Prevention of Hypokalemia during Axillary Nerve Block

To the Editor—We read with interest the report of Toyoda et al. concerning the prevention of beta-2 sympathomimetically induced hypokalemia resulting from the administration of epinephrine-containing lidocaine for axillary block. Several points relevant to their study were not mentioned in the manuscript.

An interaction between nonspecific beta blockade (propranolol) and epinephrine has been recognized. Potentially disastrous hypertension and reflex bradydyrrhythmias may result from the unopposed alpha-adrenergic agonist effects of epinephrine in patients who have received beta-adrenergic blockers. Perhaps an accidental direct intravenous injection of epinephrine containing local anesthetic solutions during axillary block may result in similar catastrophic responses.

Second, we question the clinical significance of beta-2 adrenergic induced decreases in serum potassium in this setting. No author has implicated arrhythmias secondary to this specific phenomena with worsened outcome or significant adverse effects. In fact, no arrhythmias were detected in the study by Toyoda et al. In contrast, Lampman et al., in a study of 35 patients receiving epinephrine and propranolol infusions for the measurement of insulin resistance, noted potentially significant arrhythmias in six of the 35 subjects felt to be secondary to the concurrent use of these agents.

Acute decreases in serum potassium may occur from a number of causes in the anesthetized state. Certainly, intravenous respiratory alkalosis and corresponding hypokalemia is a common event. Serum potassium falls abruptly with the onset of hyperventilation. Despite this time course, in all the reports of arrhythmias secondary to mechanical hyperventilation, the arrhythmias did not appear for hours or days. Also, hypokalemia may develop from administration of d-tubocurarine, gallamine, thiopental, halothane, and thiopental-nitrous oxide. Indeed, epinephrine levels generally increase under general anesthesia and postoperatively as a result of surgical stress. Should all persons receiving major surgical procedures under general anesthesia undergo beta-adrenergic blockade?

Last, we question the use of group 2 (received propranolol) as the control group. Although no statistical difference was shown between serum potassium before and after propranolol, there appears to be a tendency for potassium levels to increase. There is evidence to suggest that administration of propranolol is capable of inhibiting the effect of basal epinephrine levels on tissue potassium uptake. This evidence would seem to indicate that a control group consisting of patients receiving 1% lidocaine without either epinephrine or propranolol should have been included.

Although this study nicely illustrates basic science mechanisms, we feel that, in this case, the cure may be worse than the disease itself. Most certainly, there are instances in which beta-2-adrenergic induced hypokalemia may be a serious problem, such as in patients on digitalis and/or hypokalemic. To advocate, however, the general implementation of a potentially dangerous therapy for a laboratory aberration of presumed but unproven consequence seems to us a dangerous step.

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REFERENCES


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