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In Reply:—We would like to thank Dr. Roy for his careful scrutiny in methods utilized in our recent study. As he correctly points out, a gastric tube in a patient receiving mask anesthesia may increase the risk of regurgitation. However, this potential complication will not apply to the patients in our study, as all the patients were intubated. We apologize for not mentioning this aspect of anesthetic management, about which we should have said, "Following satisfactory induction of anesthesia with sodium thiopental, and endotracheal intubation with the aid of succinylcholine, a No. 18 Salem sump tube was passed into the stomach . . ."

As per Dr. Roy's comments with regard to airway management, potential risk of aspiration pneumonitis, and pharmacologic prophylaxis, we believe that, theoretically, most patients undergoing surgery under general anesthesia face the potential risk of acid aspiration, and may benefit from pharmacologic prophylaxis. However, there is, as yet, no hard evidence that the use of any of the pharmacologic agents reduces anesthetic morbidity and mortality from aspiration.

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Stress and Hypokalemia

To the Editor:—Shin *et al.*, in a recent clinical report, speculate as to the mechanism of hypokalemia in trauma patients following hypotension and resuscitation.[1] Unfortunately, serum catecholamine levels were not measured in their study. Extrarenal adrenergic modulation of potassium (K^+), first reported in 1936,[2] has been reestablished as a mechanism for hypokalemia. Brown showed that a 20- to 30-fold increase above normal of circulating epinephrine, but not isoproterenol, depresses serum K^+ by approximately 0.8 meg $\cdot$ l^{-1}, and concluded that K^+ is lowered by beta-2 receptor stimulation.[3] Elevation of epinephrine of this order of magnitude would likely be present in severely traumatized patients. A similar association between sympathoadrenal hyperactivity and hypokalemia has been observed in association with other acutely stressful clinical events; for example, acute myocardial infarction.[4]

The opposing effects of alpha- and beta-adrenergic receptor stimulation has been demonstrated, and these are reversed by specific receptor blockade.[5] Therefore, the balance between alpha- and beta-receptor stimulation determines the direction of adrenergically mediated changes in K^+. We recently reported that the stress of median sternotomy in beta-blocked patients undergoing coronary revascularization was not associated with a change in serum K^+, in spite of a 50% increase in norepinephrine.[6] In contrast, when we compared potassium levels measured on the preoperative day with immediate pre-induction levels, there was a significant decrease in K^+ (fig. 1). Errors in potassium values due to sampling and laboratory technique as a cause for this finding were eliminated.[7] Inter-

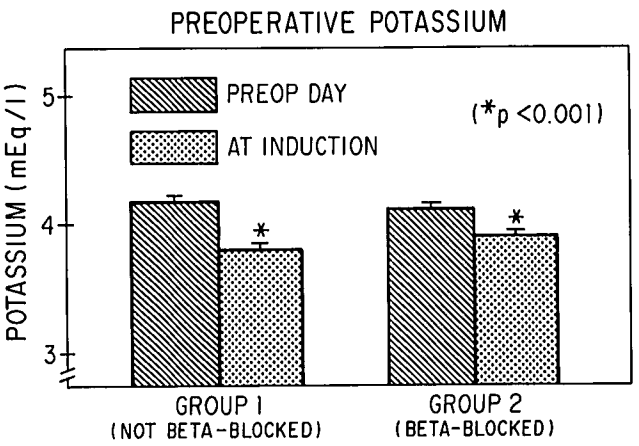

FIG. 1. Preoperative and preinduction serum potassium in cardiac surgical patients. Mean ± SEM; paired *t* tests, $P < 0.001$ for preoperative day *versus* at induction. Group 1 = patients not receiving beta-blocker therapy, n = 29; Group 2 = patients receiving propranolol, n = 33.

group analysis indicated that this change was less in beta-blocked patients, but this was not statistically significant. Epinephrine, a beta-2 agonist, is known to be elevated during the preoperative period.[8] Therefore, we postulate that the adrenergic modulation of potassium regulation is determined by both the degree and duration of sympathoadrenal hyperactivity.

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Abolishing Pain on Injection of Etomidate

To the Editor:—We have been impressed with the rapid awakening and clear postoperative sensorium in adult outpatients undergoing anesthetic induction with etomidate. Pain on injection in these alert, unpremedicated patients can be a problem. This incidence is reported to be as high as 50–60%.[1,2]

We have found a simple technique which has so far reduced the incidence of pain on injection to zero. Just prior to induction, 25–100 mg of lidocaine is given through an injection port attached directly to the intravenous catheter. As soon as the injection is made, the intravenous drip is turned off for 30 s. Etomidate is then injected.

Using this technique, we have had no pain on injection through either spontaneous complaint or direct questioning in 30 consecutive patients.

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Trunk Skin Temperature After Sympathetic Nerve Block—Is the Heat Really On?

To the Editor:—In a recent study, Chamberlain *et al.*[1] concluded, based on measurements of trunk skin temperature by infrared thermography, that sympathetic block can extend up to ten segments above the sensory block with spinal anesthesia. A surprising finding, indeed.

The authors can be congratulated for their provoking and stimulating paper, because it reminds us that we are blind when it comes to evaluation of the extent of sympathetic block with spinal or epidural anesthesia. Our assumption, based upon similar size of fibers carrying sympathetic and thermoreceptor traffic, that loss of temperature discrimination has the same level as loss of sympathetic outflow, may not be valid. Thus, the anesthesia community would receive with enthusiasm any monitor which reliably detects the level of sympathetic block, especially in the unconscious patient. Also, the