The Lack of Effect of Succinylcholine on Serum Potassium in Patients with Parkinson's Disease

To the Editor—Depolarizing muscle relaxants are known to cause hyperkalemia in a variety of neurological diseases. The effect of succinylcholine (ScH) on serum potassium in patients with Parkinson's disease has not been adequately studied and reported.1 The etiology of hyperkalemia associated with ScH and Parkinson's disease in a single patient report was clouded by the complex nature of the case.2

With approval from the internal review board, seven patients (38–58 yr) with severe Parkinson's disease who underwent elective adrenal medullary to caudate transplantation were studied. They were surgical candidates for the procedure because of poor control of their disease with medical therapy. Preoperatively, their medications for Parkinson's disease were discontinued for approximately 12 h before induction of general anesthesia.

Baseline arterial blood gases and serum K+ concentrations were determined, after which anesthesia was induced with thiopental 5 mg/kg and fentanyl 2–3 µg/kg, and tracheal intubation was facilitated with ScH 1.5 mg/kg. Patients' lungs were ventilated with 100% oxygen and end-tidal carbon dioxide levels maintained within ± 2 mmHg of preinduction values. Arterial blood gases and the serum K+ concentration were measured 3–5 min following administration of succinylcholine.

In five of the seven patients there was no change in serum K+ concentration. One patient had an increase of serum K+ of 0.2 meq/l, and one patient had a decrease of serum K+ of 0.2 meq/l. In all, patients' arterial oxygen saturation remained above 99%. Arterial carbon dioxide was within ± 2 mmHg between the two sampling periods, and arterial pH was within ± 0.02 in all seven patients during this time period. None of the seven patients had any of the EKG abnormalities associated with hyperkalemia during induction of anesthesia or during the first 60 min of the case. All seven patients successfully recovered from general anesthesia without neurologic or cardiac sequelae.

In summary, based upon the finding in this series of seven patients with Parkinson's disease, ScH-induced hyperkalemia was not an identifiable clinical problem.

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REFERENCES

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Source of Specialized Endotracheal Tubes

To the Editor—It was with interest and an appreciation of the circumstances that I read Dr. Holzman's letter regarding fabrication of an elongated endotracheal tube for intubation in a patient with tracheal resection.3 Because of the size of some species encountered in veterinary anesthesia, veterinary anesthesiologists frequently use specialized endotracheal tubes to maintain inhalation anesthesia. One of the vendors of anesthesiology and respiratory care devices for human use entered the veterinary market by applying the materials and expertise utilized for manufacture of human endotracheal tubes to needs of veterinary anesthesia (tubes of up to 30 mm ID and up to 90 cm length).

They have manufactured their veterinary product line from the same materials and under the sameCurrent Good Manufacturing Practices guidelines promulgated by the Food and Drug Administration as they have their human product line.* The only difference is that the human tubes are packaged steriley before shipment, while the veterinary tubes are not.

When anesthetizing neonatal foals, tracheal tube diameters of 6–10 mm ID are sufficient. However, because of the length of the foal's head, use of commercial human endotracheal tubes is not possible because they are too short, particularly if intubation is performed nasally.4 One of the vendor's products (fig. 1) is used for these cases and could have potential application in the case described by Holzman.


FIG. 1. Commercial 7.0 mm I.D. by 55-cm length endotracheal tube.