Fluid restriction during exercise in the heat reduces tolerance to progressive central hypovolaemia

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Abstract

This study tested the hypothesis that dehydration induced via exercise in the heat impairs tolerance to central hypovolaemia. Eleven male subjects (32 ± 7 years old, 81.5 ± 11.1 kg) walked (O₂ uptake 1.7 ± 0.4 l min⁻¹) in a 40°C, 30% relative humidity environment on three occasions, as follows: (i) subjects walked for 90 min, drinking water to offset sweat loss (Hydrated, n = 11); (ii) water intake was restricted, and exercise was terminated when intestinal temperature increased to the same level as in the Hydrated trial (Isothermic Dehydrated, n = 11); and (iii) water intake was restricted, and exercise duration was 90 min (Time Match Dehydrated, n = 9). For each trial, tolerance to central hypovolaemia was determined following exercise via progressive lower body negative pressure and quantified as time to presyncope. Increases in intestinal temperature prior to lower body negative pressure were not different (P = 0.91) between Hydrated (1.1 ± 0.4°C) and Isothermic Dehydrated trials (1.1 ± 0.4°C), but both were lower than in the Time Match Dehydrated trial (1.7 ± 0.5°C, P < 0.01). Prior to lower body negative pressure, body weight was unchanged in the Hydrated trial (−0.1 ± 0.2%), but was reduced in Isothermic Dehydrated (−0.9 ± 0.4%) and further so in Time Match Dehydrated trials (1.7 ± 0.5°C, P < 0.01). Time to presyncope was greater in Hydrated (14.7 ± 3.2 min) compared with Isothermic Dehydrated (11.9 ± 3.3 min, P < 0.01) and Time Match Dehydrated trials (10.2 ± 1.6 min, P = 0.03), which were not different (P = 0.19). These data indicate that inadequate fluid intake during exercise in the heat reduces tolerance to central hypovolaemia independent of increases in body temperature.

Some parts of the data and images are not displayed in the natural text format. The rest of the content is readable and follows the guidelines.
Haemorrhage, and subsequent central hypovolaemia and cardiovascular decompensation, is a leading cause of death in both civilian and military settings (Bellamy, 1984; Kauvar & Wade, 2005). Many individuals who are at risk for a haemorrhagic injury often undertake physical work in hot conditions [e.g. soldiers (Carter et al. 2005), miners (Brake & Bates, 2002) and firefighters (Colburn et al. 2011)], which renders them hyperthermic (i.e. elevated skin and internal temperatures) and, due to sweat loss, dehydrated (i.e. a hypovolaemic and hyperosmotic state). Notably, hyperthermia (Schlader & Crandall, 2014), dehydration (Frey et al. 1994) and physical work (i.e. exercise; Lacewell et al. 2014) can independently impair tolerance to central hypovolaemia.

Hyperthermia reduces tolerance to central hypovolaemia due, at least partly, to hyperthermia-induced decreases in central blood volume (Crandall et al. 2008) and cerebral perfusion (Wilson et al. 2006; Brothers et al. 2009; Nelson et al. 2011), together with attenuated increases in peripheral resistance during such a challenge (Crandall et al. 2010; Ganio et al. 2012; Pearson et al. 2013). Dehydration decreases the ability to withstand central hypovolaemia via similar mechanisms, such as an attenuated capacity to maintain central blood volume (Frey et al. 1994), stroke volume (Convertino, 1993; Frey et al. 1994) and cerebral perfusion (Carter et al. 2006; Romero et al. 2011) during a central hypovolaemic challenge, as well as alterations in baroreflex control of blood pressure (Convertino & Baumgartner, 1997; Charkoudian et al. 2003). Finally, exercise appears to impair tolerance to central hypovolaemia due to reductions in baroreflex sensitivity (Piepoli et al. 1993) and an attenuated ability to increase peripheral resistance during such instances (Halliwill et al. 1996; Davis & Fortney, 1997).

Our laboratory and others have investigated interactions between many of these factors on tolerance to central hypovolaemia. For instance, we identified that passively induced hyperthermia (i.e. elevated skin and internal temperatures) in combination with dehydration (1.6% body weight loss) further compromises tolerance to central hypovolaemia relative to hyperthermia during which dehydration was prevented with intravenous fluids (Lucas et al. 2013). We have also shown that hyperthermia impairs lower body negative pressure (LBNP) tolerance to a similar extent whether induced via exercise or passive heat stress when skin temperatures are similar between trials (Pearson et al. 2014). Furthermore, Davis & Fortney (1997) have identified that fluid ingestion following exercise in a moderate environment improved cardiovascular responses during central hypovolaemia, which is suggestive of improved tolerance. These studies generally support the premise that exercise together with dehydration and hyperthermia may impair tolerance to central hypovolaemia. However, interactions between dehydration, at the levels that occur during physical work in the heat without fluid replacement (i.e. drinking), and hyperthermia on the ability to tolerate central hypovolaemia are unknown. The purpose of this study, therefore, was to test the hypothesis that fluid restriction and accompanying dehydration during an exercise task performed in the heat, which is common to many occupational demands, impairs tolerance to central hypovolaemia. The testing of this hypothesis will provide important information regarding the prevention, treatment and care of individuals at risk of haemorrhagic injury and who perform physical work in the heat (e.g. soldiers, firefighters and miners). Thus, the
information obtained has direct implications for policy and practices regarding fluid consumption in many recreational and occupational settings.

**Methods**

**Subjects**

Eleven healthy, physically active men participated in this study. The subject characteristics were as follows (means ± SD): age 32 ± 7 years; height 183 ± 10 cm; weight 81.5 ± 11.1 kg; and peak oxygen uptake 3.8 ± 1.0 l min⁻¹. All subjects were non-smokers, not taking medications and were free of any known cardiovascular, metabolic, neurological or psychological diseases. Each subject was fully informed of the experimental procedures and possible risks before giving informed, written consent. The protocol and consent were approved by the Institutional Review Boards at the University of Texas Southwestern Medical Center at Dallas and Texas Health Presbyterian Hospital of Dallas. This study also conformed to the standards set by the latest revision of the Declaration of Helsinki.

Subjects visited the laboratory on four (or three; see below) occasions. Visit 1 was a screening trial, during which subjects underwent a peak exercise test using methods previously described in our laboratory (Ganio et al. 2014). The remaining visits involved the experimental trials, which are described in detail below. These trials were separated by at least 8 weeks, but completed at the same time of day (within a subject). For these trials, subjects arrived at the laboratory euhydrated (confirmed via urine specific gravity and plasma osmolality; see Table 1) and having refrained from strenuous exercise, alcohol and caffeine for a period of 24 h. Experimental testing was conducted throughout the calendar year in Dallas, TX, USA and, as a result, heat acclimatization status was not controlled.

**Instrumentation and measurements**

Approximately 60 min prior to any experimental testing, each subject swallowed a telemetry pill (HQ Inc., Palmetto, FL, USA) for the measurement of intestinal temperature. Mean skin temperature was measured as the weighted average of six thermocouples attached to the following locations: abdomen (14%), calf (11%), chest (22%), lower back (19%), thigh (14%) and upper back (19%). Heart rate was continuously recorded from an ECG (GE Healthcare, Little Chalfont, UK) interfaced with a cardiotachometer (CWE, Ardmore, PA, USA). Urine specific gravity was measured in duplicate using a refractometer (PAL-10S; Atago Inc., Bellevue, WA, USA). Body weight was measured using a standard scale (Health o meter Professional Scales, McCook, IL, USA), while oxygen uptake was measured via indirect calorimetry (Parvo Medics, Sandy, UT, USA). During LBNP (see Experimental Protocol), beat-to-beat blood pressure was measured continuously via the Penaz method (Finometer Pro; FMS, Amsterdam, The Netherlands) and confirmed intermittently via auscultation of the brachial artery by electrophygmonanometry (Tango+; SunTech, Raleigh, NC, USA). Venous blood samples were measured for haemoglobin, haematocrit (both via fluorescent flow cytometry) and plasma osmolality (via osmometry).
**Experimental protocol**

Following at least 30 min of supine rest in a thermoneutral environment, a baseline blood sample was drawn. Subjects then entered an environmental chamber maintained at 41 ± 1°C, 25 ± 4% relative humidity and exercised on a treadmill with a fan placed in front of them that produced an air velocity of 5 ± 2 m s\(^{-1}\). The speed and gradient of the treadmill were adjusted to elicit 55 ± 3% of peak oxygen uptake (1.7 ± 0.4 l min\(^{-1}\); no differences between trials, \(P = 0.560\)), which is similar to that typically observed in soldiers while on foot patrol (Buller *et al*. 2010). During exercise, oxygen uptake was measured over a 2–3 min period every 10 min during the first 30 min of exercise, and the speed and gradient were kept constant thereafter. Following thorough removal of sweat that was on the skin surface with a towel, changes in body weight (inclusive of clothing and instrumented equipment) were measured every 15 min throughout exercise.

The three experimental trials comprised different conditions that varied depending on fluid (i.e. water) consumption and exercise duration. (i) Water intake was sufficient to offset sweat losses fully throughout 90 min of exercise (Hydrated). This water was warm (38.6 ± 1.0°C), and the timing of drinking was carefully controlled such that no fluid was permitted within 5 min of measuring intestinal temperature. This prevented water temperature from influencing the measurement of intestinal temperature, which was confirmed by continuously monitoring intestinal temperature throughout the exercise, including during drinking. (ii) Water was withheld throughout exercise, and subjects exercised until they achieved the same increase in intestinal temperature as that occurring in the Hydrated trial (Isothermic Dehydrated). (iii) Water was withheld throughout exercise, and subjects exercised for the full 90 min (Time Control Dehydrated). The study was originally designed to compare only the Hydrated and Isothermic Dehydrated trials; however, given that exercise duration was substantially shorter during the Isothermic Dehydrated trial compared with the Hydrated trial (see Table 1), the Time Control Dehydrated trial was added *post hoc*. As a result, the order of the trials was not randomized. Eleven subjects completed the Hydrated and Isothermic Dehydrated trials, but only nine subjects returned to complete the final, Time Control Dehydrated trial. The characteristics of these nine subjects were as follows: age 34 ± 6 years; height 184 ± 11 cm; weight 83.9 ± 11.0 kg; and peak oxygen uptake 4.0 ± 1.0 l min\(^{-1}\).

Immediately after exercise, while remaining in the same hot environment, subjects were moved to a patient bed and placed in the supine position within the LBNP box, where they were instrumented and underwent progressive LBNP to presyncope, a model that simulates haemorrhage in humans (Cooke *et al*. 2004; Hinojosa-Laborde *et al*. 2014; Johnson *et al*. 2014). All efforts were made to ensure a rapid transition between the end of exercise and the start of LBNP, so as to mimic conditions of a person incurring a haemorrhagic injury during physical work in the heat. As a result, physiological measures were constrained to those that were considered essential for subject safety and data integrity (e.g. blood pressure and heart rate). The transition from end of exercise to the commencement of LBNP was 18 ± 3 min, which was not different between trials (\(P = 0.651\)). The LBNP commenced at 20 mmHg, with the level of LBNP increasing by 10 mmHg every 3 min until the onset of syncopal signs and symptoms, which included the following: continued self-reporting of feeling faint,
sustained nausea, rapid and progressive decreases in blood pressure resulting in sustained systolic blood pressure being <80 mmHg and/or relative bradycardia accompanied with a narrowing of pulse pressure. Notably, every LBNP trial was terminated due to haemodynamically identified syncopal signs. After exercise, the subjects were not allowed to drink fluids at any time. Venous blood samples were drawn pre-exercise (following 30 min supine rest) and immediately prior to LBNP. It should be noted that due to the relatively rapid transition between exercise and LBNP, plasma volume shifts due to changes in posture might not have been complete during the pre-LBNP blood draw (Hagan et al. 1978), which may have affected the calculated relative (percentage) changes in plasma volume. This was considered acceptable given that the primary research question involved interactions between exercise, dehydration and LBNP tolerance, while blood measures were used as indices of hydration status that were considered secondary to changes in body weight.

Data and statistical analyses

Thermal and cardiovascular data were collected at 50 Hz via a data acquisition system (MP 150; Biopac Systems Inc., Santa Barbara, CA, USA). With regard to exercise, data were analysed immediately before and at the end of exercise. During LBNP, data were analysed immediately before commencing LBNP (pre-LBNP, 60 s average) and at 20 and 30 mmHg LBNP (60 s average), which were the levels that all subjects completed fully in all trials, upon the attainment of the highest heart rate achieved during the final 2 min of LBNP (peak LBNP, 10 s average; Schlader & Crandall, 2014), and during the final 10 s of LBNP (presyncope). To isolate the effect of LBNP, these data were also analysed as the change (Δ) from pre-LBNP.

The tolerance was quantified as LBNP time, as well as via the cumulative stress index (Levine et al. 1991), which is calculated by summing the product of LBNP and the time at each level of LBNP across the trial until the test was terminated (i.e. 20 mmHg × 3 min + 30 mmHg × 3 min, etc.). Percentage changes in plasma volume from pre- to postexercise were estimated using the methods of Dill & Costill (1974).

Data from pre-exercise and the end of exercise, as well as data during LBNP, were analysed using two-way (main effects: trial × time) repeated-measures ANOVA, while data on the change from pre-exercise to the end of exercise and measures of LBNP tolerance were analysed using a one-way repeated-measures ANOVA. Where appropriate, post hoc Holm–Sidak pairwise comparisons were made. Data were analysed using SigmaPlot (version 13; Systat Software, Inc., San Jose, CA, USA). A priori statistical significance was set at P ≤ 0.05 and exact P values are reported where possible. All data are reported as mean values ± SD.

Results

Fluid restriction during exercise in the heat

Pre-exercise intestinal and mean skin temperatures, heart rate, urine specific gravity and plasma osmolality were not different between trials (P ≥0.261; Table 1). Exercise duration was 40 ± 7 min shorter during the Isothermal Dehydrated trial compared with both Hydrated
and Time Match Dehydrated trials ($P < 0.001$; Table 1). During the Hydrated trial, subjects drank 1257 ± 39 ml of water to offset sweat loss during exercise. The increase ($P < 0.001$) in intestinal temperature during exercise was greatest in the Time Match Dehydrated trial ($P < 0.001$), while, by design, the increase in intestinal temperature was not different between the Hydrated and Isothermic Dehydrated trials ($P = 0.910$; Table 1). Changes in body weight and plasma volume were graded, such that the Hydrated trial had the smallest changes with exercise, the Time Match Dehydrated trial had the greatest changes ($P < 0.001$), and the alterations occurring in the Isothermic Dehydrated trial were in between ($P ≤ 0.017$; Table 1). Plasma osmolality increased during exercise in both the Isothermic Dehydrated and Time Match Dehydrated trials ($P < 0.001$), both of which were higher than the Hydrated trial ($P < 0.001$), during which plasma osmolality decreased from pre-exercise ($P = 0.051$).

**Responses to central hypovolaemia postexercise**

During the transition from exercise to LBNP, intestinal temperature did not change relative to end-exercise values ($P ≥ 0.187$, mean difference −0.1 ± 0.4°C), such that differences between the Time Match Dehydrated trial (38.5 ± 0.5°C) compared with the Hydrated (38.0 ± 0.4°C, $P = 0.013$) and Isothermic Dehydrated trials (38.1 ± 0.3°C, $P = 0.011$) persisted at pre-LBNP. Intestinal temperature in the Hydrated and Isothermic Dehydrated trials remained not different at pre-LBNP ($P = 0.813$). Mean skin temperature increased by 1.4 ± 0.7°C from postexercise to pre-LBNP ($P < 0.001$), but this increase was not different between trials ($P = 0.667$). Not surprisingly, heart rate decreased by 41 ± 15 beats min$^{-1}$ from postexercise to pre-LBNP ($P < 0.001$), but there were no differences between trials ($P = 0.114$).

LBNP tolerance, as expressed via the cumulative stress index, was lower in the Isothermic Dehydrated ($P = 0.031$) and Time Match Dehydrated trials ($P = 0.004$) compared with the Hydrated trial, while there was no difference in tolerance between the Isothermic Dehydrated and Time Match Dehydrated trials ($P = 0.188$; Fig. 1). Likewise, LBNP time to presyncope and the final LBNP stage reached was greater in the Hydrated trial (14.7 ± 3.2 min, 60 ± 10 mmHg) compared with both the Isothermic Dehydrated (11.9 ± 3.3 min, 50 ± 10 mmHg, $P ≤ 0.031$) and Time Match Dehydrated trials (10.2 ± 1.6 min, 50 ± 10 mmHg, $P ≤ 0.019$), while there were no differences in these measures between the Isothermic Dehydrated and Time Match Dehydrated trials ($P ≥ 0.188$).

Mean arterial pressure decreased and heart rate increased throughout LBNP ($P < 0.001$); however, these changes were not statistically different between trials (trial × time interaction, $P ≥ 0.207$; Fig. 2).

**Discussion**

This study tested the hypothesis that fluid restriction during exercise in the heat impairs tolerance to central hypovolaemia and that this impairment is exacerbated with further dehydration and increases in body temperature. In support of this hypothesis, LBNP tolerance was compromised by fluid restriction when increases in internal temperature were similar (Fig. 1, see Hydrated versus Isothermic Dehydrated). In contrast to our hypothesis, however, additional dehydration (a further 1.0 ± 0.8% body weight loss) and hyperthermia (a further 0.5 ± 0.4°C increase in intestinal temperature), which occurred by matching exercise...
time, did not further compromise LBNP tolerance (Fig. 1, see Isothermic Dehydrated versus Time Match Dehydrated). The precise mechanisms underlying these alterations in LBNP tolerance are not readily apparent from the present study. However, it is clear that fluid restriction during exercise did not differentially affect the blood pressure or heart rate responses prior to or up to the point of presyncope during LBNP (Fig. 2). Overall, these data suggest that inadequate fluid intake during exercise in the heat can impair tolerance to central hypovolaemia even when it elicits only mild dehydration (~1% body weight loss). Moreover, further dehydration and increases in body temperature elicited by the time matched condition had minimal impact.

**Exercise-induced dehydration and tolerance to central hypovolaemia**

Exercise (Lacewell et al. 2014) and dehydration (Frey et al. 1994) can independently impair tolerance to central hypovolaemia, while fluid ingestion following 60 min of exercise in a moderate environment (20°C) partly alleviates cardiovascular strain during LBNP (e.g. increases in heart rate, reductions in stroke volume; Davis & Fortney, 1997). The present study extends these findings by demonstrating that fluid restriction during exercise in the heat impairs tolerance to central hypovolaemia independent of the magnitude of hyperthermia (Fig. 1). Changes in heart rate and blood pressure, both before and during LBNP, do not provide insights regarding the basic haemodynamic mechanisms of this impairment (Fig. 2). However, based on a similar study (Davis & Fortney, 1997), it can be speculated that dehydration reduced blood volume and probably attenuated the magnitude of increases in peripheral resistance. Together, these responses are likely to have compromised stroke volume (Convertino, 1993) and, ultimately, cardiac output during LBNP, which would result in an earlier precipitous drop in blood pressure and, probably, cerebral perfusion, when dehydrated.

**Hyperthermia, exercise-induced dehydration and central hypovolaemia tolerance**

Researchers in our laboratory have demonstrated that passive heating-induced hyperthermia (i.e. skin temperatures of ~38°C, increases in intestinal temperature of ~1.5°C), in the absence of dehydration, impairs LBNP tolerance and that dehydration (i.e. ~1.6% reduction in body weight) accompanying this passive heat stress exacerbates this impairment (Lucas et al. 2013). We have also observed that hyperthermia (i.e. ~1.2°C increase in intestinal temperature) impairs LBNP tolerance to a similar extent whether induced via exercise or passive heat stress when skin temperatures are not different between these trials (Pearson et al. 2014). With this background, we hypothesized that further dehydration (~1.9% reduction in body weight) and hyperthermia (~1.7°C increase in intestinal temperature) induced by exercising for 90 min without fluid consumption would further reduce LBNP tolerance in the presence of moderate skin temperatures (~35°C). Against our expectations, LBNP tolerance was not further reduced by this additional strain (Fig. 1, see Isothermic Dehydrated versus Time Match Dehydrated). These findings may be explained by a ‘basement effect’, such that mild dehydration associated with 50 min of exercise without fluid ingestion may have already reduced LBNP tolerance to such a point where further reductions would be unlikely. In support of this contention, 33% of subjects (three of nine) underwent LBNP for a slightly longer duration (by 2.4 ± 1.2 min) during the Time Match Dehydrated trial compared with the Isothermic Dehydrated trial. Notably, these three subjects were among
the four shortest LBNP durations during the Isothermic Dehydrated trial. This suggests that
the deleterious impact of mild dehydration may have exerted an effect sufficient in
magnitude to mask any further impairments induced by additional dehydration and increases
in body temperature. Alternatively, it is possible that further dehydration induced during the
time matched conditions was not severe enough to observe further reductions in the ability
of the body to tolerate central hypovolaemia. The time matched condition elicited an overall
decrease in body weight of only ~2%, and it therefore remains unknown whether greater
dehydration (e.g. 3–4% body weight loss) would further compromise tolerance to central
hypovolaemia. Nonetheless, the findings of the present study collectively suggest that the
additional effects of moderate dehydration (i.e. ~2% body weight loss) and slightly greater
increases in internal temperature (~0.5°C) elicited by the time matched condition on LBNP
tolerance are small. Therefore, mild exercise-induced dehydration that can occur during
exercise in the heat impairs tolerance to central hypovolaemia, and slightly greater elevations
in internal temperature and dehydration do not exacerbate this impairment.

**Considerations**

Due to the nature of the study design and the post hoc addition of the Time Match
Dehydrated trial, we were unable to randomize the order of the trials. Although a potential
limitation, this would appear unlikely given that LBNP tests in the present study were
conducted ~8 weeks apart. For instance, it has been demonstrated that repeated LBNP tests
in the same individuals conducted within 30 min of each other (Convertino & Sather, 2000)
and as long as 1 year apart (Convertino, 2001) produce nearly identical levels of LBNP
tolerance. Nevertheless, it must be acknowledged that the lack of randomization may have,
at least partly, masked the magnitude of the effect of dehydration on LBNP tolerance.

This study evaluated the impact of hydration status on the ability to tolerate central
hypovolaemia immediately following exercise in the heat, in an attempt to mimic conditions
of a person incurring a haemorrhagic injury during physical work in a hot environment.
Thus, in order to promote the translation of these findings to such circumstances, the time
between the end of exercise and commencement of LBNP was made as short as possible,
and instrumentation was minimized to collect only those data most important for subject
safety and data integrity. As a result, the present study provides little insight regarding the
mechanisms for the present observations. Nevertheless, given that blood pressure is the
product of cardiac output and vascular resistance, it is likely that mild dehydration reduced
the duration that cardiac output and/or vascular resistance could be sufficiently regulated to
maintain blood pressure before reaching presyncope and a precipitous drop in blood
pressure. Thus, LBNP tolerance may have been impaired during dehydration via reductions
in blood/plasma volume and/or an attenuated ability to increase vascular resistance [e.g. via
reductions in baroreflex sensitivity (Charkoudian et al. 2003)]. As a result, venous return
was likely to be lower when dehydrated for a given level of LBNP, which reduced stroke
volume and resulted in an inability to maintain cardiac output adequately. Importantly,
accurate measures of cardiac output throughout LBNP are required in order to discern the
haemodynamic mechanisms underlying impairments in tolerance to central hypovolaemia
with dehydration.
Perspectives and significance

The present study suggests that individuals performing physical work (e.g. exercise) in a hot environment will be likely to succumb earlier to a haemorrhagic injury if they fail to maintain hydration adequately prior to the insult. Importantly, the level of dehydration capable of achieving this end is relatively mild. Furthermore, slightly larger increases in internal temperature accompanied by further dehydration (to the extent assessed in the present study) do not further impair haemorrhagic tolerance. Thus, the present study has identified that even mild dehydration reduces the time line to commence treatment during a haemorrhagic injury. Such insight is important because early recognition of a haemorrhagic injury and the initiation of treatment is vital to survival after such an injury (Bellamy, 1984; McNicholl, 1994). Clearly, the results from the present study demonstrate that hydration strategies performed in operational settings that require physical work in the heat are important to protect against the adverse effects of a subsequent haemorrhagic injury.

Conclusions

The present study demonstrates that, compared with conditions when sweat losses are fully offset by drinking water, LBNP tolerance is lower when fluid is restricted during exercise in the heat and that this reduced tolerance is independent of the magnitude of hyperthermia. Furthermore, slightly greater levels of dehydration and increases in internal temperature do not further reduce LBNP tolerance. These data demonstrate that mild dehydration associated with fluid restriction during exercise in the heat can have a profound impact in impairing the ability to withstand central hypovolaemia, as occurs during a haemorrhagic injury, and that further dehydration and increases in body temperature (to the extent assessed in the present study) do not further compromise such tolerance.

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References


Ganio MS, Pearson J, Schlader ZJ, Brothers RM, Lucas RA, Rivas E, Kowalske KJ, Crandall CG. Aerobic fitness is disproportionately low in adult burn survivors years after injury. J Burn Care Res. 2014; doi: 10.1097/BCR.0b013e3182a22915


New Findings

- What is the central question of this study?
  Interactions between dehydration, as occurs during exercise in the heat without fluid replacement, and hyperthermia on the ability to tolerate central hypovolaemia are unknown.

- What is the main finding and its importance?
  We show that inadequate fluid intake during exercise in the heat can impair tolerance to central hypovolaemia even when it elicits only mild dehydration. These findings suggest that hydration during physical work in the heat has important military and occupational relevance for protection against the adverse effects of a subsequent haemorrhagic injury.
Figure 1.
Lower body negative pressure (LBNP) tolerance, expressed as the cumulative stress index, following exercise in a hot environment during which: (i) water was ingested to offset sweat losses (Hydrated, $n = 11$); (ii) water was withheld, and exercise was terminated upon the same increase in intestinal temperature relative to the Hydrated trial (Isothermic Dehydrated, $n = 11$); and (iii) water was withheld, but exercise duration was the same as that occurring during the Hydrated trial (Time Match Dehydrated, $n = 9$)
Data are mean values ± SD. Main effect of trial: $P = 0.004$. Exact $P$ values are reported for all comparisons.

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Figure 2.
Absolute values (left panels) and the change (Δ; right panels) from immediately prior to lower body negative pressure (pre-LBNP) in mean arterial pressure (top panels) and heart rate (bottom panels) at pre-LBNP, 20 mmHg LBNP, 30 mmHg LBNP, upon the attainment of the highest heart rate achieved during the final 2 min of LBNP (peak LBNP) and at presyncope.

LBNP to presyncope was undertaken following exercise in a hot environment during which: (i) water was ingested to offset sweat losses (Hydrated, n = 11); (ii) water was withheld, and exercise was terminated upon the same increase in intestinal temperature as the Hydrated trial (Isothermal Dehydrated, n = 11); and (iii) water was withheld, but exercise duration was the same as that occurring during the Hydrated trial (Time Match Dehydrated, n = 9). Data are mean values ± SD. It should be noted that peak LBNP and presyncope occurred at different absolute levels of LBNP during each trial. There were no trial × time interactions for any comparisons (P values for these interactions are reported). Changes over time, independent of trial, are indicated as follows: (1) different from pre-LBNP (P ≤ 0.006); (2) different from 20 mmHg (P < 0.001); (3) different from 30 mmHg (P ≤ 0.044); and (4) different from peak LBNP (P ≤ 0.010).
### Table 1

Thermal and hydration indices pre-exercise and at end of exercise in the heat

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Hydrated</th>
<th>Isothermic Dehydrated</th>
<th>Time Match Dehydrated</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Pre-exercise</td>
<td>End of exercise</td>
<td>Pre-exercise</td>
</tr>
<tr>
<td>Exercise time (min)</td>
<td>—</td>
<td>90 ± 0</td>
<td>—</td>
</tr>
<tr>
<td></td>
<td></td>
<td>90 ± 0</td>
<td>50 ± 19 *†</td>
</tr>
<tr>
<td>Intestinal temperature (°C)</td>
<td>37.0 ± 0.2</td>
<td>38.1 ± 0.3a</td>
<td>37.0 ± 0.2</td>
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<td></td>
<td></td>
<td>38.1 ± 0.4a</td>
<td>38.1 ± 0.4a</td>
</tr>
<tr>
<td>Δ Intestinal temperature (°C)</td>
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<td>1.1 ± 0.4</td>
<td>—</td>
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<td></td>
<td></td>
<td>1.1 ± 0.4</td>
<td>1.1 ± 0.4</td>
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<tr>
<td>Mean skin temperature (°C)</td>
<td>35.5 ± 0.4</td>
<td>34.7 ± 1.0a</td>
<td>35.3 ± 0.7</td>
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<td></td>
<td></td>
<td>34.7 ± 1.0a</td>
<td>35.2 ± 0.9</td>
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<tr>
<td>Heart rate (beats min⁻¹)</td>
<td>89 ± 13</td>
<td>127 ± 13a</td>
<td>88 ± 11</td>
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<tr>
<td></td>
<td></td>
<td>88 ± 11</td>
<td>136 ± 17a</td>
</tr>
<tr>
<td>Urine specific gravity</td>
<td>1.013 ± 0.007</td>
<td>—</td>
<td>1.013 ± 0.007</td>
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<tr>
<td>Δ Body weight (kg)</td>
<td>—</td>
<td>−0.1 ± 0.2</td>
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<td></td>
<td></td>
<td>−0.1 ± 0.2</td>
<td>−0.8 ± 0.4a</td>
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<tr>
<td>Δ Body weight (%)</td>
<td>—</td>
<td>−0.1 ± 0.2</td>
<td>−0.9 ± 0.4a</td>
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<td></td>
<td></td>
<td>—</td>
<td>−1.7 ± 0.5</td>
</tr>
<tr>
<td>Δ Plasma volume (%)</td>
<td>—</td>
<td>−5.9 ± 3.3</td>
<td>—</td>
</tr>
<tr>
<td></td>
<td></td>
<td>−5.9 ± 3.3</td>
<td>−8.1 ± 3.4a</td>
</tr>
<tr>
<td></td>
<td></td>
<td>—</td>
<td>−11.2 ± 2.6 e</td>
</tr>
<tr>
<td>Plasma osmolality (mosmol kg⁻¹)</td>
<td>288 ± 6</td>
<td>286 ± 5b</td>
<td>288 ± 2</td>
</tr>
<tr>
<td></td>
<td></td>
<td>288 ± 2</td>
<td>293 ± 3e</td>
</tr>
<tr>
<td></td>
<td></td>
<td>288 ± 3</td>
<td>295 ± 4a</td>
</tr>
</tbody>
</table>

Values are means ± SD. Trials are as follows: Hydrated, n = 11; Isothermic Dehydrated, n = 11; and Time Match Dehydrated, n = 9. Δ indicates change from pre-exercise; * significantly different from the Hydrated trial (P ≤ 0.017); † significantly different from the Time Match Dehydrated trial (P ≤ 0.002); a significantly different from pre-exercise within the trial (P ≤ 0.002); and b significantly different from pre-exercise within the trial (P = 0.051).