Effect of cold water immersion on postexercise parasympathetic reactivation

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Buchheit M, Peiffer JJ, Abbiss CR, Laursen PB. Effect of cold water immersion on postexercise parasympathetic reactivation. Am J Physiol Heart Circ Physiol 296: H421–H427, 2009. First published December 12, 2008; doi:10.1152/ajpheart.01017.2008.—The aim of the present study was to assess the effect of cold water immersion (CWI) on postexercise parasympathetic reactivation. Ten male cyclists (age, 29 ± 6 yr) performed two repeated supramaximal cycling exercises (SE1 and SE2) interspersed with a 20-min passive recovery period, during which they were randomly assigned to either 5 min of CWI in 14°C or a control (N) condition where they sat in an environmental chamber (35.0 ± 0.3°C and 40.0 ± 3.0% relative humidity). Rectal temperature (Trec) and beat-to-beat heart rate (HR) were recorded continuously. The time constant of HR recovery (HRRτ) and a time (30-s) varying vagal-related HR variability (HRV) index (rMSSD30s) were assessed during the 6-min period immediately following exercise. Resting vagal-related HRV indexes were calculated during 3-min periods 2 min before and 3 min after SE1 and SE2. Results showed no effect of CWI on Trec (P = 0.29), SE performance (P = 0.76), and HRRτ (P = 0.61). In contrast, all vagal-related HRV indexes were decreased after SE1 (P < 0.001) and tended to decrease even further after SE2 under N condition but not with CWI. When compared with the N condition, CWI increased HRV indexes before (P < 0.05) and rMSSD30s after (P < 0.05) SE1 and SE2. Our study shows that CWI can significantly restore the impaired vagal-related HRV indexes observed after supramaximal exercise. CWI may serve as a simple and effective means to accelerate parasympathetic reactivation during the immediate period following supramaximal exercise.

Heart rate variability; postexercise heart rate recovery; supramaximal exercise; cooling strategies

Supramaximal exercise is a proven time-efficient stimulus for increasing cardiorespiratory function and exercise performance (18). Although the benefits of supramaximal training are well known, the acute effects of supramaximal exercise are a severe disturbance to homeostasis and a poor cardioprotective background. This is reflected by an almost nonexistent level of parasympathetic reactivation [i.e., very low levels of vagal-related heart rate (HR) variability (HRV) indexes] in the 10-min period after a repeated sprint exercise (8). When exercise is performed in the heat, the homeostatic disturbance is confounded through a temperature-mediated increase in sympathetic outflow and decrease in parasympathetic reactivation (6, 21). Since sympathetic hyperactivity (5) or reduced cardiac vagal tone (3) after exercise may underlie ischemic heart disease and the pathogenesis of malignant ventricular arrhythmias and sudden cardiac death, studies examining the sympathetic and parasympathetic activation after supramaximal exercise in the heat are needed.

The recovery and subsequent performance effects of immersing oneself in cold water following supramaximal exercise in the heat are receiving growing attention (25, 31, 41, 42). Although immersion in cold water baths (14–18°C) has been shown to enhance subsequent endurance performance (41, 42), cold water immersion (CWI) can also acutely increase the incidence of heart arrhythmias (for review, see Ref. 15). For example, a marked tachycardia and a greater incidence of ectopic cardiac beats have been reported in both healthy and elite divers following CWI (35). Furthermore, the increased cardiac workload associated with tachycardia and cold-induced vasodilatation could result in a cardiovascular incident leading to incapacitation and drowning (39). This cold shock response observed immediately after water immersion is in fact thought to be responsible for a number of open water deaths each year (39). Whether CWI, at temperatures commonly used in the field, constitute a safe and recommendable recovery strategy must be questioned, especially after supramaximal exercise in the heat, where the contrast between body and water temperature is at its highest and where parasympathetic activity is most reduced (6, 21).

Water immersion itself can trigger parasympathetic heart control (28, 32), as well as eliciting a higher cardioprotective environment (3) compared with a (nonwater) control (N) condition. For instance, water immersion of the face induces a diving reflex characterized by bradycardia, peripheral vasodepression, and a diminished cardiac output (22). In contrast, the rise in hydrostatic pressure on the body from the surrounding water (29) leads to an increase in vagal-related HRV indexes (22), perhaps related to the rise in central blood volume, cardiac output, stroke volume, and central venous pressure (29). These higher blood pressures activate arterial high pressure and cardiopulmonary low pressure baroreflexes, which may enhance vagal nerve activity and inhibit sympathetic nerve activity (34), leading to a bradycardia (34) and an increase in vagal-related HRV indexes (36). As a result, the influence of water immersion under conditions common in the applied sport setting on autonomic activity and heart rhythm is difficult to predict. In these situations, participants are generally submerged in a seated position to the midsternal level, and the diving reflex-mediated parasympathetic stimulation might not occur. In addition, ectopic cardiac beats, associated more with the diving reflex than water immersion itself (because the time link with respiration), might be avoided with this procedure (15).

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Temperature of the water immersion bath can also alter parasympathetic heart control, through its effect on peripheral cutaneous vasoconstriction, on baroreceptor loading and the resultant vagal activation (28). Nevertheless, the cold-related decrease in sympathetic activity might not occur during short-term immersion durations common in the field sport setting, since changes in core temperature usually only occur with longer immersion duration (33). Conversely, since temperatures used in the field (i.e., <15°C) (25, 30, 41, 42) are far from thermally comfortable (24), cold baths could result in further sympathetic activation (15, 24) by creating an accentuation of sympathovagal antagonism that would counter the beneficial parasympathetic effect of the increased hydrostatic pressure.

In light of our limited understanding of the effect that short-term CWI exposure has on postexercise autonomic control, the purpose of the present study was to examine the effect of a 5-min CWI at a temperature commonly used in the applied sport setting (i.e., 14°C) on vagal-related HR-derived indexes measured after supramaximal exercises performed in the heat. From a clinical perspective, we sought to assess the safety of the CWI procedure after maximal exercise. As a consequence of the apparent absence of a diving reflex, and because of the cold shock-related increases in sympathetic activation, we hypothesized that CWI immediately after exercise would decrease parasympathetic modulation during the immersion period. In contrast, we believed that the persistent decrease in core temperature after CWI would increase markers of parasympathetic reactivation during recovery from supramaximal exercise.

METHODS

Subjects

Ten male cyclists (age, 29 ± 6 yr; stature, 181.7 ± 4.0 cm; mass, 79.3 ± 6.3 kg; and \( \text{Vo}_{2}\text{max} \), 56.5 ± 5.0 ml·kg\(^{-1} \)·min\(^{-1} \)) were recruited to participate in this study. Based on the assumption that a 2.1 ± 1.1 mHz\(^2 \) difference in postexercise high-frequency (HF) power (i.e., the power density of HRV in the HF ranges) is meaningful (8), we used Minitab 14.1 Software (Minitab, Paris, France) to determine that a sample size of at least nine participants would provide a statistical power of 0.8 at an α-level of 0.05. Subjects were provided with the procedures and risks associated with participation in the study and gave their written informed consent before participation. The study was approved by the University’s Human Research Ethics Committee.

Experimental Design

Subjects completed two exercise sessions separated by 7.0 ± 1.0 day. During the exercise sessions, subjects performed two supramaximal exercise bouts [1-km cycling time trials; supramaximal cycling exercises (SE)\(_1 \)] and SE\(_2 \); adapted from Marsh and Sleivert (25)] on a Velotron cycle ergometer (RacerMate) in an environmental chamber maintained at 35°C and 40% relative humidity (rh). For each SE, subjects sprinted from a standing start using a fixed resistance (gear ratio of 53 × 14). Before the trials, subjects were familiarized with the SE protocol, and the SEs were completed without a preceding warm-up. Immediately after SE\(_1 \), subjects began 20 min of passive recovery, during which they were randomly assigned to either 5 min of CWI in 14°C (30) or a control (N) condition where the subject remained seated in the environmental chamber (Fig. 1). CWI occurred from 7.5 to 12.5 min of the 20-min recovery period. Subjects completed SE\(_2 \) within 2 min after the specific recovery condition period. During the SEs, average power (W) and completion time (s) were recorded for analysis. Beat-to-beat HR and rectal temperature (\( T_{\text{re}} \)) were continuously recorded throughout the experimental period. The subjects’ rating of recovery was measured after the 20-min recovery period using a 10-point Likert scale, with a rating of 1 as feeling not recovered and a rating of 10 for feeling fully recovered.

\section*{CWI}

During the 5-min CWI, subjects were submerged in an inflatable water bath in the seated position to the midsternal level, wearing only their cycling shorts. Water temperature was maintained at a constant 14°C by a specially designed water refrigeration unit (iCool Porta-cover, Queensland, Australia). The 14°C water temperature selected for this study has been shown to be both effective at lowering body temperature while remaining tolerable to most subjects (30).

\( T_{\text{re}} \) was measured using a disposable rectal thermometer (Monatherm Thermostat, 400 Series; Mallinckrodt Medical, St. Louis, MO) inserted ~12 cm past the anal sphincter. \( T_{\text{re}} \) measurements were recorded via a data logger (Grant Instruments, Shepreth Cambridgehire, UK) at a sampling rate of 1 Hz. For simplicity and statistical analysis, \( T_{\text{re}} \) data are presented as an average of the last 1-min period during each HR measurement (see Fig. 1).

\section*{Beat-to-beat HR Analyses}

\textbf{Data recordings and treatment.} A Polar S810 HR monitor (Polar Electro, Kempele, Finland) was used to continuously record beat-to-beat HR during each exercise and each subsequent recovery phase (17). All R-R series were extracted on an IBM-compatible personal computer using a processing program (Polar Precision Performance SW 4.03; Polar Electro, Kempele, Finland). Occasional ectopic beats (irregularity of the heart rhythm involving extra or skipped heart beats, i.e., extrasystole and consecutive compensatory pause) were visually identified and manually replaced with interpolated adjacent R wave-to-R wave interval (R-R interval) values.

\textbf{Postexercise HR assessment.} Within 5 s of completing the exercise, subjects sat passively in the environmental chamber, during which time beat-to-beat HR was analyzed for a 6-min recovery period. HR was calculated by fitting the 6-min postexercise HR data into a first-order exponential decay curve (11). A HR time constant (HRR\(_T \)) was then produced by modeling the resultant first 5 min of HR data using an iterative technique.
Short-term resting HRV analysis. Before exercise, subjects were asked to remain quietly seated on a chair and to breathe regularly during a 5-min period in the environmental chamber (35°C; 40% rh). HRV analyses were performed at four time points throughout the exercise sessions (Fig. 1): on the last 3 min of the 5-min period preceding SE1 (pre-SE1) and SE2 (pre-SE2) and on the last 3 min of the 6-min recovery period after both SE1 (post-SE1) and SE2 (post-SE2). 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 (8).

Nevertheless, respiratory rate was always in the HF range (>0.15–0.50 Hz) and did not differ significantly during any of the three measurement periods. Mean HR at 3 to 6 min, the SD of normal R-R intervals (SDNN), the root mean square difference of successive normal R-R intervals (rMSSD), the percentage of successive R-R differences >50 ms (pNN50), and the short- (SD1) and long-term (SD2) R-R interval variability indexes of Poincaré plots were calculated for the 3 min of recorded data (Fig. 2). After the ectopic-free data were detrended and resampled (fixed linear sampling; frequency of 1/024 equally spaced points per 3-min period), a power frequency analysis was performed sequentially with a fast Fourier transform based on a nonparametric algorithm with a Welsh window. The power densities in the low-frequency (LF) band (0.04–0.15 Hz) and the HF band (>0.15–0.50 Hz) were calculated from each 3-min spectrum by integrating the spectral power density in the respective frequency bands. Poincaré plots were generated by plotting each R-R interval as a function of its previous R-R interval (40). The shapes of the Poincaré plots were also visually examined as previously reported (5A, 40).

Fig. 2. R wave-to-R wave intervals (R-R intervals) and associated Poincaré plots in a representative subject during the 3-min periods under CWI and N conditions. Each R-R interval (R-Rn+1) is plotted as a function of previous R-R interval (R-Rn).
A condition × time interaction was also not found (P = 0.16). During the N condition, when compared with baseline values (37.1 ± 0.2°C), Ts was similar after SE1 (37.5 ± 0.2°C; P = 0.49) but tended to increase thereafter after SE2 (37.6 ± 0.1°C). For the CWI condition, when compared with baseline values (37.2 ± 0.3°C), Ts did not show an increase throughout the experiment (i.e., after SE1 (37.4 ± 0.2°C) and before (37.4 ± 0.3°C) SE2). Surprisingly, there was no difference in Ts between conditions (i.e., during CWI) and after SE2.

**Effect of CWI on Peak HR and Postexercise HRR**

Peak HR values were 172.8 ± 9.1 and 175.5 ± 8.8 beats/min for SE1 and SE2 during N and 170.8 ± 7.9 and 170.6 ± 9.0 beats/min during CWI. There was neither a repetition (P = 0.65), condition (P = 0.21), nor repetition × condition interaction (P = 0.60) shown. Values for HRRr during N were 137.4 ± 53.4 and 128.3 ± 39.8 s after SE1 and SE2, respectively. During the CWI condition, HRRr values were 140.4 ± 52.4 and 128.2 ± 29.4 s after SE1 and SE2, respectively. There was neither a repetition (P = 0.46) nor condition (P = 0.91) effect and no repetition × condition interaction (P = 0.46) shown. Relative changes in HRRr were similar between both conditions (P = 0.61).

**Effect of CWI on Postexercise HRR**

Only two subjects displayed ectopic beats during CWI, but their occurrence was very low (<3%) and similar during the N condition. HRV values, before and after each exercise bout, for N and CWI conditions are presented in Table 1. R-R intervals and associated Poincaré plots in a representative subject during the 3-min periods under CWI and N conditions are illustrated in Fig. 2. Poincaré plots were comet shaped in all subjects for both conditions. Values for HF power, expressed as a percentage of pre-SE1, are illustrated in Fig. 3. All HRV indexes before SE1 were similar for N and CWI conditions (all P > 0.20). Most HRV indexes displayed comparable variations between conditions and times. For example, for HF power, there were time (P < 0.001) and condition (P = 0.02) effects, as well as a time × condition interaction (P = 0.03). During N condition, vagal-related indexes at post-SE1, pre-SE2, and post-SE2 were all significantly lower than pre-SE1 (e.g., all P < 0.01 for HF). There was no difference between post-SE1 and pre-SE2 (P = 0.60), whereas post-SE2 tended to be lower than post-SE1 (P = 0.10; ES = 1.2). During CWI, only post-SE1 and post-SE2 HF were lower than pre-SE1 HF (e.g., all P < 0.01); pre-SE2 HF tended to be lower than pre-SE1 HF (P = 0.07; ES = 0.9) and was higher than post-SE1 HF (P < 0.001). There was no difference between post-SE1 and post-SE2 HF (P = 0.99). Moreover, for the CWI condition, post-SE2 (P = 0.05; ES = 1.0) and post-SE2 (P = 0.11; ES = 1.2) HF values tended to be higher than those values during N condition.

**Effect of CWI on the Time-Varying HRV Index**

There were condition (P < 0.001) and time (P < 0.001) effects on rMSSD30, but only a tendency for a repetition effect (P = 0.10). Although there was a condition × repetition interaction shown (P < 0.01), neither a time × condition (P = 0.98) nor a time × repetition (P = 0.99) interaction was found.

![Fig. 3. Values are means ± SD. Natural log of high-frequency power density measured before and after the first supramaximal exercises (pre-SE1 and pre-SE2) and second supramaximal exercises (pre-SE1 and post-SE2), during N and CWI conditions. *Significant within condition difference vs. pre-SE1 (P < 0.05); †significant within condition difference vs. post-SE1 (P < 0.05); ‡within condition difference vs. post-SE1 with large effect size (ES >0.80); §difference vs. N condition with large ES (ES >0.80). lnHF, natural log of high frequency.](http://ajpheart.physiology.org/)

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**Table 1. HR-derived indexes of parasympathetic reactivation calculated for each recovery condition**

<table>
<thead>
<tr>
<th></th>
<th>Pre-SE1</th>
<th>Post-SE1</th>
<th>Pre-SE2</th>
<th>Post-SE2</th>
</tr>
</thead>
<tbody>
<tr>
<td>N</td>
<td></td>
<td></td>
<td></td>
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</tr>
<tr>
<td>SDNN, ms</td>
<td>84.3 ± 31.8</td>
<td>20.2 ± 6.9*</td>
<td>30.9 ± 13.7*</td>
<td>14.2 ± 3.5*</td>
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<tr>
<td>rMSSD, ms</td>
<td>56.4 ± 28.3</td>
<td>9.5 ± 4.7*</td>
<td>12.0 ± 6.7*</td>
<td>6.6 ± 1.3*†</td>
</tr>
<tr>
<td>pNN50, %</td>
<td>9.6 ± 8.1</td>
<td>0.2 ± 0.4*</td>
<td>0.6 ± 0.9*</td>
<td>0.0 ± 0.0*</td>
</tr>
<tr>
<td>SD1, ms</td>
<td>39.9 ± 22.1</td>
<td>6.7 ± 3.4*</td>
<td>8.5 ± 4.8*</td>
<td>4.7 ± 0.9*</td>
</tr>
<tr>
<td>SD2, ms</td>
<td>113.3 ± 50.2</td>
<td>27.6 ± 9.4*</td>
<td>42.6 ± 18.8*</td>
<td>19.4 ± 5.1*</td>
</tr>
<tr>
<td>LnHF, ms²</td>
<td>7.5 ± 1.2</td>
<td>4.1 ± 1.3*</td>
<td>5.7 ± 1.2*</td>
<td>3.2 ± 1.2*</td>
</tr>
<tr>
<td>HFnulu</td>
<td>0.32 ± 0.13</td>
<td>0.23 ± 0.18*</td>
<td>0.13 ± 0.05*</td>
<td>0.20 ± 0.17*</td>
</tr>
<tr>
<td>HR, beats/min</td>
<td>70.2 ± 12.4</td>
<td>102.5 ± 13.9*</td>
<td>97.5 ± 14.5*</td>
<td>103.5 ± 14.1*</td>
</tr>
<tr>
<td>CWI</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>SDNN, ms</td>
<td>76.9 ± 31.6</td>
<td>22.5 ± 7.2*</td>
<td>45.7 ± 23.4*</td>
<td>20.8 ± 7.7*</td>
</tr>
<tr>
<td>rMSSD, ms</td>
<td>47.5 ± 27.3</td>
<td>8.8 ± 3.6*</td>
<td>25.3 ± 23.0*</td>
<td>9.9 ± 4.9*§</td>
</tr>
<tr>
<td>pNN50, %</td>
<td>9.4 ± 7.0</td>
<td>0.1 ± 0.0*</td>
<td>2.5 ± 2.8*</td>
<td>0.1 ± 0.2*</td>
</tr>
<tr>
<td>SD1, ms</td>
<td>34.0 ± 20.5*</td>
<td>6.2 ± 2.6*</td>
<td>17.9 ± 16.3*</td>
<td>7.0 ± 3.4*</td>
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<tr>
<td>SD2, ms</td>
<td>102.9 ± 42.2</td>
<td>31.2 ± 10.9*</td>
<td>61.2 ± 30.4*</td>
<td>28.4 ± 10.5*</td>
</tr>
<tr>
<td>LnHF, ms²</td>
<td>6.3 ± 1.2</td>
<td>2.5 ± 1.2*</td>
<td>4.8 ± 1.5§</td>
<td>2.5 ± 1.1§†</td>
</tr>
<tr>
<td>HFnulu</td>
<td>0.26 ± 0.10</td>
<td>0.15 ± 0.08*</td>
<td>0.17 ± 0.12*</td>
<td>0.21 ± 0.16</td>
</tr>
<tr>
<td>HR, beats/min</td>
<td>72.5 ± 11.7</td>
<td>101.8 ± 13.0*</td>
<td>88.6 ± 13.1*‡</td>
<td>96.3 ± 13.3*</td>
</tr>
</tbody>
</table>

Values are means ± SD. N, control condition; CWI, cold water immersion; SE, supramaximal cycling exercises. SD of normal R wave-to-R wave (R-R) intervals (SDNN), root mean square difference of successive normal R-R intervals (rMSSD), percentage of successive R-R differences >50 ms (pNN50), short (SD1)- and long (SD2)-term R-R interval variability indexes of Poincaré plots, respectively, natural-log of low-frequency (LnLF) and high-frequency (LnHF) power density, normalized high-frequency power (HFnulu), and mean heart rate (HR) were calculated for the 3-min period. *Significant within-condition difference vs. pre-SE1 (P < 0.05); †significant within-condition difference vs. post-SE1 (P < 0.05); ‡within-condition difference vs. post-SE1 with large effect size (ES >0.80); §difference vs. N condition with large ES (ES >0.80).
evident. Post-SE2 was lower than pre-SE1 during N condition ($P = 0.01$), whereas this was not the case during CWI condition ($P = 0.78$; Fig. 4). Post-SE2 values for CWI were higher than during N condition ($P < 0.001$).

**DISCUSSION**

This study examined for the first time the effect of CWI (14°C) following supramaximal sprint cycling in the heat on postexercise autonomic function. The main findings of the study were that 1) supramaximal exercise performed in the heat reduced all vagal-related HRV indexes within a short period after exercise cessation; 2) the decrease in vagal-related HRV indexes tended to be even more pronounced after a second exercise bout in the heat, but not when CWI was applied; and 3) in contrast with our first hypothesis, HRV indexes were significantly increased during CWI. Our results indicate that short-term exposure to CWI after supramaximal exercise in the heat can significantly restore the impaired vagal modulation observed after a single supramaximal exercise session in the heat and is thus likely to preserve postexercise parasympathetic reactivation during repeated high-intensity exercise.

**Effect of CWI on Postexercise HRV Following a Single Supramaximal Exercise Bout**

Because direct measurement of cardiac parasympathetic nerve activity immediately after exercise was not feasible in the present study, we used HRV indexes to noninvasively assess cardiac autonomic activity. Limitations to HRV indexes are well known (1, 4). The current view is that HRV offers a qualitative marker of cardiac parasympathetic regulation (4, 11). A reduction in cardiac vagal activity after exercise can underlie ischemic heart disease and the pathogenesis of malignant ventricular arrhythmias and sudden cardiac death (3). Our results confirm, at least under the N condition, that supramaximal exercise performed in the heat lowered the cardioprotective background. Indeed, supramaximal exercise caused a significant decrease in vagal-related HRV indexes within a short period after the termination of exercise (i.e., 6–15 min post;

![Fig. 4](image_url)

*Fig. 4. Average root mean square of successive differences in the R-R intervals measured on successive 30-s segments (rMSSD30s) during the 6-min recovery period after the 2 supramaximal exercise bout (SE1 and SE2), as calculated for participants under N or CWI conditions. †Significant condition effect, i.e., difference vs. post-SE1 ($P < 0.05$); ‡significant condition effect, i.e., difference vs. post-SE2 with N condition ($P < 0.05$). For the sake figure clarity, error bars have been omitted.*

In addition, the HF power values after 80 s of supramaximal cycling were slightly higher ($2.5 \pm 1.2 \text{ ms}^2$) than that observed previously after a 360-s repeated sprint exercise ($1.5 \pm 1.2 \text{ ms}^2$) in a thermoneutral environment (8). Nevertheless, this finding is likely explained by the difference in exercise load (i.e., duration, intensity, and anaerobic system participation) between these two studies (8).

After the first exercise bout, we observed an increase in parasympathetic modulation in the CWI condition that almost reached preexercise values; however, this was not observed in the N condition (Fig. 3). Moreover, it is worth noting that CWI was not accompanied with a higher prevalence of arrhythmias compared with the normal recovery condition (Fig. 2, top). An increase in hydrostatic pressure on the trunk and an increase in thoracic blood volume possibly stimulated central baroreceptors (29, 34), which may have resulted in a decreased sympathetic activity and a shift toward cardiac parasympathetic (modulation) predominance (34, 36). The thermal effects of CWI on the autonomic system are, however, more difficult to isolate from the hydrostatic ones. In contrast with our first hypothesis, our data could indicate that the sympathetic activation (cold shock) (15) at the start of the CWI was not as prevalent as suggested. In addition, although CWI resulted in only a modest (and not significant) decrease in core temperature, it is still likely to have caused the reduced sympathetic activity shown compared with the N condition (24).

Alternatively, the increase in vagal activity during CWI could have been the result of a sympathovagal interaction, which generally occurs when both arms of the autonomic system are activated at high levels (23, 40). For instance, a heightened sympathetic tone can enhance the HR response to vagal nerve activity (23); however, the opposite response (i.e., attenuated vagal effect on HR) has also been demonstrated (26). 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 (26, 27). In our study, it is likely that sympathetic tone was activated at all levels by all mechanisms; therefore, it was not easy to speculate on its interaction with HR. Nevertheless, visual examination of the shape of Poincaré plots (Fig. 2, bottom) suggests that the presumed sympathovagal interaction did not occur, since normal comet-shaped scatter plots (and no torpedo-shaped or parabola-like plots) were observed for all subjects in both conditions (40). Thus we could not determine the exact mechanisms by which the CWI affected the autonomic system after exercise, and this area requires future research. Nevertheless, we can put forth that our commonly used recovery condition (5 min at 14°C) resulted in a parasympathetic dominance that was reflected by the observed bradycardia and enhancement of HRV indexes.

Our data confirm that water immersion and cooling have a cumulative influence on parasympathetic heart control (28). Therefore, the particular effectiveness of a 14°C 5-min CWI to restore parasympathetic modulation relies on beneficial interactions between the two branches of the autonomic nervous system, responsible simultaneously for thermoregulation, cardiovascular stability, and homeostasis restoration. The present findings confirm that CWI can be used to transiently improve cardioprotective background, at least after a single supramaximal exercise bout in the heat (12). The aim of the following
sections is to promote further research and understanding into how supramaximal exercise, hyperthermia, and CWI may interact to affect HRV.

**Effect of CWI on Immediate Postexercise Parasympathetic Function Following Repeated Supramaximal Exercise Bouts**

We were surprised that the CWI treatment did not improve cycling performance during the second exercise bout compared with the N condition. Nevertheless, this is possibly explained by the fact that our subjects were not given the opportunity to warm up before each exercise. Even if recovery might have effectively been enhanced (i.e., through improved system stress metabolic clearance), a lower muscle temperature after the CWI could have lowered muscle contractile ability and exercise performance (2). Nevertheless, CWI, in addition to its effect on autonomic activity, was associated with a significantly higher rating of recovery.

Changes in vagal-related HRV indexes indicate that CWI may provide a protective effect after repeated supramaximal exercises in the heat. For instance, after the second supramaximal exercise bout, vagal-related HRV indexes tended to diminish further compared with preexercise levels in the N condition; however, there was no additional decrement in parasympathetic function with CWI (Table 1 and Fig. 3). It is worth noting that, for the N condition, postexercise HRV values after the second exercise bout tended to be lower than preexercise values, although not significantly (P = 0.10). However, we observed a strong ES (ES = 1.2), suggestive at least of a clinical effect (20). Moreover, the time-varying index, which represents the time course of parasympathetic reactivation, was significantly higher for the CWI condition (Fig. 4), suggesting of a greater cardioprotective background. Differences shown between the N and CWI conditions can be exclusively attributed to the 5-min CWI intervention, since we did not observe any differences in exercise performance or maximal HR that could have confounded the kinetics of parasympathetic recovery (8). We speculate that the interaction between sympathetic and parasympathetic systems, described above (23, 40), is the likely reason for our observed differences in HRV indexes in the normal and CWI conditions. In addition, high temperatures trigger sympathetic activation (24), which improves heat dissipation (e.g., cutaneous vasodilatation, sweating) and counters postexercise hypotension (21). The observation of no change in Tₑ after the second exercise bout in the CWI condition further explains the higher parasympathetic background shown.

**Effect of CWI on HRR**

To our knowledge, the effect of CWI on HRR has not been investigated. In the present study, CWI had no significant effect on the HRR. Given the strong parasympathetic background governing HR deceleration after exercise (11), these findings were surprising. Nevertheless, the increase in parasympathetic activity (inferred from increased vagal-related HRV indexes; see Effect of CWI on Postexercise HRV Following a Single Supramaximal Exercise Bout) after CWI is likely related to an increase in vagal modulation concomitant to increases in central blood volume (9, 36) rather than vagal tone. Therefore, the lack of a CWI effect on HRR is consistent with our present understanding of physiological mechanisms underlying HRR (9, 11). Indeed, recent findings have shown HRR to be related to the acetylcholine concentration at the receptor levels (16) or to DNA sequence variation in the CHRM2 gene locus of the muscarinic receptors of the heart (19) and not systematically to variation in its concentration (i.e., in its modulation) (9, 11). Our findings provide further evidence to support the theory that HRR and HRV indexes characterize distinct independent aspects of cardiac parasympathetic function (7, 9, 11, 16).

**Study Limitations**

The main objective of this study was to assess the effect of a commonly used CWI recovery strategy [5-min in 14°C (25, 31, 41, 42)] on HR-related indexes. Nevertheless, our present study protocol did not permit us to decipher the respective effects of immersion versus cold exposure on postexercise parasympathetic reactivation. Administering warmer water and cooler ambient air conditions might have helped us to isolate the independent effects of temperature and hydrostatic pressure on postexercise HRV and should be the focus of future work. In addition, using water immersion temperatures to maintain a constant hydrostatic vasoconstriction (38) could have also helped us to quantify the presumed sympathovagal interaction phenomenon mentioned. Nevertheless, maintaining equal temperatures of the two environmental mediums would not have been easy. Subjects would have had to sit in a very cold chamber [taking into account the high thermal conductivity of water, i.e., \(\sim -5^\circ\text{C} (12)\)] or been immersed in a hot water bath at an equivalent temperature to our ambient air [35°C (13)], which would have been difficult to handle in the heat before our supramaximal exercise. Finally, we chose to recruit trained cyclist in the present study, since they were familiar and conditioned with this highly demanding protocol. We acknowledge that our choice of subjects limits the applicability of our findings to a small population of individuals. Future research in sedentary individuals or others in certain disease states (e.g., hypertension or heart failure) is warranted.

The present study supports the likely benefits of using of CWI before and after supramaximal exercise performed in the heat. When used after a single exercise session, CWI significantly restored the impaired parasympathetic function to almost preexercise levels. Thus CWI may serve as a simple and effective means of accelerating parasympathetic reactivation during the immediate period following supramaximal exercise.

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