Effect of cold or thermoneutral water immersion on post-exercise heart rate recovery and heart rate variability indices

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ABSTRACT

This study aimed to investigate the effect of cold and thermoneutral water immersion on post-exercise parasympathetic reactivation, inferred from heart rate (HR) recovery (HRR) and HR variability (HRV) indices. Twelve men performed, on three separate occasions, an intermittent exercise bout (all-out 30-s Wingate test, 5 min seated recovery, followed by 5 min of submaximal running exercise), randomly followed by 5 min of passive (seated) recovery under either cold (CWI), thermoneutral water immersion (TWI) or control (CON) conditions. HRR indices (e.g., heart beats recovered in the first minute after exercise cessation, HRR60s) and vagal-related HRV indices (i.e., natural logarithm of the square root of the mean of the sum of the squares of differences between adjacent normal R-R intervals (Ln rMSSD)) were calculated for the three recovery conditions. HRR60s was faster in water immersion compared with CON conditions [30 ± 9 beats min\(^{-1}\) for CON vs. 43 ± 10 beats min\(^{-1}\) for TWI (P = 0.003) and 40 ± 13 beats min\(^{-1}\) for CWI (P = 0.017)], while no difference was found between CWI and TWI (P = 0.763). Ln rMSSD was higher in CWI (2.32 ± 0.67 ms) compared with CON (1.98 ± 0.74 ms, P = 0.05) and TWI (2.01 ± 0.61 ms, P = 0.08; aE = 1.07) conditions, with no difference between CON and TWI (P = 0.964). Water immersion is a simple and efficient means of immediately triggering post-exercise parasympathetic activity, with colder immersion temperatures likely to be more effective at increasing parasympathetic activity.

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1. Introduction

Water immersion has been shown to be a simple and efficient method for increasing parasympathetic activity and lowering sympathetic tone at rest, as inferred from heart rate (HR) variability (HRV) measures (Miyamoto et al., 2006; Mourot et al., 2008). The hydrostatic pressure created by the head-out water immersion condition shifts peripheral blood into the thoracic vasculature (Wilcock et al., 2006), thereby increasing central blood volume, stroke volume, cardiac output and central venous pressure (Park et al., 1999). This increase in central venous pressure likely stimulates arterial high pressure and cardiopulmonary low pressure baroreflexes (Gabrielsen et al., 1996, Pump et al., 2001), which may augment parasympathetic activity and inhibit sympathetic activity (Pump et al., 2001), leading to a bradycardia (Pump et al., 2001) and an increase in vagal-related HRV indexes (Spinelli et al., 1999), Mourot et al. (2008) also showed that moderately cold (26–27 °C) exposure had a greater effect on cardiac parasympathetic activity compared with a thermoneutral immersion temperature (35–36 °C). This may be due to an arterial vasoconstriction that can induce greater increases in central blood volume or faster reductions in core temperature (Buchheit and Laursen, 2009).

Nevertheless, while the beneficial impact of hydrostatic pressure and/or moderately cold water exposure on parasympathetic activity has been well demonstrated at rest, little is known about its effect during the post-exercise period. Furthermore, the optimal water temperature likely to be most effective at accelerating post-exercise parasympathetic reactivation is also unknown. For example, 5 min of cold water immersion (14 °C) applied following supramaximal exercise in the heat induced faster and greater post-exercise parasympathetic reactivation compared with a non-immersed condition (Buchheit et al., 2009d). However, these authors did not measure the effect of other water immersion temperatures. While sympathovagal balance with water immersion is simply shifted towards parasympathetic dominance at rest, the autonomic background during the post-exercise period is more complex, with a progressive parasympathetic reactivation concomitant with a sympathetic withdrawal generally observed (Savin et al., 1982). Therefore, the effect of immersion-induced
parasympathetic activation on cardiac function is difficult to predict due to the possible occurrence of sympathovagal interactions during the post-exercise period (i.e., sympathovagal antagonism). For instance, the combination of exercise- and immersion-related increases in sympathetic and parasympathetic activity, respectively, may lead to either higher or lower vagal effects on HR, depending on the site and type of activated adrenergic receptors (i.e., cholinergic stimulation of the heart can be mediated by adrenergic mechanisms) (Levy, 1971, Tulppo et al., 1998, Miyamoto et al., 2003, Miyamoto et al., 2004).

In order to assess the most efficient post-exercise immersion temperatures to accelerate post-exercise cardiac autonomic function, we aimed to compare the effect of two commonly used temperatures in the field (i.e., cold and thermoneutral water immersions (Wilcock et al., 2006)) on HR recovery (HRR) and HRV indices. We first hypothesized that, because of changes in hydrostatic pressure, water immersion itself, irrespective of water temperature, would induce a faster and greater parasympathetic reactivation compared with the out-of-water control condition. However, since a prevailing high level of sympathetic activity from exercise might either lower or increase the vagal influence on HR (Levy, 1971, Miyamoto et al., 2003), the isolated effects of water temperature on post-exercise autonomic activity was difficult to predict.

2. Methods

2.1. Participants

Based on the assumption that a 0.49 ± 0.17 ms difference in post-exercise natural logarithm square root of the mean of the sum of the squares of differences between adjacent normal R–R intervals (Ln rMSSD) (Al Haddad et al., 2010) and an 8 ± 5 beats min⁻¹ difference in HRRR54 (i.e., absolute difference between the final HR at exercise end and the HR recorded 60 s later) was meaningful (Buchheit et al., 2007a), we used sigmaStat 3.11 Software (Systat software inc., San Jose, Calif., USA) to determine that a sample size of at least eight participants would provide a statistical power of 0.8 at an alpha level of 0.05. To further increase the statistical power of the study, we eventually recruited 12 healthy males (21.6 ± 1.4 yr; 1.80 ± 0.05 m; 76.5 ± 12.2 Kg, training 2 to 5 h per week), who volunteered for the study after providing written informed consent. Participants were all familiar with exercise testing, not taking prescribed medications and presented with normal levels of blood pressure and electrocardiographic patterns. The study conformed to ethical guidelines outlined in the Declaration of Helsinki and was approved by the local human research ethics committee.

2.2. Exercise testing

Participants were tested on four different occasions over 4 weeks. All tests were performed on an indoor synthetic track where ambient temperature ranged from 20 to 23 °C. Participants first performed an incremental test to determine a reference speed (Vegt) for the submaximal exercise bout. During the 3 consecutive weeks, they performed an exercise sequence, which was randomly followed by either 5 min seated recovery with cold water immersion (CWI), 5 min seated recovery with thermoneutral water immersion (TWI) or 5 min seated recovery without immersion (CON). HR recordings were carried out 5 min before and throughout the exercise sequence and recovery periods. To minimize circadian effects, tests were conducted at the same day of the week (i.e., Friday) and at the same time of day (10 h ± 1 h). All participants were asked to consume their last (usual) meal at least 3 h before each test session.

2.3. Maximal graded test

Participants performed, as previously described, the 30–15 intermittent fitness test [30–15IFT (Buchheit et al., 2009b)] (i.e., 30-s of running interspersed with 15-s of passive recovery). This test is a graded intermittent shuttle field test and was used to determine the running velocity of the submaximal exercise (the maximal speed reached at the end of the test being labeled Vegt).

2.4. Exercise sequence

Subsequent to a short warm-up period, participants performed a Wingate test (i.e., all-out 30-s cycling), followed after 5 min of passive seated recovery, by a 5 min continuous submaximal running exercise at 45% of Vegt (Al Haddad et al., 2010). The exercise sequence was followed by 5 min of passive seated recovery in either CWI, TWI or CON conditions. The Wingate test was performed on a Monark cycle ergometer (Monark 884E). Prior to the test, seat height was adjusted to accommodate the subject’s stature. Subjects performed a 2-min warm-up pedaling at a cadence of 80 rpm at a constant power output set at 50 W. The warm-up included two brief (2–3 s) sprinting bouts. Immediately after the warming-up period, the subjects performed the 30-s Wingate test. Standard resistance applied corresponded to 7.5% of the participant’s body mass (Inbar et al., 1996). Participants were verbally encouraged to perform maximally throughout the 30-s test. Running pace of the submaximal run was governed by a prerecorded beep that sounded at appropriate intervals in order to allow participants to adjust their running speed as they passed through specific zones of the field. The Wingate test was aimed at markedly increasing sympathetic and reducing vagal activity (Goulopoulou et al., 2006), while the intensity of the following submaximal run was chosen to ensure a rapid return of HR to baseline levels following exercise (Buchheit et al., 2009a), as required for appropriate short-term HRV analysis (i.e., steady-state signal). Finally, the association of the two tests enabled us to assess the confounding effect of sympathetic overactivity (i.e., interaction; (Levy, 1971)) on the post-exercise parasympathetic reactivation response to the three recovery conditions, whilst also eliminating the potential confounding mechanical effects of high minute ventilation on vagal-related HRV indices (Blin et al., 2005).

2.5. Water immersion and control recovery conditions

Immediately at the end of the submaximal exercise, subjects recovered passively in the seated position for 5 min in either CWI, TWI or CON conditions. In water immersion conditions, two water tanks were filled with temperature-adjusted water from a gas water-heater unit for TWI or a refrigeration unit for CWI. The water was then circulated continuously to maintain a uniform temperature, as verified with a thermometer (Neptun, TFA, Wertheim-Reicholzheim, Germany). Participants were submerged to the midternal level wearing only their running shorts. Water temperature were chosen to mimic post-exercise recovery strategies implemented in field, and was therefore kept at 14–15 °C for CWI and 33–34 °C for TWI (Wilcock et al., 2006). The 5-min CWI temperature and duration are effective at lowering body temperature while remaining tolerable (Peiffer et al., 2009). This temperature was also reported to be efficient to increase parasympathetic activity and to be a safe recovery strategy when used following exercise in the heat (Buchheit et al., 2009d).

2.6. HR measurements

HR was continuously recorded (s810 heart rate monitor, Polar Electro, Kempele, Finland) during the exercise sequence and the subsequent recovery phase. Recorded data were downloaded on a computer using compatible Polar software (Polar Precision Performance SW 5.20, Polar Electro, Kempele, Finland). All irregular heart beats were automatically identified and replaced with interpolated adjacent R–R interval values with the Polar Software (Nunan et al., 2009). Please cite this article as: Al Haddad, H., et al., Effect of cold or thermoneutral water immersion on post-exercise heart rate recovery and heart rate variability indices, Auton. Neurosci. (2010), doi:10.1016/j.autneu.2010.03.017
2.7. Post-exercise HRR assessment

HRR was calculated by fitting the 5-min post-exercise R–R data into a first-order exponential decay curve (Buchheit et al., 2007b) which is thought to be more related to the progressive withdrawal of sympathetic activity (Perini et al., 1989). An HR time constant (HRτr) was then produced by modeling the resultant first 5 min of HR data using an iterative technique (SigmaPlot 10; SPSS Science, Chicago, IL) according to the following equation: HR = HR0 + HRamp e^(-t/HRτr), where HR0 is resting (final) HR, HRamp is maximal HR (HRmax) − HR0, and T is time (s). HRR was also assessed by calculating the absolute difference between the final HR at exercise end and the HR recorded 60 s later (HR60s) which is more representative of the immediate post-exercise parasympathetic reactivation (Buchheit et al., 2007b).

2.8. Short-term resting HRV analysis

HRV indices included the mean R–R intervals (mRR), the square root of the mean of the sum of the squares of differences between adjacent normal R–R intervals (rMSSD) (i.e., estimates the parasympathetic activity) (Task Force, 1996) as well as instantaneous beat-to-beat variability (SD1) and continuous beat-to-beat variability (SD2) ratio (SD1/SD2) to assess the autonomic interaction (i.e., SD1 and SD2 are derived from the Poincaré Plot analysis) (Tulppo et al., 1996). HRV indices were calculated from the last three stationary minutes of the 5 min recovery period using the accompanying Polar software (Polar Precision Performance SW 5.20, Polar Electro, Kempele, Finland), which has been shown to provide accurate measurements of short term HRV analysis (Nunan et al., 2009). Given the correlations between the power spectral density in high frequencies and rMSSD values (Task Force, 1996), data were restricted to time domain analysis. Although respiratory rate is often controlled in HRV studies, we chose not to control respiratory rate in our participants because we did not want to perturb the natural return of HR to baseline levels; moreover vagal-related HRV indices during spontaneous or controlled breathing differ perturb the natural return of HR to baseline levels; moreover vagal-related HRV indices during spontaneous or controlled breathing differ little (Bloomfield et al., 2001). The possible occurrence of a sympathovagal interaction (Levy, 1971, Tulppo et al., 1998, Miyamoto et al., 2003, Miyamoto et al., 2004) was visually assessed through the inspection of the shape of the Poincaré plot (Tulppo et al., 1998). Tulppo et al. (1998) reported that changes in HR dynamics could be accurately described by visual inspection of Poincaré plots, with visual interpretation being more reliable than numerical methods in revealing the atypical HR behavior during autonomic interaction. Nevertheless, (SD1/SD2) was also calculated in order to support our visual interpretation, since SD1/SD2 is associated with a normal comet-shaped plot when >0.15 and with a torpedo-shaped plot when SD1/SD2 < 0.15 (Tulppo et al., 1998). The Poincaré plot was performed by plotting the R–R intervals as a function of the previous one.

2.9. Time-varying vagal-related HRV index

A time-varying vagal-related index, the square root of the mean of the sum of the squares of differences between adjacent normal R–R intervals (rMSSD), was calculated for each of the 30-s segments of recovery (rMSSD30s) for the three 5-min recovery conditions (Goldberger et al., 2006). Data were median-filtered in order to smooth out transient outliers in the HRV plots (HRV vs. time in recovery). The first and last values were not median-filtered (Goldberger et al., 2006).

2.10. Statistical analyses

The distribution of each variable was examined with the Shapiro-Wilk normality test. Homogeneity of variance was verified by a Levene test. As data were skewed, natural logarithmic transform (ln) was applied for rMSSD (ln rMSSD) and rMSSD30s (ln rMSSD30s) to obtain a normal distribution. Reliability of the Wingate power output and the mean HR during the submaximal exercise was assessed via 1) a one-way ANOVA for repeated measures, with a ‘between-recovery condition’ factor, 2) the intraclass correlation coefficient (ICC) and 3) the typical error of measurement; TE, expressed as a coefficient variation; (CV%). Differences between-recovery methods (i.e., CWI, TWI and CON) for HRR and HRV indices were compared using a one-way ANOVA for repeated measures and Tukey’s post hoc analyses. Time-varying rMSSD30s was analyzed using a two-way ANOVA for repeated measures, with one within-time (‘time’, i.e., ten consecutive measurements during recovery) and one between-recovery condition (‘condition’, i.e., CWI, TWI and CON). When a significant interaction was noted, a Bonferroni’s post hoc test was conducted to delineate the main effects and/or interactions of the recovery condition and time during recovery. These analyses were performed using SigmaStat software (SigmaStat 3.1, Systat software inc., San Jose, Calif., USA). Significance level was set at P ≤ 0.05. When no significant effects were observed, but a tendency towards significance (P < 0.1) was apparent, then adjusted effect sizes (d) were calculated (Cohen, 1988). The scale proposed by Cohen (1988) was used for interpretation. The magnitude of the difference was considered either small (d ≤ 0.2), moderate (>0.5), or large (>0.8). Data in text are presented as means ± SD.

3. Results

3.1. Maximal graded test and reliability of Wingate test and submaximal exercise

Mean V̇O2 was 19.0 ± 1.5 km·h⁻¹. Mean maximal power output for the Wingate test (743 ± 134, 772 ± 128 and 765 ± 120 W for CON, CWI and TWI, respectively, P = 0.12) and mean HR for the submaximal exercise (160 ± 12, 163 ± 13 and 160 ± 13 beats min⁻¹ for CON, CWI and TWI, respectively, P = 0.43) were similar for the three recovery conditions. ICC and TE (CV %) were 0.96 and 24 W (3.2%) for the Wingate and 0.99 and 1.1 beats min⁻¹ (0.7%) for mean submaximal exercises’ HR.

3.2. Post-exercise HRR indices

HRR indices for the three recovery conditions are reported in Table 1. Compared with CON, HRR60s was greater with CWI (P = 0.017) and with TWI (P = 0.003). When compared with CON, HRRτr was not different with CWI (P = 0.695) but was shorter in TWI (P = 0.025). No difference was found between both water immersion methods for HRR60s (P = 0.763),

<table>
<thead>
<tr>
<th>Table 1</th>
<th>Heart rate variability (HRV) and heart rate recovery (HRR) indices following the submaximal exercise bout in control, thermoneutral and cold water conditions.</th>
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<tr>
<td></td>
<td>CON</td>
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<tr>
<td>HRR indices</td>
<td></td>
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<tr>
<td>HRR60s (beats min⁻¹)</td>
<td>30 ± 9</td>
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<tr>
<td>HRRτr (s)</td>
<td>51 ± 17</td>
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<tr>
<td>HRV indices</td>
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<tr>
<td>mRR (ms)</td>
<td>549.91 ± 60.62</td>
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<tr>
<td>Ln rMSSD (ms)</td>
<td>1.98 ± 0.74</td>
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<td>SD1/SD2</td>
<td>0.17 ± 0.07</td>
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Mean ± SD of absolute difference between the final heart rate at exercise end and the heart rate recorded 60 s later (HRR60s), time constant of heart rate decay (HRτr), mean R–R interval (mRR), natural logarithm of the square root of the mean of the sum of the squares of differences between adjacent normal R–R intervals (ln rMSSD) and the ratio between instantaneous beat-to-beat variability (SD1) and continuous beat-to-beat variability (SD2) (SD1/SD2). HRR was analyzed during the last 3 min of the 5 min recovery in control (CON), thermoneutral (TWI) and cold water immersion (CWI) conditions.

a P < 0.05 vs. CON.
b Difference vs. TWI with adjusted effect size considered as moderate (>0.5).
c Difference vs. CON with adjusted effect size considered as moderate (>0.5).
but a tendency towards a shorter HRRt was shown for TWI compared with CWI ($P=0.08$, aES = $-1.02$).

3.3. Post-exercise HRV indices

Visual examination of individual HR traces for recovery conditions confirmed the stabilities of the 3-min analyzed period of spectral HRV analyses (Fig. 1). HRV indices are reported in Table 1. Poincaré plots were drawn for all participants and were comet-shaped in the three recovery conditions. R–R intervals and associated Poincaré plots in a representative participant during the 3-min recovery periods under CWI, TWI and CON conditions are illustrated in Fig. 1.

The main ‘condition’ effect was $P=0.053$ for mRR. Consequently, aES was calculated to assess the magnitude of differences between the three recovery modalities. When compared with CON, mRR tended to be greater in CWI (aES = 0.59) and in TWI (aES = 0.89). Ln rMSSD was greater for CWI ($P=0.05$) but not for TWI ($P=0.964$) when compared with CON. When comparing both water immersion methods, a tendency towards greater mRR in TWI was observed (aES = 0.61). A tendency towards a higher Ln rMSSD ($P=0.08$; aES = 1.07) was observed in CWI compared with TWI. SD1 and SD2 ratios (i.e., SD1/SD2) for the three conditions were $>0.15$ (0.17±0.07 for CON, 0.22±0.08 for TWI and 0.19±0.07 for CWI) and were not significantly different ($P=0.19$).

3.4. Post-exercise time-varying vagal-related HRV index

Fig. 2 illustrates the time course of the time-varying Ln rMSSD30s in control and water immersion (i.e., CWI and TWI) conditions. A significant ‘recovery condition’ ($P=0.036$) and a ‘time’ ($P<0.001$) effect, as well as a significant ‘time × condition’ interaction ($P=0.029$) were found. When compared with CON, we found that Ln rMSSD30s increased faster in CWI and in TWI. While the within-condition time effect was significant at 180 s ($P=0.026$) for CON, significant changes were observed at 60 s ($P<0.001$) and 90 s ($P=0.008$) with TWI and CWI, respectively. A significant ‘time × condition’ interaction was noted between CON and CWI for Ln rMSSD30s at 90 s ($P=0.008$) and at 120 s ($P=0.005$). When comparing CWI with TWI, we found that Ln rMSSD30s increased faster in the TWI compared with the CWI condition. While the within-condition time effect was significant at 90 s ($P=0.008$) for CWI, significant changes were observed at 60 s ($P<0.001$) with TWI. A higher Ln rMSSD30s was also found at 300 s ($P=0.044$) for the CWI compared with the TWI condition.

4. Discussion

This study is the first to investigate the effect of head-out water immersion temperature on post-exercise HRV and HRV indices. In accordance with our hypotheses, results showed that water immersion itself can accelerate the restoration of parasympathetic function, irrespective of water temperature. Results also show that, as during resting conditions, cold receptor stimulation via CWI has a cumulative effect on post-exercise HRV vagal-related indices compared with thermoneutral immersion.

4.1. Reliability of Wingate test and submaximal exercise

Wingate test performances were within the range of previously published data on peak power in soccer and handball players (Popadic Gacesa et al., 2009). While post-exercise lactate concentrations were not measured following the exercise sequence in the present study, the mean peak power produced by the participants reflected the maximal nature of the test. Mean Wingate test cycling peak power and submaximal exercise HR values were also comparable and showed good reliability throughout the three recovery conditions. This finding implies that the presumed oversympathetic activity applied using the Wingate test at the start of the submaximal runs was likely similar across all three conditions.

4.2. Effect of water immersion on immediate post-exercise HRR

Because direct measurement of post-exercise parasympathetic nerve activity was not feasible in this study, we used HRR and HRV indices to noninvasively estimate cardiac autonomic activity (Buchheit et al., 2007b). When compared with the control condition, we found that immediate post-exercise parasympathetic activity was higher with immersion, irrespectively of water temperature. These results confirm that water immersion itself may stimulate parasympathetic activity, presumably via baroreceptors stimulation (i.e., cardiopulmonary and

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aortic receptors) (Gabrielsen et al., 1996). These baroreceptors are known to be sensitive to deformation and may be affected by minor changes in blood volume (Lacolley et al., 1992, Taylor et al., 1995), which could occur as a result of the increased hydrostatic pressure (i.e., the shift of peripheral blood towards the thoracic vasculature, and the resultant increase in the central blood volume).

Regarding the effect of water temperature on HRR indices, we observed that HRR−τ was shorter in TWI compared with CWI (aES = −1.02). No difference was noted however between both immersion temperatures for HRR60s (Table 1). Disparity in the response of the two HRR indices may be related to the fact that these variables span different time frames, or to their different underlying mechanisms. HRR60s is thought to represent the immediate post-exercise parasympathetic reactivation, whereas HRR−τ is thought to be more related to progressive withdrawal of sympathetic activity (Buchheit et al., 2007b). Thus, delayed HRR−τ with CWI might be related to a greater activation of the peripheral sympathetic response to cold application (Wilcock et al., 2006).

4.3. Effect of water immersion on immediate post-exercise HRV

Limitations to HRV indices are well known (Task Force, 1996). In accordance with previous studies, we noted that irrespective of water temperature, immersion induced a shift towards parasympathetic dominance (i.e., inferred from a longer mRR in CWI and TWI compared with CON) (Miyamoto et al., 2006, Mourot et al., 2008, Buchheit et al., 2009d). CWI applied immediately following exercise lead to greater vagal-related HRV indices compared with the control out-of-water condition (Table 1). This beneficial effect of CWI on cardiac parasympathetic function is in accordance with previous studies showing the beneficial effects of CWI on parasympathetic activity at rest (Mourot et al., 2008) and following supramaximal exercise (Buchheit et al., 2009d). Conversely, the lack of a significant effect of TWI on absolute vagal HRV indices is in contrast with results reported by Miyamoto et al. (2006), who found an increase in vagal-related HRV indices during TWI (34 °C) at rest (with no previous exertion). These discrepancies may be related to the possible confounding effects that post-exercise body temperature (Buchheit and Laursen, 2009) and blood pressure normalisation (Franklin et al., 1993) may have on cardiac autonomic activity. Moreover, analysis of the time-varying vagal-related index (i.e., Ln rMSSD60s) revealed a faster parasympathetic reactivation in the water immersion condition, irrespective of water temperature (Fig. 2).

CWI was found to be more efficient than the TWI temperature at increasing vagal-related HRV indices (Table 1). This apparent greater impact of CWI is in accordance with the results reported by Mourot et al. (2008) during resting conditions. This might be due to the profound effect that cold application has on thermolysis (Pretorius et al., 2006), which might have accelerated heat dissipation and the restoration of post-exercise-induced hypotension in the CWI condition (Franklin et al., 1993) and consequently quickened the reinstatement of vagal-related indices (Buchheit and Laursen, 2009). In CWI, cold-induced vasconstriction might have further shifted peripheral blood into the thoracic vasculature (Mourot et al., 2008), thereby increasing central blood volume and cardiac output. This, in turn, might have helped restoration of post-exercise-induced hypotension. Nevertheless, since blood pressure was not measured in this study, this latter hypothesis could not be verified. When comparing the respective effect of water temperatures on the time-varying vagal-related index, we found a faster parasympathetic reactivation with TWI compared with CWI (i.e., significant time effect occurring earlier with TWI compared with the cold condition [Fig. 2]); and a higher parasympathetic activity in CWI when compared with TWI (i.e., significant ‘time’ × ‘condition’ interaction at 300 s [Fig. 2]). Results for the time-varying vagal-related index are thus in line with those of HRR (short term changes, i.e., within the first 1–2 min) and HRV (long term changes, i.e., after 4–5 min) indices (Table 1).

Since the immersion-induced increase in vagal activity may occur under the heightened exercise-induced sympathetic level, a sympathovagal interaction was likely in the water immersion trials of the present study. Studies have reported that when both arms of the autonomic nervous system are highly activated, the vagal effect on HR may be greater (Leyv, 1971, Tulppo et al., 1998). Conversely, sympathetic activation can also attenuate the vagal influence on HR (Miyamoto et al., 2003). Whether concomitant sympathetic tone augments the HR response to vagal stimulation would depend on the type (e.g. neural vs. humoral stimulation) and site (pre- vs. postsynaptic) of adrenergic receptors most selectively activated under a given condition (Miyamoto et al., 2003, Miyamoto et al., 2004). Our results suggest that the presumed sympathovagal interaction did not occur since normal comet-shaped scatter plots (i.e., SD1/SD2 > 0.15) without torpedo-shaped or parabola-like plots were observed (Fig. 1, bottom and Table 1) for all subjects in the three conditions (Tulppo et al., 1998). We can therefore postulate that the increase in vagal-related HRV indices under the water immersion conditions in the present study were likely related to the stimulation of pressure-dependent baroreceptors, and to the additional co-activation of cold receptors with the cold water immersion condition. It has experimentally been demonstrated that small increases in blood volume may lead to an increase in vagal-related HRV indices (Spinelli et al., 1999) and an accelerated parasympathetic reactivation (Buchheit et al., 2009c). Finally, a cumulative effect might have occurred from the joint stimulation of the water immersion-induced baroreceptor stimulus and cold receptor stimulus in reinforcing the post-exercise parasympathetic activity observed.

4.4. Limitations

Since cardiac activity was only assessed during immersion phases, it is not known whether the observed beneficial effect of water immersion on post-exercise parasympathetic reactivation is sustained beyond the acute period. In addition, our present study protocol did not permit us to decipher the respective effects of immersion vs. cold exposure on post-exercise parasympathetic reactivation. Administering cooler ambient air conditions might have helped us to isolate the independent effects of cold and hydrostatic pressure on post-exercise HRV and should be the focus of future work. Techniques such as low body negative/positive pressure, which largely affect central blood volume, could also be used in the future to examine the respective links between changes in blood volume and post-exercise parasympathetic activity. Finally, for this study we preferred recruiting moderately trained subjects because we expected that they would be capable of handling the demands of the present experimentation. Thus, clarification is needed as to whether populations with lower physical activity levels and/or lower parasympathetic reactivation levels show similar responses to the water immersion stimulus.

5. Conclusion

To conclude, while investigating for the first time the effect of water immersion and its temperature on post-exercise vagal-related HR-derived indices, we showed that water immersion immediately after exercise, irrespective of water temperature, appears to be a simple, non-invasive and effective means of immediately accelerating post-exercise parasympathetic reactivation. Given the small differences noted between the two conditions used, the choice of water temperatures should be left up to practitioners.

References


