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Optimal Duration and Temperature of Prewarming

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Background: **Core hypothermia developing immediately after induction of anesthesia results largely from an internal core-to-peripheral redistribution of body heat. Although difficult to treat, redistribution can be prevented by prewarming. The benefits of prewarming may be limited by sweating, thermal discomfort, and efficacy of the warming device. Accordingly, the optimal heater temperature and minimum warming duration likely to substantially reduce redistribution hypothermia were evaluated.**

Methods: **Sweating, thermal comfort, and extremity heat content were evaluated in seven volunteers. They participated on two study days, each consisting of a 2-h control period followed by 2 h of forced-air warming with the heater set on "medium" (≈40°C) or "high" (≈43°C). Arm and leg tissue heat contents were determined from 19 intramuscular needle thermocouples, ten skin temperatures, and "deep" foot temperature.**

Results: **Half the volunteers started sweating during the second hour of warming. None of the volunteers felt uncomfortably warm during the first hour of heating, but many subsequently did. With the heater set on "high," arm and leg heat content increased 69 kcal during the first 30 min of warming and 136 kcal during the first hour of warming, representing 38% and 75%, respectively, of the values observed after 2 h of warming. The increase was only slightly less when the heater was set to "medium."**

Conclusions: **Neither sweating nor thermal discomfort limited heat transfer during the first hour of warming. Thirty minutes of forced-air warming increased peripheral tissue heat content by more than the amount normally redistributed during the first hour of anesthesia. The large increase in arm and leg heat content during prewarming thus explains the observed efficacy of prewarming. (Key words: Anesthetic techniques: prewarming. Heat: balance; distribution. Temperature, measurement: muscle; skin; tympanic membrane. Thermoregulation: vasoconstriction; vasodilation.)**

CORE hypothermia developing immediately after induction of general[1] and regional[2] anesthesia results largely from an internal core-to-peripheral redistribution of body heat. Under test conditions, 81% of the observed 1.6°C reduction in core hypothermia during the first hour of general anesthesia represented redistribution and resulted from a flow of 46 kcal from the trunk to extremities. In contrast, only 17 kcal was redistributed during the subsequent 2 h of anesthesia.[3]

It is difficult to *treat* redistribution hypothermia both because the internal flow of heat is large and—more importantly—because heat applied to the skin surface requires considerable time to reach the core thermal compartment. However, redistribution can be *prevented* by prewarming. Cutaneous warming before induction of anesthesia has little effect on core temperature (which remains well regulated).[4,5] It does increase peripheral tissue temperature and reduce the normal core-to-peripheral temperature gradient. Subsequent induction of anesthesia then produces little redistribution hypothermia because heat can only flow down a temperature gradient.[4,6,7] The efficacy of prewarming is thus determined by the extent to which treatment increases peripheral thermal compartment (*i.e.,* extremity) tissue temperature and heat content.

Several factors potentially limit the speed and maximum efficacy of prewarming. (1) Sweating is a remarkably effective thermoregulatory response,[8] easily dissipating more heat than is provided by even the best clinical warming devices. It is regulated by core and skin temperature and the rate of skin temperature change. Sufficiently aggressive cutaneous warming may thus trigger sweating and *reduce* net cutaneous heat transfer. (2) High skin temperatures—and especially rapid increases in skin temperature—provokes thermal discomfort. Such discomfort may limit the tolerable

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duration of aggressive warming. (3) Body heat content increases as the sum of metabolic heat production and environmental loss/gain. Resting metabolic rate is essentially constant, but noninvasive warmers differ significantly in their cutaneous heat transfer rates. Forced-air is the most effective clinically available warming method[5,9] and thus potentially increases tissue temperature most rapidly. Nonetheless, tissue heat transfer at the highest temperature settings may be restricted by sweating or thermal discomfort.

Previous studies demonstrating the benefits of prewarming on redistribution hypothermia have applied moderate heat intensities for 1.5–2 h.[4,6,7] Such prolonged prewarming is, however, impractical in most hospitals. Accordingly, we evaluated sweating, thermal comfort, and the rate at which peripheral tissue heat content increases during moderate and intense forced-air warming. Our purpose was to determine the optimal heater temperature and minimum warming duration likely to substantially reduce redistribution hypothermia.

Methods

With approval from the Committee on Human Research at the University of California, San Francisco and written informed consent, we studied seven male volunteers. None was obese, was taking medication, or had a history of thyroid disease, dysautonomia, or Raynaud's syndrome. Each participated on 2 study days in March 1994.

The volunteers' height was 176 ± 8 cm (mean ± SD), weight 84 ± 15 kg, and age 32 ± 6 yr. The percentage of body fat was 17 ± 3, as determined using infrared interactance[10] (Futrex 1000, Futrex, Hagerstown, MD). Ambient temperature was maintained at 21.3 ± 0.5°C and ambient relative humidity at 36 ± 5% during the study period (Model HX93 humidity and temperature transmitter, Omega Engineering, Stamford, CT).

Studies in two of the volunteers started at approximately 9:30 AM; studies in five others began near 5 PM. All started with 2 h of exposure to a typical operating room environment (control period), which was sufficient to trigger thermoregulatory vasoconstriction. The volunteers then were warmed for two h with a Bair Hugger forced-air heater (Model 200 blower, full-body cover, Augustine Medical, Eden Prairie, MN).[5] On 1 day, the blower was set to "high" (≈43°C), and on the other it was set to "medium" (≈40°C). In each case, two cotton blankets were superimposed on the covers.

Details of the measurement techniques are described in a companion manuscript[3] and previous publications. Most values were measured continuously, and recorded on a computer at 5-min intervals. Briefly, core temperature was measured at the tympanic membrane.[11] Peripheral tissue temperatures were measured using 10 cutaneous probes, foot "deep temperature,"[12,13] and 19 thermocouple needles inserted into arm and leg muscles.[3] Extremity heat content was calculated by fitting local skin and tissue temperatures to parabolic regressions and integrating over volume.[14]

Mean skin-surface temperature and cutaneous heat transfer were calculated from measurements at 15 area-weighted sites using thermocouples incorporated into thermal flux transducers.[1,3] These transducers record heat lost *via* radiation, conduction, and convection; however, they do not detect evaporative loss. Sweating on the chest was quantified by passing anhydrous oxygen through a ventilated capsule. Cutaneous water loss was calculated from the gas flow rate, gas temperature, and relative humidity, as previously described.[15,16] As in previous studies, we considered a sweating rate of 40 $g \cdot m^{-2} \cdot h^{-1}$ as significant.

Oxygen consumption 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 5-min epochs. Metabolic heat production was calculated from oxygen consumption, as previously described.[17]

Left forearm blood flow was quantified using strain-gauge plethysmography.[18] Instead of a mercury-in-rubber gauge, we used a capacitance-based "extensometer."[19] Strain-gauge plethysmography is often used to measure cutaneous capillary blood flow, in which case arteriovenous shunts in the hand or foot are isolated by an arterial tourniquet.[20] In this study, however, we avoided a distal arterial tourniquet because we were interested in total extremity blood flow. Arteriovenous shunt flow in the finger was evaluated using volume plethysmography. Capillary vasodilation was estimated using laser Doppler flowmetry (Periflux 3, Perimed, Piscataway, NJ) with an integrating multi-probe ("wide-band" setting) positioned on the right lateral forearm.[21,22]

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.[23]

Thermal comfort was evaluated at 15-min intervals, as previously described,[24] using a 100-mm visual analog scale (VAS). Zero was defined as the coldest imaginable sensation and 100 mm defined as the warmest imaginable sensation; 50 mm identified thermal comfort.

Changes in extremity heat content also were calculated from overall heat balance. Specifically, the change was calculated as the sum of metabolic heat production, cutaneous heat gain, and the change in core temperature multiplied by the weight of the trunk (and head) and the specific heat of humans (0.83 kcal · $kg^{-1}°C^{-1}$).[25] Trunk and head weight was estimated by subtracting the calculated weight of the extremities (from the radial integration) from the total weight of each subject. Calculated changes in arm and leg heat content were compared with directly measured values using two-tailed, unpaired *t* tests.

We considered measured arm and leg heat contents maximal at the end of 2 h warming with the heater set to "high." Time-dependent changes were evaluated using repeated-measure ANOVA; values were compared with those recorded at time zero (start of warming) with Dunnett's test. Differences between the treatments ("medium" and "high" heater setting) were evaluated using paired *t* tests. Results are expressed as mean ± SD; differences were considered statistically significant when $P < 0.01$.

Results

In all volunteers, vasoconstriction was observed throughout the control period. Vasodilation occurred soon after forced-air warming started, and the volunteers remained vasodilated for the rest of the study. Forearm blood flow increased significantly from 3.3 ± 3.8 ml · min^{-1} · 100 g^{-1} to 9.6 ± 5.2 ml · min^{-1} · 100 g^{-1} when it was set to "high." Consistent with this increase, finger (arteriovenous shunt) flow increased from 0.1 ± 0.1 ml/min to ≈0.9 ml/min. Forearm capillary flow doubled on the "medium" and tripled on the "high" settings. Leg vasodilation was dramatic, with both the perfusion index on the toe and capillary flow on the calf (as evaluated using laser Doppler flowmetry) increasing significantly (table 1).

Metabolic heat production, which was nearly constant at ≈100 W before induction of anesthesia, decreased slightly during forced-air heating. Cutaneous heat loss was ≈97 W before warming was started. In contrast, the first 40 min of active warming transferred 7 ± 12 W through the skin surface on the "medium" setting, and 21 ± 15 W when the heater was set to "high." As the skin and subcutaneous tissues warmed, heat transfer subsequently decreased ≈7 and ≈17 W, respectively, at each setting. No sweating was observed during the control period or for the first hour of warming. However, the increases in core temperature during the second hour of active warming triggered detectable sweating in about half the volunteers (fig. 1).

After an initial 0.1°C increase, core temperatures gradually increased an additional ≈0.1°C during the control period. During the first 30 min of forced-air heating, core temperatures decreased ≈0.2°C at each temperature setting. Subsequently, core temperatures increased ≈0.3°C when the warmer was set on "medium" and ≈0.4°C when it was set to "high" (fig. 2). Mean skin temperature, which was ≈32°C at the beginning of the study, decreased ≈0.4°C during the control period. Subsequently, it increased to 36.7 ± 0.2°C when the forced-air warmer was set to "medium" and 37.1 ± 0.2°C when it was set to "high."

Estimated mass of the thighs and lower legs (including feet) were 20 ± 9 kg and 8 ± kg, respectively. Consequently, the legs represented ≈35% of our volunteers' total mass. Similarly, estimated mass of the upper and forearms (including hands) were 4 ± 2 kg and 3 ± 1 kg, respectively. Consequently, the arms represented ≈10% of our volunteers' total mass. The

Table 1. Extremity Blood Flow

	Control	Medium	High
Extensometer/arm (ml · min^{-1} · 100 g^{-1})	3.3 ± 3.8	9.6 ± 5.2	8.6 ± 3.5
Finger flow (ml/min)	0.1 ± 0.1	0.8 ± 0.3	1.0 ± 0.3
Laser Doppler/forearm (units)	6.0 ± 2.8	13.8 ± 3.5	16.9 ± 5.4
Laser Doppler/calf (units)	0.0 ± 0.1	3.5 ± 1.8	6.2 ± 5.6
Perfusion index/toe (units)	0.2 ± 0.2	1.1 ± 0.4	1.3 ± 0.9

There were no statistically significant differences between the control periods on the "medium" and "high" days; consequently, these results were combined. All values differed significantly from control during "medium" and "high" heating. Absolute laser Doppler values on the calf and forearm should not be compared because of differences in the probes used and instrument settings.

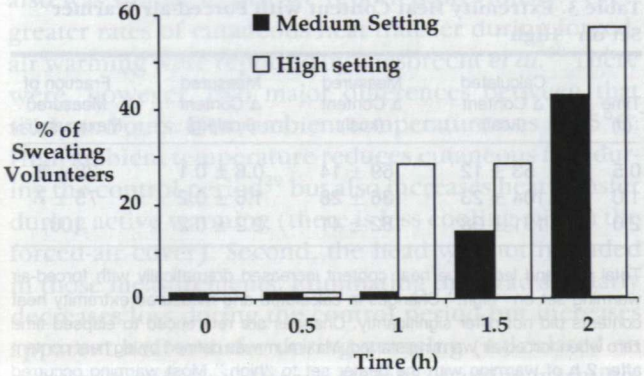

Fig. 1. Increases in core and skin temperatures during the second hour of active warming triggered sweating in about half the volunteers. Elapsed time zero identifies the beginning of forced-air warming.

parabolic regression correlation coefficients for extremity skin and tissue temperatures were generally excellent (*i.e.*, $r^2 > 0.95$).

Initial extremity heat content (at −2 elapsed hours) averaged 1393 kcal and decreased ≈50 kcal during the control period. Both arm and leg heat contents increased significantly during forced-air warming. However, leg heat content increased three times as much as that in the arms (fig. 3). With the heater set on "high," total (arm and leg) heat content increased ≈69 kcal during the first 30 min of warming and ≈136 kcal during the first hour of warming, representing 38% and

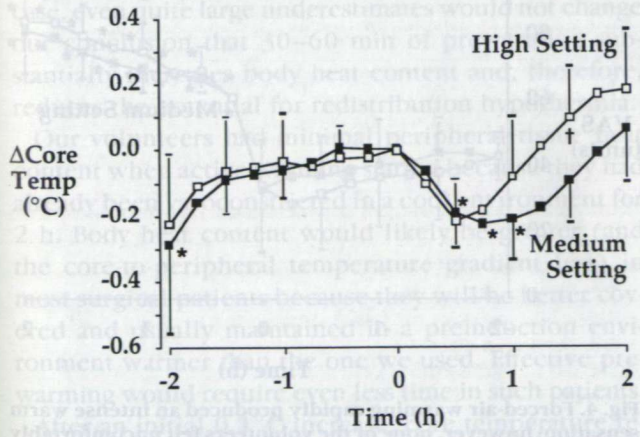

Fig. 2. Core temperatures increased ≈0.2°C during the control period, decreased ≈0.2°C during the first 30 min of forced-air heating, and then increased ≈0.3°C when the warmer was set on "medium" and ≈0.4°C when it was set to "high." Elapsed time zero identifies the beginning of forced-air warming. *Value differs significantly from time zero. †A significant difference between the heater settings.

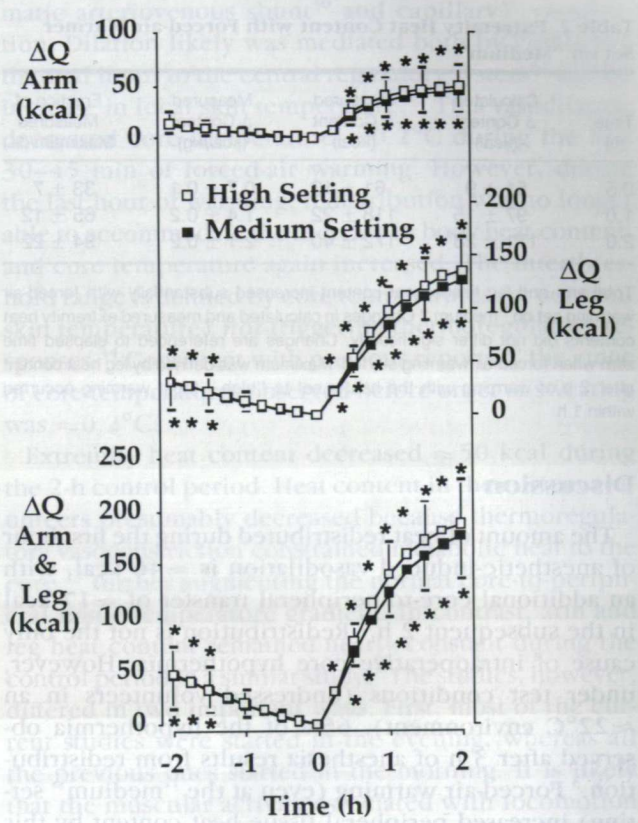

Fig. 3. Heat content of the arms and legs increased at virtually the same rate during forced-air warming with the heater set at "medium" and "high." The increase in the legs was three times as great in the arms. With the heater set on "high," total (arm and leg) heat content increased ≈69 kcal during the first 30 min of warming and ≈136 kcal during the first hour of warming, representing 38% and 75%, respectively, of the values observed after 2 h of warming. Elapsed time zero identifies the beginning of forced-air warming. *Value differs significantly from time zero.

75%, respectively, of the values observed after 2 h of warming. Extremity heat content increased only slightly faster when the forced-air warmer was set to "high" than when it was set to "medium," and maximum content was only slightly greater at the higher temperature (tables 2 and 3).

The volunteers felt slightly cool when the study started, and the cold sensation increased during the control period. Forced-air warming rapidly produced an intense warm sensation; however, none of the volunteers felt uncomfortably warm during the first hour of active heating. Subsequently, many of the volunteers were uncomfortably warm when the forced-air heater was set to "medium," and all were excessively hot when the heater was set to "high" (fig. 4).

Table 2. Extremity Heat Content with Forced-air Warmer Set on "Medium"

Time (h)	Calculated Δ Content (kcal)	Measured Δ Content (kcal)	Measured Δ Content (kcal/kg)	Fraction of Measured Maximum (%)
0.5	51 ± 9	61 ± 13	0.7 ± 0.1	33 ± 7
1.0	97 ± 15	118 ± 22	1.4 ± 0.2	65 ± 12
2.0	171 ± 23	172 ± 40	2.1 ± 0.2	94 ± 22

Total arm and leg tissue heat content increased substantially with forced-air warming set on "medium." Changes in calculated and measured extremity heat contents did not differ significantly. Changes are referenced to elapsed time zero when forced-air warming started. Maximum was defined by leg heat content after 2 h of warming with the heater set to "high." Most warming occurred within 1 h.

Table 3. Extremity Heat Content with Forced-air Warmer Set on "High"

Time (h)	Calculated Δ Content (kcal)	Measured Δ Content (kcal)	Measured Δ Content (kcal/kg)	Fraction of Measured Maximum (%)
0.5	53 ± 12	69 ± 14	0.8 ± 0.1	38 ± 5
1.0	104 ± 23	136 ± 28	1.6 ± 0.2	75 ± 7
2.0	191 ± 38	182 ± 41	2.2 ± 0.2	100

Total arm and leg tissue heat content increased dramatically with forced-air warming set on "high." Changes in calculated and measured extremity heat contents did not differ significantly. Changes are referenced to elapsed time zero when forced-air warming started. Maximum was defined by leg heat content after 2 h of warming with the heater set to "high." Most warming occurred within 1 h.

Discussion

The amount of heat redistributed during the first hour of anesthetic-induced vasodilation is ≈46 kcal, with an additional core-to-peripheral transfer of ≈17 kcal in the subsequent 2 h.[3] Redistribution is not the only cause of intraoperative core hypothermia. However, under test conditions (undressed volunteers in an ≈22°C environment), 65% of the hypothermia observed after 3 h of anesthesia results from redistribution.[3] Forced-air warming (even at the "medium" setting) increased peripheral tissue heat content by this amount within 30 min. After 1 h of warming ("high" setting), measured extremity heat content had increased 136 ± 28 kcal, explaining why prewarmed patients remain normothermic even after 3 h of major surgery.[6]

Among the factors potentially limiting efficacy of prewarming is thermoregulatory sweating provoked by high skin temperature[26] and/or a rapid increase in skin temperature.[27] There was little sweating during the first hour of prewarming, and by the end of the 2-h warming period, only about half the volunteers started to sweat. However, sweating apparently did not significantly impede transfer of heat to peripheral tissues, because extremity heat content calculated from metabolic rate, cutaneous thermal flux, and change in trunk temperature was comparable to that determined by direct measurement. More importantly, the increase in extremity heat content already was substantial by the time sweating was first detected.

All the volunteers were uncomfortably cool during the control period and appreciated forced-air warming for the first 30–60 min, even when the heater was set to "high." Subsequently, most felt excessively warm especially at the higher air temperature. However, peripheral compartment heat content was increased by clinically important amounts within 30–60 min of warming at either temperature setting. Based on these data, we predict that adequate prewarming can be administered without engendering thermal discomfort. In practice, the forced-air warmer can be set to "high" for as long as patients wish. Blower temperature[28] can be decreased as necessary to maintain a comfortable sense of warmth.

Cutaneous heat loss during the control period was similar to that we have reported at ambient temperatures near 21°C.[1] The efficacy of convective warming

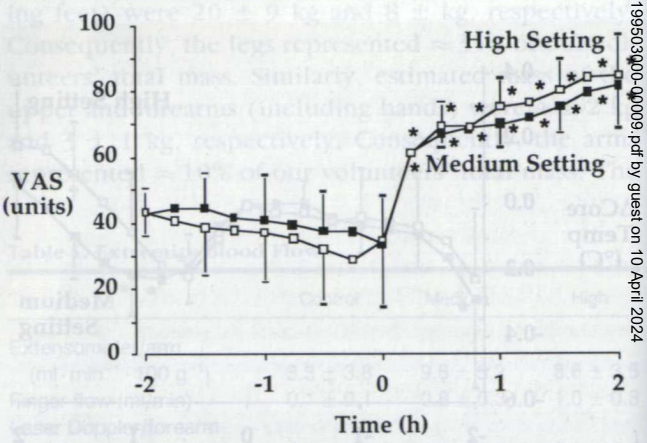

Fig. 4. Forced-air warming rapidly produced an intense warm sensation; however, none of the volunteers felt uncomfortably warm during the first hour of active heating. Thermal comfort was rated on a 100-mm visual analog scale (VAS), with zero indicating intense cold and 100 mm representing intense heat. Elapsed time zero identifies the beginning of forced-air warming. All values after induction of anesthesia differed significantly from time zero; however, there were no significant differences between the groups.

also was similar to that we reported previously.[5] Even greater rates of cutaneous heat transfer during forced-air warming were reported by Giesbrecht *et al.*[28] There were, however, two major differences between that study and ours. First, ambient temperature was ≈25°C: High ambient temperature reduces cutaneous flux during the control period[29] but also increases heat transfer during active warming (there is less cooling within the forced-air cover). Second, the head was not included in those measurements. Eliminating the head similarly decreases loss during the control period but increases apparent heat transfer during warming (calculated on a per person basis). After ≈40 min of active warming, cutaneous heat transfer gradually decreased ≈7 and ≈17 W, respectively, at each setting. Heat transfer during forced-air warming is roughly proportional to the skin-air gradient (at constant air velocity). This decrease thus simply reflects the progressive increase in skin and tissue temperature as peripheral thermal compartment heat content increased.

As previously described,[14] our direct tissue heat content estimates are limited by extrapolations from a limited number of temperature measurement sites and various assumptions of tissue homogeneity and symmetry. Measured extremity heat content increased somewhat more rapidly than did calculated arm and leg heat content. However, the values never differed significantly, and after 2 h of warming, each method provided similar estimates. It is, therefore, unlikely that the increase in peripheral compartment heat content was much less impressive than reported here. In any case, even quite large underestimates would not change our conclusion that 30–60 min of prewarming substantially increases body heat content and, therefore, reduces the potential for redistribution hypothermia.

Our volunteers had minimal peripheral tissue heat content when active warming started because they had already been vasoconstricted in a cool environment for 2 h. Body heat content would likely be greater (and the core-to-peripheral temperature gradient less) in most surgical patients because they will be better covered and usually maintained in a preinduction environment warmer than the one we used. Effective prewarming would require even less time in such patients.

After an initial 0.1°C increase, core temperature remained relatively constant during the control period, increasing only an additional 0.1°C. Nearly constant core temperatures were consistent with the relatively small decrease in mean skin temperature during this period. Active cutaneous warming then triggered dramatic arteriovenous shunt[30] and capillary[31] vasodilation. Dilation likely was mediated both by cutaneous thermal input to the central regulatory system[32] and by increase in local skin temperature.[33] This vasodilation decreased core temperature ≈0.2°C during the first 30–45 min of forced-air warming. However, during the last hour of warming, redistribution was no longer able to accommodate the increase in body heat content, and core temperature again increased. The interthreshold range is defined by core temperatures (at constant skin temperature) *not* triggering thermoregulatory responses.[34] Consistent with previous reports,[35] the range of core temperatures observed before onset of sweating was ≈0.2°C.

Extremity heat content decreased ≈50 kcal during the 2-h control period. Heat content in the current volunteers presumably decreased because thermoregulatory vasoconstriction constrained metabolic heat to the core,[14] further augmenting the normal core-to-peripheral tissue temperature gradient. In contrast, arm and leg heat content remained nearly constant during the control period in a similar study.[3] The studies, however, differed in two important ways. First, most of the current studies were started in the evening, whereas all the previous ones started in the morning. It is likely that the muscular activity associated with locomotion increases leg content over the course of the day. Second, the current investigation was conducted in March, when San Francisco was considerably warmer than in January, when most of the previous studies were done. It is thus likely that vasoconstriction was already present in volunteers in the previous studies when they arrived in the laboratory, and they thus had nearly minimal extremity temperature and heat content. Under these conditions, heat content would not decrease further during the control exposure. Consistent with this theory, initial tissue heat content was 16% (189 kcal) greater in the March volunteers than in the previous ones. These differences in the response to a standard set of laboratory conditions illustrate the importance of initial conditions and sufficiently long control periods in thermoregulatory studies.

Acute inhibition of tonic thermoregulatory vasoconstriction initiates a core-to-peripheral redistribution of body heat that is the major cause of hypothermia during the first hour of anesthesia.[1] Although arms are considerably smaller than legs, both contribute comparably to redistribution hypothermia.[3] During active warming, however, the larger leg mass absorbed considerably more heat than the arms. These results suggest that re-

distribution accesses only a fraction of the peripheral thermal compartment's potential capacity.

To minimize volunteer risk, we did not induce general anesthesia and specifically quantify redistribution hypothermia. However, the mechanism by which redistribution reduces core temperature,[1] the magnitude of heat flow,[3] and the efficacy of prewarming[4,6] are all well established. We thus can predict with reasonable certainty that redistribution hypothermia in surgical patients will be markedly reduced by 30 min of forced-air warming and virtually eliminated if active heating is maintained for an hour.

We only studied men; the specific amounts of heat absorbed during prewarming would differ somewhat in women. However, the amount required also would be less because women usually are smaller than men. It is thus likely that our general conclusions would apply comparably to women.

In summary, half the volunteers started sweating during the second hour of forced-air warming. None of the volunteers felt uncomfortably warm during the first hour of heating, but many subsequently did. With the heater set on "high," arm and leg heat content increased 69 kcal during the first 30 min of warming and 136 kcal during the first hour of warming, representing 38% and 75%, respectively, of the values observed after 2 h of warming. The increase was only slightly less when the heater was set to "medium." Neither sweating nor thermal discomfort limited heat transfer during the first hour of warming. Thirty minutes of forced-air warming increased peripheral tissue heat content by more than the amount typically redistributed from core to peripheral tissues. The large increase in extremity heat content during prewarming explains why prewarmed patients remain normothermic even after several hours of major surgery.

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