The Interactive Effects of Recovery Mode and Duration on Subsequent Repeated Sprint Performance

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Abstract

Brown, J and Glaister, M. The interactive effects of recovery mode and duration on subsequent repeated sprint performance. *J Strength Cond Res* 28(3): 651–660, 2014—The aim of this study was to examine the interactive effects of recovery mode and duration on subsequent repeated short sprint (RSS) performance. Ten male recreational athletes (age, 27.9 ± 5.0 years; height, 1.80 ± 0.07 m; mass, 81.6 ± 13.5 kg) performed 4 randomized trials consisting of a 30-second cycle sprint, followed by a specified recovery period (45 or 180 seconds), and a subsequent set of RSS (7 × 5 seconds, 20-second passive rest periods). Recovery mode was either active (AR; 70% of the power output at lactate threshold) or passive (PR). Mean heart rate and $\dot{V}O_2$ were significantly higher ($p \leq 0.05$) in AR than in PR over both recovery durations. Although the difference in $\dot{V}O_2$ reached significance after 10–15 seconds, a significant ($p \leq 0.05$) difference in heart rate was observed only after 26 seconds (45-second trials) – 75 seconds (180-second trials). Blood lactate was significantly ($p \leq 0.05$) lower in AR than in PR only after 135 seconds (mean difference, 2.16 mmol·L^{-1}; 95% likely range, 0.77–3.55 mmol·L^{-1}). Mean peak power output in the RSS test was significantly ($p \leq 0.05$) higher following PR_{45} than AR_{45} (12.0 ± 1.4 vs. 11.4 ± 1.4 W·kg^{-1}) and following AR_{180} than PR_{180} (12.7 ± 1.2 vs. 12.0 ± 1.2 W·kg^{-1}). In conclusion, when rest periods are short, a PR strategy appears to optimize subsequent RSS performance. However, as the recovery duration increases subsequent RSS performance appears to benefit from an AR strategy.

Key Words repeated sprint ability, intermittent, active, passive

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Introduction

The ability to perform repeated short (2–5 seconds) sprints (RSS) is integral to many sporting activities (17,30). In field sports, such as soccer, rugby, and hockey, the most intense periods of play are characterized by sets of up to 7 RSS, with short (≤21 seconds) recovery periods between sprints (22,30). These intense periods typically occur at critical points during the game, such as when the try-line or goal is being attacked, and are separated by periods of lower-intensity activity, the duration of which is variable (22). The ability to maintain optimal performance during repeated maximal-intensity bouts with varying recovery periods is, therefore, an important component of performance in field-based sports.

The degree to which optimal performance can be maintained throughout successive maximal efforts is dependent on the ability to recover between those efforts (5,17,18). For example, increasing the duration of the recovery periods enables a greater maintenance of RSS performance (18,30). It has also been suggested that performing low-intensity exercise may accelerate recovery from previous bouts and, therefore, enhance subsequent performance (6,29). Although several studies have demonstrated that an active recovery (AR) strategy can improve subsequent maximal-intensity performance (1,10,13,29,32), others have found passive recovery (PR) to be the most beneficial (8,14,31). Although, superficially, such observations appear contradictory, the findings may, in fact, reflect an inherent variability in the optimal recovery mode, which is dependent on the energetic demands of the protocol used.

The decline in power output (PO) typically observed during RSS is primarily associated with skeletal muscle fatigue, which in turn is thought to result from a progressive reduction in the ability of the active muscle to resynthesize adenosine triphosphate at the required rate (5,17). During a single sprint lasting 3–6 seconds, ATP resynthesis is due almost entirely to phosphocreatine (PCr) degradation (~50%) and glycolysis (~40%) (5,17,30). However, when a set of RSS is performed, glycolytic flux is progressively retarded, and the relative contribution from PCr increases (5,17,30). Indeed, the degree to which peak power

output (PPO) can be achieved in the initial seconds of successive sprints has been found to be highly correlated ($r = 0.84$, $p \leq 0.05$) with the restoration of intramuscular [PCr] (6). Moreover, because the recovery kinetics of PPO are very similar to those of PCr, this relationship is thought to be causal (5,6,23). It is therefore likely that a recovery strategy that is able to facilitate intramuscular PCr resynthesis will have the greatest benefit for subsequent RSS performance.

After high-intensity exercise, the resynthesis of intramuscular PCr displays a biexponential recovery pattern (15,17,23), the initial phase of which is rapid ($t_{1/2} \approx 22$ seconds), and is primarily rate-limited by intramuscular O_2 availability (20,27). Because exercise causes a substantial reduction in intramuscular Po_2 (~34 mm Hg at rest to ~5 mm Hg at 50% PPO) (26), an AR strategy is likely to inhibit PCr resynthesis during this initial phase. Consequently, when recovery duration is relatively short, a PR strategy is most likely to optimize subsequent performance. However, as recovery duration increases, a secondary slower phase of PCr resynthesis is observed ($t_{1/2} > 170$ seconds), the rate of which is primarily limited by intramuscular [H^+] through its influence on the equilibrium of the creatine kinase reaction (15,20,23). Because AR is known to accelerate the clearance of intramuscular H^+ (28,40), an AR strategy may facilitate PCr resynthesis during its secondary phase. Therefore, an AR strategy is likely to enhance performance when recovery duration is longer. In support of these suggestions, investigators finding PR to optimize subsequent performance have used protocols with relatively brief (15–30 seconds) recovery periods (8,14,31), and, with the exception of 1 group (29), researchers reporting enhanced performance following AR have used protocols with relatively long (3–5 minutes) recovery periods (1,10,13,32).

The combined influence of recovery duration and mode has previously been investigated by 1 research group (35–37). Toubekis et al. (36) found that when subjects performed PR during short (30 and 120 seconds) recovery periods and AR during longer (5 minutes) recovery periods, subsequent 50-m swimming performance was optimized. However, similar work by the same group failed to confirm these findings (35,37). Although the above evidence supports the possibility of an interaction between recovery mode and duration, statistical analysis of any potential interactive effect has not previously been performed.

The aim of this study, therefore, was to investigate the interactive effect of recovery mode and duration on RSS performance, using a protocol reflecting the metabolic demands of the most intense periods of play in field-based sports. It is hypothesized that after exhaustive maximal exercise, the short-term recovery of RSS performance will be enhanced by PR, whereas recovery over a longer duration will be enhanced by AR.

Methods

Experimental Approach to the Problem

Each participant performed a preliminary testing and familiarization session, followed by 4 experimental trials in random order. All of the experimental protocols comprised an initial exhaustive (30 seconds) sprint, designed to largely deplete PCr stores, followed by a set of RSS (7 × 5 seconds interspersed with 20-second rest periods). The experimental trials differed only in the mode (PR vs. AR) and duration (45 vs. 180 seconds) of the intervening recovery period before each set of RSS (Figure 1). There were, therefore, 4 experimental conditions, referred to as PR_{45}, AR_{45}, PR_{180}, and AR_{180}. All trials were performed on a preprogrammed electromagnetically braked cycle ergometer (Lode Excalibur Sport; Lode BV, Groningen, The Netherlands). The torque factor for all sprints was 0.75 $N \cdot m \cdot kg^{-1}$ (3). Participants were instructed to refrain from high-intensity exercise for 48 hours before each trial, to abstain from alcohol and ensure adequate hydration 24 hours before each trial, and to avoid consuming food and fluids (except water) for 2 hours before each trial. Participants completed a diet diary for the 24-hour period before the preliminary trial, which they were instructed to replicate before each subsequent trial. Trials for each participant were separated by at least 48 hours. Participants began each experimental trial blind to the selected protocol.

Subjects

Ten male recreational athletes (age, 27.9 ± 5 years; height, 1.80 ± 0.07 m; mass, 81.6 ± 13.5 kg), with a minimum of 7 years experience in training for their sporting activities and with experience in some sort of multiple sprint sport, volunteered to participate in the study. At the time of the investigation, participants reported that they spent 7.8 ± 3.9 $h \cdot wk^{-1}$ training for their sport. Before participation, after being informed of all relevant procedures and risks, subjects provided written consent and completed an exercise readiness questionnaire. Ethical approval for the study was obtained from St Mary's University College Ethics Committee.

Preliminary Trial

In the preliminary trials, subjects performed an incremental lactate test (see Incremental Lactate Test) to determine the power output at lactate threshold (PO_{LT}), followed by a 15-minute recovery period during which anthropometric data (height and body mass) were collected. Subjects were then familiarized with the experimental procedures by performing the same basic protocol as for the experimental trials (Figure 1), but with 120 seconds of AR (PO = 95 W) before the RSS. Performance measures obtained from the initial 30-second sprint were used as a reference to evaluate potential pacing issues in the subsequent experimental trials. In effect, based on previously reported test-retest reliability ($r > 0.94$) (7), a PPO of <95% of the subject's reference value in any of the experimental trials would be

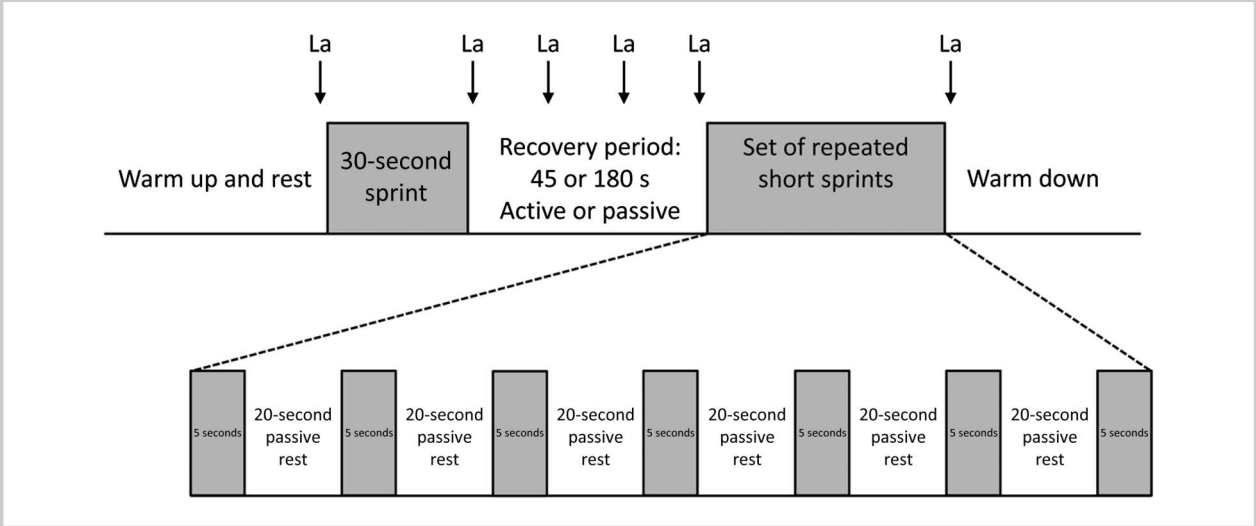

Figure 1. Schematic representation of the experimental protocol. Shaded blocks represent sprints. La = blood lactate sample (only performed in 180-second recovery trials).

regarded as the evidence of pacing and result in the trial being repeated.

Incremental Lactate Test

Individual lactate profiles were assessed using an incremental protocol consisting of 3-minute stages at a pedaling cadence of 80 revolutions per minute (RPM). Each stage was separated by a 45-second break, during which a 20 μl blood sample was drawn from the ear lobe to be analyzed for lactate concentration using an automated analyser (Boisen, C-Line; EKF Diagnostic, Ebendorfer Chaussee, Germany). Power output was increased by 20 W in each stage, with the initial PO individualized to ensure that the required lactate profile was obtained within 5–9 stages (33). Tests were terminated when the blood lactate concentration was $\geq$4 mmol·L^{-1}. Lactate threshold was identified using the log-log method, previously shown to provide a reliable evaluation of LT (4), and was performed using software developed for the purpose (25). The PO for the AR mode was set at 70% PO_{LT} for each subject.

Experimental Trials

Before the start of each experimental trial, subjects performed a 5-minute warm-up (80 RPM and PO = 120 W), incorporating a 5-second sprint at the start of minutes 2, 3, and 4. After 3 minutes of passive rest, subjects performed a maximal 30-second sprint, immediately after which they were instructed to either stop pedaling and remain seated (PR) or to carry on pedaling at 80 RPM (AR). After the specified (45 or 180 seconds) recovery period, subjects performed the RSS test. Subjects were advised 5 seconds before each sprint so that they could assume a predetermined starting position. Instruction was given to indicate the start and end points of each sprint, but no countdown was given to reduce the likelihood of pre-empting. Subjects remained seated and received strong verbal encouragement during all sprints. After the final sprint, subjects performed an active warm-down (PO $\sim$ 80 W) for $\leq$10 minutes.

Physiological and Metabolic Measures

Throughout each of the experimental trials, heart rate (HR) was recorded telemetrically at 5-second intervals (Polar S610i; Polar Electro Oy, Kempele, Finland), and $\dot{V}O_2$ was measured using breath-by-breath gas analysis (Jaeger Oxycon Pro; Jaeger Ltd., Hoechberg, Germany). Before each trial, the gas analyser was calibrated using a known concentration of oxygen and carbon dioxide (Riessner-Gase GmbH, Lichtenfels, Germany), and volume calibration was performed using a 3-L syringe (Viasys Healthcare GmbH, Hoechberg, Germany). Before analysis, data for $\dot{V}O_2$ during the recovery period after the 30-second sprint were filtered to remove any outliers and interpolated to produce readings at synchronous 5-second intervals. Blood lactate was measured during each of the AR_{180} and PR_{180} trials using the method described previously, with blood samples obtained 1 minute before the initial (30 seconds) sprint (baseline), at 0, 45, 90, and 135 seconds after the 30-second sprint, and immediately after the RSS.

Statistical Analyses

Statistical analyses were conducted using the Statistical Package for the Social Sciences (SPSS for Windows, version 15.0; SPSS, Inc., Chicago, IL, USA). Measures of central tendency and spread are presented as means $\pm$ *SD*. All PPO and $\dot{V}O_2$ data are presented relative to body mass (W·kg^{-1} and ml·kg^{-1}·min^{-1}, respectively) to reduce between-subject variation. Within-subject differences in PPO and mean PO (MPO) during the initial 30-second sprint (PPO_{30} and

MPO_{30}, respectively) were evaluated using a repeated-measures analysis of variance (ANOVA). Between-protocol differences in mean HR and mean $\dot{V}O_2$ during the recovery period after each 30-second sprint (HR_{rec} and $\dot{V}O_{2rec}$, respectively), as well as mean HR, and mean $\dot{V}O_2$ during the sets of RSS (HR_{RSS} and $\dot{V}O_{2RSS}$, respectively) were compared using 2-way (duration × mode) repeated-measures ANOVA. The influence of recovery mode on HR and $\dot{V}O_2$ at 5-second intervals during the recovery period after each 30-second sprint, as well as blood lactate at 45-second intervals during the 180-second recovery periods and immediately post-RSS, was also analyzed using 2-way (mode × time) repeated-measures ANOVA. The effects of recovery duration and mode on the PPO achieved during the RSS (PPO_{RSS}) were evaluated using 3-way repeated-measures ANOVA (duration × mode × sprint). Differences within trials between PPO_{RSS} and PPO_{30} were evaluated using a repeated-measures ANOVA. Significant interactions were followed up with post hoc analyses using Bonferonni corrections. Normality was assessed using the Shapiro-Wilk test, and sphericity was assessed using Mauchley's test. Where the assumption of sphericity was violated, the Greenhouse-Guisser statistic was used. Significance was accepted at $p \leq 0.05$ for all analyses.

Table 1. Power output data from the 30-second sprint at the start of each trial.*

	Familiarization	PR_{45}†	AR_{45}	PR_{180}	AR_{180}
PPO ($W \cdot kg^{-1}$)	12.3 ± 1.7	13.9 ± 1.5	14.5 ± 1.8	14.3 ± 1.7	14.1 ± 1.9
MPO ($W \cdot kg^{-1}$)	8.5 ± 0.9	8.7 ± 0.6	8.8 ± 0.6	8.8 ± 0.7	8.7 ± 0.7

*Values are indicated as means ± *SD*.
†PPO = peak power output; MPO = mean power output; PR_{45} = trial with 45 seconds of passive recovery; AR_{45} = trial with 45 seconds of active recovery; PR_{180} = trial with 180 seconds of passive recovery; AR_{180} = trial with 180 seconds of active recovery.

Results

Thirty-second Sprint Performance

Results from the 30-second sprints are presented in Table 1. In each experimental trial, PPO_{30} was >95% for the reference value achieved by respective subjects in their familiarization trial. Moreover, there were no significant within-subject differences in either PPO_{30} ($F_{(3,27)} = 0.55$, $p = 0.66$) or MPO_{30} ($F_{(3,27)} = 0.26$, $p = 0.86$).

Recovery Responses

Mean PO_{LT} was 175 ± 42 W, leading to a PO for the AR trials of 123 ± 29 W. There was a significant effect of recovery mode ($F_{(1,8)} = 124.9$, $p < 0.001$), duration ($F_{(1,8)} = 183.2$, $p < 0.001$), and mode × duration ($F_{(1,8)} = 12.6$, $p = 0.007$) on mean $\dot{V}O_{2rec}$ (Table 2). (Note: $\dot{V}O_2$ data are based on a sample size of 9 because of a problem with the gas analyser in one of the trials.) Similar effects of mode ($F_{(1,9)} = 25.6$, $p = 0.001$), duration ($F_{(1,9)} = 35.4$, $p < 0.001$), and mode × duration ($F_{(1,9)} = 8.8$, $p = 0.016$) were also observed for mean HR_{rec} (Table 2). The effects of recovery mode and duration on the kinetics of $\dot{V}O_{2rec}$ and HR_{rec} are presented in Figures 2 and 3, respectively. Analysis of both $\dot{V}O_{2rec}$ and HR_{rec} data revealed significant effects of mode ($p < 0.001$; apart from HR in 45-second trial, where $p = 0.01$), time ($p < 0.001$), and mode × time ($p < 0.001$).

There was no significant effect of recovery mode on blood lactate ($F_{(1,9)} = 3.1$, $p = 0.112$). There was, however, a significant effect of time ($F_{(2.6,23.6)} = 120.0$, $p < 0.001$) and a significant mode × time interaction ($F_{(1.9,16.7)} = 3.9$, $p = 0.005$). Post hoc tests revealed a significantly higher blood lactate only after 135 seconds of recovery (mean difference, 2.16 $mmol \cdot L^{-1}$; 95% likely range, 0.77–3.55 $mmol \cdot L^{-1}$) (Figure 4).

Repeated Sprint Performance

A significant interaction was observed between the effects of recovery duration and recovery mode on subsequent PPO_{RSS} ($F_{(1,9)} = 35.4$, $p < 0.001$). As shown in Table 3, post

Table 2. The effects of recovery duration and mode, following a maximal 30-second cycle sprint, on heart rate and oxygen uptake responses in a group ($n = 10$) of male recreational athletes.*

Recovery duration (s)	Recovery mode	Mean HR_{rec}† ($b \cdot min^{-1}$)	Mean $\dot{V}O_{2rec}$ ($ml \cdot kg^{-1} \cdot min^{-1}$)
45	Passive	156 ± 12‡	35.2 ± 4.4‡
	Active	164 ± 10‡§‖	45.6 ± 7.2‡§‖
180	Passive	133 ± 19	20.1 ± 2.2
	Active	150 ± 16‡	35.3 ± 4.8‡

*Values are indicated as means ± *SD*.
†HR_{rec} = recovery heart rate; $\dot{V}O_{2rec}$ = recovery oxygen uptake.
‡Significantly ($p \leq 0.05$) higher than after passive 180-second trials.
§Significantly ($p \leq 0.05$) higher than after passive 45-second trials.
‖Significantly ($p \leq 0.05$) higher than after active 180-second trials.

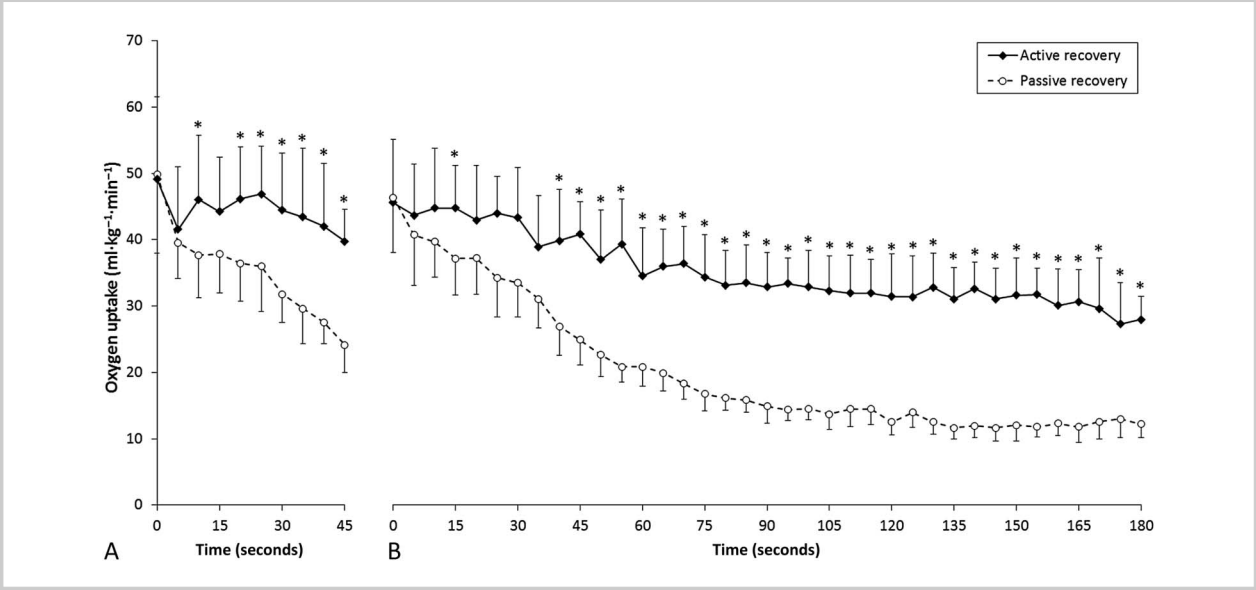

Figure 2. Oxygen uptake at 5-second intervals during 45 seconds (A) and 180 seconds (B) of active and passive recovery after a 30-second sprint. Data points represent means; bars represent *SD*. *Significantly ($p \leq 0.05$) different at the same time point.

hoc analysis revealed that after 45 seconds of recovery, mean PPO_{RSS} was significantly higher following PR than AR ($t_{(9)} = 2.8$, $p = 0.041$). Conversely, after 180 seconds of recovery, mean PPO_{RSS} was significantly higher following AR than PR ($t_{(9)} = 4.5$, $p = 0.003$). The interactive effect of recovery mode and duration on PPO_{RSS} was also significantly influenced by sprint number ($F_{(6,54)} = 2.85$, $p = 0.017$) (Figure 5). After 45 seconds of recovery, PPO was significantly higher ($p = 0.022$) following PR than AR in the first sprint, with no significant differences ($p \geq 0.05$) observed thereafter (Figure 5A). However, after 180 seconds of recovery, PPO was significantly higher following AR than PR in sprints 4 ($p = 0.04$) and 6 ($p < 0.001$) (Figure 5B). Significant differences ($p < 0.001$) between PPO_{30} and PPO_{RSS} were

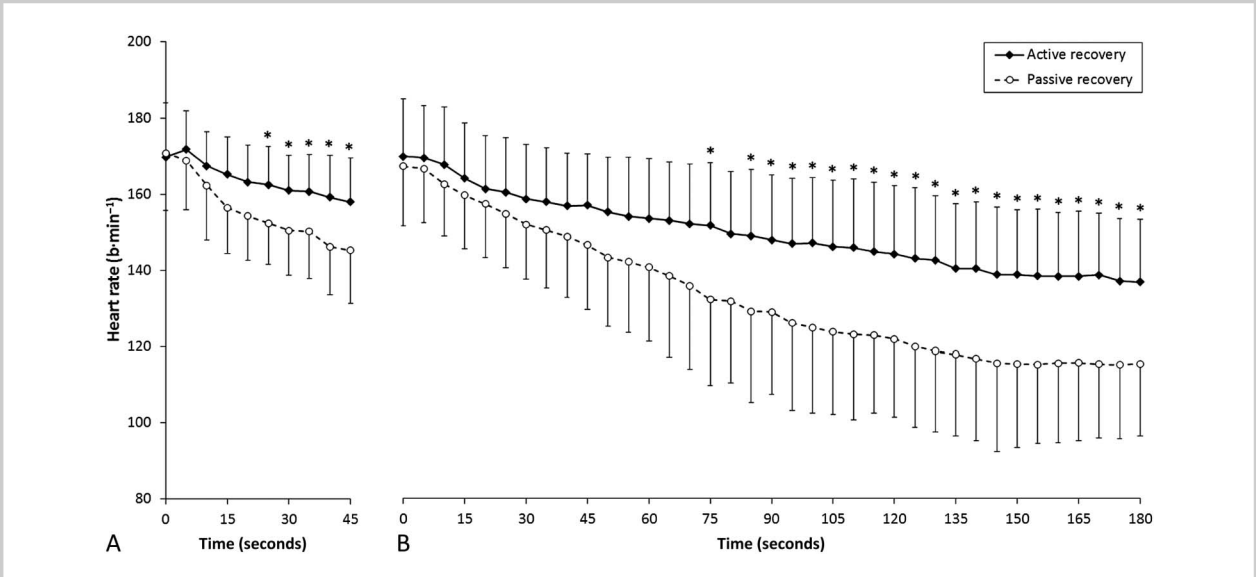

Figure 3. Heart rate at 5-second intervals during 45 seconds (A) and 180 seconds (B) of active and passive recovery after a 30-second sprint. Data points represent means; bars represent *SD*. *Significantly ($p \leq 0.05$) different at the same time point.

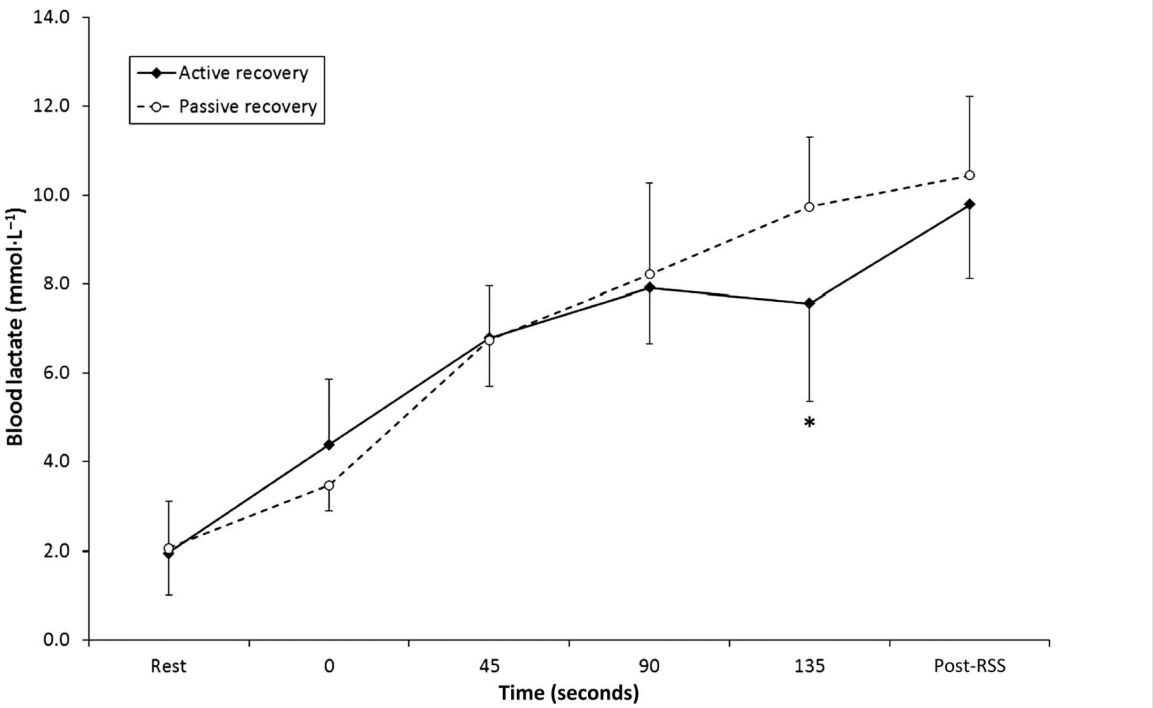

Figure 4. The effects of recovery mode (active vs. passive) on blood lactate concentrations at 45-second intervals during 180 seconds of recovery after a maximal 30-second sprint, and after a set of repeated short sprints (7 × 5 seconds sprints; 20-second rest periods). Data points represent means; bars represent *SD*. * Significantly ($p \leq 0.05$) different at the same time point.

revealed within each trial. Peak power output was significantly lower ($p \leq 0.05$) than PPO_{30} in all the RSS of the PR_{45} and AR_{45} trials (Figure 5A). In the PR_{180} trial, PPO was significantly ($p \leq 0.05$) lower than PPO_{30} in all RSS except sprint 3, whereas in the AR_{180} trial PPO was significantly ($p \leq 0.05$) lower than PPO_{30} in sprint 1 only (Figure 5B). Furthermore, in the AR_{45} trial, PPO was significantly ($p \leq 0.05$) lower in sprint 1 than in all subsequent RSS (Figure 5A), whereas there were no significant differences ($p \geq 0.05$) between sprints within any other RSS trials.

There was a significant main effect of recovery mode ($F_{(1,8)} = 27.8, p < 0.005$) and duration ($F_{(1,8)} = 22.8, p < 0.005$)

Table 3. Physiological and performance responses during a set of 7 × 5 seconds repeated maximal sprints (20-second stationary rest periods) after a maximal 30-second cycle sprint with different recovery durations (45 vs. 180 seconds) and modes (active vs. passive) in male recreational athletes ($n = 10$).*

Recovery duration (s)	Recovery mode	Mean PPO_{RSS}† ($W \cdot kg^{-1}$)	Mean HR_{RSS} ($b \cdot min^{-1}$)	Mean $\dot{V}O_{2RSS}$ ($ml \cdot kg^{-1} \cdot min^{-1}$)
45	Passive	12.0 ± 1.4‡	148 ± 14	34.6 ± 3.6
	Active	11.4 ± 1.4	154 ± 12	38.0 ± 4.7
180	Passive	12.0 ± 1.2	141 ± 16	32.3 ± 4.5
	Active	12.7 ± 1.2§	145 ± 18	35.3 ± 4.7

*Values are indicated as means ± *SD*.
†Mean PPO_{RSS}, HR_{RSS}, and $\dot{V}O_{2RSS}$ = average peak power output, average heart rate, and average oxygen uptake during the 7 × 5 seconds repeated maximal sprint protocol, respectively.
‡Significantly ($p \leq 0.05$) higher than active 45-second trial.
§Significantly higher than passive 180-second trial.

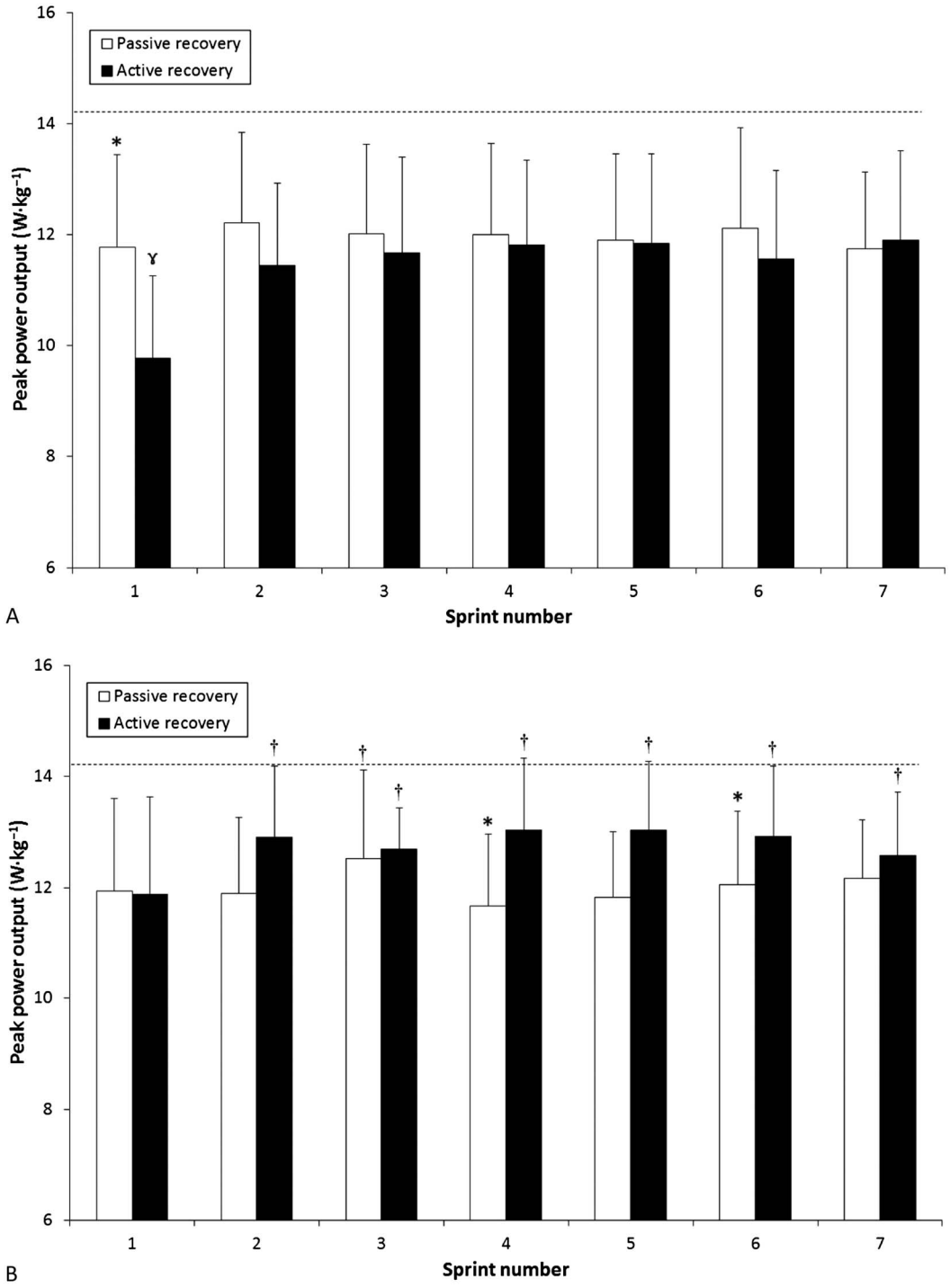

Figure 5. Peak power output (PPO) during repeated short sprints (7 × 5 seconds; 20-second rest periods) after either 45 seconds (A) or 180 seconds (B) of passive or active recovery from a maximal 30-second sprint. Values are means; bars represent *SD*. Dashed line represents 30-second sprint PPO. *Significantly ($p \leq 0.05$) different from active recovery. ˠSignificantly ($p \leq 0.05$) lower than all other sprints in the same trial. †Not significantly ($p \geq 0.05$) different from PPO in 30-second sprint.

on mean $\dot{V}O_{2RSS}$. Significant effects of recovery mode ($F_{(1,9)} = 16.9$, $p < 0.005$) and duration ($F_{(1,9)} = 13.1$, $p < 0.01$) were also observed for mean HR_{RSS}. In short, mean $\dot{V}O_{2RSS}$ and mean HR_{RSS} values were higher in AR vs. PR trials, and also in short vs. long recovery trials. However, there was no significant ($p \geq 0.05$) mode × duration interaction for either variable (Table 3).

Discussion

The aim of this study was to examine the interactive effects of recovery mode and duration on the performance of a field sport-specific set of RSS, following a standardized bout of exhaustive exercise. The study found that where recovery from the initial exhaustive bout was short (45 seconds), mean PPO_{RSS} was higher following PR than AR. However, after the longer (180 seconds) recovery period, mean PPO_{RSS} was higher following AR than PR. Moreover, the finding that PPO_{30} was achieved in 6 RSS in the AR_{180} trial, but only 1 RSS in the PR_{180} trial, further demonstrates the beneficial effect of AR during the longer recovery period (Figure 5). The results, therefore, demonstrate that a significant interaction exists between recovery duration and recovery mode, and as such, may be seen to reconcile previous reports, some of which found subsequent performance to be optimized by AR (1,10,13,29,32), whereas others found PR to be the most beneficial (8,14,31).

The performance benefit observed after AR may be because of a hemodynamic effect, which is evident over the 180 but not the 45-second recovery periods. After intense muscular contraction, blood flow is known to be critical for the optimization of both intramuscular PCr resynthesis and subsequent performance (20,23). Blood flow is enhanced during AR relative to PR because of both an elevated HR (Figure 3) and the "pumping" effect of muscular contraction, which have been shown to prevent the stagnation of blood in the dilated vasculature of the previously active muscle (11,34). Together these factors help to maintain central venous pressure and prevent the often-dramatic decline in cardiac preload and stroke volume that is observed during PR (11,34). However, as shown in this study, HR remains elevated after intense exercise, declining only gradually toward baseline even during PR (Figure 3). Consequently, the difference between HR in AR and PR reached significance only after a lag-time between 25 seconds (45-second trials) and 75 seconds (180-second trials). Similarly, the effect of the muscle-pump is reportedly evident only after a short (~2 minutes) lag-time (11). It is likely, therefore, that this delayed hemodynamic effect of AR relative to PR helps to explain why performance was enhanced following AR in the 180 seconds but not the 45-second recovery periods.

It has been suggested that the elevated blood flow observed during AR enhances subsequent performance by increasing the delivery of O_2 to the working muscle, thereby facilitating the resynthesis of PCr (13). However, as illustrated in this study by the significantly greater mean $\dot{V}O_{2rec}$ in AR than in PR over both 45 and 180 seconds, there is an elevated metabolic cost associated with AR, which has been shown to depress muscle tissue oxygenation (14,26). Consequently, any benefit derived from enhanced O_2 delivery during AR is likely outweighed by such an increase in O_2 consumption and the resultant reduction in intramuscular P_{O_2}. It is likely that these factors explain why, in the period immediately after exhaustive exercise, when intramuscular $[O_2]$ is critical to the rate of PCr resynthesis, a PR strategy appears to be optimal, until the hemodynamic benefits of AR outweigh the energetic cost of the exercise.

Skeletal muscle blood flow is a principal component of the cell-cell lactate shuttle (16). Moreover, the rate of lactate oxidation is known to increase during exercise relative to $\dot{V}O_2$ (16). Together, these factors likely explain the well-established increase in the rate of blood lactate removal during AR (19,24,37). In this study, AR resulted in a significantly lower blood lactate concentration than PR, after a lag-time of 135 seconds (Figure 4). Consequently, blood lactate was likely lower in AR than PR at the end of the 180 seconds but not the 45-second recovery periods. However, because the lactate molecule is no longer considered a direct cause of muscle fatigue (16), this reduction in blood lactate during AR is unlikely to have been responsible for the enhancement of subsequent RSS performance. Nevertheless, given that lactate and H^+ are predominantly cotransported through the monocarboxylate transport proteins MCT1 and MCT4 (21), and because AR has been also shown to accelerate the removal of intramuscular H^+ (28,40), the reduction in blood lactate observed during AR in the 180-second trials likely reflects a concomitant reduction in intramuscular $[H^+]$. Moreover, because the rate constant for the creatine kinase reaction is retarded by a low-intramuscular pH, particularly during its secondary phase (15,23), a reduction in intramuscular $[H^+]$ during AR may have accelerated the latter phase of PCr resynthesis, thereby enhancing subsequent RSS performance. Although it remains possible that the enhanced RSS performance observed after AR was the result of a reduction in the direct inhibition of muscular contraction by H^+, this mechanism has, at least under in vitro conditions, been largely discredited (38). More relevant to this work is the disparity observed between the time course of muscle force recovery and that of intramuscular pH after intense muscular contraction in vivo (2). For example, Bogdanis et al. (6) found that after 3 minutes of PR, after exhaustive exercise, 88.7% of PPO was achieved despite a high intramuscular $[H^+]$ (pH, 6.79). However, the high PO observed by the authors is likely explained by the initial rapid pH-independent phase of PCr resynthesis. In support of this, this study also found the performance of the initial 5-second sprint in the AR_{180} and PR_{180} trials to be similar, despite a lower blood lactate in the AR_{180} trial. Indeed, because intramuscular PCr stores are likely 70–84% replenished after 180 seconds of PR (6,12), and with the initial 5-second sprint requiring <55% of the total PCr store (30), this observation

is not surprising. However, Bogdanis et al. (6) also found neither PPO nor PCr to be fully recovered after a further 3 minutes (i.e., 6 minutes) of PR, suggesting that the complete restoration of PCr was, in fact, inhibited by the low intramuscular pH. In this study, therefore, it is possible that an enhanced clearance of H^+ during AR facilitated the repletion of PCr during the 180-second recovery period, thereby enabling the superior RSS performance as the set progressed.

Several observations from this study are difficult to explain from a physiological perspective and should, therefore, be highlighted. First, previous studies have consistently reported fatigue to occur during a set of RSS, as evidenced by a progressive decrement in sprint performance (17,30). However, this study found no such evidence in any of the trials. The impact of previous exercise on the mechanisms responsible for the development of fatigue during RSS, therefore, requires further investigation. Second, although PPO in the first of the RSS in the AR_{180} trial was not significantly different to the rest of the RSS in the same trial, it was the only sprint in that condition with a PPO significantly lower than PPO_{30}. Because PCr would have been almost completely replenished after 180 seconds of AR, the mechanisms responsible for this observation are difficult to elucidate. However, it is possible that the increase in PPO after the initial sprint is because of a potentiation effect, as observed in several previous studies (17). Finally, in the AR_{45} trial, PPO was significantly lower in the first RSS than in the rest of the RSS in that condition. The metabolic implication of this finding is that intramuscular PCr was replenished to a greater extent during the 20-second recovery period after the initial RSS, than in the 45-second recovery period before. However, the precise physiological mechanisms responsible for this finding are, again, difficult to identify.

The initial 30-second sprint of the experimental protocol was designed to induce a standardized level of PCr depletion, thereby providing a consistent starting point from which recovery could be measured. Individual measures of performance (i.e., PPO and MPO) during the 30-second sprint, as well as mean HR, $\dot{V}O_2$, and lactate at the end of the 30-second sprint, were similar in all trials, indicating that the metabolic impact of the initial sprint was consistent, and that the observed difference in RSS performance could be attributed to the recovery strategy. It should be noted, however, that application of the present findings to a match situation, in which sets of RSS are repeated throughout a prolonged (80–90 minutes) period, is complicated by the potential influence of AR on glycogen depletion and the associated decline in performance (9). Nevertheless, the findings are applicable to both the prescription and quantification of training stimuli because the overall training impulse is considerably influenced by the degree to which recovery is achieved between bouts (39). Future research should look to assess the impact of AR on the performance of repeated sets of RSS over the duration of a match and also to further define the relationship between postexercise cardiodeceleration and the transition of optimal recovery mode from PR to AR as recovery duration increases.

Practical Applications

This study demonstrates that after exhaustive maximal exercise, the optimal recovery mode for the enhancement of subsequent RSS performance is dependent on the duration of the recovery period. Where rest periods are short, a PR strategy appears to optimize recovery and, therefore, subsequent RSS performance. However, as recovery duration increases, subsequent RSS performance appears to benefit from an AR strategy. Although the exact point at which AR becomes beneficial remains to be elucidated, those wishing to optimize recovery between maximal-intensity efforts during training should be aware of this interactive effect and use AR and PR strategies accordingly.

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