Heat Flow and Distribution during Induction of General Anesthesia

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Abstract

Background: Core hypothermia after induction of general anesthesia results from an internal core-to-peripheral redistribution of body heat and a net loss of heat to the environment. However, the relative contributions of each mechanism remain unknown. The authors evaluated regional body heat content and the extent to which core hypothermia after induction of anesthesia resulted from altered heat balance and internal heat redistribution.

Cited Here...: Six minimally clothed male volunteers in an [nearly equal] 22 degrees Celsius environment were evaluated for 2.5 control hours before induction of general anesthesia and for 3 subsequent hours. 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 needle thermocouples, 10 skin temperatures, and "deep" foot temperature. To separate the effects of redistribution and net heat loss, we multiplied the change in overall heat balance by body weight and the specific heat of humans. The resulting change in mean body temperature was subtracted from the change in distal esophageal (core) temperature, leaving the core hypothermia specifically resulting from redistribution.

Cited Here...: Core temperature was nearly constant during the control period but decreased 1.6 plus/minus 0.3 degrees Celsius in the first hour of anesthesia. Redistribution contributed 81% to this initial decrease and required transfer of 46 kcal from the trunk to the extremities. During the subsequent 2 h of anesthesia, core temperature decreased an additional 1.1 plus/minus 0.3 degrees Celsius, with redistribution contributing only 43%. Thus, only 17 kcal was redistributed during the second and third hours of anesthesia. Redistribution therefore contributed 65% to the entire 2.8 plus/minus 0.5 degrees Celsius decrease in core temperature during the 3 h of anesthesia. Proximal extremity heat content decreased slightly after induction of anesthesia, but distal heat content increased markedly. The distal extremities thus contributed most to core cooling. Although the arms constituted only a fifth of extremity mass, redistribution increased arm heat content nearly as much as leg heat content. Distal extremity heat content increased [nearly equal] 40 kcal during the first hour of anesthesia and remained elevated for the duration of the study.

Conclusions: The arms and legs are both important components of the peripheral thermal compartment, but distal segments contribute most. Core hypothermia during the first hour after induction resulted largely from redistribution of body heat, and redistribution remained the major cause even after 3 h of anesthesia.

UNANESTHETIZED subjects do not become hypothermic when exposed to a typical cool operating room environment because thermoregulatory vasoconstriction or shivering maintains core temperature. [17] In contrast, core hypothermia develops rapidly in the hour immediately after induction of general anesthesia. [2,3].

Cooling produced by ventilation with dry, cold gases. [4] and surgical skin preparation [5] usually contributes little to the observed hypothermia. Anesthesia only slightly increases cutaneous heat loss [2] but does reduce metabolic heat production. [5,6] The resulting imbalance between heat production and loss decreases body heat content. However, the decrease appears insufficient to explain the observed initial decrease in core temperature, suggesting that redistribution of heat from core to peripheral tissues is a major cause of core hypothermia. [2,7].

The extent to which internal redistribution of body heat can contribute to core cooling after induction of anesthesia depends on the capacity of the peripheral thermal compartment. During transition from aggressive cooling to vigorous warming, the heat capacity of the peripheral thermal compartment approaches 150 kcal. [8]. However, it is unlikely that thermoregulatory vasoconstriction can maintain such an extreme steady-state core-to-peripheral temperature gradient at typical operating room temperatures. The effective heat capacity of peripheral thermal buffering tissues--and consequent importance of redistribution hypothermia--is thus presumably considerably less in the perioperative period than under extreme circumstances. However, its magnitude in perioperative circumstances remains unknown. That is, the relative contributions of changes in heat loss, heat production, and internal redistribution of body heat remain unknown. Also unknown is the physical distribution of heat within "peripheral" tissues, and how induction of general anesthesia alters this distribution.

Changes in distribution of body heat are most likely to significantly alter core temperature at two points during anesthesia. The first is during induction, when anesthetic-induced vasodilation facilitates core-to-peripheral redistribution of body heat. [2] The second is during the "plateau" phase when reemergence of thermoregulatory vasoconstriction [9-11] reestablishes the normal core-to-peripheral tissue temperature gradient. [12] We have reported changes in leg heat content during the plateau phase. [12] Accordingly, the purpose of this study was to evaluate regional body heat content and the fractional contributions of altered heat balance and internal heat redistribution to observed changes in core temperature before and after induction of general anesthesia.

Methods

With approval from the Committee on Human Research, we studied six 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 January or February 1994.

The volunteers' height was 179 plus/minus 6 cm (mean plus/minus SD), weight 82 plus/minus 13 kg. and age 28 plus/minus 5 yr. The percentage of body fat was 21 plus/minus 5, as determined using infrared interactance [13] (Futrex 1000, Futrex. Hagerstown, MD). Ambient temperature was maintained at 21.9 plus/minus 0.6 degrees Celsius and ambient relative humidity at 26 plus/minus 8% during the study period (Model HX93 humidity and temperature transmitter. 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. Lactated Ringer's solution warmed to 37 degrees Celsius was initially infused at [nearly equal] 100 ml/h. Throughout the study, minimally clothed volunteers reclined on an operating room table set in chaise-lounge position.

Application of monitoring equipment (see below) was followed by a 2.5-h control period (-2.5 to 0 elapsed hours). Most volunteers remained fully exposed to a typical operating room environment during this time; however, several started to shiver shortly before induction of anesthesia. Those volunteers were covered with a single unwarmed cotton blanket, which was was left in place throughout the study. The others remained uncovered throughout.

Anesthesia was induced without premedication, at elapsed time 0, by infusion of propofol ([nearly equal] 3 mg/kg) and fentanyl ([nearly equal] 4 micro gram/kg). Vecuronium (10 mg) was administered intravenously, and the trachea was intubated. A bolus of warmed lactated Ringer's solution (10 ml/kg) was administered during induction of anesthesia; subsequently, warmed fluid was administered again at a rate of [nearly equal] 100 ml/h.

After induction of anesthesia, ventilation was controlled by an Ohmeda ventilator incorporated into a Modulus CD integrated anesthesia machine (Ohmeda, Madison, WI). The system was modified so that respiratory gases were not rebreathed. The volunteers' lungs were ventilated with air at a rate and volume sufficient to maintain end-tidal P_{CO_2} near 35 mmHg. Airway humidification was provided by placing a Pall Biomedical Products (Glen Cove, NY) heat- and moisture-exchanging filter between the Y-piece of the circle system and the endotracheal tube. [4].

Anesthesia subsequently was maintained by infusion of fentanyl (2 micro gram

symbol kg sup -1 *symbol* h sup -1) and propofol. Propofol was administered by a computer-controlled infusion pump (Ohmeda 9000, Ohmeda, Steeton, England) to a target blood concentration of 2-4 micro gram/ml. In some volunteers, anesthesia was augmented by [nearly equal] 0.5% end-tidal isoflurane to prevent thermoregulatory vasoconstriction. Muscle relaxation was 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.

After 3 h, general anesthesia was discontinued, neuromuscular blockade antagonized, and the patients' tracheas extubated. Study measurements ceased at that point, and the volunteers were rewarmed with forced-air (Bair Hugger, Augustine Medical, Eden Prairie, MN). [15].

Monitoring

Core temperature was measured in the distal esophagus, with a probe positioned according to the formula of Mekjavic and Rempel. [16] Temperature also was recorded from the tympanic membrane using Mon-a-Therm thermocouples (Mallinckrodt, St. Louis, MO). Visual inspection with an otoscope confirmed that the ear canal was free of wax in each volunteer. The aural probe was inserted by volunteers until they felt the thermocouple touch the tympanic membrane; appropriate placement was confirmed when volunteers easily detected a gentle rubbing of the attached wire. The aural canal was occluded with cotton, the probe securely taped in place, and a gauze bandage positioned over the external ear. Trunk and head skin temperatures were calculated by assigned the following regional percentages: head 20%, chest 20%, abdomen 20%, and back 40%. [17] Skin-surface temperatures were recorded from thermocouples incorporated into thermal flux transducers (see below).

The length of the thigh (anterior iliac crest to midpatella) and lower leg (midpatella to ankle) were measured in centimeters. Circumference was measured at the mid-upper thigh (one-quarter of the distance from the anterior iliac crest to the patella), mid-lower thigh (three-quarters of the distance from the anterior iliac crest to the patella), mid-upper calf (one-quarter of the distance from the patella to the ankle), and midlower calf (three-quarters of the distance from the patella to the ankle). At four sites, right leg muscle temperatures were recorded using disposable, 8-, 18-, and 38-mm-long, 21-G needle thermocouples (Mallinckrodt) inserted perpendicular to the skin surface. After intradermal injection of [nearly equal] 0.1 ml 1% lidocaine, one needle of each length was inserted several centimeters lateral to the anterior midline of the mid-upper and mid-lower thigh. Needles were inserted similarly into the mid-upper calf and mid-lower calf. In each case, needles were inserted at the same place in which leg segment circumference was measured. Skin-surface temperatures were recorded immediately adjacent to each set of needles and directly posterior to each set.

The lengths of the right arm (axilla to elbow) and forearm (elbow to wrist) were measured in centimeters. The circumference was measured at the midpoint of each segment. As in the right leg, 8-, 18-, and 38- mm-long needle thermocouples were inserted into the upper and forearms at the same place in which arm segment circumference was measured. Skin-surface temperatures were recorded immediately adjacent to each set of needles. Additionally, adductor pollicis temperature was measured using a 22-G, 8-mm-long needle thermocouple placed directly into the muscle, 1 cm proximal to the metacarpophalangeal joint of the thumb.

Core, skin-surface, and muscle temperatures were recorded from thermocouples connected to two calibrated Iso-Thermex 16-channel electronic thermometers having an accuracy of 0.1 degrees Celsius and a precision of 0.01 degree Celsius (Columbus Instruments, Columbus, OH). Individual temperatures and appropriate averages were displayed at 1-s intervals. Additional temperatures were recorded from Mon-a-Therm 6510 two-channel thermometers having an accuracy near 0.1 degree Celsius (Mallinckrodt).

Subcutaneous temperature was measured on the ball of the foot and on the back of the hand near the fifth finger using a Coretemp (Terumo, Tokyo, Japan) "deep tissue" thermometer. This thermometer uses an active heating element to null cutaneous heat flux, a system originally described by Fox et al. [18] and subsequently refined by Kobayashi et al. [19] When cutaneous heat flux is zero, the Second Law of Thermodynamics requires equal temperatures on each side of the skin. Temperature of the thermometer thus equals cutaneous (and subcutaneous) temperature. In practice, the device records tissue temperature to [nearly equal] 1 cm below the skin surface.

Oxygen consumption before induction of anesthesia was measured using a canopy-based metabolic monitor (Deltatrac, SensorMedics, Yorba Linda, CA). The system was calibrated daily using a known mixture of gases and additionally calibrated numerous times by burning ethanol. Measurements were averaged over 1-min intervals and recorded every 5 min. After induction of anesthesia, the system was switched into ventilator mode, and recordings were continued for the duration of the study. When isoflurane was required near the end of several studies, it was added gradually to minimize artifact in the oxygen consumption measurements. [20].

Heat flux from 15 skin-surface sites was measured in watts/meter squared using thermal flux transducers (Concept Engineering, Old Saybrook, CT). Flux values for each subject were converted into watts/site by multiplying by the calculated body surface area [17] of each volunteer and assigning the same regional percentages used for calculating mean skin temperature. Contact between the flux transducers and skin was facilitated by application of a thin layer of Type 120 Thermal Joint Compound (EG&G Wakefield Engineering, Wakefield, MA) between the two surfaces. Approximately 20 cm of lead

wire to each transducer were taped to the skin to prevent artifactual cooling of the flux monitors by conduction through the wire.

Measured cutaneous heat loss was augmented by 10% to account for insensible transcutaneous evaporative loss [21] and 3% to compensate for the skin surface covered by the volunteers' shorts. We further augmented cutaneous loss by 5% of the metabolic rate (as determined from oxygen consumption) to account for respiratory loss. Respiratory loss is roughly comparable in unanesthetized subjects breathing air with a relative humidity of [nearly equal] 30% and in subjects breathing dry gas via an endotracheal tube but protected by a heat- and moisture-exchanging filter. [4].

Thermal flux transducers measure heat loss via radiation, conduction, and convection. The specified accuracy of our transducers is plus/minus 5%. Although flux transducers can underestimate flux by up to 25% when skin is immersed in water and dilated, they remain accurate with constricted or dilated skin in air. [22] Flux estimated by these transducers has been shown to correlate reasonably well with data from a water-perfused suit calorimeter. [23] One W = 1 joule/s = 0.86 kcal/h; the overall specific heat of humans is [nearly equal] 0.83 kcal *symbol* kg^{1O} C sup -1, [21] but the specific heat of muscle is 0.89 kcal *symbol* kg^{1O} C sup -1. [25] We defined flux as positive when heat traversed skin to the environment.

Forearm minus fingertip skin-surface temperature gradients were used as an index of hand arteriovenous shunt perfusion. [26] As in previous studies, [12] we considered a gradient exceeding 4 degrees Celsius to indicate vasoconstriction, and a gradient less than 0 degree Celsius to indicate vasodilation. Left forearm blood flow was quantified using strain-gauge plethysmography. [27] Instead of using a mercury-in-rubber gauge, we used a capacitance-based "extensometer." [28] Plethysmography often is used to measure cutaneous capillary blood flow, in which case arteriovenous shunts in the hand or foot are isolated by an arterial tourniquet. [29] In this study, however, we avoided a distal arterial tourniquet because we were interested in total extremity blood flow. Capillary vasodilation was estimated using laser Doppler flowmetry (Periflux 3, Perimed, Piscataway, NJ) with an integrating multiprobe ("wide-band" setting) positioned on the right lateral forearm. [30,31]

Calf minus toe skin-surface temperature gradients were used as an index of foot arteriovenous shunt perfusion. [9] As in previous studies, [12] we considered a gradient exceeding 4 degrees Celsius to indicate vasoconstriction and a gradient less than 0 degree Celsius to indicate vasodilation. Vasodilation in leg capillaries was estimated using laser Doppler flowmetry with a standard fiberoptic probe (narrow-band setting) positioned on the right lateral calf. Vascular tone also was evaluated on the second toe using the perfusion index, which is derived using the same principle as in pulse

oximeters, from absorption of two different infrared wave lengths. The index is calculated from the combined absorption of the two intensities. [32].

Heart rate was monitored continuously using three-lead electrocardiography. Oxyhemoglobin saturation (Sp_{O_2}) was measured continuously using pulse oximetry, and blood pressure was determined oscillometrically at 5-min intervals at the left ankle using the Modulus CD Anesthesia System. End-tidal gas concentrations were measured using a Rascal monitor (Ohmeda, Salt Lake City, UT); gas sampled by this monitor was returned to the Deltatrac oxygen consumption monitor. Analog and serial thermoregulatory data were recorded at 5-min intervals, using a modification of a previously described data-acquisition system. [33] Anesthetic data were recorded using IdaCare, version 1.3 (Premier Anesthesia Systems, Atlanta, GA), which is Macintosh (Apple computer, Cupertino, CA)-based patient information management software. Both systems operated asynchronously on a Macintosh FX computer.

Tissue Temperature and Heat Content

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 temperatures, and the muscle temperatures (8, 18, and 38 mm below the surface) using parabolic regression. parallel 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. This regression assumes segmental tissue temperature is radially symmetrical. Results of the parabolic regression were expressed by the equation Equation 1 where T(r) is the temperature in degrees Celsius at radius r (cm), a_0 (degrees Celsius) is the temperature at the center of the leg segment, and a_2 (degrees Celsius/cm^2) is a regression constant. Average temperature of the leg segments, T_{Ave} was determined by integrating Equation 1 from zero to r: Equation 2.

$$T(r) = a_0 + a_2 r^2,$$
Equation 1

$$T_{Ave} = a_0 + \frac{a_2 r^2}{2}.$$
Equation 2

Limb heat content was estimated from core temperature, muscle temperatures, and skin-surface temperatures using the formula: Equation 3 where Q sub (0 --> r) (cal) is heat content of the leg segment from the center to radius r, L (cm) is the length of the

leg segment (i.e., iliac crest to midthigh, midcalf to ankle), p (g/cm^3) is tissue density, and s (cal *symbol* degrees Celsius^1 *symbol* g sup -1) is the specific heat of leg tissues. The specific heat of muscle was taken as 0.89 cal *symbol* degrees Celsius sup -1 *symbol* g sup -1 and density as 1.06 g/cm^3. [25] A factor of 2 has been added to the integration to account for heat in the contralateral leg. We have described the derivation of these formulas and their limitations. [12].

$$Q_{(0 \to r)} = 2(\pi r^2 L)\rho s\left[a_0 + \frac{a_2 r^2}{2}\right],$$

Equation 3

As in previous studies, [34] we did not measure posterior leg tissue temperatures. Rather than assume full radial symmetry, we assumed only that radial temperature distribution in the posterior leg segments also would be parabolic. Accordingly, we calculated the regression constant a_2 in the posterior leg segments from Equation 1, using a_0 determined from the adjacent anterior segment and the posterior segment skin temperature. Average posterior segment tissue temperature and heat content then was determined by inserting these values into Equation 2 and Equation 3.

Foot volume was determined in each volunteer by water displacement. "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 legs were treated comparably throughout this study, thus 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. Hand volume was determined in each volunteer by water displacement. 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.

In our previous studies. [12,34] limb tissue temperatures usually have been easy to fit using parabolic regression. However, fits are occasionally suboptimal during periods of rapid change (e.g., sudden vasodilation, inception of cutaneous warming). We thus also considered a fourth-order regression, which better matches "uneven" data, particularly data obtained immediately after induction of anesthesia when cutaneous temperatures

sometimes exceeded that measured 8 mm below the skin surface. Specifically, we used the following regression equation: Equation 4 where a, (degrees Celsius/cm^4) is the coefficient of the fourth-order term.

$$T(r) = a_0 + a_2 r^2 + a_4 r^4,$$
Equation 4

Segment heat content was then estimated by integrating Equation 4 and adding a factor of 2 to account for the contralateral limb: Equation 5.

$$Q_{(0 \to r)} = 2(\pi r^2 L) \rho s \left[a_0 + \frac{a_2 r^2}{2} + \frac{a_4 r^4}{3} \right].$$
Equation 5

Trunk Heat Distribution

Preliminary data showed that core and trunk skin temperatures changed synchronously after induction of anesthesia, indicating that there were not major inhomogeneities in trunk tissue temperatures. Accordingly, changes in trunk 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 subracting the calculated weight of the extremities (from the radial integration) from the total weight of each subject.

Statistical Analysis

Overall changes in body heat were calculated as the time integral of metabolic heat production minus cutaneous heat loss. Cutaneous heat flux (in watts) was integrated over 30-min intervals and converted to kilocalories/hour. Similarly, oxygen consumption (ml/min) was converted to equivalent metabolic heat production assuming the caloric value of oxygen to be 4.82 kcal/L (respiratory quotient 0.82). [35] 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; use of a standard value thus introduces minimal error in the calculation of metabolic heat production. [35].

Two factors contributed independently to core hypothermia after induction of anesthesia: decreased overall heat balance and internal redistribution of body heat. To separate the effects, we multiplied the change in overall heat balance by body weight and the specific heat of humans (0.83 kcal *symbol* kg sup -1 degrees Celsius sup -1). [24] The resulting change in mean body temperature was subtracted from the change in core temperature, leaving the core hypothermia specifically resulting from redistribution. The amount of heat redistributed from the trunk to the extremities was

calculated by multiplying the decrease in core temperature attributed to redistribution by the weight of the trunk and the specific heat of human tissue.

Time-dependent changes were evaluated using repeated-measure ANOVA; values were compared with those recorded at time 0 (induction of anesthesia) with Dunnett's test. Results are expressed as mean plus/minus SD: differences were considered statistically significant when P < 0.01.

Results

Estimated masses of the thighs and lower legs (including feet) were 21 plus/minus 2 kg and 10 plus/minus 1 kg, respectively. Consequently, the legs represented [nearly equal] 38% of our volunteers' total mass. Similarly, estimated masses of the arms and forearms (including hands) were 4 plus/minus 1 kg and 4 plus/minus 1 kg, respectively. Consequently, the arms represented [nearly equal] 10% of our volunteers' total mass.

In all volunteers, vasoconstriction was present throughout the control period (forearm minus fingertip and calf minus toe skin-temperature gradients > 4 degrees Celsius). Vasodilation occurred after induction of anesthesia, and the volunteers remained vasodilated for the remainder of the study (gradients < 0 degree Celsius). Forearm blood flow increased significantly from 1.8 plus/minus 0.8 to 7.8 plus/minus 4.4 ml *symbol* min_1 *symbol* 100 g sup -1. Vasodilation was most dramatic on the leg, with the toe perfusion index increasing from 0.2 plus/minus to 0.2 1.5 plus/minus 0.7 U and capillary flow on the calf, as evaluated using laser Doppler flowmetry, increasing from 0.2 plus/minus 0.6 to 2.3 plus/minus 2.3 U (Table 1).

	Control Period	General Anesthesia
Forearm–fingertip gradient (°C)	6.3 ± 1.0	−1.9 ± 0.7
Extensometer/arm (ml·min⁻¹·100 g⁻¹)	1.8 ± 0.8	7.8 ± 4.4
Laser Doppler/forearm (units)	5 ± 2.7	8.3 ± 2.4
Calf–toe gradient (°C)	7.6 ± 1.6	−0.9 ± 1.8
Laser Doppler/calf (units)	0.2 ± 0.6	2.3 ± 2.3
Perfusion index/toe (units)	0.2 ± 0.2	1.5 ± 0.7

Blood flow at all measurement sites increased significantly after induction of general anesthesia. Absolute laser Doppler values on the calf and forearm should not be compared because of differences in the probes used and instrument settings.

Table 1

The respiratory quotient was 0.8 plus/minus 0.1 before induction of anesthesia and did not subsequently change significantly. Cutaneous heat loss was 74 plus/minus 13 kcal/h during the initial portion of the study. Loss increased only slightly after induction of anesthesia, and the increase was not sustained. Metabolic heat production, which was nearly constant at 71 plus/minus 14 kcal/h before induction of anesthesia, subsequently decreased 33 plus/minus 8%. As a result, overall heat balance (production minus loss) was near 0 (thermal steady state) before induction of anesthesia, but heat content subsequently decreased linearly at [nearly equal] 31 kcal/h. Changes in body heat content were similar when calculated as the sum of measured changes in the arms and legs and the change in core temperature multiplied by the weight of the core and the specific heat of human tissues (Figure 1).
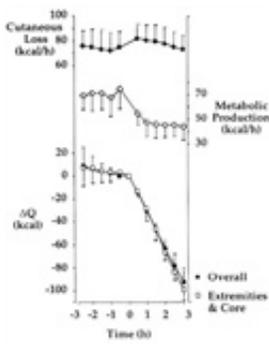
Figure 1

One volunteer could not swallow the esophageal probe. His core temperature thus was measured at the tympanic membrane. As in our previous studies, [9] tympanic membrane and esophageal temperatures were virtually identical. Trunk skin temperature was [nearly equal] 3 degrees Celsius less than core temperature during the control period, and the difference between skin and core temperature remained constant throughout anesthesia.

Core temperature, which was nearly constant during the control period, decreased precipitously after induction of anesthesia. Redistribution accounted for 87% of the decrease in core temperature during the first 30 min and 66% during the period from 30 to 60 min. Thus, after 1 h of anesthesia, core temperature had decreased 1.6 plus/minus 0.3 degrees Celsius, with redistribution contributing 81% to the decrease. This corresponded to a redistribution of 46 kcal during the first hour of anesthesia. During the subsequent 2 h of anesthesia, core temperature decreased an additional 1.1 plus/minus 0.3 degrees Celsius, to which redistribution contributed only 43%. Thus, only 17 kcal were redistributed during the second and third hours of anesthesia and redistribution still accounted for 35% of the observed decrease in core temperature. Redistribution therefore contributed 65% to the entire 2.8 plus/minus 0.5 degrees Celsius decrease in core temperature during 3 h of anesthesia (Figure 2).
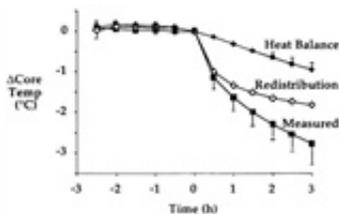

Figure 2

The parabolic regression correlation coefficients for extremity skin and tissue temperatures were generally excellent (i.e., r sup 2 > 0.95). Data fitting a parabolic regression relatively poorly (i.e., $r^2 = 0.90$) always fit a fourth-order equation well (i.e., r sup 2 = 0.98). However, changes in heat balance calculated using parabolic regression were virtually identical to those obtained from fourth-order fits. Consequently, we used and report parabolic regression throughout. Changes in "deep temperature" on back of hand were similar to those in the adductor pollicis muscle. Thus, changes in hand heat

content would have been comparable had we used that temperature instead in our calculations. Initial (-2.5 h) arm and leg heat content averaged 1,204 kcal.

Average arm tissue temperature increased [nearly equal] 1.7 degrees Celsius after induction of anesthesia and remained elevated for the duration of the study. In contrast, average leg tissue temperature only increased [nearly equal] 0.5 degrees Celsius and subsequently decreased to less than preinduction values (Figure 3). However, because the mass of the legs far exceeded that of the arms, the increases in arm and leg heat contents were comparable. Arm heat content remained elevated throughout 3 h of anesthesia: leg heat content, however, subsequently decreased to less than control values (Figure 4).
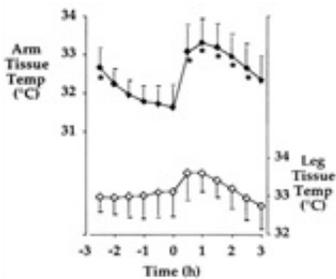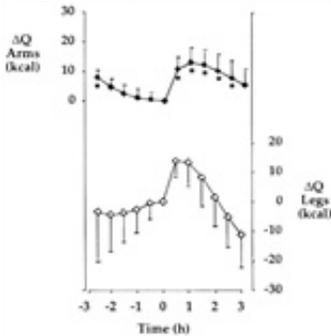

Figure 3

Figure 4

Proximal extremity (arm and thigh) tissue temperature decreased after induction of anesthesia, although the reduction was considerably less than that in core temperature. In contrast, induction of anesthesia markedly increased distal extremity (forearm, hand, calf, and foot) temperature. Average (volume-weighted) tissue temperature in the combined proximal and distal extremities thus increased only slightly after induction of anesthesia and returned to preinduction values by the end of the study (Figure 5). Proximal extremity heat content decreased after induction of anesthesia, but distal heat content increased [nearly equal] 40 kcal. Combined, proximal and distal extremity heat content increased [nearly equal] 25 kcal during the first hour of anesthesia but subsequently returned to preinduction values (Figure 6).
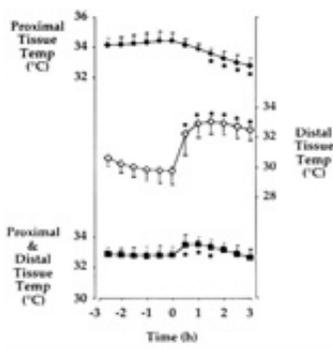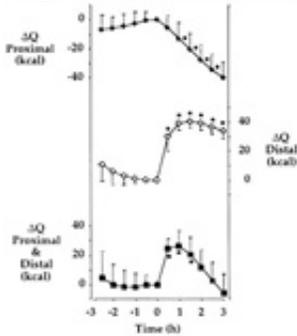
Figure 5

Figure 6

Discussion

Cutaneous heat loss and metabolic heat production were well balanced during the control period, and as a result, body heat content remained nearly constant. As in previous studies, [2] induction of general anesthesia only slightly increased cutaneous heat loss. Metabolic heat production, however, decreased by one-third, which is comparable to previous reports. [6] Consequently, overall body heat content decreased at a rate of [nearly equal] 31 kcal/h throughout anesthesia. This imbalance was small compared to the amount of redistributed heat and therefore contributed little to core hypothermia during the first hour of anesthesia. However, it subsequently became the major factor reducing core temperature during the second and third hours of anesthesia.

Our major finding thus is that redistribution hypothermia accounted for 81% of the 1.6 plus/minus 0.3 degree Celsius decrease in core temperature during the first hour of anesthesia. These results confirm and quantify our previous conclusion that internal redistribution of body heat is the major cause of hypothermia during the first hour of anesthesia. [2] The importance of redistribution is consistent with the ability of prewarming [1] and pharmacologic vasodilation [36] to minimize anesthetic-induced hypothermia. Only 43% of the 1.1 plus/minus 0.3 degrees Celsius decrease in core temperature observed during the second and third hours of anesthesia resulted from redistribution. Nonetheless, redistribution contributed 65% to the entire 2.8 plus/minus 0.5 degrees Celsius decrease in core temperature during the 3 h of anesthesia. This substantial contribution explains why prewarming remains effective during many hours

of surgery. [37].

We have divided the leg into two [12,38] or six [31] compartments in previous studies of tissue heat content. To further refine our measurements of tissue temperature and heat content, we inserted more needle thermocouples in our current volunteers and also estimated foot temperature using the "deep tissue" thermometer. Accordingly, we were able to divide the leg into nine compartments. More importantly, we also evaluated arm tissue temperature and heat content, using a three-compartment model. Although the arms constituted only a fifth of the peripheral compartment mass, redistribution increased arm heat content nearly as much as leg heat content. These data suggest that the arms, as well as the legs, are important components of the peripheral thermal compartment. It is thus likely that the [nearly equal] 50 kcal reduction in leg heat content we reported during the plateau phase [12] is accompanied by a comparable reduction in arm heat content.

Proximal extremity tissue temperature and heat content decreased consistently after induction of anesthesia, even during the first hour, when redistribution increased distal tissue heat content [nearly equal] 40 kcal. However, the temperature decreased far less than core temperature. Proximal extremities, thus, are "intermediate" tissues, sharing characteristics of both the core and peripheral thermal compartments. The distal extremities appear to be the most important components of the peripheral thermal compartment, although their mass is far less than that of the proximal limb segments. It remains likely, however, that proximal tissues would have contributed more to the peripheral thermal compartment had the environment been cooler or exposure longer.

Although we separately evaluated tissue temperature and heat content in 12 peripheral segments, our conclusions rest on a limited number of tissue and skin temperatures and assumptions of radial and axial symmetry within segments. Furthermore, hand tissue temperature was determined with a single thermocouple, and foot temperature was estimated from a single "deep temperature" site. Better measurements of hand and foot tissue heat content would be preferable because they contributed substantially to the total changes in peripheral tissue temperatures. However, such measurements may not be practical because a high density of critical structures preclude inserting many needle thermocouples into these structures, and asymmetric anatomy would have made interpretation of such temperatures difficult. Fortunately, the changes in adductor pollicis and "deep temperature" on the back of the hand were comparable, suggesting that either single temperature reasonably characterized vasodilation-induced changes in hand heat content.

We did not directly evaluate distribution of tissue heat within the trunk (including the head). However, the difference between trunk skin temperature and core temperature remained constant throughout the study, despite marked core-to-peripheral redistribution of body heat and a nearly 3 degrees Celsius decrease in core temperature.

Furthermore, the change in body heat content determined from the difference between overall heat production and loss was virtually identical to that determined by adding the measured changes in arm and leg heat content to changes in trunk heat content determined by simply multiplying core temperature, trunk mass, and tissue specific heat. Both factors suggest that distribution of heat within the trunk is not substantially altered by induction of general anesthesia and that the trunk can be modeled as a single (core) compartment.

Extremity heat content did not decrease during the control period, although we have demonstrated decreases in other studies. [39] Presumably, no decrease was observed because this investigation took place during the winter, and the volunteers already were vasoconstricted when they arrived in the laboratory and therefore had relatively low peripheral tissue heat content before the study started.

Both capillaries and arteriovenous shunts perfuse skin. [40] The shunts are specialized [nearly equal] 100-micro meter vessels that carry 10,000 times as much blood per unit length as [nearly equal] 10-micro meter capillaries. [41] Anatomically, they are restricted to peripheral tissues, most importantly the hands and feet. [42] Consistent with this distribution and previous studies, [43] calf and foot blood flow increased [nearly equal] tenfold, whereas we have previously demonstrated little increase in capillary flow on the chest. [2] It was precisely in these structures that anesthetic-inducted vasodilation most increased tissue heat content. The absolute laser Doppler values on the calf and forearm should not be compared because of differences in the probes used and instrument settings. Nonetheless, the data suggest that general anesthesia dilates forearm capillaries considerably less than those on the calf.

A limitation of this study is that only men participated. Women thermoregulate at slightly higher temperatures than men, [44,45] and anesthetic-induced changes in body heat distribution surely differ somewhat in men and women. However, it is unlikely that the differences will prove to be clinically important. Isoflurane was added to the propofol/fentanyl anesthetic in some volunteers near the end of the study when core temperatures had decreased nearly 3 degrees Celsius. The isoflurane had no perceptible effect on cutaneous heat loss or metabolic production but prevented thermoregulatory vasoconstriction, which would have markedly complicated interpretation of the data by producing a core-temperature plateau. [12] For the purpose of this heat balance study, the type of anesthesia was relatively unimportant: however, it was critical to administer an amount sufficient to prevent thermoregulatory vasoconstriction. [9].

Several of the volunteers who shivered were covered with a single blanket near the end of the control period. A blanket reduces cutaneous heat loss [nearly equal] 30%, [14] which is similar to the reduction provided by a single layer of cloth or paper surgical draping. [46] Cutaneous heat loss in the covered volunteers was thus similar to that in

fully draped patients undergoing minor surgery. Loss was greater in the undraped volunteers and would be more typical of that in patients given [nearly equal] 1 l/h of unwarmed intravenous fluid or experiencing evaporative heat loss from within a small surgical incision.

By virtue of incorporating a long control period in a cool environment, this study was designed to maximize redistribution hypothermia. The precise contributions of redistribution and heat balance to observed changes in core temperature will differ under other circumstances. For example, redistribution will contribute considerably less in patients actively prewarmed. [36,37] or simply maintained in a sufficiently warm environment before induction of anesthesia. [47] Similarly, the imbalance between heat production and loss would be greater in patients maintained in colder (or laminar flow) environments or in those undergoing large operations. [48] Such patients would become more hypothermic than the volunteers in this study, but redistribution would contribute proportionately less to the hypothermia.

In summary, proximal extremity heat content decreased after induction of anesthesia, but distal heat content increased [nearly equal] 40 kcal. Although the arms constituted only one-fifth of extremity mass, redistribution increased arm heat content nearly as much as leg heat content. The arms and legs thus are both important components of the peripheral thermal compartment, but distal segments contribute most. Redistribution hypothermia accounted for 81% of the 1.6 plus/minus 0.3 degrees Celsius decrease in core temperature during the first hour of anesthesia, and required transfer of 46 kcal from the trunk to the extremities. However, redistribution contributed only 43% (17 kcal) to the additional 1.1 plus/minus 0.3 degrees Celsius decrease observed during the subsequent 2 h. Thus, core hypothermia during the first hour after induction resulted almost exclusively from redistribution of body heat, and redistribution remained the most important cause even after 3 h of anesthesia.

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