This presentation, incidentally, illustrates a possible disadvantage of catheter epidural anesthesia, in that failures are more likely to occur with it than when single-injection epidural anesthesia is administered via a needle. When an injection is made through a needle, patient position and anatomic abnormalities may produce unilateral blockade, whereas when the injection is made through a catheter there are *at least five* possibilities for failure. This is not intended to discredit continuous techniques. The benefits of injecting through a catheter are so important that they far outweigh the possibility of an occasional failure.

Finally, it is apparent from these and other [1, 2, 4] observations that bilateral blockade may not develop when the anesthetic solution spreads unilaterally into the epidural space. Since considerable concentrations of the local anesthetic drugs appear in the cerebrospinal fluid in this situation,[8] it would appear that cerebrospinal fluid levels of the local anesthetics are unrelated to the extension and quality of the blockade. Furthermore, this would tend to support the contention [4] that transdural passage of the local anesthetic drugs is not the only blocking mechanism in epidural anesthesia.

References

1. Singh, A.: Unilateral epidural analgesia: Case report, Anaesthesia 22: 147, 1967.
2. Shanks, C. A.: Four cases of unilateral analgesia, Brit. J. Anaesth. 40: 999, 1968.
3. Gutierrez, A.: Anestesia metamerica peridural, Rev. Cir. B. Aires 12: 665, 1932.
4. Usubiaga, J. E., Wikinski, J., Wikinski, R., Usubiaga, L. E., and Pontremoli, M.: Transfer of local anesthetics to the subarachnoid space and mechanisms of epidural block, Anesthesiology 25: 752, 1964.
5. Vandam, L. D.: Quoted by Singh, A.[1]
6. Sanchez, A., Acuna, L., and Rochs, F.: An analysis of the radiological visualization of the catheters placed in the epidural space, Brit. J. Anaesth. 39: 485, 1967.
7. Usubiaga, J. E., Wikinski, J. A., and Usubiaga, L. E.: Epidural pressure and its relation to the spread of anesthetic solutions in epidural space, Anesth. Analg. 46: 440, 1967.
8. Usubiaga, J. E.: Unpublished data.

Massive Hyperkalemia after Administration of Succinylcholine

Lee H. Cooperman, M.D., George E. Strobel, Jr., M.D., Eric M. Kennell, M.D.

Hyperkalemia of sufficient magnitude to cause serious arrythmias, even to the point of ventricular fibrillation, is an often-recorded effect of administration of succinylcholine to the burned or injured patient.[1, 2] Recently we have encountered three neurosurgical patients, all with upper-motor neuron lesions and hemiplegia or paraplegia, who had electrocardiographic evidence of massive hyperkalemia after receiving succinylcholine. This is a report of the third patient.

Case Report

A 54-year-old man was admitted for evaluation of increasing disorientation and memory lapses. During his hospitalization a pneumoencephalogram was made with the patient under nitrous oxide and halothane anesthesia. Succinylcholine, 100 mg, had been injected intravenously for tracheal intubation. No electrocardiographic abnormalities were seen during anesthesia. Ventricular dilatation was noted on the pneumoencephalogram and the patient was scheduled for a right ventriculo-jugular shunt at a later date. Anesthesia again was nitrous oxide–halothane with succinylcholine, 100 mg intravenously, for intubation. Again, no electrocardiographic abnormalities occurred. Difficulty was encountered in placing the ventricular catheter and the procedure was abandoned. The next day the patient developed left hemiparesis.

Three weeks later the patient was scheduled for a left ventriculo-jugular shunt. He was premedicated with atropine, 0.5 mg intramuscularly, and anesthesia was induced with thiopental, 200 mg intravenously, followed by methoxyflurane and oxygen. An arterial catheter was placed and baseline samples for electrolytes drawn. The patient was then given succinylcholine, 60 mg intra-

Received from the Department of Anesthesia, University of Pennsylvania School of Medicine, 3400 Spruce Street, Philadelphia, Pennsylvania 19104.

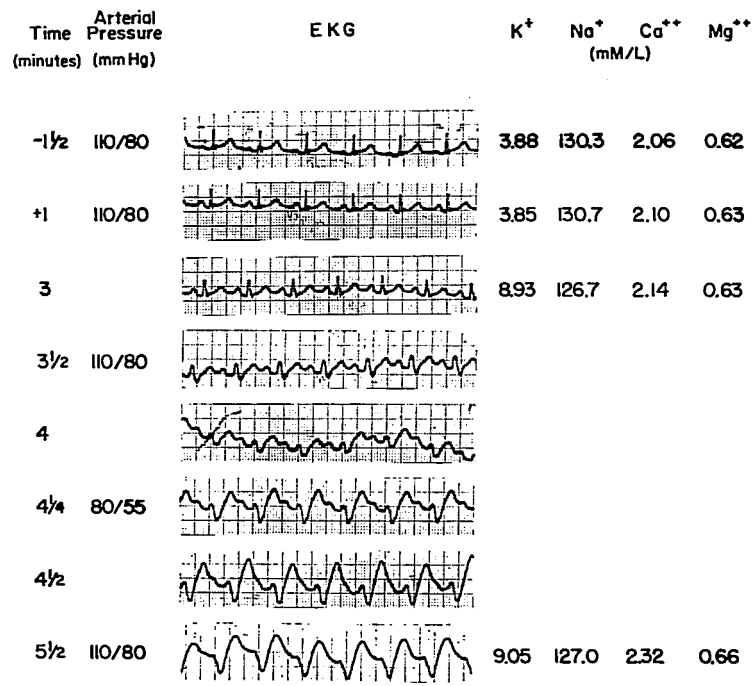

FIG. 1. Electrocardiographic and plasma electrolyte changes following succinylcholine, 60 mg, intravenously.

venously, and serial electrocardiographic tracings, arterial blood samples, and blood pressure recordings were made. Within three minutes of administration of succinylcholine the plasma potassium level reached 9 mEq/l and the electrocardiogram showed a widened QRS complex and elevated T waves. Throughout the period of hyperkalemia the heart rate did not change and systolic pressure decreased only 30 torr. The remainder of the anesthetic course was unremarkable, and postoperatively the patient's neurologic condition was unchanged.

DISCUSSION

Succinylcholine causes skeletal muscle cells to depolarize, resulting in an efflux of intracellular ions into the extracellular space and an influx of sodium into the cell. The changes in plasma electrolytes seen in our patient, as well as those in the ECG, are consistent with this mechanism. Why, though, these changes are greater in certain patients is not known, but cellular damage with increased facility for efflux of potassium may play a role. Both calcium and magnesium may be important in regulating ionic permeabilities of cell membranes; both tend to stabilize the muscle cell membrane against depolarization.[3] The low plasma calcium and magnesium levels measured in this patient were perhaps a significant factor in his atypical response.

We have now encountered three patients who had ECG evidence of massive hyperkalemia after succinylcholine (although plasma

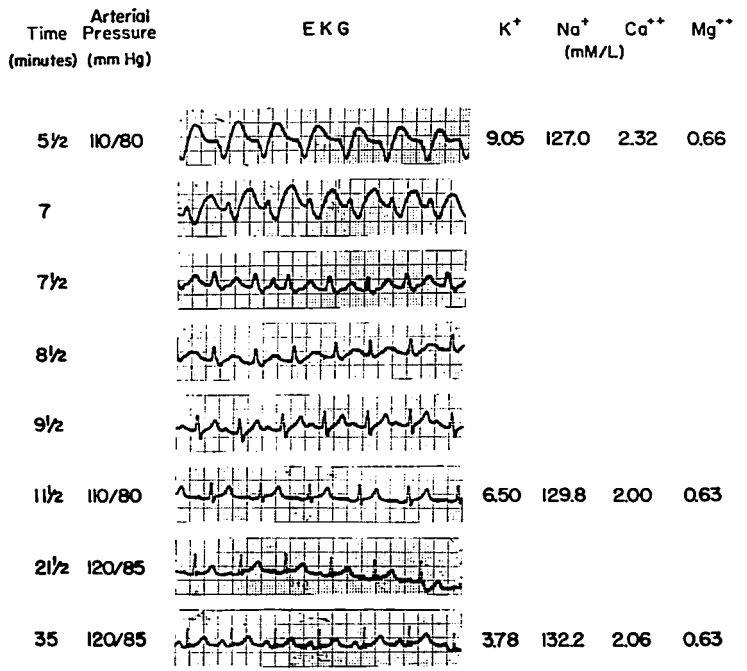

FIG. 2. Electrocardiographic and plasma electrolyte changes following succinylcholine, 60 mg, intravenously.

potassium measurements were made in only one case). These ECG and electrolyte changes were remarkably similar to those in the burned and injured patients described by Mazze and by Tolmie.[1, 2] However, instead of burns or trauma, all three patients had either hemiplegia or paraplegia for three to eight weeks before anesthesia. It is of interest that our third patient had two exposures to succinylcholine before the onset of paraplegia with no untoward effect. Perhaps there is a "vulnerable period" after neurologic injury, similar to that postulated in burned patients, when the danger of hyperkalemia is enhanced.[4]

Tammisto *et al.* reported significant increases in serum creatine phosphokinase activity after succinylcholine administration.[5] Since this enzyme is normally found in muscle cells, elevation in serum activity may indicate cellular injury. They propose that latent muscle disease may predispose to more marked rises in enzyme activity than in normal individuals. A comparable situation may exist regarding plasma potassium levels in patients with antecedent muscle paralysis, inactivity and wasting from neural damage. In any event, our experience suggests that physicians should be alert to the possibility that hyperkalemia may occur in the patient who must be anesthetized in the period immediately following a cerebral vascular accident or other upper-motor-neuron lesion.

Measurements of calcium and magnesium were made by Dr. C. P. Bianchi.

REFERENCES

1. Tolmie, J. D., Joyce, T. H., and Mitchell, G. D.: Succinylcholine danger in the burned patient, ANESTHESIOLOGY 28: 467, 1967.
2. Mazze, R. I., and Dunbar, R. W.: Intralingual succinylcholine administration in children: An alternative to intravenous and intramuscular routes? Anesth. Analg. 47: 605, 1968.
3. Constantin, L. L.: The effect of calcium on contraction and conductance thresholds in frog skeletal muscle, J. Physiol. 195: 119, 1968.
4. Lowenstein, E.: Succinylcholine administration in the burned patient, ANESTHESIOLOGY 27: 494, 1966.
5. Tammisto, T., Brander, M., and Airaksinen, M.: Hypoxia and suxamethonium-induced muscle injury, Brit. J. Anaesth. 41: 276, 1969.

Gangrene of a Thumb Following Use of the Photoelectric Plethysmograph during Anesthesia

MARTIN H. LEBOWITZ, M.D.*

The photoelectric plethysmograph is a miniaturized transducer designed to indicate volume changes of a pulsatile flow.[1-3] The device incorporates a light source and a highly sensitive photocell. It is capable of producing a large pulse-wave signal when placed over a surface artery or arteriolar bed. Several transducer models are commercially available, one model specifically for use on the finger or toe pad. The finger transducer has the photocell adjacent to the light source, relying on reflection of the light rather than transillumination. The sensor is housed in a plastic mold and attached to the finger or toe pad by means of a Velcro® strap (fig. 1).

We have found this device of great value in monitoring peripheral blood flow during anesthesia and surgery. Systolic blood pressure can be determined easily by slow deflation of a pneumatic cuff while observing the oscilloscope for the return of the pulsatile flow wave. This method of blood-pressure determination has been of particular value with small children and infants, in whom palpatory or auscultatory pressures may be difficult or impossible to obtain. In the course of many applications of the monitor its performance had been entirely satisfactory until two recent complications involving the great toe of a two-month-old child and the thumb of a newborn infant. In both cases the involved digit was observed to be markedly swollen and plethoric upon removal of the monitor. The condition rapidly improved in one and rapidly progressed to gangrene of the distal phalanx in the other.

REPORT OF TWO CASES

Patient 1. A previously healthy, 2-month-old, 4.9-kg infant girl was admitted because of signs of central nervous system irritability, manifested by hyperreflexia, vomiting, and nuchal rigidity. Meningitis was suspected, but several lumbar punctures showed only xanthochromic fluid, suggesting a subarachnoid hemorrhage. Because of seizures, oral phenobarbital therapy was initiated. The infant's condition improved after several days and she was scheduled for a cerebral angiogram under general anesthesia.

Following premedication with atropine, 0.1 mg, intramuscularly, anesthesia was induced with ketamine,† 50 mg (10 mg per kg), intramuscularly. Anesthesia was maintained with intermittent intravenous ketamine. A 4½-hour bilateral carotid and vertebral angiogram was performed via a percutaneous puncture of the right femoral artery. As a monitor of arterial flow to the right lower extremity, a photoelectric plethysmograph was attached to the right great toe at the start of anesthesia.[4] An adequate pulsatile flow wave was observed until shortly after catheterization of the artery. Thereafter, the oscilloscope showed intermittent pulse waves at diminished amplitude throughout the procedure, despite good color and warm toes.

Ventilation was spontaneous at 25–30 per minute. Rectal temperature stabilized at approximately 34 C. Vital signs remained relatively stable; systolic blood pressure was 90–110 mm Hg and cardiac rate 120 beats/min throughout

* Assistant Professor, Department of Surgery/Anesthesiology, University of California at Los Angeles, Center for the Health Sciences, Los Angeles, California 90024.

† Ketalar (CI-581) (2-(o-chlorophenyl)-2-methylamino-cyclohexanone), Parke Davis.