Anesthesiology
64:408–409, 1986

Chronic Hypokalemia and Intraoperative Dysrhythmias

To the Editor:—The article by Vitez *et al.*[1] and the accompanying editorial on hypokalemia and intraoperative arrhythmias are thoughtful additions to the available literature on this subject. They may, however, give a false sense of security about the significance of hypokalemia by addressing the issue from only one perspective. In a group of 62 patients with a mean serum potassium of 3.1 mEq/l, the incidence of intraoperative dysrhythmias was apparently no different than in a group of normokalemic controls. An equally important question, I believe, is: How much does hypokalemia contribute to the incidence of malignant ventricular arrhythmias or a deleterious outcome from anesthesia and surgery?

We are fortunate in anesthesia in having few fatal outcomes resulting from our interventions. Keats estimates the number of unexpected fatal outcomes resulting from anesthetic exposure to be about 1:3,000.[2] A recent article on the incidence of cardiac arrest related to anesthesia by Keenan and Boyan in JAMA documents 1.7 arrests per 10,000 anesthetics.[3] Goldman *et al.*, in computing their multifactorial index of cardiac risk, detected only 12 instances of postoperative ventricular tachycardia in 1,000 patients (not all of whom died).[4] Their patients were at higher risk than in the Keenan article because only patients over the age of 40 were included. If we accept the 12/1,001 figure as the incidence of life-threatening arrhythmias during anesthesia (a generous estimate based on available information), a very large sample size would be necessary to attribute excess risk to hypokalemia or any other preoperative predictor of mortality: If hypokalemia were responsible for a 50% increase in mortality from malignant arrhythmias, and such arrhythmias occurred 12 times in every 1,000 anesthetics administered, it would be necessary to study 8,600 patients to show a difference between the hypokalemic and normokalemic populations (two-tailed test, alpha = 0.05, beta = 0.10).[5] In their article, Vitez *et al.*[1] studied only 150 patients and had no malignant arrhythmias in either group. It would be impossible, therefore, based on this study, to say that hypokalemia does not contribute significantly to mortality from arrhythmias.

The assumption that a difference in the frequency of intraoperative premature ventricular contractions (PVCs) and atrial premature contractions (APCs) corresponds to potential mortality from a malignant arrhythmia is a shaky one. Many studies draw an association with death from ventricular arrhythmias and premonitory ventricular ectopy in special high-risk groups (*e.g.*, following a myocardial infarction or in patients with ischemic heart disease). Even in such situations, however, it is not uncommon for ventricular fibrillation to occur without prior evidence of ventricular irritability. If hypokalemia alone or in conjunction with other factors such as hypocarbia or cathechol release leads to ventricular fibrillation without associated early PVCs, then equating the absence of a higher rate of PVCs in the hypokalemic group with the absence of increased risk is unwarranted. In addition, Vitez *et al.* only studied ten patients with a history of cardiac disease and hypokalemia for an increase in dysrhythmias. In patients who sustain a perioperative myocardial infarction, there is considerable evidence that hypokalemia might contribute to an early demise.[6] For those patients undergoing surgery who have serious cardiac disease, therefore, the risk of hypokalemia remains undefined.

Vitez *et al.* have provided important information in confirming the infrequency of serious arrhythmias even in a population of patients with hypokalemia. By their own estimation, the maximum probability of a hypokalemic patient population similar to theirs experiencing a fatal arrhythmia was 5%. For the practitioner making a judgement about whether or not to cancel surgery in a hypokalemic patient, therefore, the issue seems to be one of perspective: Since fatal arrhythmias associated with anesthesia and surgery seem to be very infrequent, the excess mortality associated with hypokalemia must be very small (*e.g.*, if mortality increases 50% with hypokalemia—from 12/1000 to 18/1000—the excess risk would only be 6/1000). For the individual patient, however, a 50% increase in the risk of dying or an absolute increase in risk of 0.6%—especially for an elective procedure—might be unacceptable, as might the 5% outside chance of experiencing a serious arrhythmia while hypokalemic mentioned in the article.

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(Accepted for publication October 10, 1985.)

Anesthesiology
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In Reply:—Thanks for giving us an opportunity to respond to Dr. Glaser's letter. We believe some history about our article will put things in perspective. Four years ago, our group tried to reach a consensus about how to handle a recurrent clinical problem: "What should we do about the relatively well, hypokalemic patient scheduled for surgery?" The existing dogma required that surgery be postponed until some arbitrary potassium level was attained by intravenous or oral replacement. We searched for data to support this approach. We found no studies investigating the sequelae of anesthetizing hypokalemic humans. So, we performed a little clinical investigation. Our results did not seem to support the dogma. On the contrary, we found stable intraoperative rhythms in hypokalemic patients, and uncovered four cardiac arrests (three fatal) associated with attempts to replete potassium. Although we recognized the limitations to our study, we thought our results might stimulate other investigators to help delineate when hypokalemia becomes a risk.

We do not think we have proven anything; we have raised reasonable doubts about the validity of current practices surrounding hypokalemia. Dr. Glaser and others may speculate, calculate, and postulate. However, none of the referenced reports are relevant to the common situation that we addressed: The relatively well, hypokalemic patient scheduled for a routine surgical procedure. Our investigation remains as the only study about anesthetizing hypokalemic humans.

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(Accepted for publication October 10, 1985.)

Anesthesiology
64:409–410, 1986

Intravenous Nitroglycerin Dosage to Prevent Intraoperative Myocardial Ischemia during Noncardiac Surgery

To the Editor:—We were most surprised by the erroneous interpretation made by Thomson *et al.*[1] of our studies on the efficacy of intravenous nitroglycerin (iv NTG) in the prevention of intraoperative myocardial ischemia during noncardiac surgery. In our first study, published at first in abstract[2] and then in an original article,[3] the final dose of iv NTG administered during the surgical procedure was determined according to the modifications of both arterial pressure and heart rate. The infusion was started at the dose of 0.25 $\mu g \cdot kg^{-1} \cdot min^{-1}$ and increased at a rate of 0.25 $\mu g \cdot kg^{-1} \cdot min^{-1}$ every 5 min up to 1 $\mu g \cdot kg^{-1} \cdot min^{-1}$ unless systolic blood pressure decreased more than 25 mmHg. The final dose of NTG administered in the 15 patients suffering from disabling angina included in this study was $0.91 \pm 18 \mu g \cdot kg^{-1} \cdot min^{-1}$ ($x \pm SD$). Therefore, in this study, we did not use NTG at the dose of 0.5 $\mu g \cdot kg^{-1} \cdot min^{-1}$ as implied by Thomson *et al.*[1] Only three out of the 15 patients experienced intraoperative myocardial ischemia.

Since prophylactic iv NTG infusion administered at the dose of 0.5 $\mu g \cdot kg^{-1} \cdot min^{-1}$ has the apparent advantage of necessitating lesser fluid infusion than at the dose of 1 $\mu g \cdot kg^{-1} \cdot min^{-1}$, it seemed it would be interesting to determine the efficacy of these two doses in preventing myocardial ischemia. In a second study,[4] we employed a randomized protocol to compare the incidence of intraoperative ischemic episodes in patients with angina pectoris undergoing noncardiac surgery with prophylactic iv NTG infusion at the dose of 0.5 or 1 $\mu g \cdot kg^{-1} \cdot min^{-1}$. Prophylactic iv NTG was effective in preventing myocardial ischemia only when administered at the dose of 1 $\mu g \cdot kg^{-1} \cdot min^{-1}$. These findings are in agreement with both those of Thomson *et al.*,[5] who demonstrated a high incidence of myocardial ischemia with the dose of 0.5 $\mu g \cdot kg^{-1} \cdot min^{-1}$ and with those of our previous study[2,3] in which a mean dose of 0.91 $\mu g \cdot kg^{-1} \cdot min^{-1}$ was used.

In reply to Thomson's remark concerning absorption of NTG on plastics, we would like to point out that since 1978, we know that there are significant losses of NTG