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Heat Flow and Distribution during Epidural Anesthesia

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Background: **Core hypothermia after induction of epidural anesthesia results from both 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 thus 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.**

Methods: **Twelve minimally clothed male volunteers were evaluated in a ≈22°C environment for 2.5 control hours before induction of epidural anesthesia and for 3 subsequent hours. Epidural anesthesia produced a bilateral sympathetic block in only six volunteers, and only their results are reported. Shivering, when observed, was treated with intravenous meperidine. 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 esophageal or tympanic membrane (core) temperatures, leaving the core hypothermia specifically resulting from redistribution.**

Results: **Arm heat content decreased ≈5 kcal/h after induction of anesthesia, but leg heat content increased markedly. Most of the increase in leg heat content was in the lower legs and feet. Core temperature increased slightly during the control period but decreased 0.8 ± 0.3°C in the 1st hour of anesthesia. Redistribution, contributing 89% to this initial decrease, required a net transfer of 20 kcal from the trunk to the extremities. During the subsequent 2 h of anesthesia, core temperature decreased an additional 0.4 ± 0.3°C, with redistribution contributing 62%. Thus, only 7 kcal were redistributed during the 2nd and 3rd hours of anesthesia. Redistribution therefore contributed 80% to the entire 1.2 ± 0.3°C decrease in core temperature during the 3 h of anesthesia.**

Conclusions: **Core hypothermia during the 1st hour after induction of epidural anesthesia resulted largely from redistribution of body heat from the core thermal compartment to the distal legs. Even after 3 h of anesthesia, redistribution remained the major cause of core hypothermia. Despite the greater fractional contribution of redistribution during epidural anesthesia, core temperature decreased only half as much as during general anesthesia because metabolic rate was maintained and the arms remained vasoconstricted. (Key words: Anesthesia: epidural. Heat balance: distribution. Hypothermia: redistribution. Temperature, regulation: vasoconstriction; vasodilation. Temperature, measurement: core; muscle; skin. Thermoregulation.)**

HYPOTHERMIA during anesthesia results from a combination of negative heat balance[1,2] (heat loss exceeding metabolic heat production) and core-to-peripheral redistribution of internal heat.[1,3] Redistribution contributes ≈80% to the observed decrease in core temperature during the 1st hour of general anesthesia. Even after 3 h of general anesthesia, redistribution contributes more to core hypothermia than does negative heat balance.[1] Interestingly, distribution of heat to the arms and legs appears comparable, despite the considerably smaller mass of the arms.

Although a sympathetic block to the legs often accompanies epidural anesthesia,[4,5] sympathetic supply to the arms usually remains intact. It is thus likely that less heat will be redistributed from the core to peripheral tissues during major conduction anesthesia than during general anesthesia. Nonetheless, previous work indicates that core hypothermia during epidural anesthesia develops despite a positive heat balance (resulting from upper-body shivering thermogenesis).[6] That is, body heat content increases while core tem-

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perature decreases. These data suggest that redistribution also is a major cause of hypothermia during regional anesthesia.

Accordingly, we determined the extent to which redistribution of body heat contributes to core hypothermia during epidural anesthesia. This investigation differs from our previous one[6] because we now specifically quantify the internal flow and distribution of heat during regional anesthesia. Furthermore, we administered sufficient meperidine to prevent shivering, thus stimulating the common clinical situation in which shivering is blunted by age,[7,8] central thermoregulatory failure,[9,10] or administration of sedative drugs.[11,12]

Methods

With approval from the Committee on Human Research at the University of California, San Francisco, and written informed consent, we studied 12 male volunteers given epidural anesthesia. None was obese, was taking medication, or had a history of thyroid disease, dysautonomia, Raynaud's syndrome, or malignant hyperthermia.

Protocol

Studies started at approximately 9:30 AM, and volunteers fasted during the 8 h preceding each study. Throughout the study, minimally clothed volunteers reclined on an operating room table set in chaise-lounge position.

An intravenous catheter was inserted into an antecubital vein on the left arm. Using sterile technique, the skin and subcutaneous tissue over the L2–L3 or L3–L4 interspace in each volunteer were infiltrated with 2 ml 1% lidocaine. The epidural space was identified using an 18-G Tuohy needle and loss of resistance to air. An epidural catheter was advanced 2–3 cm into the epidural space and the needle removed.

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,[13] which then was left in place throughout the study.

After a 3-ml test dose of 1% lidocaine with 1:100,000 epinephrine, regional anesthesia was induced by injection of ≈15 ml 1% lidocaine into the epidural catheter. The initial volume was chosen based on subject's height. Injections were given at a rate of ≈5 ml/min. Additional 10-ml boluses were given at 10-min intervals if the level of sensory blockade was lower than the tenth thoracic dermatome as determined by loss of cutaneous cold sensation. Local anesthetic was infused into the epidural catheter at a rate sufficient to maintain a comparable sensory block level (≈10 ml/h).

An intravenous bolus of 15 ml/kg of lactated Ringer's solution (warmed to 37°C) was begun 5 min before administration of the test dose and completed over the ensuing 15 min. Blood pressure was maintained within 20% of control values by administration of additional intravenous fluid volume during onset of anesthesia. Lactated Ringer's solution warmed to 37°C subsequently was infused at ≈100 ml/h for the remainder of the study.

Shivering during epidural anesthesia was monitored by visual inspection, increases in systemic oxygen consumption, and electromyographic activity (see below). When detected at any intensity by any method, it was treated aggressively by intravenous administration of meperidine in 10–15-mg boluses.[14,15] After 3 h, epidural anesthesia was discontinued. Study measurements ceased at that point, and the volunteers were rewarmed with forced air (Bair Hugger, Augustine Medical, Eden Prairie, MN).[16]

Monitoring

Core temperature was measured in the distal esophagus, with a probe (Mallinckrodt, St. Louis, MO) positioned according to the formula of Mekjavic *et al.*[17] Two volunteers were unable to swallow the esophageal probe: their core temperatures were recorded from the tympanic membrane using Mon-a-Therm thermocouples. Changes in distal esophageal and tympanic membrane temperatures are almost identical under the circumstances of this study.[18] Energy expenditure, derived from oxygen consumption and carbon dioxide production, was measured using a calibrated metabolic monitor (Deltatrac, SensorMedics, Yorba Linda, CA). Measurements were averaged over 5-min intervals and recorded every 5 min.

Heat flux and temperature from 15 skin-surface sites were measured using thermal flux transducers (Concept Engineering, Old Saybrook, CT), as previously described.[1] As in previous studies, measured cutaneous heat loss was augmented by 10% to account for insensible transcutaneous evaporative loss[19] and 3% to com-

pensate for the skin covered by the volunteers' shorts. We further augmented cutaneous loss by 10% of the metabolic rate (as determined from oxygen consumption) to account for respiratory loss.[20] We defined flux as positive when heat traversed skin to the environment.

Calf minus toe skin-surface temperature gradients were used as an index of foot arteriovenous shunt perfusion.[18] As in previous studies,[3] we considered a leg gradient exceeding 0°C to indicate vasoconstriction. Vascular tone also was evaluated on the right second toe using the perfusion index, which is derived from absorption of two infrared wave lengths.[21] Capillary flow was estimated using laser Doppler flowmetry (Periflux 3, Perimed, Piscataway, NJ) with an integrating multiprobe ("wide-band" setting) positioned on the calf.[22,23] Left calf blood flow was quantified using capacitance-based "extensometer" plethysmography (without a distal tourniquet).[24,25] Forearm minus fingertip skin-surface temperature gradients were used as an index of hand arteriovenous shunt perfusion.[26] We considered an arm gradient exceeding 0°C to indicate vasoconstriction.

Arm and leg tissue heat contents were determined from 19 intramuscular needle thermocouples, 10 skin temperatures, and "deep" foot temperature (CoreTemp, Terumo Medical, Tokyo, Japan).[27,28] Briefly, the legs (to the illiac crests) were divided into nine compartments, and the arms were divided into three compartments. Within each, measured temperatures were fit to a parabolic regression, and the resulting equation integrated over volume to provide tissue heat content. We previously have described the details and limitations of these measurements.[1,3]

After mild skin abrasion and degreasing, silver/silver chloride monitoring electrodes were positioned to record the electrical activity of the pectoralis and trapezius bilaterally. The active electrodes were positioned 4 cm apart and oriented in the direction of the muscle fibers.[29] After appropriate amplification (model P511, Grass, Quincy, MA), the signals were recorded on a thermoelectric printer having a linear resolution to 1,000 Hz (Dash-IV, Astro-Med, West Warwick, RI).

Heart rate and oxyhemoglobin saturation were measured continuously using pulse oximetry, and blood pressure was determined oscillometrically. Thermoregulatory and anesthetic data were recorded electronically at 5-min intervals. Sensory block levels (perception of applied cold) were tested bilaterally at 15-min intervals.

Statistical Analysis

Overall changes in body heat were calculated as the time integral of metabolic heat production minus cutaneous heat loss. Two independent factors potentially contributed to core hypothermia after induction of anesthesia: decreased overall heat balance and internal redistribution of body heat. To separate the effects, we divided the change in overall heat balance by body weight and the specific heat of humans (0.83 $kcal \cdot kg^{-1} \cdot °C^{-1}$).[30] 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-measures analysis of variance; values were compared with those recorded at time zero (induction of anesthesia) with Dunnett's test. Results are expressed as mean ± SD; differences were considered statistically significant when $P < 0.05$.

Results

Epidural anesthesia induced a bilateral ≈T10 sensory block in every case, a sensory block that extended to the sacral area and both legs. However, the legs remained bilaterally vasoconstricted in four of the volunteers and unilaterally vasoconstricted in two others. A bilateral sympathetic block thus was evident in only six volunteers, and only their results are presented.

The volunteers in whom epidural anesthesia produced a bilateral sympathetic block had the following morphometric characteristics: height 178 ± 5 cm (mean ± SD), weight 77 ± 11 kg, and age 28 ± 4 yr. The percentage of body fat was 18 ± 5.[31] Ambient temperature was maintained at 22.2 ± 0.6°C and ambient relative humidity at 40 ± 4% during the study period (model HX93 humidity and temperature transmitter, Omega).

The sensory block level was T10 ± 1 segment on the left and T10 ± 1 segment on the right. Shivering was treated successfully in all cases, with 24 ± 8 mg meperidine. Estimated masses of the thighs and lower legs (including feet) were 24 ± 4 kg and 10 ± 1 kg, respectively. Consequently, the legs represented ≈43% of our volunteers' total mass. Similarly, estimated masses of the upper and lower arms (including hands)

were 5 ± 1 kg and 6 ± 1 kg, respectively. Consequently, the arms represented ≈14% of our volunteers' total mass.

Vasoconstriction was apparent in the arms and legs throughout the control period, and vasoconstriction in the arms persisted after induction of epidural anesthesia. In contrast, epidural anesthesia caused significant leg vasodilation. The toe perfusion index increased from 0.3 ± 0.2 to 2.6 ± 11 units; similarly, total calf flow increased from 1.4 ± 1.2 to 5.3 ± 4.2 $ml \cdot min^{-1} \cdot 100\ g^{-1}$. Capillary perfusion on the calf, as evaluated using laser Doppler flowmetry, also increased slightly (table 1).

Cutaneous heat loss slightly exceeded heat production throughout the study. As a result, overall heat balance (production minus loss) decreased linearly at ≈6 kcal/h. Changes in body heat content were generally similar when calculated as the difference between heat production and loss or as the sum of measured changes in arm, leg, and core heat contents. However, there was a slight disparity after induction of anesthesia (fig. 1).

Core temperature, which increased slightly during the control period, decreased precipitously after induction of anesthesia. During the 1st hour of anesthesia, core temperature decreased 0.8 ± 0.3°C, with redistribution contributing 89% to the decrease. This corresponds to a redistribution of 20 kcal during the 1st hour of anesthesia. During the subsequent 2 h of anesthesia, core temperature decreased an additional 0.4 ± 0.3°C, with redistribution contributing 62%. Thus, only 7 kcal was redistributed during the 2nd and 3rd hours of anesthesia. Redistribution therefore contributed 80% to the 1.2 ± 0.3°C decrease in core temperature during the 3 h of anesthesia (fig. 2).

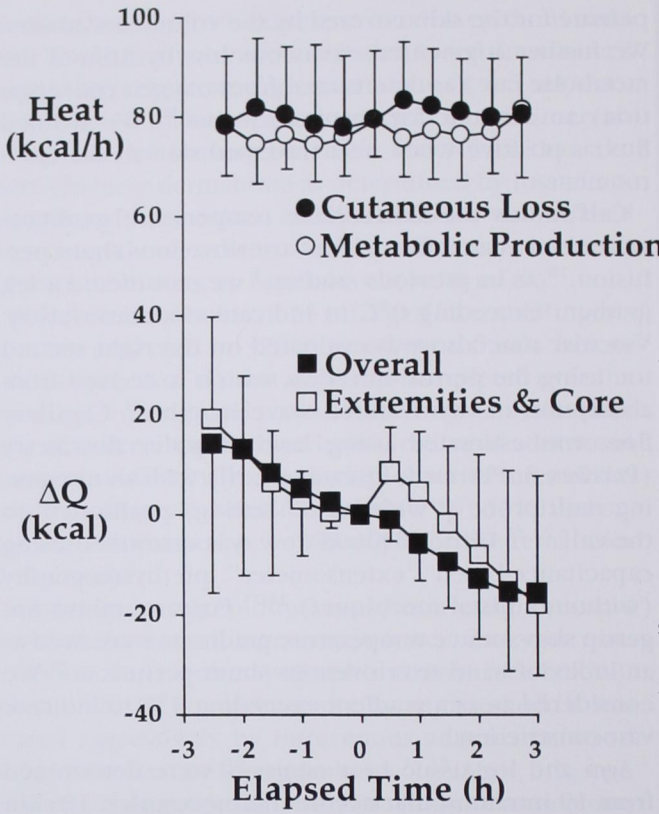

Fig. 1. Overall heat balance was only slightly negative (loss exceeding production) before induction of anesthesia and subsequently changed little. Changes in body heat content (ΔQ) were calculated two ways: (1) "overall" heat balance was calculated from the difference between metabolic heat production and heat loss, and (2) "extremity and core" changes were calculated as the sum of measured changes in the arms and legs and the change in core temperature multiplied by the weight of the trunk and the specific heat of human tissues. Although there was a slight disparity after induction of anesthesia, body heat content—calculated each way—was similar throughout the study. Body heat content results are presented as cumulative changes, referenced to induction of epidural anesthesia at elapsed time 0.

Table 1. Arm and Leg Blood Flow Responses

	Control Period	Epidural Anesthesia
Forearm–fingertip gradient (°C)	5.2 ± 2.2	5.1 ± 1.4
Vasomotor index right finger	0.1 ± 0.1	0.0 ± 0.1
Vasomotor index left finger	0.1 ± 0.2	0.0 ± 0.1
Perfusion index/toe (units)	0.3 ± 0.2	2.6 ± 1.1*
Calf–toe gradient (°C)	7.0 ± 0.7	−2.9 ± 3.6*
Extensometer/leg ($ml \cdot min^{-1} \cdot 100\ g^{-1}$)	1.4 ± 1.2	5.3 ± 4.2*
Laser Doppler/calf (units)	8.6 ± 4.4	11.3 ± 5.2

Data are mean ± SD and indicate the time averages during the respective study periods.

* Significant increases in flow after induction of epidural anesthesia ($P < 0.01$).

Arm heat content decreased ≈5 kcal in the 3 h before induction of epidural anesthesia and an additional ≈15 kcal during the 3 subsequent hours (fig. 3). Proximal and distal leg heat contents increased after induction of epidural anesthesia, but distal heat content increased most. Combined, proximal, and distal leg heat content increased ≈35 kcal during the 1st hour of anesthesia and remained increased for the remaining 2 h of study (fig. 4).

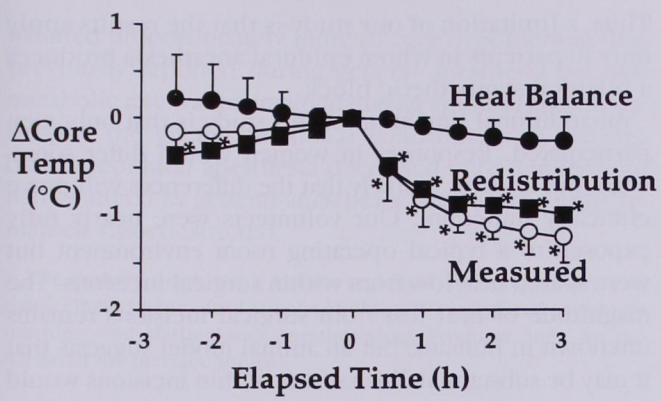

Fig. 2. To separate the contributions of decreased overall heat balance and internal redistribution of body heat to the decrease in core temperature, we divided the change in overall heat balance by body weight and the specific heat of humans. The resulting change in mean body temperature ("heat balance") was subtracted from the change in core temperature ("measured"), leaving the core hypothermia specifically resulting from redistribution ("redistribution"). After 1 h of anesthesia, core temperature had decreased 0.8 ± 0.3°C, with redistribution contributing 89% to the decrease. During the subsequent 2 h of anesthesia, core temperature decreased an additional 0.4 ± 0.3°C, with redistribution contributing 62%. Redistribution thus contributed 80% to the entire 1.2 ± 0.3°C decrease in core temperature during the 3 h of anesthesia. The increase in the "redistribution" curve before induction of anesthesia indicates that thermoregulatory vasoconstriction was constraining metabolic heat to the core thermal compartment. Such constraint is, of course, the only way in which core temperature could increase while body heat content decreased. Induction of epidural anesthesia is identified as elapsed time 0. *Values differing significantly from time 0.

Discussion

As in our previous studies of heat balance during general[1] and epidural[6] anesthesia, heat loss and metabolic heat production were similar before induction. Also in the previous studies, induction of anesthesia only slightly increased heat loss. Epidural anesthesia had little effect on metabolic rate in our current volunteers because shivering was treated with meperidine, whereas shivering thermogenesis increased heat production ≈25% in our previous investigation.[6] Core hypothermia in our current volunteers, therefore, developed with only slight assistance from a negative heat balance, whereas previously it developed despite a distinctly positive heat balance.

As in our previous studies of heat balance during general anesthesia,[1] core-to-peripheral redistribution of body heat was the major cause of core hypothermia during the 1st hour of anesthesia, contributing 89%. Redistribution continued to contribute 62% to core hypothermia during the 2nd and 3rd hours of epidural anesthesia, although it contributed only 43% during the 2nd and 3rd hours of general anesthesia.

Although core-to-peripheral redistribution of body heat contributed more to core hypothermia during epidural anesthesia than during general anesthesia, core temperature decreased more than twice as much during general anesthesia. Core temperature decreased less because: (1) heat balance was only slightly negative during epidural anesthesia, whereas body heat content decreased ≈30 kcal/h during general anesthesia[1]; and (2) vasodilation was confined to the legs during epidural anesthesia, whereas heat from the core was redistributed to both the arms and legs during general anesthesia. Our results thus confirm our previous conclusion that core-to-peripheral redistribution of body heat contributes to core hypothermia during epidural anesthesia; additionally, we now quantify the magnitude of this contribution.

As one might expect in minimally clothed volunteers exposed to a cool environment,[32] the arms were bilaterally vasoconstricted during the control period and remained constricted throughout anesthesia. Leg va-

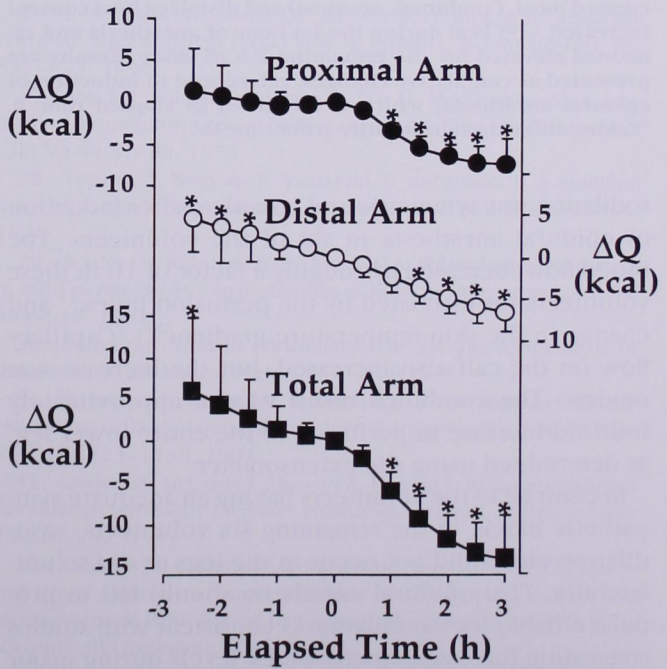

Fig. 3. Arm heat content decreased ≈5 kcal in the 3 h before induction of epidural anesthesia and ≈15 kcal during the subsequent 3 h. Induction of anesthesia is identified as elapsed time 0. Data are presented as mean ± SD. *Values differing significantly from time 0.

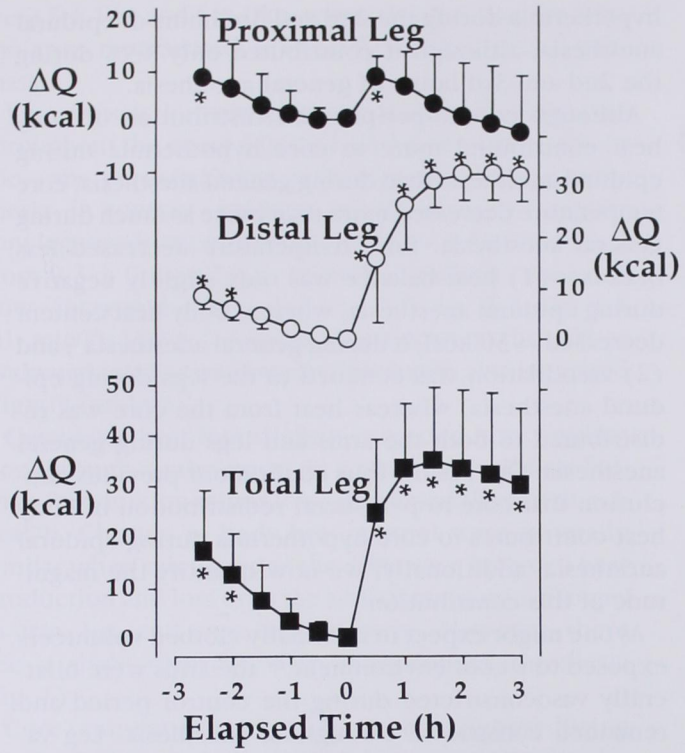

Fig. 4. Proximal and distal leg heat content increased after induction of epidural anesthesia, but distal heat content increased most. Combined, proximal and distal leg heat content increased ≈35 kcal during the 1st hour of anesthesia and remained elevated for the remaining 2 h of study. Results are presented as cumulative changes, referenced to induction of epidural anesthesia, which is identified as elapsed time 0. *Values differing significantly from time 0.

sodilation was symmetric and complete after induction of epidural anesthesia in six of the volunteers. Toe blood flow increased by roughly a factor of 10 in these volunteers (as estimated by the perfusion index[21] and change in the skin-temperature gradient[26]). Capillary flow on the calf also increased, but the increase was modest. The combined result was an approximately fourfold increase in perfusion to the entire lower leg, as determined using the extensometer.

In contrast to the volunteers having an adequate sympathetic block, in the remaining six volunteers, vasodilation either did not occur in the legs or did so unilaterally. That epidural anesthesia should fail to produce reliable leg vasodilation is consistent with studies suggesting that sympathetic block levels during major conduction anesthesia are inconsistent.[33] We did not continue studies in volunteers in whom bilateral vasodilation in the toes failed to develop, because in the absence of vasodilation, there was no redistribution. Thus, a limitation of our study is that the results apply only to patients in whom epidural anesthesia produces a bilateral sympathetic block.

An additional limitation of this study is that only men participated. Responses in women would differ somewhat, but it seems unlikely that the differences will prove clinically important. Our volunteers were nearly fully exposed to a typical operating room environment but were spared heat loss from within surgical incisions. The magnitude of heat loss from surgical incisions remains unknown in humans, but an animal model suggests that it may be substantial.[34] Loss from within incisions would contribute to a negative heat balance and increase core hypothermia. However, it would decrease the fractional contribution of redistribution. Another potential limitation of this investigation is that systemic absorption of lidocaine may have caused vasodilation. This possibility is supported by the observation that lidocaine in sufficient doses has general anesthesia properties, and all general anesthetics so far tested markedly impair thermoregulation. However, we previously demonstrated that intravenous administration of lidocaine in doses sufficient to produce plasma concentrations similar to those observed during epidural anesthesia has no important thermoregulatory effect.[35] Furthermore, vasodilation in the current investigation was restricted to the legs, whereas a systemic effect resulting from absorbed lidocaine presumably would cause vasodilation in the arms as well.

There was a transient small discrepancy between systemic heat balance and measured heat content immediately after induction of epidural anesthesia. The difference apparently resulted from acute cutaneous vasodilation, which degraded the parabolic temperature regressions from which tissue heat content is calculated. This artifactual increase highlights the numerous assumptions and extrapolations required for our heat balance estimates. (We previously described the potentially substantial limitations of the measurements in some detail.[1,3]) By the end of the study, leg tissue temperatures were better equilibrated, and the two methods again agreed. Consequently, we believe our overall conclusions are likely to be accurate because the two methods similarly estimated body heat content throughout most of the study.

In summary, core hypothermia during the 1st hour after induction of epidural anesthesia associated with sympathetic block resulted largely from redistribution of body heat from the core thermal compartment to the distal legs. Even after 3 h of anesthesia, redistribution remained the major cause of core hypothermia. Redistribution con-

tributed proportionately more to core hypothermia than previously reported during general anesthesia because metabolic rate was maintained during epidural anesthesia. Despite the greater fractional contribution of redistribution, epidural anesthesia decreased core temperature half as much as general anesthesia because the arms remained vasoconstricted.

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