An investigation of forearm vasomotor and sudomotor thresholds during passive heating, following whole-body cooling

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AN INVESTIGATION OF FOREARM VASOMOTOR AND SUDOMOTOR THRESHOLDS DURING PASSIVE HEATING, FOLLOWING WHOLE-BODY COOLING.

Joanne N. Caldwell, Åsa Nykvist, Nicholas Powers, Sean R. Notley, Daniel S. Lee, Gregory E. Peoples and Nigel A.S. Taylor

INTRODUCTION

In a previous communication to this society (MacDonald et al., 2000), it was reported that whole-body cooling, prior to an exercising heat stress, resulted in independent and opposite displacements of the sweating and cutaneous vasodilatory thresholds. If this observation was correct, it could have significant implications for our understanding of human temperature regulation. Thus, it was considered necessary to verify these observations, and so the purpose of this study was to re-evaluate the influence of whole-body cooling on the sudomotor and vasomotor thresholds, but now without a postural change, and under resting conditions.

METHODS

Eight healthy males (age 23.6 y (SD 4.0), height 178.8 m (SD 4.1), mass 74.9 kg (SD 7.9)) participated in two trials, administered in a balanced order. In each trial, subjects wearing only a water-perfusion garment and swimming costume were immersed for 45 min in the supine position (head out) in either thermoneutral water (control: 33°C) or in gradually cooled water (28-23°C). The aim of the latter trial was to extract heat without inducing shivering. Following each immersion, and without a postural change, subjects were rapidly transferred to a pre-heated climate chamber (28°C, 50% relative humidity), with pre-cooled or pre-heated clothing and blankets used to clamp body temperature. When inside the chamber, this insulation was removed and the perfusion garment was immediately perfused with water at 48°C, and passive, supine heating continued until forearm sweating and cutaneous vasodilatation were both clearly established.

Core temperature was measured from the oesophagus (Edale Instruments Ltd, U.K.) whilst skin temperatures were measured from eight sites (Edale Instruments Ltd, U.K.), with data recorded at 15-s intervals (Grant Instruments Ltd., 1206 Series Squirrel, U.K.). Mean body temperature was derived as the weighted sum of the core and mean skin temperatures (0.9 * core + 0.1 * mean skin). Sweating was evaluated from the dorsal forearm using a ventilated sweat capsule (discharged sweat: 3.16 cm²), and from changes in skin conductance recorded next to the sweat capsule (precursor sweat: SCL/SCR Data Collection System, UFI, Morro Bay, CA, U.S.A.). These data were sampled at 1-s intervals. Cutaneous blood flow was also measured from the same forearm using laser-Doppler flowmetry (TSI Laserflo BPM2 with a P-435 laser fibre optic probe, Vasamedics Inc., St. Paul, MN, U.S.A.; 37 internal refresh rate ~7 Hz), with data sampled at 20 Hz.

The thresholds for precursor and discharged sweating, and the vasomotor threshold were determined by isolating baseline (pre-threshold) and post-
threshold data (~1 min). The latter were defined as thermoeffector data that had increased above baseline, and continued to rise. First-order regression equations were derived for these data sets, and the intersection of these lines, determined using simultaneous equations, was taken as the threshold for each effector, which was then referenced to the mean body temperature.

RESULTS

The respective mean body temperatures, immediately following the control and cooling treatments, were 36.3°C (±0.12) and 35.7°C (±0.10; \( P<0.05 \)). Following the control immersion, the mean body temperature thresholds for both thermoeffectors were numerically, but not significantly different (Figure 1; \( P>0.05 \)). However, following whole-body cooling, the threshold for precursor sweating was elevated by 0.18°C (±0.06; \( P=0.07 \)), whilst that for discharged sweating was raised by 0.21°C (±0.03; \( P<0.05 \)). Conversely, the vasomotor threshold was reduced by 0.35°C (±0.11; \( P<0.05 \)). For this thermoeffector, the change in the pre-heating mean body temperature (0.41 ±0.10°C), relative to the control trial, did not differ significantly from its threshold change (\( P>0.05 \)).

![Graph showing thermoeffector thresholds](image)

**Figure 1:** Sudomotor and vasomotor thresholds (mean body temperature) during passive (supine) heating, preceded by 45 min of immersion in either thermoneutral (control: 33°C) or gradually cooled water (28-23°C). * = significant between-treatment difference (\( P<0.05 \)); † = significant difference between the within-treatment thresholds (\( P<0.05 \)).
CONCLUSION

To our knowledge, the opposite displacements of these thermoeffector thresholds have not previously been described. However, we have shown whole-body cooling to delay vasodilatation during subsequent exercise in the heat, with the mean body temperature threshold elevated by 0.59°C (P<0.05), while the sudomotor threshold was lowered by 0.37°C (P>0.05; MacDonald et al., 2000). Thus, pre-exercise cooling induced a displacement of these thresholds in opposite directions, relative to the control condition, resulting in a threshold difference of 0.85°C (P<0.05). In that experiment, subjects changed from a supine immersion to seated exercise (cycling). Notwithstanding this postural change and its affect on skin blood flow, non-thermal drives will enhance sweating and suppress skin blood flow during the early phase of an exercise transition (Kondo et al., 2010), and these affects are consistent with the observed threshold changes, and occur over similar durations.

In the current study, the opposite pattern was evident during passive, supine heating, following whole-body cooling, and this resulted in a threshold difference of 0.64°C (P<0.05) between an earlier vasodilatation and delayed discharged sweating. In the control state, one would anticipate vasodilatation to occur before the initiation of sweating, and this was observed in both trials, but it appeared significantly earlier following cooling, and the threshold displacement was equivalent to the change in mean body temperature induced by the pre-exposure treatment. This is not the first time that we have reported an equivalence between a thermoeffector threshold change and a body temperature displacement. This phenomenon was described for the reduction in the sudomotor threshold during heated exercise after pre-cooling (MacDonald et al., 2000), then following heat acclimation (Patterson et al., 2004), and again after pre-cooling (Booth et al., 2004). It is clear that these thermoeffectors show some independence of the absolute body temperature, but appear to be coupled with the change in body temperature. A second significant observation from this study is the apparent capacity of these thermoeffector thresholds to vary independently, and this characteristic is currently being explored.

REFERENCES


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