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In Reply:—We appreciate the interest of Savarese *et al.* in our publications about antagonism of mivacurium-induced paralysis.[1,2] We agree that antagonism of neuromuscular blockade is a complex process that involves both the pharmacokinetics and the inherent "reversibility" of the muscle relaxant. For most nondepolarizing muscle relaxants, administration of either edrophonium or neostigmine presumably does not alter the pharmacokinetics of the muscle relaxant; we demonstrated this for vecuronium.[1,2] The observation by Cook *et al.** that "edrophonium . . . [has] no effect on the *in vitro* metabolism of mivacurium" led to the assumption that mivacurium's elimination would not be affected *in vivo* by edrophonium.[3] However, our recent studies in patients demonstrate that both edrophonium and neostigmine alter mivacurium's elimination,[1,2] thereby indicating the greater complexity of antagonism of mivacurium. In that mivacurium is eliminated by cholinesterase, we are surprised that so little data regarding the *in vivo* effects of cholinesterase inhibitors were available before mivacurium's release to the clinical community.

Savarese *et al.* question the direct clinical applicability of our studies. Studies are sometimes designed to isolate the contribution of single variables (*e.g.*, intrinsic "reversibility" of the muscle relaxant) while maintaining constant concentrations of the anesthetic, muscle relaxant, and end-tidal PCO_2. We have acknowledged this "limitation" of our studies. A common approach to examine antagonism of muscle relaxants is to give an antagonist during recovery from a single bolus dose of a muscle relaxant. Such a design might closely resemble some clinical practice. However, it does not replicate those anesthetics during which a muscle relaxant is given repeatedly or by infusion and cumulative effects of the muscle relaxant (even mivacurium)[4] might hinder recovery. To be clinically relevant, studies should examine antagonism under a variety of adverse conditions, including prolonged administration, profound paralysis, organ dysfunction, and hypothermia.

Savarese *et al.* claim that antagonism of block >90% is probably inappropriate. Although this may be true theoretically, antagonism of profound block is probably common practice, as shown by the existence of several studies in which antagonism of profound paralysis was examined. In the absence of the sophisticated monitoring tools available to researchers, clinicians are probably unable to distinguish between twitch depression <90% and >90%. In support of this, clinicians consistently underestimate residual paralysis during recovery[5]; however, similar studies to assess profound paralysis are lacking.

Are we "way out on a limb" speculating about other routes of elimination of mivacurium? If so, we are in good company—Savarese *et al.*[6] speculated that their results regarding mivacurium's duration of action "may suggest additional routes of metabolism and/or elimination." Finally, Savarese *et al.* suggest that appropriate antagonism of mivacurium requires stopping mivacurium administration and waiting for the presence of two twitches in a train-of-four. We eagerly await studies of this regimen.

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Dennis M. Fisher, M.D.
Professor of Anesthesia and Pediatrics
Janos Szenohradszky, M.D.
Assistant Professor of Anesthesia
University of California, San Francisco
San Francisco, California 94143-0648
Paul S. Hart, M.B.
London, England

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Peripheral Nerve Stimulator for Unassisted Nerve Blockade

To the Editor:—The introduction of peripheral nerve stimulators (PNS) in the practice of regional anesthesia has resulted in a debate over whether there are any advantages to their use over the paresthesia technique. One obvious advantage is that PNS cause minimal discomfort to patients, because the low stimulating currents used (0.1–2.0 mA) readily stimulate the larger A-α motor fibers more than C pain fibers.[1] This is in contrast to paresthesia technique, which by its nature will cause varying degrees of discomfort. A second advantage of PNS is that patient cooperation is not needed during the procedure, so a block can be performed in an anesthetized patient. The incidence of nerve damage may be decreased compared with paresthesia technique.[3–8] The success rate with use of PNS is equal to or greater than that from eliciting paresthesias.[9]

Besides the initial cost of the stimulator and the continued expense of the insulated needles, a frequently cited disadvantage associated with the use of the nerve stimulator is a need for additional personnel.[10] This is because PNS usually require frequent changes in the intensity of the stimulating current during needle advancement toward the nerve. Because most anesthesiologists prefer to use sterile technique while performing a nerve block, the use of PNS is generally thought to require an extra person for manipulation of the output current.[10,11] Whereas in a teaching institution this is usually not an important issue, in a busy private practice it may present a significant disadvantage, because most anesthetic practices do not have the luxury of involving an additional person in performing regional blockade.

With this in mind, we invented a device with a foot pedal to control the current applied during the performance of the regional nerve blockade.* The invention is a combination of the Dual Stim Plus nerve stimulator (model NS-2CA, Life-Tech, Houston, TX) and a modified KORG two-channel volume pedal (model KVP-001; fig 1).

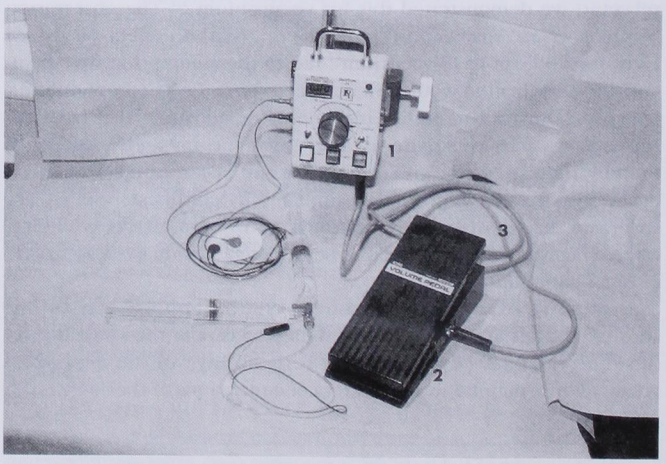

Fig. 1. (1) Peripheral nerve stimulator; (2) foot unit for control of the intensity of the stimulating current; (3) cable for electrical connection of the foot unit and the peripheral nerve stimulator.

* Peripheral Nerve Stimulation Device for Unassisted Nerve Blockade (US patent Application serial No. 08/419,419). St. Luke's-Roosevelt Hospital Center is the owner of proprietary rights in the device. If those rights are licensed, Dr. A. Hadžić and Dr. J. Vloka, as coinventors, may receive a share of the net profits, if any, on royalties paid to the Hospital Center.