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## Mean body temperature does not modulate eccrine sweat rate during upright tilt

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**Wilson, Thad E., Jian Cui, and Craig G. Crandall.** Mean body temperature does not modulate eccrine sweat rate during upright tilt. *J Appl Physiol* 98: 1207–1212, 2005. First published December 3, 2004; doi:10.1152/jappphysiol.00648.2004.—Conflicting reports exist about the role of baroreflexes in efferent control of eccrine sweat rate. These conflicting reports may be due to differing mean body temperatures between studies. The purpose of this project was to test the hypothesis that mean body temperature modulates the effect of head-up tilt on sweat rate and skin sympathetic nerve activity (SSNA). To address this question, mean body temperature ( $0.9 \cdot$  internal temperature +  $0.1 \cdot$  mean skin temperature), SSNA (microneurography of peroneal nerve,  $n = 8$ ), and sweat rate (from an area innervated by the peroneal nerve and from two forearm sites, one perfused with neostigmine to augment sweating at lower mean body temperatures and the second with the vehicle,  $n = 12$ ) were measured in 13 subjects during multiple  $30^\circ$  head-up tilts during whole body heating. At the end of the heat stress, mean body temperature ( $36.8 \pm 0.1$  to  $38.0 \pm 0.1^\circ\text{C}$ ) and sweat rate at all sites were significantly elevated. No significant correlations were observed between mean body temperature and the change in SSNA during head-up tilt ( $r = 0.07$ ;  $P = 0.62$ ), sweating within the innervated area ( $r = 0.06$ ;  $P = 0.56$ ), sweating at the neostigmine treated site ( $r = 0.04$ ;  $P = 0.69$ ), or sweating at the control site ( $r = 0.01$ ;  $P = 0.94$ ). Also, for each tilt throughout the heat stress, there were no significant differences in sweat rate (final tilt sweat rates were  $0.69 \pm 0.11$  and  $0.68 \pm 0.11 \text{ mg} \cdot \text{cm}^{-2} \cdot \text{min}^{-1}$  within the innervated area;  $1.04 \pm 0.16$  and  $1.06 \pm 0.16 \text{ mg} \cdot \text{cm}^{-2} \cdot \text{min}^{-1}$  at the neostigmine-treated site; and  $0.85 \pm 0.15$  and  $0.85 \pm 0.15 \text{ mg} \cdot \text{cm}^{-2} \cdot \text{min}^{-1}$  at the control site, for supine and tilt, respectively). Hence, these data indicate that mean body temperature does not modulate eccrine sweat rate during baroreceptor unloading induced via  $30^\circ$  head-up tilt.

microneurography; whole body heating; baroreceptor unloading

DURING HEAT STRESS, EVAPORATION of sweat secreted via eccrine sweat glands is vital to human thermoregulation. Sweat rates can be as high as 3.7 l/h systemically (3) and  $>10$  nl/min from a single eccrine gland (28). Consequently, humans can lose as much as 10–11 l/day of fluid in a hot ambient environment (1). These high sweat rates associated with passive-heat or exercise-heat stress can be problematic in the maintenance of fluid balance and therefore contribute to heat illnesses and decreases in work performance (8).

Sweat rate is controlled by means of both thermal and nonthermal factors (14, 31). Vascular pressure and/or volume has been proposed as a nonthermal factor modulating sweat rate. Fortney et al. (15) identified that acute hypovolemia

decreased the slope of the relationship between sweat rate and internal temperature compared with euvoletic conditions. Decreases in the rate of change of sweating have also been observed with acute baroreceptor unloading [via lower body negative pressure (LBNP)] during exercise-heat stress (20) and during whole body heating (32). Dodt et al. (12) extended this research by identifying decreases in skin sympathetic nerve activity (SSNA) and electrodermal activity (an index of sweating) during both low levels of LBNP and  $30^\circ$  head-up tilt in mildly heated subjects. These data suggest that acute baroreceptor unloading decreases eccrine sweat gland output, perhaps in an effort to maintain fluid balance in response to a perturbation that reduces central blood volume and/or blood pressure.

This concept, however, has been challenged by others. Vissing et al. (35) did not observe changes in SSNA or an index of sweat rate (i.e., electrodermal activity) during LBNP when cooling associated with the application of LBNP was controlled. Recently, we used pharmacological means to alter blood pressure to identify whether primarily arterial baroreceptor unloading modulates SSNA or sweat rate. During normothermia and pronounced heat stress conditions, neither acute baroreceptor unloading and loading nor more prolonged baroreceptor unloading significantly altered SSNA or sweat rate (38).

The precise reason(s) for differences between these studies remains unclear. One possibility may be related to mean body temperature before engaging the baroreflexes. Heat stress progressively decreases central venous pressure and presumably central blood volume (7, 9, 22, 26), and acute baroreceptor unloading by means of head-up tilt or LBNP also decreases central venous pressure and central blood volume (6, 21, 22). It is reasonable to postulate that, once central blood volume is sufficiently decreased via whole body heating, further decreases in central blood volume would result in less unloading of low-pressure baroreceptors. Therefore, early in a heat stress, low-pressure baroreceptors might retain the ability to modulate sweat rate but not after more pronounced heating. If such a hypothesis is correct, this may explain some of the discrepancies in the aforementioned studies. Accordingly, the purpose of this study was to test the hypothesis that mean body temperature modulates sweat rate and SSNA during low levels of baroreceptor unloading induced by head-up tilt.

Because sweating is typically not present during normothermic conditions, sweat rate was also measured within an area treated with an acetylcholinesterase inhibitor, neostigmine.

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Previously, our laboratory showed that local administration of this substance augments sweating in normothermic and heat stress conditions during perturbations known to increase sweating (30). Thus, if head-up tilt alters sweating, changes in sweating should occur earlier (i.e., lower mean body temperatures) at the neostigmine-treated site relative to the other sites.

## METHODS

**Subjects.** Thirteen healthy subjects (5 men, 8 women) completed this project. The participants' mean age was  $30 \pm 2$  yr (range = 22–42 yr), height was  $169 \pm 3$  cm (range = 152–185 cm), weight was  $67 \pm 4$  kg (range = 50–94 kg), and body surface area (13) was  $1.34 \pm 0.06$  m<sup>2</sup> (range = 1.04–1.78 m<sup>2</sup>). The protocol and informed consent received institutional approval from the University of Texas Southwestern Medical Center at Dallas and Presbyterian Hospital of Dallas. Written, informed consent was obtained from all participants before they enrolled in this study.

**Protocol.** Participants were exposed to whole body heating by perfusing 46°C water through a tube-lined suit (Carleton Technologies, Tampa Bay, FL) worn by each subject. The duration of this heat stress was 90 min or until core temperature increased by 1.0°C. Baroreceptor unloading was accomplished by means of a motorized tilt bed (Omni Technologies, Valley City, ND), which smoothly and rapidly raised the participant to 30° head-up tilt. This low level of tilt was used to cause a mild shift in central blood volume and is similar to the tilting protocol used by Dodt et al. (12). Tilting was performed during the last 2 min of every 10-min period throughout the heat stress.

**Measurements.** Heart rate was obtained from an electrocardiogram (SpaceLabs, Redmond, WA), with the signal interfaced with a cardiometer (CWE, Ardmore, PA). Arterial blood pressure was measured from the upper arm via electrophygmomanometry (Sun-Tech, Raleigh, NC). Transthoracic impedance (Biopac Systems, Santa Barbara, CA) was measured and used as an index of central blood volume (6, 16, 21). Relative change in transthoracic impedance, from supine to head-up tilt, was used to denote fluid volume shifts during tilting (e.g., an increase in transthoracic impedance indicates a decrease in thoracic fluid volume). Internal or core temperature was indexed from an ingestible pill telemetry system (HTI Technologies, Palmetto, FL;  $n = 11$ ) or a thermocouple placed in the sublingual sulcus ( $n = 2$ ). Telemetry pill measures correlate well with other internal temperature measures such as esophageal temperature (24). Mean skin temperature was measured via the weighted average of six thermocouples attached to the skin (33). Mean body temperature was calculated as  $0.9 \cdot \text{core} + 0.1 \cdot \text{mean skin temperature}$  (23, 37).

Sweat rate was measured using capacitance hygrometry (Viasala, Woburn, MA) by perfusing 100% nitrogen at a flow rate of 500 ml/min through a ventilated capsule. One capsule (surface area = 2.83 cm<sup>2</sup>) was attached within the field of innervation of the recorded nerve (see below). The other capsules (surface area = 0.50 cm<sup>2</sup>) were attached directly above 10-mm microdialysis membranes placed in dorsal forearm skin. These two microdialysis membranes ( $n = 12$ ) were placed within the dermal layer of the skin as previously reported (18, 29). A minimum of 60 min elapsed before the beginning of the experimental protocol to allow for the hyperemic response to subside after probe insertion (2). Ten minutes before the first normothermic tilt, 10 μM of neostigmine dissolved in lactated Ringer solution were perfused through one of the membranes to sensitize the sweating response at that site (29, 30). The other membrane was perfused with the vehicle (i.e., lactated Ringer solution).

Multifiber recordings of SSNA were successfully acquired throughout the entire procedure in eight subjects. SSNA was measured from a tungsten microelectrode inserted in the common peroneal nerve. A reference electrode was placed subcutaneously at a distance of 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 (11, 17). In brief, these criteria included the following: 1) light stroking of the skin within the innervated region resulted in afferent discharge, 2) deep inspiration or arousal stimuli resulted in bursts of non-pulse-synchronous activity, and 3) signal-to-noise ratio >3:1. The nerve signal was amplified, passed through a filter with a bandwidth of 700–2,000 Hz, and integrated with a time constant of 0.1 s (Iowa Bioengineering, Iowa City, IA). Mean voltage neurograms were visually displayed on an oscilloscope, were recorded on a data-acquisition system (model MP150, Biopac, Santa Barbara, CA) and chart recorder, as well as routed to a loudspeaker for monitoring throughout the study. SSNA was quantified by integrating the area under the bursts via computer software (AcqKnowledge, Santa Barbara, CA). Because of subtle site shifts that can occur during lengthy multiunit neural recordings, SSNA is reported as a percent change from supine to 30° head-up tilt for each tilt.

**Data analysis.** Data were acquired at 200 Hz throughout the protocol. Linear regression analysis was performed on the change in SSNA and sweat rate between supine and 30° head-up tilt relative to mean body temperature. The rationale for this analysis was to identify whether there was a negative relationship between the magnitude of change in sweat rate and SSNA due to head-up tilting relative to mean body temperature. Subjects attained heat stress end points (i.e., increase in internal temperature of 1.0°C or 90 min of whole body heating) at varying number of tilts during whole body heating. All subjects completed a minimum of five tilts during heat stress. These data were analyzed via repeated-measures ANOVA with main factors of thermal condition (i.e., tilt number) and tilt. Data are also reported as normothermia and end heat stress (the last tilt before reaching test end point) during 30° head-up tilts. All values are reported as means  $\pm$  SE. The  $\alpha$ -level for all statistical analyses was set at 0.05.

## RESULTS

Whole body heating significantly increased internal temperature  $0.8 \pm 0.3$ °C and mean skin temperature  $4.6 \pm 0.3$ °C (Table 1). Telemetry pill temperature measures were obtained on 11 of 13 participants; in the remaining 2 subjects, oral temperature was substituted for telemetry pill temperature for the calculation of mean body temperature. Mean body temperature steadily rose throughout whole body heating and by the end of the heat stress had increased  $1.2 \pm 0.1$ °C (Table 1).

Whole body heating resulted in large increases in heart rate ( $35 \pm 2$  beats/min), with no change in mean arterial blood

Table 1. Effects of 30° head-up tilt during normothermia and the final tilt during heat stress

	Normothermia		Heat Stress	
	Supine	Tilt	Supine	Tilt
T <sub>co</sub> , °C	37.1 ± 0.1	37.1 ± 0.1	37.9 ± 0.1*	37.9 ± 0.1*
T <sub>sk</sub> , °C	34.3 ± 0.1	34.4 ± 0.1	38.9 ± 0.1*	38.9 ± 0.1*
T <sub>b</sub> , °C	36.8 ± 0.1	36.8 ± 0.1	38.0 ± 0.1*	38.0 ± 0.1*
SR <sub>SSNA</sub> , mg·cm <sup>-2</sup> ·min <sup>-1</sup>			0.69 ± 0.11*	0.68 ± 0.11*
SR <sub>neo</sub> , mg·cm <sup>-2</sup> ·min <sup>-1</sup>			1.04 ± 0.16*	1.06 ± 0.16*
SR <sub>con</sub> , mg·cm <sup>-2</sup> ·min <sup>-1</sup>			0.85 ± 0.15*	0.85 ± 0.15*
Heart rate, beats/min	58 ± 3	61 ± 3	95 ± 3*	109 ± 4*†
MAP, mmHg	86 ± 2	88 ± 1	85 ± 1	83 ± 1*

Values are means  $\pm$  SE. T<sub>co</sub>, telemetry pill temperature ( $n = 11$ ) or sublingual temperature ( $n = 2$ ); T<sub>sk</sub>, mean skin temperature; T<sub>b</sub>, mean body temperature; SR<sub>SSNA</sub>, sweat area within the area innervated by microneurography (SSNA, skin sympathetic nerve activity); SR<sub>neo</sub>, sweat rate site perfused with neostigmine; SR<sub>con</sub>, sweat rate site perfused in vehicle control; MAP, mean arterial pressure. \*Significant difference from normothermia,  $P < 0.05$ . †Significant difference from supine posture in the specified thermal condition,  $P < 0.05$ .

pressure (Table 1). In normothermia, head-up tilt did not change heart rate or mean arterial blood pressure. In contrast, the final head-up tilt during heat stress resulted in significant increases in heart rate and decreases in mean arterial blood pressure. Head-up tilt increased thoracic impedance in both normothermic and heat stress conditions. An increase in thoracic impedance indicates a decrease in central blood volume. There were no differences in the percent change in thoracic impedance during head-up tilt between normothermia ( $4.2 \pm 0.2\%$ ) and heat-stressed conditions ( $4.2 \pm 0.1$ ,  $4.4 \pm 0.2$ ,  $3.5 \pm 0.3$ ,  $4.0 \pm 0.2$ , and  $3.6 \pm 0.2\%$  for the first five consecutive heat tilts, respectively).

A representative neurogram from one subject is illustrated in Fig. 1. This figure shows the typical increase in SSNA during heat stress. No significant differences in percent change SSNA were observed during baroreceptor unloading in normothermia or heat stress conditions. There was no significant change in SSNA relative to supine baseline (baseline = 100%) during normothermia ( $94 \pm 7\%$ ) and during the subsequent five heat stress tilts ( $96 \pm 7$ ,  $102 \pm 5$ ,  $99 \pm 10$ ,  $105 \pm 7$ , and  $106 \pm 10\%$ , respectively). No relationship existed between the percent change in SSNA from supine to  $30^\circ$  head-up tilt relative to mean body temperature ( $r = 0.07$ ). This lack of a correlation strongly indicates that mean body temperature does not modify SSNA responses to  $30^\circ$  head-up tilt.

Heat stress significantly increased sweat rate at all sites (Table 1). Similar to SSNA, sweat rates were not significantly altered during tilt (Fig. 2). Sweating threshold temperature (indexed from telemetry pill temperature) was significantly lower at the neostigmine-treated site ( $37.24 \pm 0.12^\circ\text{C}$ ) compared with the control site ( $37.38 \pm 0.11^\circ\text{C}$ ). This observation corresponded to sweating occurring  $8:08 \pm 1:23$  (min:s) earlier at the neostigmine site compared with the control site during heat stress. The neostigmine-perfused site also showed significantly higher sweat rates compared with the control site. The primary justification for using neostigmine was to identify, whether at low levels of heating (i.e., before threshold temperature for the onset of sweating at the control site), the effects of baroreceptor unloading may be identified at this sensitized site. This was not the case, because sweat rate at the neostigmine-treated site was not affected by head-up tilt, even at levels of heating that resulted in sweating at the neostigmine-treated site but not at the control site. There was no relationship between the change in sweat rate during head-up tilt and mean body temperature (dorsal foot:  $r = 0.06$ ; neostigmine site:  $r = 0.04$ ; control site:  $r = 0.01$ ). Additionally, no significant differences were observed in sweat rate within the area of afferent innervation on the dorsal foot during head-up tilt in normothermia or heat stress conditions (Fig. 3).

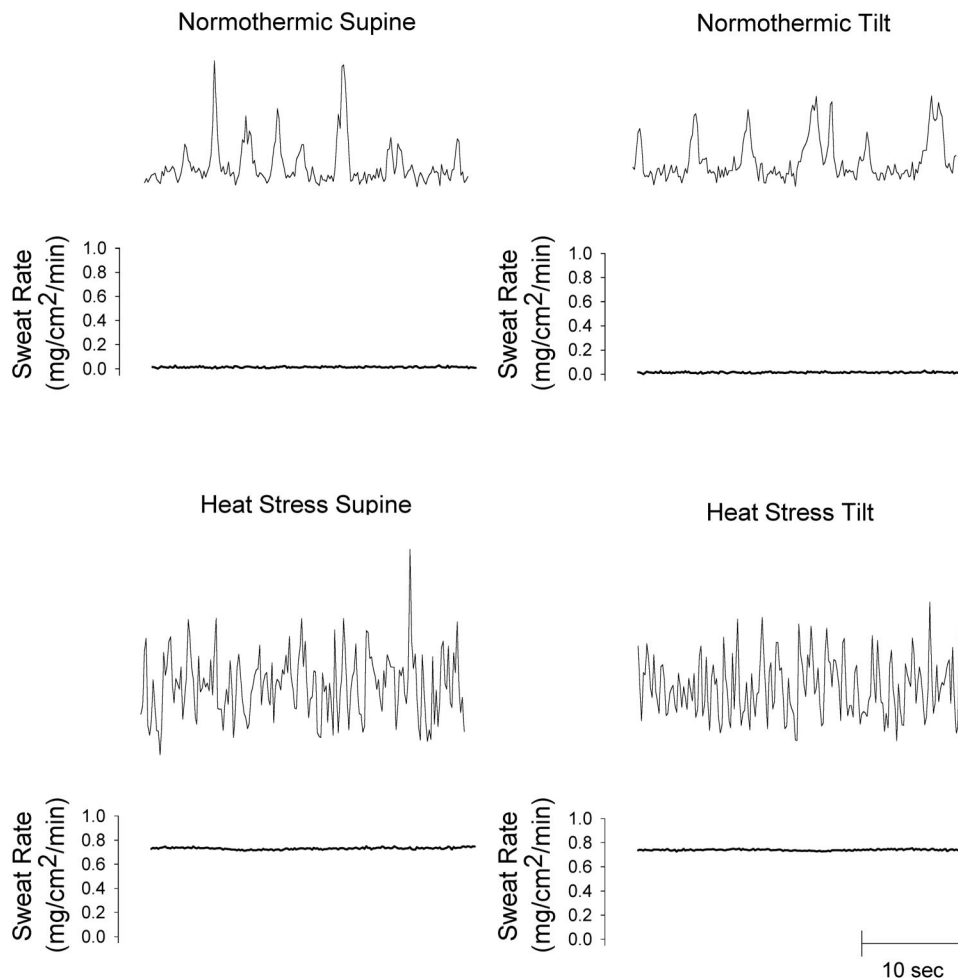


Fig. 1. Representative skin sympathetic neurograms and sweat rate from area innervated by the recorded nerve during supine and  $30^\circ$  head-up tilt in normothermia and during the final heat stress tilt.

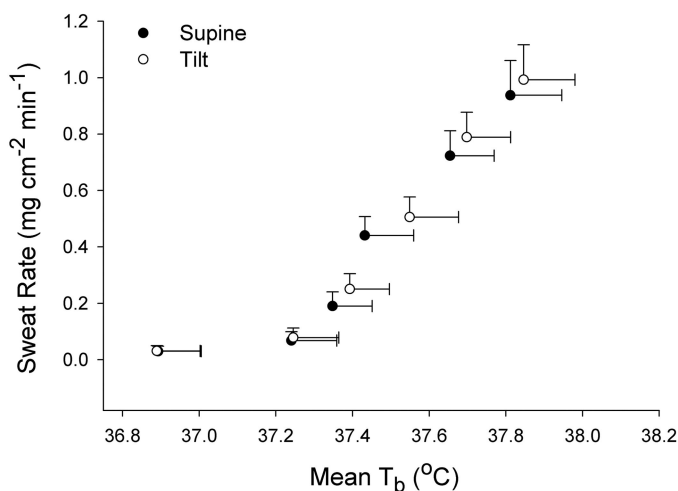
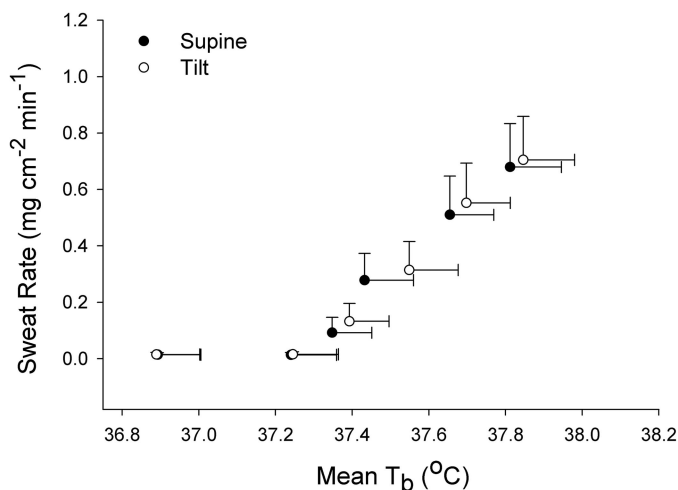
**Neostigmine Site**

**Control Site**


Fig. 2. Comparison of forearm sweat rate from neostigmine and control sites during each 30° head-up tilt that all subjects completed. *Tilt 1* was during normothermia, whereas *tilts 2–6* were during heat stress; each tilt is separated by 10 min.  $T_b$ , body temperature. Significant increases in sweat rate were observed during heat stress ( $P < 0.05$ ), but no significant differences were observed between supine and 30° head-up tilt at any time point at either site.

**DISCUSSION**

Heat stress causes progressive decreases in central venous and right atrial mean pressures, and presumably central blood volume (7, 9, 26, 27). Given this response, we hypothesized that the range through which cardiopulmonary baroreceptors would elicit a response (i.e., changes in SSNA and sweat rate) by head-up tilt would be minimized as central blood volume decreased during the heat stress. Thus it is possible that a high thermal load, and therefore low central blood volume, may reduce the capability of baroreceptors (primarily cardiopulmonary baroreceptors) to modulate SSNA and/or sweating. The present study, however, argues against this hypothesis. The major findings of the present study are 1) mean body temperature does not alter sweat rate or SSNA responses to 30° head-up tilt, and 2) sensitizing sweating responses via acetylcholinesterase inhibition did not unmask an effect of barore-

ceptor modulation of sweat rate. These data indicate that, although absolute sweat rate is integrally linked with mean body temperature, there is an absence of a significant change in SSNA and sweat rate during baroreceptor unloading via 30° head-up tilt across a wide range of mean body temperatures. Hence, these data do not support the hypothesis that mean body temperature modulates eccrine sweating during baroreceptor unloading induced via head-up tilt in humans.

Conflicting reports exist concerning the effect of baroreceptor unloading on sweat rate. In the present study, we observed no effect of baroreceptor unloading via 30° head-up tilt on SSNA or sweat rate, regardless of mean body temperature. This observation is consistent with our prior study in which arterial blood pressure was changed via pharmacological means without resulting in changes in either SSNA or sweat rate in heat-stressed subjects (38). In contrast to our prior and present findings, other investigators report that baroreceptor unloading via LBNP either decreases sweating or attenuates its rate of rise during heat stress (20, 32). However, Vissing (34) questioned the use of LBNP when assessing cutaneous responses in heat-stressed subjects due to the possible confounding influence of convective skin cooling during the LBNP procedure. When the effects of skin cooling during LBNP were controlled, neither SSNA nor an index of sweating was altered (35).

Therefore, skin surface cooling during LBNP may explain discrepancies between the aforementioned studies, as local skin temperature has pronounced effects on sweat rate (4, 10). However, this possibility is insufficient to explain the findings of Dodt et al. (12), who, in contrast to the present findings, observed decreases in SSNA and electrodermal activity during 30° head-up tilt, which would not have altered skin temperature. A few key methodological differences between the present study and that reported by Dodt et al. may explain the apparent conflicting observations. First, Dodt et al. measured SSNA from the posterior cutaneous nerve of the forearm or the cutaneous fascicles of the radial nerve, and sweat rate was

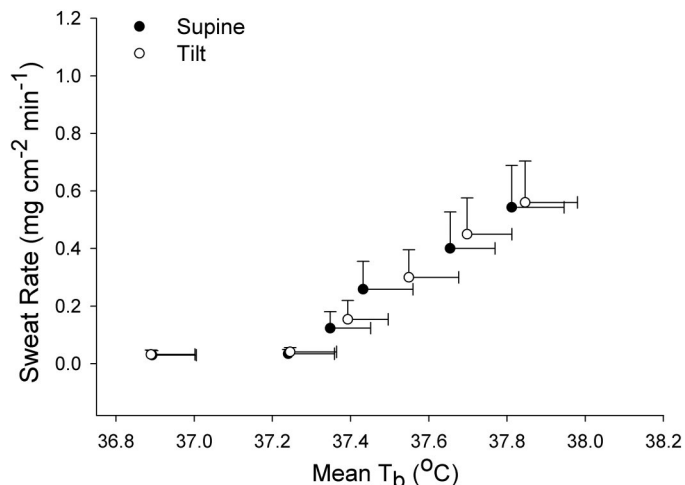


Fig. 3. Effect of thermal load on dorsal foot sweat rate during each 30° head-up tilt that all subjects completed. *Tilt 1* was during normothermia, whereas *tilts 2–6* were during heat stress; each tilt is separated by 10 min. Significant increases in sweat rate were observed during heat stress ( $P < 0.05$ ), but no significant differences were observed between supine and 30° head-up tilt at any time point at either site.

indexed by recording skin electrical resistance within the field of innervation of the recorded nerve. In the present study, SSNA was measured from the peroneal nerve, and sweat rate was measured by capacitance hygrometry. Thus it is possible that differing SSNA responses could be related to different nerves being recorded, whereas differences in sweating responses could be related to differences between measuring electrodermal activity and directly measuring evaporative water loss. Second, in the study by Dodt et al., the subjects were heated with a heating lamp. Although thermal responses to that heat stress were not reported, it was reported that the subjects were sweating profusely before LBNP or head-up tilt. However, the heating lamp was removed before LBNP and presumably head-up tilt. This is in contrast to the present study in which the subjects were continually heated throughout the experimental protocol. Nevertheless, discrepancies between the present study and that of Dodt et al. cannot be attributed to differences in subjects' mean body temperature during baroreceptor unloading.

Acetylcholinesterase inhibitors increase sweat production both systemically and locally (29, 36), and they have been used to sensitize sweat glands during conditions of low thermal load where sweating would normally not be observed (30). Such a response was observed in the present experiment, given that sweating responses during the heat stress at the neostigmine-treated site occurred earlier and to a greater extent relative to an adjacent untreated site. Assessment of sweat rate at a neostigmine-treated site was performed to identify whether baroreceptor unloading was capable of altering sweat rate early in the heat stress, before sweating was observed at the control site. However, neither the neostigmine-treated nor the vehicle control site showed any change in sweat rate during 30° head-up tilt, regardless of mean body temperature. This lack of a decrease in sweat rate at the neostigmine-treated site during baroreceptor unloading further supports the concept of an absence of baroreceptor control of sweat rate.

Although the present and prior findings suggest that baroreceptors do not modulate peroneal SSNA or eccrine sweat rate (35, 38), some studies have identified a link between cardiac interval, respiration, and blood pressure fluctuations for both multi- and single-unit SSNA signals of sudomotor origin (5, 19). It is unclear why there is a link between these more subtle pressure fluctuations and SSNA but not during more pronounced blood pressure challenges such as head-up tilt or pharmacologically induced changes in blood pressure (38). It could be that tonic SSNA firing is controlled by central sympathetic centers that are also involved in cardiovascular and respiratory regulation without being modulated by a baroreflex feedback loop. However, this hypothesis warrants further investigation.

**Limitation to the interpretation of the data.** Relatively minor levels of baroreceptor unloading occurred with 30° head-up tilt. This tilt angle was selected to be consistent with the mode of baroreceptor unloading previously used by Dodt et al. (12), who reported reductions in SSNA during tilt in heat-stressed subjects. Low levels of head-up tilt (20–40°) induced decreases in central blood volume (21, 25). Consistent with those observations, 30° head-up tilt decreased central blood volume in both thermal conditions, as indexed by thoracic impedance. Nevertheless, it remains unknown whether the absence of a baroreflex effect in modulating SSNA and sweat rate during tilt

in the present study was related to this level of baroreceptor unloading. Thus the present study does not exclude the possibility that, if greater levels of baroreceptor unloading were performed, a body temperature-dependent baroreflex modulation of SSNA and/or sweat rate may be observed. However, such a protocol would be challenging, given known reductions in orthostatic tolerance in heat-stressed individuals (27).

Because of the sequential nature of tilting during heat stress (pretilt measurements are always completed before tilt measurements), we investigated the possibility that subtle increases in mean body temperature during the 2-min tilt, compared with the pretilt period, may have affected the interpretation of the results. To test this potential limitation, responses immediately after tilt were compared with values during tilt. These posttilt values were not significantly different relative to tilt values, indicating that the slightly increased thermal load during the 2-min tilt did not confound the interpretation of the results.

**Summary.** The present data suggest that mean body temperature does not modulate eccrine sweat rate during 30° head-up tilt. This result was observed at sites treated with an acetylcholinesterase inhibitor to cause an earlier onset of sweating, as well as control sites. Although it is clear that sweat rate and SSNA are integrally linked with changes in mean body temperature, we were unable to observe an effect of baroreceptor unloading on either sweat rate or SSNA. Thus mean body temperature differences are insufficient to explain discrepancies between previous observations investigating the effects of baroreceptor unloading and sweating in humans.

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