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Test Dose for Prediction of Mivacurium Sensitivity in the Patient with Atypical Plasma Cholinesterase

To the Editor:—Mivacurium is a short-acting nondepolarizing muscle relaxant hydrolyzed by plasma cholinesterase. The *in vitro* rate of hydrolysis is approximately 70% of that of succinylcholine. Østergaard *et al.*^{1,2} showed that mivacurium, in a dose as small as $0.03 \text{ mg} \cdot \text{kg}^{-1}$, can produce an intense and prolonged neuromuscular block in patients with atypical esterase; the resulting block could not be antagonized by neostigmine except after the administration of human plasma cholinesterase.¹⁻³

Mivacurium may be the relaxant of choice in patients undergoing short surgical procedures, and it is frustrating to have unanticipated prolonged mivacurium-induced neuromuscular block after such procedures. Thus, it may be desirable to use a test dose of mivacurium to predict unusual sensitivity. Mivacurium in the general population has an ED_{50} and ED_{95} of approximately 0.043 and $0.075 \text{ mg} \cdot \text{kg}^{-1}$, respectively. A dose of $0.15 \text{ mg} \cdot \text{kg}^{-1}$ (twice the ED_{95}) is recommended for tracheal intubation. Our report suggests the use of a test dose of $0.015 \text{ mg} \cdot \text{kg}^{-1}$ mivacurium, which is one-tenth the recommended intubating dose. In five adult female patients scheduled for minor gynecologic procedures (dilation and curettage or tubal ligation), a preoperative blood sample showed a plasma cholinesterase activity and dibucaine number (DN) within the normal range (75-85). Anesthesia was induced with $2.5 \text{ mg} \cdot \text{kg}^{-1}$ propofol, and neuromuscular transmission was monitored by the Datex Relaxograph. In all five patients, administration of $0.015 \text{ mg} \cdot \text{kg}^{-1}$ mivacurium produced 0-15% neuromuscular block within 3-4 min. No block was observed in two patients, 5% block in one patient, 10% block in one patient, and 15% block in the last patient. This was considered a normal response to the test dose, and hence, the intubating dose of $0.15 \text{ mg} \cdot \text{kg}^{-1}$ mivacurium was administered and was followed by complete neuromuscular block within 2-3 min. Spontaneous recovery of neuromuscular transmission up to 25-50% was noted after 10-20 min.

The neuromuscular block of the test dose in the five normal patients was compared to that achieved in a 29-yr-old woman scheduled for tubal ligation who was known to be homozygous for the atypical genes (DN 23). In this patient, after intravenous propofol administration, $0.015 \text{ mg} \cdot \text{kg}^{-1}$ mivacurium produced about 95-98% neu-

romuscular block, and the patient showed 10% recovery of neuromuscular transmission after 30 min. At that time, $0.05 \text{ mg} \cdot \text{kg}^{-1}$ neostigmine reversed the block. Figure 1 shows the response to mivacurium in the atypical patient, as compared to one of the normal patients.

Our observations suggest that the test dose of mivacurium can predict unusual sensitivity to mivacurium in patients with atypical esterase while producing a degree and duration of block that can be reversed easily with neostigmine. In patients with normal esterase, the test dose produces only minimal or no neuromuscular block and may serve as a priming dose that enhances the onset of the subsequent intubating dose of mivacurium.

In conclusion, the use of a test dose of mivacurium of $0.015 \text{ mg} \cdot \text{kg}^{-1}$ (about 1 mg in the average adult) can predict the unanticipated intense and prolonged mivacurium-induced neuromuscular block in patients with atypical plasma cholinesterase.

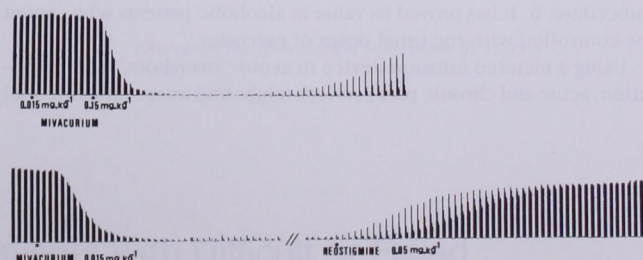


Fig. 1. Electromyographic response to ulnar nerve stimulation by a train-of-four every 20 s. In the patient with normal esterase (top), the test dose of $0.015 \text{ mg} \cdot \text{kg}^{-1}$ mivacurium produced no block, whereas the subsequent intubating dose, $0.15 \text{ mg} \cdot \text{kg}^{-1}$, resulted in complete block, which showed 50% recovery after 15 min. In the patient with atypical esterase (bottom), the test dose produced complete neuromuscular block, which showed 10% recovery after 30 min, when the block could be reversed with neostigmine $0.05 \text{ mg} \cdot \text{kg}^{-1}$.

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Intravenous Alcohol In 1945 and Beyond

To the Editor:—I reflected on the correspondence from Bergman,¹ "Intravenous Alcohol in 1831," particularly the statement, "One can speculate that, if Dupuy had used a larger dose of alcohol and achieved unconsciousness in his horses, some perceptive reader of the report might have attempted to mitigate surgical pain with this technique."

Although I was not aware of Dupuy's investigation in horses,² surgical pain in humans has been "mitigated" with alcohol.³ The summary of this article³ states, "1. Five per cent and 10 per cent alcohol intravenously increases the caloric intake and has special value in those cases with inanition. 2. It is a potent sedative and analgesic, and can be substituted for the opiates and other forms of sedation. 3. Sedation is not attended with depressed respiration. 4. It may be used in cardiac patients with relative safety because of its vasodilatory effect and minimal effect on the blood pressure. 5. It has a definite place in regional anesthesia as a supplement during the operative procedure. 6. It has proved its value in alcoholic patients who cannot be controlled with the usual doses of narcotics."

Using a metered infusion device to avoid "overshoot" and inebriation, acute and chronic pain services might find intravenous alcohol

a replacement for patient-controlled analgesia or even continuous epidural block using opioids and/or local anesthetics.

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