

Leg Heat Content Continues to Decrease during the Core Temperature Plateau in Humans Anesthetized with Isoflurane

Kumar Belani, M.D.,* Daniel I. Sessler, M.D.,* Andrew M. Sessler, Ph.D.,† Marc Schroeder, B.A.,‡ Joseph McGuire, B.S.,‡ Benjamin Merrifield, B.A.,‡ Denna E. Washington, M.D.,§ Azita Moayeri, B.A.‡

Background: Sufficient hypothermia during anesthesia provokes thermoregulatory responses, but the clinical significance of these responses remains unknown. Nonshivering thermogenesis does not increase metabolic heat production in anesthetized adults. Vasoconstriction reduces cutaneous heat loss, but the initial decrease appears insufficient to cause a thermal steady state (heat production equaling heat loss). Accordingly, the authors tested the hypotheses that: 1) thermoregulatory vasoconstriction prevents further core hypothermia; and 2) the resulting stable core temperature is not a thermal steady state, but, instead, is accompanied for several hours by a continued reduction in body heat content.

Methods: Six healthy volunteers were anesthetized with isoflurane (0.8%) and paralyzed with vecuronium. Core hypothermia was induced by fan cooling, and continued for 3 h after vasoconstriction in the legs was detected. Leg heat content was calculated from six needle thermocouples and skin temperature, by integrating the resulting parabolic regression over volume.

Results: Core temperature decreased $1.0 \pm 0.2^\circ\text{C}$ in the 1 h before vasoconstriction, but only $0.4 \pm 0.3^\circ\text{C}$ in the subsequent 3 h. This temperature decrease, evenly distributed throughout the body, would reduce leg heat content 10 kcal. However, measured leg heat content decreased 49 ± 18 kcal in the 3 h after vasoconstriction.

Conclusions: These data thus indicate that thermoregulatory vasoconstriction produces a clinically important reduction in the rate of core cooling. This core temperature plateau resulted, at least in part, from sequestration of metabolic heat to the core which allowed core temperature to remain nearly constant, despite a continually decreasing body heat content.

(Key words: Anesthesia, volatile: isoflurane. Brain: hypothalamus. Heat. Hypothermia. Measurement techniques, blood flow: laser Doppler; skin-temperature gradient; vasomotor index; volume plethysmography. Temperature, measurement: skin; esophageal. Temperature, regulation: setpoint; threshold; vasoconstriction. Thermoregulation. Vasoconstriction, vasodilation: thermoregulatory.)

CORE body temperature typically decreases rapidly during the first 1 h of anesthesia because inhibition of tonic thermoregulatory vasoconstriction permits internal redistribution of body heat from core to peripheral tissues.¹ Redistribution hypothermia is followed by a slow, linear decrease in core temperature, probably resulting from heat loss exceeding metabolic heat production.² Subsequent temperature changes have not been systematically evaluated. However, after 3–4 h of anesthesia, the progressive decrease in core temperature often stops.³ In some patients, this core temperature plateau may simply be a passive thermal steady state during which heat production equals heat loss.⁴ In others, however, the core temperature plateau is apparently accompanied by active peripheral vasoconstriction.⁵

A core temperature plateau at the time of thermoregulatory vasoconstriction indicates that vasoconstriction decreases heat loss to the environment or that nonshivering thermogenesis increases metabolic heat production. However, our previous data suggest that thermoregulatory vasoconstriction in unanesthetized⁶ and anesthetized⁵ volunteers may not immediately reduce cutaneous heat loss sufficiently to compensate for large intraoperative heat losses.⁷ Similarly, we have shown that nonshivering thermogenesis does not occur in anesthetized adults.⁸ An additional explanation for the core temperature plateau in hypothermic patients may be that vasoconstriction initially alters the internal distribution of body heat; in effect, reestablishing the normal core-to-peripheral temperature gradient.

Vasoconstriction cannot directly recover redistrib-

* Associate Professor of Anesthesia.

† Senior Scientist.

‡ Staff Research Associates

§ Clinical Instructor.

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Address reprint requests to Dr. D. Sessler: Department of Anesthesia, Room C-214, University of California, San Francisco, California 94143-0648.

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uted heat from the periphery (because heat moving *up* a temperature gradient would violate the Second Law of Thermodynamics). But the gradient could be reestablished were vasoconstriction to sufficiently reduce transfer of metabolic heat, which is largely generated in the core, to the peripheral thermal compartment. In this scenario, peripheral compartment tissue temperature, and, thus, total body heat content, would continue to decrease after vasoconstriction despite a constant core temperature, because cutaneous heat loss from peripheral tissues would continue at a relatively high rate, while less heat was supplied from the core.

Subsequently, peripheral tissue hypothermia would decrease cutaneous heat loss, which is roughly proportional to the skin-to-environmental temperature gradient. If the reduction in heat loss were sufficient to balance metabolic heat production and cutaneous heat loss, the patient would, at that point, enter a thermal steady state. If the reduction were not sufficient, however, core temperature would again begin to decrease.

Although the peripheral compartment includes the arms and the outer layers of the trunk, only the legs have sufficient mass to constitute a clinically important thermal buffer. Accordingly, we tested the hypotheses that: 1) thermoregulatory vasoconstriction causes a core temperature plateau; and 2) the resulting plateau is not initially a thermal steady state, but, instead, is accompanied for several hours by a continued reduction in body heat content.

Materials and Methods

Following approval of the University of California, San Francisco Committee on Human Research, we studied six consecutive volunteers. None was obese, was taking medication, nor had a history of thyroid disease, dysautonomia, or Raynaud's syndrome. During the study, minimally clothed volunteers reclined on a standard operating room table. Ambient temperature was maintained at $23.1 \pm 0.8^\circ\text{C}$ and ambient relative humidity at $55 \pm 3\%$ during the study period (Model HX93 humidity and temperature transmitter; Omega Engineering, Stamford, CT).

The percentage of body fat in each volunteer was determined using infrared interactance (Futrex 1000; Futrex, Hagerstown, MD).⁹ The length of the thigh (anterior iliac spine to midpatella) and lower leg (midpatella to sole of foot) were measured in centimeters. Similarly, circumference was measured at the top,

midpoint, and bottom (midpatella, ankle) of each leg segment.

Experimental Procedure

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 right arm. Lactated Ringer's solution warmed to 37°C was infused at $\approx 100\text{ ml/h}$. Anesthetic-induced redistribution hypothermia¹ was minimized by preinduction skin-surface warming¹⁰ using a forced-air warming device (Bair Hugger®; Augustine Medical, Eden Prairie, MN).¹¹

Anesthesia was induced without any premedication by inhalation of isoflurane 3–4%, nitrous oxide 70%, and oxygen. Thiopental and opioids were not administered. Vecuronium, 10 mg, was administered intravenously; muscle relaxation was subsequently maintained with an infusion of vecuronium (Program 2 syringe pump; Becton Dickinson, Lincoln Park, NJ) adjusted to maintain 0–1 twitches in response to supramaximal train-of-four electrical stimulation of the ulnar nerve at the wrist. Nitrous oxide was discontinued after induction, and the trachea intubated.

A Siemens Servo Ventilator 900-C (Siemens Life Support Systems, Schaumburg, IL) was used to control end-tidal P_{CO_2} at $37 \pm 1\text{ mmHg}$. Anesthesia was maintained with isoflurane in air; the end-tidal isoflurane concentration was gradually reduced to 0.8% over $\approx 50\text{ min}$, and then kept at that concentration. End-tidal gas concentrations were measured using a mass spectrometer (Medspect®, St. Louis, Missouri). Airway humidification was provided by placing a Pall Biomedical Products (Glen Cove, NY) heat-and-moisture exchanger between the Y-piece of the circle system and the endotracheal tube.¹²

Forced-air warming was discontinued immediately after induction of anesthesia. A 30-cm-diameter fan was positioned 1.5 m behind and 1.5 m above each volunteer's head and directed along the body axis toward the toes. Cardboard shields were positioned to prevent direct convective cooling of the fingers. Fan speed was increased to "medium" over the next 20 min, but the setting was not subsequently altered during the study period. We know from preliminary studies that this configuration produces an initial cutaneous heat loss of $\approx 2\text{ W/kg}$.

Painful stimulation (similar to that produced by surgery) increases the threshold for vasoconstriction $\approx 0.4^\circ\text{C}$.¹³ Consequently, a 10-s, 100-Hz, 65-mA te-

tanic current was passed through needle electrodes inserted into the skin of the abdomen at 10-min intervals to provide stimulation analogous to surgical pain (Digi Stim III; Neuro Technology, Houston, TX).[†]

Measurements

Core temperatures were measured in the distal fourth of the esophagus using disposable YSI Series 700 thermistor probes (Mallinckrodt Anesthesia Products, St. Louis, MO) connected to a Model 5831 thermometer (Omega, Stamford, CN) with a precision of 0.01° C. Left leg muscle temperatures were recorded using disposable, 8-, 18-, and 38-mm, 21-G, needle thermocouples (Mallinckrodt Anesthesia Products) inserted perpendicular to the skin surface. One needle of each length was inserted several centimeters lateral to the anterior midline of the thigh and calf.

Area-weighted mean skin-surface temperature was computed from measurements at 11 sites by assigning the following regional percentages to each site: head—6%, upper arms—9%, forearms—6%, hands—4.5%, back—19%, chest—9.5%, abdomen—9.5%, anterior thigh—12%, posterior thigh—7%, calves—11.5%, and feet—6%.¹⁴ Muscle and skin-surface temperatures were monitored using an Iso-Thermex (Columbus Instruments International, Columbus, OH) 16-channel electronic thermometer with an accuracy of 0.1° C and a precision of 0.01° C.

Calf-toe, skin-surface temperature gradients were used as an index of leg perfusion. We have previously demonstrated that calf-toe gradients correlate well with forearm-fingertip skin-temperature gradients.¹⁵ Additionally, vasoconstriction on the great toe was quantified using laser Doppler flowmetry (Periflux 3; Perimed, Piscataway, NJ). There is a fair correlation between the laser Doppler flow index and other measures of cutaneous blood flow.¹⁶⁻¹⁹

Absolute left middle fingertip blood flow, resulting primarily from arteriovenous shunt flow,^{20,21} was quantified using venous-occlusion volume plethysmography at 5-min intervals.²² Finger blood flow was also quantified using the Vasomotor Index ([finger-ambient temperature]/[core-ambient temperature]) (VMI). The value of this index varies from 1.0 (fully vasodilated) to 0 (fully constricted). Because ambient tem-

perature is incorporated into the index, it is relatively insensitive to variations in the environment.

Extensive preliminary study indicated that the core temperature plateau occurred when the calf minus toe, skin-temperature gradient increased to 0° C (this gradient probably represents a ≈ twofold reduction in toe blood flow²³ and indicates that thermoregulatory vasoconstriction is beginning). Consequently, a leg gradient of 0° C was considered an elapsed time of zero. Data recorded from each volunteer at 5-min intervals were averaged into 15-min epochs (indexed to elapsed time zero) using a database program; mean values for the entire group of volunteers were then calculated from these individual averages.

Heart rate and oxyhemoglobin saturation (S_pO_2) were monitored continuously using three-lead electrocardiography and a Nellcor N200 pulse oximeter (Hayward, CA). Blood pressure at the ankle was determined oscillometrically at 5-min intervals (Dinamap™ 1846 SX; Critikon, Tampa, FL). Analog and serial data from the laser Doppler, thermometers, and pulse oximeter were recorded at 5-min intervals, using a modification of a previously described data-acquisition system.⁶

Leg Heat Content

Thigh and lower leg heat content was estimated from core temperature, muscle temperatures, and skin-surface temperatures using the formula (see Appendix):

$$Q_{(0 \rightarrow r)} = 2\pi L \rho s r^2 [a_0 + a_2 r^2 / 2], \quad (1)$$

where $Q_{(0 \rightarrow r)}$ (cal) is heat content of the leg segment from the center to radius r (cm), L (cm) is the length of the leg segment (iliac crest to knee, knee to sole of foot), ρ (g/cm³) is tissue density, s (cal · ° C⁻¹ · g⁻¹) is the specific heat of leg tissues, a_0 (° C) is the temperature at the center of the leg segment ($r = 0$), and a_2 (° C/cm²) is a parabolic regression constant. The specific heat of muscle was taken as 0.89 cal · ° C⁻¹ · g⁻¹ and density as 1.06 g/cm³.²⁴ Average leg temperature was calculated similarly:

$$T_{Ave} = a_0 + a_2 r^2 / 2. \quad (2)$$

Average temperature for the entire leg was calculated by weighting the values from each segment in proportion to the estimated mass of that segment.

Statistical Analysis

Changes in values over time were analyzed using repeated-measures ANOVA and Dunnitt's tests; reference values were those obtained at zero elapsed minutes.

[†]From subsequent consultation with a biomedical engineer, we learned that similar stimulation can be provided with greater safety using silver/silver chloride pads positioned >10 cm apart on well prepared skin.

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Linear regression was used to correlate Log[absolute finger blood flow] and the Vasomotor Index. Differences were considered significant when $P < 0.01$. Results are presented as means \pm SD.

Results

The volunteers weighed 62 ± 6 kg, were 170 ± 10 cm tall, and had $19 \pm 9\%$ body fat. Four of the six were male. Estimated masses of the thighs and lower legs were 20 ± 2 kg and 8 ± 1 kg, respectively. Consequently, the legs represented $\approx 45\%$ of our volunteers' total mass.

Peripheral thermoregulatory vasoconstriction was similarly indicated by absolute finger blood flow, finger vasomotor index, toe laser Doppler perfusion index, and the calf minus toe, skin-temperature gradient (fig. 1). There was a good correlation between log[finger blood flow] and the Vasomotor Index: $VMI = 0.46 \cdot \text{Log}[\text{Flow}] + 0.88$, $r^2 = 0.92$ (fig. 2). When the

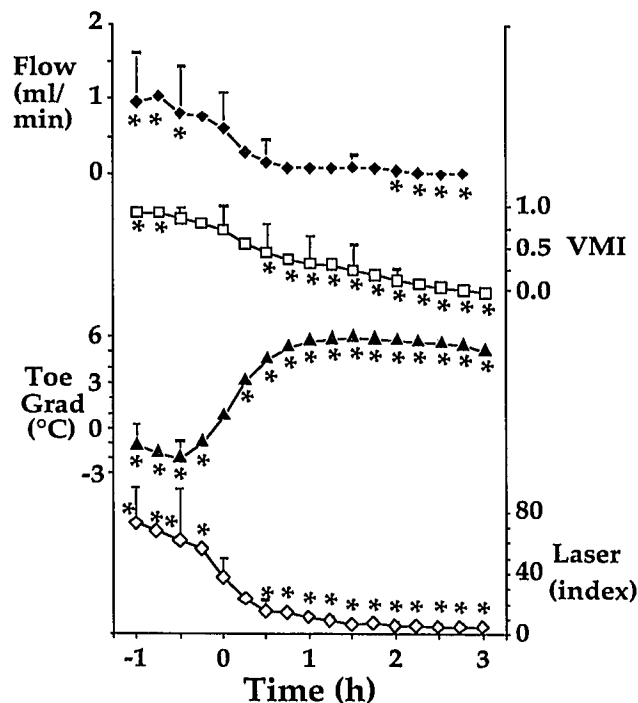


Fig. 1. Peripheral thermoregulatory vasoconstriction was similarly indicated by absolute finger blood flow ("Flow"), finger Vasomotor Index ("VMI"), toe laser Doppler perfusion index ("Laser"), and calf-toe gradient ("Toe Grad"). Elapsed time of zero indicates a calf minus toe, skin-temperature gradient of 0°C . Alternate error bars were omitted for clarity; error bars also were omitted when they overlapped individual data points. Asterisks indicate values significantly different from those obtained at elapsed time zero.

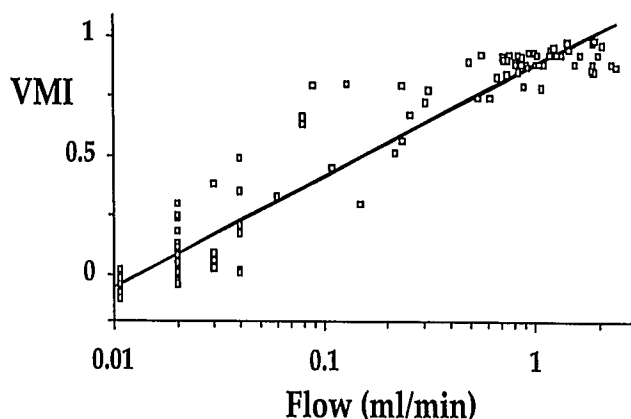


Fig. 2. There was a good correlation between log[finger blood flow] and the Vasomotor Index: $VMI = 0.46 \cdot \text{Log}[\text{Flow}] + 0.88$, $r^2 = 0.92$. When the regression was calculated for each individual, the r^2 ranged from 0.87 to 0.98 (0.93 ± 0.04). Each point represents the values recorded in one volunteer during one acquisition epoch; each volunteer contributed the same number of data points.

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Thermoregulatory vasoconstriction decreased mean skin temperature $2.7 \pm 0.6^\circ\text{C}$ in the period from -1 to $+1$ elapsed hour, but only an additional $1.2 \pm 0.2^\circ\text{C}$ in the subsequent 2 h. Arm and leg temperatures decreased throughout the study; in contrast, head and trunk temperature and heat loss remained relatively constant after vasoconstriction (fig. 3).

Leg tissue temperatures continued to decrease after the core temperature reached a plateau, indicating that body heat content also continued to decrease (fig. 4). Figure 5 shows the radial temperature distribution in the thigh and lower leg (calculated using the parabolic regression coefficients a_0 and a_2). The average r^2 for the thigh and lower leg parabolic temperature regressions were 0.97 ± 0.03 and 0.89 ± 0.06 , respectively.

Changes in core and leg temperatures and heat contents are shown in table 1. During the 3 h after vasoconstriction, average leg temperature decreased $2.0 \pm 0.8^\circ\text{C}$ and leg heat content decreased 49 ± 18 kcal. During the same period, core temperature decreased only $0.4 \pm 0.3^\circ\text{C}$.

Discussion

Most of the complications of perioperative hypothermia are directly related to core temperature. These include prolonged drug action,²⁵ negative postoperative nitrogen balance,²⁶ and impaired coagulation.²⁷ In

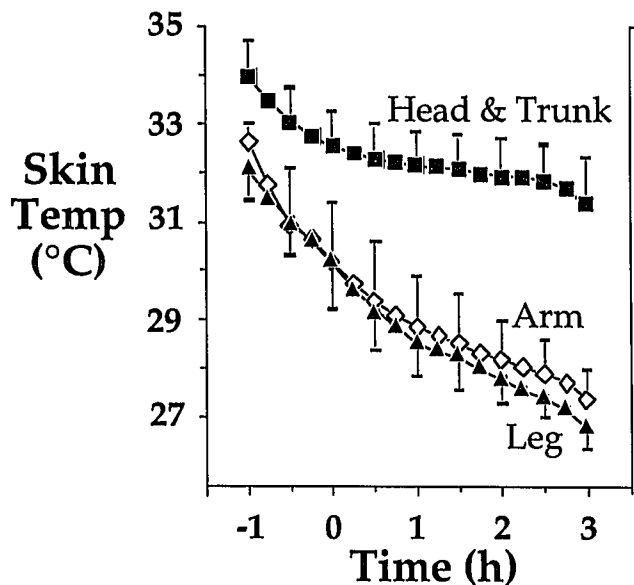


Fig. 3. Arm and leg skin temperatures decreased throughout the study; in contrast, head and trunk skin temperature remained nearly constant after vasoconstriction. Elapsed time of zero indicates a calf minus toe, skin-temperature gradient of 0°C . Alternate error bars were omitted for clarity. All measurements were significantly different from values at the time of vasoconstriction before -0.5 h and after $+0.5\text{ h}$.

the 3 h after thermoregulatory vasoconstriction, core temperature in our volunteers decreased only 0.4°C , although it had decreased a full 1°C in the 1 h before constriction. Vasoconstriction thus appears to be remarkably effective, and offers clinically important protection against further core hypothermia.

The thresholds for thermoregulatory vasoconstriction depend on the administered anesthetic type,^{5,17,28} anesthetic dose,^{15,29} patient age,^{30,31} and a number of other factors.¹³ Once triggered, the effect of vasoconstriction on core temperature in specific individuals will depend on a variety of factors, including cutaneous heat loss, metabolic heat production, body size and shape, body fat content, and intensity of constriction.^{3,32,33} Although our volunteers did not experience evaporative loss from surgical incisions,⁷ their cutaneous loss probably far exceeded that in most patients² because they were uncovered and the skin surface was fanned.^{1,3} In patients with especially large heat loss, vasoconstriction may simply decrease the rate of core cooling, rather than completely prevent further hypothermia. Similarly, simultaneous activation of vasoconstriction and nonshivering thermogenesis may even increase core temperature in hypothermic infants.

The intensity of arteriovenous shunt vasoconstriction

is comparable with, and without, anesthesia.³ Nonetheless, the observed protective effects of vasoconstriction could not easily be predicted from physiologic studies in unanesthetized individuals, because shivering thermogenesis usually would also contribute to heat balance. An additional difficulty in extrapolating from studies without anesthesia is that shivering *per se* substantially increases blood flow to peripheral muscles.³⁴

The small observed core temperature decrease, evenly distributed throughout the body, would reduce leg heat content only 10 kcal. These data thus indicate that the core temperature plateau resulted, at least in part, from altered distribution of heat within the body, not simply decreased cutaneous heat loss or increased metabolic heat production. Specifically, vasoconstriction (which we previously have shown to be, initially, relatively ineffective in preventing heat loss from skin to the environment^{3,6}) apparently decreased loss of metabolic heat from the core thermal compartment to the periphery. Although the site of vasoconstriction was

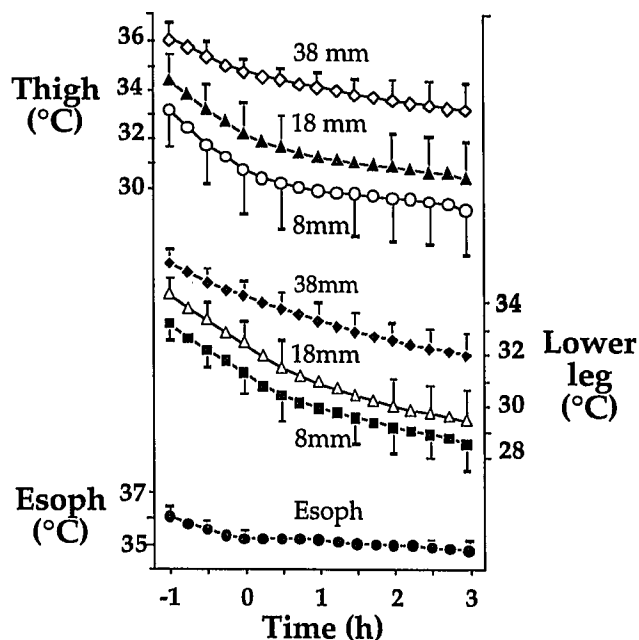


Fig. 4. Thigh and lower leg tissue temperatures continued to decrease after the core temperature reached a plateau, indicating that body heat content also continued to decrease. In contrast, esophageal temperature remained nearly constant after constriction. Elapsed time of zero indicates a calf minus toe, skin-temperature gradient of 0°C . Alternate error bars were omitted for clarity. All muscle temperatures differed significantly from values at the time of vasoconstriction at elapsed times before -0.25 h and after $+0.5\text{ h}$. In contrast, core temperatures (Esoph) differed significantly from values at the time of vasoconstriction before -15 h and after $+1.75\text{ h}$.

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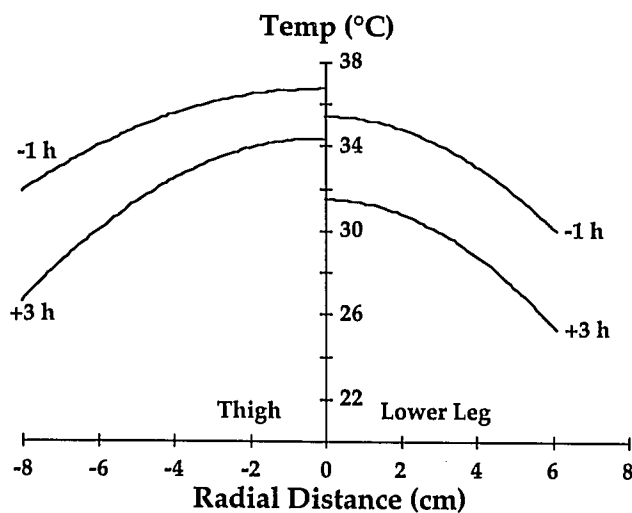


Fig. 5. Radial thigh and lower leg temperatures 1 h before and 3 h after vasoconstriction. Temperatures were estimated using the parabolic regression coefficients calculated from individual leg muscle temperatures (see Equation 3 in Appendix). Average thigh temperature decreased 3.4° C and average lower leg temperature decreased 4.4° C in this period. See figure 4 and tables 1 and 2 for estimates of variance.

peripheral arteriovenous shunts,^{35,36} its effect was to sequester heat to the core. Sequestration of metabolic heat to the core allowed core temperature to remain nearly constant, despite a continually decreasing body heat content.

Continued loss of heat from peripheral tissues eventually decreased leg temperature; in effect, reestablishing the normal core-to-peripheral compartment temperature gradient. Because cutaneous heat loss is roughly proportional to the difference between skin and environmental temperatures, lower leg skin temperature eventually will decrease loss of body heat. Depending on the ambient temperature and efficacy of thermoregulatory vasoconstriction, heat loss may

eventually decrease sufficiently to produce a thermal steady state. In other cases, however, net heat loss will continue until heat content in peripheral thermal buffering tissues is minimal, at which point core temperature will again decrease.

One consequence of a continuing decrease in body heat content during the core temperature plateau is that measured temperature may substantially underestimate the loss of body heat and falsely reassure clinicians. Eventually, the heat deficit will need to be replaced, often by shivering.¹⁹ The intensity of postanesthetic shivering is directly related to intraoperative heat loss.³⁷

Positioning the fan above the volunteer and behind the head facilitated near-simultaneous arm and leg constriction. Preliminary studies (with different cooling regimens) indicated that arm constriction alone was not sufficient to produce a clinically important core temperature plateau. In contrast, leg constriction alone usually was sufficient. This observation is consistent with the far larger mass of the legs and indicates that the legs constitute the bulk of the peripheral thermal compartment.

Because leg constriction was our primary interest in this protocol, we used a calf minus toe, skin-temperature gradient¹⁵ rather than measures of finger flow as we have in previous studies. Using finger flow would have increased the variability to the data, but not substantially altered the results. Similarly, in other studies, we chose a gradient of 4° C because this value identified definitive—and, essentially, complete—vasoconstriction. As might be expected, however, preliminary studies indicated that the core temperature plateau developed when vasoconstriction started. We, therefore, used a skin-temperature gradient of zero to identify initiation of this response. Using a gradient of 4° C as

Table 1. Changes in Temperatures and Heat Contents

	-1 h	+1 h	+2 h	+3 h
ΔT_c (° C)	1.0 ± 0.2	0.0 ± 0.2	-0.2 ± 0.2	-0.4 ± 0.3
ΔT_T (° C)	1.7 ± 0.4	-0.8 ± 0.4	-1.2 ± 0.7	-1.7 ± 1.0
ΔT_{LL} (° C)	1.7 ± 0.5	-1.4 ± 0.4	-2.1 ± 0.4	-2.7 ± 0.5
ΔT_L (° C)	1.7 ± 0.4	-1.0 ± 0.3	-1.5 ± 0.5	-2.0 ± 0.8
ΔQ_T (kcal)	29 ± 6	-14 ± 8	-21 ± 12	-30 ± 16
ΔQ_{LL} (kcal)	13 ± 3	-10 ± 2	-15 ± 3	-19 ± 3
ΔQ_L (kcal)	42 ± 9	-24 ± 9	-36 ± 14	-49 ± 18

Changes in core temperature (ΔT_c), thigh temperature (ΔT_T), lower leg temperature (ΔT_{LL}), whole leg temperature (ΔT_L), thigh heat content (ΔQ_T), lower leg heat content (ΔQ_{LL}), and whole leg heat content (ΔQ_L). Initial (-1 elapsed h) core temperature was 36.2 ± 0.3° C, initial thigh temperature was 34.4 ± 1.0° C, initial lower leg temperature was 34.0 ± 0.7° C, and initial whole leg temperature was 34.3 ± 0.9° C. Elapsed time of zero indicates a calf minus toe, skin-temperature gradient of 0° C. Values at all times differed significantly from those at elapsed time zero.

our criterion would have shifted the curves in figures 1, 3, and 4 slightly to the left, without significantly changing their shapes or the conclusion of the study.

Our calculation of leg heat content assumes radial symmetry: To minimize risk to our volunteers, we did not verify this assumption by inserting more than three needles in each leg segment. It remains likely that tissue heat content in the posterior third of the leg decreased somewhat less than estimated because heat loss from this region was minimized by the foam insulation covering the operating table. Nonetheless, given the cylindrical shape of the leg and the central position of its major blood vessels, tissue temperatures are likely to be roughly symmetrical. An assumption of angular symmetry is used in nearly all models of heat balance, including some that have been extensively validated.³⁸

Our calculations make the further assumption that each leg segment is cylindrical. Although not strictly accurate, a cylinder is a reasonable approximation of leg shape and simplifies analysis. Once again, most heat balance models make this assumption and still produce experimentally verifiable results.³⁸ And, finally, we had no way to independently determine the specific heat of leg tissues in our volunteers. Although the average specific heat of human tissues is thought to be $0.83 \text{ cal} \cdot ^\circ\text{C}^{-1} \cdot \text{g}^{-1}$,³⁹ the specific heat of muscle, $0.89 \text{ cal} \cdot ^\circ\text{C}^{-1} \cdot \text{g}^{-1}$, is somewhat higher.²⁴ Because leg tissue is mostly muscle, we used the higher value to calculate leg heat content. More importantly, legs do not have important radial differences in tissue type, indicating that this value can be extrapolated to all radii.

We did not measure arm tissue temperatures, but it is likely that vasoconstriction markedly reduces arm heat content. Thus, even large errors in estimating leg heat content (which are unlikely) would not change our conclusion that body heat content continued to decrease substantially, despite a nearly constant core temperature. A limitation of our study is that we did not quantify heat balance by measuring heat loss and metabolic heat production. Nonetheless, the large observed decrease in leg heat content during the core temperature plateau indicates that thermoregulatory vasoconstriction significantly alters distribution of body heat.

In summary, these data indicate that thermoregulatory vasoconstriction is remarkably effective in preventing further core hypothermia. Vasoconstriction apparently decreases loss of metabolic heat from the core thermal compartment to the periphery. This sequestration of metabolic heat allows core temperature to remain

nearly constant, despite a continually decreasing body heat content. The initial core temperature plateau thus results, at least in part, from altered distribution of heat within the body. In effect, vasoconstriction during anesthesia restores the normal, preinduction core-to-peripheral tissue temperature gradient.

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References

1. Sessler DI, McGuire J, Moayeri A, Hynson J: Isoflurane-induced vasodilation minimally increases cutaneous heat loss. *ANESTHESIOLOGY* 74:226–232, 1991
2. Hynson J, Sessler DI: Intraoperative warming therapies: A comparison of three devices. *J Clin Anesth* 4:194–199, 1992
3. Sessler DI, Hynson J, McGuire J, Moayeri A, Heier T: Thermoregulatory vasoconstriction during isoflurane anesthesia minimally decreases heat loss. *ANESTHESIOLOGY* 76:670–675, 1992
4. Sessler DI, Rubinstein EH, Eger II EI: Core temperature changes during N_2O /fentanyl and halothane/ O_2 anesthesia. *ANESTHESIOLOGY* 67:137–139, 1987
5. Sessler DI, Olofsson CI, Rubinstein EH, Beebe JJ: The thermoregulatory threshold in humans during halothane anesthesia. *ANESTHESIOLOGY* 68:836–842, 1988
6. Sessler DI, Moayeri A, Støen R, Glosten B, Hynson J, McGuire J: Thermoregulatory vasoconstriction decreases cutaneous heat loss. *ANESTHESIOLOGY* 73:656–660, 1990
7. Roe CF: Effect of bowel exposure on body temperature during surgical operations. *Am J Surg* 122:13–15, 1971
8. Hynson JM, Sessler DI, Moayeri A, McGuire J: Absence of non-shivering thermogenesis in anesthetized adults (abstract). *Anesth Analg* 72:S119, 1991
9. Conway JM, Norris KH, Bodwell CE: A new approach for the estimation of body composition: Infrared interactance. *Am J Clin Nutr* 40:1123–1130, 1984
10. Moayeri A, Hynson JM, Sessler DI, McGuire J: Pre-induction skin-surface warming prevents redistribution hypothermia (abstract). *ANESTHESIOLOGY* 75:A1004, 1991
11. Sessler DI, Moayeri A: Skin-surface warming: Heat flux and central temperature. *ANESTHESIOLOGY* 73:218–224, 1990
12. Bickler P, Sessler DI: Efficiency of airway heat and moisture exchangers in anesthetized humans. *Anesth Analg* 71:415–418, 1990
13. Washington DE, Sessler DI, McGuire J, Hynson J, Schroeder M, Moayeri A: Painful stimulation minimally increases the thermoregulatory threshold for vasoconstriction during enflurane anesthesia in humans. *ANESTHESIOLOGY* 77:286–290, 1992
14. DuBois D, DuBois EF: A formula to estimate the approximate surface area if height and weight be known. *Arch Intern Med* 17: 863–871, 1916
15. Støen R, Sessler DI: The thermoregulatory threshold is inversely proportional to isoflurane concentration. *ANESTHESIOLOGY* 72:822–827, 1990

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16. Holloway Jr GA, Watkins DW: Laser Doppler measurement of cutaneous blood flow. *J Invest Dermatol* 69:306-309, 1977
17. Sessler DI, Olofsson CI, Rubinstein EH: The thermoregulatory threshold in humans during nitrous oxide-fentanyl anesthesia. *ANESTHESIOLOGY* 69:357-364, 1988
18. Fagrell B: Dynamics of skin microcirculation in humans. *J Cardiovasc Pharmacol* 7:S53-S58, 1985
19. Sessler DI, Rubinstein EH, Moayeri A: Physiological responses to mild perianesthetic hypothermia in humans. *ANESTHESIOLOGY* 75:594-610, 1991
20. Coffman JD: Total and nutritional blood flow in the finger. *Clin Sci* 42:243-250, 1972
21. Coffman JD, Cohen AS: Total and capillary fingertip blood flow in Raynaud's phenomenon. *N Engl J Med* 285:259-263, 1971
22. Burch GE, Cohn AE, Neumann C: A study by quantitative methods of the spontaneous variations in volume of the finger tip, toe tip, and postero-superior portion of the pinna of resting normal white adults. *Am J Physiol* 136:433-447, 1942
23. Rubinstein EH, Sessler DI: Skin-surface temperature gradients correlate with fingertip blood flow in humans. *ANESTHESIOLOGY* 73:541-545, 1990
24. Cooper TE, Trezek GJ: Correlation of thermal properties of some human tissue with water content. *Aerospace Med* 42:24-27, 1971
25. Heier T, Caldwell JE, Sessler DI, Miller RD: Mild intraoperative hypothermia increases duration of action and spontaneous recovery of vecuronium blockade during nitrous oxide-isoflurane anesthesia in humans. *ANESTHESIOLOGY* 74:815-819, 1991
26. Carli F, Emery PW, Freemantle CAJ: Effect of preoperative normothermia on postoperative protein metabolism in elderly patients undergoing hip arthroplasty. *Br J Anaesth* 63:276-282, 1989
27. Valeri RC, Cassidy G, Khuri S, Feingold H, Ragno G, Altschule MD: Hypothermia-induced reversible platelet dysfunction. *Ann Surg* 205:175-181, 1987
28. Hynson JM, Sessler DI, Belani K, Washington D, McGuire J, Merrifield B, Schroeder M, Moayeri A, Crankshaw D, Hudson S: Thermoregulatory vasoconstriction during propofol/nitrous oxide anesthesia in humans: Threshold and Sp_{O_2} . *Anesth Analg* 75:947-952, 1992
29. Washington D, Sessler DI, Moayeri A, Merrifield B, Prager M, McGuire J, Belani K, Hudson S, Schroeder M: Thermoregulatory responses to hyperthermia during isoflurane anesthesia in humans. *J Appl Physiol* 74:82-87, 1993
30. Tankersley CG, Smolander J, Kenney WL, Fortney SM: Sweating and skin blood flow during exercise: Effects of age and maximal oxygen uptake. *J Appl Physiol* 71:236-242, 1991
31. Wagner JA, Robinson S, Marino RP: Age and temperature regulation of humans in neutral and cold environments. *J Appl Physiol* 37:562-565, 1974
32. Frim J, Livingstone SD, Reed LD, Nolan RW, Limmer RE: Body composition and skin temperature variation. *J Appl Physiol* 68:540-543, 1990
33. LeBlanc J: Subcutaneous fat and skin temperature. *Can J Biochem Physiol* 32:354-358, 1954

**Parabolic regression constants were determined by reflecting temperatures around the radius and fitting the values to a second-order polynomial least-squares regression. Using this technique, the coefficient of the linear term is zero, leaving a parabolic equation.

34. Johnson JM, Rowell LB: Forearm skin and muscle vascular responses to prolonged leg exercise in man. *J Appl Physiol* 6:6, 1975
35. Hales JRS: Skin arteriovenous anastomoses, their control and role in thermoregulation, Cardiovascular Shunts: Phylogenetic, Ontogenetic and Clinical Aspects. Edited by Johansen K, Burggren W. Copenhagen, Munksgaard, 1985, pp 433-451
36. Rowell LB: Cardiovascular aspects of human thermoregulation. *Circ Res* 52:367-379, 1983
37. Just B, Delva E, Camus Y, Lienhart A: Oxygen uptake during recovery following naloxone. *ANESTHESIOLOGY* 76:60-64, 1992
38. Wissler EH: Comparison of computed results obtained from two mathematical models—a simple 14-node model and a complex 250-node model. *J Physiol* 63:455-458, 1971
39. Burton AC: Human calorimetry: The average temperature of the tissues of the body. *J Nutr* 9:261-280, 1935

Appendix: Leg Heat Content

Tissue temperature as a function of mean radial distance from the center of the leg segment was calculated using skin-surface temperature and the muscle temperatures (8, 18, and 38 mm below the surface) 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 was estimated from the regression equation with no similar assumption. This regression assumes that tissue temperature is radially symmetrical. Results of the parabolic regression were expressed by the equation

$$T(r) = a_0 + a_2 r^2, \quad (3)$$

where $T(r)$ is the temperature in °C at radius r (cm), a_0 (°C) is the temperature at the center of the leg segment, and a_2 (°C/cm²) is a regression constant.

From the radial tissue temperature distribution, heat content was calculated using the assumptions that specific heat and density of tissues were similar at all points and that leg segment volume could be approximated by a cylinder the length of the segment with an average radius equaling the mean of the radii at the top, midpoint, and bottom of the segment:

$$Q = 2\pi\rho sL \int_0^r [a_0 + a_2 r^2] r dr, \quad (4)$$

where Q (cal) is heat content of the leg segment, L (cm) is the length of the leg segment, s (cal · °C⁻¹ · g⁻¹) is the specific heat of leg tissues, and ρ (g/cm³) is tissue density.

This equation was integrated and a factor of two added to account for the volunteers' other leg.

$$Q_{(0 \rightarrow r)} = 2\pi L \rho s r^2 [a_0 + a_2 r^2 / 2], \quad (5)$$

where $Q_{(0 \rightarrow r)}$ (cal) is heat content of the leg segment from the center to radius r . The specific heat of muscle was taken as 0.89 cal · °C⁻¹ · g⁻¹ and density as 1.06 g/cm³.

Average temperature of the leg segments, T_{Ave} , was determined by dividing Equation 5 by the volume of the segment and its specific heat and density, and deleting the factor of two (added to the heat equation to account for the second leg).

$$T_{Ave} = a_0 + a_2 r^2 / 2. \quad (6)$$