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

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Background: Core temperature decreases rapidly after induction of anesthesia, largely because heat is redistributed to peripheral tissues. The hypothesis that warming peripheral tissues before induction of general anesthesia (prewarming) minimizes hypothermia was tested. Because circulating blood volume may be greater during exposure to heat compared to cold, the hypothesis that prewarming decreases the amount of hypotension associated with induction of anesthesia was tested also. Finally, the hypothesis that the difference between direct radial arterial blood pressure and blood pressure measured osccillometrically at the brachial artery depends on thermoregulatory and anesthetic conditions was tested.

Methods: Six volunteers underwent general anesthesia (propofol and nitrous oxide) twice on the same day. Each anesthetic lasted 1 h and was preceded by 2 h of active warming with forced air or 2 h of passive cooling by exposure to a typical operating room environment. After induction of each anesthetic, volunteers were fully exposed to the ambient environment. Volunteers recovered for 2 h before starting the second preinduction treatment.

Results: Initial tympanic membrane temperatures were similar before each preinduction treatment: 36.7 ± 0.4°C when volunteers were not warmed and 36.7 ± 0.6°C when volunteers were warmed. Tympanic membrane temperature did not change during the preinduction period without warming but increased slightly (ΔT = 0.4 ± 0.2°C) during warming. After induction of anesthesia, core temperatures decreased to 36.1 ± 0.4°C over 1 h when volunteers were prewarmed but decreased to 34.9 ± 0.4°C when they were not. Radial arterial systolic, diastolic, and mean blood pressures were lower before induction of anesthesia when volunteers were warmed compared to when no warming was given. Oscillometric diastolic and mean pressures also were lower during prewarming; however, oscillometric systolic pressure did not differ significantly. Prewarming did not result in less hypotension after induction. Without warming, the difference (radial arterial minus oscillometric) in systolic blood pressure measurements was ≈17 mmHg. Warming was associated with a reversal of the systolic pressure difference to ≈−6 mmHg. After induction of anesthesia, the differences in systolic and mean pressure measurements became more negative with respect to the preinduction values regardless of preinduction warming treatment.

Conclusions: These data confirm our hypothesis that redistribution hypothermia can be minimized by preinduction warming of peripheral tissues. Prewarming decreases blood pressure but does not prevent subsequent hypotension after induction. The difference between radial arterial blood pressure and oscillometric blood pressure depends on thermoregulatory vasomotor changes but also may be influenced by vasodilation associated with administration of propofol and nitrous oxide. (Key words: Anesthetics, gases: nitrous oxide. Anesthesics, intravenous: propofol, Blood pressure, measurement: direct arterial, noninvasive: oscillometric. Hypothermia. Measurement techniques, blood flow: skin-temperature gradients. Thermoregulation.)

INTERNAL redistribution of heat after induction of general anesthesia rapidly reduces core temperature 0.5−1.5°C. The quantity of heat redistributed, or transferred, from core to peripheral tissues largely depends on their relative temperatures and heat contents before induction. This core-to-peripheral temperature gradient is determined by the intensity of vasoconstriction, environmental temperature, and duration of exposure to that environment. Active warming may increase the temperature of peripheral tissue to nearly that of core tissue. Total body heat content can vary by ≈600 kJ in an average-sized adult human without a change in core temperature. Warming peripheral tissues before induction of anesthesia thus may decrease the potential heat sink presented by the peripheral tissue mass. Accordingly, we tested the hypothesis that redistribution hypothermia can be minimized by
warming peripheral tissues before induction of general anesthesia.

Thermoregulatory and cardiovascular homeostasis are closely related. Cutaneous blood flow is determined by the often competing influences of cardiovascular and thermoregulatory control. Heat stress is associated with increased cardiac output and redistribution of blood flow and volume to cutaneous vascular beds. Passive movement of blood out of splanchnic veins in response to decreased central venous pressure is thought to provide this translocated blood volume, though splanchnic vasoconstriction probably contributes. In contrast, hypothermia decreases circulating blood volume. We postulated that inducing and maintaining cutaneous vasodilation via skin-surface warming for 2 h would increase circulating blood volume sufficiently to blunt the hypotensive response to induction of anesthesia.

Thermoregulatory and anesthetic-induced changes in arteriovenous shunt tone and extremity blood flow might effect the relative accuracy of different blood pressure measurement techniques. Therefore, we tested the hypothesis that the difference, between noninvasive oscillometric (measured at the brachial artery) and direct radial arterial blood pressure measurements, varies with thermoregulatory and anesthetic state.

Methods

Following approval of the University of California, San Francisco Committee on Human Research, we studied six young, healthy volunteers. None was obese or taking medications. All abstained from food and liquids for 8 h before the study. During the study, volunteers wore only shorts or a two-piece bathing suit and reclined on a standard operating room table covered by a 5-cm-thick foam pad. Lactated Ringer’s solution (≈1 ml·kg⁻¹·h⁻¹) was infused continuously into a right antecubital vein of each volunteer.

Study Protocol

Ambient temperature was maintained near 21°C. To maintain a thermoneutral state, volunteers were covered with two cotton blankets for the ≈1 h required to attach monitoring equipment. Volunteers acted as their own controls by undergoing two separate general anesthetics on the same day. Each anesthetic lasted ≈1 h and was preceded in one case by 2 h of active cutaneous warming (prewarming) and in the other by 2 h of passive cooling produced by exposure to the ambient room environment (no warming). Treatment order was assigned randomly. The 2-h period was chosen to ensure adequate time for heat transfer and to reach near steady-state thermal conditions before induction of anesthesia.

During prewarming, volunteers were heated with forced air (Bair Hugger, Augustine, Eden Prairie, MN) with the blower set at medium (≈40°C). A full-length warming blanket (model 300, Augustine) covered the trunk and legs of each volunteer, but the arms remained exposed. Forced-air warming was discontinued at anesthetic induction and volunteers subsequently were fully exposed to the ambient environment. Thus, thermal management after induction of anesthesia always was the same regardless of the preinduction treatment.

After intravenous administration of 40 mg lidocaine to reduce pain during propofol injection, general anesthesia was induced by intravenous injection of 3 mg/kg propofol and inhalation of 70% N₂O/O₂. Anesthesia was maintained with an intravenous propofol infusion at 100 μg·kg⁻¹·min⁻¹ (Program 2 syringe pump, Becton Dickinson, Lincoln Park, NJ) and 70% N₂O delivered via face mask. Volunteers breathed spontaneously during anesthesia and were monitored by electrocardiography, pulse oximetry, and capnography. No external airway humidification was provided. Anesthesia was discontinued after 60 min, and volunteers recovered for ≈2 h before the start of the second preinduction period. During recovery, volunteers sat in a chair for at least 30 min. Tympanic membrane temperatures were verified to be within 0.5°C of each volunteer’s initial temperature, before starting the second preinduction treatment.

Measurements

Temperatures were monitored using Mon-a-Therm tympanic membrane and skin-surface thermocouple probes (Mallinckrodt, St. Louis, MO) connected to Mallinckrodt model 8700 two-channel electronic thermometers. Core temperature was measured with a thermocouple placed at the tympanic membrane. Peripheral thermoregulatory vasoconstriction was assessed by measuring the forearm-minus-fingertip, skin-surface temperature gradient on the right arm, as previously described. Mean skin-surface temperature was computed from measurements at ten sites using the following regional percentages: head 6%, upper arms 9%, forearms 6%, hands 4.5%, back 19%, chest 9.5%, abdomen 9.5%, thigh 19%, calves 11.5%, and feet 6%.
Total cutaneous heat loss was determined by measuring heat flux at ten skin-surface sites (W/m²) using thermal flux transducers (Concept Engineering, Old Saybrook, CT). These values were converted into W/site by multiplying by the calculated body surface area [area (m²) = weight⁰.⁶⁴²⁵ (kg) · height⁰.⁹²⁵ (cm) · 0.007184] of each volunteer and assigning the same regional percentages as used for calculating mean skin temperature. We defined flux as positive when heat traversed skin to the environment. We previously described the details of thermal flux measurements.

Right radial arterial blood pressure was measured using a 5.1-cm-long, 20-G catheter (Angiocath, Deseret, Sandy, UT) connected to 213 cm of arterial pressure extension tubing (Abbott, North Chicago, IL). The liquid-filled component of the monitoring system was attached to a Transpac disposable transducer (Sorensen, Abbott). Visible bubbles were purged from the tubing and transducer housing. A Tektronix 414 pressure monitor (SpaceLabs, Redmond, WA; band width 0 to 20 Hz) was used to condition and display the transduced pressure signal. The transducer was zeroed at the level of the right atrium and volunteers remained in the same horizontal position throughout the study. Left upper arm blood pressure was measured oscillometrically using an Ohmeda 2140 monitor (Madison, WI). Systolic, diastolic, and mean blood pressures obtained by each method were recorded at 5-min intervals except during the first 15 min after the start of induction, when data were recorded every minute. The difference between measurement techniques, for each blood pressure parameter, was defined as radial arterial minus oscillometric pressure.

The dynamic characteristics of the catheter-transducer system were determined by measuring the damping coefficient and undamped natural frequency in situ every 30 min using the fast-flush technique. Recordings of the pressure waveforms and fast-flush transients were made on a physiologic chart recorder (model 220, Gould, Valley View, OH; band width 0 to 100 Hz). Damping coefficients ranged from 0.16 to 0.31, and the undamped natural frequency from 13 to 20 Hz.

Arterial blood samples for propofol levels were obtained preinduction and at 3, 5, 10, 15, 30, and 60 min after induction of anesthesia. Whole blood was stored at 4°C and subsequently analyzed for propofol concentration using high-pressure liquid chromatography. This assay has an accuracy exceeding 0.2 µg/ml for concentrations ranging from 0.1 to 5.0 µg/ml and an accuracy exceeding 0.2 µg/ml for concentrations ranging from 5.0 to 10.0 µg/ml.

Data Analysis

Temperature and heat flux data recorded from each volunteer at 2-5-min intervals were averaged into 10-min epochs using a database program; these individual averages were used to calculate means for the entire group. As in previous studies, we prospectively defined significant peripheral vasoconstriction as a forearm-minus-fingertip, skin-temperature gradient exceeding 4°C. Additionally, we defined significant vasodilation as a gradient <0°C (fingertip warmer than forearm).

An estimate of the cumulative difference in heat content (kJ) was calculated by integrating, over time, the difference in total cutaneous heat loss between the prewarming and no-warming treatments. Because metabolic rate was not measured, this method provides only an approximation of the actual difference in heat content. For calculating the equivalent change in mean body temperature, the specific heat of human tissue was taken as 3.5 kJ·°C⁻¹·kg⁻¹.

Repeated-measures analysis of variance and Dunnett's tests were used to evaluate time-dependent changes in temperatures and cutaneous heat loss within each treatment. Reference values were those obtained during the last acquisition epoch before induction of anesthesia. Differences between treatments at each time interval were compared using two-tailed, paired t tests.

The averages of each blood pressure parameter (systolic, diastolic, and mean) during the last 15 min of the preinduction period and the minimum for each blood pressure parameter during the first 15 min after induction were compared between the prewarming and no-warming treatments using two-tailed, paired t tests. The change in blood pressure parameters with induction (defined as the difference between the 15-min preinduction averages and the 15-min postinduction minimums) were similarly compared. The same analysis was applied to both oscillometric and radial arterial blood pressure measurements.

The difference between radial arterial and oscillometric blood pressure under different conditions was evaluated by grouping and averaging data on the basis of thermal treatment and anesthetic state. Four periods were defined: preinduction warming, preinduction no-warming, anesthesia prewarmed, and anesthesia not-warmed. Preinduction periods were defined as the second hour of treatment, because a thermoregulatory steady-state may not have been achieved throughout.
the first hour. Anesthesia periods were defined as the last 50-min of anesthesia, because hemodynamic conditions were unstable during the first 10 min. For each volunteer, the mean difference within each period was calculated by averaging the difference determined at 5-min intervals. The average difference and standard deviation for all volunteers during each period was determined from the individual averages. The difference was compared among periods using paired t and Scheffé’s F tests.

All data are reported as mean ± SD. Differences were considered statistically significant when $P < 0.05$.

Results

The age of volunteers was 26 ± 3 yr, weight 72 ± 15 kg, and height 177 ± 14 cm. There were two women and four men. Ambient temperatures during anesthesia were similar, 21.8 ± 0.7°C after prewarming versus 21.9 ± 0.8°C without prewarming. Blood propofol concentrations during anesthesia ranged from 1.7 to 9.0 μg/ml. Propofol concentrations determined before induction of the second anesthetic always were lower than 0.1 μg/ml. There were no statistically significant or clinically important differences between propofol concentrations during the second and first anesthetics. Similarly there were no significant differences in propofol concentrations during the anesthetic preceded by warming compared to that given without prewarming.

Temperature and Heat

Vasoconstriction (skin-temperature gradients >4°C) was present throughout the second hour of the preinduction period without warming, but vasodilation (skin-temperature gradient <0°C) occurred by the first recording epoch (10 min) after induction of anesthesia. Vasodilation was present in all volunteers during the second hour of preinduction warming and continued after induction of anesthesia. In both groups, vasodilation remained throughout anesthesia.

Initial tympanic membrane temperatures were similar before each preinduction treatment, 36.7 ± 0.4°C without warming and 36.7 ± 0.6°C with warming. Without warming, tympanic membrane temperatures did not change significantly before induction (fig. 1). In contrast, tympanic membrane temperature increased slightly during the 2-h preinduction warming ($ΔT = 0.4 ± 0.2°C$). Immediately before induction of anesthesia, tympanic membrane temperatures were 36.8 ± 0.2°C without warming and 37.1 ± 0.4°C with warming. During the subsequent hour of general anesthesia, core temperatures decreased significantly less when volunteers had been prewarmed, −1.1 ± 0.3°C than when they were not, −1.9 ± 0.3°C (fig. 1). Tympanic membrane temperatures after 1 h of anesthesia were significantly higher when volunteers had received prewarming than when they had not, 36.1 ± 0.4°C versus 34.9 ± 0.4°C (fig. 1).

Mean skin-surface temperatures were 33.0 ± 0.7°C at the beginning of the preinduction treatment without warming and subsequently decreased to 32.0 ± 0.7°C before induction ($P < 0.05$). Skin temperatures were 33.9 ± 0.8°C before preinduction warming and increased to 36.1 ± 0.8°C after 2 h of warming ($P <$
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0.05). Heat loss decreased from 111 ± 20 W at the start of the preinduction period to 89 ± 20 W at the end when volunteers were not warmed (fig. 2). During prewarming, heat loss averaged 18 ± 20 W. After induction of anesthesia (and after warming was discontinued), heat loss was slightly greater after volunteers were prewarmed, as expected from their slightly greater skin temperatures (fig. 2).

At the end of the preinduction period, the calculated difference in heat content when volunteers were prewarmed versus when they were not was 573 ± 196 kJ, which corresponds to ≈2.3°C difference in mean body temperature. The calculated difference after 1 h of anesthesia was 510 ± 192 kJ.

**Hemodynamics**

There were no significant differences in heart rate among the four defined periods. Blood pressure data are shown in figure 3. Compared to no-warming, prewarming was associated with significantly lower systolic (108 ± 16 versus 138 ± 15 mmHg), diastolic

- No Warming pre-Induction
- Warming pre-Induction

Fig. 3. Prewarming was associated with lower radial arterial systolic, diastolic, and mean pressures compared to no-warming. Oscillometric diastolic and mean pressures also were lower during prewarming. However, there was no statistically significant difference between the oscillometric systolic pressure during prewarming versus no-warming. Volunteers were exposed to the given preinduction treatment by the time of the first pressure measurements. Similar hypotension was observed after induction regardless of the preinduction treatment. However, because of the difference in preinduction blood pressures, the change in blood pressure with induction was significantly less when volunteers were prewarmed then when they were not. This difference was statistically significant for all parameters considered, i.e., radial arterial and oscillometric systolic, diastolic, and mean pressures. Data are presented as mean ± SD. Error bars are shown only for every third data point.

(56 ± 10 versus 68 ± 10 mmHg), and mean (75 ± 12 versus 88 ± 10 mmHg) radial arterial blood pressures during the 15-min period immediately before induction of anesthesia (P = 0.0001, 0.01, and 0.004, respectively; fig. 3). Prewarming also was associated with lower preinduction oscillometric diastolic and mean pressures (56 ± 8 versus 70 ± 8 mmHg, P = 0.02, and 74 ± 9 versus 90 ± 10 mmHg, P = 0.01, respectively). However, oscillometric systolic blood pressures did not

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systolic pressure, which decreased 58 ± 12 mmHg when volunteers were not warmed versus 30 ± 9 when they were prewarmed (P < 0.001). The smallest difference was noted for oscillometric systolic pressure, 26 ± 7 mmHg when volunteers were not warmed versus 18 ± 4 mmHg when they were prewarmed (P = 0.025).

Significant differences were observed in the difference between radial arterial and oscillometric blood pressure among the defined periods (fig. 4). The difference for systolic blood pressure measurements varied the most, 17 ± 13 mmHg during the no-warming preinduction period versus −6 ± 8 mmHg during prewarming (P < 0.05). Propofol/nitrous oxide anesthesia was associated with an even greater negative difference in systolic pressure measurements regardless of preinduction treatment (fig. 4).

Mean blood pressure measurement difference also varied significantly among periods (fig. 4). The difference was significantly more negative (radial arterial less than oscillometric pressure) after induction of anesthesia than it was before. The mean blood pressure measurement difference after induction was similar regardless of preinduction treatment (≈−12 mmHg). The difference between diastolic blood pressure measurements was small and without statistically significant differences among the defined periods.

Qualitatively, the radial arterial blood pressure waveforms changed dramatically between periods. Figure 5 shows typical radial arterial pressure waveforms recorded from one volunteer during each of the four defined periods.

Discussion

General anesthesia facilitates redistribution of heat from the core to peripheral tissues because anesthesia inhibits thermoregulatory vasoconstriction. The initial rapid decrease in core temperature after induction of general anesthesia is largely due to internal redistribution of body heat. In the present study, prewarming of peripheral tissues significantly reduced the initial decrease in core temperature after induction of anesthesia. This effect likely was due to increased peripheral tissue temperatures and heat contents after prewarming, because core temperatures increased only slightly during preinduction warming. Skin temperatures remained higher after induction of anesthesia, and consequently, cutaneous heat loss was greater after volunteers were prewarmed than when they were not. De-
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Fig. 5. Typical radial artery blood pressure waveforms recorded from one volunteer. The waveform during the no-warming preinduction period has a typical jagged appearance with a sharp systolic peak and two secondary peaks. The waveform recorded during prewarming appears slightly smoother, and the second peak is nearly absent. Waveforms recorded after induction of anesthesia appear much smoother, with no secondary peaks. Despite the smooth appearance of the waveforms after induction, the catheter-transducer system still had characteristics of an underdamped system. $\zeta = $ damping coefficient.

spite greater heat loss to the environment after prewarming, core temperature decreased less.

These data confirm our hypothesis that redistribution hypothermia can be prevented largely by preinduction warming of peripheral tissues. (The initial hypothermia after induction of epidural anesthesia similarly is caused by internal redistribution of heat and also can be minimized by preinduction warming.) The effectiveness of prewarming contrasts markedly with the failure of warming after induction of anesthesia to prevent redistribution hypothermia in patients.

Our results in volunteers are consistent with those reported by Trevien et al. for surgical patients. In their study, prewarmed patients remained nearly normothermic (typanic membrane temperatures 36.5 $\pm$ 0.1 $^\circ$C) after $\approx$3 h of general anesthesia and surgery. In contrast, core temperatures in control patients, who did not receive prewarming, were 35.2 $\pm$ 0.2 $^\circ$C after the same length of time. Less hypothermia than that observed in our study was likely due to intraoperative warming, which was given to both groups of patients. Additionally, their patients were covered by surgical drapes, whereas most of the skin was exposed in our volunteers.

Our estimated difference in heat content between the preinduction treatments, 573 kJ, is consistent with previous studies of heat storage in the human body. The estimated difference in the heat content at the end of 1 h of anesthesia, 510 kJ, suggests that several additional hours might be required before the core temperature curves would converge. It is therefore likely that, had we extended the anesthetic for 2 to 3 h, core temperature would have remained significantly higher when volunteers were prewarmed.

We chose a long period of gradual warming (with the forced-air warmer set at medium) to slowly increase tissue heat content. Had we warmed more aggressively, rapid changes in skin temperature may have triggered sweating. Sweating would greatly increase evaporative heat loss during and after discontinuation of warming and possibly would negate the benefit of prewarming. A much shorter period of warming would have warmed the most superficial tissues (mainly skin) but would likely not have prevented redistribution of heat to other tissues.

We observed a slight, statistically insignificant, decrease in core temperature during the first 20 min of warming. In contrast, we previously observed that 30 min of forced-air warming (using a higher air temperature of $\approx$43 $^\circ$C) slightly, but significantly, decreased core temperature. In that study, core hypothermia likely resulted when previously vasoconstricted vol-
unteers became vasodilated on exposure to a sudden increase in skin temperature. Vasodilation would lead to redistribution of heat from core to cooler peripheral tissues (although forced-air warming increases skin-surface temperature, most peripheral compartment tissues initially remain well below core temperature). In contrast, volunteers in the present study were covered with cotton blankets and maintained in a higher ambient temperature before active warming started. Consequently, they were somewhat vasodilated and presumably had smaller initial core-to-peripheral thermal gradients.

Our present results illustrate the importance of preinduction thermal status on subsequent core temperature patterns. Preoperative thermal status should be considered, particularly in patients undergoing surgical procedures for which maintenance of normothermia is critical. Thermal management of these patients may be facilitated by preventing redistribution hypothermia.

Many factors contribute to hypotension on induction of anesthesia. For example, propofol not only reduces preload, \textsuperscript{17} afterload, \textsuperscript{18} and contractility, \textsuperscript{19} but also inhibits the baroreceptor reflex. \textsuperscript{20} To some extent, decreased preload and afterload may result from inhibition of thermoregulatory vasoconstriction. We previously showed that propofol/nitrous oxide anesthesia significantly decreases the threshold for thermoregulatory vasoconstriction. \textsuperscript{21} Because heat stress and vasodilation are associated with a slight increase in central blood volume, \textsuperscript{5} we postulated that increased circulating blood volume during prewarming might help to maintain preload and thus decrease the hypotensive response to induction of anesthesia.

In our volunteers, prewarming did not prevent hypotension after induction of anesthesia. Likely, the direct vasodilation produced by propofol is more substantial than that produced by mild heating. Furthermore, prewarming would not have prevented decreases in contractility and baroreceptor response. Our volunteers were only exposed to mild heating, to avoid sweating. Therefore, maximal thermoregulatory vasodilation was not provoked. Under extreme heat stress, cutaneous blood flow can increase to as high as 7.5 l/min, \textsuperscript{5} greater than the entire resting cardiac output. Although more profound thermoregulatory vasodilation before induction might have prevented hypotension, changes in fluid status and central venous pressure due to sweating losses would have been difficult to control.

Despite failing to prevent absolute hypotension, pre-warming decreased the change in blood pressure associated with induction of anesthesia. This reduced relative hypotension was due to higher preinduction blood pressures when volunteers were not actively warmed. Thermal discomfort during exposure in a relatively cool environment may have contributed to higher preinduction pressures. However, for radial arterial systolic pressure, the larger decrease when volunteers were not warmed also was likely due to a sudden change from the sharply peaked waveform present during vasoconstriction to the smoothed waveform observed after induction.

Despite well known limitations of invasive blood pressure monitoring, particularly at peripheral sites, radial arterial pressure is widely regarded as the standard against which other methods are compared. Numerous studies have compared radial arterial blood pressure and noninvasive blood pressure measurements. \textsuperscript{22-24} In general, the correlation is only fair and considerable variability in the difference between measurement techniques exists among individuals. Interestingly, a study comparing oscillometric blood pressure measurements to central aortic measurements found a better correlation. \textsuperscript{25} Our present results indicate that, even within a given individual, the difference between measurement techniques is not constant. Both thermoregulatory and anesthetic-induced vasomotor changes may affect the direction and magnitude of this difference.

The difference between techniques varied most with respect to systolic pressure measurements. Systolic pressures often are used clinically to assess anesthetic depth, state of sympathetic activation, and adequacy of tissue perfusion. Our results suggest that radial arterial systolic blood pressure may be exaggerated during thermoregulatory vasoconstriction particularly when using an underdamped measurement system.

We can speculate on the reason for the change in direction of the blood pressure difference. Thermoregulatory vasoconstriction may increase the effect of factors that increase the pulse pressure as the pressure wave travels peripherally. The most important of these is the peripheral impedance, because this determines the degree of wave reflectance. \textsuperscript{26} Blood flow through thermoregulatory arteriovenous shunts varies more than tenfold, depending on whether shunts are open or closed. \textsuperscript{27} Thus, arteriovenous shunts affect peripheral impedance and, therefore, may modify the systolic pressure measured at the radial artery.
Anesthesia with propofol and nitrous oxide was associated with substantially lower radial arterial systolic and mean pressures compared to those measured oscillographically over the brachial artery. Normally, mean pressure in peripheral arteries is essentially the same as mean pressure in more central arteries: if no gradient existed, there would be no net forward blood flow. However, substantial gradients in mean blood pressure (aorta-to-radial artery) commonly develop during rewarming from cardiopulmonary bypass and have been attributed to decreased hand vascular resistance.²⁸,²⁹ Because anesthesia with propofol and nitrous oxide is associated with dilation of thermoregulatory arteriovenous shunts as well as direct arterial and venous dilation, decreased hand vascular resistance may have contributed to the higher brachial oscillometric, (vs. radial arterial) blood pressures measured in this study.

Unfortunately, we did not measure direct blood pressure from a more proximal site, such as the brachial or axillary artery. It remains possible that the oscillometric method underestimates systolic pressure during thermoregulatory vasomotor constriction and overestimates systolic and mean pressure during anesthesia with propofol and nitrous oxide. Others have reported oscillometric brachial artery pressures higher than direct radial artery pressures when radial artery pressures were less than 80 mmHg.²² These authors also did not have the benefit of more central invasive blood pressure measurements.

In summary, core temperature decreased significantly less during 1 h of propofol/nitrous oxide anesthesia when volunteers were warmed for 2 h before induction of anesthesia, indicating that prewarming minimizes redistribution hypothermia. Prewarming did not reduce hypotension with induction of general anesthesia but did decrease the preinduction blood pressure. Consequently, prewarming decreased the change in blood pressure, or relative hypotension, after induction of anesthesia. Significant changes in the difference between radial arterial and oscillometric blood pressure were noted under different thermal and anesthetic conditions. These changes likely reflect a greater sensitivity of radial arterial blood pressure to thermoregulatory and anesthetic-induced peripheral vasomotor changes and suggest that, under varying conditions, oscillometric blood pressure more consistently reflects central blood pressure.

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