Succinylcholine is Different in Humans than in Dogs

To the Editor.—Although Lanier et al.1 have indicated that the results of their study, “The cerebral stimulating effects of succinylcholine in dogs,” may not be applicable to the human situation, they certainly suggest that the results are directly transferable. I would like to offer another reason why the results of the injection of 1 mg/kg of succinylcholine intravenously in dogs may not be relevant to the clinical situation. Any investigator who has looked at the dose of succinylcholine necessary for neuromuscular blockade in the dog has seen that a bolus dose of 0.1–0.2 mg·kg\(^{-1}\) intravenously is sufficient to produce adequate muscle relaxation for almost any intervention with the duration of action relatively equivalent to a dose of 1 mg·kg\(^{-1}\) in humans. A dose of 1 mg·kg\(^{-1}\) in the dog will last up to 30 min. Consequently, it is likely that the effects of such a dose on muscle spindles, or the CNS directly, will be greater in the dog than an equivalent dose in humans. Although initially it was thought that the reason for this was a different plasma cholinesterase in canines, this has not been entirely substantiated.2,3 It is still unclear as to why there is the difference in kinetics, but there appears to be no controversy as to the existence of this difference. I believe that clinicians should take this species difference into account before transferring the results of this investigation to the clinical situation.

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In reply.—As stated by Dr. Merin, dogs are more sensitive than humans to the neuromuscular relaxant properties of succinylcholine (SCh); however, this information does not allow us to predict species differences in the cerebral effects of SCh. We hypothesized that cerebral stimulation following SCh was primarily related to increases in afferent muscle spindle activity and secondarily to SCh-induced increases in arterial P\(_{\text{CO}_2}\). Information regarding the potency of SCh as a relaxant is based on SCh effects on extrafusal, contractile muscle fibers, while EEG arousal following SCh is felt to be related to the activity of intrafusal, afferent muscle fibers.3–5 The effects of SCh on afferent fibers are reported to be qualitatively dissimilar to its effects on extrafusal fibers.4

While large doses of SCh can be expected to provide prolonged neuromuscular paralysis, large doses may offer no more cerebral stimulation than smaller doses. When compared with modest doses of SCh, excessive doses of SCh have been reported not to further increase afferent muscle spindle activity5 or muscle CO\(_2\) production.7

The dog model for evaluating the intracranial pressure (ICP) increases following SCh has now been criticized on all fronts. While Dr. Merin apparently feels the dog model will overestimate the cerebral effects of SCh, others have criticized the dog model for its underestimation of the cerebral effects of SCh.8 The purpose of our study was not to compare a dose–response curve in dogs and humans, but instead to gain insight into the mechanisms by which SCh may alter cerebral function. In this regard, the results of our study in dogs given SCh 1 mg·kg\(^{-1}\) closely parallel the results in humans given SCh 1 mg·kg\(^{-1}\). This dose of SCh produced 5–10 min of EEG arousal in humans receiving 0.7 to 1.3 MAC halothane,3,4 and 5 min of EEG arousal in five of six dogs receiving 1.0 MAC halothane.3 In lightly anesthetized humans, Minton et al. have recently reported that SCh 1 mg·kg\(^{-1}\) produced clinically meaningful and statistically significant increases in ICP that peak 3 min after SCh administration.9 ICP increases were not related to central venous pressure increases and were prevented by prior onset of nonde-
polarizing neuromuscular blockade. These findings are strikingly similar to those noted in our dog study.

We share Dr. Merin’s concern that Sch-induced cerebral stimulation in the dog may differ somewhat in intensity and duration when compared with humans. However, the alert clinician should be aware that Sch may produce clinically meaningful cerebral effects in humans. While differences in the cerebral response to Sch between healthy dogs and brain-injured humans may be attributed to differences in species susceptibility, probably far more important are variations in anesthetic depth, baseline cerebral function, cerebral compliance, and the prior administration of nondepolarizing neuromuscular relaxants.

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An Additional Use for the Esophageal Stethoscope

To the Editor.—During endotracheal intubation in adults, occasional tearing of the cuff occurs, particularly when McGill’s forceps are used for assistance. We would like to share one solution to this problem. We now insert a well-lubricated pediatric esophageal stethoscope (size 12 Fr) through a new endotracheal tube (ideally 7.0 mm ID or larger) and advance it 2-3 cm past the end of the tube. With direct visualization, the McGill's forceps are again used, but now to grasp and guide the esophageal stethoscope through the vocal cords; the endotracheal tube is now advanced over the esophageal stethoscope through the vocal cords. The esophageal stethoscope is then removed from the endotracheal tube, auscultation performed, and the tube secured when bilateral breath sounds are determined to be equal.

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