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Thermoregulatory Vasoconstriction Decreases Cutaneous Heat Loss

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To determine the extent to which thermoregulatory vasoconstriction decreases heat loss to the environment, we measured regional heat flux, average skin temperature, and tympanic membrane temperature before and after thermoregulatory vasoconstriction in five minimally clothed volunteers maintained in a 30.8 ± 0.1° C environment. Thermoregulatory vasoconstriction was induced by central venous infusion of cooled fluid. Peripheral cutaneous blood flow was evaluated with venous-occlusion volume plethysmography and skin-surface temperature gradients. Laser Doppler flowmetry was used to measure vasoconstriction in centrally located skin. This model mimics the common clinical situation in which patients in a warm environment are centrally cooled by administration of cold intravenous fluids or by lavage of internal cavities with cold fluids. Tympanic membrane temperature decreased 1.5 ± 0.3° C in the first 15 min after the cold fluid infusion was started and remained ≈1° C below control values during the rest of the study. Average skin-surface temperature decreased slowly to ≈0.7° C below control. Flow in capillaries of centrally distributed skin, determined with laser Doppler flowmetry, decreased only ≈40%. Total heat flux, and flux from the arms and legs decreased ≈25% (15.5 ± 0.3 W). Heat loss from the trunk and head decreased only 17%, whereas, loss from the hands and feet (10.5% of the body surface area) decreased ≈50%. All measured values decreased significantly following vasoconstriction ($P < 0.01$). Therefore, thermoregulatory vasoconstriction in a thermoneutral environment appears to decrease cutaneous loss of metabolic heat ≈25%. (Key words: Brain: hypothalamus. Hypothermia. Temperature, measurement: skin, tympanic membrane. Temperature, regulation: set-point, threshold. Heat, measurement: thermal flux transducers Vasoconstriction, thermoregulatory.)

THERMOREGULATORY VASOCONSTRICTION decreases heat loss to the environment by reducing blood flow in cutaneous arteriovenous shunts and capillaries.[1–3] Whereas decreased flow through anatomic shunts lowers skin temperature and heat loss in only a small fraction of the skin surface (for instance, the hands, feet, or nose), capillary constriction may affect temperature and heat loss over the entire body.[3–6] Total cutaneous heat loss cannot be deduced from fluctuations in central temperature alone, because central temperature is influenced by shivering,[7] nonshivering thermogenesis,[8,9] and redistribution of heat within the body.[10] Therefore, we directly measured thermal flux** across the skin in unanesthetized volunteers to determine the distribution of cutaneous heat loss and the extent to which thermoregulatory vasoconstriction decreases flux. To assure that we achieved near-maximal vasoconstriction, we documented shivering (which normally is preceded by thermoregulatory vasoconstriction) in each volunteer.[11]

Methods

With approval from the University of California, San Francisco, Committee on Human Research and with written informed consent from the volunteers, we evaluated cutaneous thermoregulatory vasoconstriction in one woman and four men aged 21–35 yr. None was obese, was on medication, or had a history of smoking, thyroid disease, dysautonomia, or Raynaud's syndrome. Volunteers refrained from coffee, tea, and food during the 8 h preceding study. Each study started near 9:30 AM.

All volunteers were minimally clothed, and reclined on their backs on a standard operating room table (5-cm-thick foam mattress). Ambient temperature was maintained at ≈30.8° C and relative humidity near 35%. A 16-G catheter was placed into the superior vena cava *via* the internal jugular vein by the standard technique. After 30 min of control measurements, lactated Ringer's solution at ≈3° C was infused into the central catheter at 1.8 $ml \cdot kg^{-1} \cdot min^{-1}$ for 15 min, and then at 0.8 $ml \cdot kg^{-1} \cdot min^{-1}$ for an additional 45 min. Ringer's solution was cooled by passing it through an aluminum cardiopulmonary bypass heat exchanger immersed in an ice, water, and salt slurry.

Heat flux from ten skin-surface sites was measured in W/m^2 with the use of thermal flux transducers (Concept Engineering, Old Saybrook, CT). These values were converted into watts per site by multiplying by the calculated body surface area (area $[m^2]$ = $weight^{0.425}$ [kg] · $height^{0.725}$ [cm] · 0.007184) of each volunteer and assigning the following regional percentages: head 6%, up-

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** Net transfer of heat from an organism/object to the environment.

per arms 9%, forearms 6%, hands 4.5%, back 19%, chest 9.5%, abdomen 9.5%, thigh 19%, calves 11.5%, and feet 6%.[12] (The basal metabolic rate in adult humans is $\approx$ 100 W, 1 W = 0.86 kcal/h, and the specific heat of humans is $\approx$ 0.83 kcal $\cdot$ kg^{-1} $\cdot$ ° C^{-1}.[13])

The transducer measuring heat loss from the head was placed in the middle of the forehead; others were placed in the center of each anatomic area. The transducers were 25-mm-diameter circles that were firmly attached to the skin surface with a ring of thin, double-sided adhesive film of trivial thermal resistance. All transducers (except that between the back and mattress) were exposed to ambient air during the study. We defined flux as positive when heat traversed skin to the environment. Output from the transducers (0–5 mV) was amplified 2,000-fold with the use of a simple laboratory-built device.

Thermal flux transducers measure total heat loss (or gain) *via* radiation, conduction, and convection; they do not detect evaporative losses. (However, sweating would not be expected in a thermoneutral environment, and none was observed during the study.) These transducers, in effect, compare temperatures on each side of a known thermal resistance. They actually are calibrated thermopiles that produce a voltage proportional to flux. Their physical properties include an emissivity of 0.93 (similar to that of human skin) and thermal conductivity of 0.294 W $\cdot$ ° C^{-1} $\cdot$ m^{-1}, which allows rapid detection of physiologic changes in flux. The transducers are individually calibrated by the manufacturer and have an accuracy of 5%, but a precision near 1%. Estimates of total body heat loss obtained using thermal flux transducers are similar to values measured by direct calorimetry.[14]

Temperatures were monitored with Mon-a-Therm® (St. Louis, MO) tympanic membrane and skin-surface thermocouple probes connected to Mallinckrodt® model 8700 (St. Louis, MO) two-channel electronic thermometers with analog output. The manufacturer specifies that these thermometers have an accuracy of $\approx$0.1° C; extensive calibrations in our laboratory have confirmed this value. Thermocouple probes were placed at the central venous catheter insertion site, in contact with the tympanic membrane, and under each thermal flux transducer.

Analog data from the thermometers and heat flux transducers were acquired with an electrically isolated Macintosh® II computer (Apple, Inc., Cupertino, CA) equipped with two NB-MIO-16L® 16-channel analog–digital converters (National Instruments, Inc., Austin, TX). Data were digitized asynchronously at 4 Hz in 48-s epochs and individually scaled to degrees Celsius and watts per site using individual first- or second-order corrections and body surface area estimated from each volunteer's height and weight. The results were averaged, displayed graphically on the computer screen, and recorded in spreadsheet format on a hard disk at $\approx$1-min intervals. Only data free of electrical and other artifact were recorded and analyzed. The process was controlled by a "virtual instrument" (computer program emulating hardware) written with LabVIEW® graphic signal processing software (National Instruments, Inc., Austin, TX).††

Fingertip vasoconstriction (resulting primarily from decreased arteriovenous shunt flow) was quantified with venous-occlusion volume plethysmography performed at 5-min intervals.[15] Volume plethysmography probably is the most reliable technique for evaluating peripheral blood flow. For comparison, we also evaluated peripheral flow according to forearm–fingertip skin-surface temperature gradients[16–18]: we have previously demonstrated that this index correlates well with laser Doppler flowmetry and volume plethysmography.[19] As in our previous studies, significant vasoconstriction was prospectively defined as a skin-temperature gradient $\geq$ 4° C. Vasoconstriction in capillaries of central skin surfaces was quantified with laser Doppler flowmetry (Periflux 3; Perimed Inc., Piscataway, NJ) with the fiberoptic probe positioned midchest, at approximately the level of the fifth thoracic dermatome.

Shivering was evaluated qualitatively at 5-min intervals, on a scale from 0 (no tremor) to 2 (intense, continuous shivering).

Data recorded at 1- to 5-min intervals from each volunteer were averaged into 5-min epochs by a database program; these individual averages were used to calculate the means ($\pm$ SD) for the entire group of volunteers. Changes in skin temperature, thermal flux, fingertip blood flow, skin-temperature gradient, and laser Doppler perfusion index were analyzed by repeated measures analysis of variance and Dunnett's tests. The last 5-min acquisition epoch before starting the cold infusion was considered the reference for intragroup comparisons. The average laser Doppler flow index during the entire control period was considered 100%. $P < 0.01$ identified significant differences.

Results

The mean age of the volunteers was 28 $\pm$ 6 yr, weight was 69 $\pm$ 10 kg, and height was 170 $\pm$ 5 cm. Average ambient temperature was 30.8° C, and did not vary more than 0.1° C during any experiment. Central venous infusion of cold lactated Ringer's solution produced no local discomfort. Grade-2 (*i.e.*, intense) shivering was detected in each volunteer within 5 min of starting the cold fluid infusion. Grade-1 or grade-2 shivering continued throughout the study.

Changes in fingertip blood flow and skin-surface temperature gradients are shown in figure 1. Fingertip blood

†† Dr. Sessler will make this program available to interested investigators.

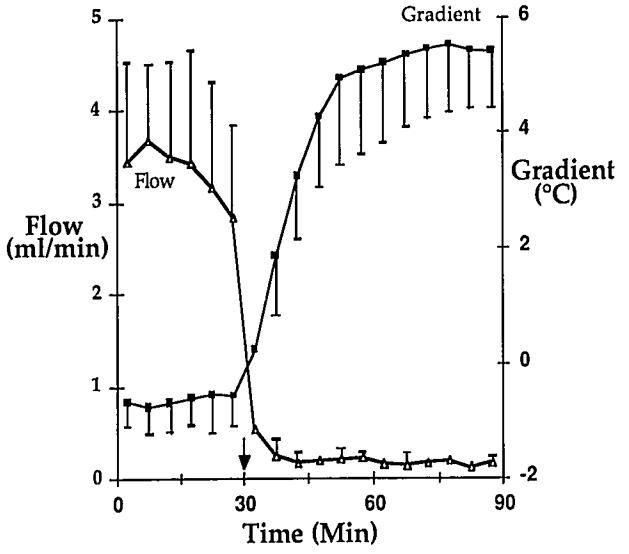

FIG. 1. Changes in fingertip blood flow and skin-surface temperatue gradients. Infusion of cold lactated Ringer's solution started at the arrow, and continued for 60 min. Fingertip blood flow decreased ~10-fold within a few minutes after cold fluid infusion started, and continued to decrease for an additional 5–10 min. Skin-temperature gradients increased to ≥4° C (our prospectively defined index of significant vasoconstriction) ~15 min after the infusion was started. All values obtained for epochs >35 min differed significantly from control.

flow decreased ≈ ten-fold within a few minutes after cold fluid infusion began, and continued to decrease for an additional 5 min. Skin-temperature gradients increased to ≥ 4° C (our prospectively defined index of significant vasoconstriction) ≈ 15 min after the infusion was started.

Tympanic membrane temperature decreased 1.5 ± 0.3° C in the first 15 min after cold fluid infusion was started. During the remainder of the study, when the infusion rate was lower, central temperature returned to ≈ 1° C below control values. Average skin-surface temperature decreased slowly to ≈ 0.7° C below control. The most prominent decrease in skin temperature occurred in the hands and feet (fig. 2). Flow in capillaries of centrally distributed skin (determined with laser Doppler flowmetry) decreased ≈ 40% (fig.3).

Heat flux from the ten measured sites were grouped into three categories, indicating losses from the head and trunk (forehead, back, chest, and abdomen), from the legs and arms (upper arm, lower arm, thigh, and calf), and from the hands and feet. Total heat flux, and flux from the arms and legs decreased ≈ 25%. Heat loss from the trunk and head decreased 17%, whereas loss from the hands and feet decreased ≈ 50% (fig.4).

Analysis of the volume plethysmography, skin-temperature gradient, laser Doppler, skin-surface temperature, and heat flux data indicated that none of the five control epochs (0–25 min) differed significantly from the epoch immediately preceding the intravenous infusion (26–30 min). In contrast, values obtained during all epochs > 35 min differed significantly from control.

Discussion

Our study modeled the common clinical situation in which central temperature is decreased by administration of cold intravenous fluids to a patient in a comfortable thermal environment. Cold intravenous fluids are known

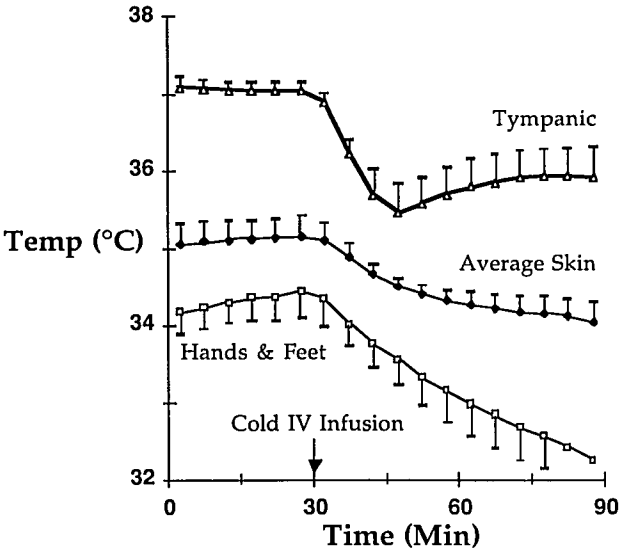

FIG. 2. Tympanic membrane temperature decreased ~1.5° C in the first 15 min after cold fluid infusion began (arrow). During the rest of the study, when the infusion rate was lower, central temperature decreased further to ~1° C below control. Average skin-surface temperature decreased slowly to ~0.7° C below control. The most prominent decrease in skin temperature occurred in the hands and feet (consistent with the known distribution of thermoregulatory arteriovenous shunts). All values obtained for epochs >35 min differed significantly from control.

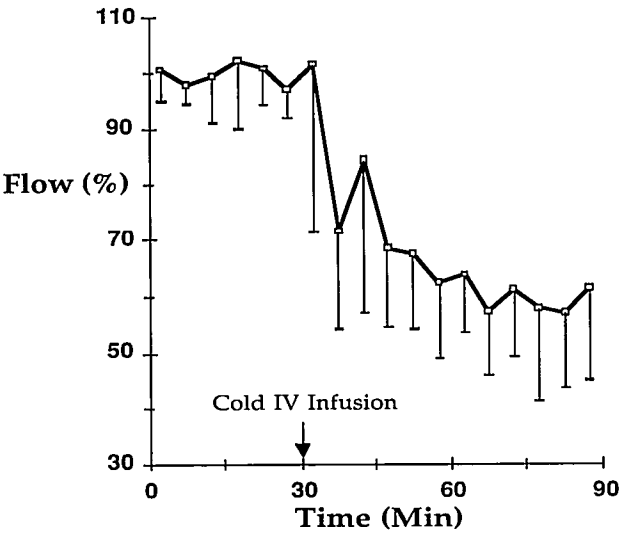

FIG. 3. Flow in capillaries of centrally distributed skin determined using laser Doppler flowmetry decreased ~40% after infusion of cold fluid (arrow). All values obtained for epochs >35 min differed significantly from control.

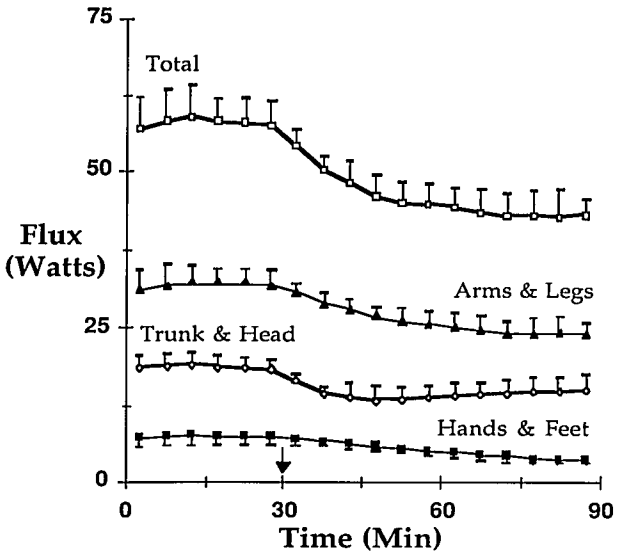

FIG. 4. Heat flux from the ten measured sites were grouped to indicate losses from the head and trunk (head, back, chest, and abdomen); legs and arms (upper arm, lower arm, thigh, and calf); and hands and feet. Total heat flux and flux from the arms and legs decreased ~25% after infusion of cold fluid (arrow). Heat loss from the trunk and head decreased only 17%; in contrast, loss from the hands and feet decreased ~50%. All values obtained for epochs >35 min differed significantly from control. Heat losses indicated in the lower three curves (arms and legs; trunk and head; hands and feet) comprise the total loss indicated at the top of the figure.

to contribute to shivering during epidural anesthesia.[20-22] An analogous problem occurs during transurethral resection of the prostate when the bladder is irrigated with large amounts of fluid at ambient temperatures. Our results also serve as a baseline against which anesthetic-induced vasodilation and thermoregulatory vasoconstriction during general anesthesia can be compared.[16-18]

Thermal steady state requires that metabolic heat production equals heat loss to the environment.[23] Steady state usually is actively maintained in unanesthetized humans by thermoregulatory effectors that increase metabolic heat production (nonshivering thermogenesis and shivering), decrease environmental heat loss (active vasoconstriction and behavioral maneuvers), or increase heat loss (active vasodilation and sweating). Most metabolic heat traverses the skin; the remainder is lost *via* respiration.[1,24,25] Vasoconstriction is the first thermoregulatory response to hypothermia, presumably because preserving heat is more efficient than generating it. Shivering is believed to occur only when maximal vasoconstriction (and behavioral maneuvers) are insufficient to maintain an appropriate mean body temperature.[11,26]

Preliminary studies showed that preventing active thermoregulatory vasoconstriction in unanesthetized volunteers during the control period required ambient temperatures > 30° C. Decreasing ambient temperature would have rapidly induced vasoconstriction, but also would have markedly increased cutaneous heat loss (which is determined primarily by the difference between skin-surface and ambient temperatures). Thus, we decreased central temperature (a much more important thermoregulatory input) by the administration of iced intravenous fluids.

Central hypothermia (≈ 1.5° C below control temperatures) induced by administration of cold intravenous fluids did not produce the intense thermal discomfort we have previously observed during skin-surface cooling.[27] The minimal discomfort (qualitatively evaluated) in this study is especially notable because discomfort in the previous study was *not* associated with a decrease in central temperature. We have demonstrated previously that thermal comfort during epidural anesthesia is determined by skin-surface, not central, temperature.[28,29] A minimal subjective response to central hypothermia also is consistent with studies in monkeys that demonstrate that behavioral responses, which are easy to elicit by skin-temperature alterations, rarely occur after isolated changes in brain temperature.[30]

Thermoregulatory changes in cutaneous blood flow are greatest (*i.e.*, > 10-fold) in arteriovenous shunts of the hands, feet, ears, lips, and nose.[1-3] However, even in these areas, flow also decreases in the more numerous capillaries.[3-6] In our volunteers, thermoregulatory vasoconstriction only decreased heat loss 15.5 W. This is similar to the savings produced by active airway humidification in intubated patients[31,32] or by placement of a circulating water blanket beneath patients.‡‡

Although the fractional changes in heat flux during vasoconstriction were largest in tissues known to have thermoregulatory arteriovenous shunts, most of the savings occurred elsewhere. These data suggest that capillary constriction in skin without arteriovenous shunts (approximately 40% decrease estimated by laser Doppler flowmetry) also contributes to cutaneous thermoregulation. However, the relatively small decrease in flux across the largest skin surfaces may have resulted, in part, from a passive decrease in skin temperature induced by central hypothermia. Because central hypothermia is the only practical way to induce thermoregulatory vasoconstriction without changing ambient temperature, it was not possible to determine the extent to which regulated capillary constriction and passive central hypothermia contributed to decreased skin temperature and thermal flux. *Active* vasodilation (during heat stress) markedly increases capillary flow over large areas of the skin; heat flux surely increases significantly under such circumstances. Some active vasodilation is likely at 30.8° C.

Because we found ten heat flux transducers to be the practical limit in this study, flux across each transducer (in watts per meter-squared) was multiplied by the adjacent estimated surface area (to produce watts per site).

‡‡ Unpublished data.

This extrapolation introduced some error in our regional heat flux measurements, but the error was minimized by distributing a large number of transducers on skin surfaces in which changes were greatest. Cutaneous heat losses are approximately two-fold higher in typical surgical or recovery environments (ambient temperatures 21–23° C) than in our study environment (≈ 30.8° C).[33] However, the absolute number of watts saved by thermoregulatory vasoconstriction during anesthesia is likely to be less than that observed in this study, because active capillary vasodilation does not occur in normothermic or hypothermic anesthetized patients.

We conclude that thermoregulatory vasoconstriction decreased cutaneous heat loss ≈ 25%; the largest fractional decrease occurred in skin known to have arteriovenous shunts.

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