Heat Loss Through the Glabrous Skin Surfaces of Heavily Insulated, Heat-Stressed Individuals

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Insulation reduces heat exchange between a body and the environment. Glabrous (nonhairy) skin surfaces (palms of the hands, soles of the feet, face, and ears) constitute a small percentage of total body surface area but contain specialized vascular structures that facilitate heat loss. We have previously reported that cooling the glabrous skin surfaces is effective in alleviating heat stress and that the application of local subatmospheric pressure enhances the effect. In this paper, we compare the effects of cooling multiple glabrous skin surfaces with and without vacuum on thermal recovery in heavily insulated heat-stressed individuals. Esophageal temperatures (T_{es}) and heart rates were monitored throughout the trials. Water loss was determined from pre- and post-trial nude weights. Treadmill exercise (5.6 km/h, 9–16% slope, and 25–45 min duration) in a hot environment (41.5°C, 20–30% relative humidity) while wearing insulating pants and jackets was used to induce heat stress ($T_{es} \geq 39°C$). For postexercise recovery, the subjects donned additional insulation (a balaclava, winter gloves, and impermeable boot covers) and rested in the hot environment for 60 min. Postexercise cooling treatments included control (no cooling) or the application of a 10°C closed water circulating system to (a) the hand(s) with or without application of a local subatmospheric pressure, (b) the face, (c) the feet, or (d) multiple glabrous skin regions. Following exercise induction of heat stress in heavily insulated subjects, the rate of recovery of T_{es} was $0.4 \pm 0.2°C/h (n=12)$, but with application of cooling to one hand, the rate was $0.8 \pm 0.3°C/h (n=12)$, and with one hand cooling with subatmospheric pressure, the rate was $1.0 \pm 0.2°C/h (n=12)$. Cooling alone yielded two responses, one resembling that of cooling with subatmospheric pressure ($n=8$) and one resembling that of no cooling ($n=4$). The effect of treating multiple surfaces was additive (no cooling, $\Delta T_{es} = -0.4 \pm 0.2°C$; one hand, $-0.9 \pm 0.3°C$; face, $-1.0 \pm 0.3°C$; two hands, $-1.3 \pm 0.1°C$; two feet, $-1.3 \pm 0.3°C$; and face, feet, and hands, $-1.6 \pm 0.2°C$). Cooling treatments had a similar effect on water loss and final resting heart rate. In heat-stressed resting subjects, cooling the glabrous skin regions was effective in lowering T_{es}. Under this protocol, the application of local subatmospheric pressure did not significantly increase heat transfer per se but, presumably, increased the likelihood of an effect.
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Keywords: insulation, heat stress, heat loss, glabrous skin, subcutaneous vasculature

1 Introduction

Many military, recreational, and industrial activities require the wearing of heavy insulating materials while performing demanding physical activities in thermally stressful conditions (e.g., body armor, mission oriented protective posture (MOPP) gear, protective padding, hazmat suits, and firefighting turnout gear). The accumulation of internal heat can be performance limiting and even life threatening. A practical means to increase heat transfer despite the external insulation would allow individuals to perform at higher levels or for longer durations in thermally stressful environments.

A stable internal body temperature is maintained by balancing heat production and heat loss. Heat is produced as a byproduct of cellular metabolism—the greater the metabolic effort, the greater the heat production. Heat is lost to the environment across the surface of the body. High ambient temperature, humidity, thermal radiation, and low air movement reduce heat dissipation capacity and can even result in net heat gain from the environment. External insulation further reduces radiative, conductive, convective, and evaporative heat losses. Nonhuman mammals maintain relatively constant internal temperatures despite variations in environmental conditions and internal heat production, and a fixed layer of external insulation. To effectively remove heat from the body, internal heat must be delivered to noninsulated surface areas for dissipation to the environment.

The role of the cardiovascular system in temperature regulation is to transport heat to exposed body surfaces. Although heat transfer occurs across the entire body surface and the entire skin is perfused by blood, regional distributions of blood flow to the skin (as well as external insulation layers) determine the contributions of the various skin regions to temperature regulation. Immersion of the hands and forearms and/or feet and lower legs in cold water has been demonstrated to be effective for reducing core tempera-

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ture in heat-stressed subjects during periods of rest [1–7]. A substantially similar benefit can be derived by using subatmospheric pressure to enhance heat transfer through the glabrous skin regions of the distal appendages only [8–10].

Humans have two types of skin: (1) glabrous skin is characterized by an absence of hair follicles and a dense packing of subcutaneous vascular structures (collectively referred to as retia venosa), and (2) nonglabrous skin is characterized by the presence of hair follicles and the absence of densely packed subcutaneous vascular structures other than those associated with the hair follicles [11]. Glabrous skin covers only the soles of the feet, the palms of the hands, and regions of the face and ear pinnae. Blood flow through the glabrous subcutaneous vascular structures is under vasomotor control and regulated according to thermoregulatory requirements [12–15]. With heat stress, local blood flow through the glabrous skin regions can be an order of magnitude greater than local blood flow through the nonglabrous skin regions [16–18].

A means for facilitating heat transfer through the glabrous skin regions of the hands and feet has been described [8,9,19]. Through the combined local application of subatmospheric pressure to expand the filling of the retia venosa and an appropriate heat sink, it is possible to manipulate core temperature using only a single hand or foot [8–10,19]. Use of this heat extraction method during fixed-load aerobic exercise in a hot environment by appropriately clad, fit, and active individuals decreases the rate of core temperature rise and improves physical performance [8,10].

Cardiac output increases during exercise, but the increased blood flow must service both the metabolic demands of the active muscles and the thermoregulatory demands associated with increased internal heat production. Cardiac output of resting individuals increases during heat stress when there are no metabolically active peripheral tissues competing for the increased cardiac output. It was unclear whether a local pressure differential would provide a benefit to heat-stressed resting individuals who already have large volumes of blood flowing through the subcutaneous heat exchange vascular structures.

The objective of the current study was to replicate, and expand on, previous studies that assessed the effects of distal appendage cooling on heat-stressed, resting individuals [1–7,20]. The specifics to be addressed in this study were as follows: (1) Can heat extraction through the glabrous skin substantially hasten recovery from heat stress despite an insulation layer covering the general body surface? (2) Does application of a local subatmospheric pressure to the glabrous skin facilitate recovery from heat stress? (3) Will the treatment of multiple glabrous skin regions provide additive benefits?

2 Materials and Methods

Subjects. Seventeen adult males (age range: 22–64) participated in the study. All subjects engaged in regular exercise programs. Informed consent was obtained from each subject using an instrument approved by the Stanford University Institutional Review Board (IRB). Each subject was assigned an alphanumeric identifier, which was used thereafter in accordance with Health Insurance Portability and Accountability Act (HIPAA) guidelines.

Facilities and monitoring equipment. The trials were conducted in a $2.4 \times 3.3 \times 2.4$ m^3 (width, length, and height) temperature-controlled environmental chamber. The ambient conditions inside the environmental chamber were $41.5 \pm 0.5\,^{\circ}C$, and relative humidity of 20–35%. Treadmills (model SC7000, SciFit, Tulsa, OK) housed in the experimental chamber were used for the exercise portion of the trials.

Esophageal temperature (T_{es}) and heart rate were measured throughout the trials. T_{es} was measured with a commercially available general purpose thermocouple probe (Mon-a-Therm No. 503-0028, Mallinckrodt Medical Inc., St. Louis, MO). These probes were self-inserted by the subjects through the nose or mouth to a depth of 38–39 cm and held in place by a loop of surgical tape (Transpore, 3M Corporation, St. Paul, MN) adhered to the skin adjacent to the nostril opening or lower lip. The probes were connected to a laptop-based thermocouple transducer/data collection system (GEC instruments, Gainesville, FL), which recorded temperature data at 1 s intervals. Heart rate monitors/data loggers (model S810, Polar Electro Oy, Kempele, Finland) collected heart rate at 5 s intervals. Water loss was calculated by subtracting post-trail nude weight from pretrial nude weight (cargo scale, model c-12, OHAUS, Pine Brook, NJ).

At the end of each trial, heart rate and temperature data were downloaded to a central desktop computer and transferred to spread sheets (Microsoft Excel) for subsequent off-line analysis. Hand-noted data logs were also maintained for each trial. Subject identifier, date, treatment, pre- and post-trial nude weights, exercise duration, and miscellaneous comments were recorded on these data sheets along with temperature and heart rate measurements noted at 3 min intervals.

Heat extraction devices. The custom-built heat extraction device for use on the hand(s) consisted of a rigid chamber into which one hand was inserted through an elastic sleeve that formed a flexible airtight seal around the wrist, where the palm rested on a curved metal surface perfused with $10-11\,^{\circ}C$ water circulated at (1.0–2.0 l/min). The rigid chamber was connected to a pressure sensor and a vacuum pump. When activated, the vacuum pump created a low level subatmospheric pressure inside the chamber (-40 mm Hg). For heat extraction from the feet, commercially available urethane-coated nylon water perfusion pads (36.5×21.6 cm^2, Plas-tech, Corona, CA) were wrapped around the foot such that the entire sole was in contact with the pad and held in place with Velcro straps. For heat extraction from the face, two urethane-coated nylon water perfusion pads (10.0×20.0 cm^2, Plas-tech, Corona, CA) were sewn into a balaclava (cloth headgear covering the whole head, exposing only limited regions of the face around the eyes and mouth) so that pads directly abutted the ear pinnae and cheek areas of the face. Water ($10-11\,^{\circ}C$) was circulated through the head and feet water perfused interfaces at a rate of 1.0–2.0 l/min.

Protocol. Prior to each trial, nude weight was measured and each subject was equipped with a heart rate monitor and T_{es} probe. During each trial, the subjects wore military clothing (battle dress uniforms under MOPP jackets and pants). MOPP overgarments are water impermeable and have a high insulative value (≈ 1.7 clo). Heat stress was induced by having the subjects walk at 5.6 kph (3.5 mph) uphill on a treadmill. The slope of the treadmill was adjusted for individual subjects so that heat stress (defined as $T_{es} \geq 39\,^{\circ}C$) was achieved in 25–45 min. The stop criteria for exercise were $T_{es} = 39\,^{\circ}C$, a heart rate 95% of calculated maximum (220 bpm (beats/min) − years of age) or subjective fatigue. At the termination of exercise, the subjects donned additional insulation (polypropylene balaclava (Tullahoma Industries, Tullahoma, TN), cold weather gloves (Sovereign, Grandoe Corporation), and commercially available disposable waterproof boot covers) and sat quietly on a chair in the hot room for a minimum of 60 min (Fig. 1). After the 60 min minimum rest/recovery period, the heart rate monitor and thermocouple probes were removed from the subject and post-trial nude weight was measured.

The experimental manipulations were performed during the rest/recovery phase. The insulating overlayers were removed from the regions of the body being treated. Seventeen subjects were subjected to three treatments: no treatment (control), the combined application of a cool thermal sink and subatmospheric pressure to a single hand, or the combined application of the cool thermal sink and subatmospheric pressure to both hands. Twelve subjects participated in an additional trial during which a cool thermal sink was applied to the palm of one hand without the application of subatmospheric pressure. Eight subjects participated in three additional trials during which a cool thermal sink was applied to the face, the feet, or all five glabrous skin regions (the face, feet, and hands (with the pressure differential)). All

Fig. 1 Photographs of the experimental setup and experimental devices. Left panel: a subject clad in MOPP gear, balaclava, winter glove, and boot covers, and one hand in the experimental heat transfer device during postexercise rest in the experimental chamber (T_{es}=41 °C). Right panel: two hands placed in the experimental devices (see Sec. 2 for description of devices). The external neoprene insulation covering the experimental device had been removed for the photograph to better display the details of the device.

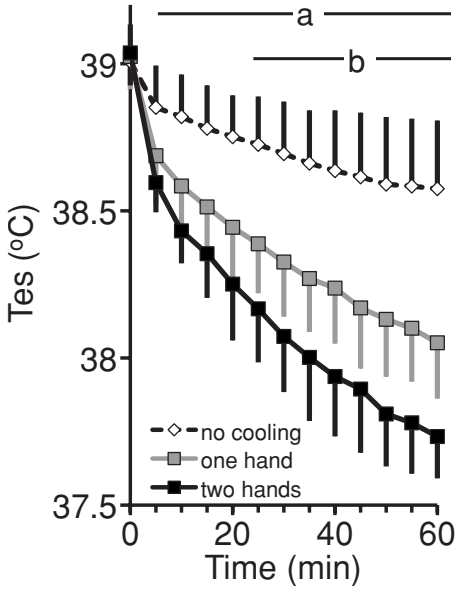

Fig. 2 T_{es} versus time during recovery after exercise in thermally stressful conditions. Treatments: control (no cooling), heat extraction from one hand, and heat extraction from two hands (n=17). Heat extraction entailed the combined application of a heat sink to the glabrous skin of the hand and subatmospheric pressure to the entire hand (mean±SD). t-test results: (*a*) control versus one hand, $p<0.001$; (*b*) one hand versus two hands, $p<0.005$.

experimental trials on an individual subject were separated by a minimum of 2 days and the subjects returned to the laboratory at the same time of day for all of their trials. The order of the treatments was randomized. No water was consumed during the trials, but each subject consumed the volume of fluid equivalent to his weight loss during the trial prior to leaving the facility.

Statistical analysis. The raw T_{es} and heart rate data were plotted as a function of time for each trial, the plots were screened for artifact, and the artifacts were removed from the data set. The sizes of the data sets were then reduced by sampling the data at 30 s intervals. The data from the resting portion of the trial were selected and plotted as a function of time.

The individual trial recovery T_{es} data were then grouped according to treatment. The grouped data were further reduced by sampling the data at 5 min intervals. Mean and standard deviation of the mean were calculated for each treatment group at each 5 min time point. The mean treatment data were plotted against time. The main effects of factors "treatment," "time," and "subject" were analyzed by one- or two-factor analysis of variance (ANOVA) (Microsoft Excel), with repeated measures where appropriate.

Pre-exercise heart rate (5 min mean), maximum heart rate (at the end of exercise), and final resting heart rates (5 min mean) were sorted by treatment and analyzed for the main effects of factors treatment, time, and subject by one- and two-way ANOVAs (n=8). Water losses were calculated from changes in nude weights. The water loss data were sorted by treatment and analyzed for the main effects of factors treatment and subject by one- and two-way ANOVAs.

Post hoc paired t-tests were used for subsequent statistical analysis of the T_{es}, heart rate, and water loss data.

3 Results

The T_{es} at the end of exercise for all trials was 39.0±0.1 °C (mean±SD, n=84).

3.1 Core Temperature. *One hand versus two hand treatment*. The combined application of cooling and subatmospheric pressure to one hand resulted in a decrease in T_{es} of 1.0±0.2 °C in 60 min of treatment compared with 0.4±0.2 °C with no cooling (Fig. 2). Cooling two hands further enhanced the treatment effect: T_{es} decreased by 1.3±0.2 °C in 60 min when cooling and subatmospheric pressure were applied to two hands. Two-way ANOVA with repeated measures revealed significant effects of the factors treatment and time (treatment $p \ll 0.001$, time $p \ll 0.001$, and interaction $p \ll 0.001$). Post hoc t-tests determined significant differences between the no treatment and one and two hand treatment groups ($p<0.001$) at time points of 5–60 min, and between the one hand and two hand treatment groups ($p<0.005$) at time points of 25–55 min.

One hand treatment with and without a pressure differential. In the 60 min of treatment, T_{es} decreased by 0.4±0.2 °C (n=12) with control treatment, by 0.8±0.3 °C (n=12) with cooling only, and by 1.0±0.2 °C (n=12) with cooling and subatmospheric pressure (Fig. 3(*a*)). Two-way ANOVA with repeated measures revealed significant effects of the factors treatment and time (treatment $p \ll 0.001$, time $p \ll 0.001$, and interaction $p=0.005$). Post hoc t-tests determined significant differences ($p<0.001$) between no treatment and cooling with subatmospheric pressure treatments at time points of 5–60 min, and between no cooling and cooling-alone at time points of 10–60 min. Cooling with subatmospheric pressure trended to be different from cooling-alone at time points of 15–60 min ($p<0.1$).

A more detailed analysis revealed that cooling-alone treatment yielded two discrete T_{es} response patterns, one resembling that of cooling with subatmospheric pressure and one resembling that of no cooling (Fig. 3(*b*)). In 8 of the 12 subjects, T_{es} decreased by 1.0±0.3 °C with cooling-alone compared with 1.0±0.2 °C with cooling and pressure differential and 0.3±0.2 °C with no cooling (control treatment). In these eight subjects, post hoc t-tests revealed that the data from cooling alone were significantly different from control (p=0.001) but was not significantly different from the cooling with pressure differential (p=0.53). In 4 of the 12 subjects, T_{es} decreased by 0.5±0.2 °C with cooling-alone, compared with 0.9±0.2 °C with cooling and a pressure differential

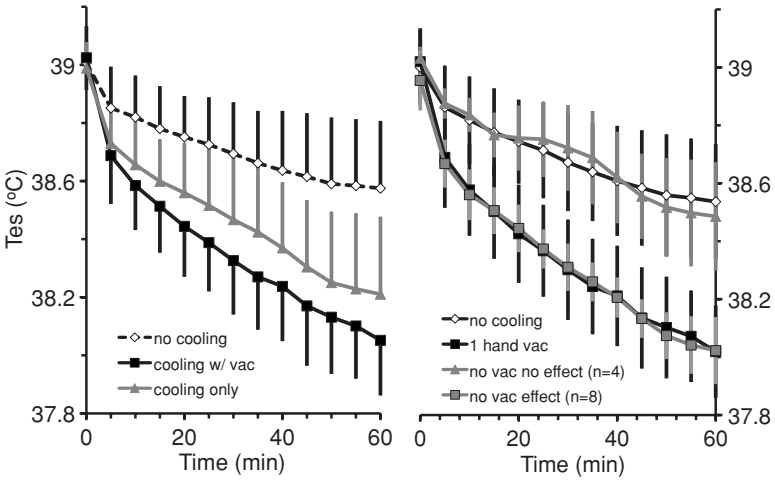

Fig. 3 T_{es} versus time during recovery after exercise in thermally stressful conditions; the effect of cooling one hand, with or without application of subatmospheric pressure. Left: group results $n=12$. Right: cooling only (no pressure differential) group data sorted according to effect and compared with the complete data set for one hand cooling with a pressure differential and no cooling. In eight subjects the response to cooling-alone resembled the response to cooling with subatmospheric pressure. In four subjects, the response to cooling-alone resembled the no treatment response.

and $0.5 \pm 0.1\,^{\circ}\mathrm{C}$ with control treatment. For these four subjects cooling-alone and control treatments were not different ($p=0.38$), while cooling-alone and cooling with a pressure differential trended to be different ($p=0.07$).

Multiple glabrous skin area treatments. Eight subjects participated in a series of six trials that assessed the effects of the application of a thermal sink to the various glabrous skin regions alone and in combination (Fig. 4). The cooling of a single glabrous skin region (one hand or the face) for 60 min doubled the decrease in T_{es} compared with no treatment (one hand, $\Delta\ T_{es}=-0.9 \pm 0.3\,^{\circ}\mathrm{C}$; face, $-1.0 \pm 0.3\,^{\circ}\mathrm{C}$; and no cooling, $-0.4 \pm 0.2\,^{\circ}\mathrm{C}$). Cooling of two glabrous skin regions (two hands or two feet) augmented the cooling effect ($\Delta\ T_{es}=-1.3 \pm 0.1\,^{\circ}\mathrm{C}$ (two hands) and $-1.3 \pm 0.3\,^{\circ}\mathrm{C}$ (two feet)). Application of a thermal sink to all five glabrous skin regions further enhanced the cooling effect. Treatment of the face, feet, and hands (FFH) resulted in a T_{es} decrease of $1.6 \pm 0.2\,^{\circ}\mathrm{C}$ in 60 min with the bulk of the drop in T_{es} occurring in the initial 35 min of treatment ($\Delta\ T_{es}=-1.5 \pm 0.3\,^{\circ}\mathrm{C}$ in 35 min). ANOVA revealed significant effects of the factors treatment and time (treatment $p \ll 0.001$, time $p \ll 0.001$, and interaction $p \ll 0.001$). Post hoc t-tests determined significant differences ($p < 0.001$) between the no treatment and all other treatment groups from 10 min to 60 min, between the single glabrous skin region (one hand or face) treatment groups and two glabrous skin region (two hands or two feet) treatment groups from 25 min to 55 min, and between the two glabrous skin region and the five glabrous skin region (FFH) treatment groups from 15 min to 60 min. There were no significant differences between the one hand treatment and face treatment groups or between the two hands treatment and two feet treatment groups.

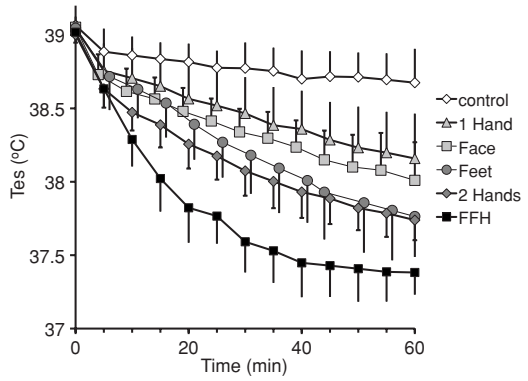

Fig. 4 T_{es} versus time during recovery after exercise in thermally stressful conditions; the effect of cooling the various combinations of glabrous skin regions ($n=8$, mean±SD). FFH: foot, face, and hand cooling.

3.2 Heart Rate and Water Loss. The treatment affected post-recovery heart rates. For the 48 individual trials (eight subjects each participating in six trials), mean heart rate increased by 76 bpm during exercise and decreased by 61 bpm after recovery (pre-exercise, 100 ± 17 bpm; postexercise, 176 ± 13 bpm; postrecovery, 115 ± 17 bpm). There were no differences between treatments in pre-exercise (ANOVA, $p=0.55$) or maximum heart rates (ANOVA, $p=0.78$), but treatment significantly affected post-treatment heart rate (ANOVA, $p<0.01$). Postrecovery heart rates after treatment FFH (103 ± 20 bpm) were indistinguishable from the pre-exercise heart rates but significantly different from heart rates after two hand (112 ± 20 bpm), feet (114 ± 16 bpm), face (117 ± 20 bpm), one hand (121 ± 14 bpm), and control (123 ± 9 beats/min) treatments (ANOVA, $p<0.01$).

The treatment also affected water loss (ANOVA, $p \leq 0.0001$). Compared with control treatment water loss (2.3 ± 0.8 l), cooling of a single hand reduced water loss by <0.1 l (2.2 ± 0.7 l), cooling of the face by 0.3 l (2.0 ± 0.7 l), cooling of two hands by 0.3 l (2.0 ± 0.7 l), cooling of the feet by 0.6 l (1.7 ± 0.8 l), and cooling of the five glabrous skin regions (FFH) by 0.9 l (1.4 ± 0.7 l). Treatment FFH reduced water loss by over 34%. Paired t-tests determined that water losses during the FFH treatment were significantly different from water losses during all other treatments ($p \leq 0.01$).

4 Discussion

These results demonstrate that heat can be effectively removed from a thermally stressed individual despite insulating overgarments that reduce heat transfer across the general body surface.

Furthermore, these results are in opposition to what would be expected if heat transfer across the surface of the body were uniform.

An accepted tenet about heat transfer in humans is that the effectiveness of a heating or cooling procedure is dependent on the size of the surface area being treated and the magnitude of the thermal gradient between the skin and the external thermal medium (for example, see Refs. [21–24]). This tenet is based on the premise that either (1) the vascular composition of the skin (and thus, heat transfer across the skin) is uniform and that vasomotor responses are generalized to all skin vascular structures [22,23] or (2) vasomotor tone is not relevant for heat transfer [24,25]. As a result, equipment developed for the manipulation of core body temperature has focused on the delivery of a thermal load across the general skin surface (e.g., forced-air for peri-anesthesia temperature management [26], and cooling vests for combating heat stress [27,28]). However, the premises on which the heat transfer tenets are based are not supported by a large body of data available in published research reports.

Heat is moved around the body via the circulating blood. To effectively dissipate heat, heat must be moved to the surface of the body. A series of elegant studies in the late 1960s and early 1970s examined changes in blood flow to various organs during thermal stress (see review in Ref. [29]). These studies demonstrated that during extreme thermal stress in resting subjects: (1) cardiac output increased by 6–7 l/min, (2) blood flow to the visceral organs decreased (splanchnic and renal blood flow decreased by 30%), (3) skeletal muscle blood flow remained unchanged, (4) mean arterial pressure decreased (despite a doubling of cardiac output), and (5) the only observed increase in blood flow was a six-fold increase in blood flow through the forearm as measured by plethysmography. It was concluded that, by default, the increased cardiac output was flowing through the skin [29]. How the skin could accommodate such an enormous increase in cardiac output was not known, but it was suggested that the skin vasculature had the ability to expand in capacity to handle the increased cardiac output.

The decrease in mean arterial pressure that accompanied the six-fold increase in blood flow in the forearm suggests that the increased blood flow was not accommodated by the cutaneous vasculature associated with metabolic function (terminal arterioles, capillary loops, and postcapillary venules). An essential role of blood flow through the skin is to support metabolic function, which requires small-diameter membrane-permeable microvessels (capillaries) for gas and nutrient exchange. A six-fold increase in flow through those nutrient microvessels would require an increase—rather than a decrease—in head pressure propelling the blood (i.e., mean arterial pressure). It has previously been suggested (e.g., Ref. [30]) that, in the distal aspects of the appendages, there exist low-resistance bypass routes for blood flow that can accommodate the high rates of blood flow associated with heat stress.

The precise anatomy of the unique subcutaneous microvascular structures that underlie the glabrous skin regions has only recently been revealed [11,31]. The vascular anatomy of the cutaneous layers (papillary layer and dermal-epidermal junction) of the dorsal and palmar skin regions are similar (characterized by loops of capillaries and support vasculature). In contrast, the hypodermal layers (subcutaneous space) of the same two regions are dramatically different. The hypodermal layer of the dorsal hand skin is only sparsely populated with the ovoid structures made up of the capillaries feeding hair follicles, while the hypodermal layer of the palmar skin is densely packed with sweat glands, vascular support structures for the sweat glands, arteriovenous anastomoses, and glomerular-shaped vessels. The densely packed vascular structures in the palmar hypodermic layer are aligned in parallel and of relatively large diameter (compared with capillaries). Such an arrangement of vessels provides a low-resistance pathway capable of accommodating the volumes of blood flow associated with vasodilation and heat stress.

Laser Doppler measurements of local skin blood flow during exercise and heat stress have revealed regional differences in blood flow patterns consistent with the subcutaneous vascular anatomy [17,18,32,33]. During heat stress at a constant exercise work load, subsurface blood flow through the palmar region of the hand can be an order of magnitude greater than the cutaneous blood flow through the forearm and dorsal hand regions (>600 laser Doppler flux (LDF) units of flow through the glabrous skin compared with <75 LDF units of flow through the nonglabrous skin) [17]. During sinusoidal workload cycle exercise trials, cutaneous vascular conductance (CVC) at all measuring sites oscillated with a period linked to the workload cycle but, unlike in the nonglabrous skin regions, the CVC cycle of the glabrous skin regions was 140 deg out of phase with the workload cycles (i.e., blood flow through the glabrous skin increased during the falling phase of the heart rate and work load cycle) [18,32,33]. These results suggest that changes in blood flow through the nonglabrous skin are determined primarily by changes in central blood pressure, while changes in blood flow through the glabrous skin are regulated by changes in vascular resistance.

The distribution of the anatomical structures that directly facilitate heat transfer is limited. A substantial portion of the controlled heat transfer between the body core and the external environment occurs across the glabrous skin. If blood flow through the subcutaneous heat exchange vascular structures of the glabrous skin is unimpeded (i.e., vasodilated), heat transfer will be facilitated and determined by the thermal gradient between the circulating blood and the external environment. With vasodilation, the body core will receive direct and immediate effects as the cooled (or heated) blood returns to the heart and mixes with the remainder of the circulating blood. However, if blood flow is reduced through vasoconstriction or by an uncontrolled obstruction, heat transfer through the heat exchange vascular structures will be impeded and sensible heat transfer across the general body surface will be the dominant heat transfer medium. Utilizing the circulating blood to conduct heat between the skin surface and the body core is a more efficient method for transferring heat than is conduction through peripheral tissues (see Ref. [19]).

Skin temperature and central drive influence vasomotor tone. If the local temperature of the glabrous skin region being treated is below the vasomotor threshold, blood flow through the underlying subcutaneous heat exchange vascular structures will be dramatically reduced [13,14]. Local temperature vasomotor thresholds are influenced largely by core temperature [15]. During treatment for heat stress, as core temperature returns to the desired temperature range, the vasomotor threshold will rise and heat transfer capacity through the glabrous skin will diminish. One way to prevent local temperature-induced vasoconstriction is to increase the temperature of the glabrous skin surface.

Our interpretation of the mixed results from the single hand cooling trials without the addition of the pressure differential (Fig. 3) is that eight of the test subjects' hands were vasodilated during treatment, while four of the subjects' hands were vasoconstricted. There should be an additive effect when treating numerous vasodilated glabrous skin regions. However, the additive effect should diminish as core temperature returns to the desired range and vasomotor tone (vascular resistance through the glabrous skin regions) increases. This notion is supported by the results presented in Fig. 4. The diminishing rate of change in core temperature over time would be expected when blood flow through the heat exchange vasculature is reduced by a progressive increase in vasomotor tone.

The decrease in water loss and reduction in heart rates associated with the cooling treatments make sense; when heat stress is reduced, heat dissipation responses will diminish. Decreasing the thermoregulatory demands on cardiac output enables utilization of

the circulating blood for other functions like increased work capacity. Decreasing sweat rate reduces body water loss. The reduction in water loss can change fluid resource management requirements during activities in heat stress conditions and potentially reduce logistical requirements of supporting groups of individuals working in hot environments.

When rewarming mildly hypothermic individuals, rates of change in core temperatures of 13°C/h have been reported when heat and a pressure differential were applied to a single hand [19] and of 10°C/h when heat was delivered through the feet and lower legs [34]. In the current study on cooling heat-stressed subjects, under the optimal cooling conditions (cooling through the hands, feet, and face), core temperature decreased at a rate of less than 5°C/h. While it is difficult to make direct comparisons between these sets of results (due to numerous factors including the different experimental protocols and conditions of the subjects), it is interesting to speculate about the possible causes of the different effects of seemingly similar thermal manipulations in hypothermic and hyperthermic individuals. One possible explanation for the differences in rates of core temperature changes between the cooling and rewarming studies is that it is the mass of the perfused tissues (rather than the total body mass) that determine the rate of core temperature changes. The hypothermic individuals were vasoconstricted and, thus, circulating blood flow was confined to the central core organs—less than 10% of the total body mass. Conversely, heat-stressed individuals are vasodilated with blood flowing through both the subcutaneous vasculature structures and other peripheral regions of the body. Thus, compared with the hypothermic postanesthesia patients, a greater percent of the total body mass is being perfused in the heat-stressed individuals and, therefore, a larger total mass was being affected by the thermal manipulation. The differences in the perfused mass of the hypothermic and hypothermic individuals could account for the differences in the rates of core temperature changes.

Summary. Heat is lost across the surface of the body. Passive, uncontrolled heat loss occurs across the general body surface. Active, regulated heat transfer is dependent on the controlled delivery of heat to the skin surface via the circulating blood. Only the glabrous skin regions have the anatomical structures necessary to accommodate large volumes of blood flow and therefore large changes in heat dissipation. Thermoregulatory vasodilation primarily effects blood flow through glabrous skin regions. External insulation reduces heat loss. Nonetheless, a substantial amount of heat can be extracted from the body by appropriate treatment of the glabrous skin regions. Heat transfer through the glabrous skin is tightly regulated. If local skin temperature is below the vasomotor threshold, blood flow through the heat exchange vascular structures will be reduced and heat exchange compromised. The relative contributions of the glabrous and nonglabrous skin regions to heat loss are determined primarily by ambient conditions, insulation, and subcutaneous blood flow.

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Disclosures

Patents have been issued for the technology discussed in this manuscript (D. Grahn and H. C. Heller (Inventors); Stanford University (Assignee)), and Stanford University has entered into a licensing agreement with AVAcore Technologies, Inc., for the commercialization of the technology. Included in the license is a royalty agreement that grants Stanford University a percentage of the net sales of the technology, which will be shared by the University and the inventors. D. Grahn and H. C. Heller are founders of AVAcore Technologies but receive no ongoing compensation from the company and AVAcore Technologies provides no financial support for research. To assure that potential conflicts of interest do not influence the outcome of the research, Stanford University requires that Grahn and Heller have no participation in the recruitment of subjects, the conduct of the experimental trials, or the initial analysis of the data.

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