Heat Balance and Distribution during the Core-Temperature Plateau in Anesthetized Humans

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Background: Once triggered, intraoperative thermoregulatory vasoconstriction is remarkably effective in preventing further hypothermia. Protection results from both vasoconstriction-induced decrease in cutaneous heat loss and altered distribution of body heat. However, the independent contributions of each mechanism have not been quantified. Accordingly, we evaluated overall heat balance and distribution of heat within the body during the core-temperature plateau.

Methods: Nine minimally clothed male volunteers were anesthetized with propofol and isoflurane and maintained in an 18°C environment. They were monitored for 2 h before vasoconstriction and for 3 h subsequently. Overall heat balance was determined from the difference between cutaneous heat loss (thermal flux transducers) and metabolic heat production (oxygen consumption). Arm and leg tissue heat contents were determined from 19 intramuscular temperatures, ten skin temperatures, and “deep” foot temperature. Heat constrained by vasoconstriction to the trunk and head was calculated by subtracting the expected change in that region (overall heat balance multiplied by the fractional weight of the trunk and head) from the actual change (change in distal esophageal temperature multiplied by the specific heat of human tissue and the weight of the trunk and head); the result represents the amount by which core heat exceeded that which would be expected based on overall heat balance, assuming that the change was evenly distributed throughout the body.

Results: Vasoconstriction and passive tissue cooling decreased heat loss but not to the level of heat production. Consequently, heat loss exceeded metabolic heat production throughout the study. Core temperature decreased 1.3°C during the 2-h prevasoconstriction period; however, core temperature remained virtually constant during the subsequent 3 h. In the 3 h after vasoconstriction, arm and leg heat content decreased 57 ± 9 kcal, and vasoconstriction constrained 22 ± 8 kcal to the trunk and head.

Conclusions: These results confirm the efficacy of thermoregulatory vasoconstriction in preventing additional core hypothermia. Decreased cutaneous heat loss and constraint of metabolic heat to the core thermal compartment contributed to the plateau. (Key words: Anesthetics, intravenous: propofol. Anesthetics, volatile: isoflurane. Brain: hypothalamus. Heat. Hypothermia. Thermoregulation: vasoconstriction; vasodilation. Temperature, regulation: setpoint; threshold; vasocostriction.)

VASOCONSTRICTION is the only major thermoregulatory defense against hypothermia usually available during surgery. Once triggered, \( ^1 \) intraoperative thermoregulatory vasoconstriction is remarkably effective in minimizing further hypothermia. \( ^2, ^4 \) Protection results in part from a vasoconstriction-induced decrease in cutaneous heat loss. \( ^5 \) However, we demonstrated in a previous investigation that leg heat content decreased 50 kcal in the 3 h after thermoregulatory vasoconstriction—although core temperature decreased only slightly. \( ^6 \) These data suggested that altered distribution of body heat contributed to the core-temperature plateau.

Our previous study investigating the effects of thermoregulatory vasoconstriction on body heat distribution had several limitations. First, we evaluated leg heat content by using a two-compartment model; we have recently switched to a nine-compartment model that presumably provides greater accuracy. Second, we did not evaluate changes in arm heat content, although a subsequent study has indicated that the arms (despite their small size) contribute significantly to thermal buffering. \( ^7 \) Third, we did not measure overall heat balance and therefore were unable to separate completely the effects of decreased body heat content from changes in heat distribution.

The ability of vasoconstriction to alter distribution of body heat is clinically important because it deter-
mines the extent to which core temperature can be maintained during periods of negative heat balance. It also defines the amount by which body heat content can decrease without altering clinicians by reducing core temperature. This reduction is potentially important because intraoperative heat loss must be replaced postoperatively, often by shivering. Accordingly, we evaluated overall heat balance and distribution of heat within the body before and after thermoregulatory vasocostriction in anesthetized volunteers. This allowed us to test the hypothesis that constraint of metabolic heat to the core thermal compartment contributes to the core-temperature plateau.

Materials and Methods

With approval from the Committee on Human Research and written informed consent, we studied nine male volunteers. None was obese; was taking medication; or had a history of thyroid disease, dysautonomia, Raynaud's syndrome, or malignant hyperthermia. Each participated on a single study day in November or December 1994.

The volunteers' height was 175 ± 4 cm (mean ± SD), weight was 74 ± 11 kg, and age was 31 ± 6 yr. The percentage of body fat was 18 ± 4 (Futrex 1000, Futrex, Hagerstown, MD). Ambient temperature was maintained at 21.6 ± 0.4°C and ambient relative humidity at 34 ± 8% during the study period (humidity and temperature transmitter HX93, Omega Engineering, Stamford, CT).

Protocol

Studies started at approximately 9:30 AM, and volunteers fasted during the 8 h preceding each study. An intravenous catheter was inserted into an antecubital vein on the left arm. Throughout the study, the minimally clothed volunteers reclined on an operating room table set in a chaise-lounge position. Approximately 10 ml/kg lactated Ringer's solution warmed to 37°C was infused immediately before induction of anesthesia. Subsequently, fluid was given at a rate of 100 ml/h.

Anesthesia was induced without any premedication by administration of propofol 0.7 mg/kg and midazolam 5 mg. Vecuronium, 10 mg, was administered intravenously, and the trachea was intubated. Muscle relaxation was subsequently maintained with an infusion of vecuronium adjusted to maintain 0–1 twitches in response to supramaximal train-of-four electrical stimulation of the ulnar nerve at the wrist. Ventilation was controlled by a ventilator incorporated into a Molulus CD integrated anesthesia machine (Ohmeda, Madison, WI). The system was modified so respiratory gases were not rebreathed. The volunteers' lungs were ventilated with air at a rate and volume sufficient to maintain end-tidal carbon dioxide tension near 35 mmHg.

The volunteers were not covered during the study. To simulate heat loss that might be encountered during surgery, a fan set to low power was positioned 1 m from the volunteers' feet, with the air stream directed toward the head. Anesthesia was maintained with an infusion of propofol (=150 µg·kg⁻¹·min⁻¹). When necessary to prevent peripheral vasocostriction (see below), isoflurane was added. Sufficient propofol and isoflurane were administered to prevent thermoregulatory vasocostriction for at least 2 h after induction of anesthesia. Subsequently, isoflurane administration was gradually discontinued and the propofol infusion rate decreased to trigger arteriovenous shunt vasocostriction. Vasocostriction was then maintained for the remaining 5 h of the study.

Heat Balance Monitoring

Heat flux from 15 skin-surface sites was measured with thermal flux transducers (Concept Engineering, Old Saybrook, CT). As in a previous study, measured cutaneous heat loss was augmented by 10% to account for insensible transtrunci cutaneous evaporative loss and 3% to compensate for the skin covered by the volunteers' short pants. We further augmented cutaneous loss by 10% of the metabolic rate (as determined from oxygen consumption) to account for respiratory loss, and we defined flux as positive when heat traversed the skin to the environment.

Oxygen consumption was measured using a metabolic monitor (DeltaTrac, SensorMedics, Yorba Linda, CA). The system was calibrated daily using a known mixture of gases and also calibrated numerous times by burning ethanol. Measurements were averaged over 1-min intervals and recorded every 5 min. Oxygen consumption (milliliters per minute) was converted to equivalent metabolic heat production (watts) assuming the caloric value of oxygen to be 4.82 kcal/l (respiratory quotient = 0.82), and using a conversion of 1 kcal/l = 1.16 W. We chose a standard value for the respiratory quotient because the caloric value of oxygen varies only slightly over the full range of respiratory quotients; the use of a standard value thus in...
produc es minimal error in the calculation of metabolic heat production.\textsuperscript{14}

**Blood Flow and Anesthetic Monitoring**

Right calf minus toe, skin-surface temperature gradients were used as an index of foot arteriovenous shunt perfusion.\textsuperscript{1} A gradient exceeding 0ºC indicated vasoconstriction.\textsuperscript{6} Total digital flow also was evaluated on the right second toe using the perfusion index, which is derived, using the same principle as in pulse oximeters, from absorption of two infrared wavelengths. The index is calculated from the combined absorption of the two wavelengths.\textsuperscript{15} Left calf blood flow was quantified using capacitance-based “extensometer” plethysmography.\textsuperscript{16,17} Plethysmography is often used to measure cutaneous capillary blood flow, in which case arteriovenous shunts in the hand or foot are isolated by an arterial tourniquet.\textsuperscript{18} In this study, however, we avoided a distal tourniquet because we were interested in total extremity blood flow.

Right forearm minus fingertip, skin-surface temperature gradients were used as an index of hand arteriovenous shunt perfusion.\textsuperscript{19} We again considered a gradient exceeding 0ºC to indicate vasoconstriction. Absolute right middle fingertip blood flow was quantified using venous-occlusion volume plethysmography at 5-min intervals.\textsuperscript{19} Vasoconstriction was also estimated using laser Doppler flowmetry (Periflux 3, Perimed, Piscataway, NJ) with an integrating multiprobe (wideband setting) positioned on the right fourth finger.\textsuperscript{20,21} Thermoregulatory data were recorded electronically at 5-min intervals.

**Tissue Temperature and Heat Content**

Core temperature was measured in the distal esophagus, with a probe positioned according to the formula of Mekjavic and Rempel.\textsuperscript{22} Trunk and head skin temperatures were calculated by assigning the following regional percentages: head 20%, chest 20%, abdomen 20%, and back 40%.\textsuperscript{23} Skin-surface temperatures were recorded from thermocouples incorporated into thermal flux transducers.

Arm and leg tissue temperatures were determined as previously described.\textsuperscript{7} In brief, the length of the thigh (groin to mid-patella) and lower leg (mid-patella to ankle) were measured in cm. Circumference was measured at the mid upper thigh, mid lower thigh, mid upper calf, and mid lower calf. At each circumference, right leg muscle temperatures were recorded with 8-, 18-, and 38-mm 21-G needle thermocouples (Mallinckrodt Anesthesiology Products, St. Louis, MO) inserted perpendicular to the skin surface. Skin-surface temperatures were recorded immediately adjacent to each set of needles and directly posterior to each set. Subcutaneous temperature was measured on the ball of the foot using a Coretemp (Terumo Medical, Tokyo, Japan) deep-tissue thermometer.\textsuperscript{24} This device estimates tissue temperature ±1 cm below the skin surface.

The lengths of the right arm (axilla to elbow) and forearm (elbow to wrist) were measured in centimeters. The circumference was measured at the mid-point of each segment. As in the right leg, 8-, 18-, and 38-mm needle thermocouples were inserted into each segment. Skin-surface temperatures were recorded immediately adjacent to each set of needles. In addition, an 8-mm needle thermocouple was inserted directly into the adductor pollicis.\textsuperscript{25} Core, skin-surface, and muscle temperatures were recorded from thermocouples connected to two calibrated Iso-Thermex 16-channel electronic thermometers (Columbus Instruments International, Columbus, OH) and Mon-a-Therm 6510 two-channel thermometers (Mallinckrodt Anesthesiology Products).

The leg was divided into five segments: upper thigh, lower thigh, upper calf, lower calf, and foot. Each thigh and calf segment was further divided into an anterior and posterior section, with one third of the estimated mass considered to be posterior.

Anterior segment tissue temperatures, as a function of radial distance from the center of the leg segment, were calculated using skin-surface and muscle temperatures using parabolic regression. Temperature at the center of the thigh was set to core temperature. In contrast, temperature at the center of the lower leg segments was estimated from the regression equation with no similar assumption. Limb heat content was estimated from these temperatures, as previously described,\textsuperscript{6} using the formula:

\[
Q_{0\rightarrow r_1} = 2(\pi r^2)L\rho s a_0 + \frac{a_2 r^2}{2},
\]

where \(Q_{0\rightarrow r_1}\) (calories) = heat content of the leg segment from the center to radius \(r\); \(L\) (centimeters) = the length of the leg segment (i.e., groin to mid-thigh, mid calf to ankle); \(\rho\) (grams per cubic centimeter) = tissue density; \(s\) (calories per degree Celsius per gram) = the specific heat of leg tissues; \(a_0\) (degrees Celsius) = the temperature at the center of the leg segment; and \(a_2\) (degree Celsius per squared centimeter) = the parabolic regression constant. The specific heat of muscle...
was taken as 0.89 cal·C⁻¹·g⁻¹ and density as 1.06 g/cm³.²⁶

Rather than assume full radial symmetry, we assumed only that radial temperature distribution in the posterior leg segments would also be parabolic. Accordingly, we calculated the regression constant a₂ in the posterior leg segments from a₀ determined from the adjacent anterior segment and the posterior segment skin temperature. Posterior segment tissue heat contents were then determined from equation 1. Average segment tissue temperatures (Tₐₘ) were determined by the equation:

\[ T_{av} = a_0 + \frac{a_2 r^2}{2} \]  

We have previously described the derivation of these equations and their limitations.⁶

'Deep' temperature, measured on the ball of the foot, was assumed to represent the entire foot. Foot heat content thus was calculated by multiplying foot temperature by the mass of the foot and the specific heat of muscle. Average temperatures of the thigh and lower leg (calf and foot) were calculated by weighting values from each of the nine segments in proportion to their estimated masses. The right and left leg were treated comparably throughout this study, so we assumed that average tissue temperatures in the two limbs were similar.

Arm tissue temperature and heat content were calculated from parabolic tissue temperature regressions and the above equations. In the arms, we assumed full radial symmetry and thus did not separately calculate posterior segment values. Adductor pollicis temperature was assumed to represent that of the entire hand. Hand heat content thus was calculated by multiplying adductor pollicis temperature by the mass of the hand and the specific heat of muscle. As in the leg, average temperatures of the arm and forearm (forearm and hand) were calculated by weighting values from each of the three segments in proportion to their estimated masses.

Changes in trunk and head heat content were modeled simply by multiplying the weight of the trunk and head by the change in core temperature and the average specific heat of human tissues. Trunk and head weight was estimated by subtracting the calculated weight of the extremities (from the radial integration) from the total weight of each subject.

Statistical Analysis

Results from the 1st h after induction of anesthesia were discarded because redistribution is the most important cause of core hypothermia during this period. All subsequent results were indexed to the time when both arm and leg skin-temperature gradients first exceeded 0°C (elapsed time zero). The preanesthesia period thus extended from 1 h after induction of anesthesia until elapsed time zero.

Changes in mean body temperature were determined by multiplying the difference between heat production and loss by the average specific heat of human tissue (0.83 cal·C⁻¹·g⁻¹) and body mass.²⁷ Heat constrained by vasoconstriction to the trunk and head was calculated by subtracting the expected change in that region (overall heat balance multiplied by the fractional weight of the trunk and head) from the actual change (change in distal esophageal temperature multiplied by the specific heat of human tissue and the weight of the trunk and head).

Overall decreases in body heat were calculated first as the time integral of metabolic heat production minus cutaneous heat loss. Decreases in heat content were also estimated by the sum of the change in extremity heat content and the change in core temperature multiplied by the specific heat of human tissue and the weight of the trunk and head.

Metabolic heat production and mean body and core temperatures were correlated using linear regression. Time-dependent changes were evaluated using repeated-measures analysis of variance; values were compared with those recorded at elapsed time zero (arm and leg gradients of 0°C) with Dunnett’s test. Results are expressed as means ± SD; differences were considered statistically significant when P < 0.01.

Results

Estimated mass of the thighs and lower legs (including feet) were 15 ± 3 kg and 9 ± 1 kg, respectively. Consequently, the legs represented ~32% of our volunteers’ total mass. Similarly, estimated mass of the arms and forearms (including hands) were 5 ± 1 kg and 4 ± 1 kg, respectively. Consequently, the arms represented ~12% of our volunteers’ total mass.

Vasoconstriction occurred at a core temperature of 33.2 ± 0.4°C, 3.3 ± 0.5 h after induction of anesthesia. Consequently, the length of the preanesthesia period (excluding the first h of anesthesia) exceeded 2 h in six of nine volunteers and 1.5 h in eight of nine volunteers. All measures of blood flow indicated intense vasoconstriction at elapsed time zero. From ~2 to 5 h elapsed, finger flow decreased more than tenfold.
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Fig. 1. Vasoconstriction and passive tissue cooling decreased cutaneous heat loss (adjusted for evaporative and respiratory loss) \( \approx 25 \) kcal/h. However, heat loss always exceeded heat production. Consequently, mean body temperature, which decreased at a rate of \( \approx 0.6^\circ\)C/h before vasoconstriction, subsequently decreased at a rate of \( \approx 0.2^\circ\)C/h. Core temperature also decreased at a rate of \( \approx 0.6^\circ\)C before vasoconstriction but remained virtually constant during the subsequent 3 h. Because mean body temperature and body heat content continued to decrease, constraint of metabolic heat to the core thermal compartment contributed to the core-temperature plateau. That is, vasoconstriction reestablished the normal core-to-peripheral temperature gradient by preventing metabolic heat (which is largely generated in the core) from escaping to peripheral tissues. Constrained heat is presented cumulatively, referenced to vasoconstriction at elapsed time zero. Values significantly different from those obtained at elapsed time zero.

While arm and leg gradients increased from \( \approx -2^\circ\)C to \( \approx +5^\circ\)C. During this period, laser Doppler, perfusion index, and extensometer flows all decreased approximately tenfold. All measures of flow at all times before 0.75 h and after 1.25 h differed significantly from those at elapsed time zero.

Initial metabolic heat production (as determined from oxygen consumption), which was \( 56 \pm 9 \) kcal/h, decreased linearly as mean body temperature decreased (slope \( = 4\%/{^\circ}\)C; \( r^2 = 0.78 \)). However, when heat production was correlated with core temperature, the decrease was \( 8\%/{^\circ}\)C. Initial cutaneous heat loss (adjusted for evaporative and respiratory loss) was \( 99 \pm 10 \) kcal/h but decreased to \( 59 \pm 5 \) kcal/h by the end of the study. Consequently, heat loss always exceeded heat production and mean body temperature decreased throughout the study.

Core temperature decreased \( \approx 1.2^\circ\)C during the 2-h vasoconstriction period; however, core temperature remained virtually constant during the subsequent 3 h. This core-temperature plateau resulted from vasoconstriction that decreased cutaneous heat loss and constrained \( 22 \pm 8 \) kcal to the trunk and head (fig. 1). "Constraint," in this context, refers to accumulation of heat in the core thermal compartment in excess of what would be predicted by the change in overall body heat content (assuming the change were evenly distributed throughout the body). It indicates the extent to which vasoconstriction has reestablished the normal core-to-peripheral temperature gradient.

The difference between core and mean trunk skin temperature increased only \( 0.8^\circ\)C in the 3 h after vasoconstriction, indicating that vasoconstriction only slightly altered distribution of heat within this tissue mass. Consequently, changes in body heat content were similar when calculated as (1) the integrated difference between heat loss and production and (2) the sum of measured changes in the arms and legs and the change in mean temperature multiplied by the weight of the core and the specific heat of human tissues (fig. 2).
extremity tissue heat content similarly decreased at a rate of \( \approx 10 \text{ kcal/h} \) during this period. After vasoconstriction, distal tissue heat content decreased at a rate of \( \approx 12 \text{ kcal/h} \), whereas proximal heat content decreased only \( \approx 7 \text{ kcal/h} \). Average tissue heat content in the combined proximal and distal extremities thus decreased \( \approx 57 \text{ kcal} \) during the 3 h after vasoconstriction (Fig. 4).

**Discussion**

Core temperatures decreased at a rate of \( \approx 0.6^\circ\text{C/h} \) before vasoconstriction but remained virtually constant during the subsequent 3 h of anesthesia. We confirm that vasoconstriction was markedly effective in preserving core temperature, as observed previously. Heat was dissipated at a comparable rate during vasoconstriction. The decrease in linear rate of heat loss was derived from decreased tissue perfusion and skin temperature. The subcutaneous fat resulted from a vasoconstriction of the core-to-peripheral tissues. Metabolic heat production was a function of mean body and skin temperature. Heat loss, however, remained relatively constant throughout the study. The observed decrease in mean body surface area over the period. Heat loss was expected to contribute to the plateau.

We noted an alteration in the distal heat content during vasoconstriction. Heat content also decreased by \( \approx 50 \text{ kcal} \) (Fig. 4). Arm and leg heat contents did not change substantially throughout the study. The plateau in heat content could be due to the combined proximal and distal extremities. The distal extremity tissue temperature decreased at a rate of \( \approx 0.8^\circ\text{C/h} \) before vasoconstriction. Distal extremity tissue temperature similarly decreased at a rate of \( \approx 1.1^\circ\text{C/h} \) during this period. After vasoconstriction, distal tissue temperature decreased at a rate of \( \approx 0.4^\circ\text{C/h} \). Average tissue temperature in the combined proximal and distal extremities thus decreased \( \approx 2^\circ\text{C} \) during the 3 h after vasoconstriction.

Proximal extremity tissue heat content decreased at a rate of \( \approx 14 \text{ kcal/h} \) before vasoconstriction. Distal extremity tissue heat content similarly decreased at a rate of \( \approx 10 \text{ kcal/h} \) during this period. After vasoconstriction, distal tissue heat content decreased at a rate of \( \approx 12 \text{ kcal/h} \), whereas proximal heat content decreased only \( \approx 7 \text{ kcal/h} \). Average tissue heat content in the combined proximal and distal extremities thus decreased \( \approx 57 \text{ kcal} \) during the 3 h after vasoconstriction. Results are presented cumulatively, referenced to vasoconstriction at elapsed time zero. All values before \(-0.5\) h and after \(+1\) h differed significantly from those at vasoconstriction.
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during the subsequent 3 h of anesthesia. These results confirm that vasoconstriction, once triggered, is remarkably effective in preventing further core hypothermia.\textsuperscript{2, 4}

As observed previously,\textsuperscript{5} cutaneous heat loss decreased at a comparable rate before and after vasoconstriction. The decrease before vasoconstriction presumably resulted from decreasing core (and therefore skin) temperature. The subsequent decrease, however, resulted from a vasoconstriction-induced increase in the core-to-peripheral tissue temperature gradient.

Metabolic heat production decreased linearly as a function of mean body and core temperatures, as might be expected. Heat loss, however, never decreased to the level of metabolic heat production; consequently, overall heat balance remained negative throughout the study. The observed decrease in body heat content—were it evenly distributed—would be expected to decrease core temperature 0.7 ± 0.3°C in the 3 h after vasoconstriction. However, core temperature remained nearly constant, indicating that another mechanism also contributed to the plateau. That mechanism appears to be an alteration in the distribution of body heat.\textsuperscript{6}

Leg heat content decreased 36 ± 6 kcal during the 3 h after vasoconstriction. But importantly, arm heat content also decreased by 20 ± 5 kcal. These data indicate that the arms, despite their relatively small size, contribute substantially to thermal buffering. This result is consistent with the observation that the arms contribute significantly to redistribution hypothermia.\textsuperscript{7}

We stopped the study after 3 h of vasoconstriction to limit total anesthesia time to ≈ 6 h. Combined arm and leg heat content decreased 57 ± 9 kcal during vasoconstriction. This value is less than half the estimated maximum buffering capacity of the peripheral thermal compartment,\textsuperscript{2, 29} and core temperature remained constant, even at the end of this period. It thus appears that the capacity of peripheral tissues to buffer decreased body heat content was not exhausted and that a negative heat balance could have been sustained even longer without decreasing core temperature.

Thermoregulatory physiologists often divide the body into core and peripheral thermal compartments.\textsuperscript{30} The core is defined functionally by tissues maintaining high and similar temperatures, even during rapid thermal perturbations and extreme cold exposure. All other tissues are considered "peripheral." (Although peripheral tissue temperatures can equal or exceed core temperature under some circumstances, these tissues remain part of the peripheral compartment.) Despite the formal functional definition of thermal compartments, tissue volumes in this study were assigned anatomically (e.g., the posterior third of the lower thigh). Nonetheless, it is roughly correct to consider the trunk and head to be core and the arms and legs to be peripheral.\textsuperscript{30}

In previous similar investigations we considered the thigh to extend from the knee to the anterior iliac crest.\textsuperscript{6, 7} In one,\textsuperscript{7} however, temperature and heat content changes in the upper thigh differed both in direction and magnitude from those in more peripheral tissues. Temperature changes in the upper thigh resembled those in core, rather than those in peripheral buffering tissues. Consequently, we currently define the thigh as extending only to the groin. This more restrictive definition increases trunk mass at the expense of leg volume but is more consistent with considering the arms and legs to be peripheral. As one would expect from restricting leg volume, leg heat content decreased only ≈ 36 kcal in our current study, whereas a decrease of 49 ± 18 kcal was reported previously.\textsuperscript{6}

Patients undergoing surgery lose heat by evaporation from within incisions,\textsuperscript{31} although the amount remains to be quantified. Our volunteers of course did not lose heat through surgical incisions, but they were nearly completely exposed to a cool, convective environment during the study. Cutaneous heat loss was thus at least 30% greater than in surgical patients covered with even a single layer of surgical draping.\textsuperscript{32} Furthermore, the rate of core cooling before vasoconstriction was ≈ 0.6°C/h, which is typical for the linear phase. These observations suggest that overall cold exposure in our volunteers was similar to that experienced by most surgical patients. Efficacy of the core-temperature plateau is thus likely to be comparable in most surgical patients, and constraint of metabolic heat to the core thermal compartment is again likely to be an important mechanism protecting core temperature. Vasoconstricted surgical patients may, therefore, lose up to ≈ 60 kcal without any change in core temperature to alert clinicians.

In hypothermic surgical patients, thermoregulatory vasoconstriction usually prevents further decrease in core temperature. However, the ability of thermoregulatory vasoconstriction to minimize core hypothermia depends on the amount of heat loss. When large operations or extreme cold exposure excessively increase heat loss, vasoconstriction may produce a decreased rate of core cooling rather than a plateau.\textsuperscript{3, 4} Conversely,
in a warm environment, vasocostriction may even increase core temperature.3

Addition of a volatile anesthetic potentially confounds our measurements of metabolic heat production because the Deltrac monitor is not designed to compensate for tissue absorption of the vapor. Using different systems, it is possible to compensate for tissue absorption of volatile anesthetics.25 However, the potential error is trivial as long as inspired and expired concentrations of the volatile anesthetic are similar.33

In a previous similar heat balance investigation, we augmented heat loss by 5% of the metabolic rate to compensate for evaporative and conductive respiratory losses.2 However, we used a heat-and-moisture-exchanging filter in that study but not in the current investigation. We thus augmented heat loss by 10% in this study, which approximates respiratory heat loss during in subjects whose lungs are ventilated with dry, cold gases with a nonbreathing system.13

Rather than directly evaluate distribution of heat within the trunk and head, we considered these tissues to be uniform and applied changes in core temperature to the entire mass. This assumption is surely not strictly valid: the difference between core and trunk skin temperatures increased slightly during the study, suggesting that tissue temperature in outer portions of the trunk decreased relative to the core. We thus at least slightly underestimated the reduction in trunk and head heat content after vasocostriction.

Our use of heat flux transducers to estimate overall heat loss is fraught with potential errors: (1) transducers are only certified to ±5%, and the calibration process is tricky31; (2) flux across individual transducers was extrapolated to the entire region assigned to each; and (3) we augmented flux to account for insensible cutaneous and respiratory heat losses without directly measuring either. Similarly, the potential error in estimating metabolic heat production from oxygen consumption is probably near 10%. Finally, our estimates of tissue heat content involve numerous assumptions, including (1) minimal conduction along the stainless steel shaft of the needle thermocouples, (2) radial and axial symmetry within extremity segments; and (3) homogeneous tissue specific heats.

To the extent that measurement errors were randomly distributed, they would only add variability to the results without substantially influencing mean values. In contrast, systematic errors (such as recording of a falsely low metabolic rate in every volunteer) would produce potentially serious bias in the results that could not be detected statistically. Nonetheless, our estimates of overall changes in body heat balance were similar using two independent methods, suggesting that our results reasonably describe the consequences of intraoperative vasocostriction on body heat distribution.

In summary, vasocostriction during anesthesia decreased heat loss but not to the level of heat production. Consequently, heat loss continued to exceed metabolic heat production. Core temperature decreased ≈1.3°C during the 2-h prevasocostriction period but remained virtually constant during the subsequent 3 h. In the 3 h after vasocostriction, arm and leg heat content decreased 57 ± 9 kcal and vasocostriction constrained 22 ± 8 kcal to the trunk and head. These results confirm the efficacy of thermoregulatory vasocostriction in preventing additional core hyperthermia. Decreased cutaneous heat loss and constraint of metabolic heat to the core thermal compartment contribute to the core-temperature plateau.

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