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ORIGINAL ARTICLE

Heart rate variability during exertional heat stress: effects of heat production and treatment

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Abstract
Purpose We assessed the efficacy of different treatments (i.e., treatment with ice water immersion vs. natural recovery) and the effect of exercise intensities (i.e., low vs. high) for restoring heart rate variability (HRV) indices during recovery from exertional heat stress (EHS).
Methods Nine healthy adults (26 ± 3 years, 174.2 ± 3.8 cm, 74.6 ± 4.3 kg, 17.9 ± 2.8 % body fat, 57 ± 2 mL·kg·$^{-1}$ min^{-1} peak oxygen uptake) completed four EHS sessions incorporating either walking (4.0–4.5 km·h^{-1}, 2 % incline) or jogging (~7.0 km·h^{-1}, 2 % incline) on a treadmill in a hot-dry environment (40 °C, 20–30 % relative humidity) while wearing a non-permeable rain poncho for a slow or fast rate of rectal temperature (T_{re}) increase, respectively. Upon reaching a T_{re} of 39.5 °C, participants recovered until T_{re} returned to 38 °C either passively or with whole-body immersion in 2 °C water. A comprehensive panel of 93 HRV measures were computed from the time, frequency, time–frequency, scale-invariant, entropy and non-linear domains.
Results Exertional heat stress significantly affected 60/93 HRV measures analysed. Analyses during recovery demonstrated that there were no significant differences between HRV measures that had been influenced by EHS at the end of passive recovery vs. whole-body cooling treatment ($p > 0.05$). Nevertheless, the cooling treatment required statistically significantly less time to reduce T_{re} ($p < 0.001$).
Conclusions While EHS has a marked effect on autonomic nervous system modulation and whole-body immersion in 2 °C water results in faster cooling, there were no observed differences in restoration of autonomic heart rate modulation as measured by HRV indices with whole-body cold-water immersion compared to passive recovery in thermoneutral conditions.

Keywords Exercise-induced hyperthermia · EHS · Heart rate variability · HRV · Core temperature · Hyperthermia

Abbreviations

ANS	Autonomic nervous system
ECG	Electrocardiogram
EHS	Exertional heat stress
FH + C	Fast heating with whole-body cooling session
FH + N	Fast heating with natural recovery session
HRV	Heart rate variability
NN	Normal-to-normal heart rate variability
RMSSD	Root mean square of differences of successive normal-to-normal intervals
R–R interval	Time between two consecutive R waves in the electrocardiogram
SH + C	Slow heating with whole-body cooling session
SH + N	Slow heating with natural recovery session
T_{re}	Rectal temperature
$\dot{V}O_{2peak}$	Peak oxygen consumption

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Introduction

Exertional heat stroke is caused by an imbalance between heat production and loss during physical exercise in a hot environment leading to a core temperature >40 °C and is a serious medical condition with potentially tragic consequences (Casa et al. 2012; Armstrong et al. 2007). More than ten serious exertional heat stroke cases occur in major athletic events such as the Boston or the Athens marathon (Armstrong et al. 1994; Casa et al. 2005). Therefore, this condition represents an important aspect of acute and emergency care requiring extensive knowledge and planning to ensure optimal onsite treatment. Moreover, its increased prevalence during the past 8 years (Casa et al. 2012) highlights the need for further research into its causative agents and treatment.

Since the key diagnostic criterion of exertional heat stroke is a disturbance in central nervous system function [i.e., confusion, delirium, coma and convulsions (Casa et al. 2005)], it is crucial to investigate neurophysiological elements that can assess treatment efficacy. Heart rate variability (HRV) has been identified as a window over the autonomic nervous system (ANS), being affected by sympatho-vagal modulation, physiological oscillators function (e.g., vasomotor and respiratory centers) and humoral factors (Task Force of the European Society of Cardiology and the North American Society of Pacing and Electrophysiology 1996; Dinas et al. 2013). Because there is no clear mapping between HRV and physiological variables, several measures of variability have been developed, and categorized in different domains, such as time, frequency, time–frequency, scale-invariant, entropy and non-linear (Bravi et al. 2011). Nevertheless, the vast majority of HRV studies focus only on the time and frequency domains.

Surprisingly, the literature contains little information on the effects of exertional heat stroke on ANS functioning. Previous reports using exertional heat stress (EHS) as a model (which is not a heat illness per se, but can lead to a heat illness) showed that sympathetic activity was elevated during and following exercise in the heat, compared to the same exercise performed in a thermoneutral environment (Brenner et al. 1997; Leicht et al. 2009). Immersion in 14 °C water following supramaximal exercise has shown greater HRV and parasympathetic reactivation compared with a passive recovery in warm ambient conditions of 35 °C air temperature (Buchheit et al. 2009). Similarly, ice packs administered to the armpits, groins, and back of the neck, or industrial fan cooling with intermittent water spray resulted in higher parasympathetic reactivation following EHS compared to 4 °C saline infusion (Leicht et al. 2009). However, the available evidence for exertional heat stroke survival rates suggests that the best cooling strategy for this condition is ice water immersion (Casa et al. 2007). To our knowledge, the effect of ice water immersion on ANS functioning during EHS remains unknown.

A unique and currently unexplained feature of exertional heat stroke is the time required for its development, which may vary from 20 min to several hours (Casa and Armstrong 2003). Indeed, the rate at which core temperature increases can range extensively during sporting and occupational heat stress depending on factors such as exercise intensity, ambient conditions, and clothing. As the development of exertional heat stroke can occur slowly or quickly, it is critical to understand whether different rates of EHS development can affect ANS function during treatment. Our purpose was to compare the efficacy of different treatments (i.e., treatment with ice water immersion vs. natural recovery) and the effect of exercise intensities (i.e., low vs. high) for restoring ANS function during recovery from EHS. Based on the evidence for exertional heat stroke survival rates suggesting that cold-water immersion is the best cooling strategy for this condition (Casa et al. 2007), as well as the superior capacity of water for heat transfer compared to air (Proulx et al. 2003), we hypothesized that treatment with ice water immersion would result in a similar, yet more rapid, restoration of ANS function compared to natural recovery.

Materials and methods

Participants

The study received approval from the University of Ottawa Health Sciences and Science Research Ethics Board. Following appropriate determination of sample size (see "Statistical analysis") nine healthy [no history of respiratory, metabolic, or cardiovascular conditions; 26 ± 3 years (range 18–45 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, 57 ± 2 $mL \cdot kg^{-1} \cdot min^{-1}$ peak oxygen consumption ($\dot{V}O_{2peak}$); mean ± SD] and physically active individuals from the university population and the general community were informed of all experimental procedures, associated risks, and discomforts, and provided written consent.

Experimental design

During a preliminary session, participants received an orientation to the instrumentation and experimental protocols, 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 to ensure their safety for participation, and performed body composition and fitness assessments. Body

density was measured using hydrostatic weighing and percentage of body fat was estimated via the Siri equation (Siri 1956). The $\dot{V}O_{2peak}$ was assessed using a treadmill protocol and open-circuit indirect calorimetry. Participants began running at a self-selected pace where the treadmill incline was increased 1 % each minute to a maximum of 10 %, after which, speed and incline were increased alternatively each minute by 0.8 km·h^{-1} and 1 % incline, respectively, until the subject could no longer continue. The $\dot{V}O_{2peak}$ was defined as the highest oxygen uptake during the test, taken as the mean of the three highest consecutive 15-s recordings.

Following the preliminary session, all participants completed four EHS sessions separated by ≥5 days. 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 sat upright rested for 30 min of baseline measurements. 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, 20–30 % 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 (to avoid peaking at a high T_{re} during the passive recovery condition), participants removed only the rain poncho and recovered either naturally (passive recovery upright seated in a ~29 °C ambient environment) or with whole-body cooling treatment (2 °C cold-water immersion, upright seated with legs straight in front) until T_{re} returned to 38 °C (to avoid overcooling). Thus, the four EHS sessions, which were performed in a counterbalanced manner, included fast heating with whole-body cooling (FH + C), fast heating with natural recovery (FH + N), slow heating with whole-body cooling (SH + C), and slow heating with natural recovery (SH + N).

Physiological measurements

Core temperature

A pediatric thermocouple probe (Mon-a-therm Nasopharyngeal Temperature Probe, Mallinckrodt Medical, St. Louis, USA) inserted approximately 15 cm beyond the anal sphincter was used to measure T_{re} continuously.

Heart rate variability

R–R interval data were extracted from 175 Hz ECG waveforms throughout all EHS sessions using a 5-lead ECG Zymed Holter system (Philips DigiTrack Plus Recorder, Andover, USA) and those deemed to be normal-to-normal (NN) were retained for further analysis. Using the NN interval time series of each subject (total of nine NN interval series; 2-min windowed analysis with 30 s time step), 93 measures of HRV analyses were performed employing the continuous individualized variability analysis—CIMVA™ software (http://ohridal.org/cimva/CIMVA-Core-Description.pdf) to extract a total of 93 measures of variability computed from the time, frequency, time–frequency, scale-invariant, entropy and non-linear domains.

Statistical analysis

The minimum required sample size was determined using data from a recent study (Leicht et al. 2009), where the high-frequency index of HRV was measured during exercise in the heat (until core temperature reached 40 °C) and a subsequent recovery using different cooling treatments: fridge-cold saline infusion (2 L of 0.9 % sodium chloride solution), ice packs administered to the armpits, groins and back of the neck, and industrial fan cooling with intermittent water spray. Sample size calculations were conducted using G*Power 3.0 (Faul et al. 2007). The ARE 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 was determined by calculating the effect size *d*. Using the aforementioned published data (Leicht et al. 2009), the resulting minimum required sample size was seven participants. In order to confidently detect a reasonable departure from the null hypothesis, we recruited a total of nine participants.

As some of the HRV data were not normally distributed, non-parametric statistics were used for the HRV analyses. Considering the four EHS sessions, the following distributions were computed for each of the 93 variability time series: variability at baseline (10 min), variability during the last 10 min of exercise, and variability during the last 3 min of recovery. Each sample in the distribution represented the median value of a given measure for one subject in the specified time frame. The Friedman test was used to assess differences in the medians of the three distributions. As the test was repeated for all 93 measures of variability, the robust false discovery rate method (Pounds and Cheng 2006) was used to adjust for multiple comparisons, by selecting the appropriate threshold to reject the null hypothesis of equal medians. A rate of 0.01 false positives was imposed. Two-tailed Wilcoxon signed-rank tests were used for post hoc analysis to compare recovery HRV within the same EHS session (i.e., FH + N vs. FH + C and

SH + N vs. SH + C). These tests were repeated to compare recovery HRV between exercise intensities (i.e., FH + N vs. SH + N and SH + C vs. FH + C). Finally, factorial analysis of variance was used to compare the duration of each recovery across the two exercise protocols with a level of significance set at $p \leq 0.05$.

Results

The data analysis included all 93 indices of HRV extracted. Given the inability to graphically illustrate the results for all 93 indices extracted, selected indices covering most HRV domains are presented in Fig. 1 (mean RR: statistical domain; standard deviation of RR: statistical domain; low/high-frequency ratio Lomb–Scargle: energetic domain) and Fig. 2 [sample entropy: informational domain; Hurst exponent: invariant domain; root mean square of differences of successive NN intervals (RMSSD): statistical domain]. Exercise and heat stress resulted in reduced HRV, standard deviation of HRV, and low/high-frequency ratio (derived from the Lomb–Scargle periodogram) (Fig. 1). Similar changes were observed in Hurst exponent, sample entropy, and RMSSD (Fig. 2), suggesting significant vagal withdrawal.

Differences between the three distributions and the results from Friedman's test appear in Table 1. As we only considered NN intervals, we were unable to compute the analysis for the FH + N and the FH + C sessions due to a very large number of abnormal beats with this higher intensity exercise. The results for the SH + N and the SH + C data demonstrated that HRV was markedly affected by exercise, EHS, and the EHS treatments as indicated by the large number of HRV measures that detected a change from

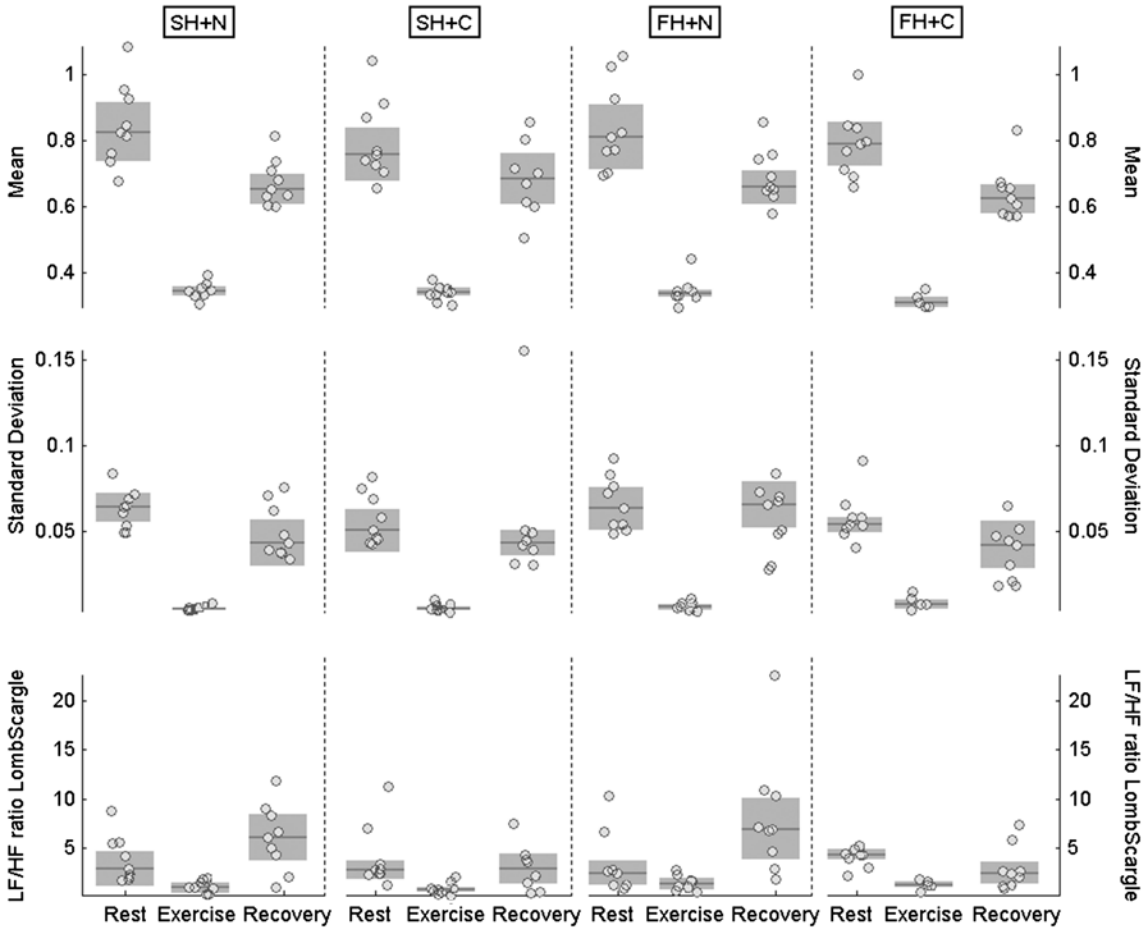

Fig. 1 Results for mean RR, standard deviation of RR, and low/high-frequency ratio Lomb–Scargle extracted from the R–R interval time series of the subjects during each phase (rest, exercise and recovery) in each of the four exertional heat stress sessions (SH + N: slow heating with natural recovery, SH + C: slow heating with whole-body cooling, FH + N: fast heating with natural recovery, FH + C: fast heating with whole-body cooling). For each heart rate variability marker, the *gray line* represents the mean value, while the *shaded area* represents the 95 % confidence interval. As shown, exercise and heat stress resulted in reduced HRV, standard deviation of HRV, and low/high-frequency ratio, indicating significant vagal withdrawal

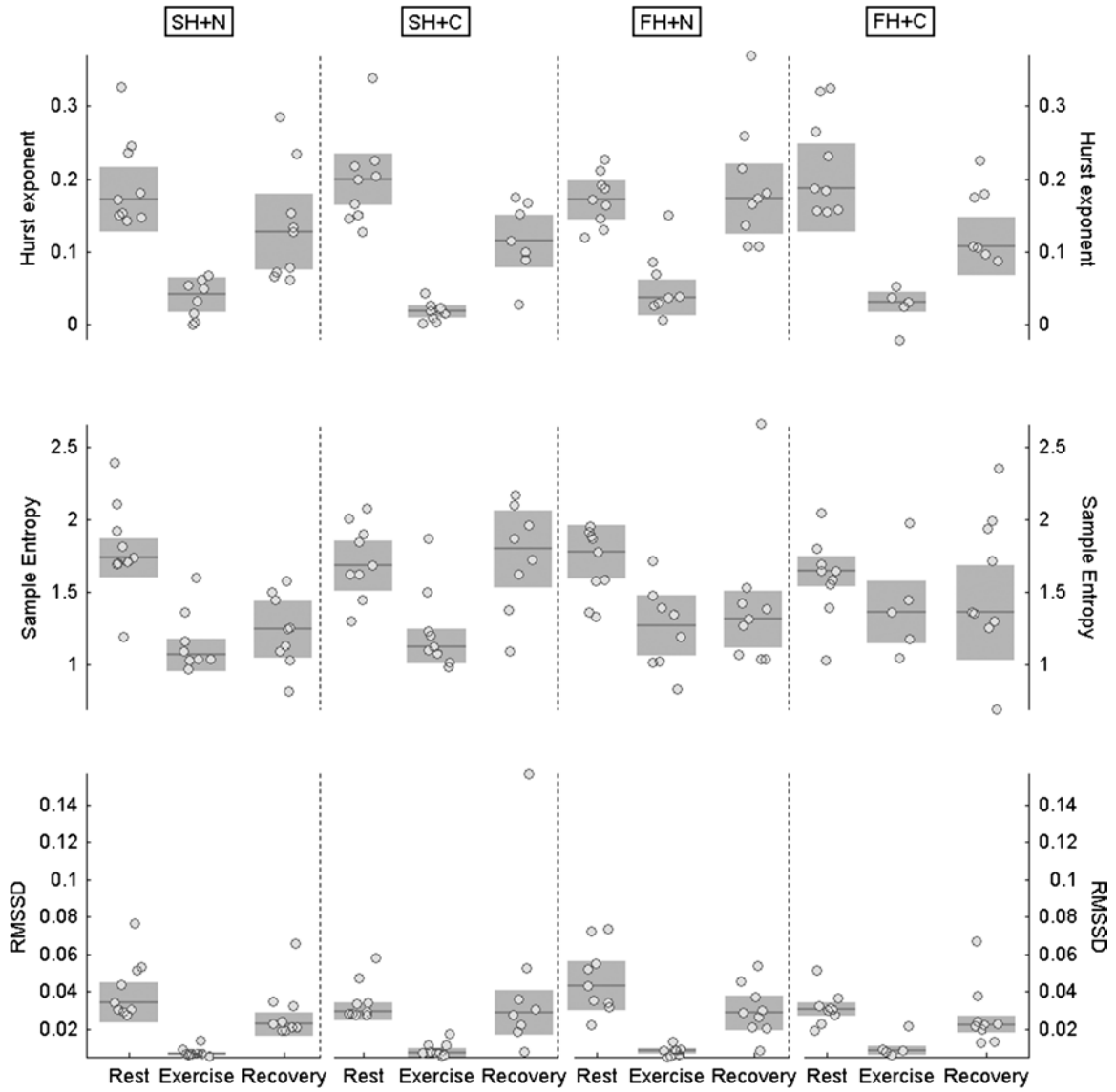

Fig. 2 Results for sample entropy, Hurst exponent (computed through the scaled windowed variance method), and root mean square of differences of successive NN intervals (RMSSD) extracted from the R–R interval time series of the subjects during each phase (rest, exercise and recovery) in each of the four exertional heat stress sessions (SH + N: slow heating with natural recovery, SH + C: slow heating with whole-body cooling, FH + N: fast heating with natural recovery, FH + C: fast heating with whole-body cooling). For each heart rate variability marker, the *gray line* represents the mean value, while the *shaded area* represents the 95 % confidence interval. As shown, exercise and heat stress resulted in reduced Hurst exponent, sample entropy, and root mean square of differences of successive NN intervals, indicating significant vagal withdrawal

baseline during exercise and recovery (Table 1; Figs. 1, 2), indicating a significant vagal withdrawal. Using only the HRV measures that were affected by heat stress, post hoc analysis Wilcoxon signed-rank tests compared recovery HRV within the same exercise intensity (i.e., FH + N vs. FH + C; SH + N vs. SH + C; Table 1). Results demonstrated that all the HRV measures that had been influenced by heat stress were similar at the end of recovery for both the FH and the SH sessions ($p > 0.05$). The analyses were repeated with different time intervals (larger than 10 min for the exercise phase, and 3 min for the recovery phase), however all measures were similar at the end of recovery ($p > 0.05$). Furthermore, these tests were repeated to compare recovery HRV between exercise intensities. The results demonstrated that exercise intensity did not affect HRV at the end of recovery [FH + C vs. SH + C: 0.57 ± 0.43; FH + N vs. SH + N: 0.60 ± 0.36 (median p value ± interquartile range)].

To reduce T_{re} to 38 °C, the EHS treatments (i.e., cold-water immersion treatment and natural recovery) were applied for

Table 1 Results of the Friedman's test assessing the differences between the three distributions and the post hoc Wilcoxon signed-rank tests comparing treatments at the EHS recovery stage

	Friedman's test		Wilcoxon test	
	SH + N dataset	SH + C dataset	SH + N vs. SH + C	FH + N vs. FH + C
Number of measures rejecting the null hypothesis of equal median	68/93	60/93	0	0
p values of the measures rejecting the null hypothesis (median ± interquartile range)	$0.15 \times 10^{-3} \pm 0.34 \times 10^{-3}$	$0.25 \times 10^{-3} \pm 0.14 \times 10^{-2}$	0.45 ± 0.29	0.35 ± 0.24
Alpha value for rejection	0.03	0.04	0	0

As we only considered NN intervals, we were unable to compute the analysis for the FH + N and the FH + C sessions due to a very large number of abnormal beats with this higher intensity exercise

EHS: exertional heat stress, SH + N: slow heating with natural recovery, SH + C: slow heating with whole-body cooling, FH + N: fast heating with natural recovery, FH + C: fast heating with whole-body cooling

Table 2 Rectal temperature at resting baseline and the start of recovery, and exercise and recovery times, for the four conditions

Condition	SH + N	SH + C	FH + N	FH + C
Resting T_{re} (°C)	36.90 ± 0.36	36.93 ± 0.23	36.95 ± 0.36	37.04 ± 0.23
Time to T_{re} 39.5 °C (min)	76.20 ± 15.30*	86.10 ± 20.30*	34.80 ± 7.90	39.80 ± 12.30
T_{re} start recovery (°C)	39.57 ± 0.13	39.57 ± 0.14	39.81 ± 0.25	39.72 ± 0.22
Time to T_{re} 38.0 °C (min)	53.80 ± 15.90*†	6.60 ± 2.10	76.10 ± 24.00†	11.10 ± 7.20

T_{re}: rectal temperature, SH + N: slow heating with natural recovery, SH + C: slow heating with whole-body cooling, FH + N: fast heating with natural recovery, FH + C: fast heating with whole-body cooling

* Significantly ($p < 0.05$) different from the FH session incorporating similar treatment at the recovery phase

† Significantly ($p < 0.05$) increased compared to cooling treatment sessions

76.1 ± 24.0, 53.8 ± 15.9, 11.1 ± 7.2, and 6.6 ± 2.1 min during FH + N, SH + N, FH + C, and SH + C, respectively (Table 2). Following a grouping in terms of EHS treatment (i.e., FH + N with SH + N; FH + C with SH + C), our results show that the cold-water immersion treatment was applied for 8.8 ± 5.7 min, while the natural recovery treatment was applied for 64.9 ± 22.9 min ($p < 0.001$). Factorial analysis of variance demonstrated statistically significant main effects of exercise intensity (i.e., FH + N and FH + C were different from SH + N and SH + C; $p = 0.01$) and recovery type (i.e., FH + N and SH + N were different from FH + C and SH + C; $p < 0.001$).

Discussion

This is the first study to show that the HRV indices affected by EHS can be restored with whole-body immersion in 2 °C water as effectively as with natural recovery, confirming our initial hypothesis. It is important to note that the present HRV analysis went beyond the typical time and frequency domain measures (Dinas et al. 2013), assessing for the first time a comprehensive panel of variability markers in the EHS literature.

Heat exposure is known to reduce parasympathetic activity, while exercise alone causes a progressive decrease in parasympathetic tone which tends to become evident at exercise intensities >50 % of $\dot{V}O_{2peak}$ (Brenner et al. 1997, 1998; Carrillo et al. 2013). When combined, exercise and heat stress result in even greater vagal withdrawal (Brenner et al. 1997; Leicht et al. 2009). Given that the exercise intensity in our study was relatively low, it seems reasonable to suggest that the marked reduction in parasympathetic tone observed was induced by EHS. Indeed, we found that EHS has a significant effect on ANS modulation as assessed by HRV. The data obtained during the low intensity exercise suggest that EHS resulted in reduced HRV, standard deviation of HRV, low/high-frequency ratio, Hurst exponent, sample entropy, and root mean square of differences of successive NN intervals indicating a marked ANS disturbance characterized by sympathetic dominance during EHS. Following the end of exercise, we found that all the variability measures that had been influenced by EHS were similar at the end of recovery during both natural recovery and whole-body immersion in 2 °C water. The changes observed at the end of the recovery in low/high-frequency ratio and root mean square of differences of successive NN intervals suggest that the HRV restoration was

mainly caused by vagal restoration. This finding is supported by a recent study showing that immersion in 14 °C water following supramaximal exercise generated greater HRV and parasympathetic nervous system reactivation compared with a passive recovery in 35 °C (Buchheit et al. 2009). Furthermore, we found that the above results were evident at the end of recovery in both exercise intensities.

Although the natural recovery and the ice water immersion treatments demonstrated similar capacities for restoring ANS function following EHS, ice water immersion was able to rapidly restore T_{re} requiring only ~13 % of the time needed by natural recovery. This confirmed our hypothesis that treatment with ice water immersion would result in a faster restoration of ANS function compared to natural recovery, and represents a critical advantage since the most important determinant of exertional heat stroke outcome is the time required to reduce core temperature to near-normal levels (Casa et al. 2012). The primary goal of exertional heat stroke treatment is to lower core temperature below 40 °C within 30 min from collapse (Armstrong et al. 2007; Casa et al. 2012), in line with survival rate data suggesting that the best cooling strategy for exertional heat stroke is ice water immersion (Casa et al. 2007, 2012). In this light, our results provide support to the notion that ice water immersion is the most appropriate treatment for exertional heat stroke as it is capable of quickly reversing hyperthermia without any adverse effects on ANS function. The latter includes intense shivering which was not observed during our data collection, probably due to the short duration of the water immersion. Indeed, a previous study that immersed hyperthermic individuals into 2 °C water for the same time reported no shivering (Proulx et al. 2003).

Unfortunately, we were unable to compare autonomic nervous system function between the two rates of EHS development at the end of exercise. This was due to the large number of abnormal beats recorded during exercise in the high intensity conditions and did not allow us to investigate whether different rates of EHS development can affect ANS function during treatment. On the other hand, indices of HRV (particularly high-frequency power) are considerably influenced by breathing patterns (Dinas et al. 2013; Flouris and Cheung 2009). Given the severity of stress involved in our EHS sessions, we did not control the frequency of breathing and tidal volume. It is, therefore, possible that differences between the EHS sessions and/or treatments may have affected respiratory patterns to some degree and, in turn, the obtained HRV frequency domain indices. However, we believe that our findings are robust since they are based on a thorough analysis of 93 variability measures computed from the time, frequency, time–frequency, scale-invariant, entropy and non-linear domains.

We used absolute exercise intensities to generate a slow (4.0–4.5 km·h^{-1}, 2 % incline) and fast (~7.0 km·h^{-1}, 2 % incline) rate of T_{re} increase. Although our sample was relatively homogeneous in terms of fitness levels (57 ± 2 mL·kg^{-1}·min^{-1} $\dot{V}O_{2peak}$), using an absolute exercise intensity may have impacted our results since training can influence the ability to tolerate (or not) heat stress. The T_{re} endpoints in our study were chosen to ensure the participants' well-being. The 39.5 °C endpoint was chosen because T_{re} continues to increase after the end of exercise if a cooling treatment is not applied (Flouris and Cheung 2006, 2010). Similarly, during the cold-water immersion, an endpoint T_{re} of 38 °C was utilized for all participants to avoid the potential of overcooling during FH + C and SH + C. A previous study that immersed hyperthermic individuals into 2 °C water for the same time reported a significant postcooling afterdrop with a T_{re} of 35.7 °C (Proulx et al. 2003). Thus we used a 38 °C endpoint to maximize the time period in which ANS function could be examined while, at the same time, ensure the participants' well-being.

It is concluded that EHS has a significant effect on ANS modulation. Treating EHS with whole-body immersion in 2 °C water resulted in faster cooling, yet similar restoration of ANS function, as compared to passive recovery in thermoneutral conditions. Therefore, our results are compatible with the conclusion that ice water immersion is the most appropriate treatment for exertional heat stroke as it is capable of quickly reversing the hyperthermic state of the individual without any adverse effects on ANS function.

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Conflict of interest Andrew J. E. Seely is the founder and Chief Science Officer, and Geoffrey Green is the Product Manager of Therapeutic Monitoring Systems (TMS). TMS aims to commercialize patented continuous individualized multi-organ variability analysis (CIMVA) technology, with the objective of delivering variability-directed clinical decision support to improve quality and efficiency of care. All the other authors have no conflicts of interest to disclose.

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