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ONSET AND STEADY STATE DISTRIBUTION OF ECCRINE SWEATING

James D. Cotter, Mark J. Patterson and Nigel A.S. Taylor,
Department of Biomedical Science,
University of Wollongong,
Wollongong, NSW 2522, AUSTRALIA

INTRODUCTION

Eccrine sweating is critical to thermoregulation during exercise, and heat stress. Evaporative cooling is maximised when sweating is initiated early, when it increases rapidly in relation to heat storage (high gain) and when it attains high steady state levels and optimal spatial distribution.

The spatial distribution of sweat Onset or steady state excretion ($\dot{m}_{sw}$) in humans has not been thoroughly defined. A caudal to rostral pattern of sweat onset has been demonstrated in resting males (1,2,3), though not in exercising males (4; n=3). The onset threshold (1) and gain characteristics (4) of *each* skin region influence the steady state distribution of $\dot{m}_{sw}$, which appears to be greatest *on* the forehead and least *on* the arms (1) and extremities (5). A large inter-individual variability in distribution is apparent (1,4,5), although this variability may be reduced at higher $\dot{m}_{sw}$ (3). The purpose of this study was to further investigate the patterns of onset and *steady* state distribution of $\dot{m}_{sw}$ in humans exposed to combined exercise and heat stress.

METHODS

Six healthy, trained males (29 ±7.3 yrs ($\bar{X}$ ±S.D.); A_D= 1.99 ±0.10 m^2) participated in this study. An exercise ramp test (Quinton, Excalibur) to volitional exhaustion was first completed to determine the steady-state work rate (40% peak power; 166 ±19 W). Subjects completed four 42 min heat stress trials: 2 min rest and 40 min cycling in 36.6 ±0.6°C (dry bulb; relative humidity = 46.0 ±2.3%). Trials were undertaken at the same time of day and were separated by a minimum period of three days. Four trials, presented in balanced order, were necessary to obtain sweat onset and steady state $\dot{m}_{sw}$ for *each* of the following regions: forehead (all *trials*), medial lower chest, lateral abdomen, lateral scapula, medial lower back, anterior mid-thigh, anterior *calf*, dorsal foot, dorsal hand, anterior upper-arm, and at three forearm sites: distal ventral, mid-ventral and mid-dorsal. Sweat excretion was monitored from four ventilated capsules (2.19 cm^2, LiCl saturated air, 400 $cm^3.min^{-1}$) using capacitance hygrometry (Multi-site sweat monitor, Clinical Engineering, Sydney). Saturated solutions for calibration were LiCl, NaI, KCl, NaCl and KH_2PO_4. The test-retest correlation and intrasubject coefficient of variation of the procedure were 0.85 ±0.10 and 15.0 ±11.8%, respectively (forehead steady state $\dot{m}_{sw}$).

Skin blood flow was monitored on the ventral *aspect* of the forearm, over the midpoint of the radius (TSI Laserflo BPM², Vasamedics). The velocimeter uses a gallium aluminium arsenide semiconductor to generate light (λ=780 nm), emitted via a 50 μm probe core. The two 100 μm core receiving fibres are spaced 0.5 mm from the transmitting fibre.

Auditory canal temperature (T_{ac}) was monitored using Zero gradient thermometry (Keatinge, London Hospital) during all trials. Oesophageal temperature (T_{es}) was monitored during each subject's second trial (YSI, type 401). Skin temperature was–measured at each sweat capsule site (YSI mini-thermistor, Type EU), and mean skin temperature derived (T_{sk}; after 6). Temperatures, with the exception of T_{ac}, were recorded on a squirrel data logger (Grant, 1200 series) at 0.2 Hz. Remaining data were converted to digital equivalents (Computerboards Inc, PPIO-A108) and recorded at 1 Hz *on* a portable IBM compatible PC.

Sweat onset times and $\dot{m}_{sw}$ were standardised between trials, *such* that responses were normalised to the largest forehead $\dot{m}_{sw}$ response. The initial $\dot{m}_{sw}$ following onset ('transient $\dot{m}_{sw}$') was calculated as the mean $\dot{m}_{sw}$ during the five min after onset. Steady state $\dot{m}_{sw}$ was calculated as the mean for the period 18.5-23.5 min after onset. (Data from the *calf* site were considered inaccurate beyond 23.5 min (n=2), due to alteration of the pressure exerted *on* these capsules whilst improving the seal). Dependent variables were analysed using ANOVA and Tukeys HSD post hoc procedure. Pearson's correlation coefficient was used to examine the relation between variables.

RESULTS

The total whole–body, area-corrected $\dot{m}_{sw}$, estimated from the mean $\dot{m}_{sw}$ of each region, was 1.31 ±0.10 mg·cm^{-2}·min^{-1} (X ±S.E.M.). This did not significantly differ from, nor correlate with, the mass loss (1.36 ±0.08 mg·cm^{-2}·min^{-1}, *Paired* t-test: $t(5)$ –0.58, p=0.59; r=0.36, p>0.05).

The T_{ac} and T_{es} at sweat onset were equivalent across regions: **37.01** ±0.03°C (p=0.30) and **36.45** ±0.01°C (p=0.86), respectively. The $\bar{T}_{sk}$ at onset was obtained for **15** of the **24** trials, ranging **34.82** ±0.22°C (forearm, n=4) to **35.15** ±0.10°C (abdomen and scapula, n=4), with a mean of **35.02** ±0.02°C between regions. T_{skl} at onset was lower at the foot **(33.11 i0.42)** and calf **(34.56 i0.19)** than at the scapula **(35.97 i0.19)** and forehead **(36.00** ±0.31°C; p=0.00). Adjacent regions were grouped prior to analysis of sweat onset recruitment, for the purpose of reducing the number of treatment levels. The lower torso sweat onset (45.5 ±42.0 s) preceded that of the head **(126.5** ±34.8 s, p=0.02) but was not significantly different from those of the legs **(66.6** ±25.7 s), upper torso **(80.2** ±36.8 s) or arms **(108.6 i31.2** s). The sweat onset (ventral forearm) preceded vasodilation onset by **119.0** ±55.2 s (p<0.05), however, the linear relation between onset times was not significant (r=0.68, p>0.05).

All subjects exhibited an initial rapid gain in $\dot{m}_{sw}$ for a period of approximately seven min, although the subsequent phase of reduced gain was somewhat variable. The foot was excluded from analyses of $\dot{m}_{sw}$ due to an inadvertently greater pressure exerted upon this capsule in two subjects. The (pooled sites) mean transient $\dot{m}_{sw}$ ranged from **0.39** ±0.09 mg·cm^{-2}·min^{-1} on the lower limb to **0.91** ±0.25 mg·cm^{-2}·min^{-1} on the upper limb, though did not differ statistically between regions (p=0.12). The mean transient $\dot{m}_{sw}$ (n=8 sites) was not correlated with the estimated mean gland density for European males (r=0.50, p>0.05, cited in (7)).

The mean steady state $\dot{m}_{sw}$ on the forehead (3.20 ±0.51 mg·cm^{-2}·min^{-1}) was not significantly greater than on the scapula (2.9 ±0.4), forearm (2.2 ±0.5), hand (2.1 ±0.4), abdomen (2.0 ±0.4) or lower back (1.9 i0.3; descending order), but was greater (p=0.00) than the chest (1.6 ±0.2), upperarm (1.6 ±0.2), calf (1.5 ±0.3) and thigh (1.0 i0.2). The mean regional steady state $\dot{m}_{sw}$ (n=8 sites) was significantly correlated with the estimated mean gland density (r=0.75, p<0.05, (7)). The ventral and distal-ventral forearm steady state $\dot{m}_{sw}$ were correlated mast closely with the mean body, area-corrected, steady state $\dot{m}_{sw}$ (1.83 i0.ll mg·cm^{-2}·min^{-1}): r=0.89 and 0.85 (p<0.05), respectively. This steady state 'maximum' $\dot{m}_{sw}$ was inversely related to surface area (r=0.84, p<0.05).

CONCLUSIONS

Whilst only the lower torso sweat onset significantly preceded that of the forehead, the trend within sweat onset times tends to support previous indications that recruitment occurs caudally to rostrally (1,2,3), and that this pattern is not due to T_{skl} dismbution (1) or controller output (i.e. core and mean skin temperature). Spinal facilitation (2,3) or regional differences at the glandular level may be responsible. However, above threshold, the rapid elevation in $\dot{m}_{sw}$ occurred at similar times and rates of gain between regions, indicating equivalent transient response to efferent activity, although the power of this analysis was modest. A distinct inter-regional variability in gland density (7), and a poor relation with this estimated density (r^2=0.25), indicates that this rapid, widespread elevation in $\dot{m}_{sw}$ may be due to an increase in output per gland prior to the recruitment of additional glands.

Progression toward maximal steady state $\dot{m}_{sw}$ revealed large inter-individual variability between regions, in which central regions did not predominate. The estimated gland density (7) appeared to account for some of the observed regional steady state distribution (r^2=0.57), however, acclimation status (8) and the high level of heat stress may also have contributed to the variability within the present sample, and between this and earlier studies of the distribution of steady state $\dot{m}_{sw}$ (1,4,5).

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