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Succinylcholine-Induced Hyperkalemia In Children: The Case That Led To The Studies

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Background: Since its introduction into clinical practice in the early 1950s, succinylcholine rapidly became the gold standard for rapid onset of profound muscle relaxation and became an integral component of the standard anesthetic induction sequence for tracheal intubation. Side effects of succinylcholine, however, soon became evident. The first identifiable side effect was prolonged muscle relaxation that was ultimately correlated with delayed drug metabolism caused by abnormal plasma cholinesterase. By the late 1950s, there were reports of cardiac arrest in burn patients following succinylcholine administration that were eventually found to be secondary to hyperkalemia. Reports of succinylcholine-induced hyperkalemia in patients with neurologic diseases in the early 1970s led to warnings about the potential for hyperkalemia in many neurologic disorders, including children with cerebral palsy (CP) and myelomeningocele (MM). We had used succinylcholine in many patients with CP or MM without adverse effect and had discussed on many occasions the need to formally study this. Eventually, we had a case that stimulated us to proceed with a series of studies.

Case Report: The patient was a two-year-old male with severe developmental delay who presented for cataract extraction (March 1, 1983). His hospital chart was extensive and every clinic note stated that he had cerebral palsy with spastic quadriplegia. He was evaluated the day before surgery and the plan for general anesthesia discussed with his mother. Prior to induction, an intravenous catheter was inserted after dermal anesthesia with lidocaine with a 30 gauge needle. Induction was performed with atropine, thiopental and succinylcholine. Soon after the injection of succinylcholine, peaked T waves developed on the ECG followed by widening of the QRS complex and onset of ventricular tachycardia. Tracheal intubation was performed, closed chest massage was initiated and calcium chloride was administered intravenously. The ECG quickly returned to normal and vital signs stabilized. A potassium level obtained several minutes after the ECG change had reverted to normal was 8.1 mEq/L. Surgery proceeded without incident and the patient was extubated several hours later in the PACU. Several hours after release from the PACU, he developed respiratory distress and was intubated and transferred to the ICU. A creatine kinase (CK) level obtained in the ICU the day after surgery was 22,620. It was suggested to the patient's neurologist that the patient might have a primary myopathy. The neurologist felt that the CK elevation was secondary to the cardiac arrest. He remained in the ICU for 10-14 days and was discharged from the hospital on March 24, 1983. In July, 1983 he underwent a muscle biopsy utilizing local anesthesia and the diagnosis of muscular dystrophy was made.

Conclusion: Based on this experience, we decided to proceed with formal studies on the effect of succinylcholine on plasma potassium in normal children and children with CP or MM. These studies were published in 1984 (normal), 1985 (CP), and 1986 (MM). All three studies showed no significant change in plasma potassium levels after succinylcholine.^{1,2,3}

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