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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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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 abstract² 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 $0.25 \mu\text{g} \cdot \text{kg}^{-1} \cdot \text{min}^{-1}$ and increased at a rate of $0.25 \mu\text{g} \cdot \text{kg}^{-1} \cdot \text{min}^{-1}$ every 5 min up to $1 \mu\text{g} \cdot \text{kg}^{-1} \cdot \text{min}^{-1}$ 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 $0.91 \pm 18 \mu\text{g} \cdot \text{kg}^{-1} \cdot \text{min}^{-1}$ ($x \pm \text{SD}$). Therefore, in this study, we did not use NTG at the dose of $0.5 \mu\text{g} \cdot \text{kg}^{-1} \cdot \text{min}^{-1}$ 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 $0.5 \mu\text{g} \cdot \text{kg}^{-1} \cdot \text{min}^{-1}$ has the apparent advantage of necessitating lesser fluid infusion than at the dose of $1 \mu\text{g} \cdot \text{kg}^{-1} \cdot \text{min}^{-1}$, 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 $1 \mu\text{g} \cdot \text{kg}^{-1} \cdot \text{min}^{-1}$. Prophylactic iv NTG was effective in preventing myocardial ischemia only when administered at the dose of $1 \mu\text{g} \cdot \text{kg}^{-1} \cdot \text{min}^{-1}$. 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 $0.5 \mu\text{g} \cdot \text{kg}^{-1} \cdot \text{min}^{-1}$ and with those of our previous study^{2,3} in which a mean dose of $0.91 \mu\text{g} \cdot \text{kg}^{-1} \cdot \text{min}^{-1}$ 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