Succinylcholine-induced Hyperkalemia in a Patient with a Closed Head Injury

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The hyperkalemic responses following succinylcholine administration in a variety of clinical conditions are well documented.1–5 This case report illustrates a previously undescribed condition, hyperkalemia following administration of succinylcholine to a patient with a closed head injury without peripheral paralysis.

REPORT OF A CASE

A previously healthy 18-year-old white youth sustained a closed head injury while sledding on snow. On initial examination he was semicomatose, with head and facial contusions, but he was able to move all extremities in response to painful stimulation. Reflexes were 2+ bilaterally, with positive ankle clonus. The Babinski test was positive bilaterally, with minimal weakness. Pupils were dilated and nonreactive. There was no skull fracture. Cranial tomography on admission revealed an intracerebral hematoma in the left temporal lobe.

The patient was brought to the operating room 37 days after the initial injury for a ventriculo-peritoneal shunt. He was able to move all extremities in response to pain, but was not able to communicate. Anesthetic induction began with 1 mg pancuronium, followed 3 min later with 250 mg thiopental and 120 mg succinylcholine. Endotracheal intubation was uneventful, and anesthesia was maintained with enflurane, oxygen, and nitrous oxide. Fifteen days later the patient returned to the operating room for debridement of a hip decubitus. Preoperative serum potassium was 3.9 mEq/l. The conduct of anesthesia was similar to that of the preceding anesthesia. After preoxygenation, a defasciculating dose of 1 mg pancuronium bromide, a slow dose of 250 mg thiopental, and 120 mg of succinylcholine were administered intravenously. Immediately following intubation of the larynx the patient was found to have ventricular tachycardia, without an audible blood pressure. Resuscitation was successfully achieved by use of lidocaine, sodium bicarbonate, and countershock. Ten minutes after initiation of resuscitation procedures, serum potassium was 3.5 mEq/l. A ventricular tap during resuscitation, revealed no increase in intracranial pressure. Eight days following this episode, and 60 days after the initial trauma, the patient returned to the operating room for revision of the ventriculoperitoneal shunt and debridement of a hip ulcer. A radial arterial catheter was placed percutaneously and samples of blood for determination of arterial blood gases, potassium, and sodium were drawn at one-minute intervals following induction of anesthesia. The patient was preoxygenated, and given 200 mg thiopental and 40 mg succinylcholine without a defasciculating dose of pancuronium. Initial blood-gas values, breathing room air, were pH 7.47, PaO₂ 36 torr, PaCO₂ 85 torr, and bicarbonate 25 mEq/l. The initial serum potassium was 3.8 mEq/l.

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Figure 1 summarizes the values of the serum potassium obtained throughout induction. There was a rapid increase of serum potassium from the baseline of 3.8 mEq/l to a peak of 6.8 mEq/l 4 min after succinylcholine administration. Following this peak there was a gradual reduction in potassium values toward baseline. The EKG pattern during these electrolytic changes showed a widened QRS complex with peaking of the T waves. Arrhythmias did not occur. Arterial blood-gas values after 10 min, breathing 40 per cent oxygen, were pH 7.47, PaO₂ 156 torr, PaCO₂ 36 torr, and bicarbonate 25 mEq/l. The remainder of the anesthesia was uneventful. The patient was transferred to another hospital in the custodial care of his parents 76 days after admission. He was able to move all extremities, but was unable to communicate.

DISCUSSION

Three reasons led us to repeat succinylcholine following a previous cardiac arrest. First, we were not sure that the dysrhythmia was the result of succinylcholine hyperkalemia, especially since the patient had been pretreated with 1 mg pancuronium and the potassium concentration in blood drawn after the cardiac arrest was only 3.5 mEq/l. Second, the patient did not have paraplegia, quadriplegia from cerebral injury, or hemiplegia from cardiovascular accident, as previously described.1 He, in fact, could move both sides in response to pain and had spontaneous movement such that he needed restraint. Third, if he, indeed, were sensitive to succinylcholine,
then reducing the dose of succinylcholine would provide the information while giving added protection against harmful side effects, as shown by the original work of Tolmie, Joyce, and Mitchell.\(^5\)

The time course of this patient's sensitivity correlates closely with the three to eight week "vulnerable period" described by Cooperman.\(^3\)

While it is known that succinylcholine-induced hyperkalemia can occur in association with both upper and lower motor-neuron diseases,\(^4,6\) it is probably not correct to lump all neuromuscular diseases in these categories. First, it is not clear what makes these patients hyperkalemic. Second, there is no common denominator for the cases thus far reported. Certainly the lesions of cerebral palsy, encephalitis, Parkinson's disease, multiple sclerosis, muscular dystrophy, and now cerebral hematoma are different.

Some have suggested that disuse atrophy from prolonged bed rest is a common denominator, but Gronert and Theye demonstrated that this had only a minor effect, at least in dogs.\(^6\) Nutrition may play a role, but definitive studies have not been done. This patient was receiving 2,400 kcal per day, with additional multivitamins, through a nasogastric tube. His weight was stable, without gross edema or obvious muscle wasting.

Although it has been shown that pretreatment with a nondepolarizing muscle relaxant prior to succinylcholine administration will attenuate the hyperkalemia in susceptible individuals,\(^7\) this case illustrates that this therapy is not entirely protective. In summary, closed head injuries should be added to the growing list of disorders that have the potential for serious hyperkalemia following intravenous administration of succinylcholine.

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**Preoperative Clonidine Withdrawal Syndrome**

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Clonidine is an antihypertensive drug available only in oral form. Rebound hypertension following abrupt cessation of clonidine therapy was described in 1973,\(^1,2\) and since then, more reports have appeared.\(^3-8\) Brodsky and Bravo described the case of a patient in whom clonidine withdrawal syndrome manifested postoperatively in the recovery room,\(^8\) but there has been no published report of this problem occurring preoperatively.

**Report of Two Cases**

**Patient 1.** A 54-year-old man who had an ischemic leg was scheduled for a femoral-popliteal bypass operation, to begin about noon. He had been receiving clonidine, digoxin, hydralazine, propranolol, furosemide and nitroglycerin. Since midnight, all drugs except nitroglycerin had been discontinued. Premedicated with meperidine, 25 mg, and atropine, 0.6 mg, the patient arrived in the operating room shortly after noon. He had just taken nitroglycerin for moderate chest pain, with resultant relief of this discomfort. Radial and pulmonary arterial lines were inserted promptly and provided pressure readings of 280 torr systolic and 30 torr in the occluded (PAOP, "wedge") position, respectively. The abnormal PAOP indicated cardiac failure and the

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