

Evidence for metaboreceptor stimulation of sweating in normothermic and heat-stressed humans

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1. Isometric handgrip (IHG) exercise increases sweat rate and arterial blood pressure, and both remain elevated during post-exercise ischaemia. The purpose of this study was to identify whether the elevation in arterial blood pressure during post-exercise ischaemia contributes to the increase in sweating.
2. In normothermia and during whole-body heating, 2 min IHG exercise at 40% maximal voluntary contraction, followed by 2 min post-exercise ischaemia, was performed with and without bolus intravenous administration of sodium nitroprusside during the ischaemic period. Sodium nitroprusside was administered to reduce blood pressure during post-exercise ischaemia to pre-exercise levels. Sweat rate was monitored over two microdialysis membranes placed in the dermal space of forearm skin. One membrane was perfused with the acetylcholinesterase inhibitor neostigmine, while the other was perfused with the vehicle.
3. In normothermia, IHG exercise increased sweat rate at the neostigmine-treated site but not at the control site. Sweat rate remained elevated during post-exercise ischaemia even after mean arterial blood pressure returned to the pre-IHG exercise baseline. Subsequent removal of the ischaemia stimulus returned sweat rate to pre-IHG exercise levels. Sweat rate during post-exercise ischaemia without sodium nitroprusside administration followed a similar pattern.
4. During whole-body heating, IHG exercise increased sweat rate at both neostigmine-treated and untreated sites. Similarly, regardless of whether mean arterial blood pressure remained elevated or was reduced during post-exercise ischaemia, sweat rate remained elevated during the ischaemic period.
5. These results suggest that sweating in non-glabrous skin during post-IHG exercise ischaemia is activated by metaboreflex stimulation and not via baroreceptor loading.

Increases in sweating during heat stress occur secondarily to increases in internal and skin temperatures. In addition to these thermal factors, non-thermal factors, such as central command, muscle mechano/metaboreceptor activation and baroreflexes, have been shown to modulate sweating during whole-body heating and during exercise (Van Beaumont & Bullard, 1963; Solack *et al.* 1985; Mack, 1995; Yamazaki *et al.* 1996; Kondo *et al.* 1999; Kondo, 2001). However, independent roles of these non-thermal factors in the control of sweating, particularly during exercise, remain unclear.

Isometric handgrip (IHG) exercise increases heart rate, arterial blood pressure, and muscle and skin sympathetic nerve activity (Mark *et al.* 1985; Vissing *et al.* 1991; Joyner, 1992). In warm conditions IHG exercise increased

sweat rate due to non-thermal factors since internal and skin temperatures did not increase during the bout of IHG exercise (Kondo *et al.* 1999). Moreover, this increase in sweating was attributed to muscle metaboreceptor stimulation since sweat rate remained elevated during post-IHG exercise ischaemia under moderately warmed and hyperthermic conditions (Crandall *et al.* 1998; Kondo *et al.* 1999). However, given prior findings that baroreceptors modulate sweating (Mack *et al.* 1995), coupled with elevations in mean arterial blood pressure during exercise and post-exercise ischaemia, it may be that baroreceptor loading contributes to increases in sweat rate during IHG exercise and post-exercise ischaemia. To our knowledge, the contribution of elevated arterial blood pressure during exercise on the control of sweat rate remains unknown.

Under normothermic conditions IHG exercise does not increase sweat rate in non-glabrous skin (Crandall *et al.* 1995, 1998) despite prior findings demonstrating that this perturbation increases skin sympathetic nerve activity without causing sustained changes in cutaneous vascular resistance (Vissing *et al.* 1991). We recently reported that local inhibition of acetylcholine hydrolysis, via microdialysis administration of neostigmine, modified sweating responses in non-glabrous skin resulting in earlier increases in sweating during local acetylcholine administration and during whole-body heating (Shibasaki & Crandall, 2001). Moreover, in that study some subjects showed measurable sweat explosions at the neostigmine-treated site prior to the administration of acetylcholine or prior to the onset of heating. It is possible that the aforementioned increases in skin sympathetic nerve activity during IHG exercise observed by Vissing *et al.* (1991) was sudomotor in nature; however, insufficient quantities of acetylcholine were released from these nerves relative to the quantity necessary to overcome acetylcholine hydrolysis sufficient to evoke measurable sweating responses. Thus, it remains unclear whether responses associated with IHG exercise would increase sweat rate under conditions when acetylcholine hydrolysis was inhibited. Such a finding would provide new insight pertaining to the control of sweating in non-glabrous skin during exercise when neither skin nor core temperatures are elevated.

Therefore the primary purpose of this study was twofold: (1) to test the hypothesis that baroreceptor loading during post-exercise ischaemia contributes to the elevation in sweating rate due to muscle metaboreceptor stimulation; and (2) to test the hypothesis that IHG exercise, and specifically muscle metaboreceptor stimulation, under normothermic conditions (i.e. normothermic skin and internal temperatures) is capable of increasing sweat rate if acetylcholine hydrolysis is inhibited.

METHODS

Nine healthy subjects (eight men, one woman; aged 22–37 years) participated in this study. To minimize potential variability in responses associated with the menstrual cycle, the female subject was tested during the early follicular phase of the menstrual cycle. All subjects were healthy and were of normal weight (73 ± 2 kg) and height (173 ± 3 cm). Each subject was informed of the purpose and risks of this study before providing their written consent. The consent form was approved by the Institutional Review Boards at the University of Southwestern Medical Center at Dallas and Presbyterian Hospital of Dallas and conforms with the Declaration of Helsinki.

Upon entering the laboratory (room temperature, 22–23°C), each subject was instrumented for the measurement of mean skin temperature, from the weighted electrical average of six thermocouples placed on the skin (Physitemp, BAT-10), and electrocardiogram. The subject was then dressed in a suit lined with tubes through which water could be perfused to control skin temperature. The suit covered the entire body surface except for the head, feet and forearms. Each subject rested in the supine position, while two microdialysis probes were placed at least 3 cm apart in the dermal space of dorsal forearm

skin. The depth of probe placement was not determined, although Kellogg *et al.* (1999) using the same procedure reported a placement depth of 0.3–1.0 mm. The probes were constructed in our laboratory from a semi-permeable cellulose membrane (Spectrum Inc., 18000 molecular weight cut off) glued between two polyimide tubes (MicroLumen Inc.) and reinforced with a 51 μm diameter stainless-steel wire placed in the lumen of the membrane and tubes (Crandall *et al.* 1997; Kellogg *et al.* 1999). The membrane window for each probe was 10 mm. To place the probe, a 25 gauge hypodermic needle was inserted into the dermal space without local anaesthesia, the tip exiting 20–25 mm away from the point of entry. The microdialysis probe was then inserted through the lumen of the needle and the needle was withdrawn, leaving the probe in place. After placement, the probes were perfused with Ringer solution at a rate of $2 \mu\text{l min}^{-1}$. Chambers with a small window (10 mm \times 5 mm, i.e. surface area, 0.5 cm^2) were positioned over each membrane to allow measurement of sweat rate by the ventilated capsule method (Shibasaki & Crandall, 2001), using compressed nitrogen as the perfusion gas, delivered at a rate of 150 ml min^{-1} . Capsule positioning was aided by the use of markings on the polyimide tubing that indicated the centre of the probe membrane. The effluent gas was sensed via a humidity/temperature probe (HMP 35E, Vaisala Inc.), positioned 1.2 m from the capsule on the skin. The humidity/temperature probe interfaced with a humidity data processor (HMI 38, Vaisala Inc.) which calculated absolute humidity from the measurement of relative humidity and temperature at the probe. Absolute humidity (expressed as mg m^{-3}) was then converted to sweat rate (expressed as $\text{mg cm}^{-2} \text{ min}^{-1}$) using the following formula:

$$\text{Sweat rate} = \text{absolute humidity} \times \text{nitrogen flow/chamber window area.}$$

A catheter was then placed in an antecubital vein. Arterial blood pressure was measured by arterial tonometry (Colin Inc.) over the radial artery at the wrist of the hand not performing IHG exercise. Mean arterial blood pressure was calculated by integration of the arterial waveform. Sublingual temperature was measured with a thermister and used as an index of internal temperature. Heart rate was obtained from the electrocardiogram signal interfaced with a cardiometer (CWE, Inc.). Data collection did not begin for at least 60 min after microdialysis probe placement to allow for the hyperaemic response associated with probe placement to subside. Following this period of time, the acetylcholinesterase inhibitor neostigmine (10 μM) was administered through one microdialysis membrane while the other membrane was perfused with the vehicle (Ringer solution). Using the opposite arm, subjects performed three maximum voluntary isometric handgrip contractions. The maximal workload of these three contractions was used to calculate the effort during the ensuing data collection period. No data were collected for at least 30 min after the last maximal voluntary contraction.

The subject then performed 2 min IHG exercise at 40% maximum voluntary contraction using the same arm. During the final 5 s of the 2 min exercise bout, a cuff around the upper portion of the exercising arm was inflated to 250 mmHg and remained inflated for 2 min. Within 10 s following the end of exercise, during the period of post-exercise ischaemia, a bolus injection of sodium nitroprusside (150 μg) was administered through the intravenous catheter with the aim of returning mean arterial blood pressure to the pre-exercise level. IHG exercise and post-exercise ischaemia were also performed without administration of sodium nitroprusside. The order of these tests (i.e. with and without sodium nitroprusside administration during post-exercise ischaemia) was randomized. The subjects were then heated by perfusing warm water through the suit that each subject wore. The aforementioned tests were then repeated after sublingual temperature had increased by $\sim 0.5^\circ\text{C}$ and sweating at the control site was apparent. The order of the tests during heat stress was also randomized.

Data collection and analysis

All data were recorded at 200 Hz via a 16 bit A/D converter (Biopac, MP100) and stored as 20 s averages. Absolute humidity from 0 to 50 g m⁻³ represented an analog voltage of 0–5000 mV. Each bit of the 16 bit A/D conversion represents a sweat rate of 0.0009 mg cm⁻² min⁻¹. Thus, the sweat detection system was not limited by the resolution of the data collection system. Moreover, others have used similar systems to measure transient and pulsatile increase in sweat rate of < 0.05 mg cm² min⁻¹ in association with skin sympathetic nerve bursts (Sugenoya *et al.* 1998). Data presented in the figures represent sweating as a change from baseline, while data listed in Table 1 represent a change in sweating from the period immediately prior to exercise regardless of the thermal status. Data represent an average of the final 20 s from each of the following stages: a pre-exercise period, minutes 1 and 2 of IHG exercise, minutes 1 and 2 of post-exercise ischaemia, and after 2 min following release of ischaemia. These data were statistically compared using one-way repeated measures ANOVA followed by Dunnett's test when significant main factors were identified. The effects of neostigmine and sodium nitroprusside on sweat rate were statistically analysed via two-way repeated measures ANOVA. All data are expressed as means ± S.E.M. The level of statistical significance was set at $P \leq 0.05$.

RESULTS

Temperature and haemodynamic responses

Whole-body heating increased sublingual temperature from 36.8 ± 0.1 to 37.3 ± 0.1 °C ($P < 0.001$), which resulted in skin blood flow increasing from 22 ± 4 to 88 ± 11 laser-Doppler flowmetry units ($P < 0.001$). Sublingual and skin temperatures during each bout of IHG exercise and post-exercise ischaemia did not change substantially regardless of the thermal condition of the subject (Figs 1 and 2). Although in some cases significant differences in sublingual and skin temperatures were observed during IHG exercise ($P \leq 0.05$) the physiological significance of the differences is probably unimportant, relative to the large increase in sweat rate during IHG exercise. In normothermia and during whole-body heating, IHG exercise increased mean arterial blood pressure and heart rate (Table 1). As expected, during post-exercise ischaemia (without sodium nitroprusside administration) mean arterial blood pressure remained elevated relative to pre-

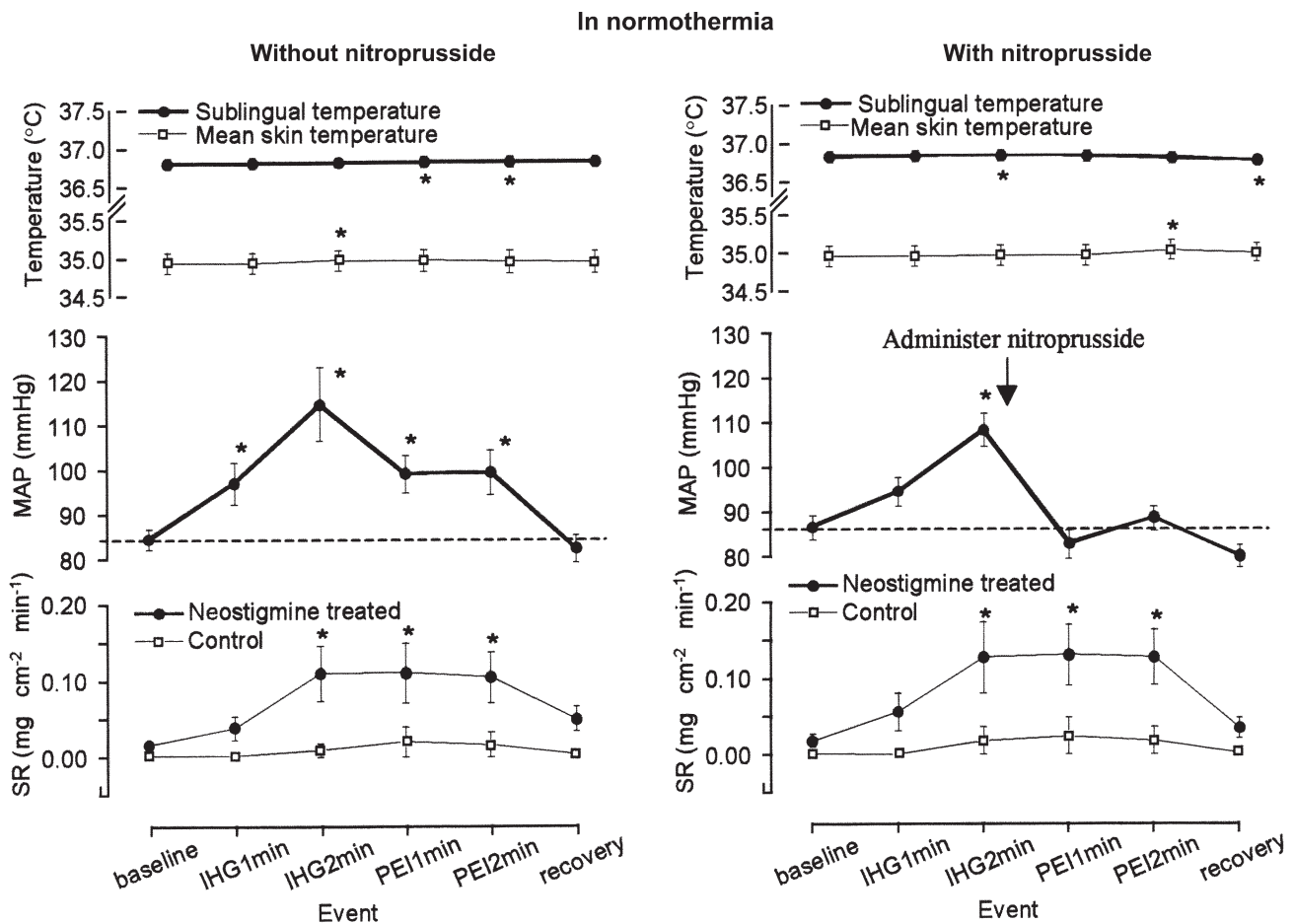


Figure 1. Influence of isometric exercise on sweating rate in normothermia

In normothermia, isometric exercise increased sweating rate (SR) at the neostigmine-treated site but not at the control site. Sweat rate remained elevated during post-exercise ischaemia (PEI) at the neostigmine-treated site regardless of whether mean arterial blood pressure (MAP) remained elevated during PEI (left panel) or was reduced via bolus infusion of sodium nitroprusside (right panel; see arrow). * Significantly different from baseline ($P < 0.05$).

Table 1. Effects of isometric handgrip exercise and post-exercise ischaemia in normothermic and heat stress conditions

| | | IHG exercise | | | Post-exercise ischaemia | | Recovery |
|--------------|---------------------|--------------|-------------|--------------|-------------------------|--------------|--------------|
| | | Baseline | 1 min | 2 min | 1 min | 2 min | |
| Normothermia | | | | | | | |
| Without SNP | MAP | 84 ± 2 | 97 ± 5* | 115 ± 8* | 99 ± 4* | 100 ± 5* | 82 ± 3 |
| | HR | 61 ± 3 | 71 ± 4* | 77 ± 4* | 58 ± 3 | 59 ± 3 | 61 ± 4 |
| | ΔSR _{neo} | — | 0.02 ± 0.1 | 0.09 ± 0.03* | 0.09 ± 0.04* | 0.09 ± 0.03* | 0.03 ± 0.01 |
| | ΔSR _{cont} | — | 0.00 ± 0.00 | 0.01 ± 0.01† | 0.02 ± 0.02† | 0.01 ± 0.01† | 0.00 ± 0.00† |
| With SNP | MAP | 87 ± 3 | 95 ± 3* | 108 ± 4* | 83 ± 3‡ | 89 ± 3 | 80 ± 2 |
| | HR | 64 ± 4 | 71 ± 4 | 74 ± 4* | 78 ± 4*‡ | 66 ± 4*‡ | 59 ± 4 |
| | ΔSR _{neo} | — | 0.04 ± 0.02 | 0.11 ± 0.04* | 0.11 ± 0.03* | 0.11 ± 0.03* | 0.02 ± 0.01 |
| | ΔSR _{cont} | — | 0.00 ± 0.00 | 0.02 ± 0.02† | 0.02 ± 0.02† | 0.02 ± 0.02† | 0.00 ± 0.00† |
| Heat stress | | | | | | | |
| Without SNP | MAP | 86 ± 3 | 102 ± 6* | 120 ± 7* | 96 ± 4* | 96 ± 4* | 84 ± 3 |
| | HR | 78 ± 4 | 88 ± 4* | 98 ± 4* | 77 ± 5 | 80 ± 5 | 79 ± 4 |
| | ΔSR _{neo} | — | 0.05 ± 0.02 | 0.15 ± 0.02* | 0.12 ± 0.02* | 0.16 ± 0.03* | 0.13 ± 0.04* |
| | ΔSR _{cont} | — | 0.04 ± 0.02 | 0.15 ± 0.03* | 0.14 ± 0.04* | 0.18 ± 0.04* | 0.15 ± 0.05* |
| With SNP | MAP | 84 ± 3 | 98 ± 4* | 112 ± 5* | 84 ± 3‡ | 92 ± 3* | 83 ± 2 |
| | HR | 78 ± 4 | 90 ± 2* | 91 ± 5* | 96 ± 5*‡ | 86 ± 4*‡ | 75 ± 5‡ |
| | ΔSR _{neo} | — | 0.05 ± 0.02 | 0.14 ± 0.03* | 0.17 ± 0.03* | 0.18 ± 0.04* | 0.10 ± 0.04* |
| | ΔSR _{cont} | — | 0.04 ± 0.02 | 0.13 ± 0.03* | 0.16 ± 0.04* | 0.18 ± 0.04* | 0.09 ± 0.04 |

SNP, sodium nitroprusside; MAP, mean arterial blood pressure (mmHg); HR, heart rate (beats min⁻¹); ΔSR_{neo}, change in sweat rate at the neostigmine-treated site relative to the period prior to isometric exercise (mg cm⁻² min⁻¹); ΔSR_{cont}, change in sweat rate at the control site relative to the period prior to isometric exercise (mg cm⁻² min⁻¹). *Significant difference compared with baseline; †significant difference compared with neostigmine-treated site; ‡significant difference relative to the condition without sodium nitroprusside.

IHG exercise, while heart rate returned to baseline. Bolus intravenous administration of sodium nitroprusside during post-exercise ischaemia returned mean arterial blood pressure to pre-IHG exercise levels within the first minute following administration of the drug under both thermal conditions, and caused a baroreflex-mediated elevation in heart rate that was significantly greater than baseline.

Sweat rate responses in normothermia

IHG exercise during normothermic conditions significantly increased sweat rate by ~0.1 mg cm⁻² min⁻¹ at the neostigmine-treated site (Fig. 1 and Table 1). In contrast, no change in sweat rate was observed at the control site during IHG exercise or post-exercise ischaemia. At the neostigmine-treated site, the elevation in sweat rate observed during IHG exercise persisted throughout the post-exercise ischaemia period. Moreover, sweat rate remained elevated when arterial blood pressure was reduced during post-exercise ischaemia via bolus infusion of sodium nitroprusside. Finally, sweat rate at the neostigmine-treated site returned to values not different from baseline upon release of the occlusion cuff following post-exercise ischaemia.

Sweat rate responses during whole-body heating

Whole-body heating significantly increased sweat rate at both control (0.44 ± 0.08 mg cm⁻² min⁻¹ from baseline) and neostigmine-treated (0.73 ± 0.10 mg cm⁻² min⁻¹ from baseline) sites before IHG exercise in the heat. Sweat rate at the neostigmine-treated site was significantly higher compared with the control site, which is consistent with our previous work (Shibasaki & Crandall, 2001). Prior to the final bout of IHG exercise during heat stress, sweat rate was slightly elevated at both sites (control, 0.50 ± 0.11 mg cm⁻² min⁻¹; neostigmine, 0.78 ± 0.11 mg cm⁻² min⁻¹) relative to the aforementioned sweat rate before IHG exercise in the heat ($P > 0.05$).

IHG exercise during heat stress increased sweat rate significantly at both control and neostigmine-treated sites by ~0.15 mg cm⁻² min⁻¹ relative to the period immediately before the bout of exercise (Fig. 2 and Table 1). The magnitude of the increase in sweat rate during IHG exercise was not different between control and neostigmine-treated sites ($P > 0.05$). During post-exercise ischaemia, sweat rate remained elevated at both sites regardless of whether mean arterial blood pressure was reduced to pre-IHG exercise levels via bolus sodium

nitroprusside administration. Sweat rate decreased upon release of the occlusion cuff after post-exercise ischaemia. However, 2 min after the release of the occlusion cuff sweat rate remained elevated relative to the baseline period before IHG exercise (see Fig. 2 and Table 1).

DISCUSSION

The primary finding of this study is that increased sweating during post-exercise ischaemia occurs via metaboreflex stimulation, and is not due to baroreceptor loading associated with increased blood pressure during the ischaemic period. A secondary finding is that IHG exercise can cause sweating in normothermic individuals if hydrolysis of acetylcholine is inhibited via local administration of neostigmine. These findings are supported by the observations that, regardless of the thermal condition, sweat rate remains elevated during post-exercise ischaemia in combination with the lowering

of arterial blood pressure (Figs 1 and 2), and that sweat rate increases significantly at the neostigmine-treated site in normothermia during IHG exercise and post-exercise ischaemia (Fig. 1).

In the present study, each subject performed IHG exercise at 40% maximal voluntary contraction for 2 min, and mean arterial blood pressure during subsequent post-exercise ischaemia was elevated 15 ± 3 and 12 ± 2 mmHg relative to pre-exercise pressures in normothermia and during whole-body heating, respectively. Given previous findings that demonstrate a relationship between muscle metaboreceptor stimulation and the elevation in blood pressure and muscle sympathetic nerve activity during post-exercise ischaemia (Victor *et al.* 1988; Nishiyasu *et al.* 1994), it is clear that muscle metaboreceptors during post-exercise ischaemia were activated in the present procedure. Since baroreceptors have been shown to modulate sweat rate (Mack *et al.* 1995), it was possible

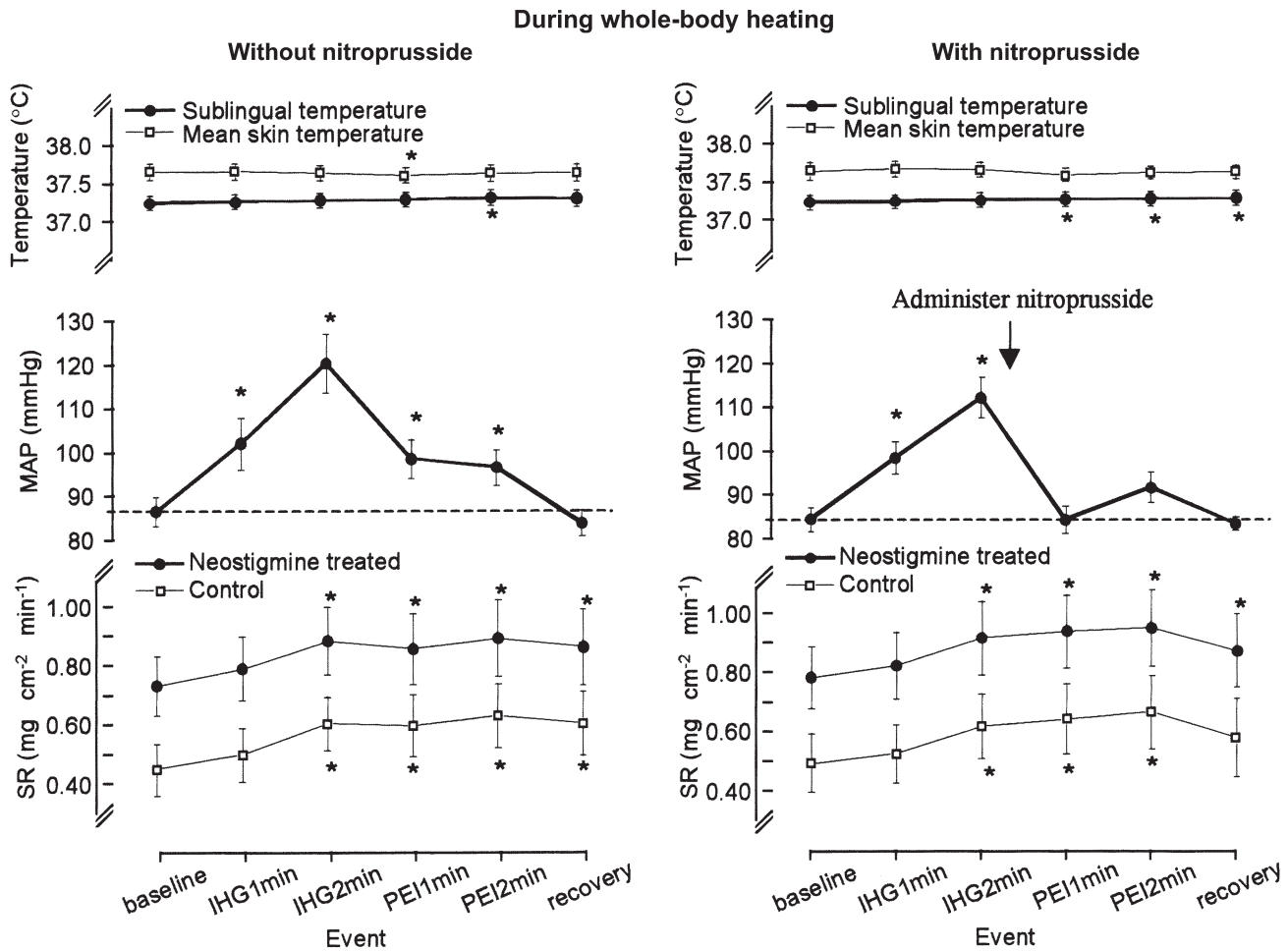


Figure 2. Influence of isometric exercise on sweating rate during whole-body heating

During whole-body heating, isometric exercise increased sweating rate (SR) at both neostigmine-treated and untreated sites. Sweat rate remained elevated during post-exercise ischaemia (PEI) at both sites regardless of whether mean arterial blood pressure (MAP) was returned to baseline during PEI. *Significantly different from baseline ($P < 0.05$).

that elevated mean arterial blood pressure in association with IHG exercise and post-exercise ischaemia contributed to the elevation in sweat rate. Data from the present protocol do not support this hypothesis since returning mean arterial blood pressure to pre-exercise levels did not decrease sweat rate at the neostigmine-treated site in normothermia or at either site during heat stress (Figs 1 and 2). These results clearly suggest that increases in sweating during post-exercise ischaemia occur via muscle metaboreflex stimulation and not via baroreceptor loading during the perturbation.

The present data suggest that baroreceptor loading associated with IHG exercise does not modulate sweat rate regardless of the thermal conditions. This observation is in contrast to the findings of Mack *et al.* (1995) who reported decreases in sweat rate with baroreceptor unloading during lower body negative pressure in subjects performing dynamic exercise in a warm (28°C) room. There are several key differences between these studies that may explain this apparent discrepancy. First, in heated individuals changes in skin temperature can greatly affect skin sympathetic nerve activity and sweat rate (Vissing *et al.* 1991; Okamoto *et al.* 1994). Given that lower body negative pressure is capable of reducing skin temperature (Vissing *et al.* 1991; Mack *et al.* 1995), it is possible that the reductions in sweat rate observed by Mack *et al.* (1995) were due to skin cooling and not baroreceptor unloading. In the present study skin temperature was not altered by pharmacologically induced decreases in mean arterial blood pressure. Second, in the present study mean arterial blood pressure was pharmacologically reduced during post-exercise ischaemia, a condition in which central command has no influence on cardiovascular variables and sweat rate. Thus, it is possible that the inclusion of central command and/or responses associated with stimulation of muscle metaboreceptors during static *versus* dynamic exercise may lead to different findings (Sundblad & Linnarsson, 1996). Third, it is possible that the primary stimulus leading to modulation of sweat rate originates from cardiopulmonary, not arterial baroreceptors. Dibner-Dunlap *et al.* (1996) showed that bolus administration of nitroprusside, in similar doses to that used in the present study, did not change central venous pressure, and therefore presumably did not unload cardiopulmonary baroreceptors. Thus, it is unlikely that bolus sodium nitroprusside administration in the present study reduced cardiopulmonary baroreceptor loading. This is in contrast to the substantial reductions in cardiopulmonary baroreceptor loading that are likely to occur during lower body negative pressure as used by Mack *et al.* (1995).

Sweating responses at the neostigmine-treated site in normothermia were similar to those observed at treated and untreated sites after whole-body heating. This similarity occurred even though sublingual and skin temperatures did not change appreciably during IHG exercise and post-exercise ischaemia regardless of the

thermal conditions. In contrast, IHG exercise and post-exercise ischaemia did not change sweat rate at the control site in normothermia, which is consistent with our previous studies (Crandall *et al.* 1995, 1998). In normothermia, upon release of the occlusion cuff sweat rate at the neostigmine-treated site rapidly returned to values not different from baseline (i.e. pre-exercise). This observation clearly indicates that sudomotor activity in non-glabrous skin increases due to non-thermal factors such as IHG exercise and post-exercise ischaemia in normothermic individuals. However, due to rapid hydrolysis of acetylcholine this response can only be observed if acetylcholinesterase is inhibited.

Saito *et al.* (1990) and Vissing *et al.* (1991) reported that in normothermic individuals skin sympathetic nerve activity, measured from the tibial and peroneal nerves, respectively, increased during IHG exercise but returned to baseline during post-exercise ischaemia. Findings from the present study clearly show that in normothermia sweat rate remains elevated during post-exercise ischaemia at the neostigmine-treated site. There are three possible explanations for the increases in sweat rate observed in the present study during post-exercise ischaemia, without the increases in skin sympathetic nerve activity during post-exercise ischaemia reported by others: (a) the origin of the signal leading to increases in sweat rate is not governed by skin sympathetic nerve activity, (b) subtle changes in skin sympathetic nerve activity leading to increases in sweat rate are not detectable with current microneurography technology, or (c) skin sympathetic nerve activity to the lower limbs responds differently during post-exercise ischaemia relative to skin sympathetic nerve activity innervating the dorsal forearm. Although forearm skin sympathetic nerve activity was not measured in the present study, Sugeno *et al.* (1998) suggested that 80% of spontaneous skin sympathetic bursts in mildly heated individuals led to sweat expulsion. In the present study, spontaneous sweat expulsion was observed at the neostigmine-treated site prior to IHG exercise in many subjects, as well as during IHG exercise and post-exercise ischaemia in all subjects. If sweat expulsion is indicative of bursts of sympathetic skin nerve activity, the observed sweat expulsion during post-exercise ischaemia suggests that skin sympathetic nerve activity to the forearm remains elevated during post-exercise ischaemia.

Conclusions

The results from the present study indicate that increases in sweat rate during post-exercise ischaemia occur via muscle metaboreceptor stimulation, not via baroreceptor loading associated with increases in blood pressure during post-exercise ischaemia. Moreover, sudomotor activity governing sweat rate in non-glabrous skin can be activated by non-thermal factors in normothermia if acetylcholinesterase is inhibited, as well as at both control and neostigmine-treated sites during heat stress.

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