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Prevention of Hypokalemia during Axillary Nerve Block

To the Editor:—We read with interest the report of Toyoda *et al.*[1] 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.[2-4] Potentially disastrous hypertension and reflex bradydysrhythmias may result from the unopposed alpha-adrenergic agonist effects of epinephrine[2,4] in patients who have received beta-adrenergic blockers. Perhaps an accidental direct intravascular 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.*,[4] 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, iatrogenic respiratory alkalosis and corresponding hypokalemia is a common event. Serum potassium falls abruptly with the onset of hyperventilation.[5] Despite this time course, in all the reports of arrhythmias secondary to mechanical hyperventilation, the arrhythmias did not appear for hours or days.[5] Also, hypokalemia may develop from administration of d-tubocurarine, gallamine, thiopental, halothane, and thiopental-nitrous oxide.[6] Indeed, epinephrine levels generally increase under general anesthesia and postoperatively as a result of surgical stress.[7] 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.[8] 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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In Reply:—We thank Drs. Lunn and Narr for their comments on our report.[1] We admit that we failed to describe the potentially paradoxical hypertensive response relating to the interaction of epinephrine and propranolol, though rarely reported.[2-5]

Use of the noun "prevention" in our title has obviously led to misunderstanding. It was not our intention to imply routine prophylaxis. It was shown by Brown *et al.*[6] that epinephrine-induced hypokalemia results from β2-adrenoceptor stimulation. Since a selective β2-receptor antagonist was not available, the available nonselective β-adrenoceptor blocker propranolol was employed to establish the hypothesis that hypokalemia in the course of regional nerve block is produced by lidocaine containing epinephrine. Parenthetically, we have since learned that

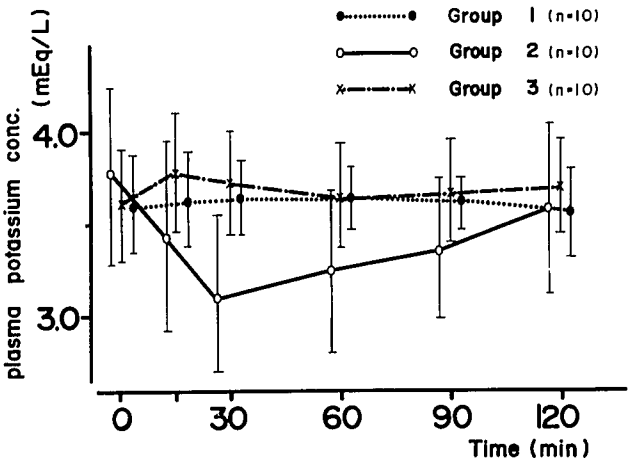

FIG. 1. Changes in plasma potassium concentration after axillary nerve block. Group 1: blocked with plain 1% lidocaine. Group 2: blocked using 1% lidocaine with epinephrine 1:200,000. Group 3: pretreatment with propranolol 2 mg iv and nerve block was done by the same agent in group 2.

Hahn[7] also observed development of hypokalemia during the course of epidural nerve block where epinephrine was used, although he did not implicate epinephrine as the source.

We have given approximately 1800 axillary nerve blocks with lidocaine containing epinephrine, but we did not use propranolol prophylactically. We have not encountered dysrhythmias severe enough to require treatment. Furthermore, we did not recommend pretreatment with propranolol but suggested avoidance of epinephrine in patients with K^+ deficiency. The incidence of potentially significant arrhythmias in Lampman's[8] study might have been influenced by the large dose of propranolol employed (5 mg IV followed by a continuous infusion of 0.08 mg/min as compared with 2 mg IV) or a drug interaction.

In our study, the results of blood gas analysis indicated that respiratory alkalosis did not develop during the course of operation, and was thus ruled out as a cause of hypokalemia. Hypokalemia following general anesthesia and use of neuromuscular blockers is not the issue here. It is not our practice to use beta-adrenergic blockers during general anesthesia or the postoperative period unless absolutely indicated.

In regard to a study of lidocaine without either epinephrine or propranolol, since 1987, we have measured changes in plasma K^+ level resulting from lidocaine and epinephrine in 1:200,000 concentration during axillary nerve block with the same technique. Changes in plasma K^+ are shown in figure 1. Results of the reported experiments indicated that plain lidocaine did not produce changes in plasma K^+ (group 1), lidocaine and epinephrine 1:200,000 concentration also produce hypokalemia (group 2), the latter similarly prevented by pretreatment with propranolol heretofore described (group 3). The amplitude of the T wave was decreased in all cases of group 2 at 30 min after blockade. A U wave was observed in two cases of group 2.

In conclusion, we as anesthesiologists should be aware of the potential dangers of drug interactions and that lidocaine containing epinephrine produces hypokalemia. Therefore, we must closely observe not only the patient, but also the ECG during operation even with regional nerve block.

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The Needle Tilt Test: An Aid to Epidural Needle Insertion

To the Editor:—Many techniques and devices have been recommended to help identify the loss of resistance that signals that a needle has entered the epidural space. But uncertainty often arises earlier when an advancing needle is passing through compliant tissue spaces