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CORRESPONDENCE

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In Reply:—Both Waltuck and Pas speculate that the administration of epinephrine in the epidural local anesthetic solution was a likely contributing factor to the neurologic deficit observed in our case. We contend that there is no scientific evidence to support this assumption. We are unaware of any research data in humans examining the relation of epinephrine administered by either the subarachnoid or epidural route to spinal cord blood flow. On the contrary, in an animal model, Kozody et al.* reported that lumbar subarachnoid injection of 200 µg epinephrine had no effect on spinal cord blood flow in the cervical, thoracic, and lumbosacral regions. It should be noted that 200 µg epinephrine frequently is administered with local anesthetics in the subarachnoid area to augment surgical anesthesia in a wide variety of patients (including patients with severe peripheral vascular disease) without apparent detrimental neurologic effects. Given that the total dose of epinephrine used in our case was 40 µg and given the lack of support from either clinical observation or animal studies of adverse outcome with neuraxial (epidural or subarachnoid) administration of epinephrine on spinal cord blood flow. we suggest that the contribution of epinephrine in this case remains speculative and highly unlikely

We do not regard the selection of a thoracic epidural for postoperative analgesia in this case as either "overzealous" or "aggressive."

* Kozody R, Palahniuk RJ, Wade JG, Cumming MO: The effect of subarachnoid epinephrine and phenylephrine on spinal cord blood

Our patient had inadequate analgesia, with evidence of deteriorating pulmonary status (inability to cough and poor oxygen saturations) despite patient-controlled analgesia morphine. He was facing the possibility of reintubation and ventilation when the surgical service asked us to consider epidural catheter placement. The benefits of thoracic epidural analgesia are well recognized in this setting. The incidence of adverse neurologic events from thoracic epidurals is unknown and probably exceedingly rare. As such, decisions regarding postoperative analgesia should be made on an individual basis and with the clinical situation in mind.

Dermot R. Fitzgibbon, M.B., B.Ch., F.F.A.R.C.S.I. Acting Assistant Professor
L. Brian Ready, M.D., F.R.C.P.(C.)
Professor and Director of Pain Service
Department of Anesthesiology
University of Washington Medical Center
Seattle, Washington 98105

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Hotline Fluid Warming Fails to Maintain Normothermia

To the Editor:—In recent months, Anesthesiology and other journals have carried advertisements from Level 1 Technologies, Inc. guaranteeing that 90% of patients in whom Hotline fluid warming is used will arrive in the recovery room with "final temperature \geq 36°C or net temperature decrease \leq 0.2°C." The claim that fluid warming alone will maintain normothermia does not agree with much of what is known about perioperative heat balance and seems inconsistent with the laws of thermodynamics.

Intraoperative hypothermia results from a combination of anesthetic-induced inhibition of thermoregulatory control and exposure to a cold environment. Hypothermia during the first hour of surgery results largely from an internal core-to-peripheral redistribution of body heat. Redistribution typically reduces core temperature 0.5–1.5°C^{2,3} and is difficult to treat even with active cutaneous warming. Subsequently, mean body temperature decreases when heat loss (in-

cluding that required to warm administered fluids) exceeds metabolic heat production. During this phase, core temperature is most influenced by cutaneous insulation/heating, fluid warming, and other manipulations altering heat balance. Because radiative and evaporative losses associated with surgery can be enormous, active warming usually is required to prevent hypothermia.

One liter of crystalloid at ambient temperature or a unit of refrigerated blood reduces mean body temperature $\approx 0.25\,^{\circ}\text{C}$ in adults. It is therefore easy to cool patients by administering unwarmed intravenous fluids. Conversely, it is not possible to significantly warm patients by administering heated intravenous fluids. The reason is that intravenous fluid temperature cannot much exceed $40\,^{\circ}\text{C}$ without harming blood cells.

Heat transferred by fluid warming therefore can be calculated as the difference between fluid and mean body temperature multiplied by the volume of fluid. For example, administration of 21 of fluid at 40°C to a patient with a mean body temperature of 36°C increases body heat content 8 kcal. Assuming a patient weight of 70 kg and a tissue specific heat of 0.86 cal·°C⁻¹·g⁻¹,⁸ 8 kcal will increase mean body temperature only 0.1°C. This trivial amount of warming will rarely compensate for ongoing cutaneous and surgical losses.^{4,5} Heat delivered by warmed fluids has the advantage of being directly inserted into the core thermal compartment; it is, of course, rapidly dissipated to peripheral tissues.

The Hotline warmer improves conventional designs by preventing fluid cooling within intravenous tubing. However, the amount of cooling at typical flow rates is of no consequence in adults. In addition, this design compromises efficacy, and the device warms fluids only to 38°C at a flow rate of 1 l/h. The above calculation thus overestimates the benefits of Hotline warming.

To evaluate the Level 1 Technologies, Inc. claim, I conducted the following study. With Institutional Review Board approval, distal esophageal temperature (probes: RSP, Inc., Irvine, CA; monitor: SpaceLabs, Redmond, WA) was measured in all patients aged 18 yr or older to whom I administered general anesthesia during a 1-month period (excluding those undergoing cardiopulmonary bypass). Anesthesia was induced with fentanyl, methohexital, and rocuronium and maintained with desflurane in oxygen. Total fresh gas flow was 900 ml/min; ventilation was controlled to an end-tidal PCO2 near 35 mmHg in others. Ambient temperature was $19.3 \pm 1.3\,^{\circ}\text{C}$ (normal at NT Enloe Hospital), and the patients were covered with a single layer of surgical drape. Fluids were administered at a rate of 10 ml · kg $^{-1}$ · h $^{-1}$, always using a Hotline warmer.

Twenty-two patients had surgery lasting at least 1 h $(1.5\pm0.7\ h$ (mean \pm SD); among these, only seven underwent open abdominal procedures. Final core temperatures averaged $35.7\pm0.4^{\circ}C$ and equaled or exceeded $36^{\circ}C$ in only six. In addition, the decrease in core temperature exceeded $0.2^{\circ}C$ in all but two cases. Consequently, 68% of the cases failed to meet the criteria "absolutely guaranteed" by Level 1 Technologies, Inc. To their credit, and as offered in the advertisement, a representative of Level 1 Technologies, Inc. immediately offered to refund the purchase price of the Hotline unit and replace the disposable materials. Nonetheless, my results suggest that the Level 1 Technologies, Inc. advertisement is based more on wishful thinking than usual clinical experience.

It would be relatively easy to design a study in which use of the Hotline would maintain temperature of patients ≥36°C. Level 1 Technologies, Inc. may be able to cite such a study, although I am not aware that one has been published. Final core temperatures often will exceed 36°C if the study population is restricted to well covered patients 12 undergoing relatively noninvasive procedures 13.14 in a warm environment. 15 Numerous studies demonstrate that fluid warming alone will not maintain normothermia in patients undergoing typical procedures in a typical (≈19–21°C) environment. 4.6.7 My data are consistent with these peer-reviewed publications.

In summary, only $\approx 32\%$ of patients given fluids warmed with a Hotline were normothermic at the end of surgery. That fluid warming

alone would fail to maintain normothermia is expected from previous studies of intraoperative heat balance and the laws of thermodynamics.

Victor Werlhof, M.D. Attending Anesthesiologist NT Enloe Hospital Chico, California 95926

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