There was one failure after three trials. All procedures were accomplished with no complications such as arterial puncture or pneumothorax.

The high rate of success (99.2%) of the present technique was comparable to that previously reported by us (99.3%)[2] and suggests that the characteristics of our curved needle are suitable for piercing only the anterior wall of the IJV.

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Atropine Facilitates Neostigmine Reversal of Vecuronium-induced Neuromuscular Blockade

To the Editor:—In their article, Baurain *et al.*[1] demonstrate facilitation of neostigmine reversal of vecuronium-induced neuromuscular blockade by larger doses of concomitantly administered atropine (15–20 μg/kg) when compared with smaller doses of atropine (10 μg/kg) in anesthetized patients. I wish to propose a pharmacokinetic explanation for this observation. Atropine has a more rapid onset of action than neostigmine. Administered simultaneously, I would expect an increase in heart rate and cardiac output to precede cardiovascular effects of the anticholinesterase. Was this change significantly greater in the higher dose atropine group? Increased delivery of the neostigmine to muscle may have influenced recovery of neuromuscular function. The conclusion of the study is based upon a single measurement of 100-Hz tetanic fade, 15 min after the atropine and neostigmine doses were administered. No measurement of tetanic fade was done after 15 min, so there is no basis on which to compare the recovery time for the tetanic fade between the groups.

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In Reply:—Unfortunately, cardiac output was not measured as part of our study.[1] Thus the influence of atropine upon blood flow to muscle must, for the moment, remain hypothetical.

The reasons for limiting tetanic fade stimulations to one measurement performed 15 min after the administration of the atropine and neostigmine mixture were the following: first, high-frequency stim-

ulations produce marked changes in subsequent low-frequency elicited mechanical responses—twitch height[2] or train-of-four (TOF).[3] Therefore, tetanic stimulation cannot be mixed with repetitive TOF recordings. Second, when recovery from an infusion of neuromuscular blocking drug is monitored using twitch height, the peak action of neostigmine appears to be centered between 10 and 15 min.[4] Third, in many clinical conditions in which atropine and neostigmine mixtures or atropine and edrophonium mixtures are used, more than 10 min is required to reach a TOF value greater than 0.70.[5] Fourth, 15 min seems to represent for many anesthesiologists a realistic time to assess the effect of the reversal agents.

Thus, since only one high-frequency tetanic stimulation pattern can be used, it seems quite logical to do so 15 min after giving atropine and neostigmine.

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Hyperkalemia from Nonelectrolyte Solutions

To the Editor:—A "regulatory volume decrease" through a shift of potassium from cells was suggested by Hirose *et al.* to explain the increase of the serum potassium concentration from nonelectrolyte solutions, such as mannitol and glycine.[1,2] Studies made at this institution provide additional information about the nature of this hyperkalemia.

The increase in potassium is three times greater following intravenous infusion of isosmotic (2.2%) glycine compared to isosmotic (5%) mannitol solution in male volunteers, despite similar degrees of hyponatremia.[3] This suggests that increased serum potassium is *not* related to hyponatremia *per se* and, hence, cannot be explained by dilution acidosis.

Glycine solution does not cause hemolysis or impair urinary excretion of potassium,[3,4] and therefore, a shift of intracellular potassium remains the only possible cause of this hyperkalemia.

The intensity of the hyperkalemic response can be obtained by measuring serum potassium at the end of a short (20-min) intravenous infusion of nonelectrolyte solution. Results from such experiments in young male volunteers[3] and in patients undergoing prostatectomy[4] show that increase in serum potassium was 0.91 ± 0.35 mM/L from a 2.2% solution and 0.36 ± 0.26 mM/L from a 1.5% solution of glycine.[3,4] The effect of tonicity would promote a more pronounced cell swelling from the hypo-osmotic solution, and this also is evidenced by a smaller dilution of the serum sodium during infusion of 1.5% glycine. However, if cell swelling triggers the hyperkalemia, which is implied by Hirose *et al.*, one would expect the serum potassium concentration to correlate inversely with the glycine concentration of the infusion; but this is not the case.

Our data suggest that hyperkalemia is related directly to how much nonelectrolyte solution is transported into the cells rather than to the degree of cell swelling that triggers the "regulatory volume decrease." This hypothesis is consistent with the referenced changes in serum potassium. As the distribution volume of glycine at the end of glycine infusion is double the size of the extracellular space,[3,5] one can assume that more glycine enters the cells during infusion of 2.2% glycine, and therefore, hyperkalemia is more pronounced.

This hypothesis is supported further by experiments in sheep. The distribution volumes at the end of infusions of 1.8 L 2.2% glycine solution and 4.0 L 1.5% glycine were only 15–19 L[6] and about 16 L, respectively. These volumes are similar in size to the expected extracellular space in these animals, which indicates that a surplus amount of glycine is transported into the cells much more slowly than in humans. There was no increase in serum potassium with the glycine, which is consistent with the view that hyperkalemia depends on the rate of intracellular accumulation of the nonelectrolyte solution. On the other hand, if hyperkalemia was triggered by cell