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ORIGINAL ARTICLE

Treatment of exertional heat stress developed during low or moderate physical work

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Abstract
Purpose We examined whether treatment for exertional heat stress via ice water immersion (IWI) or natural recovery is affected by the intensity of physical work performed and, thus, the time taken to reach hyperthermia.
Methods Nine adults (18–45 years; 17.9 ± 2.8 percent body fat; 57.0 ± 2.0 mL kg^{-1} min^{-1} peak oxygen uptake) completed four conditions incorporating either walking or jogging at 40 °C (20 % relative humidity) while wearing a non-permeable rain poncho. Upon reaching 39.5 °C rectal temperature (T_{re}), participants recovered either via IWI in 2 °C water or via natural recovery (seated in a ~29 °C environment) until T_{re} returned to 38 °C.
Results Cooling rates were greater in the IWI [T_{re}: 0.24 °C min^{-1}; esophageal temperature (T_{es}): 0.24 °C min^{-1}] than the natural recovery (T_{re} and T_{es}: 0.03 °C min^{-1}) conditions ($p < 0.001$) with no differences between the two moderate and the two low intensity conditions ($p > 0.05$). Cooling rates for T_{re} and T_{es} were greater in the 39.0–38.5 °C (T_{re}: 0.19 °C min^{-1}; T_{es}: 0.31 °C min^{-1}) compared with the 39.5–39.0 °C (T_{re}: 0.11 °C min^{-1}; T_{es}: 0.13 °C min^{-1}) period across conditions ($p < 0.05$). Similar reductions in heart rate and mean arterial pressure were observed during recovery across conditions ($p > 0.05$), albeit occurred faster during IWI. Percent change in plasma volume at the end of natural recovery and IWI was 5.96 and 9.58 %, respectively ($p < 0.001$).
Conclusion The intensity of physical work performed and, thus, the time taken to reach hyperthermia does not affect the effectiveness of either IWI treatment or natural recovery. Therefore, while the path to hyperthermia may be different for each patient, the path to recovery must always be immediate IWI treatment.

Keywords Exercise-induced hyperthermia · Cooling · Cold water immersion · Recovery · Core temperature

Abbreviations

EHS	Exertional heat stress
FH	Fast heating condition with no cooling recovery
FH + IWI	Fast heating condition with ice water immersion recovery
IWI	Ice water immersion
SH	Slow heating condition with no cooling recovery
SH + IWI	Slow heating with ice water immersion recovery
T_{re}	Rectal temperature
T_{es}	Esophageal temperature
$\dot{V}O_{2peak}$	Peak oxygen consumption

Introduction

Exertional heat-related illness is a risk to all individuals who are physically active in temperate or hot environments

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and represents a serious medical condition that can lead to severe consequences such as multi-system organ failure and cell death (Casa et al. 2012; Armstrong et al. 2007). Despite the severity of this issue, currently we do not know whether the treatment for exertional heat-relates illness should vary depending on the nature of the physical effort that caused hyperthermia. This knowledge gap is an important one to address to reduce the number of fatalities due to exertional heat-related illness.

Individuals suffering from exertional heat-related illness demonstrate markedly attenuated whole-body heat loss (Kenny et al. 2006; Flouris and Cheung 2010). Under these conditions, cooling treatments which enhance heat dissipation by increasing conductive and/or convective heat transfer and/or evaporative heat loss are critical for the victim's survival (Casa et al. 2007). This is because the primary goal of acute and emergency care in exertional heat-related illness is to restore core temperature to near-normal levels as rapidly as possible (Armstrong et al. 2007; Casa et al. 2012). Ice water immersion (IWI, circulated water of 2 °C) provides some of the highest core temperature cooling rates (0.35 °C min^{-1}) (Proulx et al. 2003). It is not surprising, therefore, that IWI has been proposed as the optimum treatment for exertional heat-related illness (Casa et al. 2007, 2012).

Much of our understanding of the benefits of IWI in the treatment of exertional heat-related illness stems from studies that used exertional heat stress (EHS) as a model. These studies measured cooling effectiveness during an elevated state of hyperthermia of ≥39.5 °C generated by a short duration (~35–45 min) moderate intensity [~65–70 % peak oxygen consumption ($\dot{V}O_{2peak}$)] exercise performed in the heat (~39–42 °C) (Proulx et al. 2003; Gagnon et al. 2010; Lemire et al. 2008, 2009). However, even lighter exercise intensities may lead to exertional heat-related illness in individuals exposed to extreme heat. For example, specific occupations such as industrial workers, military personnel (Rohe 2012), and athletes, as well as the general population (Nelson et al. 2011) commonly experience elevated levels of hyperthermia while performing light physical work in uncompensable heat stress conditions (Brake and Bates 2002; Cuddy and Ruby 2011; Hunt et al. 2013). This occurs in cases where the environmental temperature and humidity are very high and/or the necessity to wear protective clothing restricts the body's ability to dissipate internal heat (Cheung et al. 2000; Flouris 2011). In these circumstances, the severity of EHS has been associated with the duration of core temperature elevation (Armstrong et al. 2007; Smith 2005). However, differences in the rate of increase in core temperature may also be a potential factor affecting the time-line development and severity of physical signs and symptoms of EHS (Hales and Richards 1988; Febbraio 2000).

We recently assessed the effectiveness of different EHS treatments on restoring heart rate variability (Flouris et al. 2014). Our objective in this paper was to examine whether treatment for EHS via IWI or natural recovery is affected by the intensity of physical work performed and, thus, the time taken to reach a hyperthermic state of 39.5 °C. We evaluated the hypothesis that core cooling rates during IWI in individuals rendered hyperthermic would not differ between the exercise intensity conditions despite exercise-induced differences in thermal and cardiovascular responses. Specifically, we hypothesized that any potential effects of differences in core temperature heating rates (e.g., differences in tissue blood flow and/or heat distribution) would be minimized by the very powerful cooling capacity of IWI which was demonstrated previously in our afore-mentioned study (Proulx et al. 2003). We also hypothesized that marked differences in the thermal and cardiovascular responses would also be observed between exercise intensity conditions during the natural recovery condition.

Materials and methods

Participants and procedures

The current experimental protocol was approved by the University of Ottawa Health Sciences and Science Research Ethics Board, in accordance with the Declaration of Helsinki. Following appropriate determination of sample size (see "Statistical analysis"), seven men and two women aged 18–45 years who were healthy (no history of respiratory, metabolic, or cardiovascular conditions) and physically active were recruited from the university population and general community. Participants were informed of all experimental procedures, associated risks, and discomforts prior to their participation in the study and provided written consent. Females were tested during the early-to-mid follicular phase of their menstrual cycle. The physical characteristics of the participants were: 26.0 ± 2.8 years, 174.2 ± 3.8 cm, 74.6 ± 4.3 kg, 1.89 ± 0.07 m^2 body surface area, 17.9 ± 2.8 percent body fat, and 57.0 ± 2.0 mL kg^{-1} min^{-1} $\dot{V}O_{2peak}$ (mean ± SD). The current data were collected as part of a larger study, the main paper of which was recently published in this journal (Flouris et al. 2014).

Experimental protocol

The participants completed the American Heart Association/American College of Sports Medicine Pre-Participation Screening (Balady et al. 1998) and the Canadian Society for Exercise Physiology Physical Activity Readiness

(CSEP 2002) questionnaires during a preliminary session to ensure their safety for participation. Aerobic fitness and body composition assessments were also conducted at that time using standardized protocols (Flouris et al. 2014).

Following the preliminary session, all participants completed four EHS sessions separated by ≥5 days in a random order using a crossover design. Participants were asked to refrain from intense exercise (running, swimming, cycling, weight lifting, etc.), alcohol, and the use of over-the-counter medications for 24 h before, and caffeine for 12 h before each EHS session. During the EHS sessions, participants remained rested for 30 min of baseline measurements in an upright seated position. Thereafter, to attain a similar rate of increase in rectal temperature (T_{re}), participants either walked (4.0–4.5 km h^{-1}, 2 % incline, ~0.3 °C min^{-1}) or jogged (~7.0 km h^{-1}, 2 % incline, ~0.7 °C min^{-1}) on a treadmill in a hot-dry environment (40 °C, 20 % relative humidity) while wearing shorts and a non-permeable rain poncho for a slow or fast rate of T_{re} increase, respectively. Upon reaching a T_{re} of 39.5 °C, participants were asked to remove the rain poncho and recovered until T_{re} returned to 38 °C either naturally (passive recovery upright seated in a ~29 °C ambient environment) or with IWI treatment (immersion in 2 °C water, upright seated with legs straight in front). Thus, the four EHS sessions, which were performed in a counterbalanced manner, included: fast heating with no cooling recovery (FH), fast heating with ice water immersion recovery (FH + IWI), slow heating with no cooling recovery (SH), and slow heating with ice water immersion recovery (SH + IWI).

For the IWI recovery, the participants were transferred to the immersion bath 5 min after the end of exercise. They were immersed up to the upper part of their chest, slightly below the level of the clavicle. Their arms were supported at the level of the heart and were not immersed in the water at any time for the purpose of measuring blood pressure. The participants wore neoprene boots during the immersion period to protect the extremities from cold injury. Once participants' T_{re} returned to 38 °C, they were removed from the immersion bath and towel dried. Thereafter, they were monitored until T_{re} returned to ~0.5 °C of baseline.

Physiological measurements

Core and skin temperatures

T_{re} was measured continuously using a general purpose thermistor (Mon-a-therm Nasopharyngeal Temperature Probe, Mallinckrodt Medical, St. Louis, MO, USA) inserted approximately 15 cm beyond the anal sphincter. To ensure that the thermistor did not move during changes in body position, the thermistor was affixed to the skin of the dorsal pelvic region with surgical tape (Transpore, 3M Health Care, St. Paul, USA). The same thermistor was used to measure esophageal temperature (T_{es}). The probe was inserted 40 cm past the entrance of the nostril while the participants sipped water (100–300 mL) through a straw. Skin temperature was measured at ten sites using thermistors (Concept Engineering, Old Saybrook, CT, USA) attached to the skin with surgical tape. Mean skin temperature (T_{sk}) was subsequently calculated using a 10-point weighting of the regional proportions as previously described (Hardy and DuBois 1938). Temperature data were collected using a HP Agilent data acquisition module (model 3497A) every 15 s and simultaneously displayed and recorded in spreadsheet format on a personal computer with LabVIEW software (Version 7.0, National Instruments, TX, USA).

Cardiovascular measures

Systolic and diastolic blood pressures were measured using the Welch Allyn® ABPM 6100 (Welch Allyn®, Skaneateles Falls, NY, USA) at baseline, the start of recovery, and every 2 min throughout the IWI recovery conditions or 5 min throughout the natural recovery conditions for the calculation of mean arterial pressure (MAP).

Plasma volume

Venous blood was collected via an indwelling intravenous catheter (Jelco I.V. 18G Catheter, Smiths Medical International, Rossendale, Lancashire, UK), inserted into an antecubital vein, with a 21 in. extension line. Blood was collected at rest, at a T_{re} of 38.0, 39.0, and 39.5 °C during EHS, at the beginning of recovery, and at a T_{re} of 39.0 and 38.0 °C during recovery. Blood was drawn into sterile plastic syringes and transferred immediately into 5.4 mg plasma K2EDTA BD Vacutainer tubes (BD, Franklin Lakes, NJ, USA). EDTA blood was mixed by inversion and used to measure hematological parameters (Beckman Coulter, Miami, FL, USA). Blood hemoglobin and hematocrit values were used to estimate the percent change in plasma volume (Dill and Costill 1974).

Statistical analysis

The minimum required sample size was determined using data from a recent study that assessed the efficacy of different EHS cooling treatments (Leicht et al. 2009). Sample size calculations were conducted using G*Power 3.0 (Faul et al. 2007). The A.R.E. method of the "Wilcoxon signed-rank test" incorporated in the "*t* tests" family with "a priori" as the type of power analysis was used to calculate the power of the within effect. A two-tailed test was selected. Statistical power and α error probability were set to 0.95 and 0.05, respectively. The minimum required sample size

Table 1 Exercise time and physiological measures (mean ± SE) at the end of the exercise-induced hyperthermia for the four conditions

	FH	FH + IWI	SH	SH + IWI
Exercise time (min)	34.8 ± 2.6	39.8 ± 4.1	76.2 ± 5.1	86.1 ± 6.8[a]
Warming rate (°C min^{-1})				
T_{re}	0.07 ± 0.00	0.07 ± 0.01	0.03 ± 0.00	0.03 ± 0.00[a]
T_{es}	0.09 ± 0.01	0.08 ± 0.01	0.03 ± 0.00	0.03 ± 0.00[a]
End temperature (°C)				
T_{re}	39.50 ± 0.03	39.51 ± 0.00	39.50 ± 0.01	39.51 ± 0.00
T_{es}	39.84 ± 0.17	39.81 ± 0.11	39.40 ± 0.08	39.41 ± 0.05[a]
T_{sk} (°C)	37.45 ± 0.12	37.11 ± 0.16	37.37 ± 0.08	37.13 ± 0.19
Heart rate (beats min^{-1})	188 ± 3	191 ± 3	181 ± 3	183 ± 4
MAP (mmHg)	90 ± 1	86 ± 2	86 ± 1	87 ± 1
Plasma volume (% change)	−9.58 ± 0.95	−9.35 ± 1.88	−12.53 ± 1.31	−11.29 ± 1.47

FH fast heating with no cooling recovery, *FH + IWI* fast heating with ice water immersion recovery, *SH* slow heating with no cooling recovery, *SH + IWI* slow heating with ice water immersion recovery, T_{re} rectal temperature, T_{es} esophageal temperature, T_{sk} skin temperature

[a] Significantly different from the fast heating conditions ($p < 0.05$)

was determined by calculating the effect size *d*. Using the afore-mentioned published data (Leicht et al. 2009), the resulting minimum required sample size was seven participants. To confidently detect a reasonable departure from the null hypothesis, we recruited a total of nine participants.

A one-way ANOVA was used to assess changes in T_{re} and T_{es} between rest, end of exercise, start of recovery, and end of recovery. A one-way ANOVA was used to compare the exercise times, the recovery times, the warming and cooling rates between the experimental conditions, and the T_{re} and T_{es} change between the end of exercise and start of recovery (immediately upon immersion/start of natural recovery). During exercise, a two-way ANOVA with repeated measures of condition (FH, FH + IWI, SH, and SH + IWI) × time (to the common point of 25 min) was performed on the heart rate and T_{sk}. During recovery, a two-way ANOVA with repeated measures of condition (FH, FH + IWI, SH, and SH + IWI) × T_{re} (39.5, 39.0, and 38.0 °C) was performed on T_{sk}, heart rate, percent change in plasma volume, and MAP. If a significant *F* ratio was obtained, a Newman–Keuls post hoc analysis was used to isolate differences among treatment means. Results are reported as mean ± SE. Effects were deemed statistically significant at $p < 0.05$, while trends were identified for $0.05 < p < 0.1$.

Results

Exercise

The warming rates to reach a T_{re} of 39.5 °C and the physiological measures at the end of exercise are shown in Table 1. No differences were observed for the rate of increase in T_{re} or T_{es} between FH and FH + IWI ($p = 0.128$ and $p = 0.187$, respectively) as well as between SH and SH + IWI ($p = 0.539$ and $p = 0.728$, respectively). There was no main effect of condition at rest for T_{re} ($p = 0.785$) or T_{es} ($p = 0.986$) as well as at the end of exercise for T_{re} ($p = 0.840$). However, T_{es} was significantly higher at the end of exercise following FH and FH + IWI compared to SH and SH + IWI ($p < 0.05$). At the end of exercise, there were no differences in heart rate ($p = 0.133$) or T_{sk} ($p = 0.250$) between the four conditions. Also, the percent change in plasma volume tended to be larger in the SH and SH + IWI compared to the FH and FH + IWI conditions, yet the differences were not statistically significant ($p = 0.09$).

Recovery

The changes in T_{re} and T_{es} during the recovery phase of the four conditions are illustrated in Fig. 1. During recovery, no main effect of condition was observed between FH and SH for T_{re} ($p = 0.075$) or T_{es} ($p = 0.233$) or between FH + IWI and SH + IWI for T_{re} ($p = 0.396$) or T_{es} ($p = 0.638$). A main effect of condition was observed for T_{es} at the end of recovery, such that T_{es} was significantly greater during the no-cooling compared to the IWI conditions ($p < 0.001$). Compared to rest, T_{es} was significantly elevated at the end of recovery for FN (37.78 ± 0.09 °C, $p < 0.001$) and SN (37.78 ± 0.06 °C, $p < 0.001$), yet not for FC + IWI (35.94 ± 0.44 °C, $p = 0.062$) or SC + IWI (36.27 ± 0.29 °C, $p = 0.093$).

Table 2 summarizes the T_{re} and T_{es} cooling rates, where overall cooling rates were calculated as a function of the time for T_{re} to return to 38.0 °C from the start of recovery. No differences existed for the range of cooling rates

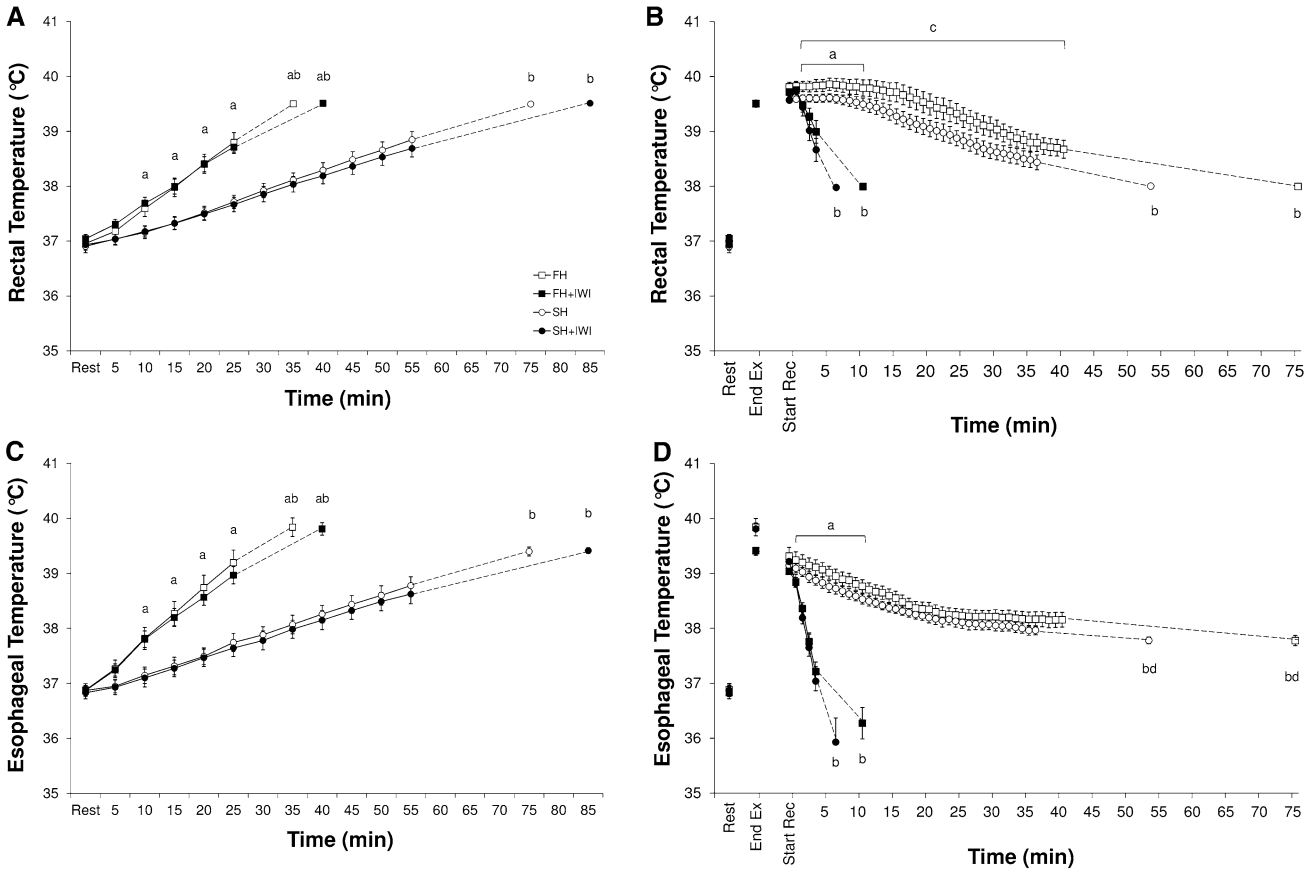

Fig. 1 Rectal and esophageal temperatures (mean ± SE) during the heating (**A**, **C**) and the recovery (**B**, **D**) phases of the four conditions. *Note* [a]significant differences in temperature between fast heating and slow heating conditions ($p < 0.05$); [b]significant difference in time between fast heating and slow heating conditions ($p < 0.05$); [c]significant differences in temperature between fast and slow heating conditions ($p < 0.05$); [d]significant differences in temperature between cooling and no cooling conditions at the end of recovery ($p < 0.05$); *End Ex* end of exercise, *Start Rec* start of recovery phase

for T_{re} or T_{es} between the two natural recovery conditions (Table 2); however, the T_{re} rate of cooling was greater from 39.0 to 38.5 °C compared to the start of recovery to 39.0 °C ($p = 0.006$) and greater from 39.0 to 38.5 °C compared to the rate from 38.5 to 38.0 °C ($p = 0.005$). Similarly, no differences existed for the range of cooling rates for T_{re} or T_{es} between the two IWI recovery conditions (Table 2); however, the rates of cooling were greater from 39.0 to 38.5 °C for T_{re} ($p < 0.001$) and T_{es} ($p < 0.001$), and from 38.5 to 38.0 °C for T_{re} ($p = 0.002$) and T_{es} ($p < 0.001$), compared to the start of recovery to 39.0 °C. Table 3 outlines the continued increase in T_{re} and decrease in T_{es} during the transition period from the end of exercise to the start of recovery/immersion (mean 6.25 ± 0.55 min), accounting for the reduced subject numbers for some T_{es} cooling rates presented in Table 2, given that some individuals T_{es} had already cooled below the temperature at which the cooling range was calculated (Fig. 1).

A condition × T_{re} interaction was observed for T_{sk} ($p < 0.001$), such that T_{sk} was greater during the no-cooling recovery compared to the IWI recovery at the start of recovery, a T_{re} of 39.0 °C, and the end of recovery at a T_{re} of 38.0 °C. However, we detected no differences in T_{sk} between FH and SH ($p = 0.491$) or between FH + IWI and SH + IWI ($p = 0.156$) (Fig. 2).

Similar reductions in heart rate ($p = 0.449$) and MAP ($p = 0.632$; Fig. 3) were observed with decreases in core temperature from the start until the end of recovery for the four conditions, albeit occurred more quickly during the IWI compared to the no-cooling conditions given the shorter duration to reach a T_{re} of 38.0 °C in the IWI conditions. Compared to rest, MAP remained significantly reduced for FH ($p = 0.025$) and SH ($p = 0.027$) at the end of recovery, however, had returned to similar resting values for FH + IWI ($p = 0.618$) and SH + IWI ($p = 0.265$).

A condition × T_{re} interaction was observed for percent change in plasma volume ($p = 0.001$). Specifically, with decreases in T_{re} during recovery, percent change in plasma volume remained at higher levels during the FH (−5.46 ± 1.64 %) and SH (−6.45 ± 2.22 %)

Table 2 Core temperature cooling rates (°C min^{-1}; mean ± SE) during recovery for the four conditions

	FH	FH + IWI	SH	SH + IWI
T_{re} (°C)				
39.5–39.0	0.027 ± 0.003	0.175 ± 0.026	0.028 ± 0.03	0.196 ± 0.021
39.0–38.5	0.035 ± 0.007[a,b]	0.295 ± 0.063[a]	0.051 ± 0.006[a,b]	0.363 ± 0.048[a]
38.5–38.0	0.027 ± 0.005	0.297 ± 0.078[a]	0.030 ± 0.004	0.292 ± 0.026[a]
Overall	0.026 ± 0.002	0.219 ± 0.040	0.031 ± 0.002	0.263 ± 0.027
T_{es} (°C)				
39.5–39.0	0.061 ± 0.016	0.251 ± 0.058	0.068 ± 0.011	0.147 ± 0.029
39.0–38.5	0.041 ± 0.008	0.700 ± 0.076[a]	0.053 ± 0.008	0.450 ± 0.040[a]
38.5–38.0	0.037 ± 0.009	0.570 ± 0.062	0.033 ± 0.008	0.470 ± 0.106[a]
Overall	0.022 ± 0.003	0.367 ± 0.048	0.031 ± 0.002	0.429 ± 0.034

FH fast heating with no cooling recovery, *FH + IWI* fast heating with ice water immersion recovery, *SH* slow heating with no cooling recovery, *SH + IWI* slow heating with ice water immersion recovery, T_{re} rectal temperature, T_{es} esophageal temperature

[a] Significantly different from 39.5 to 39.0 °C cooling rate ($p < 0.05$)

[b] Significantly different from 38.5 to 38.0 °C cooling rate ($p < 0.05$)

Table 3 Core temperatures (°C; mean ± SE) at the end of exercise and start of recovery for the four conditions

	FH	FH + IWI	SH	SH + IWI
T_{re}				
End exercise	39.50 ± 0.03	39.51 ± 0.00	39.5 ± 0.01	39.51 ± 0.00
Start recovery	39.81 ± 0.08[a]	39.72 ± 0.07	39.57 ± 0.04	39.57 ± 0.05
Change	0.31 ± 0.07[a,b]	0.21 ± 0.08	0.08 ± 0.04	0.06 ± 0.05
T_{es}				
End exercise	39.84 ± 0.16[a,b]	39.81 ± 0.11[a,b]	39.40 ± 0.08	39.41 ± 0.05
Start recovery	39.31 ± 016	39.22 ± 0.12	39.14 ± 0.07	39.04 ± 0.06
Change	−0.53 ± 0.07[a]	−0.59 ± 0.05[a,b]	−0.26 ± 0.05	−0.38 ± 0.06

FH fast heating with no cooling recovery, *FH + IWI* fast heating with ice water immersion recovery, *SH* slow heating with no cooling recovery, *SH + IWI* slow heating with ice water immersion recovery, T_{re} rectal temperature, T_{es} esophageal temperature

[a] Significantly different from SH ($p < 0.05$)

[b] Significantly different from SH + IWI ($p < 0.05$)

compared to FH + IWI (−10.33 ± 2.20 %) and SH + IWI (−8.82 ± 1.97 %) conditions ($p < 0.001$) due to the shorter duration to reach a T_{re} of 38.0 °C in the IWI conditions. However, no statistically significant differences were observed between the FH and SH ($p = 0.402$) or between the FH + IWI and SH + IWI ($p = 0.978$) conditions.

Discussion

The key finding of this study is that the exercise-induced rate of increase in core temperature does not influence core cooling rates in EHS individuals during either IWI treatment or natural recovery. Thus, the etiology of EHS may be different in each case but the treatment must always be immediate IWI. Despite the significantly longer exercise times during the SH and SH + IWI, the levels of thermal and cardiovascular strain were comparable at end exercise. However, both T_{re} and T_{es} showed a greater transient increase over the 5-min transition period between end exercise and start of recovery during FH and FH + IWI, most likely reflecting differences in tissue blood flow and, therefore, tissue heat distribution (Scott et al. 2004). Ultimately, this greater residual increase in core temperature resulted in a longer recovery time following the fast heating protocol during the natural recovery condition (despite the observed similar cooling rates). While cooling recovery time to a T_{re} of 38.5 °C tended to be longer following fast compared to slow heating, the powerful cooling of IWI negated any effect of differences on tissue heat distribution.

Evidence suggests that the body's ability to dissipate heat is acutely compromised following a dynamic exercise bout (Kenny et al. 2006; Flouris and Cheung 2010). Factors determining post-exercise cardiovascular status have

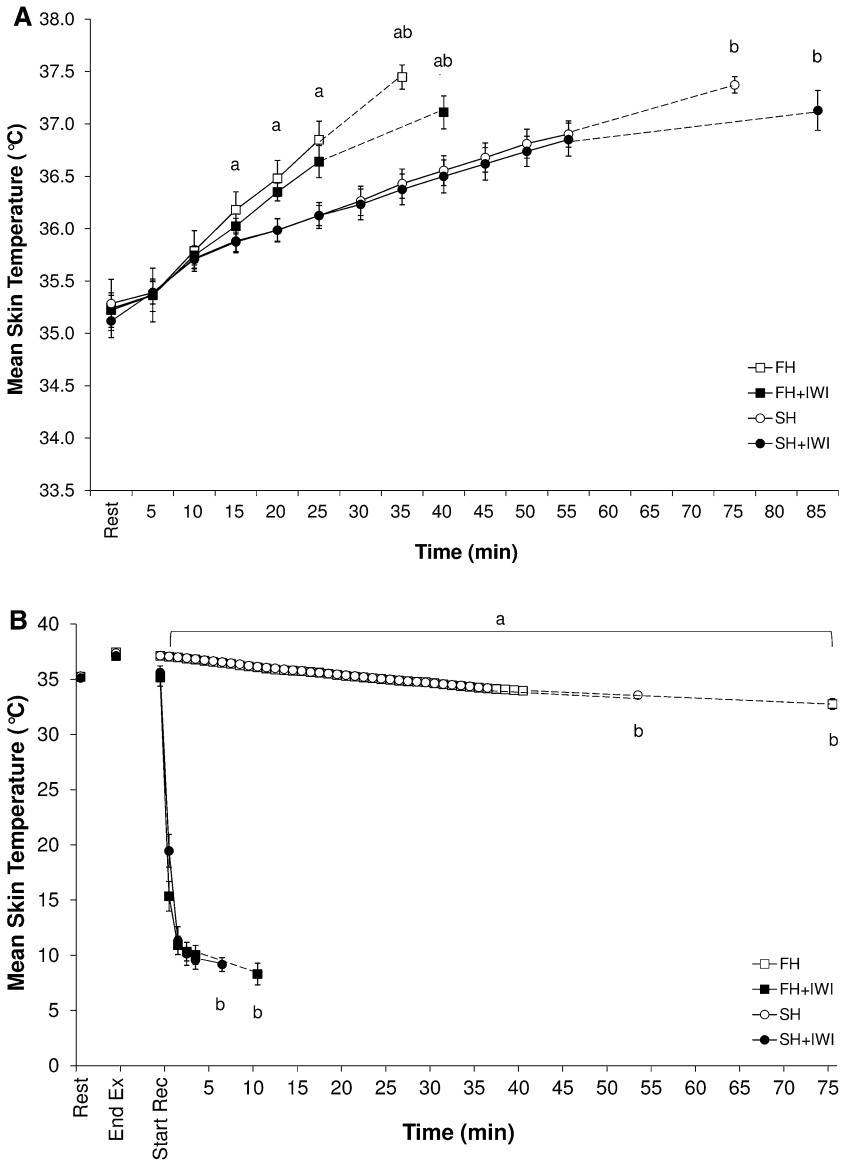

Fig. 2 Mean skin temperature (mean ± SE) during the heating (**A**) and the recovery (**B**) phases of the four conditions. *Note* [a]significant differences in temperature between fast heating and slow heating conditions ($p < 0.05$); [b]significant difference in time between fast heating and slow heating conditions ($p < 0.05$); *End Ex* end of exercise, *Start Rec* start of recovery phase

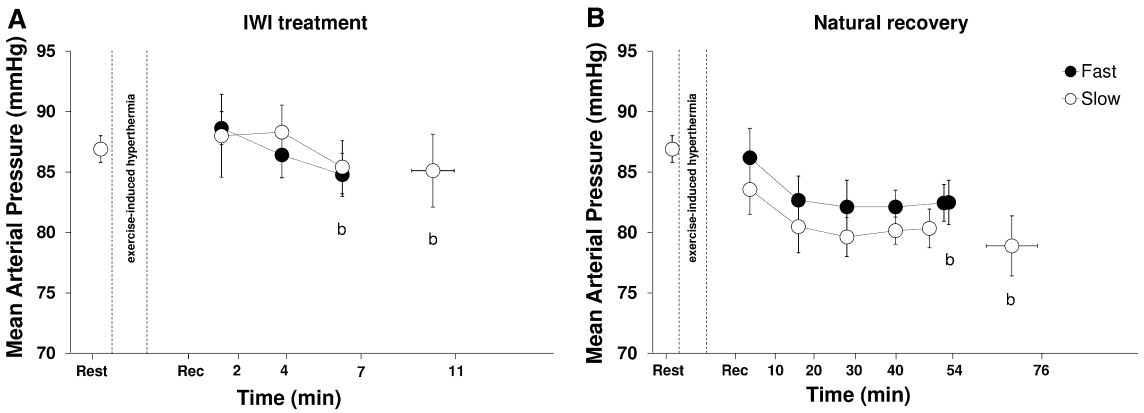

Fig. 3 Mean arterial pressure (mean ± SE) during the recovery phase in the ice water immersion (IWI) treatment (**A**) and the natural recovery (**B**) conditions. *Note* [b]significant difference in time between fast heating and slow heating conditions ($p < 0.05$); *Rec* start of recovery phase

been shown to have a strong influence on thermoregulatory control of heat loss responses (Kenny et al. 2008). During exercise, a large proportion of blood is redistributed to the skin and the working muscles (Rowell 1974). During post-exercise recovery, a significant portion of this blood remains pooled in previously active musculature (Halliwill 2001) resulting in a sustained reduction in mean arterial blood pressure. The current MAP reduction of ~5 mmHg from baseline levels is in line with previous data from our group for this exercise intensity (Kenny et al. 2006). While the observed MAP attenuation may appear physiologically insignificant, studies show that this level of post-exercise reduction in blood pressure is paralleled by a concomitant decrease in heat loss responses of skin blood flow and sweating which results in a decrease in the rate of core and muscle temperature decay (Kenny et al. 2006; Jackson and Kenny 2003). Thus, it is important to note that MAP was restored to near-resting levels at the end of recovery in the IWI trials but not in the natural recovery trials, suggesting improved heat loss. Nevertheless, the current design does not allow us to assess whether this was attributed to the hydrostatic effect of sitting in water in the IWI condition. Factors such as hydration status, the level of hyperthermia, exercise intensity, exercise duration, etc. have been shown to independently influence the magnitude of the response (Kenny et al. 2003a, b). Indeed, we found that the rate of T_{es} decay is attenuated in the mid-stage of recovery and the magnitude of this attenuation is comparable in both natural recovery and IWI conditions. This response is consistent with a non-thermal suppression of heat loss associated with the post-exercise hypotension response (Kenny et al. 2006). It was recently shown that such actions of non-thermal reflexes are lessened in the presence of a greater core temperature (Gagnon et al. 2010). Despite different rates of heat production employed in this study, exercise was continued until a similar level of hyperthermia was achieved. Taken together, these findings demonstrate that the level of hyperthermia is the main factor affecting post-exercise heat dissipation in both natural recovery and IWI conditions.

Previous studies demonstrated that the significant hemodynamic adjustments following exercise can have marked effects on regional heat distribution. Indeed, the increase in post-exercise hypotensive response, induced by exercise of increasing intensity, is paralleled by an increase in the magnitude and recovery time of the post-exercise esophageal and muscle temperatures (Kenny et al. 2006). In the current study, no differences in post-exercise hypotension were observed between the two FH (i.e., FH and FH + IWI) and the two SH (i.e., SH and SH + IWI) conditions and this was paralleled by similar T_{es} responses. As such, the differences in time of recovery are more likely associated with differences in hemodynamic responses and, therefore, tissue heat distribution during the exercise per se and the very early stages of recovery. Nevertheless, the fact remains that for the same reduction in MAP and, therefore, level of cardiovascular strain, individuals experienced a greater level of thermal strain and over a longer period of recovery during the FH conditions.

In a recent paper from the same study, we assessed the effectiveness of different EHS treatments on restoring heart rate variability (Flouris et al. 2014). Our analyses showed that IWI and natural recovery have similar capacities for restoring autonomic nervous system function following EHS, yet IWI requires only ~13 % of the time needed by natural recovery to restore T_{re}. In the current paper, we focus on whether treatment for EHS via IWI or natural recovery is affected by the intensity of physical work performed and, thus, the time taken to reach a hyperthermic state. To achieve this purpose, we studied both thermal (i.e., core/skin temperatures, cooling rates) and non-thermal (i.e., cardiovascular indices and plasma volume) factors. Despite the similar level of thermal strain achieved during exercise (as defined by T_{re} of 39.5 °C), the time required to achieve the end recovery target temperature of 38.0 °C was significantly greater following the fast heating protocol. Yet as noted above, we observed similar core cooling rates. Upon closer examination, despite achieving similar end exercise T_{re} (i.e., 39.5 °C), the corresponding T_{es} was significantly different (i.e., 39.81 vs. 39.57 °C for FH and SH, respectively). Furthermore, during the 5-min transition period between end exercise and start of recovery, a significantly greater residual increase in T_{re} (0.23 °C) was measured for the FH conditions relative to SH. As a result, despite similar core cooling rates, the time required to achieve the same end-point varied as a result of the different T_{re} at the start of recovery. In contrast, T_{es} showed a sustained and more pronounced decrease (0.53 and 0.26 °C for FH and SH, respectively) during this same period which likely explains why T_{es} demonstrated a similar response pattern between conditions. Combined, these observations suggest that differences in tissue blood flow associated with the very different metabolic demands and duration of each exercise condition resulted in marked differences in tissue heat distribution despite the similar levels of hyperthermia at end exercise. However, it is important to recognize that this may reflect the fact that our criterion measure for ending exercise was T_{re} which is known to respond slowly to changes in heat balance.

The practical applications of the current work are noteworthy. The risk of EHS is always present when laborers, athletes, and soldiers perform exercise in temperate or hot environments, particularly in cases where protective equipment is worn. While the intensity and duration that leads to the onset of EHS can vary significantly in these circumstances (Rohe 2012; Nelson et al. 2011; Brake and Bates 2002; Cuddy and Ruby 2011; Hunt et al. 2013), our

results demonstrate that the treatment should not depend on the type/length/intensity of the activity. Thus, the current paper is unique because it is the first to show that the path to hyperthermia may be different, but the path to recovery should not be. Indeed, as the magnitude of hyperthermia and the time spent at an elevated core temperature are directly related to the severity of heat-related injuries (Smith 2005), restoring core temperature to near-normal resting levels is vital (Armstrong et al. 2007; Flouris et al. 2014). Therefore, once EHS is recognized, aggressive whole-body cooling via IWI should begin immediately and on-site (Casa et al. 2010). If this is not feasible, then an alternative effective treatment (albeit to some degree less so) should be implemented immediately and continue until IWI treatment can begin. However, more than often the appropriate treatment is not applied or possible (i.e., remote location, individual exercise on their own, etc.), and individuals suffering from exertional heat illness are left to recuperate via natural recovery.

Based on the data presented in this study, we conclude that the intensity of physical work performed and, thus, the time taken to reach an elevated state of hyperthermia does not affect the effectiveness of either IWI treatment or natural recovery. Therefore, while the path to hyperthermia may be different for each EHS patient, the path to recovery must always be immediate IWI treatment.

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