HIGHLIGHTS

Prevention of Intraoperative Hypothermia by Preoperative Skin-Surface Warming

Bernard Just, M.D., Véronique Trévien, M.D., Eric Delva, M.D., André Lienhart, M.D.

The Effects of Preinduction Warming on Temperature and Blood Pressure during Propofol/Nitrous Oxide Anesthesia

James M. Hynson, M.D., Daniel I. Sessler, M.D., Azita Moayeri, B.A., Joseph McGuire, B.S., Mark Schroeder, B.S.

HYPOTHERMIA during anesthesia has been described and documented for many years; many techniques have been advocated to prevent or treat such hypothermia. Recently, a better understanding of the causes, characteristic pattern, and consequences of such hypothermia has been obtained thanks to the work of several investigative groups, most prominently that headed by Dr. Daniel Sessler. Sessler and his colleagues have postulated that the initial sharp (1–2° C) decrease in core temperature after induction of general anesthesia is due to admixture of blood from the warmer core with that of blood from the cooler periphery due to anesthesia-induced vasodilation. Subsequently, body temperature is a function of metabolic heat production and heat loss to the environment. It would seem logical that a period of peripheral and core warming before induction of anesthesia might reduce the temperature gradient between the two compartments and therefore prevent intraoperative hypothermia. Questions as to the duration and extent of preoperative warming, the duration of the effect of such warming, and consequences of preoperative warming need answers.

Two articles in this issue of ANESTHESIOLOGY address several of these questions. In the article by Just *et al.* (page 214), prewarming was done in patients undergoing total hip arthroplasty. In the article by Hynson *et al.* (page 219), it was done in volunteers. Just *et al.* randomly divided patients into two groups: one group of eight patients were warmed with an electric warming blanket set at 42–43° C for at least 90 min before induction of anesthesia. The other group was not warmed preoperatively. During the anesthetic consisting of fentanyl, enflurane, and nitrous oxide, heat and moisture exchangers were not used and the ambient room temperature was maintained at 21–22° C. Following preoperative skin surface warming, mean skin and core temperature increased 4.2 ± 0.1° C and 0.5 ± 0.1° C, respectively. In the first hour after induction of anesthesia, core temperature decreased in both groups, but the decline was more rapid in the unwarmed than in the prewarmed patients. At the end of 3 h of surgery, core temperature was similar to basal values in the prewarmed patients, but unwarmed patients were hypothermic. Seven unwarmed patients shivered; no prewarmed patients shivered. In effect, prewarming created a reservoir of stored heat available to the patient during the procedure. It was believed that it was necessary to prewarm the patient for more than an hour because of the time necessary to stabilize skin temperature and to ensure transfer of heat to the core.

Hynson *et al.* studied the effects of preinduction warming in subjects anesthetized with propofol/nitrous oxide. Each volunteer served as his/her own control undergoing a period of either prewarming using a forced-air warming device for 2 h or no prewarming followed by a 1-h anesthetic. In addition to core, skin, and mean body temperature measurements, the investigators examined the effect of warming and induction of anesthesia on the configuration at the radial artery pulse trace as well as the difference between blood pressure measured oscillometrically at the brachial artery and by radial artery catheter.

Blood propofol levels were measured to ensure comparability of depth of anesthesia; nitrous oxide was administered in all cases at a concentration of 70%. A long period of gentle warming was carried out to gradually increase tissue heat content and avoid the possibility of sweating and evaporative heat loss before induction of anesthesia and exposure to the cooler environment of the operating room. During the warming period, tympanic membrane temperatures increased slightly from 36.8 ± 0.2° C to 37.1 ± 0.4° C. However, at the end of the warming period, the heat content of those who were prewarmed was 573 ± 196 kJ (corresponding to be about 2.3° C increase in mean body temperature).

After the 1-h anesthetic, the nonwarmed group experienced an approximately 2° C decrease in core temperature whereas those who were prewarmed experienced a 1° C decrease in core temperature. Of course, heat loss to the environment was greater after prewarming as opposed to nonprewarming, but this was not significant enough to lead to core hypothermia, again because the body had stored a significant amount of heat.

Thus, both articles suggest that a period of 1–2 h of gentle prewarming may serve to prevent significant hypothermia after induction of general anesthesia in procedures lasting for 1–3 h in which there is not extensive blood loss or exposure of body cavities to low ambient temperature. Because hypothermia is associated with its own set of side effects/complications, ultimately the effects of prewarming and subsequent maintenance of core temperature on several measures of outcome should be tested.

Henry Rosenberg, M.D.

Clonidine Pretreatment Reduces the Systemic Toxicity of Intravenous Bupivacaine in Rats

M. De Kock, M.D., B. Le Polain, M.D., D. Henin, M.D., F. Vandewalle, M.D., J. L. Scholtes, M.D.

CARDIOTOXICITY remains the most feared complication from the use of bupivacaine in regional anesthesia. In this report by DeKock *et al.* (page 282), the α_2-adrenergic agonist clonidine reduced bupivacaine-induced cardiotoxicity in rats. A much larger dose of intravenous bupivacaine was required to produce ventricular dysrhythmias with clonidine than without. Though both drugs can produce cardiovascular depression, addition of clonidine did not worsen bupivacaine-induced hypotension or bradycardia.

This animal study has two important implications. First, it addresses an important safety concern in the use of clonidine–bupivacaine combinations for epidural anesthesia, as commonly employed in countries outside the United States. Clonidine has been demonstrated to enhance epidural bupivacaine anesthesia, and this study suggests it would not be expected to enhance and might lessen cardiotoxicity for accidental intravenous bupivacaine injection. Second, this study confirms the role of vagal tone in cardiac repolarization and generation of dysrhythmias. Bupivacaine can produce dysrhythmias in part by actions in the brainstem to decrease vagal tone. α_2-Adrenergic drugs, such as clonidine, protect against cardiac dysrhythmias produced by halothane and epinephrine by a central action, primarily to increase vagal tone. This study in rats extends these observations to the clinically relevant cardiac dysrhythmic effects of bupivacaine, which are reduced by clonidine.

James C. Eisenach, M.D.