are only used infrequently and the effects are transient. The misleading readings for isoflurane, enfurane, and CO₂ partial pressures with these types of propellants are easily obviated by not dispensing medication with such a propellant while the mass spectrometer is sampling. When sampling is continuous, removing the sample port briefly, 10–15 s, from the circuit during administration of medication would prevent misreadings. Readings from a clinical, fixed collector mass spectrometer system must be interpreted cautiously when gases that the system was not designed for are being used.

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Using a Priming Dose of Relaxant Is Not New

To the Editor:—The use of a priming dose of relaxant as a means of improving conditions for tracheal intubation is a novel but not a new idea. The use of a “test” dose of tubocurarine (usually 5 mg) given 2–3 min before the main dose was taught as a routine procedure in Liverpool when I was a resident in the late 1940s, early 1950s. It was also accepted practice to give the full “relaxing” dose of tubocurarine (usually 20–40 mg) immediately before the thiopentone. I have personally used this technique as a routine for intubation in thousands of patients with all the available competitive relaxants and, with the exception of laudexium, all produced good conditions for passage of the tube. It is particularly valuable with vecuronium, after which intubation can be as easy as with suxamethonium.

The reasons for this technique, as originally described by Gray and Halton, were twofold. The early administration of tubocurarine was to identify patients with latent myasthenia gravis where the period of apnoea would be very prolonged. Sensitivity would be suspected if the response to the test dose was other than mild ptosis. Giving the full relaxant dose before the thiopentone aims at synchronizing the maximum effect of two drugs with different onset times. This technique demands an “open vein”—a practice not generally accepted at the time—and with this precaution I have never encountered a patient who complained of breathlessness at induction.

The detailed monitoring used in the recent papers of Schwarz et al. and Mehta et al. was not available when Gray popularized his technique (which has appeared in standard British textbooks). While the rationale behind the use of relaxants in divided doses may be different in recent studies, nevertheless the use of the technique by earlier pioneers should be acknowledged.

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Patients with Huntington's Chorea May Respond Normally to Succinylcholine

To the Editor:—A single report of prolonged paralysis in a patient with Huntington's chorea is the basis for the recommendation that succinylcholine be avoided in such patients. We recently found it necessary to use succinylcholine to facilitate endotracheal intubation in a patient who had this hereditary disorder, and wish to report our experience.

A 46-yr-old, 55-kg man came to surgery for placement of a feeding gastrostomy. He had a 10-yr history of dementia and chorea, which had progressed to include swallowing dysfunction, with chronic drooling, and regurgitation. His father, brother, maternal aunt, and two paternal uncles had died from Huntington's chorea; a daughter was also affected. Medications included haloperidol 2 mg daily and benztropine 1 mg bid. He appeared awake but did not respond appropriately to verbal commands, was unable to communicate, and made frequent, unpredictable, forceful athetoid movements. The remainder of the physical and laboratory examination was unremarkable. Because he was completely uncooperative, we felt that local or regional anesthesia would not be feasible. To minimize the risk of aspiration during induction of general anesthesia, we planned "rapid-sequence" endotracheal intubation with cricoid pressure and muscle paralysis.

We applied surface electrodes over the patient's ulnar nerve at the wrist, applied supramaximal stimulation at 1 Hz, and measured the resulting muscle twitches using a Grass FT-10 strain gauge. After "preoxygenation," we administered fentanyl (150 μg), thiopental (200 mg), and succinylcholine (35 mg, 0.6 mg/kg); with cricoid pressure applied, we accomplished tracheal intubation without difficulty. Nitrous oxide (70%) in oxygen maintained anesthesia, while we documented recovery from succinylcholine paralysis by continually recording muscle twitch strength. When twitch recovery was complete, we added isoflurane (up to 2% inspired) for the remainder of the 90-min procedure. Emergence from anesthesia was smooth; extubation and recovery were uneventful.

Analysis of the twitch-strength record revealed that muscle strength returned to 50% of control 555 s after succinylcholine and to 90% of control within 660 s. These recovery times are within the ranges (mean ± 2 SD) previously reported for normal patients receiving similar doses of succinylcholine.

Gualandi and Bonfani's report of a patient with Huntington's chorea in whom succinylcholine 50 mg lasted 2 h has been excerpted in case reports as well as textbooks. Anesthesiologists subsequently avoided giving succinylcholine to patients with Huntington's chorea. The unproven association has become accepted as fact!

Conversely, the diminished cholinesterase activity in Gualandi and Bonfani's patient may have been completely unrelated to his Huntington's chorea. Even in the absence of a "linkage" between Huntington's chorea and atypical cholinesterase, a patient with Huntington's chorea has an approximately 1 in 2,500 chance of being homozygous for atypical cholinesterase. Our findings suggest that patients with Huntington's chorea recover normally from succinylcholine and that this drug may be administered safely to these patients when clinically indicated.

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