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# Impaired Cutaneous Vasodilation and Sweating in Grafted Skin During Whole-Body Heating

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The aim of this investigation was to identify the consequences of skin grafting on cutaneous vasodilation and sweating in split-thickness grafted skin during indirect whole-body heating 5 to 9 months after surgery. In addition, thermoregulatory function was examined at donor skin sites on a separate day. Skin blood flow and sweat rate (SR) were assessed from both grafted (n = 14) or donor skin (n = 11) and compared with the respective adjacent control skin during indirect whole-body heating. Cutaneous vascular conductance (CVC) was calculated from the ratio of skin blood flow (arbitrary units; au) to mean arterial pressure. Whole-body heating significantly increased internal temperature ( $37.0 \pm 0.1$  °C to  $37.8 \pm 0.1$  °C;  $P < .05$ ). Cutaneous vasodilation (ie, the increase in CVC from baseline,  $\Delta$ CVC) during whole-body heating was significantly attenuated in grafted skin ( $\Delta$ CVC =  $0.14 \pm 0.15$  au/mm Hg) compared with adjacent control skin ( $\Delta$ CVC =  $0.84 \pm 0.11$  au/mm Hg;  $P < .05$ ). Increases in sweat rate ( $\Delta$ SR) were also significantly lower in grafted skin ( $\Delta$ SR =  $0.08 \pm 0.08$  mg/cm<sup>2</sup>/min) compared with adjacent control skin ( $\Delta$ SR =  $1.16 \pm 0.20$  mg/cm<sup>2</sup>/min;  $P < .05$ ). Cutaneous vasodilation and sweating during heating were not significantly different between donor sites ( $\Delta$ CVC =  $0.71 \pm 0.19$  au/mm Hg;  $\Delta$ SR =  $1.04 \pm 0.15$  mg/cm<sup>2</sup>/min) and adjacent control skin ( $\Delta$ CVC =  $0.50 \pm 0.10$  au/mm Hg;  $\Delta$ SR =  $0.83 \pm 0.17$  mg/cm<sup>2</sup>/min). Greatly attenuated or absence of cutaneous vasodilation and sweating suggests impairment of thermoregulatory function in grafted skin, thereby, diminishing the contribution of this skin to overall temperature control during a heat stress. (J Burn Care Res 2007;28:427–434)

Increases in skin blood flow and sweating are critical responses for humans to appropriately regulate internal temperature during exercise and/or hyperthermic exposure. To thermoregulate, the skin must be properly vascularized and neurally innervated. Wounds

such as burns seriously damage the skin, requiring, in many cases, excising the damaged tissue with subsequent skin grafting to the damaged area. Split-thickness skin grafting results in the removal of the epidermis and a portion of the dermis from a donor site, followed by attachment of the harvested graft to a damaged recipient area. Unless grafted skin becomes appropriately revascularized and reinnervated, the grafted site will not be able to effectively contribute to thermoregulatory responses.

Little is known regarding the consequences of skin grafting with respect to the control of skin blood flow in the grafted tissue. Revascularization and angiogenesis between the recipient bed and the graft begins within 48 to 72 hours after grafting.<sup>1–3</sup> Depending on the thickness of the graft, some degree of circulation usually is restored by the fourth to seventh day after grafting.<sup>4</sup> However, it is unknown whether autonomic control of the cutaneous vasculature, with

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respect to thermoregulatory responses, is restored in healed split-thickness grafts.

A functioning sudomotor (ie, sweating) system also is critical for adequate thermoregulation during exercise and/or heat exposure. Most studies show an absence of sweating from split-thickness grafts.<sup>5-8</sup> This response is thought to be the result of a combination of the initial injury destroying sweat glands at the recipient tissue and a lack of sweat glands in the harvested tissue in most split-thickness grafts.<sup>9</sup>

Altered autonomic control of skin blood flow and sweating may be responsible for the observed higher rectal temperatures in burn patients during a thermal challenge relative to nonburned counterparts.<sup>6,8,10,11</sup> In the small amount of research that has been conducted, information regarding thermoregulatory responses in grafted skin is limited. Freund and colleagues<sup>5</sup> reported varying degrees of cutaneous vasodilation during whole-body heating in grafted skin, with normal vasodilatory responses in some individuals as soon 7 to 9 weeks after injury. However, these varied responses, coupled with a small subject number ( $n = 6$ ) with a wide range of graft maturities (7 weeks to 5 years after surgery), complicates the interpretation of these data.<sup>5</sup> In addition, Freund et al<sup>5</sup> assessed cutaneous vasodilation via forearm venous occlusion plethysmography, which does not allow for the regional assessment of cutaneous blood flow responses between grafted skin and adjacent control skin. Finally, little is known regarding the effects of harvesting skin on cutaneous vasomotor and sweating responses from the donor site, at which the cutaneous vascular bed, neural connections, and the duct portion of the sweat gland are disrupted.

The primary aim of the present investigation was to test the hypothesis that cutaneous vasodilation and sweating responses during indirect whole-body heating are attenuated in split-thickness grafted skin 5 to 9 months after surgery compared with adjacent control skin. A secondary aim was to test the hypothesis that cutaneous vasodilation and sweating responses during indirect whole-body heating are also attenuated at donor skin sites when compared with adjacent control skin.

## METHODS

### Protocol 1: Grafted Skin

**Human Subjects.** Fourteen individuals (7 men, 7 women) who had undergone split-thickness autograft application after tangential excision to viable fat within the prior 5 to 9 months participated in this study. Patients with shallow and deep dermal exci-

sions were not enrolled. The mean age, height, and weight of the subjects were  $32.6 \pm 2.6$  yrs,  $168.1 \pm 2.6$  cm, and  $82.1 \pm 4.8$  kg, respectively (mean  $\pm$  SEM). Participants provided informed written consent before testing. All protocols were approved by the Institutional Review Board at the University of Texas Southwestern Medical Center at Dallas and Presbyterian Hospital of Dallas and were conducted in accordance with the Declaration of Helsinki principles. Subjects were not taking any medications that would affect cutaneous vasodilatory or sweating responses. All subjects refrained from caffeine, alcohol, and exercise for 24 hours before the study.

**Instrumentation.** Heart rate was obtained from an electrocardiogram (Agilent, Palo Alto, CA), with the signal interfaced with a cardiometer (CWE, Ardmore, PA). Arterial blood pressure was measured from the upper arm via electrophygmomanometry (SunTech, Raleigh, NC). Internal temperature was indexed from an ingestible pill telemetry system (HQ, Inc., Palmetto, FL). The telemetry pill correlates well with other methods of internal temperature measurement.<sup>12</sup> Mean skin temperature was measured via the weighted average of six thermocouples attached to the skin.<sup>13</sup> Skin blood flow was measured continuously from integrating laser-Doppler flowmetry probes (model PF413, Perimed, Sweden), each housed in a 3-cm diameter heater element (Perimed, Sweden) placed on grafted skin and adjacent control skin. The integrating laser-Doppler flow probes continuously measured skin blood flow throughout the entire protocol, but over a small area ( $\sim 0.28$  cm<sup>2</sup>). Skin blood flow at the grafted site and adjacent control skin was also assessed with a laser-Doppler imager (Moor LDI, Moor Instruments, UK) at normothermic baseline and at peak heat stress. The laser Doppler imager uses a scanning laser to measure blood flow over a larger area of skin compared with the integrating laser Doppler flowmetry probes (Table 3). Sweat rate was measured using capacitance hygrometry (Vaisala, Woburn, MA) by perfusing 100% nitrogen at a flow rate of 300 ml/min through a ventilated capsule (surface area = 2.83 cm<sup>2</sup>) placed on grafted skin and adjacent control skin.

**Protocol.** Individuals were dressed in a tube-lined suit that permitted the control of skin and core temperature by changing the temperature of water perfusing the suit (Med-Eng, Ottawa, Canada). The perfusion suit covered the entire body with the exception of the head, hands, feet, and instrumented area (Table 1). Because instrumented areas were not in contact with the suit, observed changes in skin blood flow or sweating associated with heat stress were not caused by the effects of locally heating the skin but

**Table 1.** Location of split-thickness skin graft and skin donor site

Subject No.	Sex	Graft Site	Donor Site
1	Female	Left forearm	Right thigh
2	Male	Right forearm	Right calf
3	Male	Left thigh	Left thigh
4	Male	Right forearm	Right thigh
5	Female	Right hand/wrist	Right thigh
6	Female	Right forearm	Right thigh
7	Female	Left calf	Right thigh
8	Female	Left calf	Right thigh
9	Male	Right forearm	Right thigh
10	Male	Left forearm	Right thigh
11	Female	Right arm	Right thigh
12*	Male	Left hand/wrist	Left thigh
13*	Male	Right forearm	Right thigh
14*	Female	Left calf	Right thigh

\* Individuals who did not participate in Protocol 2.

rather were an autonomic response associated with increases in internal temperature. Data were collected with the subject in the supine position. Baseline measurements were obtained while perfusing the suit with 34 °C water. After normothermic data collection, a whole-body heat stress ensued by perfusing 46 °C water through the suit until internal temperature increased ~0.8 °C. On completion of the whole-body heat stress, cool water was perfused through the suit and local heating was performed by increasing local skin temperature to 42 °C via the heating elements housing the laser-Doppler flow probes. Local temperature was held at this level for 30 minutes to elicit maximal cutaneous vasodilation.<sup>14</sup> Skin blood flow was then normalized relative to maximal vasodilation for each site.

### Protocol 2: Donor Skin

**Human Subjects.** Eleven individuals (5 men, 6 women) returned a minimum of 48 hours after completing Protocol 1 and repeated the whole-body heating protocol to assess responses from the donor site.

**Instrumentation and Protocol.** Subjects were instrumented in the same manner as described above with the exception that integrating laser-Doppler flowmetry probes, local heaters, and sweat capsules were placed on donor skin sites and adjacent control skin (Table 1). This protocol was performed exactly as specified above for Protocol 1.

### Data and Statistical Analysis

For both protocols, data were continuously acquired at a sampling rate of 50 Hz using a data collection

system (Biopac System, Santa Barbara, CA). One-minute-averaged responses were calculated at the final minute of normothermic baseline and whole-body heating. Cutaneous vascular conductance (CVC) was calculated from laser Doppler-derived skin blood flow divided by mean arterial blood pressure. CVC data were also normalized to maximal vasodilation obtained during the final minute of local heating at 42 °C and expressed as percentage of CVC maximum (CVC<sub>max</sub>).

Student's paired *t*-tests were used to compare the magnitude of the increase in CVC from normothermic baseline between grafted skin and adjacent control skin, as well as between donor sites and control skin adjacent to the donor site, during the heat stress. Student's paired *t*-tests also were used to compare the differences in maximal CVC obtained during local heating, as well as the increases in sweating during whole body heating for each area (graft and donor). Statistical significance was accepted at *P* < .05. All data are presented as mean ± SEM.

## RESULTS

### Protocol 1: Grafted Skin

Baseline CVC was significantly higher in grafted skin (0.53 ± 0.08 au/mm Hg) compared with adjacent control skin (0.31 ± 0.04 au/mm Hg; *P* = .04). This difference was more apparent when data were expressed relative to percentage of CVC<sub>max</sub> (graft: 36.0 ± 6.1 %CVC<sub>max</sub>; graft control: 12.8 ± 0.9 %CVC<sub>max</sub>; *P* = .002) because of differences in maximal vasodilation obtained during local heating discussed herein.

Typical thermal and cardiovascular responses associated with whole-body heating were observed (Table 2). This level of heating resulted in approximately a 3-fold increase in CVC in the adjacent control skin (ΔCVC = 0.84 ± 0.11 au/mm Hg) compared with minimal increases from baseline in grafted skin (ΔCVC = 0.14 ± 0.15 au/mm Hg; *P* = .001; Table 3). This difference in the change in CVC was also apparent when data were expressed relative to percentage of CVC<sub>max</sub> (graft: ΔCVC = 2.2 ± 5.2 %CVC<sub>max</sub>; graft control: ΔCVC = 39.0 ± 5.2 %CVC<sub>max</sub>; *P* < .001; Table 3). Large differences in the change in CVC between sites during the heat stress were also confirmed with laser-Doppler scanning (graft: ΔCVC = 0.24 ± 0.11 au/mm Hg; graft control: ΔCVC = 1.28 ± 0.16 au/mm Hg; *P* < .001; Table 3). Laser Doppler scanner images from a representative subject during normothermia and at the end of whole-body heating illustrate the large

**Table 2.** Thermal and cardiovascular responses during normothermic baseline (normothermia) and at the end of indirect whole-body heating (WBH) in Protocol 1 (graft) and Protocol 2 (donor)

Variable	Protocol 1: Graft (n = 14)		Protocol 2: Donor (n = 11)	
	Normothermia	WBH	Normothermia	WBH
T <sub>core</sub> (°C)	37.0 ± 0.1	37.8 ± 0.1*	36.9 ± 0.1	37.7 ± 0.1*
Mean T <sub>sk</sub> (°C)	34.7 ± 0.1	38.2 ± 0.2*	34.5 ± 0.2	37.9 ± 0.2*
HR (beats/min)	71.2 ± 3.7	97.7 ± 3.8*	69.3 ± 5.1	90.1 ± 5.3*
MAP (mm Hg)	83.6 ± 2.3	84.0 ± 2.5	87.3 ± 2.0	82.7 ± 3.2*

T<sub>core</sub>, core temperature; T<sub>sk</sub>, skin temperature; HR, heart rate; MAP, mean arterial pressure.

Values are expressed as means ± SEM.

\* Difference from normothermia ( $P < .05$ ).

difference in cutaneous vasodilation between these areas of skin (Figure 1). Increases in sweating during whole-body heating were also lower in grafted skin ( $\Delta SR = 0.08 \pm 0.08$  mg/cm<sup>2</sup>/min) relative to adjacent control skin ( $\Delta SR = 1.58 \pm 0.21$  mg/cm<sup>2</sup>/min;  $P < .001$ ; Figure 2).

Maximal CVC caused by local heating at 42 °C tended to be lower in grafted skin ( $1.80 \pm 0.28$  au/mm Hg) compared with adjacent control skin ( $2.46 \pm 0.26$  au/mm Hg;  $P = .11$ ; Table 3).

### Protocol 2: Donor Skin

Baseline CVC was similar between the donor site ( $0.25 \pm 0.05$  au/mm Hg) and adjacent control skin ( $0.27 \pm 0.05$  au/mm Hg;  $P = .72$ ). However, when data were expressed as a percentage of maximum, the donor site ( $10.8 \pm 1.8$  %CVC<sub>max</sub>) tended to be

lower than adjacent control skin ( $15.1 \pm 2.3$  %CVC<sub>max</sub>;  $P = .11$ ), likely because of differences in maximal vasodilation obtained during local heating discussed below.

Similar to Protocol 1, typical thermal and cardiovascular responses associated with whole-body heating also were observed (Table 2). Increases in CVC from baseline in response to whole-body heating were not different between the donor site and adjacent control skin regardless of whether the data were expressed in absolute units (donor:  $0.71 \pm 0.19$  au/mm Hg; donor control:  $0.50 \pm 0.10$  au/mm Hg;  $P = .17$ ; see Table 4) or as %CVC<sub>max</sub> (donor:  $27.7 \pm 4.5$  %CVC<sub>max</sub>; donor control:  $26.9 \pm 4.8$  %CVC<sub>max</sub>;  $P = .88$ ; Table 4). When assessed by laser Doppler scanning, increases in CVC from baseline were also similar between the donor site ( $\Delta CVC = 0.92 \pm 0.26$  au/mm Hg) and adjacent control skin ( $\Delta CVC = 0.99 \pm 0.33$  au/mm Hg;  $P = .87$ ; Table 4 and Figure 3). No differences were also observed in sweating at the donor site ( $\Delta SR = 1.04 \pm 0.15$  mg/cm<sup>2</sup>/min) compared with adjacent control skin ( $\Delta SR = 0.84 \pm 0.17$  mg/cm<sup>2</sup>/min;  $P = .40$ ; Figure 4).

Maximal CVC responses to local heating at 42 °C tended to be greater at donor sites ( $2.46 \pm 0.36$  au/mm Hg) when compared with adjacent control skin ( $1.74 \pm 0.11$  au/mm Hg;  $P = .06$ ; Table 4).

**Table 3.** Cutaneous vascular conductance (CVC) at the end of indirect whole-body heating and local heating in grafted (graft) and adjacent control (graft control) skin

	Graft	Graft Control
Whole-body heating (laser-Doppler probes)		
ΔCVC (au/mm Hg)	0.14 ± 0.15*	0.84 ± 0.11
ΔCVC (%CVC <sub>max</sub> )	2.2 ± 5.2*	39.0 ± 5.2
Whole-body heating (laser-Doppler scanner)		
ΔCVC (au/mm Hg)	0.24 ± 0.11*	1.28 ± 0.16
Scanner area (cm <sup>2</sup> )	1.16 ± 0.12	1.16 ± 0.12
Local heating (laser-Doppler probe)		
CVC (au/mm Hg)	1.80 ± 0.28	2.46 ± 0.26

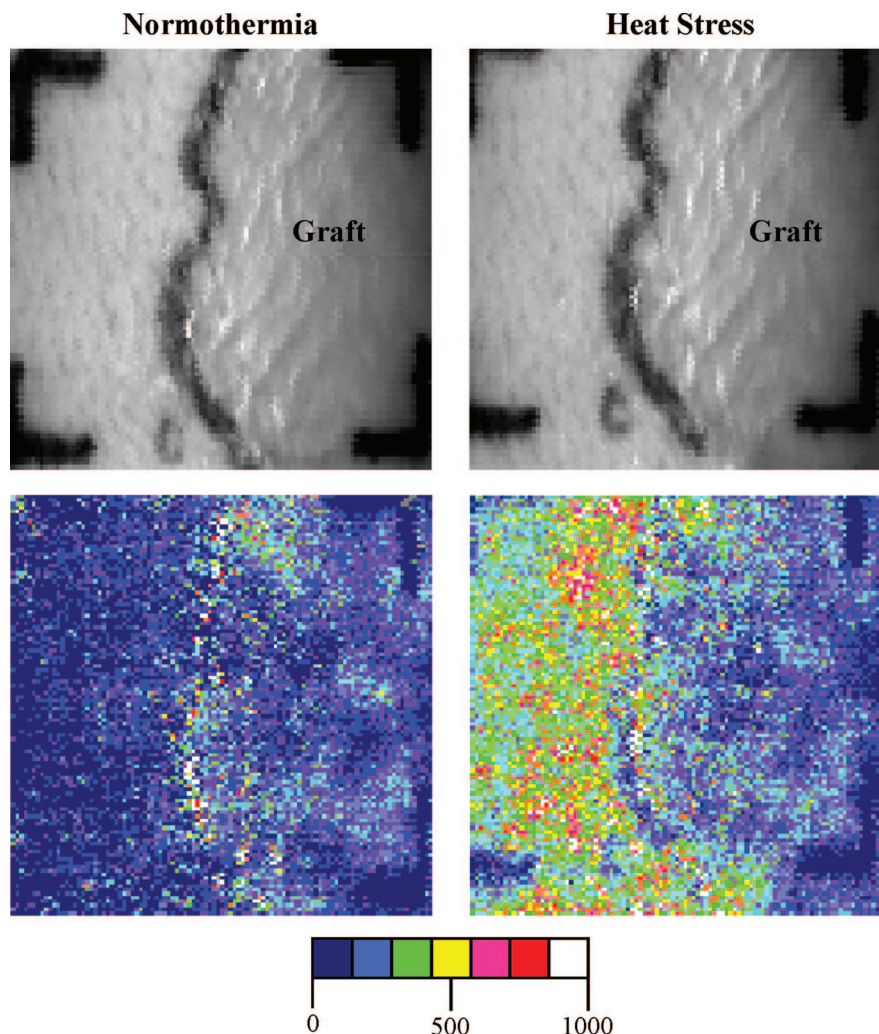
ΔCVC represents changes from normothermic baseline expressed in both absolute units (au/mm Hg) and relative to maximum CVC (%CVC<sub>max</sub>).

Values are expressed as means ± SEM.

\* indicates difference from Graft Control ( $P < .05$ ).

## DISCUSSION

The primary finding of this investigation is that cutaneous vasodilation and sweating during indirect whole-body heating are attenuated in grafted skin 5 to 9 months after surgery when compared with adjacent control skin, suggesting that grafted skin has reduced thermoregulatory capacity during a heat stress. An additional important finding of this study is that cutaneous vasodilation and sweating are not impaired at donor skin sites.

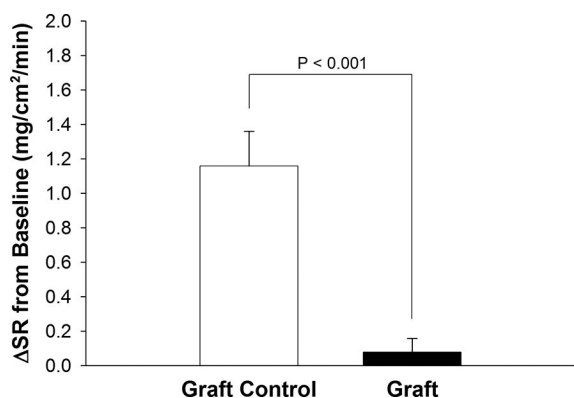


**Figure 1.** Laser-Doppler scanner image (top: photo image; bottom: flux image) from a representative subject during normothermia (left column) and indirect whole-body heating (right column) at a grafted and adjacent control site. Progressively greater skin blood flows were observed in adjacent control skin (expressed in green, yellow, and red) compared with lower skin blood flows in grafted skin (depicted in shades of blue).

Control of skin blood flow in humans occurs through two distinct sympathetic pathways. The first pathway involves sympathetic vasoconstrictor nerves, whereas the second pathway modulates skin blood flow through a non-adrenergic sympathetic active vasodilator system.<sup>15,16</sup> Upon exposure to warm/hot environments and/or exercise, the initial increase in skin blood flow occurs via withdrawal of the aforementioned cutaneous vasoconstrictor system.<sup>17</sup> Further increases in skin blood flow during whole-body heating in humans are accomplished through engagement of the sympathetic active vasodilator system, which contributes upwards to 85% to 95% of the rise in skin blood flow in nonglabrous (ie, hairy) skin during a heat stress.<sup>15-19</sup> Importantly, active cutaneous vasodilation is absent in denervated

skin<sup>16,18,20,21</sup>; thus, an intact functioning cutaneous sympathetic active vasodilator system must be present for the vasculature of the skin to dilate during indirect whole-body heating.

In the present investigation, the magnitude of cutaneous vasodilation observed in grafted skin was quite small in some patients and absent in others. These findings are in stark contrast to the findings of Freund and colleagues suggesting that cutaneous vasodilatory responses can be normal in grafted skin in some subjects.<sup>5</sup> These conflicting findings may be attributed to differences in methodology, as well as the maturity of the graft. Laser-Doppler flowmetry (probe and scanner) was used in the current study allowing the simultaneous assessment and comparison of cutaneous blood flow in grafted skin and ad-



**Figure 2.** Changes in sweat rate (mg/cm<sup>2</sup>/min) from normothermic baseline during whole-body heating from grafted skin (graft) and adjacent control skin (graft control).

adjacent control skin. Freund et al. measured forearm blood flow via venous occlusion plethysmography as an index of cutaneous vasodilation, which would not differentiate between blood flow responses in the grafted and adjacent control skin.<sup>5</sup> Finally, the current study tightly controlled for maturity of the skin graft (5–9 months after surgery), whereas Freund et al. investigated cutaneous vasodilatory responses in grafted skin from a small group of individuals ( $n = 6$ ) with a wide range of graft maturities (7 weeks to 5 years after surgery).<sup>5</sup>

Attenuated increases in cutaneous blood flow in grafted skin may be the result of diminished sympathetic neural function (ie, inappropriate or absence of required sympathetic innervation and/or decreased

neurotransmitter release). Consistent with this hypothesis, elevated baseline CVC in grafted skin during normothermia may be attributed to a lack of tonic sympathetic vasoconstrictor activity. Taken together, these findings may suggest altered sympathetic neural function of both the vasoconstrictor and active vasodilator pathways. Another possibility is altered postsynaptic function (ie, decreased sensitivity to vasodilator neurotransmitters), which also may contribute to an attenuation of cutaneous blood flow in grafted skin during heating. Although this work focuses on overall cutaneous vascular and sweating responses in grafted and donor skin, the effects of skin grafting specifically on postsynaptic function were examined and are reported in the companion manuscript.<sup>22</sup>

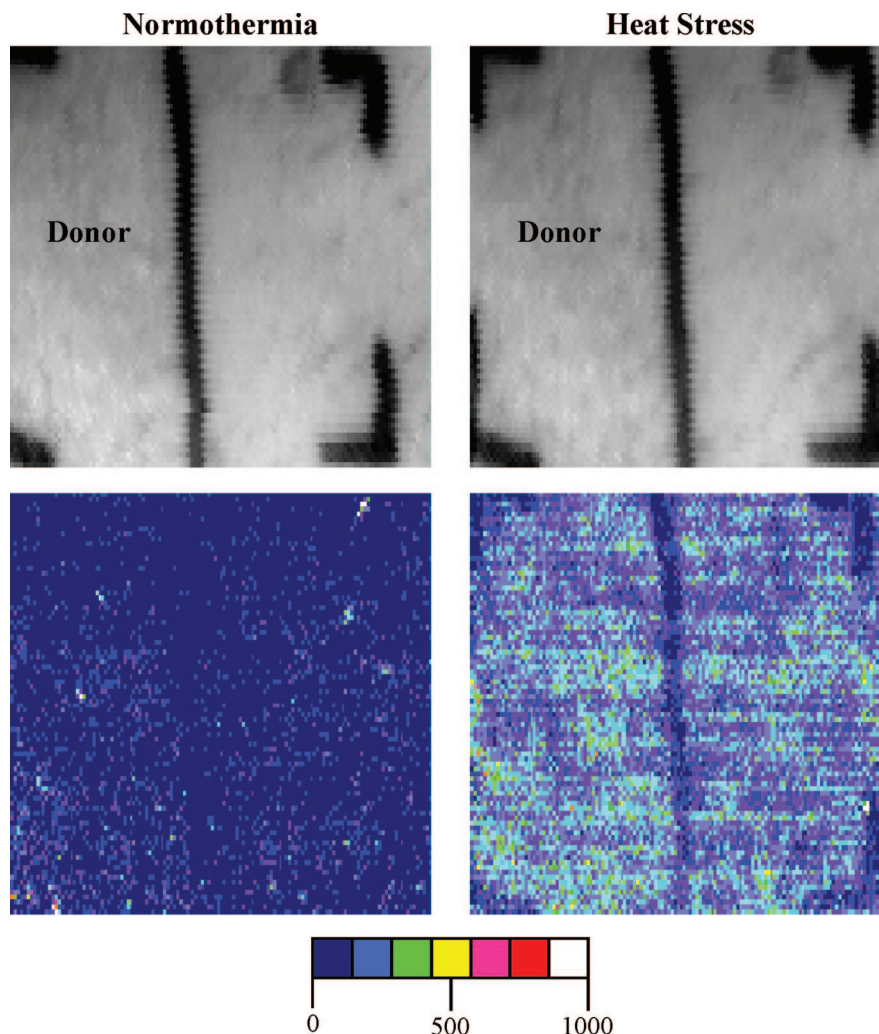
Local heating of the skin (ie, directly heating the skin where blood flow is measured) at 42 °C for 30 minutes causes maximal cutaneous vasodilation.<sup>15</sup> This vasodilator response is not abolished by nerve blockade at the site or proximal to the site where skin blood flow is assessed.<sup>23</sup> However, the response is greatly attenuated when nitric oxide synthase inhibitors are administered, indicating cutaneous vasodilation during sustained local heating is primarily nitric oxide dependent.<sup>23,24</sup> In the current study, both adjacent control and grafted skin were locally heated to assess whether grafting altered local heating-induced vasodilation. Increases in CVC were observed at both areas of skin during local heating. However, the magnitude of the increase in CVC tended to be less in grafted skin when compared to adjacent control skin ( $P = .11$ ; Table 3). Whether this response is the result of alterations in nitric oxide release or vascular responsiveness to nitric oxide remains unknown. If grafting impairs nitric oxide mediated vasodilation, it may partially explain attenuated cutaneous vasodilation during whole-body heating, given that approximately 30% of cutaneous vasodilation during whole-body heating is nitric oxide dependent.<sup>25,26</sup> However, such an effect alone would be unlikely to completely explain differences in the elevation of CVC between sites during indirect whole-body heating as many subjects had a complete absence of an increase in CVC with the whole-body heat stress.

Also necessary for adequate thermoregulatory responses to exercise and/or heat exposure is a functioning sudomotor (ie, sweating) system. Minimal sweating was observed in the grafted skin in the present study. This finding is consistent with previous studies documenting an absence of sweating from split-thickness grafts.<sup>6–8</sup> The reported lack of sweating in split-thickness skin grafts is likely the result of a combination of absent and/or disrupted sweat glands in the injured area, coupled with a lack of sweat glands

**Table 4.** Cutaneous vascular conductance (CVC) at the end of indirect whole-body heating and local heating at the donor site (donor) and adjacent control skin (donor control)

	Donor	Donor Control
Whole-body heating (laser-Doppler probe)		
ΔCVC (au/mm Hg)	0.71 ± 0.19	0.50 ± 0.10
ΔCVC (%CVC <sub>max</sub> )	27.7 ± 4.5	26.9 ± 4.8
Whole-body heating (laser-Doppler scanner)		
ΔCVC (au/mm Hg)	0.92 ± 0.26	0.99 ± 0.33
Scanner area (cm <sup>2</sup> )	1.45 ± 0.21	1.45 ± 0.21
Local heating (laser-Doppler probe)		
CVC (au/mm Hg)	2.46 ± 0.36	1.74 ± 0.11

ΔCVC represents changes from normothermic baseline expressed in both absolute units (au/mm Hg) and relative to maximum CVC (%CVC<sub>max</sub>). Values are expressed as means ± SEM.



**Figure 3.** Laser-Doppler scanner image (top: photo image; bottom: flux image) from a representative subject during normothermia (left column) and indirect whole-body heating (right column) at a donor and adjacent control site. Areas of increased skin blood flows are expressed in green, yellow, and red compared to lower skin blood flows depicted in shades of blue. Similar increases in skin blood flow during whole-body heating were observed at the donor site compared to adjacent control skin.

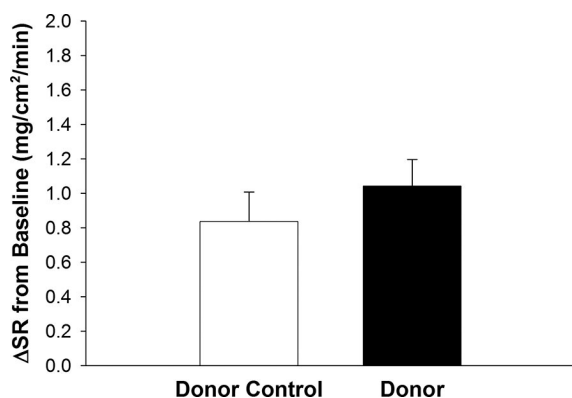
in the donor tissue of most split-thickness grafts.<sup>9,27</sup> However, the potential contribution of denervation of the sweat gland has not been investigated, nor is it known whether sweat glands regenerate in split-thickness grafted tissue as the graft matures.

The attenuation or absence of active cutaneous vasodilation and sweating in grafted skin observed in this study could account for the greater rectal temperatures observed in some burned individuals during a thermal challenge.<sup>6,8,10,12</sup> Individuals with burns covering a greater percentage of their body surface area (ie, greater than 40% TBSA) appear to be impacted to a greater extent during a thermal challenge, indicating thermoregulatory function may become more compromised with increased areas of grafted skin.<sup>6,8,10,12</sup>

Despite the disruption and removal of the epidermal layer and a portion of the dermal layer, the magnitude of the increase in CVC and sweating during heat stress were similar between donor sites and adjacent control skin. These data suggest that the donor site has maintained thermoregulatory responses. That said, CVC at the donor site tended to be higher than the control site during local heating induced maximal cutaneous vasodilation ( $P = .06$ , Table 4). The reasons for a greater maximum CVC at the donor site are unclear but may be related to healing processes.

## CONCLUSION

In summary, split-thickness skin grafts 5 to 9 months after surgery have impaired cutaneous vasodilation



**Figure 4.** Changes in sweat rate ( $\text{mg}/\text{cm}^2/\text{min}$ ) from baseline during whole-body heating from the donor site (Donor) and adjacent control skin (Donor Control).

and sweating in response to indirect whole-body heating. In addition, grafted skin has reduced maximal vasodilatory responsiveness to a local heating stimulus. These impairments, taken together, indicate that grafted skin has a greatly attenuated capability to contribute to thermoregulation, and are likely the key reasons why individuals are at an increased risk of a heat related injury if the grafted region covers a large area of the skin's surface. It remains unknown whether cutaneous vasodilation and possibly sweating responses are restored in grafted skin later than 5 to 9 months after surgery. However, thermoregulatory function is not impaired at donor sites despite split-thickness graft harvesting.

## ACKNOWLEDGMENTS

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