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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 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 ± 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

when the drug is run through polyvinylchloride (PVC) infusion sets[6] and that we used non-PVC-type, polyethylene–polypropylene administration sets.

The results of our two investigations should be appreciated with respect to the age of the patients studied, the severity of their coronary artery disease, their incidence of previous myocardial infarction, and to the presence of a peripheral vascular disease. Most of the patients studied were unfit for coronary arteriography or for cardiac surgery because of their age or their peripheral vascular disease. In such patients, left ventricular function is often depressed,[7] which probably permits the full exertion of the beneficial hemodynamic and antianginal effects of iv NTG.[6]

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A Potential Hazard: Interchanging Fentanyl and Sufentanil

To the Editor:—A recent probable drug administration error prompts us to share details of the episode and express a note of concern.

During a lengthy upper-extremity operation performed under axillary block, a Physical Status I, 38-yr-old man received 5 ml of fentanyl over 2.5 h initially for sedation and then, subsequently, for tourniquet pain. A second 5-ml ampul of fentanyl was ordered and 1 ml administered. Approximately 2–3 min later, the patient was noted to be apneic, cyanotic, unresponsive, and demonstrating tonic–clonic movement of the unanesthetized arm. Following an uncomplicated resuscitation, the patient breathed with a large tidal volume and a spontaneous respiratory rate of 4 breaths/min for some time thereafter. Naloxone was not given. Initially, the cause for this event was not known, but a recheck of the operating room narcotic log revealed a one-ampul surplus of fentanyl and a one-ampul shortage of sufentanil. Unfortunately, because it had been recently emptied, the glass disposal container could not provide a missing ampul of either possible drug. Consequently, we classified this as a probable, rather than certain, error.

Although the ultimate responsibility for the incident described above rests with the individual who administered the drug, we believe that the manufacturer should seriously reconsider the advisability of marketing a narcotic at a concentration such that 1 ml administered by the only route suggested constitutes *the* dose, rather than an increment. Yes, numerous other drugs (*e.g.*, epinephrine, phenylephrine) are available in similarly concentrated forms, and we have seen all of these drugs involved in errors of administration, occasionally with significant morbidity. Anesthetized patients do not need an additional source for such error, convenience to the practitioner notwithstanding.

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In Reply:—Janssen Pharmaceutica recognizes the concern that Ward and Sanford have expressed. We have been in the process, over the past several months, of acquiring specially designed equipment and obtaining the necessary Food and Drug Administration (FDA) approval to market our intravenous anesthesia products in paper-