

Sprint Cycling Performance Is Maintained with Short-Term Contrast Water Immersion

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ABSTRACT

CRAMPTON, D., B. DONNE, M. EGANA, and S. A. WARMINGTON. Sprint Cycling Performance Is Maintained with Short-Term Contrast Water Immersion. *Med. Sci. Sports Exerc.*, Vol. 43, No. 11, pp. 2180–2188, 2011. **Purpose:** Given the widespread use of water immersion during recovery from exercise, we aimed to investigate the effect of contrast water immersion on recovery of sprint cycling performance, HR and, blood lactate. **Methods:** Two groups completed high-intensity sprint exercise before and after a 30-min randomized recovery. The Wingate group ($n = 8$) performed 3×30 -s Wingate tests (4-min rest periods). The repeated intermittent sprint group ($n = 8$) cycled for alternating 30-s periods at 40% of predetermined maximum power and 120% maximum power, until exhaustion. Both groups completed three trials using a different recovery treatment for each trial (balanced randomized application). Recovery treatments were passive rest, 1:1 contrast water immersion (2.5 min of cold (8°C) to 2.5 min of hot (40°C)), and 1:4 contrast water immersion (1 min of cold to 4 min of hot). Blood lactate and HR were recorded throughout, and peak power and total work for pre- and postrecovery Wingate performance and exercise time and total work for repeated sprinting were recorded. **Results:** Recovery of Wingate peak power was 8% greater after 1:4 contrast water immersion than after passive rest, whereas both contrast water immersion ratios provided a greater recovery of exercise time (~10%) and total work (~14%) for repeated sprinting than for passive rest. Blood lactate was similar between trials. Compared with passive rest, HR initially declined more slowly during contrast water immersion but increased with each transition to a cold immersion phase. **Conclusions:** These data support contrast water immersion being effective in maintaining performance during a short-term recovery from sprint exercise. This effect needs further investigation but is likely explained by cardiovascular mechanisms, shown here by an elevation in HR upon each cold immersion. **Key Words:** EXERCISE RECOVERY, HIGH-INTENSITY EXERCISE, THERMOTHERAPY, CRYOTHERAPY, CONTRAST THERAPY

The effects of water immersion as a short-term recovery intervention have not been extensively explored in regard to subsequent exercise performance. Indeed, there are limited data to support the generalized use of water immersion for recovery from exercise. Despite this, recent evidence confirms that water immersion forms part of typical postexercise regimens among elite-level competitive sporting teams (37), with some published literature and many anecdotal reports suggesting that water immersion has been in use for some time by athletes and teams at a variety of competitive levels (9,28). From this, water immersion practices seem relatively equally distributed between using continuous cold/ice water immersion and using contrast water immersion that utilizes brief alternating periods of cold and hot water immersion.

Suggested rationales for implementing water immersion during recovery are for treatment of soft tissue injury (5,11), for relieving factors associated with muscle soreness and microinjury (1,31,34), or as a method to assist in clearance of metabolites and restoration of physiological systems to a preexercise state in preparation for subsequent exercise (4,17).

Cold water immersion after unaccustomed or eccentric exercise that typically produces muscle damage or microinjury has shown little effect in improving exercise performance or the recovery of muscle strength for up to 7 d after the exercise bout (1,18,24,31,34). And although a few studies have reported cold water immersion to limit muscle soreness and circulating blood markers of muscle damage during recovery (1,34), more often, these indicators have been reported to be unchanged by cold water immersion (8,18,24,31).

In relatively well-conditioned athletes, water immersion after nondamaging exercise has been shown to vary in its effectiveness at improving subsequent performance the day after a fatiguing exercise bout (20,33), after repetitive use over consecutive days (30,33), and during recovery from exercise in the heat (25,26,32). Although these studies used a variety of protocols to investigate both cold and contrast water immersion for up to 15 min immediately after the initial exercise bout, this comprises a small component of the time of recovery before the subsequent exercise bout.

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Therefore, despite the applied nature of these experimental protocols, this leaves substantial time during recovery whereby influences other than water immersion may affect the subsequent performance. In particular, if other practices typically performed during recovery are undertaken such as an active recovery or warm-down (4,15,21,28), stretching and massage (29,38,39), and passive rest. Although it is assumed that these practices would be constant for individual subjects, this is perhaps not the case between subjects. Therefore, to examine the effect of water immersion on subsequent performance, it is necessary to conduct the subsequent exercise in the immediate period after the immersion treatment so that the recovery is not complicated by other influences.

One recent study investigated a 20-min recovery between high-intensity short-duration (6–10 min) bouts of rock climbing in experienced female climbers (15). Cold water immersion was performed for the entire recovery period and reported to be effective in maintaining a better climbing performance and more rapid lactate clearance than passive rest (15). This protocol was not complicated by other factors and, therefore, provides a suitable method for investigating the effectiveness of water immersion on subsequent performance, at least over the short term. In addition, such a recovery duration is similar to that which may be experienced in a variety of settings after high-intensity exercise including mid/half-time game breaks in sports such as Gaelic football and hurling, soccer, and rugby, as well as breaks between performances in a variety of athletic and gymnastic events often scheduled on the same day during competitions.

The reasoning behind selecting to use either cold or contrast water immersion during recovery lacks scientific basis apart from cold water immersion being the method of choice in the treatment of symptoms of muscle soreness and injury (1,5,11,31,34). There is some suggestion that contrast water immersion may be more beneficial for subsequent performance by providing a more rapid restoration of normal muscle function and clearance of circulating metabolites (4,17).

Therefore, the aim of the present study was to investigate the effect of two contrast water immersion protocols implemented for the entire duration of a short-term and, as such, uncomplicated recovery period after two models of high-intensity sprint exercise on the recovery of blood lactate, HR, and subsequent exercise performance. It was hypothesized that contrast water immersion would facilitate a more rapid recovery and, therefore, provide a suitable technique to allow maintenance of performance in events with limited time available for recovery.

METHODS

Subjects

Sixteen healthy male Gaelic football players volunteered to participate in this study. Although playing positions varied among subjects, training for these players is conducted as a team through on-field drills and track/fitness work. As

such, the capability for sprint and endurance performance was consistent and similar across all subjects. Each subject was randomly assigned to one of two experimental groups that were defined according to the type of exercise bouts to be performed (described below): a Wingate cycling group (WG; $n = 8$; mean \pm SD = 25 \pm 3 yr, 82 \pm 6 kg, 180 \pm 9 cm) or a repeated intermittent sprint cycling group (RIS; $n = 8$; mean \pm SD = 23 \pm 1 yr, 81 \pm 5 kg, 184 \pm 4 cm). Each subject completed a medical questionnaire, provided written informed consent, and was then examined by a registered medical practitioner for approval for participation. All subjects were involved in vigorous exercise two to three times per week, and all had prior experience of water immersion as a recovery practice. However, as the present study was conducted on amateur Gaelic football players during the “off-season” period, subjects were not currently using water immersion during recovery from exercise. All subjects were nonsmokers and required to refrain from consuming caffeine and alcohol for 12 h before each laboratory session. In addition, subjects were tested at the same time of day for each trial and did not undertake exercise for 24 h before each laboratory session. The experimental protocol was approved by the Faculty of Health Sciences Research Ethics Committee, Trinity College Dublin.

Experimental Protocol

Subjects were required to complete three trials separated by 7 d. For each trial, subjects completed a short high-intensity cycling exercise bout (Ex1) followed immediately by a 35-min recovery period, which in turn was followed by a second identical high-intensity cycling exercise bout (Ex2). All cycling was completed in the exercise laboratory with the bulk of the recovery period completed within a separate dedicated recovery room. Air temperature in both rooms was regulated to 21°C \pm 2°C.

Recovery. The recovery period comprised a 2.5-min transition from the exercise laboratory to the recovery room, followed by a 30-min treatment period, then a further 2.5-min transition period back to the exercise laboratory to complete Ex2. During the first transition, subjects removed cycling shoes and socks and changed into swimming shorts, changing back into exercise clothing during the second transition. Towels were provided for subjects after all water immersion treatments so subjects could dry themselves before redressing for Ex2.

For each trial, during the 30-min treatment period, subjects undertook one of three possible treatments, with a different treatment used in each trial for each subject. These treatments were administered in a balanced randomized fashion for each group across the duration of the study. The treatments were (A) passive nonimmersed seated rest in air (PAS), (B) contrast water immersion with an alternating cold-hot treatment ratio of 1:1 (2.5:2.5 min, respectively) (OT1), and (C) contrast water immersion with an alternating cold-hot treatment ratio of 1:4 (1:4 min, respectively) (OT4). Immersion

bath temperatures were regulated to $8^{\circ}\text{C} \pm 1^{\circ}\text{C}$ for cold and $40^{\circ}\text{C} \pm 1^{\circ}\text{C}$ for hot. During all recovery treatments (PAS, OT1, and OT4), subjects were seated upright in a custom-built 330-L bath (Sturdy Products, Co. Wicklow, Ireland) with the knees bent so that the feet remained flat on the bottom of the bath. Subjects were required to keep the hands and arms rested on the sides of the bath and to minimize any active movement. This was above the level of the water in OT1 and OT4. The level of the water for the immersion treatments was to the top of the iliac crest, which ranged from 5 to 7 cm above the thigh level. The transitions between baths during OT1 and OT4 were nonvigorous and lasted no more than 5–6 s each. Aside from the initial entry into and final exit from the baths, this equated to 11 transitions for both OT1 and OT4. To allow for these transitions, the duration of each recovery treatment was set to 30 min. So in total, there were six cold immersion phases and six hot immersion phases, which was similar to other investigations (20).

In addition, the 30-min recovery was not complicated by other external factors, and the treatment duration was similar to that used in previous investigations (up to 20 min), albeit slightly longer but with an overall recovery duration within the range of those published previously (5 min to 24 h) (15,20,33,35). This provided many contrast immersion transitions that were expected to be sufficient to examine the response of HR and blood lactate with adequate resolution under these conditions throughout the early and late phases of recovery from sprinting. It was also expected that 30 min would be short enough so that exercise performance would not yet have recovered to baseline levels, thereby providing a model by which the effect of the recovery treatments on sprint exercise performance could be examined (14).

Exercise bouts (Ex1 and Ex2). All exercise was performed on an electromagnetically braked cycle ergometer (Excalibur Sport; Lode B.V., Groningen, The Netherlands). Ex1 and Ex2 were identical within groups but different between the WG and RIS groups. All subjects were familiarized with the exercise requirements for all trials in the week before commencing participation in the study.

WG group. For the WG group, each exercise bout required subjects to complete three standard 30-s Wingate tests in sequence, with each preceded by 4-min cycling at 50 W. During each bout, the cycle ergometer was controlled via a PC running Lode Wingate software (v1.0.12; Lode B.V.). For each Wingate test, the braking force applied was 7% body mass, and subjects were encouraged to cycle “all out” at the highest cadence possible for the duration of the 30-s test period. Total exercise time for each bout was 13.5 min. Average data normalized to body mass for peak power (PP) over the three Wingate tests and for total work (TW) for the exercise bout were recorded to examine short-term high-intensity cycling performance of the PAS, OT1, and OT4 trials in this group.

RIS group. For the RIS group, 1 wk before completing the first of the PAS, OT1, or OT4 trials, all subjects were required to complete an incremental cycling test to volitional

exhaustion to determine maximum power output (P_{max}), i.e., at $\dot{V}\text{O}_{2\text{max}}$. For this test, subjects initially cycled at 100 W. The workload then increased by 50 W every 3 min until 9 min, after which the workload increased by 25 W each minute until failure. P_{max} was determined as the highest workload able to be sustained for a minimum period of 30 s.

For the PAS, OT1, and OT4 trials, each exercise bout required subjects to complete a series of RIS, each lasting 30 s. Each trial commenced with an initial warm-up period of 6 min at 40% P_{max} , followed by the repeated sprint sequence that comprised cycling at 40% P_{max} for 30 s, followed by 120% P_{max} for 30 s, and this was repeated until subjects could not complete a single 30-s period. Invariably, this was a period at 120% P_{max} . Exercise time to failure and TW normalized to body mass were recorded for Ex1 and Ex2 to examine short-term high-intensity cycling performance of the PAS, OT1, and OT4 trials in this group.

Measurements

Blood lactate. For each trial, forearm venous blood samples were obtained before and after Ex1 and Ex2, at 1, 2.5, and 5 min into the recovery treatment and subsequently every 5 min for the duration of each recovery treatment. All samples were collected via a 20-gauge indwelling catheter connected to an extension line set, into 4.5-mL Vacutainers containing K^+EDTA anticoagulant (BD, Franklin Lakes, NJ). The catheter was flushed with and cleared of sterile saline both after and before each sample collection, respectively.

Each whole blood sample was immediately analyzed for blood lactate by injecting a 25- μL aliquot into an automated lactate analyzer via a syringepet (1500 Sport; YSI, Yellow Springs, OH).

HR. Subjects wore a standard chest strap and transmitter that recorded HR every 5 s throughout all trials (S725X; Polar Electro Oy, Kempele, Finland). Data were uploaded to a PC running Polar Precision Performance analysis software (v4.03.050), and data were extracted for analysis for before and after Ex1 and Ex2, and for every 30 s during the recovery period in the PAS, OT1, and OT4 trials.

Data Presentation and Statistical Analyses

Unless otherwise indicated, all data are expressed as mean \pm SEM. WG and RIS performance data were analyzed using a two-way repeated-measures ANOVA (trial (PAS, OT1, OT4) \times time (Ex1, Ex2)) followed by a Tukey–Kramer *post hoc* test. HR and blood lactate data were similarly analyzed using a two-way repeated-measures ANOVA (trial \times time (all sampling points)) followed by a Tukey–Kramer *post hoc* test. For both the WG and RIS groups, no differences were reported between subjects or trials for all measured variables taken before the start of each experimental trial. All statistical analyses were performed using the NCSS software package (v2007; NCSS, Kaysville, UT) with the level of significance set to $P < 0.05$. In addition, an *a priori* power analysis was conducted for expected outcomes of performance variables with power

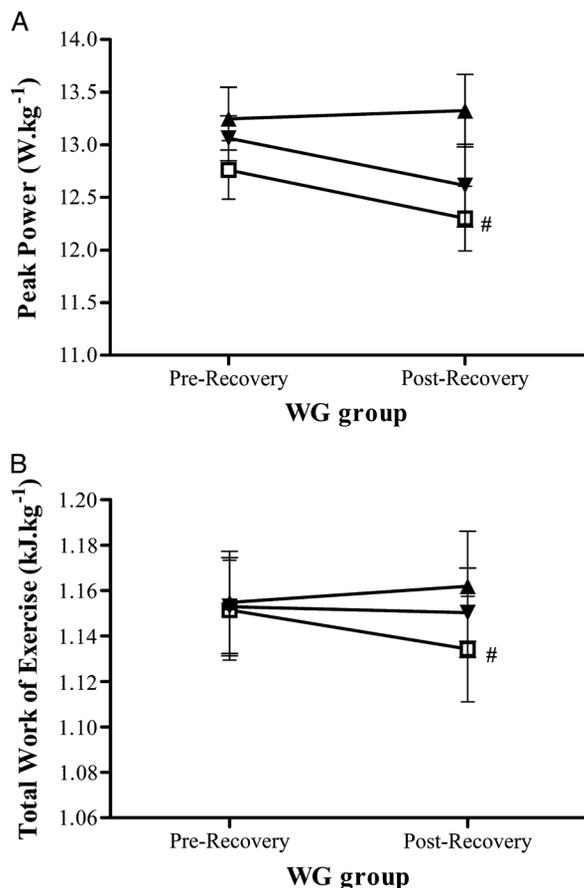


FIGURE 1—WG group performance data for the PAS (□), OT1 (▼), and OT4 (▲) trials. A, Mean normalized PP showed no main effect for time but a significant main effect for trial where PP is significantly greater in OT4 than in PAS for Ex2 ($\#P < 0.05$). B, Mean normalized TW showed no main effect for trial or time but a significant interaction showed TW to be significantly greater in OT4 than in PAS for Ex2 ($\#P < 0.05$).

set to 0.8 and provided $n = 8$ to be deemed suitable (G*Power v3.0.10 free software; Institute of Experimental Psychology, Heinrich Heine University, Dusseldorf, Germany).

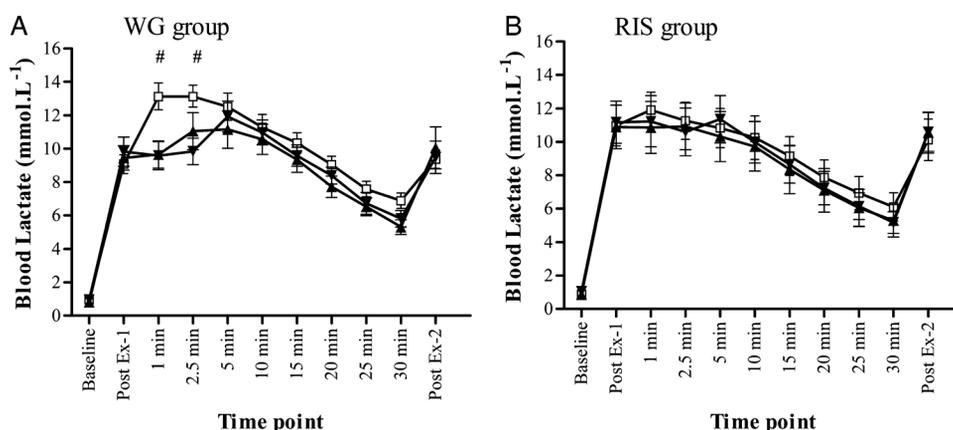


FIGURE 2—The exercise-induced increase in blood lactate and the subsequent clearance during recovery for PAS (□), OT1 (▼), and OT4 (▲). A, The WG group shows a continued increase in blood lactate during the initial recovery period in PAS that is absent in OT1 and OT4 ($\#P < 0.05$), after which no differences are observed between trials. B, The RIS group shows similar changes in blood lactate over time in all trials.

RESULTS

WG Group

Performance. Mean PP generated during the Wingate tests for Ex1 (i.e., before recovery) was not different between trials (for PAS = $12.8 \pm 0.3 \text{ W}\cdot\text{kg}^{-1}$) (Fig. 1A). However, PP for Ex2 was 8% greater in OT4 ($13.3 \pm 0.3 \text{ W}\cdot\text{kg}^{-1}$) than in PAS ($12.3 \pm 0.3 \text{ W}\cdot\text{kg}^{-1}$) ($P < 0.05$), whereas PP for Ex2 in the OT1 trial ($12.6 \pm 0.4 \text{ W}\cdot\text{kg}^{-1}$) was not different from that recorded in the PAS or OT4 trials for Ex2. This was despite no significant difference (i.e., no main effect) for PP between Ex1 and Ex2 in any trial.

TW was not different between trials (i.e., no main effect) for Ex1 (for PAS = $1.15 \pm 0.02 \text{ kJ}\cdot\text{kg}^{-1}$) or Ex2 (for PAS = $1.13 \pm 0.02 \text{ kJ}\cdot\text{kg}^{-1}$) (Fig. 1B). There was also no difference in TW in any trial from Ex1 to Ex2. However, a significant interaction ($P < 0.05$, trial \times time) suggests that TW declined from Ex1 to Ex2 in PAS and as such that TW was lower at Ex2 compared with OT4 ($1.16 \pm 0.02 \text{ kJ}\cdot\text{kg}^{-1}$). OT1 was not different from either PAS or OT4 at Ex1 or Ex2.

Blood lactate. Blood lactate increased similarly in all trials from rest (for PAS = $1.0 \pm 0.1 \text{ mmol}\cdot\text{L}^{-1}$) to after Ex1 (for PAS = $9.1 \pm 0.6 \text{ mmol}\cdot\text{L}^{-1}$) (Fig. 2A). However, during recovery, blood lactate continued to rise during the first 5 min in PAS ($t = 1 \text{ min}$, $13.2 \pm 0.8 \text{ mmol}\cdot\text{L}^{-1}$) to a level significantly greater when compared with both OT1 ($t = 1 \text{ min}$, $9.6 \pm 0.9 \text{ mmol}\cdot\text{L}^{-1}$) and OT4 ($t = 1 \text{ min}$, $9.7 \pm 0.8 \text{ mmol}\cdot\text{L}^{-1}$) ($P < 0.05$). For the remainder of the recovery period, blood lactate was not different between trials and, although continuing to decline, was still significantly elevated compared with baseline by the end of recovery (for PAS: $t = 30 \text{ min}$, $6.9 \pm 0.5 \text{ mmol}\cdot\text{L}^{-1}$) ($P < 0.05$). Blood lactate then increased again in all trials after Ex2 to a level similar to that for Ex1.

HR. WG group HR data are shown in Figure 3A, whereas Figure 4 shows a stylized representation of HR during recovery from exercise both with (solid line) and without (dashed line) contrast water immersion (Fig. 4 uses a 1:1 immersion ratio as the example). The change in HR from rest to after

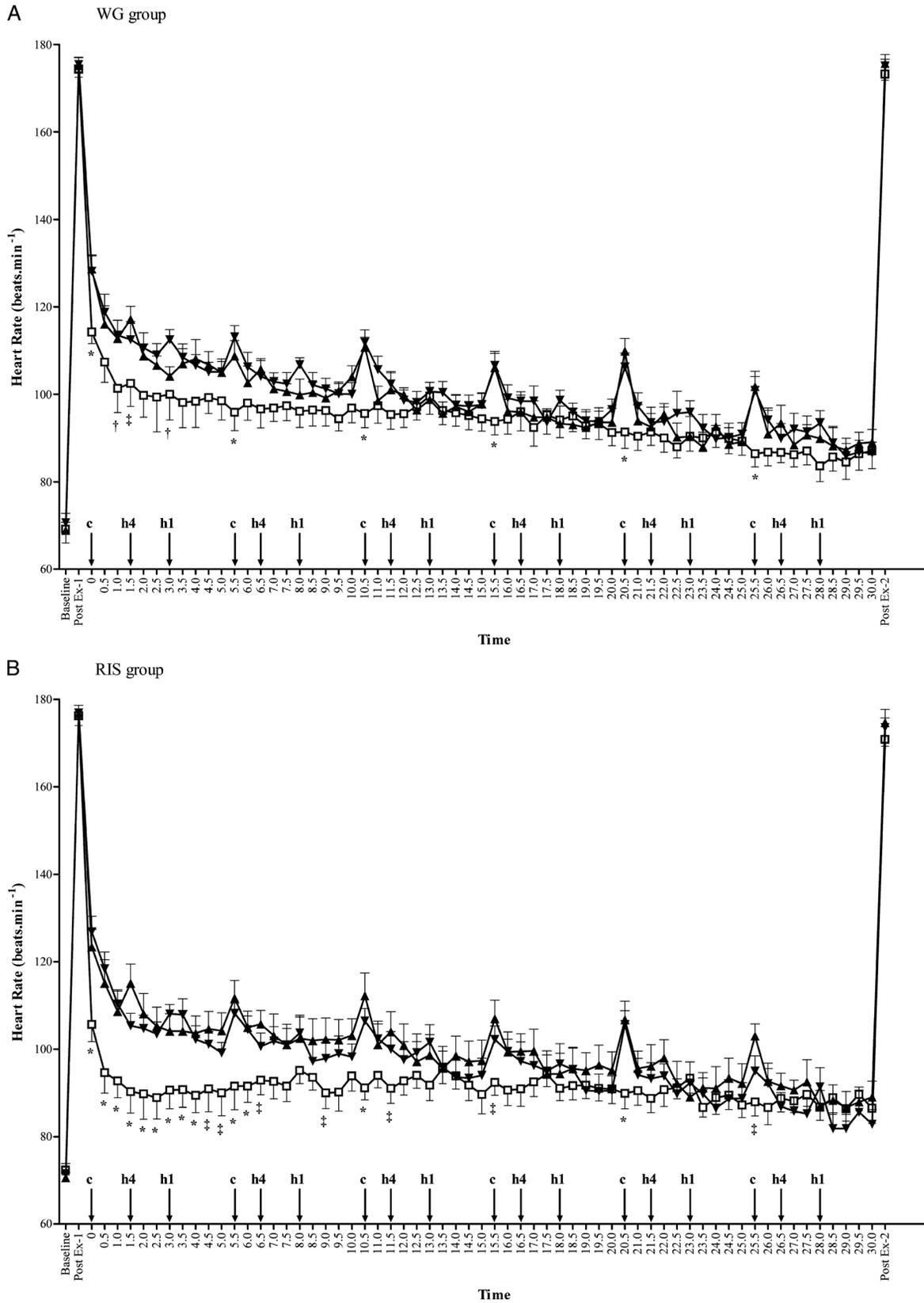


FIGURE 3—The exercise-induced increase in HR and subsequent decline during recovery for PAS (□), OT1 (▼), and OT4 (▲). The WG group (A) and the RIS group (B) show similar HR responses such that HR is maintained above that for PAS in OT1 and OT4 during the initial minutes of treatment during recovery. However, HR remains similar between groups for the remainder of the decline during recovery except after transitions to cold immersion in both OT1 and OT4 where HR increases briefly. Arrows indicate transition into cold (c), hot during the OT1 trial (h1), and hot during the OT4 trial (h4). **P* < 0.05 for PAS versus both OT1 and OT4. †*P* < 0.05 for PAS versus OT1 only. ‡*P* < 0.05 for PAS versus OT4 only.

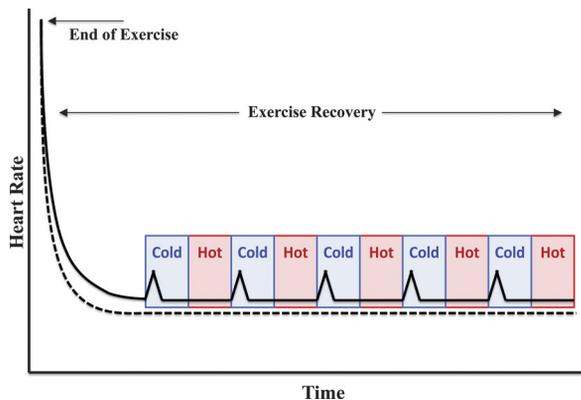


FIGURE 4—Stylized model of the recovery of HR when undertaking passive rest (*dashed line*) or when undertaking contrast water immersion (*solid line*; an example of a 1:1 immersion ratio is depicted). Note the significant increase in HR with immersion into each cold water phase (*shaded blue*), while each hot water phase does not affect the recovery of HR.

exercise was similar in all trials with no differences observed between trials (for PAS = 69 ± 3 to 174 ± 2 beats·min⁻¹). However, during recovery, HR remained elevated in OT1 and OT4 upon immersion in the initial cold phase of treatment during the immediate recovery period ($t = 0$) (Fig. 3A). During the next few minutes of the recovery period, HR then converged and became similar in all trials before declining slowly to the end of the recovery period but without returning to baseline levels. HR then increased again during Ex2 with these changes being similar between trials (for PAS = 87 ± 3 to 173 ± 1 beats·min⁻¹). It is clearly noticeable, though, that throughout the treatment period but particularly during the later slow decline, HR increased briefly with each cold immersion, while remaining unchanged with each hot immersion (Fig. 4). This was evident in both OT1 and OT4 when compared with PAS (Fig. 3A). Apart from the differences at these brief periods, at all other times from 5 min into the recovery until the end of the recovery period, HR was not different between trials.

RIS Group

Performance. Exercise time to failure for Ex1 was not different between trials (for PAS = 11.8 ± 0.4 min) (Fig. 5A). However, this declined by 11% for Ex2 after recovery in PAS (10.6 ± 0.4 min) ($P < 0.001$) but was maintained similar to Ex1 for OT1 (Ex2 = 11.7 ± 0.4 min) and OT4 (Ex2 = 11.6 ± 0.4 min). As such, exercise time for Ex2 was 10.4% and 9.3% greater in OT1 and OT4, respectively, when compared with PAS ($P < 0.001$).

Similar to exercise time to failure, TW for Ex1 was not different between trials (for PAS = 1.82 ± 0.1 kJ·kg⁻¹) (Fig. 5B). However, this declined by 15% for Ex2 after recovery in PAS (1.55 ± 0.1 kJ·kg⁻¹) ($P < 0.001$) but was maintained similar to Ex1 for OT1 (Ex2 = 1.78 ± 0.1 kJ·kg⁻¹) and OT4 (Ex2 = 1.76 ± 0.1 kJ·kg⁻¹). As such, TW for Ex2 was 14.8% and 13.5% greater in OT1 and OT4, respectively, when compared with PAS ($P < 0.001$).

Blood lactate. Blood lactate was not different between trials at any stage during exercise or recovery (Fig. 2B). However, a main effect for time ($P < 0.001$) showed blood lactate to increase similarly in all trials from rest (for PAS = 0.9 ± 0.1 mmol·L⁻¹) to after Ex1 (for PAS = 11.0 ± 1.2 mmol·L⁻¹) and then to decline progressively to the end of recovery without returning to baseline (for PAS at $t = 30$, 6.1 ± 0.9 mmol·L⁻¹), before increasing again after Ex2 (for PAS = 10.1 ± 1.2 mmol·L⁻¹).

HR. RIS group HR data are shown in Figure 3B, whereas again, Figure 4 presents a model of the effect of contrast water immersion (solid line) in comparison with passive rest (dashed line) on the recovery of HR after exercise. The change in HR was again similar in all trials from rest to after exercise, with no differences observed between trials (for PAS = 72 ± 1 to 176 ± 2 beats·min⁻¹). However, during recovery, HR remained elevated in OT1 and OT4 upon the initial cold immersion phase of treatment during the immediate

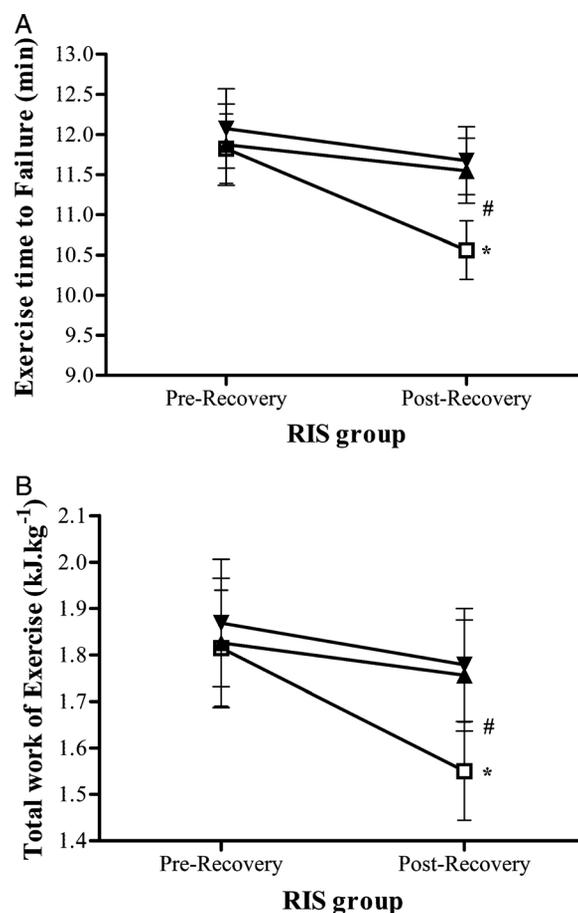


FIGURE 5—RIS group performance data for the PAS (□), OT1 (▼), and OT4 (▲) trials. A, Exercise time was maintained in OT1 and OT4 between Ex1 and Ex2 but not in PAS. Exercise time in PAS declined from Ex1 to Ex2 to a level that was also significantly lower than that for OT1 and OT4. B, Mean normalized TW was maintained in OT1 and OT4 between Ex1 and Ex2 but not in PAS. TW done in PAS declined from Ex1 to Ex2 to a level that was also significantly lower than that for OT1 and OT4. * $P < 0.001$ between Ex1 and Ex2 for PAS. # $P < 0.001$ between PAS and both OT1 and OT4 for Ex2.

rapid recovery period ($t = 0$) (Fig. 3B). These differences were generally maintained until at least 5 min into the treatment period during recovery, after which HR became similar between trials before declining slowly to the end of the recovery period but without returning to baseline levels. HR then increased again during Ex2 with these changes being similar between trials (for PAS = 87 ± 3 to 171 ± 2 beats·min⁻¹). Again, it is noticeable that throughout the treatment period and, in particular, the later slow declining phase of the recovery, HR increased briefly with almost every cold immersion (Fig. 4). This is evident in OT4 for all cold phases and for most of these phases in OT1 when compared with PAS (Fig. 3B). HR again was unaffected by each hot immersion phase.

DISCUSSION

The present study used a counterbalanced randomized crossover design to investigate the effect of contrast water immersion undertaken for the entire duration of a short-term (30 min) recovery period after high-intensity sprint cycling on subsequent exercise performance, the clearance of blood lactate, and recovery of HR. The major finding was that subsequent high-intensity sprint cycling performance was superior after a recovery comprising contrast water immersion in comparison with a recovery comprising passive rest and that this superior performance was independent of other complicating influences such as an active warm-down/recovery, stretching, or nutritional practices that may be typical during a longer recovery period.

This effect of contrast water immersion was more clearly demonstrated in the RIS group where performance after recovery in both OT1 and OT4 was approximately 10% greater than that for passive rest, and this superior performance was similar for both ratios of cold-hot contrast water immersion, that is, for OT1 and OT4. For the WG group, however, when compared with PAS, this effect of contrast water immersion during recovery on subsequent performance was also apparent, but only for the OT4 trial (~8%). Despite the similarity between the groups for total exercise time (RIS = ~12 min, WG = 13.5 min), the apparently more consistent effect of contrast water immersion independent of the ratio used during the application in RIS may be explained by the difference between the exercise types in each group where the RIS group completed exercise to exhaustion and therefore ~60% greater TW than the WG group (RIS = ~ 1.85 kJ·kg⁻¹, WG ~ 1.15 kJ·kg⁻¹).

In addition to being one of only a few studies to investigate contrast water immersion on subsequent exercise performance (10,13,19,34,36), we believe this is the first study to compare the effects of different contrast water immersion ratios implemented for recovery from exercise. From these data, OT4 seemed marginally more effective in restoring performance after recovery. As such, one might be tempted to suggest that for high-intensity exercise performance after a short-term recovery period whereby contrast water im-

mersion is undertaken, implementing a low ratio of cold-hot contrast water immersion (in the case of the present study, 1:4) would provide a greater benefit to recovery of performance than a cold-hot contrast water immersion ratio that approaches 1:1. However, such a generalized approach to implementation of contrast water immersion would seem premature given that there is no other research that has attempted to identify the most appropriate contrast water immersion ratio for recovery of performance. Moreover, the effect on performance is probably also dependent on the time of immersion and absolute temperature of immersion during each phase, as well as the total treatment time during recovery, none of which seem to have been previously examined in a systematic manner.

In the present study, the treatment ratios were determined from the absolute period of the cold water immersion phase, with the hot water immersion phase comprising the balance of each 5-min immersion period (80% hot for OT4, 50% hot for OT1). Consequently, the absolute immersion times for the cold phase and, therefore, the hot phase, were different between trials. It is unknown whether similar treatment ratios implemented with different base cold water immersion periods, for example, from 30 s up to 2, 3, or even 5 min, are more or less effective. However, the limited research investigating contrast water immersion has only investigated cold immersion durations between 1 and 2 min as part of contrast treatments using ratios from 1:1 to 1:4 (13,16,19,23,27). Moreover, the longer the base immersion period for a particular treatment ratio, the fewer the transitions that may be possible within the overall time available for recovery. Because it is these transitions that are speculated to be responsible for driving the physiological effects that result in the observed performance effect, it would seem that the focus of contrast water immersion research should in part be to optimize the methodology that produces the greatest benefit from the recovery, and this seems more likely with transitions occurring more regularly and most probably at the very least every 5–6 min (12,40).

It has been speculated that contrast water immersion provides a “pumping effect” on the vascular system despite little evidence to support such effects under passive conditions in the treatment of injury or during recovery from exercise (9,17,40). In the present study, there is some evidence for an effect on cardiac function with a significant response of HR observed throughout the course of treatment in both OT1 and OT4, whereby HR increased on each transition to cold immersion when compared with PAS (Fig. 4). This is supported by similar data examining HR recovery and parasympathetic function after exercise, but only after a single 5-min phase of cold water immersion (6,7). However, contrast water immersion has been shown to cause a fluctuation in limb blood flow (12) as well as skin blood flow (27), but these data are again at rest, and not after exercise. One recent study did investigate limb blood flow after exercise but, as easily predicted, showed only that an active recovery maintained a greater limb blood flow than when resting during a cold water immersion (35). A

comparison with a passive resting control was not performed, and therefore, this study provides little value in understanding the cardiovascular effect of water immersion during recovery, or indeed any physiological effect. As such, the present study is unique in showing evidence of an effect of contrast water immersion on cardiovascular function not only during recovery in the early rapid period of recovery from exercise but also during the later slow declining phase of recovery from exercise that is more likely to provide a cardiovascular effect on previously active muscle. However, this study does not provide an explanation as to how this may affect subsequent sprint performance, and so, further work is required to more fully examine the cardiovascular changes experienced during water immersion, such as cardiac output, stroke volume, and blood pressure, as well as blood flow to muscle and skin.

Blood lactate concentration does not seem to shed light on the observed improvement in performance in the present study. Only during the initial stages of immersion in cold during both OT1 and OT4 in the WG group was blood lactate different from PAS. In this case, it was significantly lower. However, it is difficult to reconcile this being the reason for the improvement in performance. Other studies have shown blood lactate to be cleared more effectively when using water immersion during recovery (13,22), but this research has been unable to clearly demonstrate that this provides a benefit to performance. In addition, no clear mechanism has been suggested by which water immersion may induce a more rapid clearance of blood lactate. For an active recovery, however, it is well accepted that persistent low-intensity activity primarily increases lactate clearance by maintaining muscle blood flow to allow continued efflux of lactate from active muscle in conjunction with uptake by inactive muscle, whereas there remains potential for other sites capable of removing significant amounts of lactate from the circulation such as the liver (2,3). Although contrast water immersion is a passive mode of recovery, any changes in blood lactate clearance must stem from the application of a temperature stimulus to the skin, from the added hydrostatic pressure, or from a combination of both. Although we show an effect on HR during transitions into cold immersion and this likely suggests the response is via a change in temperature in the skin, there is no evidence in the present study or otherwise that this translates to an effect on blood flow and, subsequently, lactate clearance. Further investigation is therefore warranted, to examine the effect of water immersion on skin, muscle, and core temperatures, as well as muscle and systemic blood flow.

One limitation of the present study was that during the PAS trial, subjects were not required to move from one empty bath to another to mimic the “activity” associated with changing

baths that was present in the OT1 and OT4 trials. However, it does not seem that this contributed to the small effect on blood lactate in the early stages of the recovery because the persistent activity from the changing of baths in OT1 and OT4 lasted the entire duration of the recovery. Similarly, this activity was nonvigorous and short lived, comprising <1 min in total of the 30 min of water immersion, and, therefore, is unlikely to play a role in the development of the observed performance effect. It is acknowledged, however, that there is difficulty in selecting conditions for the control trial (PAS). The present study used the same seated position to that of the immersion treatments but in an empty bath at a standard laboratory room temperature ($21^{\circ}\text{C} \pm 2^{\circ}\text{C}$). Although these conditions may have contributed to a more clear observation of the effects of the water immersion responses to the application of both a temperature stimulus and a hydrostatic pressure stimulus, future research should consider investigating other control conditions such as immersion in water at either room temperature or a thermoneutral temperature.

Similarly, the data from the present study do not seem to be related to other metabolic factors such as glycogen depletion or muscle damage given that the exercise is of short duration despite being of high intensity (14) and is expected to be nondamaging because it comprised concentric cycling exercise in accustomed subjects involved in regular exercise training.

In conclusion, we have shown that contrast water immersion undertaken after high-intensity sprint exercise results in an improved maintenance of exercise performance compared with passive rest. This is likely a result of the effect of alternating cold and hot water immersion on cardiovascular function where we provide good evidence of an HR response with the transition into each cold water immersion phase during the recovery. These data are not complicated by other recovery practices given that the experimental protocol examined exercise performance immediately either side of the recovery treatment. This result provides support to the mechanism that water immersion affects cardiovascular function when used during recovery from exercise, which has previously only been speculated. As such, further investigation is warranted to explore cardiac and vascular function during water immersion in recovery from exercise.

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