starting nitroglycerin infusion prevents an unwanted decrease in blood pressure.

YORAM SHAPIRA, M.D.
Instructor of Anesthesia

MORRIS GERTEL, M.D., F.R.C.P.(C)
Senior Anesthetist

Department of Anesthesia
Hadassah University Hospital
Ein Karem, Jerusalem 91120,
Israel

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In reply:—We thank Drs. Shapira and Gertel for their letter and welcome the opportunity to discuss the recent publication of Coriat *et al.*[1] We need no introduction to the work of Coriat *et al.*, as our recent work[2] was, in part, inspired by a previous communication from that group that was published first in abstract[3] and later as an original article.[4] The original work of Coriat *et al.*[4] demonstrated that a 0.5 $\mu g \cdot kg^{-1} \cdot min^{-1}$ nitroglycerin (NTG) infusion was superior to no infusion for the prevention of ischemic ST-segment changes in patients with angina undergoing noncardiac surgery, under "balanced anesthesia" with thiopental, succinylcholine, fentanyl, and pancuronium (incidence of ischemia 3/15 *vs.* 15/16, $P < 0.001$). We were unable to duplicate this result in a double-blind randomized comparison of NTG 0.5 $\mu g \cdot kg^{-1} \cdot min^{-1}$ *versus* placebo in patients undergoing fentanyl–pancuronium–oxygen anesthesia for coronary artery surgery (incidence of ischemia 5/9 *vs.* 5/11, P = ns). The recent paper by Coriat *et al.* demonstrates that a 1.0 $\mu g \cdot kg^{-1} \cdot min^{-1}$ NTG infusion was superior to a 0.5 $\mu g \cdot kg^{-1} \cdot min^{-1}$ NTG infusion under circumstances similar to their first study. The incidence of ischemia in the group receiving 1.0 $\mu g \cdot kg^{-1} \cdot min^{-1}$ NTG was 4/23 *versus* 14/22 in the group receiving the lower dose, $P < 0.001$. Thus, Coriat *et al.* attribute the failure of NTG infusion to prevent ischemia in our patients to the use of an inadequate dose of NTG. Only at the 1.0 $\mu g \cdot kg^{-1} \cdot min^{-1}$ dose, they claim, can iv NTG be expected to prevent ischemia. Initially, this appears to resolve the issue. However, Coriat *et al.* do not explain the contradictory results of their own two trials. The 0.5 $\mu g \cdot kg^{-1} \cdot min^{-1}$ NTG infusion used in their first experiment[4] appears to have been significantly more effective than the same dose utilized under similar circumstances in their second trial[1] (incidence of ischemia 3/15 *vs.* 14/22, $P < 0.01$, chi-square) and equal in efficiency to the 1.0 $\mu g \cdot kg^{-1} \cdot min^{-1}$ NTG infusion used in the second trial (3/15 *vs.* 4/23, P = ns). We feel that these two contradictory results from the same investigators possibly may be explained by the influence of investigator bias in nonblind trials. As the investigators were aware of which treatment group patients belonged to, they may unconsciously have managed the patients differently intraoperatively, especially with respect to the rapidity and extent to which supplemental fentanyl was administered. Unfortunately, the authors did not even report the total fentanyl dose employed in their studies.[1,4]

A second issue arises with respect to the actual NTG dose administered. We utilized an infusion system proven *in vitro* not to adsorb NTG. Thus, there is no doubt as to the actual NTG dose administered to our patients. Coriat *et al.* did not describe the NTG infusion system used in their studies. This probably implies that standard polyvinylchloride (PVC) infusion sets were used. Such sets are known to adsorb significant quantities of NTG, resulting in the administration of reduced and unpredictable doses of NTG. If PVC infusion sets were used by Coriat *et al.*, then the dose of nitroglycerin employed in their studies is indeterminate, and comparison with our studies is precluded.

At any rate, because of obvious differences in population, surgery, and anesthetic technique, it is probably unwise to compare our results with those of Coriat *et al.* under any circumstances. Thus, the inevitable questions that arise can only be addressed by further trials. Unfortunately, the high incidence of ischemia associated with the anesthetic techniques used in all these studies means that they cannot ethically be repeated. Thus, the debate will likely continue unresolved.

I. R. THOMSON, M.D.
W. A. C. MUTCH, M.D.
J. D. CULLIGAN, M.D.
Department of Anesthesia
St. Boniface General Hospital
409 Tache Avenue
Winnipeg, Hanitoba
Canada R3H 2A6

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Early Detection of Malignant Hyperthermia

To the Editor:—It has been suggested that end-tidal CO_2 should be useful in the early detection of malignant hyperthermia (MH) and should be followed to gauge the effectiveness of therapy.[1] Although capnographs and mass spectrometers are proliferating in United States operating rooms, most anesthesiologists still do not have the ability to routinely monitor expired CO_2. Gronert and Theye[2] found parallel increases in CO_2 production and O_2 consumption. By closing the circuit, the anesthesia machine becomes a "metabolic laboratory" and the measurement of whole-body O_2 consumption becomes straightforward.[3] Moderate leaks in the system are easily measured and have little effect on the accuracy of the O_2 consumption measurement. Therefore, all operating rooms have the capability to detect the early metabolic changes of MH.

On the basis of animal models, the O_2 consumption would be expected to increase by at least 30–50% if the patient has MH develop. Effective therapy with dantrolene will also be demonstrated by a decrease in O_2 consumption.[4] During treatment of MH, cooling is desirable so the circuit should be opened with high flows of oxygen and intermittently closed for 3–5-min periods to follow O_2 consumption. Expiratory CO_2 monitoring would be a valuable complement to measurements of O_2 consumption. Whenever MH is suspected, the anesthesiologist should measure O_2 consumption.

R. K. BAUMGARTEN, M.D.
WILLIAM J. REYNOLDS, M.D., PHD.
630 Alamo Heights Boulevard
San Antonio, Texas

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Respiratory Monitoring for Children Undergoing Radiation Therapy

To the Editor:—Pediatric patients often require sedation or general anesthesia to prevent movement during high-voltage radiation. Because personnel cannot be present during this 2- to 3-min period of radiation, the anesthesiologist usually monitors such patients using closed-circuit television. However, resolution of the television monitor is poor, and movements of the chest or anesthesia bag are often impossible to see. Therefore,