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Encephalitis and a Hyperkalemic Response to Succinylcholine

D. BRUCE COWGILL, M.D.,* LUCILLE A. MOSTELLO, M.D.,* HARVEY M. SHAPIRO, M.D.†

Hyperkalemic responses secondary to the administration of succinylcholine have been observed in patients with traumatic or burn injuries to muscle,[1,2] muscular dystrophies,[3] spinal cord lesions, and upper motor neuron disease associated with strokes.[4–6] This case study documents a heretofore unreported association of succinylcholine-induced hyperkalemia with encephalitis.

Report of a Case

A 26-year-old woman had been in good general health until five months prior to hospitalization, when she sought medical help because of emotional problems. Shortly after admission to another hospital, she developed weakness of the left leg, followed by confusion, incoherent speech, and incontinence. There was no history of drug abuse or poisoning. Analysis of blood for heavy metals, routine bacteriologic evaluation, and serum viral titers were negative. Roentgenograms of the skull, a pneumoencephalogram, brain scan, and bilateral carotid arteriograms were all normal. Serial electroencephalograms revealed nonspecific, diffuse slowing. Lumbar puncture disclosed leukocytosis, erythrocytosis, decreased alpha-globulin, and normal CSF pressure. The tuberculin skin test was positive. Treatment with isoniazid was begun; this, in addition to dexamethasone, was continued to the time of her transfer to the Hospital of the University of Pennsylvania for further evaluation, including a brain biopsy.

On admission the patient was unable to follow simple commands and had increased tone and hyperactive deep tendon reflexes in both arms and the left leg. Generalized muscle atrophy was apparent. Response to pin prick was normal, as was the cranial nerve examination.

The clinical impression was that of a progressive, degenerative brain disease. Immediate preoperative studies included serum glucose, BUN, sodium, potassium, chloride, and bicarbonate—all of which were normal. Roentgenograms of the chest revealed no active disease; an electrocardiogram showed sinus tachycardia with occasional premature atrial contractions and S-T elevation in leads V_{1-4}, suggestive of early repolarization. Hemoglobin was 13.4 g/100 ml. Leukocyte count was 9,500/cu mm.

Prior to biopsy of the brain anesthesia was induced with sodium thiopental, 150 mg, and nitrous oxide–oxygen, 4:2 l/min. Succinylcholine, 80 mg, was administered intravenously and the trachea was intubated without difficulty. The electrocardiogram was monitored; an immediate progressive increase in the height of the T-waves was noted (fig. 1). Blood samples for potassium analysis were obtained from a 16-gauge intracath placed in the basilic vein prior to induction of anesthesia (fig. 1). Ninety seconds after succinylcholine administration serum K^+ increased from a control value of 3.59 to 10.52 mEq/l. After 5 and 30 minutes values were 7.29 and 6.13 mEq/l, respectively. Immediate treatment consisted of controlled hyperventilation,[7] which resulted in a rapid decline in the amplitude of the T-wave. Cardiovascular stability was evident throughout the episode and it was not necessary to administer additional treatment. After a few minutes of hyperventilation,

* Resident in Anesthesia.
† Assistant Professor in Anesthesia.
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Reprint requests should be directed to Dr. Shapiro.

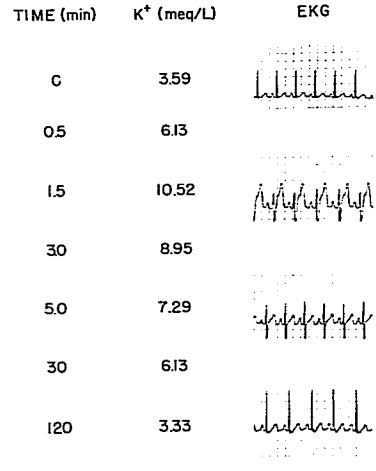

FIG. 1. Serum potassium levels (mEq/l) and electrocardiograms obtained during induction and subsequent anesthesia in a patient with encephalitis.

the electrocardiographic pattern returned to that of the preanesthetic period, although serum K^+ values remained somewhat above normal. The remainder of the anesthetic course was unremarkable, and the patient's neurologic condition was unchanged following anesthesia. Creative phosphokinase in blood obtained two days later was normal, and the electromyogram of the left biceps muscle and nerve conduction velocity of the right median nerve were normal. These studies are compatible with upper motor neuron disease.

DISCUSSION

The disease pattern in this case is that commonly ascribed to encephalitis, probably caused by viral infection. The first signs are mood changes, which are progressively followed by intellectual deterioration, and akinetic mutism, or complex movement disorders.[8] The end stage of such an infection may be widespread loss of cortical ganglion cells and clinical decortication. The condition of our patient at the time of operation was approaching end-stage encephalitis.

We doubted whether a diffuse central nervous system disease not associated with hemiplegia or other focal neurologic signs could produce sensitization of muscle to succinylcholine. Nevertheless, we carefully monitored the EKG and made provision for blood sampling during the administration of succinylcholine to our patient. The extent of hyperkalemia which occurred was unexpected. Nervous system lesions associated with pronounced potassium efflux from muscle either originate in peripheral nerves or spinal cord[9] or are associated with the stroke syndrome and severe focal motor abnormality.[4] Marked focal motor abnormalities were not a major manifestation in our patient.

Speculations about this patient's response to succinylcholine included the contributions of chronic steroid administration and muscle wasting produced by long-term bed rest associated with her encephalitis. Solandt and colleagues[10] found that disuse atrophy produced by experimental skeletal fixation enhanced potassium release resulting from depolarizing relaxants. Gronert *et al.*[11] believe that muscle atrophy of any etiology is a relative contraindication to the use of succinylcholine. However, a review of the literature failed to reveal any clinical study of either of these as a primary or contributing factor in the hyperkalemic response in man. Many patients taking steroids and on chronic bed rest receive anesthetics but have not been reported to react adversely to succinylcholine.

We think it most likely that this patient with encephalitis had hyperkalemia as a result of a denervation type of sensitization of motor units to the effects of succinylcholine. Our findings emphasize that any central nervous system lesion may at some stage be likely to potentiate succinylcholine-induced potassium release from muscle. Individuals with encephalitis and its frequently puzzling neurologic manifestations should now be added to the list of those patients who may become hyperkalemic following administration of succinylcholine.

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A Training Aid for Direct Laryngoscopy and Intubation

PAUL E. BERKEBILE, M.D.,* HUGO H. PFAEFFLE, M.D.,†
R. BRIAN SMITH, M.D.,‡ PAUL REHKOPF§

Teaching direct laryngoscopy and intubation is often hampered because the instructor and the trainee are both trying to look down a small orifice simultaneously. We have devised a technique permitting adequate observation of laryngoscopy and intubation by an instructor without interference with the student performing the procedure. This has improved direct laryngoscopy and intubation as performed by trainees at the Eye and Ear Hospital in Pittsburgh, and has provided a more efficient and relaxed atmosphere for training.

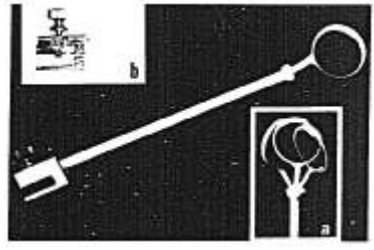

FIG. 1. Mirror and detachable clip.

DESCRIPTION

The equipment consists of a disposable mirror, handle, and clip (fig. 1). The mirror is

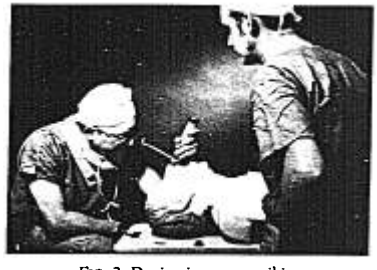

FIG. 2. Device in use, manikin substituted for a patient.

* Assistant Professor of Anesthesiology.
† Assistant Professor of Clinical Anesthesiology.
‡ Associate Professor of Anesthesiology.
§ Design Engineer, Department of Ophthalmology, Eye and Ear Hospital, Pittsburgh, PA 15213.

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Address reprint requests to Paul E. Berkebile, M.D., Department of Anesthesiology, Eye and Ear Hospital, 230 Lothrop Street, Pittsburgh, PA 15213.