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Orthostatic challenge does not alter skin sympathetic nerve activity in heat-stressed humans

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Abstract

Perturbations that load or unload baroreceptors do not alter skin sympathetic nerve activity (SSNA) in normothermic individuals. However, in pronounced heat-stressed individuals, when a significant component of the SSNA signal is sudomotor and possibly vasodilator in origin, the effects of baroreceptor unloading via an orthostatic stress on SSNA remain unclear. The purpose of the present study was to test the hypothesis that low and moderate levels of orthostatic stress via lower body negative pressure (LBNP) alter SSNA in pronounced heat-stressed individuals. In both normothermic and heat-stressed conditions, progressive LBNP at −3, −6, −9, −12, −15, −18, −21 and −40 mm Hg were applied to 11 subjects for 2 min per stage. Whole-body heating increased sublingual temperature by 0.7±0.1 °C, heart rate by 28±2.1 bpm, SSNA by 259±76 %, forearm skin blood flow by 631±142% and forearm sweat rate to 0.68±0.14 mg/cm^2/min (all $p<0.005$), but did not change mean arterial blood pressure (MAP) ($p>0.05$). LBNP did not change total SSNA in normothermic or heat-stressed conditions (both $p>0.05$), although skin blood flow and sweat rate decreased during moderate levels of LBNP while heat stressed. These data suggest that in pronounced heat-stressed individuals, when a significant component of the SSNA signal contains sudomotor and possibly cutaneous active vasodilator activities, low and moderate levels of baroreceptor unloading via LBNP do not alter total SSNA. This observation, coupled with reductions in skin blood flow and sweating during moderate levels of LBNP, suggests that integrated SSNA should not be used as an indicator of baroreflex modulation of the cutaneous vasculature or sweat rate in heat-stressed subjects.

Keywords: SSNA; Autonomic nervous system; Baroreflexes; Sudomotor; Hyperthermia

1. Introduction

Humans are more susceptible to fainting during orthostatic stress and gravitational acceleration when combined with heat stress (Lind et al., 1968; Allen and Crossley, 1972; Wilson et al., 2002). However, the mechanisms responsible for this observation remain unclear. During a heat stress, upwards of 50% of cardiac output is directed towards the skin (Rowell, 1974), and thus, baroreflex control of cutaneous vascular conductance is important for short-term blood pressure control in this thermal condition, while baroreflex control of sweat rate may be important for long-term blood pressure control through attenuation of fluid loss. A key factor in these responses is whether the neural signal leading to alterations in skin blood flow and sweating (i.e., skin sympathetic nerve activity; SSNA) is modulated by baroreceptor unloading.

Under normothermic conditions, SSNA is primarily vasoconstrictor in origin (Bini et al., 1980). In this thermal condition, perturbations such as direct stimulation of the carotid sinus nerve (Wallin et al., 1975), Valsalva manoeuvres, spontaneous variations in blood pressure (Delius et al., 1972; Hagbarth et al., 1972), low and high levels of lower body negative pressure (LBNP) (Vissing et al., 1994, 1997),

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and pharmacologically induced changes in arterial blood pressure (Wilson et al., 2001) do not alter multiunit SSNA. These studies strongly suggest that in normothermia, baroreceptors do not modulate SSNA.

Pronounced increases in skin blood flow and sweat rate during a heat stress originate from increases in cutaneous sudomotor/vasodilator SSNA signals that are not present under normothermic conditions. Thus, despite prior data suggesting an absence of baroreflex control of SSNA under normothermic conditions, it may be that during a heat stress, when SSNA contains primarily sudomotor and possibly cutaneous vasodilator signals, baroreceptors become capable of modulating SSNA. However, prior studies investigating this question report controversial findings.

Our laboratory recently demonstrated that in heat-stressed subjects, multiunit SSNA was unaffected by pronounced changes in arterial blood pressure via bolus infusions of nitroprusside and phenylephrine (Wilson et al., 2001). This perturbation primarily loads and unloads arterial baroreceptors (Ebert and Cowley, 1992; Rudas et al., 1999) with minimal (Martin et al., 2004) or no (Dibner-Dunlap et al., 1996) changes in central venous pressure. Although informative, our prior study did not identify the effects of heat stress on baroreceptor control of SSNA during global baroreceptor unloading as would occur during orthostatic stress.

Other studies investigating these questions yield mixed results (Vissing et al., 1994; Dodt et al., 1995). Dodt et al. (1995) reported decreases in SSNA during LBNP and upright tilting in heated individuals, which suggests that baroreceptor unloading can modulate SSNA in this condition. In contrast, Vissing et al. (1994) observed that baroreceptor unloading via low levels of LBNP did not modulate SSNA. A primary limitation of these studies is that the thermal stress was mild, and the magnitude of the contribution of sudomotor and active vasodilator signals in the overall SSNA recording was likely small. Consequently, it remains unknown whether orthostatic stress and accompanying global baroreceptor unloading is capable of modulating SSNA in pronounced heat-stressed subjects when a significant component of the SSNA signal contains sudomotor and possibly active vasodilator activities. Thus, the purpose of present study was to test the hypothesis that baroreceptor unloading alters SSNA in pronounced heat-stressed individuals.

2. Materials and methods

A total of 11 healthy subjects (6 males, 5 females) participated in this study. The subjects' average age was 32±2 (S.E.) years, and all were of normal height (168±3 cm) and weight (70±4 kg). Subjects were normotensive (supine blood pressures <140/90 mm Hg), were not taking medications and were free of any known cardiovascular, neurological and metabolic diseases. All subjects refrained from caffeine, alcohol and exercise 24 h prior to the study. A written informed consent, which was approved by the Institutional Review Boards at the University of Texas Southwestern Medical Center and Presbyterian Hospital of Dallas, was obtained from each subject before participation in this study.

2.1. Instrumentation and measurements

Internal temperature (T_{sl}) was measured from a thermistor placed in the sublingual sulcus. Mean T_{sk} was measured via the weighted average of six thermocouples attached to the skin (Taylor et al., 1989). Two of the six thermocouples were placed on the abdomen and lower back, which locations are more sensitive to changes in T_{sk} caused by possible air leakage during LBNP. Each subject was dressed in a tube-lined suit that permitted the control of T_{sk} by changing the temperature of water perfusing the suit. Subjects were placed in a Plexiglas box, which was sealed at the level of the iliac crests. The water-perfused suit comprised of an upper and lower garment, and thus, the LBNP device was sealed directly to the subject's skin, which minimizes cooling during LBNP. Suction was provided by a vacuum pump and controlled with a variable autotransformer. The pressure difference between the LBNP chamber and the atmosphere was measured with a digital manometer. LBNP was used to simulate orthostasis since it permits discrete and reproducible levels of orthostatic stress (i.e., incremental changes at 3 mm Hg) without inherent challenges of maintaining a SSNA signal during upright tilt or potentially confounding influences of postural muscle contraction.

Multifiber recordings of SSNA were obtained with a tungsten microelectrode inserted in the common peroneal nerve. A reference electrode was placed subcutaneously 2–3 cm from the recording electrode. The recording electrode was adjusted until a site was found in which SSNA bursts were clearly identified using previously established criteria (Delius et al., 1972; Vallbo et al., 1979), i.e., (1) integrated nerve activity nonsynchronous with the heart beat; (2) irregular burst activity; (3) the generation of reflex bursts during mental or somatosensory stimuli (i.e., loud sound and light stroking of the innervated region) and (4) an absence of an increase in activity during inspiratory apnea. The nerve signal was amplified, passed through a bandpass filter with a bandwidth of 500–5000 Hz and integrated with a time constant of 0.1 s (Iowa Bioengineering, Iowa City, IA). Mean voltage neurograms were displayed on a chart recorder. The nerve signal was also routed to an oscilloscope and a loudspeaker for monitoring throughout the study. Heart rate was obtained from the electrocardiogram signal (SpaceLabs, Redmond, WA) interfaced with a cardiotachometer (1000 Hz sampling rate; CWE, Ardmore, PA). Blood pressure was recorded by auscultation of the brachial artery (SunTech, Medical Instruments Raleigh, NC). Respiratory frequency was monitored using piezoelectric pneumography. Impedance cardiograph (EBI 100 C, Biopac System, Santa Barbara, CA) was used to measure transthoracic impedance (Zo) as an index of shifts in central blood volume. Skin blood flow was measured via laser Doppler flowmetry, using integrating flow probes (Perimed, North Rayalton, OH, USA), from dorsal forearm skin and from the area within the innervation field of the SSNA being recorded (usually dorsal foot). Cutaneous vascular conductance (CVC) was calculated from the ratio of skin blood flow to mean arterial blood pressure (MAP). MAP was calculated as diastolic blood pressure plus one-third pulse pressure. After the entire procedure, a 3-cm-diameter heater element (Perimed), which housed the laser Doppler flow probe, was engaged to elevate local T_{sk} to 42 °C. Local temperature was held at this level for 30 min to elicit maximal cutaneous vasodilation (Taylor et al., 1984). Skin blood flow and CVC were normalized relative to maximal vasodilation for each site. Sweat rate was measured adjacent to the laser Doppler flow probes via capacitance hygrometry (Viasala, Woburn, MA, USA) by perfusing 100% nitrogen at a flow rate of 300 ml/min through a ventilated capsule (surface area=2.83 cm^2) attached to the surface of the skin.

2.2. Protocol

In both normothermic and heat-stressed conditions, progressive LBNP was applied at −3, −6, −9, −12, −15, −18, −21 and−40 mm Hg for 2 min per stage. However, LBNP was released for 3 min between −12 and −15 mm Hg LBNP stages. The purpose for this pause in the LBNP protocol was to identify if baseline SSNA had changed during the heating period, given the possibility that SSNA may increase due to an increase in thermal load throughout the LBNP procedure. Following the normothermic LBNP protocol, T_{sl} was elevated ~0.5–0.7 °C via the water-perfused suit. Upon reaching this objective, the temperature of the water perfusing the suit was slightly reduced to attenuate the rate of rise of internal temperature throughout the ensuing LBNP protocol.

LBNP was terminated if the subject developed signs and/or symptoms of presyncope, such as sudden onset of nausea, sweating (normothermic trial), light headedness, bradycardia, or hypotension (sustained systolic blood pressure, ≤80 mm Hg). In the present study, all subjects completed the protocol under normothermic conditions. In the heat-stressed condition, LBNP was terminated in two subjects at −21 mm Hg and another two subjects at −40 mm Hg.

2.3. Data analysis

Data were sampled at 200 Hz via a commercial data acquisition system (Biopac System) and analyzed using LabView software (National Instruments, Austin, TX). SSNA bursts were first identified in real time by visual inspection of data plotted on a chart recorder, coupled with the burst sound from the audio amplifier. SSNA recordings with indications of electrode movement during LBNP or whole-body heating were not analyzed. In the present study, data from 3 subjects (who were not included in the presented 11 subjects) were discarded for this reason. Integrated SSNA was normalized by assigning a value of 100 to the mean amplitude of the largest sympathetic bursts (top 10% of identified bursts) during normothermic pre-LBNP baseline (Taylor et al., 1998; Halliwill, 2000; Cui et al., 2004). Subsequent bursts in the neurogram were normalized against that value. To assess total activity of SSNA, baseline was carefully identified, and the area of the integrated neurogram above this baseline was calculated at 10-s intervals from the digitized record. Segments with aberrant electrical noise were excluded from the calculation. Average heart rate, CVC, sweat rate and total activity of SSNA during the second minute of each LBNP stage was obtained and statistically analyzed.

Statistical analyses were performed using a commercial software package (SigmaStat 2.03). Differences in LBNP-induced responses between normothermic and heat stress trials were evaluated via post hoc analysis after repeated-measures two-way analysis of variance ANOVA. Main factors of that ANOVA were LBNP stage and thermal condition. Differences in thermal and hemodynamic responses within normothermic and heat stress trials were evaluated using one-way repeated-measures ANOVA. Differences in baseline (i.e., pre-LBNP) thermal and hemodynamic responses were statistically analyzed via paired *t*-tests. All values are reported as means±S.E. *P* values of <0.05 were considered statistically significant.

3. Results

Whole-body heating increased mean T_{sk} ~3.2 °C and T_{sl} ~0.7 °C resulting in significant increases in heart rate, SSNA, skin blood flow and sweat rate (Table 1). MAP did not change throughout whole body heating. T_{sl} did not change during LBNP, although T_{sk} decreased slightly, but

Table 1
Thermal and hemodynamic responses to the heat stress

	Normothermia	Heat stress
T_{sl} (°C)	36.5±0.1	37.2±0.1*
T_{sk} (°C)	34.6±0.1	37.8±0.1*
MAP (mm Hg)	83±2	82±2
HR (beats/min)	57±2	84±3*
Total SSNA (units/min)	317±80	697±90*
Forearm SkBF (% max)	8.2±1.4	50.8±3.5*
Forearm SR (mg/cm^2/min)	–	0.68±0.14*
Leg SkBF (% max)	9.6±1.2	42.7±5.1*
Leg SR (mg/cm^2/min)	–	0.58±0.09*

Data show baseline values before LBNP in both thermal conditions. Mean arterial blood pressure (MAP) was calculated as diastolic blood pressure plus one-third pulse pressure. Skin blood flow (SkBF) was normalized relative to maximum skin blood flow and is expressed as a percent of maximum. T_{sl}: sublingual temperature; T_{sk}: mean skin temperature; HR: heart rate; SSNA: skin sympathetic nerve activity; SR: sweat rate.

* Significantly different from normothermia (P<0.05).

Table 2
Hemodynamic and thermal responses during progressive LBNP under both thermal conditions

LBNP (mm Hg)	Baseline	−3	−6	−9	−12	Rest	−15	−18	−21	−40
Normothermic										
T_{sk} (°C)	34.6±0.1	34.6±0.1	34.6±0.1	34.6±0.1	34.6±0.1	34.6±0.1	34.6±0.1	34.6±0.1	34.6±0.1	34.5±0.1
T_{sl} (°C)	36.5±0.1	36.5±0.1	36.5±0.1	36.5±0.1	36.5±0.1	36.5±0.1	36.5±0.1	36.5±0.1	36.6±0.1	36.5±0.1
ΔTI (Ω)	–	0.05±0.11	0.30±0.10*	0.59±0.12*	0.81±0.17*	0.07±0.15	1.01±0.28*	1.26±0.29*	1.60±0.33*	2.66±0.51*
Heat stress										
T_{sk} (°C)	37.8±0.1†	37.8±0.1†	37.7±0.1*,†	37.6±0.1*,†	37.6±0.1*,†	37.8±0.1	37.7±0.1*,†	37.7±0.1*,†	37.7±0.2*,†	37.4±0.2*,†
T_{sl} (°C)	37.2±0.1†	37.2±0.1†	37.2±0.1†	37.2±0.1†	37.2±0.1†	37.2±0.1†	37.2±0.1†	37.2±0.1†	37.2±0.1†	37.2±0.1†
ΔTI (Ω)	–	0.14±0.24	0.39±0.25*	0.59±0.29*	0.84±0.28*	0.05±0.26	1.13±0.32*	1.53±0.34*	1.67±0.37*	2.98±0.64*

T_{sk}: mean skin temperature; T_{sl}: sublingual temperature; ΔTI: change in thoracic impedance from the baseline. Subject number=11, except for −21 mm Hg (N=9) and −40 mm Hg (N=7) LBNP in the heat-stressed condition.

* Significantly different from baseline ($P<0.05$).

† Significantly different from the normothermic condition ($P<0.05$).

significantly, due to LBNP during the heat stress trial (Table 2). Importantly, there were no differences in skin and internal temperatures, skin blood flow, or sweat rate between baseline (pre-LBNP) and the 3-min resting period between −12 and −15 mm Hg of LBNP. These findings suggest an absence of a progressive increase in thermal stress during the LBNP protocol.

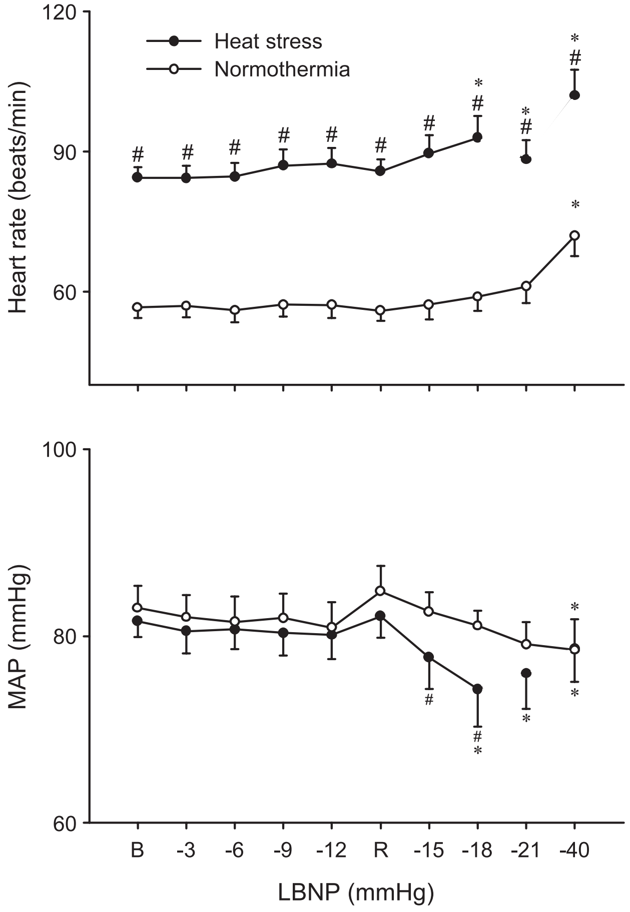

Fig. 1. Effect of heat stress on mean arterial blood pressure (MAP) and heart rate in response to progressive LBNP. N=11 for all stages of LBNP for the normothermic trial. For the heat stress trial, N=11 for baseline (B) through −18 mm Hg, N=9 for −21 mm Hg and N=7 for −40 mm Hg due to pre-syncopal symptoms and subsequent stopping of LBNP in some subjects. R: a 3-min period without LBNP between −12 and −15 mm Hg LBNP. *$P<0.05$ compared with baseline prior LBNP for the specified thermal condition. $^{\#}P<0.05$ compared with normothermia.

Application of LBNP from −3 to −12 mm Hg did not alter heart rate or MAP regardless of the thermal condition, while increases in heart rate were observed around LBNP levels of −15 to −21 during the heat stress trial (Fig. 1). Heart rate increased, and MAP decreased during −40 mm Hg LBNP in both normothermic and heat-stressed conditions (Fig. 1). Progressive LBNP did not induce significant changes in SSNA regardless of the thermal condition (Figs. 2 and 3). No correlation existed in normothermia (r=−0.06±0.15) or heat stress (r=0.14±0.15) between the change in SSNA and the change in an index of central blood volume (i.e., thoracic impedance (Pawelczyk et al., 1994; Cai et al., 2000)).

During the combination of LBNP and heat stress, slight decreases were observed in forearm and leg sweat rates (Fig. 4). However, there was no significant correlation between the change in sweat rate and the change in thoracic impedance (forearm mean r=−0.18±0.15, leg mean r=−0.40±0.18). These data suggest that these slight reductions in sweat rate were probably not due to changes in central blood volume and presumably baroreceptor unloading, but may have been due to subtle decreases in T_{sk} during LBNP. CVC did not change significantly during low levels of LBNP regardless of the thermal condition; however, higher levels of LBNP significantly decreased forearm and leg CVC during the heat stress trial (Fig. 5).

4. Discussion and conclusion

The integrated SSNA signal contains neural activity leading to vasoconstrictor, pilomotor, sudomotor and possibly vasodilator efferent responses (Janig et al., 1983); the latter two being prominent during heat stress. The major novel finding of the present study is that in

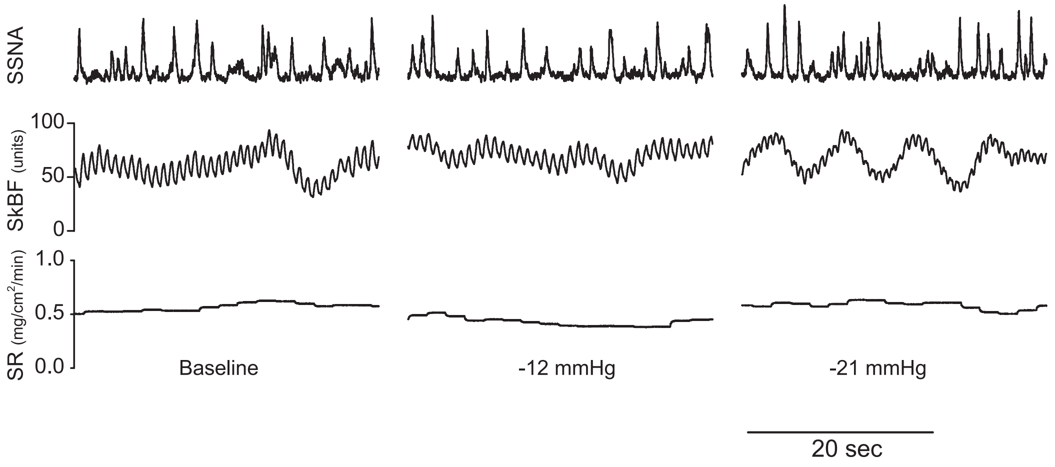

Fig. 2. Effects of LBNP at −12 and −21 mm Hg on skin sympathetic nerve activity (SSNA), skin blood flow (SkBF) and sweat rate (SR) in a representative subject during the heat stress. There was no detectable change in SSNA during progressive LBNP.

pronounced heat-stressed subjects, global baroreceptor unloading associated with low and moderate levels of LBNP did not alter total SSNA. Despite the absence of a change in SSNA, both CVC and sweat rate were reduced during LBNP in the heat. This observation suggests a functional change in the end organ response during LBNP in the heat without a detectable change in the neural signal thought to mediate that response. Consistent with prior findings (Vissing et al., 1994, 1997), baroreceptor unloading of normothermic subjects did not alter SSNA.

LBNP decreases both central blood volume and central venous pressure in normothermic (Johnson et al., 1974; Victor and Leimbach, 1987; Scherrer et al., 1988) and heat-stressed conditions (Peters et al., 2000). Some degree of arterial baroreceptors unloading likely occurs during progressive LBNP (Taylor et al., 1995; Pannier et al., 1995; Hisdal et al., 2001, 2002). Thus, it is expected that in the present procedure, both cardiopulmonary and arterial baroreceptors were unloaded by LBNP, especially during the higher levels of LBNP during the heat stress trial. Taken together, the present data do not support the hypothesis that global baroreceptor unloading alters SSNA in heat-stressed individuals.

Previously, Vissing et al. (1994) showed that in slightly warmed subjects, LBNP did not alter SSNA when changes in T_{sk} during LBNP were accounted for. In

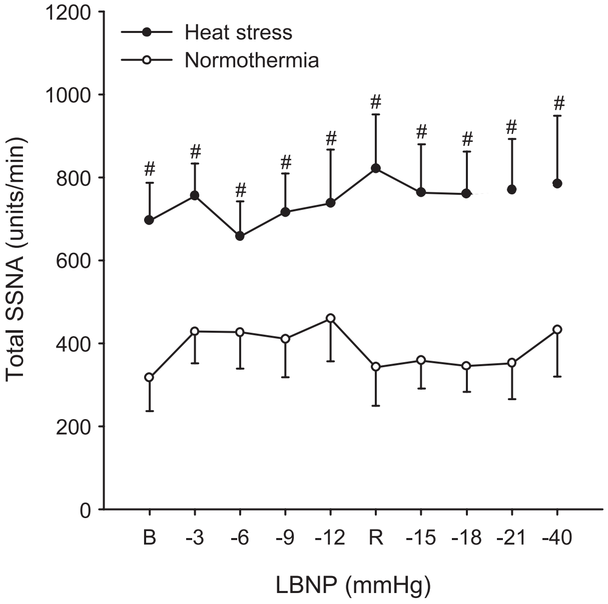

Fig. 3. Effect of heat stress on average SSNA responses to progressive LBNP. Progressive LBNP did not significantly change mean SSNA from pre-LBNP levels in either thermal condition, although whole body heating significantly elevated SSNA. R: a 3-min period without LBNP between −12 and −15 mm Hg LBNP. Subject numbers are as described in Fig. 1. $^{\#}P<0.05$ compared with normothermia.

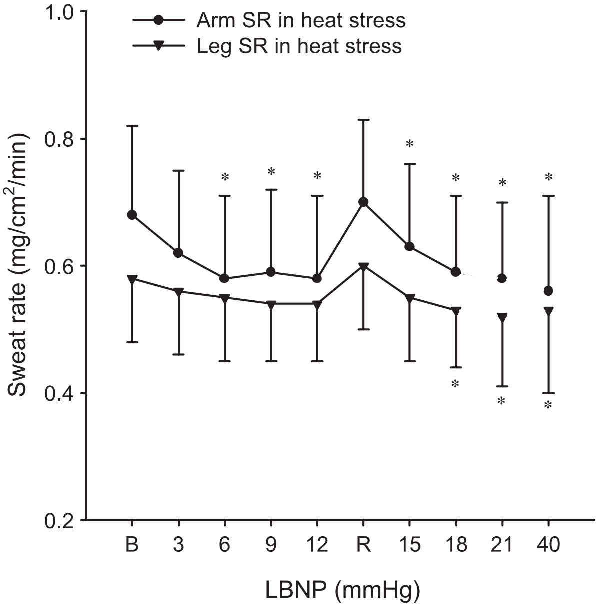

Fig. 4. Sweat rate during progressive LBNP in the heat-stressed condition. Sweat rate was recorded from dorsal forearm skin and from the area within the field of innervation of the nerve being recorded (usually dorsal foot). Subject numbers are as described in Fig. 1. $^{*}P<0.05$ compared with baseline prior LBNP for the specified location.

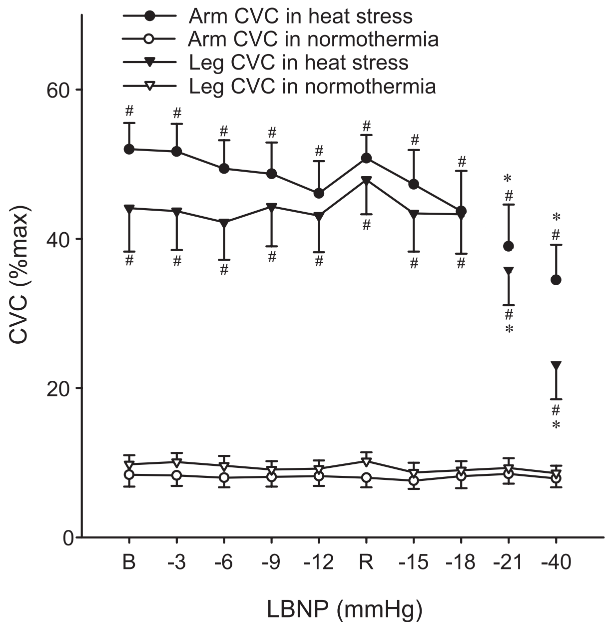

Fig. 5. Effect of heat stress on cutaneous vascular conductance (CVC) in response to progressive LBNP. Skin blood flows were recorded from dorsal forearm and from the area within the field of innervation of the nerve being recorded (usually dorsal foot). CVC was calculated from the ratio of skin blood flow to mean arterial blood pressure. Data are normalized relative to maximum CVC obtained during local heating. Subject numbers are as described in Fig. 1. *$P<0.05$ compared with baseline prior LBNP for the specified site and thermal condition. $^{\#}P<0.05$ compared with normothermia.

that study, the thermal status of the individuals was not reported other than a small increase (0.9 °C) in T_{sk}. Given this slight increase in T_{sk}, coupled with an absence of an increase in heart rate, which is typically observed during pronounced heat stress (see Table 1), it is unlikely the subjects in that study (Vissing et al., 1994) were sufficiently heated such that the SSNA signal contained significant sudomotor and/or cutaneous vasodilator components. In contrast, in the present study, heat stress led to pronounced increases in T_{sk} (~3.2 °C), T_{sl} (~0.7 °C), heart rate (~27 beats/min), CVC (about sixfold increase) and sweat rate (0.68 mg/cm^2/min), thereby strongly suggesting that the SSNA signal contained substantial sudomotor and cutaneous active vasodilator activities. Nevertheless, data from the present study extend the findings of Vissing et al. (1994) by show that LBNP during more pronounced heating also does not alter SSNA.

Dodt et al. (1995) showed that low levels of LBNP (−5 and −10 mm Hg) and upright tilting (30°) in mildly heat-stressed individuals induced a prompt inhibition of SSNA recorded from an upper limb. An explanation for differences between the present findings and those of Dodt et al. (1995) is not readily apparent. However, it may be that these differences are related to the nerve recorded (peroneal vs. forearm or radial nerves) and/or the level of heat stress. With respect to the latter, it is difficult to identify the thermal status of the participants in Dodt et al.'s study, as internal temperature was not reported and relatively small elevations in heart rate and T_{sk} were observed. An additional difference between the present study and that of Dodt et al. is the method of heating the subjects. In the aforementioned study, the subjects were heated with a heat lamp, which was removed during LBNP and presumably upright tilt. Thus, the subjects were not continually heated during the orthostatic challenge. This is in contrast to the present study in which the subjects were continually heated with the water perfused suit. It remains unclear whether these or other mechanisms are responsible for differences in the present study and that of Dodt et al. (1995).

Previously, we reported an absence of a change in SSNA during pharmacological induced changes in arterial blood pressure in heat-stressed individuals (Wilson et al., 2001). However, bolus infusions of vasoactive drugs used in that study load and unload arterial baroreceptors with minimal (Martin et al., 2004) or no effect (Dibner-Dunlap et al., 1996) on central venous pressure, and presumably cardiopulmonary baroreceptors. This is in contrast to LBNP, which more closely simulates orthostasis, being that substantial cardiopulmonary baroreceptor unloading occurs in combination with arterial baroreceptor unloading. Taken together, these observations do not support the hypothesis that primarily arterial baroreceptor unloading (Wilson et al., 2001), or a combination of cardiopulmonary and arterial baroreceptor unloading, modulate SSNA during an orthostatic challenge of normothermic or pronounced heat-stressed individuals.

Sweat rate decreased slightly (less than 0.2 mg/cm^2/min) when LBNP was applied during whole-body heating (see Fig 4). Despite the objective to minimize changes in T_{sk}, the mean T_{sk} decreased during LBNP in heat-stressed condition (see Table 2). Thus, we cannot conclusively identify whether decreases in sweat rate with LBNP were due to baroreceptor unloading (Dodt et al., 1995; Mack et al., 1995) or were due to skin cooling associated with LBNP (Solack et al., 1985; Vissing et al., 1994). However, there was an absence of a significant correlation between the change in sweat rate relative to the change in thoracic impedance (index of central blood volume) during LBNP in the heat-stressed condition. This observation suggests that the reduction in sweat rate during LBNP was probably due to skin cooling, although the present data do not completely exclude the possibility of some baroreceptor involvement.

Forearm CVC decreased during the combination of LBNP and heat stress; however, this reduction only occurred at the two highest levels of LBNP. This observation is consistent with prior findings by us and others (Crandall et al., 1996; Peters et al., 2000), which suggests that moderate arterial baroreceptor unloading and/or substantial cardiopulmonary baroreceptor unloading must occur to reduce CVC in heat-stressed individuals.

It is interesting to note that despite clear reductions in sweat rate and CVC at higher levels of LBNP in the heat-stressed condition, SSNA innervating the area from which sweat rate and CVC were measured did not change. Thus, the SSNA signal was not consistent with the efferent response (i.e., sweat rate or CVC) thought to be controlled by that neural signal. Multiple effector activities within the integrated SSNA signal may be responsible for the absence of a change in total SSNA during LBNP while heat-stressed despite reductions in CVC and sweat rate. For example, it is possible that the sudomotor/vasodilator component of SSNA decreased during LBNP, while the vasoconstrictor component of SSNA increased; the sum of which resulted in a lack of measurable change in integrated SSNA. Unfortunately, these components within multiunit SSNA recording cannot be separated by current technology, and thus, we are unable to confirm this intriguing hypothesis.

In conclusion, the present data show for the first time that global baroreceptor unloading does not alter SSNA in pronounced heat-stressed individuals, when a significant component of the SSNA signal contains sudomotor and possibly cutaneous active vasodilator activities. However, CVC and sweat rate were reduced during moderate levels of LBNP when the subjects were heat-stressed. These data suggest integrated sympathetic outflow to skin should not be used as an indicator of baroreflex control of skin blood flow and sweating in normothermic and heat-stressed individuals.

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