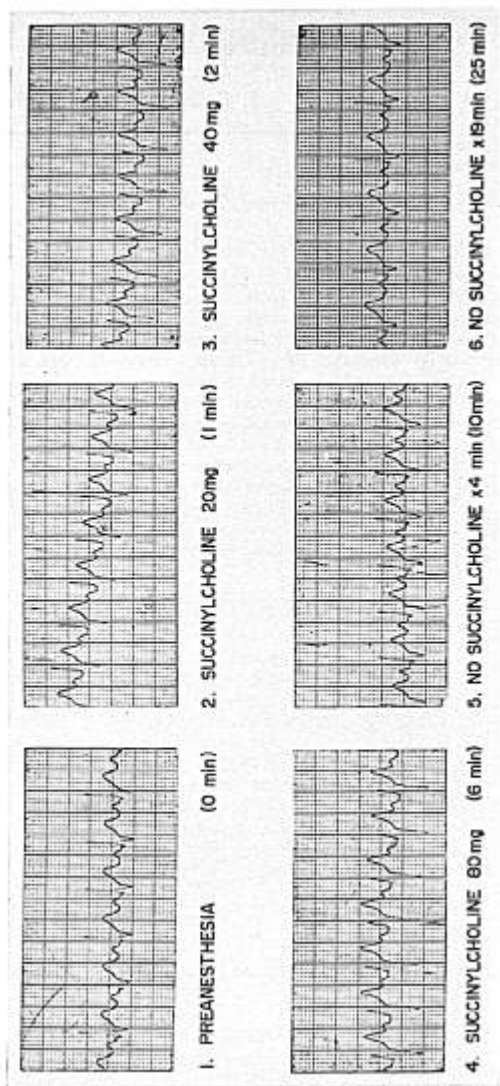


Fig. 2. ECG of Patient 2. Note peaking of T₁ wave and return to normal.

Fig. 3 (below). ECG of Patient 4.



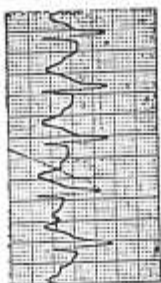
1. PRE-ANESTHESIA (0 MIN)



3. (3 MIN)

	SUP	INF
K ⁺	3.4	6.8
Na ⁺	134	135
PoO ₂	400mmHg	
Paco ₂	35mmHg	
pH	7.36	
SUCCINYLCHOLINE 10mg (2 MIN)		

2.



	INF
K ⁺	6.8
Na ⁺	124

6.

(12 MIN)



	SUP	INF
K ⁺	7.2	13.6
Na ⁺	133	130
SUCCINYLCHOLINE 20mg		

4.

(4 MIN)

5. EXTERNAL CARDIAC MASSAGE



	SUP	INF
K ⁺	4.2	4.0
Na ⁺	128	135

7.

sure revealed serum potassium levels between 5.3 and 6.4 mEq/l. It was speculated that the succinylcholine precipitated the cardiac arrests by producing hyperkalemia. Subsequently, two patients with spinal-cord injuries were anesthetized by the same technique except that succinylcholine was omitted. Complications did not occur. A clinical study was then designed to determine if the concentration of serum potassium became abnormally high following administration of succinylcholine to paraplegics.

PROCEDURE

Four paraplegics were premedicated with pentobarbital (Nembutal), 100 mg, and atropine, 0.6 mg, intramuscularly, or meperidine (Demerol), 75 mg, and atropine, 0.6 mg, intramuscularly. A venous catheter was inserted into either a jugular vein or a subclavian vein and advanced into the superior vena cava. The lower part of the inferior vena cava was cannulated via a femoral vein and a third intravenous catheter was placed in an arm or hand vein for the administration of drugs. Only 5 per cent dextrose in water was infused during the test period. An indwelling arterial needle was placed in the brachial or radial artery for arterial blood gas sampling. Lead II of the ECG was monitored on a Corbin-Farnsworth monitor and a permanent record was obtained by a Sanborn electrocardiograph. A venous blood sample for determination of serum sodium, potassium, chloride, bicarbonate and calcium was obtained prior to induction of anesthesia.

The patients breathed 100 per cent oxygen for three minutes, and venous samples from the superior vena cava and inferior vena cava were drawn simultaneously. Thiopental (200 to 275 mg, intravenously) was administered and an infusion of 0.1 per cent succinylcholine started. Anesthesia was maintained with 1 per cent halothane in oxygen. As the dose of succinylcholine increased, the ECG pattern began to change, with peaking of the T waves, depression of the R waves, deepening of the S waves and prolongation of the QRS complex and T wave. As soon as a change in the ECG pattern was apparent, the succinylcholine was stopped. Venous blood samples were drawn

from both superior and inferior vena cava and, at random times, arterial samples were obtained. Tracheal intubation was not attempted until the ECG, blood pressure and pulse had all returned to normal, and was then accomplished without muscle relaxant. In each case except the last, technical difficulties were encountered, so that not all parameters were measured or correlated for every patient.

RESULTS

In three of the patients potassium concentrations rose to abnormally high levels after the infusion of succinylcholine began and returned to control levels after it was stopped (fig. 1). Peak levels were 7.3 and 11.0 mEq/l. The ECG pattern of Patient 1 had obvious peaking of the T waves, a decrease in amplitude of the R wave and deepening of the S wave. A permanent record was not made of the ECG on this patient. All the venous blood samples from Patient 2 hemolyzed before analysis could be made, but the ECG pattern changed from a normal complex to an even more abnormal pattern of the same type (fig. 2). In Patient 3 the ECG change was recorded and correlated with the high potassium concentration. In Patient 4, after only 20 mg of succinylcholine, T waves developed which completely obliterated the P wave and QRS complex; in fact, the pattern of T waves simulated ventricular tachycardia (fig. 3). In the first three patients the ECG reverted to normal when the succinylcholine infusion was stopped. In Patient 4, circulatory arrest occurred, with blood pressure and peripheral pulse unobtainable for one minute and 40 seconds. External cardiac massage for approximately 20-30 seconds produced a spontaneous reversion towards a more normal ECG pattern with an effective pulse and gradual increase in blood pressure.

Serum chloride, bicarbonate and calcium were all essentially unchanged. Sodium did not change in Patient 1, but in Patient 3 and Patient 4 sodium decreased from 133 mEq/l and 138 mEq/l to as low as 112 mEq/l and 119 mEq/l, respectively. The greatest decrease in concentration occurred as the potassium levels and ECG pattern returned to normal. Arterial blood gases were obtained suc-

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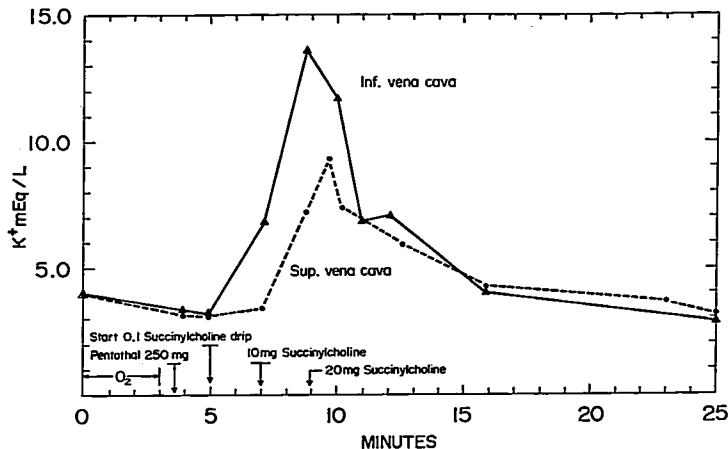


FIG. 4. Changes in serum potassium in the superior vena cava and the inferior vena cava in Patient 4.

cessfully for Patient 4 only, and were as would be expected with 99 per cent inspired concentration of oxygen and adequate ventilation, i.e., Pa_{O_2} 400 mm Hg, Pa_{CO_2} 35 mm Hg, and pH 7.36.

Patients 1, 2, and 3 became hypotensive as the study progressed but only at the end of the test periods after normal ECG patterns had returned. This was attributed to the acute blood loss, amounting to a total of 300 ml to 600 ml, for samples. In Patient 4, circulatory arrest developed in conjunction with the marked ECG changes after only 100 ml of blood had been withdrawn for samples.

No changes in the ECC or potassium occurred in any of the patients during tracheal intubation, accomplished without muscle relaxants between 20 and 25 minutes after the start of anesthesia.

DISCUSSION

Many reports documenting the occurrence of cardiac arrhythmias and/or cardiac arrest with the use of succinylcholine have appeared.¹⁻⁵ There has been controversy as to the cause of this complication, with hypovolemia,⁷ anesthetic overdose, digitalization⁴

and potassium efflux⁶ most often suggested. In the report by Tolmie *et al.*,⁵ three successive cardiac arrests and an episode of ventricular tachycardia occurred in a severely-burned patient; there was little question that hyperkalemia secondary to succinylcholine was the cause. Likewise, in the report by Escue *et al.*,⁹ three of 19 severely-traumatized patients had cardiovascular collapse within four to five minutes after 1 mg/kg succinylcholine. In the former instance serum potassium levels rose to more than 8.0 mEq/l and in the latter, to 9.2 mEq/l.

In our patients one serum potassium level reached 13.6 mEq/l within two minutes of the start of 0.1 per cent succinylcholine infusion and a total dose of only 20 mg. In the two others for whom potassium levels were determined, 7.3 mEq/l was reached after 30 mg of succinylcholine and 11.0 mEq/l after 80 mg. The highest potassium concentration came from the inferior vena caval blood of Patient 4 (fig. 4), and differed considerably from the concentration in the superior vena cava. This supports the hypothesis that the source of the excess potassium may be the abnormal skeletal muscles below the spinal-cord lesion.

Each of these patients had had at least one surgical operation within 24 hours of wounding, but there were no reports of cardiac arrest or arrhythmia during these procedures. Records are available, and each had been given 60 to 100 mg of succinylcholine in a single intravenous dose. Patient 3 was anesthetized 53 days after being wounded and was given 80 mg of succinylcholine intravenously without difficulty, although an ECG monitor was not used. Thirteen days later he had cardiac arrest after only 60 mg succinylcholine.

The reason the sodium concentration fell so markedly after the ECG values returned to normal in two of the patients but not in the other is not apparent. None of these patients exhibited the usual fasciculations that follow succinylcholine.

The patients reported here and the experiences of others with severely-burned patients and those with massive tissue injuries support the hypothesis that a sensitive period occurs sometime after severe injury, reaching a peak and then subsiding. Further studies are needed to define this period. I conclude that succinylcholine must be used cautiously, if at all, in paraplegics after the first 24 to 48 hours of injury.

Intermittent Cuff Inflation during Prolonged Positive-pressure Ventilation

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The use of tracheostomy and prolonged intermittent positive-pressure ventilation (IPPV) in the care of patients with respiratory insufficiency has been accompanied by an increasing incidence of tracheal mucosal erosion, bleeding, and tracheoesophageal fistula.¹ It is becoming increasingly clear that the major contributing factor in these cases is prolonged inflation of the occlusive cuff, with high lateral

pressures on the mucosa and resultant interruption its blood supply.

Various methods of alleviating this problem have been suggested, including frequent intermittent deflation of the cuff, prestretching the cuff, use of fluted cuffs and, recently, the use of long, large-residual-volume, low-pressure, large-contact-area, evenly-inflating cuffs. This last method, which has been studied intensively by Carroll *et al.*¹ and Hedden *et al.*² appears promising.

Another procedure which has been suggested is to inflate the cuff automatically during the inspiratory phase, allowing it to de-

REFERENCES

1. Leigh, M. D., McCoy, D. D., Belton, M. K., and Lewis, C. B.: Bradycardia following intravenous administration of succinylcholine chloride to infants and children, *ANESTHESIOLOGY* 18: 698, 1957.
2. Craythorne, N. W., Brian, M. D., Turndorf, H., and Dripps, R. D.: Changes in pulse rate and rhythm associated with the use of succinylcholine in anesthetized children, *ANESTHESIOLOGY* 21: 465, 1960.
3. Galindo, A. H., and Davis, T. B.: Succinylcholine and cardiac excitability, *ANESTHESIOLOGY* 23: 32, 1962.
4. Dowdy, E. G., and Fabian, L. W.: Ventricular arrhythmias induced by succinylcholine in digitalized patients: A preliminary report. *Anesth. Analg.* 42: 501, 1963.
5. Tolmie, J. D., Joyce, T. H., and Mitchell, G. D.: Succinylcholine danger in the burned patient. *ANESTHESIOLOGY* 28: 467, 1967.
6. Escue, H. M., Houston, J. B., Rudman, H. L., and Hansen, H. R.: Succinylcholine Induced Hyperkalemia: A Potential Hazard Following Severe Trauma or Burns. Scientific exhibit American Society of Anesthesiologists' Meeting, October 1968.
7. Lowenstein, E.: Succinylcholine administration in the burned patient, *ANESTHESIOLOGY* 27: 494, 1966.
8. List, W. F.: Serum potassium changes during induction of anesthesia, *Brit. J. Anaesth.* 39: 480, 1967.

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Received from the Anesthesiology Service, Wilford Hall USAF Medical Center (AFSC), Lackland Air Force Base, Texas 78236.

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