

## THE CLINICAL IMPORTANCE OF HYPERKALAEMIA FOLLOWING SUXAMETHONIUM ADMINISTRATION

BY

F. ROTH AND H. WÜTHRICH

### SUMMARY

A 3-year-old child with severe tetanus, at the end of the third week of illness, developed circulatory arrest after suxamethonium injection. A similar incident also occurred in an adult patient with tetanus. Both incidents were attributable to acute hyperkalaemia induced by suxamethonium. In another patient with severe tetanus, after injection of suxamethonium 100 mg, the potassium level rose within 2 minutes from 3.8 m.equiv/l. to 7.4 m.equiv/l. Cardiac arrest followed suxamethonium injection also in two patients with uraemia. One further patient developed ventricular fibrillation when given suxamethonium three weeks after a road accident in which he sustained multiple injuries. It is suggested that in these last three instances the increase of serum potassium caused by the injected suxamethonium was responsible for the circulatory arrest.

A rise in the level of serum potassium following suxamethonium administration is well recognized (Klupp, Kobinger and Kraupp, 1954; Paton, 1956, 1959; Stevenson, 1960b; List, 1967; Striker and Morrow, 1968) and is usually of the order of 0.5 m.equiv/l. Tolmie, Joyce and Mitchell (1967) drew attention to the large rise in serum potassium and the consequent ventricular fibrillation following suxamethonium in a burned patient. This mechanism is the most likely explanation for the instances of cardiac arrest occurring in these patients (Forrest, 1959; Finer and Nylén, 1959; Fleming et al., 1960; Allan, Cullen and Gillies, 1961; Bush, Graham and Littlewood, 1962; McCaughey, 1962; Belin and Karleen, 1966). Several instances of cardiac arrest that have been encountered recently suggest the possibility that an abnormal rise in serum potassium following the administration of suxamethonium may have been responsible for these disturbances in cardiac action, and that patients suffering from disease processes other than burns may also be at risk. The purpose of this paper is to draw attention to this complication and to suggest in which conditions is the administration of suxamethonium contraindicated.

#### CASE 1 (treated in 1967).

A 3-year-old girl weighing approximately 15 kg was admitted 9 days after injuring herself in the left big toe. Within 36 hours severe tetanus developed. Intuba-

tion was followed by tracheostomy. During the surgical toilet a splinter of wood was located. Muscle relaxation was instituted and maintained for a period of 12 days with alcuronium and the lungs artificially ventilated. Although generalized cramps were no longer apparent, the child still was very spastic and soon developed bilateral talipes equinus. Consequently, 8 days after the discontinuation of the alcuronium therapy passive mobilization using suxamethonium was planned. She was given a mixture of nitrous oxide and oxygen (6:3 l./min) via the tracheostomy using a Rees modification of the Ayre's T-piece. As soon as she was asleep atropine 0.3 mg was injected i.v.; 2 minutes later suxamethonium 25 mg was administered i.v. Barely a minute later the child became pale. Almost simultaneously the pulse disappeared. Immediately external heart massage was performed. Directly following this the colour improved. The heart massage was stopped and a strong relatively slow pulse was palpable. Atropine 0.2 mg was injected i.v. and the pulse quickened. Muscle tone reappeared. The bradycardia was ascribed to the suxamethonium administration. Atropine 0.2 mg was given i.v. followed by a further 25 mg of suxamethonium. Even before the child's muscles had relaxed properly, pallor reappeared together with other indications of circulatory arrest (no pulse, no heartbeat, dilated pupils). External heart massage was performed again. A cardio-oscilloscope showed ventricular tachycardia. The tachycardia soon was interrupted by slowly expanding, wavy complexes. Ventricular tachycardia reappeared. Calcium gluconate (2 ml of 10 per cent) was injected rapidly followed by 5 m.equiv of sodium bicarbonate. The situation suddenly improved. Normal complexes were evident on the oscilloscope, although the rate was very rapid (approximately 180 beats/min).

F. ROTH, DR.MED.; H. WÜTHRICH, CAND.MED.; Section for Resuscitation and Intensive Therapy of the Department of Anesthesiology of the University Clinic and the Inselspital in Bern, Switzerland.

A pulse was detected peripherally. The heart massage was discontinued. The child now was reactive, and no cerebral damage had occurred. The following day the same procedure was carried out, alcuronium being used for the relaxation. The heart action was followed continuously on the oscilloscope but no disturbance was observed.

#### CASE 2 (treated in 1965).

A 56-year-old woman had been curarized with alcuronium for 21 days for severe tetanus. She was, however, still very spastic and it was intended to do passive mobilization under suxamethonium 2 days after the discontinuation of the relaxant. The procedure was carried out exactly as in the previous case (a mixture of nitrous oxide and oxygen, atropine 0.5 mg i.v., then suxamethonium 100 mg). Mobilization of the knee and hip joints had just begun when the patient became pale and all the indications of a circulatory arrest appeared. Immediately external heart massage was applied. Adrenaline 0.5 mg was injected i.v. and a peripheral pulse was soon detected, followed by general recovery.

#### CASE 3 (treated in 1968).

A 62-year-old farmer was admitted with very severe tetanus following an impalement injury. Incubation time was 3 days and onset period was several hours. After 20 days of relaxation with alcuronium and artificial ventilation no more spasms were observed. However, considerable amounts of diazepam were still needed to make the general spasticity bearable. During this transitional phase his respiration was assisted by means of a Bird ventilator. On the fourth day after the discontinuation of alcuronium the patient's muscles were relaxed using suxamethonium 100 mg in order that passive mobilization could be carried out. The patient was connected to a semiclosed system using a mixture of nitrous oxide and oxygen (6:3 l./min). As premedication he had received 45 minutes earlier atropine 0.5 mg i.v. Immediately before the initiation of narcosis a further 0.5-mg dose of atropine was injected followed by diazepam 5 mg. A Bardic Intracath (No. 1814), which 2 weeks earlier had been inserted in the superior vena cava through a puncture in the subclavian vein, permitted blood sampling for estimation of the serum potassium. The e.c.g. (lead II) was observed on a cardio-oscilloscope and registered by a continuous recorder stylus. The blood pressure was measured according to the Riva-Rocci technique. As soon as the patient was no longer reactive four blood samples were taken as controls, then suxamethonium 100 mg was injected. Twelve further blood samples were taken at 30-second intervals followed by sampling at 60-second intervals. During the entire mobilization the patient's circulatory state was good. The blood pressure never deviated more than 15 mm from the initial value. On the oscilloscope a definite pattern of hyperkalaemia, particularly high, pointed T-waves were observed. The serum potassium level of the blood samples was analyzed by flame photometry. Double determinations were always performed but the values never differed more than 0.1 m.equiv. From an initial value of 3.8 m.equiv/l. the potassium level increased within 2 minutes after the injection of suxamethonium to 7.4 m.equiv/l. and then gradually dropped again as is shown in figure 1. The behaviour of the e.c.g. is also indicated. The changes in the

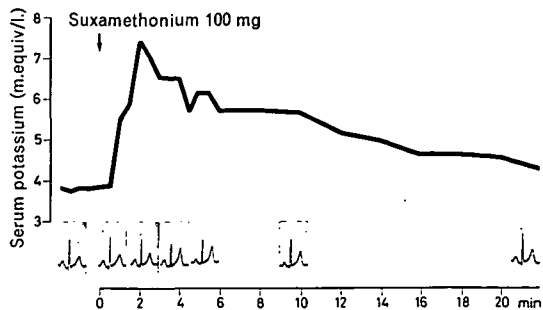


FIG. 1

Serum potassium after injection of suxamethonium 100 mg in a case of tetanus. After some delay the signs of hyperkalaemia appear in the electrocardiogram.

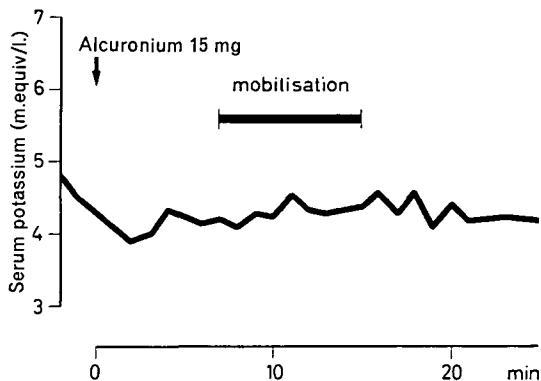


FIG. 2

Serum potassium after injection of alcuronium 15 mg and passive mobilization of a patient with tetanus.

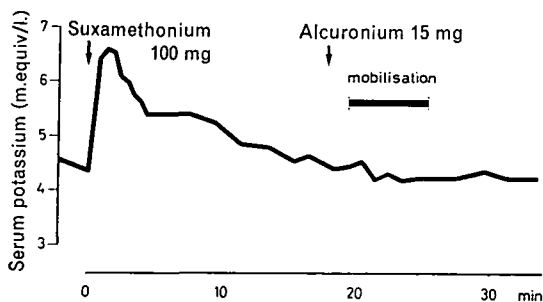


FIG. 3

Serum potassium after injection of suxamethonium 100 mg in a tetanus patient 8 days after the first and 4 days after the second mobilization. Further relaxation for passive mobilization by alcuronium 15 mg.

electrocardiogram occurred after the changes in serum potassium.

Four days later a second mobilization was performed. The patient was relaxed with alcuronium 15 mg and was then exercised passively up to extreme positions for 8 minutes by a physiotherapist and a physician in all of the important joints. Premedication, narcosis, blood sampling, etc., were carried out just as in the first experiment. The results of the potassium determinations are presented in figure 2. Neither the relaxation nor the mobilization brought about a significant increase in potassium levels.

Four days later a third mobilization was undertaken. The relaxation necessary for mobilization was obtained by means of one dose of suxamethonium followed by alcuronium. Otherwise the procedure was the same as that for the previous occasions. As is evident in figure 3, a considerable potassium increase again took place. Within 2 minutes of injection of suxamethonium the potassium increased from an average of 4.4 m.equiv/l. to 6.6 m.equiv/l. The absolute increase, however, was about 1.4 m.equiv/l. below that obtained on the first occasion.

#### CASE 4 (treated in 1968).

A 74-year-old woman with renal insufficiency (BUN = 162 mg/100 ml, potassium 6.2 m.equiv/l.) required an emergency operation for intestinal perforation. Intravenous premedication consisted of atropine 0.5 mg and diazepam 5 mg. Anaesthesia was induced with an oxygen-ether mixture by facepiece. When the patient was asleep suxamethonium 100 mg was injected and intubation carried out smoothly. Approximately 2 minutes after the intubation the pulse weakened and disappeared. All indications of a circulatory arrest became apparent. The usual techniques for resuscitation were performed to no avail.

#### CASE 5 (treated in 1962).

A 2-year-old boy weighing 14 kg with congenital valvular stenosis of the urethra, bilateral pyohydro-nephrosis, and renal insufficiency, was scheduled for an emergency nephrostomy. The same day the serum potassium level was 6.5 m.equiv/l. Premedication was atropine 0.3 mg. Anaesthesia was induced with a mixture of nitrous oxide, oxygen and ether by facepiece. Suxamethonium 25 mg was given i.v. and intubation performed. Shortly afterwards a bradycardia appeared followed by cardiac arrest. External heart massage was performed. About 10 minutes after the administration of adrenaline and calcium the cardiac activity resumed, but only temporarily. Pulmonary oedema and death followed about an hour later. Blood had been withdrawn following cardiac arrest for the determination of electrolytes. The serum potassium level was 8.9 m.equiv.

#### CASE 6 (treated in 1968).

A patient, aged 22 years, was admitted following a road accident. The diagnoses then were: multiple fractures including the left femur; depressed fracture of the skull with some brain damage; partial lesion of the spinal cord. Three weeks later he was scheduled for closure of a persistent fistula with leakage of cerebrospinal fluid. Since the injury the patient had

almost permanently high temperatures and his haemoglobin level had decreased to 50 per cent. He had been given three anaesthetics before without any disturbances. After a premedication of atropine, pethidine and diazepam, anaesthesia was induced with nitrous oxide/oxygen and ether. He then was given suxamethonium 100 mg and intubated. About 2-3 minutes after injection of suxamethonium the patient developed the signs of circulatory arrest. External massage was started immediately. The cardio-oscilloscope showed ventricular fibrillation. Four electroshocks were ineffective. A blood sample taken 10-15 minutes after injection of suxamethonium showed a serum potassium of 7.1 m.equiv/l. Sodium bicarbonate and calcium gluconate were given and the heart was finally defibrillated 40 minutes after the circulatory arrest. Half an hour after successful defibrillation the serum potassium was 3.5 m.equiv/l.

#### *Comment.*

Until recently it has been the authors' practice to use suxamethonium in the mobilization of tetanus patients and this procedure has been performed on about 100 occasions without trouble, at least without actual arrest occurring. And if something happened another explanation could easily be found, as in Case 2 where at that time one thought about pulmonary embolism. But if we now review all these cases, as far as this is possible after some years, we remember several instances when the patient developed severe tachycardia and looked rather pale following suxamethonium injection. If the severe reactions described in Cases 1 and 2 had occurred before we became familiar with external cardiac massage both might have led to a fatal outcome.

We have quite often seen complications after endotracheal intubation in uraemic patients but these were attributed to some nervous reflex mechanism which was thought to be more likely because of the presence of metabolic acidosis. We now believe that an increase in serum potassium after injection of suxamethonium would be a much more reasonable explanation for the incidents encountered in Cases 4 and 5. At least in Case 5 this possibility is supported by the detection of a serum potassium level of 8.9 m.equiv/l. shortly after cardiac arrest and in both cases it was clearly observed that some time elapsed between intubation and cardiac arrest. This fact makes us believe that cardiac arrest was related to hyperkalaemia following suxamethonium because it is not typical of a reflex mechanism.

It might be of interest that in Cases 4, 5 and 6 (all the non-tetanus cases) ether was used for

induction of anaesthesia. It must first be stated that in this department ether is most commonly used for induction of anaesthesia in patients who are not considered to be fit enough to receive thiopentone. In our own investigations (Wüthrich, 1968, unpublished) concerning the increase of serum potassium level in healthy adults following suxamethonium we had to exclude the effect of ether. Like Stevenson (1960a) we could not demonstrate any increase of serum potassium level due to ether alone.

When cardiac arrest occurred recently in Case 6 we were able to profit from the experiences of tetanus patients. We first thought about the role of potassium when the electroshocks proved completely ineffective. Considering the results shown in figures 1 and 3 it can be assumed that the potassium level would have been very high just after the injection of suxamethonium because it was still 7.1 m.equiv/l. 10–15 minutes later. On consideration of this case it would appear that dangerous increase in serum potassium level can occur in patients other than those suffering from burns, tetanus or uraemia. However, it is not possible at this stage to define the circumstances in which this may occur.

#### DISCUSSION

There are several publications in which it is reported that following suxamethonium injection the serum potassium level definitely increases (Klupp, Kobinger and Kraupp, 1954; Paton, 1956, 1959; Stevenson, 1960b; List, 1967; Striker and Morrow, 1968). Paton in 1956 was able to demonstrate in an animal experiment that the potassium circulating in the blood comes from the musculature. The question remains open as to whether it results from ionic exchanges which take place during depolarization in the region of the endplate, or directly from damaged muscle cells. The latter assumption is believed to be more likely for the following reasons. During voluntary muscle activity the synchronously appearing action potential brings about a synchronous depolarization and contraction of the approximately 1,000 muscle fibres of a motor unit. However, that which takes place during depolarization induced by suxamethonium is completely different. Following intravenous injection of suxamethonium the threshold concentration for

depolarization is reached in different muscle fibres at different times. It is conceivable that the further course of events would be the following: a fibre is already contracting while those next to it are still relaxed. They contract shortly afterwards, meanwhile the first fibre relaxes. Mechanical damage to the cell wall might thus result and, as a consequence of this, liberation of potassium. According to this theory the explanation of the muscular pain following the administration of suxamethonium is simple since it results from the injury inflicted by the muscle cells on each other through asynchronous depolarization and contraction. Paton (1959) also developed this theory among others in the course of his reflections on the mechanism underlying muscular pain after suxamethonium application. Also Lenggenhager (1965) came to similar conclusions in his work on the origin of pain in muscle cramps. The theory according to which the liberated potassium primarily should come from mechanically damaged muscle cells is supported by the investigations of Tammisto and Airaksinen (1966), and Tammisto, Leikkonen and Airaksinen (1967) who demonstrated an increase in serum creatine kinase following suxamethonium. Furthermore, Airaksinen and Tammisto (1966) and Bennike and Jarnum (1964) described myoglobinuria following suxamethonium.

According to various authors (Paton, 1959; List, 1967; Striker and Morrow, 1968) and our own investigations (Wüthrich, 1968, unpublished) in healthy adults an increase in the serum potassium of hardly more than 0.5 m.equiv/l. occurs following the injection of 50–100 mg of suxamethonium.

Remaining for discussion is the question as to why in patients with burns or tetanus such a massive liberation of potassium takes place. If one combines these two groups, it can be argued that tetanus as well as the burn represents a severe illness in which the organism and its metabolic processes probably are profoundly disturbed. With limited laboratory investigation available the pathophysiology of both conditions can be explored only to a limited extent at present. For the tetanus patient the explanation may be that the musculature as such is affected by the disease. Brody and Hatcher (1967) on the basis of their animal experiments came to the conclusion that

an increase in the creatine phosphokinase (CPK) must be attributed to a direct influence of the tetanus toxin on the muscle rather than an over-exertion of the muscle. On the other hand, the enzyme determinations performed on tetanus patients by Stirnemann (1966) and Stirnemann and others (1967) support the concept of the CPK increase as resulting from the increased stress on the musculature, because in curarized patients—among whom the severest cases primarily are included—fewer high values were found than in those who were not curarized. In this connection the results of Eyrich and associates (1967), who conducted not only enzyme studies on tetanus patients but also electromyographic and histological studies, are interesting. They detected from the end of the second week of illness definite degeneration and necrosis of the muscle fibres. Using electron microscopy they were also able to confirm the damage in the postsynaptic segment of the endplate.

Since in uraemia profound disturbances in the metabolic processes take place it is plausible that after suxamethonium an abnormal increase in the serum potassium also occurs. On the other hand, it could be argued that on a pre-existing level of 6.5 m.equiv/l. a further increase of 0.5 m.equiv/l. could be sufficient to cause trouble, e.g. for bringing about ventricular tachycardia and circulatory arrest. We hope to undertake some definite investigations in response to these questions in due course. We do not think that reflex vagal activity would be a satisfactory explanation in our uraemic cases (4 and 5) because they were both given atropine and the arrest did not occur immediately after intubation but about 1–2 minutes later. In a recent article in this journal Jacobsen, Christiansen and Lunding (1968) state that suxamethonium had been used in the majority of their patients with acute renal failure and that no untoward effects had been observed. We wonder if some of the cardiac arrests they mention could not also be explained by a rise in serum potassium following suxamethonium. They are to be congratulated on the small number of complications in their series. This might be explained by the fact that their patients were better prepared before operation than were ours.

As a result of these clinical experiences we offer the following recommendations.

#### *Burns.*

Since receiving personal information from Bush in 1960, the authors administer exclusively non-depolarizing relaxants (formerly tubocurarine or gallamine, in recent years alcuronium) and with this procedure have never had any incidents. (In this instance the authors were guided less by the fear of cardiac arrest than by the possibility of a prolonged apnoea following suxamethonium injection. In view of the latest knowledge, this procedure takes on a much more vital significance.) Consequently, in this department, the rule applies that in cases of burns only on the first day is intubation to be performed with the aid of suxamethonium; thereafter, without exception, non-depolarizing relaxants are to be administered.

#### *Tetanus.*

On the basis of the experience described in this paper, it is recommended that for the mobilization of tetanus patients only non-depolarizing relaxants should be administered.

#### *Uraemia.*

Uraemia with increased serum potassium is regarded as an absolute contraindication to the use of suxamethonium. Uraemia with normal serum potassium levels, until more specific investigation has been completed, is believed to be a relative contraindication.

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### L'IMPORTANCE CLINIQUE DE L'HYPERKALIEMIE APRES L'ADMINISTRATION DE SUXAMETHONIUM

#### SOMMAIRE

Un enfant de 3 ans avec tétanie grave présenta à la fin de la troisième semaine de sa maladie, un arrêt de la circulation consécutivement à l'injection de suxaméthonium. Un accident similaire survint également chez un adulte avec tétanos. Dans les deux cas, l'accident était attribuable à l'hyperkaliémie aiguë, causée par suxaméthonium. Le taux de potassium s'éleva chez un autre malade avec tétanos dans les deux minutes après l'injection de 100 mg de suxaméthonium, de 3,8 m.equiv/l. à 7,4 m.equiv/l. L'arrêt cardiaque se produisit également après l'injection de suxaméthonium chez deux patients avec urémie. Un autre malade manifesta une fibrillation ventriculaire lorsqu'il reçut du suxaméthonium 3 semaines après un accident de route avec blessures multiples. Les auteurs sont d'avis que dans les trois derniers cas l'augmentation du taux sérique de potassium, causée par le suxaméthonium, était responsable de l'arrêt circulatoire.

### DIE KLINISCHE BEDEUTUNG DER NACH APPLIKATION VON SUXAMETHONIUM AUFTRETENDEN HYPERKALIÄMIE

#### ZUSAMMENFASSUNG

Bei einem dreijährigen Kind mit schwerem Tetanus entwickelte sich am Ende der dritten Krankheitswoche nach einer Injektion von Suxamethonium ein Kreislaufversagen. Zu einem ähnlichen Vorfall kam es auch bei einem erwachsenen Tetanus-Patienten. In beiden Fällen wurde die durch Suxamethonium induzierte Hyperkaliämie verantwortlich gemacht. Bei einem anderen Patienten mit schwerem Tetanus stieg nach der Injektion von 100 mg Suxamethonium die Kaliumkonzentration innerhalb von zwei Minuten von 3,8 m.Äq./l. auf 7,4 m.Äq./l. an. Bei zwei Patienten mit Urämie kam es nach einer Injektion von Suxamethonium zum Herzstillstand. Bei einem weiteren Patienten, der durch einen Verkehrsunfall multiple Verletzungen erlitten hatte, entwickelte sich drei Wochen nach dem Unfall nach Verabreichung von Suxamethonium Kammerflimmern. Es wird vermutet, daß in diesen drei letzten Fällen das Versagen von Herz und Kreislauf durch das Ansteigen der Serumkaliumkonzentrationen infolge der Injektion von Suxamethonium verursacht wurde.