

Research article

Effects of In-Water Passive Recovery on Sprint Swimming Performance and Heart Rate in Adolescent Swimmers

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Abstract

The aim of the present study is to test the hypothesis that sprint swimming performance is enhanced by in-water passive recovery (IN) after sprint swimming bouts in well-trained adolescent swimmers. Using a randomized crossover study design, twelve well-trained adolescent swimmers performed two tests at the swimming pool after preliminary testing. They performed 5 bouts of 100m all-out swimming separated by 5 minutes of passive rest. Their individual in- or out-of-water passive recovery condition was randomized on the first day. In their second visit to the swimming pool the opposite recovery condition was indicated. More than 60% of the subjects which rested in-water were faster in the 5th bout when compared to the OUT group. However, no significant differences were found in blood lactate when IN and OUT were compared. After the first bout peak heart rate (HR peak) was lower in subsequent bouts for IN recovery when compared with OUT ($p < 0.001$). Thus, coaches and researchers should take into account that IN passive recovery may decrease loss of performance and diminish HR peak during sprint swimming bouts. This is particularly important given the use that many coaches give to HR as a tool in daily training.

Key words: Swimmers, test, sprint, recovery.

Introduction

High-intensity exercise describes physical exercise that is characterized by brief, intermittent bursts of vigorous activity, interspersed by periods of rest or low-intensity exercise. It is an effective alternative to traditional endurance training to improve health-related markers in both healthy individuals and diseased populations (Gibala et al., 2012). The success of high-intensity training in sports resides in its efficiency. There is strong evidence that sprint training increases exercise performance (Bangsbo et al., 2009; Iaia et al., 2008; 2009), and that it also maintains (Iaia et al., 2009) or even increases (Bangsbo et al., 2009) muscle oxidative capacity.

By reducing volume and increasing intensity, well-trained runners (Laursen and Jenkins, 2002; Smith et al., 2003), cyclists (Laursen and Jenkins, 2002; Stepto et al., 1999; Weston et al., 1997), and soccer players (Thomassen et al., 2010) increase their performance. However, little is known about swimming. One study shows that well trained swimmers maintain swimming speed after training for four weeks at an intensity around the lactate threshold (Faude et al., 2008). More recently, speed training has

been proposed for elite swimmers in order to increase training efficiency (Kilen et al., 2014).

Swimming is performed as an under-water dynamic activity, which may influence exercise adaptations and acute responses. Water immersion induces hydrostatic pressure, which can cause the displacement of fluids within a person from the extremities towards the central cavity. This displacement of fluids increases the translocation of substrates from the muscles, increases cardiac output, reduces peripheral resistance, and increases the ability of the body to transport substrates (Wilcock et al., 2006). Water immersion per se may actually play an important role in muscle recovery after exercise, since hot water immersion is able to restore muscle mechanical function after exercise (Vaile et al., 2008).

In-water (IN) passive recovery increased performance during a set of 6 repetitions of a 50 m swimming sprint every 120 s when compared with out-of-water (OUT) recovery (Buchheit, 2010). This effect was associated with a lower heart rate peak and lower blood lactate in adult swimmers (Buchheit, 2010). Swimmers are often young when they reach elite level competition, with peak freestyle speed achieved between the ages of 21 and 23 (Rüst et al., 2014). It must be also highlighted that among the swimming medalists in the 2012 Olympics ($n = 78$), 25 were under 21. Little is known about the incidence of sprint swimming on adolescent swimmers. Adolescents produce lower lactate levels at a given intensity (Tolfrey and Armstrong, 1995), which may alter the effects that IN recovery has on performance and lactate production.

Buchheit et al. (2010) tested 50m sprint bouts. However, final turns during a 100 m and 200 m are performed under fatigue, and sprint swimming training should take care of the specific needs of the swimming race (Maglischo, 2003). Thus, the 100 m swimming distance could be more beneficial for well-trained swimmers in helping fulfill the physiological needs of swimming races. In this regard, cold water (16°C) immersion does not enhance 100 m swimming sprint performance despite a greater perception of recovery (Parouty et al., 2010). However, swimmers usually train at a temperature of 26-28°C. By doing recovery at this temperature re-heating is not necessary, as shown by Parouty et al. (2010) after immersion in 16°C water, but it may retain the beneficial effects of the hydrostatic pressure.

The aim of the present study was to test the hypothesis that 5 minutes of IN recovery, in thermoneutral water, will increase performance relative to OUT during a

5 x 100 m sprint swim. The secondary hypothesis is that IN recovery will decrease lactate production and peak heart rate (peak HR).

Methods

Participants

Twelve well-trained adolescent swimmers participated in the study. Their mean (\pm SD) age, height, and body mass were 15 ± 0.8 years, 1.75 ± 0.05 m, and 72.0 ± 5.5 kg, respectively. All swimmers had competed in regional and national championships in the previous twelve months, and were experienced in 100 m front crawl sets such as the one used in this study. They normally trained ten to twelve hours per week. Swimmers were informed about the purpose of the study and any known risks, and then gave their written consent to participate. The Ethical Advisory Committee of the University of Jaén (Spain) approved the test protocols and all procedures. All experimental work conforms to requirements stipulated in the Declaration of Helsinki.

Study design

The study employed a randomized crossover design where each subject completed two 5 x 100 m all-out swimming separated by at least 48 h and a maximum of 5 days. The study was carried out in a 25 m indoor swimming pool, environmental temperature was 30 ± 0.5 °C and water temperature was 26.7 ± 0.3 °C. The protocol applied during these two testing days was the same with the exemption of recovery between bouts. The 5 x 100 all-out protocol was carried out by swimming front crawl style interspersed by 5 minutes of in- or out-of-water passive recovery. First day IN or OUT condition was chosen randomly for each swimmer, and the second day the opposite recovery condition was applied. The reason why they were instructed to take 5 minutes of rest is that in order to achieve adaptations in response to sprint training a recovery period of 4 to 6 times the duration of the exercise is needed (Iaia and Bangsbo, 2010). A longer period of rest might have magnified the effect of hydrostatic pressure.

Preliminary tests

Two weeks before the start of the study fifteen swimmers from local swimming clubs were recruited. In order to avoid any learn effect the swimmers performed the 5 x 100 m test at least once before the start of the study without attending to the recovery condition. Over the five repetitions, the fastest (RSb) and mean (RSm) times were recorded. Fatigue was assessed during the protocol as a percentage of sprint speed decrement which was calculated as follows: $100 - ([Rsm/RSb] \times 100)$. This method is thought to provide a reliable method for assessing fatigue during all-out bouts (Glaister et al., 2004). After this preliminary test 3 subjects were eliminated because their fatigue was higher than 7%. These subjects had not performed regular training during the 3 weeks prior to testing, because they were in the transition phase between two macrocycles.

Testing procedures

The day before testing, swimmers performed a low-intensity warm-up (~1000 m). On the day of testing swimmers reported having abstained from caffeine intake. They were suggested to take notes on their food intake in order to be able to repeat the same food pattern on the second experimental day. In the experimental sessions, swimmers performed a standardized warm-up (200 m free swim, 200 m kick, 200 m pull, 200 m medley, 8 x 25 m progressive swim, 100 m easy swim). After 10 minutes of rest, swimmers performed 5 repetitions of maximal 100 m swimming bouts separated by 5 minutes of recovery. All tests were performed at the same time. Every swimming bout started from inside the pool in order to avoid differences in start performance. Participants were encouraged to perform each repetition as fast as possible. During both IN and OUT recovery swimmers remained passively with water at the chest level (IN) or seating on a chair out of the water (OUT). Swimmers were asked to assume the ready position 10 to 5 prior to the start signal. During swimming bouts heart rate (HR) was continuously recorded.

Blood lactate

Capilar blood lactate (5 μ L) was measured from the fingers of swimmers in the 2nd and ~5th minute within the 1st, 3rd, and 5th recovery period (Lactate Pro, Japan). The measurement at minute 2 was chosen because we found a lactate peak two minutes after the all-out swimming in the preliminary tests. The measurement at minute 5 was chosen in order to find out the levels of capilar lactate before starting the next swimming bout. In addition, the difference between lactate at 2' and 5' provided information about blood lactate removal.

Heart Rate

A Polar S810 HR monitor (Polar Electro, Kempele, Finland) was used to record heart rate (HR). During the protocol, the band was wrapped around the chest of swimmers to allow continuous recording. Higher HR achieved after/during each swimming bout was considered as HR peak, and the lowest HR recorded during the rest period was considered as minimal HR. HR peak was located during the last 10 seconds of the sprint and the first 10 seconds of the recovery. Chlorine is known to negatively affect HR measurement (Polar Electro, personal communication). Thus, during the experimental tests water chlorine was reduced (0.6 - 0.9 mg/L) in order to allow a reliable HR measure. These measurements were checked at preliminary testing.

Statistical analysis

Results are presented as mean \pm SD. Homoscedasticity and normality were tested by Levene and Kolmogorov-Smirnov tests respectively. Results were analyzed using two-factor 2(group: OUT vs IN) x 5(time) analysis of variance (ANOVA) with repeated measures to assess time, blood lactate and HR. For each variable, P value was calculated for group (between-subjects), time (within-subjects) and interaction (group x time) effect. We calculated the P value for within-group differences by group when a significant interaction was present. Multiple

comparisons were adjusted by Bonferroni's correction. Specific differences between groups were located using Student's t-test for independent samples. Size effect was also calculated for time and blood lactate using Hedges's *g*, since it provides a better estimate when small samples are used (Tejero-Gonzalez et al., 2012). In addition, probability value is also shown (in brackets) using the standard normal distribution table (Vicent, 2005). Pearson's correlational analysis was performed in order to assess a possible relation between time taken in each 100 m bout, blood lactate, and fatigue development index. The level of significance was considered at $P < 0.05$. All the analyses were performed using the Statistical Package for Social Sciences (SPSS, version 19.0 for Windows; SPSS, Inc., Chicago, IL, USA).

Results

The Kolmogorov-Smirnov test showed that all data presented a normal distribution and the Levene test showed an equality of variances. Results from the ANOVA 2(group) x 5(time) analysis performed on swimming performance showed a time main effect ($p < 0.001$, $F(4,88) = 20.546$, $\text{Eta}^2 = 0.483$, $1-\beta = 1.000$) but no differences were found in any bout when IN and OUT recovery are compared (Figure 1). Post-hoc time main effect analysis revealed, however, a significant increased time in bout 3 ($p = 0.008$), bout 4, and bout 5 ($p < 0.001$), when compared to the first bout. Although no differences were found ($p > 0.05$; IN vs OUT) using Student's t-test, Hedges's *g* analysis showed that in B3, B4, and B5 more than 60% of the subjects which rested IN-water were faster when compared to OUT (Figure 1). In addition, % fatigue was similar between recovery conditions (IN -2.46 ± 1.21 ; OUT -2.88 ± 1.74 ; $p > 0.05$). It is important to note that no differences were found in each 25 m partial time or in the stroke rate between recovery conditions.

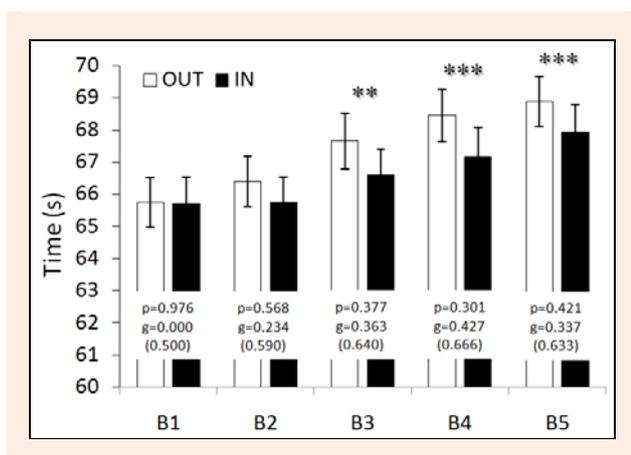


Figure 1. Time analysis during 5 x 100 m all-out swimming. Results are presented as mean \pm SD. Analysis from the ANOVA 2(group) x 5(time) showed a significant main effect for time by increasing swimming time in the 3rd, 4th, and 5th repetitions for both IN and OUT conditions. **($p = 0.01$) and ***($p = 0.001$) relative to the first swimming bout (B1). *G* = Hedges's *g* analysis. Probability is presented in brackets.

Regarding capilar blood lactate, the results of the ANOVA analysis showed a significant time main effect

($p < 0.001$, $F(5,110) = 91.355$, $\text{Eta}^2 = 0.806$, $1-\beta = 1.000$; (Figure 2). Post hoc analysis revealed higher blood lactate levels in both the IN and OUT conditions after the 3rd and 5th repetition, compared to the first swimming bout ($p < 0.001$). There was no significant main effect for group ($p = 0.568$) or for group x time interaction ($p = 0.630$). No lactate removal (difference between the 2nd and the 5th minute) was found within 5 minutes of recovery at any point ($p > 0.05$). Hedges's *g* showed that IN recovery maintained or decreased blood lactate at the 3rd bout (5' recovery) in 72% of the subjects and at the 5th bout (2' recovery) in 61% of the subjects (Figure 2).

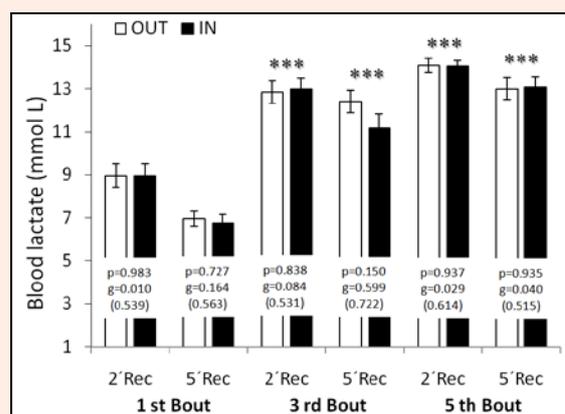


Figure 2. Blood lactate (mmol/L) after the 1st, 3rd, and 5th bouts. It was measured at the 2nd and 5th minute of rest. No differences were found when IN and OUT conditions were compared. ***($p = 0.001$) relative to the first swimming bout. *G* = Hedges's *g* analysis. Probability is presented in brackets.

HR rate was continuously monitored during the exercise. Results derived from the ANOVA analysis on maximal and minimal HR are presented in Figure 3. HR peak data showed a main group effect ($p < 0.002$, $F(1,21) = 12.56$, $\text{Eta}^2 = 0.374$, $1-\beta = 0.922$), but also a main time effect ($p < 0.004$, $F(4,84) = 4.125$, $\text{Eta}^2 = 0.164$, $1-\beta = 0.904$) and a significant group x time interaction ($p < 0.023$, $F(4,84) = 3.003$, $\text{Eta}^2 = 0.125$, $1-\beta = 0.777$). The group x time interaction post hoc analysis revealed a higher HR peak within OUT recovery in the 2nd ($p < 0.001$), 3rd ($p < 0.001$), 4th ($p < 0.001$), and 5th ($p < 0.001$) swimming bouts when compared with IN recovery. On the other hand, significant time main effect was found in minimal HR ($p < 0.001$, $F(4,88) = 22.620$, $\text{Eta}^2 = 0.507$, $1-\beta = 1.000$) but no significant group x time interaction was found when IN and OUT recovery were compared ($p > 0.05$).

Pearson's correlational analysis showed no significant correlation between time taken during each swimming bout and any of the other variables measured, whether in IN or OUT conditions ($p < 0.05$). No significant correlation was apparent when blood lactate was compared with each variable (data not shown).

Discussion

A randomized crossover study design was used to compare 5 minutes of in- vs out-of-water recovery between 5 bouts of 100 m sprint swimming on the performance and

physiological responses of adolescent swimmers. The key finding is that IN recovery is associated with a lower HR peak, although there is no change in minimal HR when compared with OUT recovery. Despite no statistically significant effects, Hedges's *g* analysis showed that IN-water recovery had a lower decrease in performance and lactate concentration was lower in bout 3.

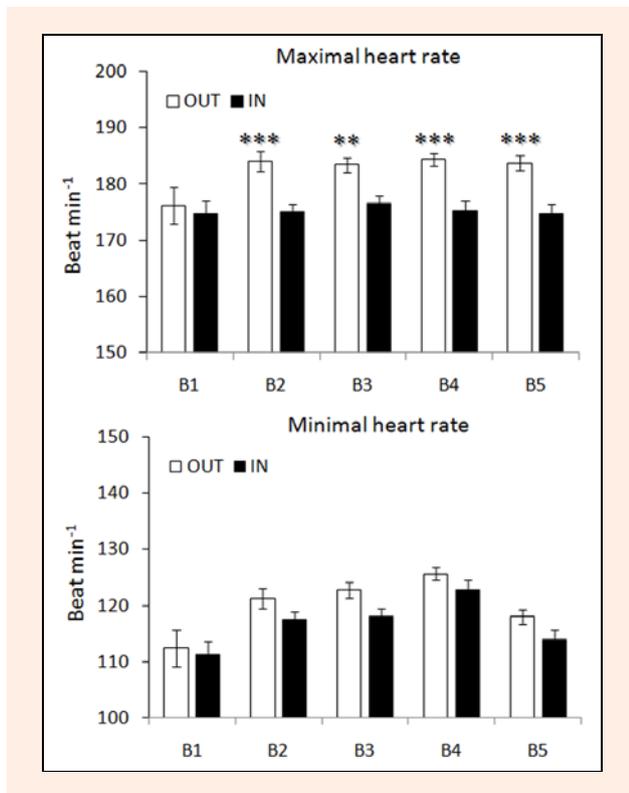


Figure 3. Heart rate data. Peak heart rate was considered as the higher recorded during the last 10 seconds of the swimming bout and the first 10 seconds of the recovery period. Minimal heart rate was the lower heart rate during the rest period. Maximal heart was significantly lower within IN recovery in the 2nd, 3rd, 4th, and 5th swimming bouts. No differences were found when looking at minimal heart rate. *** ($p < 0.001$) and ** ($p < 0.01$) when comparing the IN and OUT conditions.

It appears that athletes in sports involving intense exercise may benefit from periodically reducing the amount of work load and undertaking speed training on a regular basis (Bangsbo et al., 2009; Iaia et al., 2008; 2009; Laursen and Jenkins, 2002; Thomassen et al., 2010). Sprint training consists of maximal all-out bouts separated by rest periods of 4-6 times the duration of the exercise (Iaia and Bangsbo, 2010). It is a time-efficient strategy for markedly improving performance using very intense short-term exercise (from 30 seconds to 3 min; Iaia and Bangsbo, 2010).

There is a net decrease in blood lactate production which occurs with time. This may suggest an inhibition of glycolysis and explain the loss in sprint performance in the 3rd, 4th, and 5th bouts (Bishop, 2012). Thus, this kind of swimming training may be beneficial for swimming races in which glycolysis is compromised. The novelty of our study is that it applies to adolescent swimmers: at a given intensity adolescents show lower blood-lactate levels than adults (Tolfrey and Armstrong, 1995) as the 3rd decade of life (20-30 years of age) is thought to be the

age at which the lactate generated in response to exercise is higher (Beneke et al., 2005). Our data show a maximal blood lactate of 14.0 ± 1.1 for OUT and 14.0 ± 0.9 for IN. Compared with another study that used a set of 6 repetitions of 50 m bouts, and which produced 13.3 ± 1.6 for IN and 13.3 ± 0.8 for OUT (Buchheit et al., 2010), glycolysis is further stimulated using 100 m swimming bouts.

The 100 m sprint swimming performance in response to water immersion has been previously assessed. The authors tested the effect of cold water (14-15°C) immersion on swimming performance, and despite a greater perception of recovery there was a reduced sprint performance with a greater pre-race parasympathetic activation (Parouty et al., 2010). This might be due to the fact that a strong rewarm-up is needed after cold water immersion in order to increase exercise performance (Crowe et al., 2007; Pieffer et al., 2010; Schiniep et al., 2002). In spite of no statistically significant differences due to the sample size, in the last 3 bouts of 100 m more than 60% of the subjects which rested in-water were faster when compared to those who rested out-of-water. Thus, thermoneutral water immersion may have the beneficial effects of cold water immersion but no rewarm-up is needed to achieve them. Actually, our results show a lower HR peak for the in-water recovery group, similarly to the vascular response in adult swimmers (Buchheit et al., 2010). This effect also appears in swimmers after cold-water immersion (Parouty et al., 2010).

Immersion has several consequences on body physiology. In the present study swimmers in the IN condition rested in the pool, in thermoneutral temperature (26°C; Wilcock, 2006). In this situation water exerts a compressive force on the body: the hydrostatic pressure. During head-out immersion, hydrostatic pressure on the central cavity reduces the residual air volume of the lungs, and increases the displacement of fluids into the thorax (Farhi and Linnarsson, 1977). The lower HR peak in response to IN recovery may be a response from water immersion per se, as it is known that head-out water immersion decreases HR by increasing stroke volume (Arborelius et al., 1972; Johansen et al., 1997; Park et al., 1999; Yun et al., 2004). Thus, the lower HR peak in the IN condition may increase stroke volume and imply a higher economic state, thus explaining that in B3, B4, and B5 60% of the subjects decreased their loss of performance. In addition, immersion to the neck decreases muscular vascular resistance which in turn increases blood flow by 49% (Gabrielsen et al., 2000). Greater muscle blood flow may allow improved removal of metabolites and an increased ability to replenish energy stores. In fact, Hedges's *g* analysis showed that only after the 3rd bout lactate was lower in 72% of the subjects after 5 minutes of recovery. As it is well known that water immersion increases blood lactate removal (Coffey et al., 2004; Nakamura et al., 1996), we suggest that in spite of vascular effects in response to IN recovery (i.e. HR peak), 5-minute immersions in thermoneutral water between exercise bouts might not be enough to induce lactate removal.

In-water recovery between bouts was performed in a standing position, with head out of the water while OUT of water recovery was performed seating on a chair. Thus,

position recovery may have an effect on the comparison of IN and OUT. The differences induced by body posture on heart rate are mainly due to gravitational effects. When standing upright, blood from the central nervous system is shifted to the lower extremities, and the sympathetic system is activated for the preservation of arterial blood pressure (Takashi et al., 2000). Nevertheless, no significant differences are found when comparing cardiovascular response and parasympathetic activation in response to seating and standing recovery after exercise (Buchheit et al., 2010b). Furthermore, the gravitational effect associated with the standing position is unlikely to affect in-water recovery because of the reduction of gravitational forces due to buoyancy.

Several limitations of the present study must be mentioned, first of all, our sample does not include elite adolescent swimmers, so we do not know if the effects showed in the present study will be the same in a more trained sample. In addition, although it is very unlikely that the effects showed within IN recovery can be due to a lower core temperature, we cannot exclude this possibility because data on core temperature (i.e. rectal temperature) is not included in the present study.

Conclusion

In conclusion, in-water recovery minimizes the loss in swimming performance, probably by increasing blood flow. In fact, HR peak was lower in the IN condition. This might explain why 72% of the subjects showed lower blood lactate levels within IN recovery only in bout 3, allowing enhanced metabolite removal by increasing muscle-blood flow. This might be relevant for swimming coaches, since HR is commonly used in daily training in order to control exercise intensity.

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Key points

- In-water passive recovery minimizes the loss of performance during high intensity swimming
- Maximal HR is significantly reduced by in-water recovery
- Coaches should take this information into account when using HR to control swimming intensity
- Future research should study long-term effects induced by in-water passive recovery

AUTHORS BIOGRAPHY

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Employment

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