

## Thermoregulatory Vasoconstriction Does Not Impede Core Warming during Cutaneous Heating

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**Background:** Although forced-air warming rapidly increases intraoperative core temperatures, it is reportedly ineffective postoperatively. A major difference between these two periods is that arteriovenous shunts are usually dilated during surgery, whereas vasoconstriction is uniform in hypothermic postoperative patients. Vasoconstriction may decrease efficacy of warming because its major physiologic purposes are to reduce cutaneous heat transfer and restrict heat transfer between the two thermal compartments. Accordingly, we tested the hypothesis that thermoregulatory vasoconstriction decreases cutaneous transfer of applied heat and restricts peripheral-to-core flow of heat, thereby delaying and reducing the increase in core temperature.

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# Mort TC, Rintel TD, Altman F: Shivering in the cardiac patient: Evaluation of the Bair Hugger warming system (abstract). *ANESTHESIOLOGY* 1990; 73:A239.

**Methods:** Eight healthy male volunteers anesthetized with propofol and isoflurane were studied. Volunteers were allowed to cool passively until core temperature reached 33°C. On one randomly assigned day, the isoflurane concentration was reduced, to provoke thermoregulatory arteriovenous shunt vasoconstriction; on the other study day, a sufficient amount of isoflurane was administered to prevent vasoconstriction. On each day, forced-air warming was then applied for 2 h. Peripheral (arm and leg) tissue heat contents were determined from 19 intramuscular needle thermocouples, 10 skin temperatures, and "deep" foot temperature. Core (trunk and head) heat content was determined from core temperature, assuming a uniform compartmental distribution. Time-dependent changes in peripheral and core tissue heat contents were evaluated using linear regression. Differences between the vasoconstriction and vasodilation study days, and between the peripheral and core compartments, were evaluated using two-tailed, paired *t* tests. Data are presented as means  $\pm$  SD; *P* < 0.01 was considered statistically significant.

**Results:** Cutaneous heat transfer was similar during vasoconstriction and vasodilation. Forced-air warming increased peripheral tissue heat content comparably when the volunteers were vasodilated and vasoconstricted:  $48 \pm 7$  versus  $53 \pm 10$  kcal/h. Core compartment tissue heat content increased similarly when the volunteers were vasodilated and vasoconstricted:  $51 \pm 8$  versus  $44 \pm 11$  kcal/h. Combining the two study days, the increase in peripheral and core heat contents did not differ significantly:  $51 \pm 8$  versus  $48 \pm 10$  kcal/h, respectively. Core temperature increased at essentially the same rate when the volunteers remained vasodilated ( $1.3^\circ\text{C/h}$ ) as when they were vasoconstricted ( $1.2^\circ\text{C/h}$ ).

**Conclusions:** The authors failed to confirm their hypothesis that thermoregulatory vasoconstriction decreases cutaneous transfer of applied heat and restricts peripheral-to-core flow of heat in anesthetized subjects. The reported difference between intraoperative and postoperative rewarming efficacy may result from nonthermoregulatory anesthetic-induced vasodilation. (Key words: Hypothermia: postoperative. Metabolism: metabolic rate. Temperature: core; heat balance; heat flux; skin; tissue. Thermoregulation: vasoconstriction.)

FORCED-AIR warming rapidly increases intraoperative core temperatures, with temperatures typically differing  $\approx 0.75^\circ\text{C/h}$  compared to those of unwarmed control patients.<sup>1-3</sup> In contrast, forced-air warming is reportedly ineffective<sup>4,5</sup> or minimally effective<sup>6</sup> postoperatively, although available skin surface area—



and thus potential heat transfer—is usually greater. A major difference between these two periods is that during surgery, arteriovenous shunts are usually dilated whereas vasoconstriction is routine in hypothermic postoperative patients.<sup>7</sup>

Thermoregulatory vasomotion influences cutaneous heat transfer, flow of heat between core and peripheral tissues, and internal distribution of body heat. For example, thermoregulatory vasoconstriction decreases cutaneous heat loss in unanesthetized<sup>8</sup> and anesthetized<sup>9</sup> subjects. Similarly, core hypothermia after induction of general<sup>10</sup> and epidural<sup>11</sup> anesthesia results largely from anesthetic-induced inhibition of tonic thermoregulatory vasoconstriction and subsequent core-to-peripheral redistribution of body heat. Onset of vasoconstriction in patients becoming sufficiently hypothermic also alters heat balance, in this case by decreasing cutaneous heat loss and constraining metabolic heat to the core thermal compartment.<sup>12</sup>

Major purposes of thermoregulatory vasoconstriction thus include limitation of cutaneous heat transfer and restriction of heat transfer between peripheral and core thermal compartments. Decreased flow of heat across the skin surface allows humans to retain metabolic heat and protect core temperature.<sup>8,9</sup> Similarly, functional separation of the two compartments allows peripheral tissues to act as a thermal buffer, absorbing or dissipating heat, as necessary, to maintain core temperature.<sup>12</sup> Consistent with the importance of thermoregulatory vasomotion, induction of therapeutic hypothermia is more difficult in patients demonstrating arteriovenous shunt vasoconstriction than in those remaining vasodilated.<sup>13</sup> Available data thus suggest that the efficacy of forced-air warming will be reduced by thermoregulatory vasoconstriction. Accordingly, we tested the hypothesis that thermoregulatory vasoconstriction decreases cutaneous transfer of applied heat and restricts peripheral-to-core flow of heat, thereby delaying and reducing the increase in core temperature.

## Methods

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

istics included: age  $32 \pm 7$  yr, height  $177 \pm 3$  cm, weight  $73 \pm 9$  kg, and body fat  $18 \pm 2\%$ .<sup>14</sup>

### Protocol

Volunteers fasted for the 8 hours preceding the studies, which started at approximately 8:30 AM. Throughout the study, minimally clothed volunteers reclined on an operating room table set in chaise lounge position. An intravenous cannula was inserted in the right antecubital fossa, and lactated Ringer's solution warmed to  $37^\circ\text{C}$  was infused at a rate of 100 ml/h.

Anesthesia was induced by intravenous administration of 2 mg/kg propofol, 2 mg midazolam, and 0.1 mg/kg vecuronium bromide. The volunteers' tracheas were intubated and mechanical ventilation was adjusted to maintain end-tidal partial pressure of carbon dioxide near 35 mmHg. Anesthesia was maintained with propofol ( $\approx 3 \text{ mg} \cdot \text{kg}^{-1} \cdot \text{h}^{-1}$ ) and isoflurane ( $\approx 1\%$ ) in 30% oxygen and air. An infusion of vecuronium was adjusted to maintain one mechanical twitch in response to supramaximal train-of-four electrical stimulation of the ulnar nerve at the wrist.

Volunteers were allowed to cool passively until core temperature reached  $33^\circ\text{C}$ . On one randomly assigned day, the isoflurane concentration was reduced to  $\approx 0.3\%$  to provoke thermoregulatory arteriovenous shunt vasoconstriction (see later), which was maintained for 1 h. On the other study day, sufficient isoflurane was administered to prevent vasoconstriction ( $\approx 0.7\%$ ). The volunteers were then warmed on both study days using forced-air (Model 200 set on "high," Cover #300, Augustine Medical, Eden Prairie, MN). To prevent locally mediated vasodilation,<sup>15-17</sup> the hands and feet were protected from direct heating. Active warming was continued for 2 h. Subsequently, neuromuscular block was antagonized, anesthesia discontinued, and the trachea extubated.

### Measurements

End-tidal isoflurane and carbon dioxide concentrations were monitored using a Capnomac Ultima (Datex Medical Instruments, Tewksbury, MA). Blood pressures, arterial saturation, and heart rates were measured using monitors incorporated into an Ohmeda Modulus CD anesthesia machine (Ohmeda, Salt Lake City, UT).

Energy expenditure, derived from oxygen consumption and carbon dioxide production, was measured using a calibrated metabolic monitor (Deltatrac, Sensor-Medics, Yorba Linda, CA). Measurements were averaged over 5-min intervals and recorded every 5 min. Area-



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weighted heat flux and temperatures from 15 skin-surface sites were measured using thermal flux transducers (Concept Engineering, Old Saybrook, CT).<sup>10</sup> As in previous studies, measured cutaneous heat loss was augmented by 10% to account for insensible transcutaneous evaporative loss<sup>18</sup> and 3% to compensate 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.<sup>19</sup> We defined flux as positive when heat traversed skin to the environment.

Core temperatures were recorded from the distal quarter of the esophagus. All temperatures were recorded using Mon-a-Therm thermocouples (Mallinckrodt Anesthesiology Products, St. Louis, MO) or probes incorporated into thermal flux transducers. Temperatures were recorded from thermocouples connected to calibrated Iso-Thermex 16-channel electronic thermometers having an accuracy of 0.1°C and a precision of 0.01°C (Columbus Instruments International, Columbus, OH). Temperatures and thermal flux were measured at 1-s intervals, then averaged and recorded every 5 min by a data acquisition system.

Right index fingertip blood flows were quantified using volume plethysmography.<sup>20</sup> As in previous investigations, we considered flows <0.25 ml/min to indicate intense vasoconstriction whereas flows exceeding 1 ml/min identified vasodilation.<sup>21</sup> Vascular tone also was evaluated on the right second toe using the perfusion index, which is derived from absorption of two infrared wavelengths.<sup>22</sup> Values of the perfusion index <0.5 indicate vasoconstriction whereas values near 2 identify vasodilation. Both measures of flow were recorded at 5-min intervals.

Arm and leg tissue heat contents were determined from 19 intramuscular needle thermocouples, ten skin temperatures, and "deep" foot temperature (Core-Temp, Terumo Medical, Tokyo, Japan).<sup>23,24</sup> 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 leg tissue temperature distributions were calculated using fourth-order regressions. In contrast, arm and posterior leg segment temperatures were evaluated using parabolic regressions. The resulting equations were integrated over volume to provide tissue heat content. We previously described the details and limitations of these measurements.<sup>10,25</sup>

### Data Analysis

The beginning of forced-air warming was designated elapsed time zero. Mean body temperatures were calculated from the weighted average of peripheral tissue and core temperatures. As in previous investigations,<sup>10,11</sup> changes in whole-body heat content on each study day were calculated using two independent methods: (1) time integral of metabolic heat production minus cutaneous heat loss, and, (2) sum of extremity and core tissue heat contents. The effect of increasing mean and core body temperatures on metabolic rate were evaluated using linear regression, incorporating data between -1 and +2 elapsed hours from both study days.

Core rewarming rates, and changes in peripheral and core heat contents, were evaluated using linear regression incorporating values between 0.5 and 2.0 elapsed hours. Individual regression slopes for each compartment on each treatment day were compared using paired, two-tailed *t* tests. The combined rates of peripheral and core compartment warming were similarly compared. Time-dependent differences within treatment groups were evaluated using repeated-measures analysis of variance with Dunnett's test for comparison to time zero. Differences between the vasoconstriction and vasodilation study days were evaluated using two-tailed, paired *t* tests. Results are expressed as means  $\pm$  standard deviations. Differences in tissue temperatures and heat content were considered statistically significant when  $P < 0.01$ ; differences in potential confounding factors such as anesthetic concentration and hemodynamic responses were considered significant when  $P < 0.05$ .

### Results

End-tidal partial pressure of carbon dioxide and ambient temperatures and humidity were comparable on the two treatment days. By design, end-tidal isoflurane concentrations were significantly less on the vasoconstriction day; consequently, mean arterial blood pressures were significantly greater (table 1). The arms weighed  $8 \pm 2$  kg and the legs weighed  $19 \pm 3$  kg; consequently, peripheral tissues constituted  $\approx 36\%$  of the volunteers' total body mass. Changes in body heat content were virtually identical when calculated as the difference in heat production and loss or as the sum of peripheral and core tissue heat contents. Increasing mean body temperature increased metabolic rate  $7 \pm$



Table 1. Anesthetic, Hemodynamic, and Environmental Data

	Vasodilation	Vasoconstriction
End-tidal [isoflurane] (%)	0.68 ± 0.15	0.26 ± 0.04*
Heart rate (beats/min)	61 ± 8	61 ± 8
Mean arterial blood pressure (mmHg)	70 ± 16	90 ± 8*
Ambient temperature (°C)	22.7 ± 0.8	22.7 ± 0.9
Ambient humidity (%)	42 ± 2	40 ± 2

Data were averaged within individual treatments over the period from 0 to 2 elapsed hours and then averaged among volunteers. Results are presented as means ± SD.

\*  $P < 0.05$ .

4%/°C ( $r^2 = 0.6 \pm 0.3$ ); increasing core temperature increased metabolic rate  $8 \pm 4\%/^{\circ}\text{C}$  ( $r^2 = 0.6 \pm 0.3$ ).

Volume plethysmography on the index finger and the perfusion index on the toe both indicated that vaso-

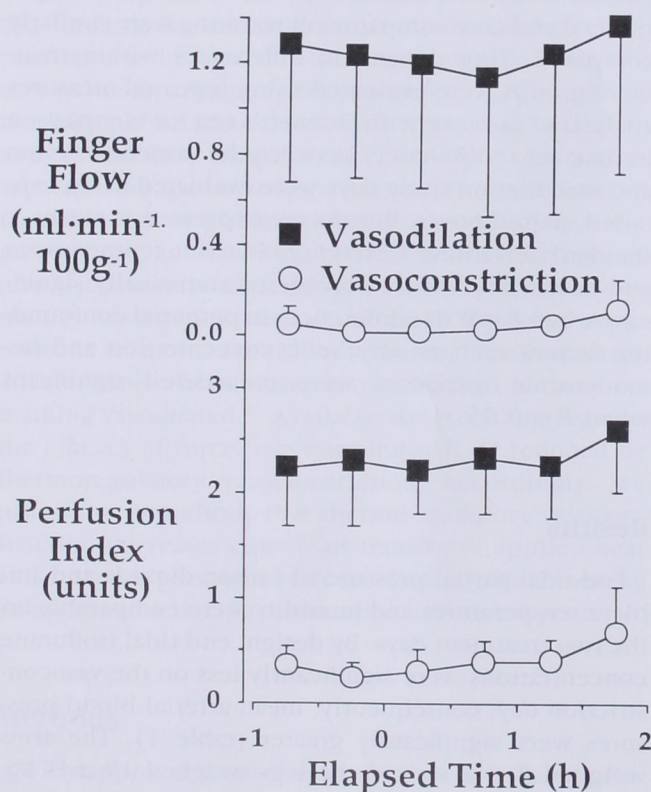


Fig. 1. Volume plethysmography on the index finger and the perfusion index on the toe both indicated that vasoconstriction was maintained on one study day whereas vasodilation was maintained on the other. Flow differences on the two treatment days differed significantly at every time point. Results are presented as means ± SD. (Error bars are not displayed when the SD was smaller than the mean value symbol.)

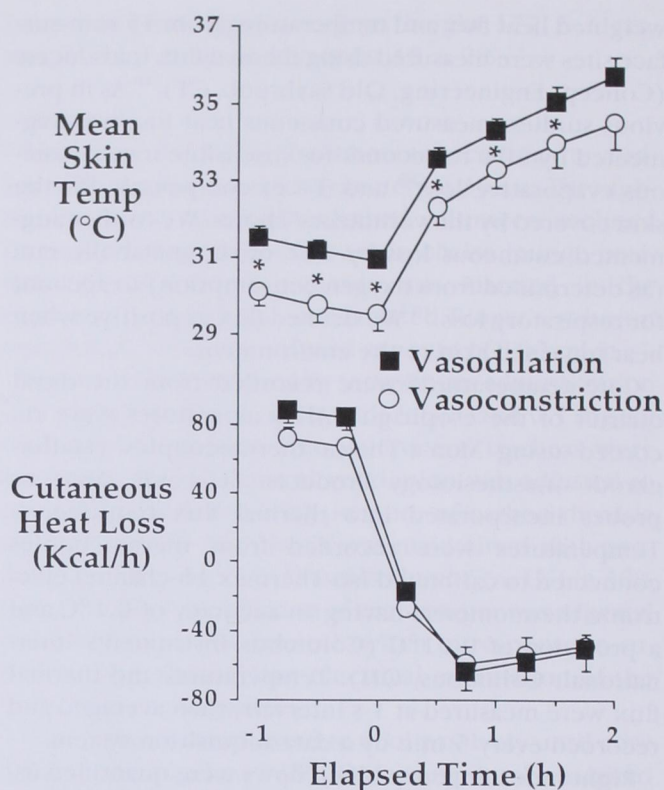


Fig. 2. Mean skin temperatures differed significantly on the two study days, whereas cutaneous heat flux was comparable. All values after elapsed time zero differed significantly from time zero. Results are presented as means ± SD; \* $P < 0.01$  compared to vasodilation.

constriction was maintained on one study day whereas vasodilation was maintained on the other (fig. 1). Mean skin temperatures differed significantly on the two study days. This difference resulting largely because temperature of the distal extremities (which were protected from direct heating) were significantly greater when the volunteers were vasodilated. Cutaneous heat flux was nonetheless similar on the two study days (fig. 2).

Peripheral (arm and leg) tissue temperatures at elapsed time zero were  $33.5 \pm 0.7^{\circ}\text{C}$  during vasodilation and  $31.2 \pm 1.0^{\circ}\text{C}$  during vasoconstriction ( $P = 0.02$ ). Average peripheral compartment tissue temperatures thus differed by  $2.3^{\circ}\text{C}$  at elapsed time zero. Peripheral heat contents at elapsed time zero were  $797 \pm 136$  kcal during vasodilation and  $743 \pm 131$  kcal during vasoconstriction ( $P = 0.02$ ). Warming immediately increased peripheral tissue heat content, with the increase being comparable when the volunteers were vasodilated and vasoconstricted:  $48 \pm 7$  versus  $53 \pm 10$  kcal/h ( $P = 0.1$ ). The increase in core com-



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partment heat content was delayed only 20 min, and the delay was similar during vasoconstriction and vasodilation. Core compartment tissue heat content increased similarly when the volunteers were vasodilated and vasoconstricted:  $51 \pm 8$  versus  $44 \pm 11$  kcal/h. This difference was neither clinically nor statistically significant ( $P = 0.1$ ), being only 10 kcal after 2 h (this value is less than might be expected from the regression slopes because they exclude the first 0.5 h during which core heat content increased only slightly). Combining the two study days, peripheral and core heat contents increased at essentially the same rate:  $51 \pm 8$  versus  $48 \pm 10$  kcal/h ( $P = 0.4$ ; fig. 3).

Core temperature decreased  $\approx 0.9^\circ\text{C}$  from  $-1$  to 0 elapsed hours during vasodilation. In contrast, core temperature was virtually constant during this period with vasoconstriction. Forced-air warming was associated with  $\approx 10$  min of afterdrop ( $\approx 0.1^\circ\text{C}$ ) during vasodilation, but core temperature began to increase after 20 min of warming. There was no afterdrop when the volunteers were vasoconstricted, but core temperature elevation was nonetheless delayed  $\approx 20$  min. Subsequently, core temperature increased only slightly faster when the volunteers remained vasodilated ( $1.3^\circ\text{C/h}$ ) than when they were vasoconstricted ( $1.2^\circ\text{C/h}$ ). There was no statistically significant ( $P = 0.1$ ) or clinically important difference in the rewarming rates, with distal esophageal temperatures differing by only  $\approx 0.3^\circ\text{C}$  after 2 h of active warming (fig. 4).

## Discussion

Vasoconstriction, once triggered, decreases cutaneous heat loss and constrains metabolic heat to the core thermal compartment.<sup>12</sup> Consequently, core temperatures, before application of forced-air warming, were virtually constant when the volunteers were vasoconstricted whereas they continued to decrease on the vasodilated day. Because vasoconstriction alters body heat content and distribution, it proved impossible to start warming under exactly the same thermal conditions on each day. We elected to maintain a similar environment and began warming at the same core temperature on each study day. A consequence of this decision was that peripheral tissue temperature and heat content were greater at elapsed time zero when the volunteers were vasodilated than during vasoconstriction.

The term afterdrop is defined by new or continued core cooling after initiation of surface warming. It ap-

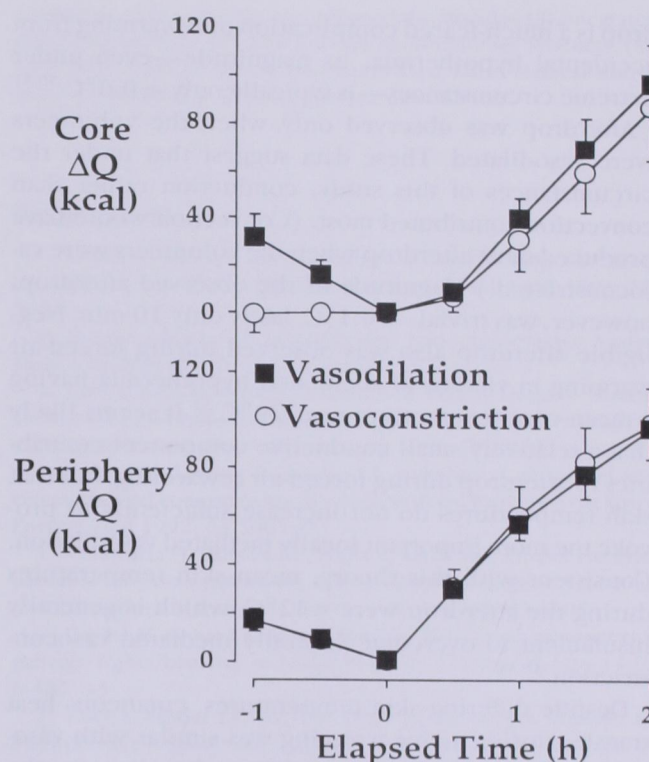


Fig. 3. Peripheral (arm and leg) heat contents at elapsed time zero were  $797 \pm 136$  kcal during vasodilation and  $743 \pm 131$  kcal during vasoconstriction ( $P = 0.02$ ). Warming immediately increased peripheral tissue heat content, with the increase being comparable when the volunteers were vasodilated and vasoconstricted:  $48 \pm 0.2$  versus  $53 \pm 10$  kcal/h ( $P = 0.1$ ). The increase in core compartment heat content was delayed only 20 min, and the delay was similar during vasoconstriction and vasodilation. Core compartment tissue heat content increased only slightly faster when the volunteers were vasodilated:  $51 \pm 8$  versus  $44 \pm 11$  kcal/h. The difference, however, was not clinically important or statistically significant ( $P = 0.1$ ), being only 10 kcal after 2 h. Combining the two study days, peripheral and core heat contents increased at essentially the same rate:  $51 \pm 8$  versus  $48 \pm 10$  kcal/h ( $P = 0.4$ ). Changes in tissue heat contents ( $\Delta Q$ ) are relative to elapsed time zero. All values after elapsed time zero differed significantly from time zero. Results are presented as means  $\pm$  SD.

parently results from both conduction and convection.<sup>26,27</sup> Conduction contributes when the core continues to lose heat to cooler surrounding tissues, even after surface warming has begun; this is a passive phenomenon and can be demonstrated in a bag of gelatin and leg of beef (without an attached cow).<sup>28</sup> In contrast, convection contributes when aggressive surface warming causes locally mediated vasodilation despite continued core hypothermia. This local dilation allows peripheral-to-core flow of blood cooled by surface tissues, and consequent core cooling.<sup>29</sup> Although after-



drop is a much-feared complication of rewarming from accidental hypothermia, its magnitude—even under extreme circumstances—is typically only  $\approx 0.6^{\circ}\text{C}$ .<sup>30,31</sup>

Afterdrop was observed only when the volunteers were vasodilated. These data suggest that under the circumstances of this study, conduction rather than convection contributed most. (Convection would have produced most afterdrop when the volunteers were vasoconstricted.) Magnitude of the observed afterdrop, however, was trivial:  $\approx 0.1^{\circ}\text{C}$ , lastly only 10 min. Negligible afterdrop also was observed during forced-air warming in victims of accidental hypothermia having a mean core temperature near  $29^{\circ}\text{C}$ .<sup>32</sup> It seems likely that a relatively small conductive component contributes to afterdrop during forced-air rewarming, but that skin temperatures do not increase sufficiently to provoke the more important locally mediated vasodilation. Consistent with this theory, mean skin temperatures during the afterdrop were  $<32^{\circ}\text{C}$ , which is generally insufficient to overcome centrally mediated vasoconstriction.<sup>33,34</sup>

Despite differing skin temperatures, cutaneous heat transfer during active warming was similar with vasodilation and vasoconstriction. These data disprove the first part of our hypothesis, that thermoregulatory vasoconstriction decreases cutaneous transfer of applied heat. Peripheral tissue heat content increased comparably when the volunteers were vasodilated and vasoconstricted; core compartment tissue heat content also increased at virtually the same rate when the volunteers were vasodilated and vasoconstricted. The increase in core compartment heat content was delayed only 20 min, and the delay was similar during vasoconstriction and vasodilation. Peripheral heat content increased at essentially the same rate as core heat content, indicating that vasoconstriction did not much impede peripheral-to-core transfer of cutaneously applied heat. We thus also failed to confirm the second portion of our hypothesis, that thermoregulatory vasoconstriction restricts peripheral-to-core flow of heat.

A minimal effect of vasomotor status on active warming is consistent with the results of a recent study showing that administration of sodium nitroprusside sufficient to decrease systolic arterial blood pressure by 10% did not speed intraoperative core rewarming.<sup>35</sup> However, our results do not support the theory that minimal efficacy of postoperative forced-air warming results from vasoconstriction decreasing peripheral-to-core heat transfer. The most likely remaining explanation for the reported efficacy difference between

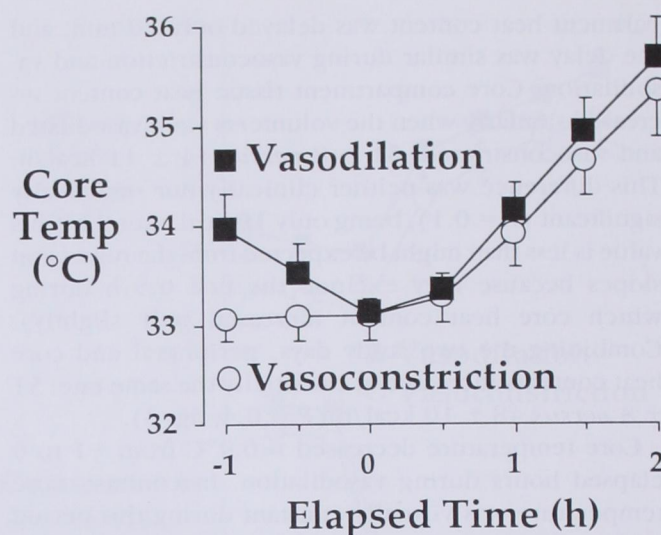


Fig. 4. Core temperature decreased  $\approx 0.9^{\circ}\text{C}$  from  $-1$  to  $0$  elapsed hours when the volunteers were vasodilated. In contrast, core temperature was virtually constant during this period when the volunteers were vasoconstricted. Forced-air warming was associated with  $\approx 10$  min of afterdrop ( $\approx 0.1^{\circ}\text{C}$ ) during vasodilation, but core temperature began to increase after 20 min of warming. There was no afterdrop when the volunteers were vasoconstricted, but core temperature nonetheless required  $\approx 20$  min to begin increasing. Subsequently, core temperature increased only slightly faster when the volunteers remained vasodilated ( $1.3^{\circ}\text{C/h}$ ) than when they were vasoconstricted ( $1.2^{\circ}\text{C/h}$ ;  $P = 0.1$ ). The difference in the rewarming rates was not clinically important, with core temperatures differing by only  $\approx 0.3^{\circ}\text{C}$  after 2 h of active warming. All values after elapsed time zero differed significantly from time zero. Results are presented as means  $\pm$  SD.

intraoperative<sup>1-3</sup> and postoperative<sup>4-6</sup> warming is an effect of anesthesia *per se*. Specifically, that anesthesia may produce a nonthermoregulatory vasodilation sufficient to markedly increase peripheral-to-core heat transfer. Consistent with this possibility, volatile anesthetics are known to increase muscle blood flow. However, it remains to be determined whether anesthetic-induced vasodilation is appropriately distributed and of sufficient magnitude to explain the reported intraoperative *versus* postoperative efficacy difference.

Relatively little oxygen was consumed by our volunteers, as would be expected because general anesthesia *per se* decreases metabolic rate 20–30%.<sup>36</sup> Increasing body temperature increased metabolic rate  $\approx 7\text{--}8\%/^{\circ}\text{C}$ . This increase is similar to those reported previously and does not result from nonshivering thermogenesis.<sup>31</sup> Metabolic rates were comparable during vasoconstriction and vasodilation, however, and thus did not contribute to the (slightly) different core rewarming rates on the two study days.



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We previously described the substantial limitations of our heat balance and tissue temperature measurements.<sup>25,11</sup> Under the circumstances of this study, however, changes in body heat content were virtually identical when independently calculated as the difference in heat production and loss or as the sum of peripheral and core tissue heat contents, suggesting that neither method contained major errors. But more importantly, the major outcome of our study was the change in core temperature—a result not depending on heat balance or peripheral tissue temperatures. Because forced-air warming increased core temperatures comparably on the two treatment days, we can conclude that there was no clinically important effect of vasoconstriction on heat transfer rates.

To avoid locally mediated vasodilation,<sup>15-17</sup> we protected the hands and feet from direct warming. Had we not taken this precaution, it would have proved difficult to maintain vasoconstriction during warming. Total cutaneous heat transfer and tissue warming rates would have been greater on both study days had we directly warmed the distal extremities simply because more surface would have been available. Furthermore, it is likely that heat transfer through the distal extremities would have been greater when the volunteers were vasodilated. However, increased local heat transfer does not appear to be the most important effect of arteriovenous shunt vasodilation; instead, increased heat transfer apparently results largely from augmented tissue blood flow in the remainder of the extremity (which was actively warmed). It is thus unlikely that directly warming the hands and feet would have produced a clinically important increase in the core rewarming rate on the vasodilated study day. Although we do not report capillary blood flow in this investigation, we have previously demonstrated that, except during sweating,<sup>37</sup> nonshunt flow is minimally altered by thermoregulatory status during anesthesia.<sup>9</sup>

In summary, cutaneous heat transfer in anesthetized subjects was similar during vasoconstriction and vasodilation. Furthermore, peripheral heat content increased at nearly the same rate as core heat content. Consequently, core temperature increased at essentially the same rate when the volunteers remained vasodilated (1.3°C/h) as when they were vasoconstricted (1.2°C/h). We therefore failed to confirm our hypothesis that thermoregulatory vasoconstriction decreases cutaneous transfer of applied heat and restricts peripheral-to-core flow of heat.

Ohmeda (Salt Lake City, Utah) loaned the Modulus CD integrated anesthesia machine and Mallinckrodt Anesthesiology Products, (St. Louis, MO) donated the thermocouples used. Datex Medical Instruments (Tewksbury, MA) loaned a Capnomac Ultima.

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