TITLE: PRIMING BEFORE VECURONIUM: CURARE VS. VECURONIUM

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Introduction Vecuronium (V) is often used as an alternative to succinylcholine during rapid sequence induction. Priming doses of relaxant (1,2) speed the onset of neuromuscular blockade; however, they often produce unpleasant muscular weakness (3). Researchers have claimed that administration of prejunctionally acting relaxants (e.g., curare (dTC)) will potentiate alcuronium, a compound similar to V (4). The present study determines whether priming with dTC more effectively potentiates V than an equivalent priming dose of V.
Methods After IRB approval, in part 1 of our study, we randomly assigned 20 consenting patients to receive either dTC (70 mcg/kg) or placebo in a blinded fashion. ED_{95} of V was determined in both groups by administering 10 mcg/kg boluses until 95% twitch depression was measured. Trains of 4 supramaximal stimuli were delivered to the ulnar nerve while adductor pollicis isometric tension was measured using a strain gauge. In part 2 of our study, using the ED_{95} data collected in part 1, we randomly assigned 20 additional patients to receive either dTC (70 mcg/kg) or the equivalent dose of V (in a blinded fashion) 3 min prior to an intubating dose of V (2 x ED_{95}). Data are reported as means+SEM. Groups were compared using repeated measures ANOVA and student's t test.
Results ED_{95} of V was 46±3 mcg/kg after dTC and 63±2 mcg/kg after placebo. The difference, 17 mcg/kg V, was used in part 2 as the V priming dose. In part 2, after both the priming and intubation relaxant doses were given, the mean twitch depression was greater at all time points in dTC group than in the V group. Intubating conditions (i.e., twitch amplitude depression of 95% or greater) were obtained in 101±7 sec (dTC+V) or 113±12 sec (V+V). The group difference at this individual time point did not achieve statistical significance. Mean recovery from block was less at all time points in the dTC group than in the V group. The delay to 25% twitch height recovery was 55±5 min (dTC+V) and 46±4 min (V+V). These were not significantly different.
Discussion The use of priming doses and drug combinations have been advocated to speed the onset of neuromuscular blockade (1,2,4). Our study, which combines both techniques, confirms that dTC speeds the onset of subsequently administered V at least as much as a larger dose (as a fraction of the ED_{95} dose and in terms of its ability to reduce twitch amplitude on its own) of V. However, we were unable to confirm the common clinical impression that relaxant combinations produce intubating conditions more rapidly than comparable doses of a single relaxant. Our dTC priming dose (which was only marginally larger than a "defasciculating" dose) was not associated with subjective weakness in any of these patients (in contrast to reported problems with single relaxant priming (3,5)). Moreover, the dTC prime appeared to produce less effect on the T4 than the V prime. Thus, dTC may be a safer and (slightly) more effective priming drug than V (prior to V) for rapid sequence induction.

References
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TITLE: **EFFECT OF RAPID SEQUENCE INDUCTION ON LOWER LEG FASCIAL COMPARTMENT PRESSURE**

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Osteofascial compartment syndrome (CS) frequently develops in patients with a closed tibial fracture, and fasciotomy often is required to preserve viability of the limb. Since these patients present to the operating room emergently, induction of general anesthesia requires a rapid sequence induction technique (RSI), usually with thiopental and succinylcholine. We wished to determine if fasciculation of skeletal muscle and/or hemodynamic instability would effect compartment pressure.

After Institutional Review Board approval, seven consenting patients who were 21 to 42 years old were studied. All patients had closed tibial fracture and symptoms compatible with compartment syndrome; each required a rapid sequence induction of anesthetic. A 14G introducer was placed percutaneously into a muscle compartment at risk for CS. A transducer-tipped catheter (Camino Laboratories) was inserted through the introducer and attached to a digital monitor and a recorder. After administration of 100% oxygen for 3 to 5 min., application of cricoid pressure, and injection of thiopental (2.0 to 4.0 mg/kg, IV) followed by succinylcholine (1.0 to 1.5 mg/kg, IV), the trachea was intubated. Anesthesia was maintained with isoflurane and 70% nitrous oxide and oxygen. Fentanyl was infused to supplement anesthesia when necessary. Compartment pressure was recorded prior to, and for 15 min after, induction of anesthesia. Data are summarized as mean ± SD; differences were statistically evaluated using an analysis of variance.

Rapid sequence induction caused a significant increase in compartment pressure: from 36±8 mm Hg to 49±12 mm Hg (p=.003). Fifteen minutes later, the average compartment pressure was 37±9 mm Hg. Rapid sequence induction lead to a significant rise in compartment pressure in all patients. The increase was transient in five patients and persistent in one.

Increase in compartment pressure during anesthetic induction may be caused by increased muscle tone upon administration of succinylcholine, hypertension secondary to laryngoscopy and intubation, or by increased muscle blood flow secondary to hypoventilation and increased $PaCO_2$. The transient rise in compartment pressure probably is not significant in the majority of patients and does not warrant alteration of anesthetic technique. However, the anesthesiologist should be aware that a rapid sequence induction of anesthesia consistently increases fascial compartment pressure. This increase in pressure may be detrimental to muscle blood flow in some patients.