European Journal of Sport Science, 2016
http://dx.doi.org/10.1080/17461391.2016.1226391

Routledge
Taylor & Francis Group

ORIGINAL ARTICLE

Thermoneutral immersion exercise accelerates heart rate recovery: A potential novel training modality

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Abstract
This study compared heart rate recovery (HRR) after incremental maximal exercise performed at the same external power output (P_{ext}) on dry land ergocycle (DE) vs. immersible ergocycle (IE). Fifteen young healthy participants (30 ± 7 years, 13 men and 2 women) performed incremental maximal exercise tests on DE and on IE. The initial P_{ext} on DE was 25 W and was increased by 25 W/min at a pedalling cadence between 60 and 80 rpm, while during IE immersion at chest level in thermoneutral water (30°C), the initial P_{ext} deployment was at a cadence of 40 rpm which was increased by 10 rpm until 70 rpm and thereafter by 5 rpm until exhaustion. Gas exchange and heart rate (HR) were measured continuously during exercise and recovery for 5 min. Maximal HR (DE: 176 ± 15 vs. IE 169 ± 12 bpm) reached by the subjects in the two conditions did not differ ($P > .05$). Parasympathetic reactivation parameters (ΔHR from 10 to 300 s) were compared during the DE and IE HR recovery recordings. During the IE recovery, parasympathetic reactivation in the early phase was more predominant (HRR at Δ10–Δ60 s, $P < .05$), but similar in the late phase (HRR at Δ120–Δ300 s, $P > .05$) when compared to the DE condition. In conclusion, incremental maximal IE exercise at chest level immersion in thermoneutral water accelerates the early phase parasympathetic reactivation compared to DE in healthy young participants.

Keywords: *Maximal exercise; heart rate recovery; thermoneutral conditions; immersed ergocycle*

Introduction

Cardiac autonomic recovery after exercise has important physiological and clinical significance because it provides a deeper insight into neural cardiovascular regulation with physiological stress (Heffernan, Kelly, Collier, & Fernhall, 2006). Post-exercise heart rate recovery (HRR) is commonly used to assess non-invasive cardiac autonomic regulation and more particularly cardiac parasympathetic reactivation. In fact, after immediately ceasing exercise, heart rate (HR) returns to pre-exercise resting values in a mono-exponential fashion (Dupuy et al., 2012; Perini, Milesi, Biancardi, Pendergast, & Veicsteinas, 1998).

The nature of the recovery kinetics (2 phases) is an intrinsic property of the cardiovascular system and is modulated by the autonomic nervous system (Savin, Davidson, & Haskell, 1982). The early rapid decay of the HR kinetics is mainly determined by a restoration of cardiac parasympathetic reactivation whereas the second slow phase is associated with a decrease in cardiac sympathetic activity. A prolonged HRR, from a clinical standpoint, reflects delayed post-exercise parasympathetic reactivation and has been associated with several cardiovascular conditions, such as coronary heart disease, heart failure, hypertension and diabetes and is an independent predictor of all-cause and/or cardiovascular mortality (Cole, Blackstone, Pashkow, Snader, & Lauer, 1999; Gayda et al., 2012). Alternatively, from a performance standpoint, HRR has been suggested to be of similar usefulness, as it appears sensitive to fatigue and

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performance changes (Buchheit, 2014; Daanen, Lamberts, Kallen, Jin, & Van Meeteren, 2012). Post-exercise HRR can be quantified by several approaches: (1) the absolute difference between HR measured immediately at the end of maximal exercise and after 60 s of recovery (HRR_{60s}); (2) calculating the time constant of the HR decay by fitting the post-exercise HRR to a first-order exponential decay curve (HRRτ) or (3) analysing the first 30 s of HRR via semi-logarithmic regression analysis (T30) (Buchheit, Papelier, Laursen, & Ahmaidi, 2007).

Exercise training in water (running and cycling) is becoming more popular as it appears to be suitable for different populations such as athletes, sedentary subjects, elderly and cardiac patients (Benelli, Ditroilo, & De Vito, 2004; Garzon, Juneau, et al., 2014). Under head-out water immersion conditions, the external hydrostatic pressure increases the central blood volume that raises stroke volume, cardiac preload, cardiac output and venous return (Christie et al., 1990; Garzon, Juneau, et al., 2014; Sheldahl et al., 1984). These acute cardiovascular changes stimulate arterial baroreflexes (Gabrielsen et al., 1996; Pump et al., 2001) that may improve parasympathetic activity and reduce sympathetic activity, leading to a reduction in HR at rest and during exercise (Buchheit, Peiffer, Abbiss, & Laursen, 2009; Pump et al., 2001).

The importance of recovery for training adaptation and maintenance of performance has stimulated the use of various strategies to promote faster recovery from intense exercise (Roberts, Nosaka, Coombes, & Peake, 2014). In young subjects, thermoneutral and cold water immersions at midsternal level, after acute sprint exercise (on land), have been shown to accelerate parasympathetic reactivation (Al Haddad et al., 2010; Buchheit et al., 2009). Improving vagal nerve activity by exercising in thermoneutral water could confer some cardioprotective effect on the heart. To our knowledge, parasympathetic reactivation measured after a maximal incremental exercise in immersion condition (immersible ergocycle: IE) at the same external power output (P_{ext}) as compared to the same maximal incremental exercise performed on dry land ergocycle condition (DE) has not been studied yet. Therefore, the aim of this study was to compare the early decay of HRR, a marker of parasympathetic reactivation, after a maximal incremental exercise on IE vs. DE in healthy young subjects.

Methods

Subjects

Fifteen healthy young subjects (30 ± 7 years, body mass (kg) 69.8 ± 9.1, height (cm) 173 ± 6, BMI (kg/m^2) 23.3 ± 2.913) gave their informed written consent to participate in the study. This study was approved by the Research Ethics Committee of the Montreal Heart Institute. Body mass was measured with bio-electrical impedance (Tanita, model BC418, Japan). Inclusion criteria were age over 18 years. Exclusion criteria included: (1) inability to perform a maximal cardiopulmonary exercise test and (2) any documented cardiovascular, pulmonary, musculo-skeletal or metabolic diseases. Following a thorough briefing and medical screening, all participants performed, on two separate occasions and in a random order, a maximal incremental exercise test in a laboratory room at stable air temperature (21°C) and in a thermoneutral swimming pool water temperature of 30°C (Christie et al., 1990; Gayda, Juneau, Guiraud, Lambert, & Nigam, 2010). In this group, 13 subjects were normally fit as defined by a % $\dot{V}O_2$ peak predicted value ≥100%, and 2 subjects were less fit with a % $\dot{V}O_2$ peak predicted value <100%. The mean % $\dot{V}O_2$ peak predicted value was 126 ± 23% for the group.

Maximal incremental exercise test

Participants performed a maximal incremental exercise test in a random order on IE and DE with measurement of gas exchanges (Cosmed $K4b^2$, Cosmed, Rome, Italy) and HR (PhysioFlow®, Enduro model, Manatec, France) to verify that each participant performed a maximal effort. The tests were performed on two different bicycle ergometers: in the laboratory the DE was an electronically braked ergocycle (Ergoline 800S, Bitz, Germany), and in the swimming pool the IE was a Hydrorider Aquabike professional (Bologna, Italy), both being equipped with pedals straps. The IE was installed at a depth that allowed the participants to be immersed up to the chest level, as previously described (Garzon, Juneau, et al., 2014). In both conditions, resting measures were assessed during 3 min in a seated position on either the DE or IE. Initial external power output (P_{ext}) on the DE was set at 25 W and increased by 25 W every minute until exhaustion. The pedalling rate (rpm) was at a minimum cadence of 60 rpm. Nonetheless, participants were instructed to maintain a pedalling cadence of 80 rpm since previous studies have shown that in conditions simulating those seen during prolonged competitive cycling, higher cadences (i.e. 100 rpm vs. 80 rpm) are less efficient, resulting in greater energy expenditure and reduced peak power output (Stebbins, Moore, & Casazza, 2014). External power (P_{ext}) on the IE was controlled by pedalling cadence as in previously published studies (Garzon, Gayda, et al., 2014; Yazigi et al.,

2013). Initial pedalling cadence was set at 40 rpm (corresponding to a P_{ext} of 25 W). Pedalling cadence was then increased by 10 rpm until 70 rpm and thereafter by 5 rpm until exhaustion. Pedalling cadence was controlled with a metronome (Qwik Time Quartz metronome, China) and a pedalling rpm meter (Cateye®, Echowell F2, Taiwan). Each maximal incremental exercise test was followed by a 5-min recovery period (2 min at 25 W or 40 rpm and 3 min at 0 W or 0 rpm). The external forces during exercise on an IE are caused primarily by the mechanical components of the pedalling system (paddles, pedals and rods) and by leg movement drag (calf, foot and thigh) that are dependent on the surface area of the lower limbs and pedalling rate (rpm). The external power (P_{ext}) expressed in watts (W) was calculated by multiplying the total net force (F) overcoming the resistance of the system movement (pedalling system and legs) by the velocity (m/s) of pedal displacement. For more details on P_{ext} calculation methods on IE, we refer to our previously published articles (Garzon, Gayda, et al., 2014; Garzon, Juneau, et al., 2014).

Maximal exercise test criteria

Gas exchange data were measured every four respiratory cycles during testing and then were averaged every 15 s for minute ventilation ($\dot{V}E$, in L min^{-1} body temperature and pressure, saturated), oxygen uptake ($\dot{V}O_2$, in L min^{-1}, standard temperature and pressure, dry (STPD)), carbon dioxide production ($\dot{V}CO_2$, in L min^{-1} STPD). Maximal exercise tests on DE and IE lasted until the attainment of primary maximal criteria: (1) a plateau of $\dot{V}O_2$ despite an increase in cadence and (2) R.E.R. > 1.15, or one of the two secondary maximal criteria: (1) inability to maintain the required workload or (2) subject exhaustion with cessation caused by general fatigue that required exercise cessation. The highest $\dot{V}O_2$value reached during the exercise phase of each test was considered as the $\dot{V}O_2$ peak (Garzon, Juneau, et al., 2014; Gayda et al., 2010). The two tests were separated from each other by 1 week.

Post-exercise parasympathetic reactivation parameters

HR was measured continuously at rest, during exercise and recovery using impedance cardiography (Physioflow, Enduro, France) and was averaged every 10 consecutive heartbeats (Garzon, Juneau, et al., 2014). Several indexes were used to characterise post-exercise HRR (Buchheit et al., 2007; Dupuy et al., 2012). The first index was the D (in beats min^{-1}), defined as the absolute difference between HR immediately at the end of exercise and after several seconds of recovery (Cole et al., 1999). Since the HR declines after exercise in two phases, we calculate Δ10, Δ20, Δ30 and Δ60 for the short-term kinetic of HRR (Ostojic et al., 2010; Ostojic, Stojanovic, & Calleja-Gonzalez, 2011) and Δ120 Δ180, Δ240, Δ300 for the second slower phase of the kinetic. The second index was the T30 (in s), defined as the negative reciprocal of the slope of the regression line between the natural logarithm of HR and elapsed time from the 10th to the 30th second of exercise (Imai et al., 1994). Finally, the overall kinetics of HR during the 5-minute transition from exercise to rest was fitted with a mono-exponential function:

$$HR(t) = a_0 + a_1 \times e^{(-t/\tau)}, \quad (1)$$

where a_0 is the asymptotic value of HR (in beats min −1), a_1 is the decrement below the HR value at the end of exercise for $t = \infty$ (in beats min−1) and t is the time constant (i.e. the time needed to reach 63% of the gain, in s) (Adabag and Pierpont 2013; Buchheit et al., 2007; Dupuy et al., 2012; Dupuy, Bherer, Audiffren, & Bosquet, 2013; Perini et al., 1989). Measurement of post-exercise cardiac autonomic control parameters are illustrated in Figure 1.

Statistical analysis

Standard statistical methods were used for the calculation of means and standard deviations. Normal Gaussian distribution of the data was verified by the Shapiro–Wilk test. When distribution was abnormal, a logarithmic transformation was performed. The compound symmetry, or sphericity, was checked by

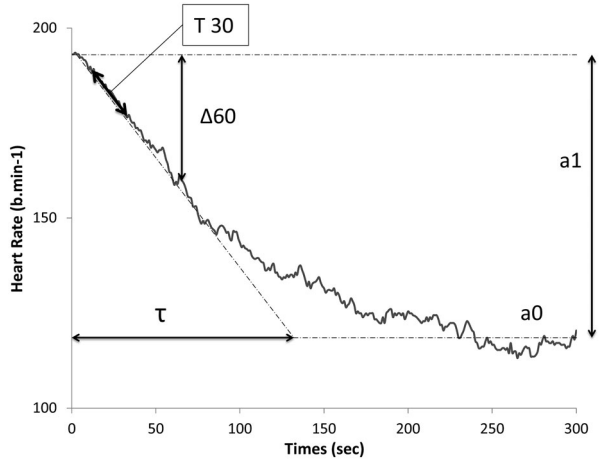

Figure 1. Graphical representation of HR measures to assess cardiac parasympathetic reactivation in this study.

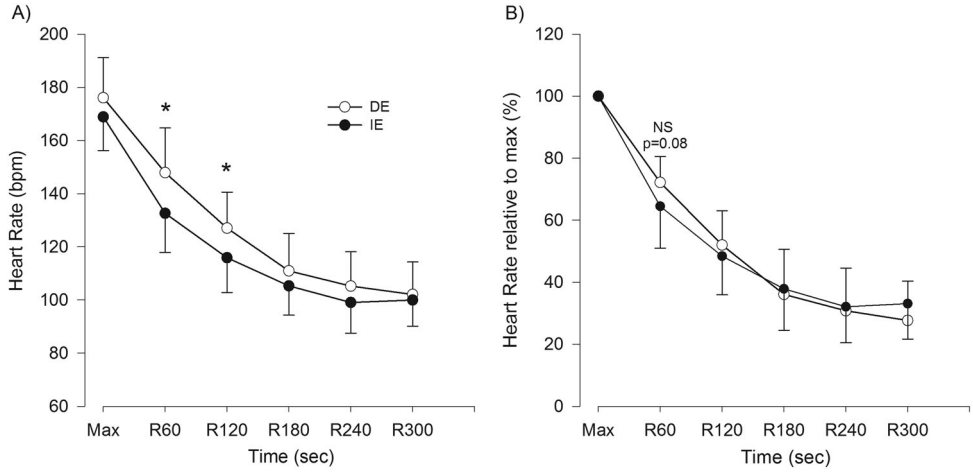

Figure 2. HRR according to post-exercise time recovery. (a) HRR expressed in beats per minute (bpm) according to time (s); (b) HRR expressed relative to the condition (IE or DE exercise) respective maximal HR (%) according to time (s). * $P < .05$ between IE and DE. Max is maximal HR reached at the end of exercise and R60 to R300 represents the post-exercise recovery time expressed in seconds; IE, immersible ergocycle; and DE, dry land ergocycle.

the Mauchley test. When the assumption of sphericity was not met, the significance of F ratios was adjusted according to the Greenhouse–Geisser procedure when the epsilon correction factor was <0.75, or according to the Huynh–Feldt procedure when the epsilon correction factor was >0.75 (Dupuy et al. 2013). A two-way ANOVA (condition × time) with repeated measures was performed to test the null hypothesis that physiological responses after maximal graded exercise test were similar when performed on dry land or in water. Multiple comparisons were made with the Bonferroni *post hoc* test. The magnitude of differences between conditions was evaluated by using Hedge's g (g). Hedge's is more adapted in our study than Cohen's d because the latter, in small samples, tends to overestimate the absolute value. The scale proposed by Cohen was used for interpretation. The size of the difference was considered either small ($0.2 < g < 0.5$), moderate ($0.5 < g < 0.8$) or large ($g > 0.8$) (Dupuy et al. 2013). Statistical significance was set at $P < .05$ level for all analyses.

Results

The P_{ext} (DE: 268.1 ± 59.2 vs. IE: 262.9 ± 63.9 W), R.E.R. (DE: 1.15 ± 0.10 vs. IE: 1.15 ± 0.14) and maximal HR (DE: 176 ± 15 vs. IE 169 ± 12 bpm) reached by the subjects in the two conditions did not differ ($P > .05$). The maximal rpm on IE was 86 ± 7. The $\dot{V}O_2$ peak reached with the DE was significantly higher when compared to the IE $\dot{V}O_2$ peak (3337 ± 802 vs. 2591 ± 173 mL/min, respectively, $P < .001$). The absolute and relative HRR are illustrated in Figure 2(a) and (b). Figure 2(a) shows that the absolute HRR with IE was significantly different ($P < .05$) to DE during the first two minutes of recovery. However, as shown in Figure 2(b), no significant difference was observed between IE and DE when HRR was normalised to their respective max HR, except at R60 that was near significance ($P = .08$). When the HRR was expressed in subsequent differences of ($HR - HR_{max}$) every 10 s the significant difference ($P < .05$) between IE and DE was only present during the first minute of recovery (Figure 3). The effect size of conditions (Hedge's g) on Δ HRR is presented in Figure 4. The effect size on HRR (decelerating effect) was large during the first

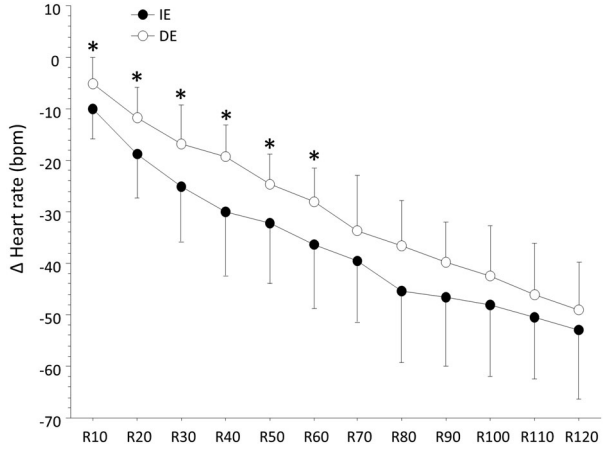

Figure 3. The difference in heart rate (ΔHR) that was obtained by subtracting HR at every 10 s from the condition's (IE or DE exercise) respective maximal HR, as a function of time (s). *$P < .05$ between IE and DE. R10–R120 represents the post-exercise recovery time expressed in seconds; IE, immersible ergocycle; and DE, dry land ergocycle.

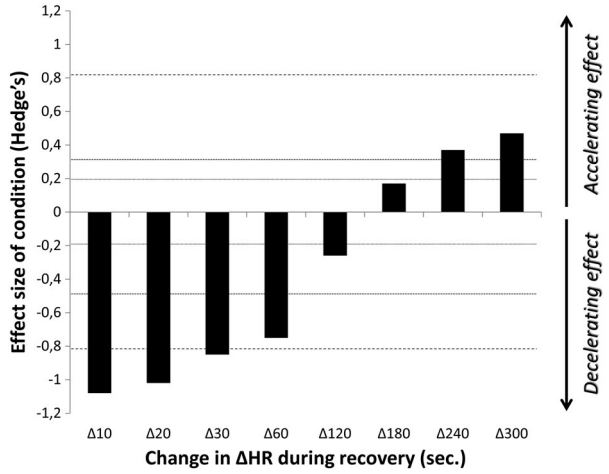

Figure 4. Effect size of condition (IE vs. DE, see Figure 1 legend) on HRR according to post-exercise recovery time. Δ10 to Δ300 represents the post-exercise recovery time expressed in seconds.

minute (Δ10 to Δ60) and disappeared thereafter. When compared to dry condition, we found a large decrease in the immersion condition of τ (DE: 128.6 ± 24 vs. IE: 85.9 ± 27.2 s; $P < .001$, $g = 1.57$). T30 also decreased in the immersion condition (DE: 325.6 ± 108.2 vs. IE: 278.3 ± 115.2 s, $P < .05$, $g = 0.4$). We also observed a large increase of a_1 in the dry condition compared to the immersion (DE: 89.4 ± 17.1 vs. IE: 69.4 ± 17.0 s, $P < .001$, $g = 1.11$). We found no difference between conditions in a_0 (IE: 89.9 ± 14.6 vs. DE: 94.9 ± 10 s, $P = .09$).

Discussion

The principal new finding of this study is that parasympathetic reactivation indexes (ΔHRR, τ and T30) were accelerated after maximal incremental exercise during cycling in immersion in thermoneutral conditions as compared to the dry land condition in healthy young subjects. To our knowledge, this is the first study to assess the effect of thermoneutral water immersion exercise on parasympathetic reactivation after a maximal incremental exercise performed on both IE and DE at the same P_{ext}. Since parasympathetic nerve activity cannot be measured directly in the immersion conditions, we have used non-invasive indexes (ΔHRR, τ and T30) of parasympathetic activity after maximal incremental exercise for both conditions. These non-invasive indexes have been previously studied (such as T30: Imai et al., 1994) with pharmacological blockage in DE conditions, and others (ΔHRR, τ) were used in immersion condition after sprint exercise performed on dry land (Al Haddad et al., 2010; Buchheit et al., 2009).

In our study, water immersion was associated with a more rapid deceleration of HR during the first minute of recovery after incremental maximal exercise (Δ10–Δ60). Our findings are similar to those reported by Al Haddad et al. (2010) who showed that the difference between the final HR at the end of exercise and the heart rate recorded 60 s later (HRR_{60s}) was greater in cold water immersion when compared to thermoneutral water immersion or control conditions (non-water). In their study, however, exercise was performed on dry land only, where the participants were instructed to immerse immediately into the water after the dry land exercise. This contrasts with our study, where HRR was monitored in exercise performed either on land or in water.

Several mechanisms have been proposed to explain why immersion accelerates HRR after exercise. After exercise in control condition (non-water), HR decreases mono-exponentially and is largely mediated by cardiac parasympathetic reactivation (Perini et al., 1998). Some known potential mechanisms, include the triggering of parasympathetic heart regulation with immersion when compared to a control condition (non-water) (Mourot et al., 2008; Perini et al., 1998), and changes in haemodynamic functions, such as an increase in central blood volume, cardiac output, stroke volume and central venous pressure (Garzon, Juneau, et al., 2014; Miyamoto, Oshima, Ikuta, & Kinoshita, 2006; Park, Choi, & Park, 1999). Previous studies suggested that immersion itself is responsible for a higher post-exercise parasympathetic activity by stimulating the baroreceptors (cardiopulmonary and aortic receptors) (Al Haddad et al., 2010; Buchheit et al., 2009; Gabrielsen et al., 1996). These baroreceptors are known to be sensitive to blood volume changes, and we previously demonstrated that stroke volume was higher in immersion during recovery from maximal incremental exercise as compared to dry land condition (Garzon, Juneau, et al., 2014). Therefore, we believe that these hemodynamics mechanisms would be mainly responsible of this higher parasympathetic reactivation seen in this study. Indeed, the exponential nature of this kinetics is modulated by the autonomic nervous system, and the rapid decline of HR after exercise is mediated by the restoration of parasympathetic activity on the cardiac sinus node (Kannankeril & Goldberger, 2002; Savin et al., 1982).

In the current study, the main effect of immersion on HRR only affected the early phase (Δ10–Δ60 s) and not the late phase (greater than Δ70 s), as shown in Figure 2. Our results are in agreement with those of Imai et al. (1994) where they showed that in healthy adults, athletes and patients with chronic heart failure, the vagal reactivation was the

principal determinant of the decrease in HR during the first 30 s of recovery. Thus, it is appropriate to suggest that the HRR in thermoneutral water immersion exercise with IE may have potentiated the early vagal reactivation. Since the rapid decay of HRR is mainly influenced by parasympathetic reactivation and the late phase by an amalgam between parasympathetic and sympathetic activity, our results confirm that water immersion has a selective effect on parasympathetic reactivation and not on sympathetic reactivation in post-exercise condition.

We, and others, have recently shown that water immersion is generally associated with an increase in SV (Garzon, Gayda, et al., 2014; Sheldahl et al., 1987) and a decrease in sympathetic activity that usually reduces absolute HRmax. In the current study, however, absolute HRmax was not significantly different between both experimental conditions (DE vs. IE). Thus, the quicker HRR on IE at Δ10 to Δ60 ($P < .001$) compared to DE indicates that parasympathetic reactivation in immersion is greater than on dry land and not attributed to an initial lower peak HRmax. Our results are in agreement with an increase in parasympathetic tone caused by immersion, as shown by Bastos et al. (2012) and Buchheit et al. (2009), but also other indices of cardiac autonomic regulation (i.e. HR variability) may be involved, such as a decrease in sympathetic activity. These differences are evident despite similar peak power outputs and respiratory exchange ratio (R.E.R) in both conditions.

Since recovery between exercise training sessions and competitive events is a key determinant of long-term training adaptation and successful performance (Roberts et al., 2014), the results of this study are interesting because it shows that after maximal incremental exercise at chest level thermoneutral water immersion, the recovery in the first 60 s is accelerated when compared at the same time point in dry land condition. In fact, it would be interesting to study further the effects of repeated bouts (HIIT, high-intensity interval training) of immersion exercise on parasympathetic adaptations in healthy subjects, and also in people with functional cardiac or other limitations with the purpose of rehabilitation. In addition, athletes may benefit from immersion exercise during preparation periods, given that immersion favours a higher efficiency of the external work (lower $\dot{V}O_2$ at same workload in water as compared to dry land condition) during incremental exercise and recovery, while in addition increasing cardiac output and stroke volume compared to exercise on dry land at the same external power output (Garzon, Juneau, et al., 2014).

In conclusion, the current study showed that immersion exercise accelerates the early phase HRR when compared to dry land exercise in healthy young participants. The acceleration of early phase HRR is probably due to higher stimulation of baroreceptors via an augmented central blood volume (higher post-exercise stroke volume in immersion). Our results were obtained in young healthy subjects and it would be of interest to determine whether patients with cardiovascular risk factors or cardiac diseases would demonstrate similar responses. An examination of the impact of an immersion training programme would also be an important question for the future.

Acknowledgments

The authors wish to express extreme gratitude to Julie Lalongé, Philippe Sosner and Joffrey Drigny for technical assistance.

Disclosure statement

No potential conflict of interest was reported by the authors.

Funding

Funding was provided by the ÉPIC Foundation and the Montreal Heart Institute Foundation.

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