SWEATING AND SKIN BLOOD FLOW CHANGES DURING PROGRESSIVE DEHYDRATION.

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INTRODUCTION
Cutaneous vasodilatation is essential for the convective delivery of heat from the body core to the periphery, whilst the evaporation of sweat dissipates this heat from the skin surface. Both of these physiological mechanisms must continue to function optimally for effective body temperature regulation to be sustained when exercising in the heat.

Unfortunately, in situations of protracted and high sweat secretion, the body water content will become progressively reduced, and the blood volume slowly declines. This is well tolerated within healthy individuals. However, the regulation of blood pressure has a higher homeostatic priority than does temperature regulation, since low pressures cannot support adequate tissue perfusion, and will eventually result in cell damage, regardless of tissue temperature. Therefore, a chronotropically and inotropically mediated increase in cardiac output ensues, simultaneously with an elevation in total peripheral resistance. Vasoconstriction is initially directed towards the visceral structures, but as the body water deficit becomes greater, constriction can also be expected within the cutaneous vascular beds to ensure that blood pressure is not compromised. When this occurs, sweating may also be impaired, and an undesirable elevation in body heat storage can result.

Previous research has established the existence of these physiological mechanisms. Indeed, reduced sweating and skin blood flow have been observed across a range of dehydration levels, accompanied by significant increments in body core temperature and heart rate (Sawka et al., 1985; Montain and Coyle, 1992). However, these observations are based upon inter-investigations comparisons, or on data from experiments in which physiological responses from dehydrated individuals were examined across hydration states evaluated on different days, rather than during a single experimental exposure. In the current study, subjects were progressively dehydrated to a 7% water deficit, in 1% increments, within one experiment, and skin blood flow and sweat rates were evaluated across these dehydration levels.

METHODS
Dehydration was gradually induced in ten healthy and physically active young males, using intermittent (upright) cycling in hot-humid conditions maintained at 35.6°C (±0.4) and 56.0% humidity (±1.0), in which the black globe temperature averaged 35.6°C (±0.3) and wind velocity was less than 0.05 m.s⁻¹. Subjects wore only shorts and running shoes. Tests were conducted at approximately the same time of day for each person, using fully-hydrated subjects: pre-experimental mean urine specific gravity across these trials was 1.006 (SD 0.006). The average total trial duration (excluding data collection phases (~10 min) at each target) was 5.27 h.
Water deficits were determined from body mass changes, and the dehydration targets were secured with precision, and within 0.06% of the targets. Two local sweat rates, forearm skin blood flow, arterial blood pressure, body core temperature (auditory canal) and heart rate were measured at baseline, and then at each 1% dehydration target, during either steady-state cycling (5 min at 40% peak work rate) or seated rest immediately thereafter. Furthermore, whole-body sweat rates and forearm vascular conductance were computed for each dehydration target.

Sweat rates from the forehead and over the scapula were evaluated for 2 min during steady-state cycling. These data were collected at 1-s intervals using ventilated sweat capsules connected to a sweat monitor system (Clinical Engineering Solutions, NSW, Australia). Forearm blood flow data were measured during seated rest (2 min) using venous-occlusion plethysmography (EC 4 Plethysmograph, D.E. Hokanson, Inc, U.S.A.), with a mercury-in-silastic strain gauge placed around the largest circumference of the forearm. It was assumed that changes in blood flow under these conditions would reflect changes in the cutaneous component only (Johnson and Rowell, 1975). Data were sampled at 20 Hz using an eight channel, 12-bit analog-to-digital converter (Computer Boards Inc., PPIO-A18, Mansfield, U.S.A.). Body core temperature, heart rate and arterial blood pressure were also measured during rest. One-way Analysis of Variance was used to evaluate differences in all physiological variables throughout the trial (baseline and dehydration states). Tukeys HSD post hoc tests were used to isolate sources of significant differences, with *alpha* set at the 0.05 level.

**RESULTS**

Data from baseline, 1%, 3%, 6% and 7% dehydration are reported here (Table 1). During the dehydration protocol, all physiological variables differed significantly from the baseline state. Furthermore, body core temperature and heart rate increased, and sweat rates (whole-body and forehead) decreased as the individuals became more dehydrated (*P* < 0.05). However, the variations in forearm blood flow and vascular conductance, and the changes in sweating from the upper back (scapula) region did not attain statistical significance (*P* >0.05).

**DISCUSSION**

In this study, it seems that, during an extended-duration dehydration protocol (> 5 h), physiological adjustments initially occurred to match the increased thermoregulatory demand dictated by using the controlled-hyperthermia technique. However, as the pressure, volume and osmolarity regulation of the blood gradually became more critical, accompanying increments in the water deficit, mechanisms to facilitate pressure and body-fluid homeostasis would be activated. Indeed, the decreased sweat responses at the highest dehydration levels, despite a significantly increased thermal strain, are consistent with such a homeostatic priority, and these observations are in agreement with previous research (Sawka *et al*., 1985).

On the other hand, scapula sweat rate, forearm blood flow and vascular conductance did not vary significantly throughout the trial, even when subjects had lost 6-7% of their body mass. Some have previously reported that whole-body sweat rate remains constant, even during dehydration up to 4% (Montain and Coyle, 1992), so it is highly likely that some regions will experience a reduction in sweat secretion, whilst in others, this will be maintained. However, the cardiovascular observations appear to indicate that blood pressure regulation was not excessively
challenged, and could be successfully defended by elevating cardiac output and central vasoconstriction, despite the progressive fluid loss over >5 h. Indeed, isosmotic hypovolaemia has been observed for 3% dehydrated subjects, with a pronounced increment in plasma osmolality occurring only when these individuals were 7% dehydrated (Sawka et al., 1985), so it is possible that electrolyte homeostasis was not adversely affected.

Table 1: Physiological variables at baseline, 1%, 3%, 6% and 7% dehydration. Data are means with standard error of the means. * significantly different from baseline (P<0.05); † significantly different from 1% dehydration (P<0.05); ‡ significantly different from 3% dehydration (P<0.05); § 6% versus 1%: 0.05<P<0.10.

<table>
<thead>
<tr>
<th>Variable</th>
<th>Baseline</th>
<th>1%*</th>
<th>3%*</th>
<th>6%*</th>
<th>7%*</th>
</tr>
</thead>
<tbody>
<tr>
<td>Core temperature (°C)</td>
<td>36.52(0.10)</td>
<td>37.52(0.10)</td>
<td>37.92(0.10)</td>
<td>38.25(0.12) †</td>
<td>38.46(0.23) †</td>
</tr>
<tr>
<td>Heart rate (beats.min⁻¹)</td>
<td>63(3.2)</td>
<td>94(5.6)</td>
<td>114(5.7) †</td>
<td>115(5.4) †</td>
<td>124(4.1) †</td>
</tr>
<tr>
<td>Forearm blood flow (mL.100 mL⁻¹.min⁻¹)</td>
<td>2.00(0.17)</td>
<td>8.58(0.63)</td>
<td>10.77(1.12)</td>
<td>8.84(1.17)</td>
<td>8.58(0.91)</td>
</tr>
<tr>
<td>Forearm vascular conductance (mL.100 mL⁻¹.min⁻¹.mmHg⁻¹)</td>
<td>1.93(0.08)</td>
<td>9.67(0.64)</td>
<td>12.68(1.47)</td>
<td>10.49(0.96)</td>
<td>9.65(1.01)</td>
</tr>
<tr>
<td>Forehead sweat rate (mg.cm⁻².min⁻¹)</td>
<td>0.20(0.02)</td>
<td>4.76(0.30)</td>
<td>4.38(0.27)</td>
<td>3.71(0.26) § (N=8)</td>
<td>3.39(0.34) † (N=7)</td>
</tr>
<tr>
<td>Scapula sweat rate (mg.cm⁻².min⁻¹)</td>
<td>0.12(0.03)</td>
<td>2.41(0.47)</td>
<td>2.46(0.52)</td>
<td>2.24(0.30) (N=9)</td>
<td>2.08(0.24) (N=7)</td>
</tr>
<tr>
<td>Whole-body sweat rate (mL.min⁻¹)</td>
<td>-----</td>
<td>17.25(1.39)</td>
<td>22.24(1.51)</td>
<td>12.45(1.32) ‡</td>
<td>11.82(1.09) ‡</td>
</tr>
</tbody>
</table>

It is possible, of course, that the failure to observe statistical differences for some of these physiological responses was due to the intrinsic inter-subject variability usually observed for skin blood flow and sweating, together with methodological artefacts that may have further increased this variability. However, it is also possible that regional differences in the sweating response existed as dehydration progressed. For instance, secretion from some skin sites may be better maintained, while that from other regions is suppressed by dehydration. The net result of these regional differences may be reduced whole-body sweating, but not an omnipresent reduction. Finally, since the current dehydration experiment was actually the third trial in a series of experiments involving exercise- and heat-induced dehydration (Taylor et al., 2009), it is possible that these subjects had adapted to some extent to these experimental conditions, and were better able to defend mean body temperature, blood pressure, and its volume and osmolality. Our thermal data are consistent with this possibility, since all subjects had to exercise harder to achieve the same target core temperatures in this third trial, whilst at the same time producing greater sweat flows.

REFERENCES


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