Effects of active and passive recovery on performance during repeated-sprint swimming

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Abstract
The effect of active and passive recovery on repeated-sprint swimming bouts was studied in eight elite swimmers. Participants performed three trials of two sets of front crawl swims with 5 min rest between sets. Set A consisted of four 30-s bouts of high-intensity tethered swimming separated by 30 s passive rest, whereas Set B consisted of four 50-yard maximal-sprint swimming repetitions at intervals of 2 min. Recovery was active only between sets (AP trial), between sets and repetitions of Set B (AA trial) or passive throughout (PP trial). Performance during and metabolic responses after Set A were similar between trials. Blood lactate concentration after Set B was higher and blood pH was lower in the PP (18.29 ± 1.31 mmol/L and 7.12 ± 0.11 respectively) and AP (17.56 ± 1.22 mmol/L and 7.14 ± 0.11 respectively) trials compared with the AA (14.13 ± 1.56 mmol/L and 7.23 ± 0.10 respectively) trial (P < 0.01). Performance time during Set B was not different between trials (P > 0.05), but the decline in performance during Set B of the AP trial was less marked than in the AA or PP trials (main effect of sprints, P < 0.05). Results suggest that active recovery (60% of the 100-m pace) could be beneficial between training sets, and may compromise swimming performance between repetitions when recovery durations are short (< 2 min).

Keywords: Sprint swimming, active recovery, passive recovery, repeated sprints, fatigue

Introduction
One of the most common practices during the training of swimmers is to use sets of repeated sprints. The aim of the swimmer in such training sets is to maintain the highest possible speed in each repetition (Maglischo, 2003), but this is not usually attainable (Peyrebrune, Nevill, Donaldson, & Cosford, 1998). The inability of the swimmer to maintain maximal speed in each repetition is associated with fatigue and is commonly related to high muscle lactate and hydrogen ion (H+) concentrations and low phosphocreatine (Bogdanis, Nevill, Boobis, Lakomy, & Nevill, 1995). The removal of H+ and restoration of phosphocreatine is important for maintaining the highest speed in a subsequent maximal effort. Active recovery is used to accelerate recovery between repeated bouts of sprint exercise, aimed at increasing muscle blood flow, which in turn enhances the lactate/H+ removal, and possibly facilitates the rate of phosphocreatine resynthesis.

Swimming studies have focused on the post-exercise decrease in blood lactate concentration as a measure of recovery (Cazorla, Dufort, & Cervetti, 1983; McMaster, Stoddard, & Duncan, 1989), although blood lactate concentration is not related to subsequent performance during repeated sprints (Bogdanis et al., 1995; Toubekis, Douda, & Tokmakidis, 2005; Toubekis, Smilios, Bogdanis, Mavridis, & Tokmakidis, 2006) and is no longer considered an agent of fatigue (Gladden, 2004).

Cycling studies have shown that performance during repeated sprints is enhanced after active compared with passive recovery (recovery duration 4 min) (Bogdanis, Nevill, Lakomy, Graham, & Louis, 1996; Dorado, Sanchis-Moysi, & Galbet, 2004; Spierer, Goldsmith, Baran, Hrynewicz, & Katz, 2004). In contrast to the findings in cycling studies, a decrease in performance was observed during repeated 25-m swimming sprints with active recovery of 45 or 120 s duration, and different
intensities of active recovery (i.e. 50 or 60% of the
100-m pace; Toubekis et al., 2005, 2006). However,
swimming race performance over 100 m and 200
yards was improved following 5–10 min active rather
than passive recovery (Greenwood, Moses, Bernar-
dino, Gaesser, Weltman, 2008; Toubekis et al.,
2008). A variety of exercise modes and interval
durations as well as a number of sprint repetitions
has been applied in previous studies; however, there
is no consensus on the effect of active recovery on
swimming performance. Employing active recovery
may not be beneficial in all cases for performance
improvements, and a more specific examination to
identify any limitations is required.

No studies have examined the effect of active
recovery during repeated-sprint swimming (i.e. 50
yards/metres) by employing protocols that simulate
swimming training practices, including repeated sets
of bouts and recovery times of relatively short
duration (∼1 min 30 s to 5 min). Such sets are
commonly used to improve anaerobic metabolism
and active recovery is usually recommended between
sets (Maglischo, 2003).

The aim of the present study, therefore, was to
examine the effects of active and passive recovery on
performance and metabolism during and between
sets of repeated-sprint swimming bouts, similar to
tasks commonly used during swimming training.
Performance and metabolism in a subsequent set
may be affected by the intensity of the previous set of
bouts. For this reason, we decided to apply a
standardized high-intensity tethered swimming set
of bouts to determine the effect of active recovery on
a subsequent set. It was hypothesized that perfor-
mance times would be faster when active recovery
was performed between repetitions and between sets
of repetitions.

Methods

Participants

Eight elite male swimmers from a university swim-
ing team participated in the study. All participants
had competed in Senior National Championships in
the previous 12 months, and normally trained seven
to nine times per week for 2 h per session. Weekly
training distance swum was between 40,000 and
50,000 m. 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 Loughborough University
approved the test protocols and all procedures.
Participants’ mean (± s) age, height, body mass,
and tethered swimming maximum oxygen uptake
(V\textsubscript{O\textsubscript{2}max}) were 20 ± 2 years, 1.82 ± 0.05 m, 75.0 ±
5.5 kg, and 4.2 ± 0.3 litres·min\textsuperscript{-1} respectively.
The participants’ personal best times for 100-m
front crawl (53.92 ± 1.39 s) were all recorded in a
25-m pool within the previous year. Swimmers were
experienced in performing sprint training sets such
as the one used in this study and were accustomed
with the tethered swimming apparatus. During the
test period, sessions were mostly endurance based,
with one intensive sprint training set per week. This
higher intensity set was not performed in the 2 days
before any trial. All tests were performed in a 25-yard
(22.86-m) indoor swimming pool with a mean water
temperature throughout the trials of 26 ± 1°C.

Preliminary tests

On separate days, swimmers performed a V\textsubscript{O\textsubscript{2}max}
test and a 30-s maximal swimming sprint using a
tethered swimming apparatus. During the V\textsubscript{O\textsubscript{2}max}
test, swimmers performed three to five stages with
the resistive force increased by 4.9 or 9.81 N every
3 min until exhaustion. Expired air was collected in
the last minute of each 3-min stage (Bonen, Wilson,
Yarkony, Belcastro, 1980). The 30-s maximal sprint
was performed with the tethered mechanism in a
fixed position. A force transducer interfaced to a
computer measured the force applied by the swim-
ner at zero velocity to a non-elastic rope attached
around the swimmer’s waist. The signal was dis-
played on a computer screen (BBC model B
microcomputer) and stored on disk for further
analysis. Before each test the force transducer was
calibrated using known resistive forces.

Main trials

Swimmers took part in three randomly assigned
main trials, separated by 3–7 days and performed at
the same time of the day (± 1 h). Each trial was split
into two sections (Set A and Set B; Figure 1).

The first section (Set A) consisted of 4 × 30-s bouts
of semi-tethered swimming separated by 30 s of
passive rest. During all three trials, the swimming
intensity of Set A was standardized by setting the
applied resistive force equal to 95% of the individual
mean force attained during 30 s of the maximal
tethered swimming sprint. The resistive force on
the rope was measured by the force transducer while
the loaded basket was suspended and was used for the
analysis of the results. Swimmers’ arm strokes were
recorded by depressing the space bar of a micro-
computer once per stroke cycle. The time to complete
each stroke cycle in addition to total number of strokes
was recorded and stored on a disk. The stroke rates
produced in the first main trial during the 4 × 30-s
tethered swimming bouts were retrieved as bleeps in
the next two trials. The swimmer was asked to follow
the same stroke rate according to the bleeps. This
provided a standardized volume and intensity of fatiguing exercise performed during Set A on each of the three trials. On completion of Set A, swimmers approached the shallow end of the pool where they could stand and be detached from the belt within 15 s.

The second section of the test (Set B) consisted of 4 × 50-yards of maximal front crawl swimming at intervals of 2 min (exercise time plus recovery time = 2 min), each beginning with a push start from within the pool. The two sections (Set A and B) were separated by 5 min of recovery. On one occasion, recovery between Sets A and B and during the repetitions of Set B was passive, with swimmers standing in the water at the shallow end of the pool (PP trial). On a second occasion, recovery between Sets A and B was 1 min passive, 3 min active, and 1 min passive, but during Set B passive (AP trial). On a third occasion, recovery between sets A and B and during the recovery between repetitions of Set B was active (AA Trial). The design of the protocol during the AP and AA trials (1 min passive, 3 min active, and 1 min passive) allowed capillary blood collection after Set A and before Set B (Figure 1). Swimmers in the AA trial began active recovery within 5 s of each 50-yards repetition and stopped swimming 15 s before the start of the next repetition. Recovery on completion of Set B was always passive. Active recovery pace on all occasions was 60% of the mean speed calculated from the individual’s best time for 100-m front crawl. Swimmers were comfortably able to adhere to the prescribed pace. During Set B, the time to complete each 50-yards repetition, the 25-yards split time, and the strokes completed for each 25 of the 50-yards swim were recorded in duplicate by experienced timekeepers. The mean of these two times was used in the analysis of the results. Strokes counted during the first and second 25 yards of each 50-yards repetition were used to calculate the mean stroke rate (SR) and mean stroke length (SL) as follows:

\[
SR \text{ (cycles} \cdot \text{min}^{-1}) = (\text{stroke cycles to complete the distance/time in seconds to complete the distance}) \times 60
\]

\[
SL \text{ (m} \cdot \text{cycle}^{-1}) = \text{distance to swim/stroke cycles to complete the distance.}
\]

The time spent during and the distance covered after each turn and glide from the wall after the push-off was not included in the calculation of stroke rate and stroke length. This is a limitation that may lead to an overestimation of stroke length and underestimation of stroke rate by 5% (Craig, Skehan, Pawelczyk, & Boomer, 1985). However, these errors occur in a systematic way and do not influence the comparison across trials.

Heart rate was recorded at rest and every 5 s during the preliminary tests and main trials using short-range radio telemetry (Polar Electro PE 3000, Kempele, Finland). A transmitter was strapped to the chest, level with the sternum, and secured under a lycra “triathlon top” (Speedo Europe Ltd.) to ensure constant contact between the electrodes and the skin. The participants were required to refrain from consuming alcohol and perform only light swimming (4–5 km day⁻¹) in the 24 h before tests. For 3 days before trial 1, swimmers recorded all food and drink intake in a diary, and were asked to follow the same diet and eating schedule before trials 2 and 3. A standardized warm-up (approximately 20 min: 400-yards swim, 200-yards pull, 200-yards kick, 4 × 50-yards at increasing speed, 100-yards easy swim, 4 × 10-yards sprint, 100-yards easy swim) was performed in preparation for each test. On completion of the warm-up, the swimmer was attached to the belt, dried, kept warm, and rested on a chair at the side of the pool. Tests began 15 min after the end of the warm-up.

**Blood collection and analysis**

Capillary blood samples were taken 3 min before the start of each main trial, 30 s after Set A, 30 s before the start of Set B, and 1, 3, 5, and 8 min after Set B. Blood samples were taken simultaneously from the thumbs of both hands. Duplicate 20-μl samples of blood were obtained from one thumb to determine lactate concentration. Blood samples were dispensed into tubes containing 200 μl of 2.5% perchloric acid, mixed, and centrifuged for 3 min. The tubes were then stored.
at −20°C and assayed enzymatically later, using the method described by Maughan (1982). A further capillary blood sample was collected from the other thumb in a heparinized tube (90–105 μl) and was immediately analysed for pH (Radiometer ABL 5, Copenhagen, Denmark). The intra- and inter-assay coefficients of variation for repeated analysis of blood lactate were 1.8–2.7% and 2.3–4.3% respectively.

Expired air collection and analysis

Expired air samples were collected in Douglas bags during each 30-s bout of Set A (tethered swimming) using a two-way valve adapted from the design of Toussaint and colleagues (1987). All expired samples collected were analysed for O₂ and CO₂ concentration using a paramagnetic oxygen analyser (570A Servomex) and infrared carbon dioxide analyser (LIRA Gas monitor 3250). Analysers were previously calibrated with gases of a known mixture. Gas volumes were measured using a Harvard dry gas meter. Values obtained were corrected for STPD and the Haldane transformation used to correct for differences in inspired and expired air volumes.

Statistical analysis

Normal distribution of the data was tested using the Kolmogorov–Smirnoff test. A fully repeated-measures factorial analysis of variance (ANOVA) (3 trials × 4 sprints), with follow-up Tukey post-hoc tests as appropriate, was used to compare means. Sphericity was verified by the Mauchly’s test. When the assumption of sphericity was not met, the significance of F-ratios was adjusted according to the Greenhouse-Geisser procedure. The statistical power (p) of the analysis was determined for each factor using the SPSS statistical package. Cohen’s d was used for the calculation of effect sizes (ES):

\[ d = (M_1 - M_2)/s_{M1} \]

where \( M_1 \) = the mean of the first condition or sprint, \( M_2 \) = the mean of the second or third condition or sprint, and \( s_{M1} \) = the standard deviation of the first mean (Rhea, 2004). Relationships between variables were examined using the Pearson product–moment correlation coefficient. Statistical significance was set at \( P < 0.05 \) and the results are presented as means ± standard deviations.

Results

Performance

Mean resistive force recorded during the 30-s tethered swimming sprint was 138.6 ± 18.1 N and 95% of the mean force attained during the 30-s tethered swimming sprint was applied during Set A of the three main trials (132 ± 12 N); this was successfully reproduced during each repetition in Set A in all three trials (\( P > 0.05 \); Table I). Oxygen uptake and stroke rate during Set A were not different between trials (approximately 70–75% \( \dot{V}O_2 \text{max} \) and 42–43 stroke cycles · min⁻¹ respectively; Table I). The applied resistive force in Set A corresponded to ∼156% of that attained in the \( \dot{V}O_2 \text{max} \) test.

There were no differences between conditions in performance time [mean ± s (95% confidence intervals) – PP: 25.58 ± 0.84 s (24.88 to 26.28); AP: 25.32 ± 1.03 s (24.46 to 26.19); AA: 25.94 ± 1.03 s (25.09 to 26.80); \( P = 0.136 \), ES = 0.3 to 0.4, \( p = 0.4 \)]. Mean AP trial times were 1.0 ± 3.2% (95% confidence intervals: −1.7 to 3.6%) and 2.4 ± 2.1% (95% confidence intervals: 0.7 to 4.1%) faster than the PP and AA trials respectively. Performance times in all three trials slowed in the second, third, and fourth sprint compared with the first (main effect sprints: \( P = 0.001 \), ES = 0.6 to 0.7, \( p = 0.9 \); interaction: \( P = 0.152 \), \( p = 0.6 \)). The first 50-yards sprint time was not different across conditions (PP: 24.82 ± 0.98 s; AP: 25.11 ± 1.31 s; AA: 25.27 ± 1.12 s; \( P > 0.05 \)), and performance time in the AP trial during sprints 2, 3, and 4 was similar to the first sprint (25.22 ± 1.01 s, 25.47 ± 1.19 s, and 25.50 ± 0.85 s respectively; ES = 0.1 to 0.3, \( P = 0.268 \)). Performance times for the last three sprints were slower than the first in the PP trial (25.65 ± 1.02 s, 25.96 ± 0.94 s, and 25.89 ± 0.81 s respectively; ES = 0.9 to 1.2, \( P = 0.00 \)) and AA trial (26.23 ± 1.23 s, 26.18 ± 1.04 s, 26.12 ± 1.25 s respectively; ES = 0.8 to 0.9; \( P = 0.02 \)) (Figure 2).

The fatigue index during Set B (expressed as the percentage of the time difference between the first 25 yards of the first sprint and the second 25 yards of the fourth sprint) was no different between trials (PP: 9.01 ± 3.84%; AP: 6.29 ± 3.46%; AA: 7.86 ± 5.28%; \( P = 0.441 \)).

Stroke rate and stroke length

Mean stroke rate during set B was lower in the AA trial than in the AP and PP trials (main effect trials: \( P = 0.041 \), ES = 0.4 to 0.5, \( p = 0.6 \)). Stroke rate also declined during the second, third, and fourth sprints compared with the first (main effect sprints: \( P = 0.004 \), ES = 0.2 to 0.3, \( p = 0.9 \); interaction: \( P = 0.487 \), \( p = 0.4 \)). A tendency for maintenance of stroke rate was observed in the AP trial [(sprints 1–4) PP: 41.6 ± 3.7, 40.1 ± 3.6, 39.5 ± 4.0, 40.4 ± 3.6 cycles · min⁻¹; AP: 40.8 ± 6.2, 40.5 ± 5.7, 40.3 ± 4.9, 40.6 ± 4.6 cycles · min⁻¹; AA: 40.2 ± 4.3, 38.1 ± 4.5, 38.0 ± 3.8, 38.5 ± 3.3 cycles · min⁻¹]
respectively]. Mean stroke length did not change during the four repetitions in all three trials and was not different between trials (PP: $2.67 \pm 0.22$ m \cdot cycle$^{-1}$; AP: $2.71 \pm 0.29$ m \cdot cycle$^{-1}$; AA: $2.75 \pm 0.23$ m \cdot cycle$^{-1}$; main effect trials: $P=0.159$; main effect sprints: $P=0.360$; interaction: $P=0.770$).

Metabolic responses

Blood lactate concentration 30 s after completion of Set A was $13.0 \pm 3.1$, $12.4 \pm 3.4$, and $11.7 \pm 3.5$ mmol \cdot L$^{-1}$ for the PP, AP, and AA trial respectively ($P<0.05$). Values remained similar until the end of the 5-min recovery period (Figure 3). Peak blood lactate concentration occurred 1 min after Set B in all three trials. The highest peak blood lactate was observed in the AP and PP trials ($17.6 \pm 3.4$ and $18.3 \pm 3.7$ mmol \cdot L$^{-1}$ respectively) while peak blood lactate was lower in the AA trial ($14.1 \pm 4.5$ mmol \cdot L$^{-1}$, $P=0.01$). These blood lactate differences were still evident 8 min into

### Table 1. Resistive forces, oxygen uptake, stroke rate, stroke length, and heart rate during Set A of each trial (mean $\pm$ s).

<table>
<thead>
<tr>
<th>Trial</th>
<th>PP</th>
<th>AP</th>
<th>AA</th>
</tr>
</thead>
<tbody>
<tr>
<td>Repetition</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>Force (N)</td>
<td>$129 \pm 9$</td>
<td>$129 \pm 10$</td>
<td>$129 \pm 9$</td>
</tr>
<tr>
<td>$O_2$ uptake (ml kg$^{-1}$ min$^{-1}$)</td>
<td>$126 \pm 11$</td>
<td>$126 \pm 10$</td>
<td>$127 \pm 10$</td>
</tr>
<tr>
<td>Stroke rate (cycles min$^{-1}$)</td>
<td>$47 \pm 4$</td>
<td>$47 \pm 3$</td>
<td>$45 \pm 4$</td>
</tr>
<tr>
<td>Heart rate (beats min$^{-1}$)</td>
<td>$179 \pm 11$</td>
<td>$179 \pm 11$</td>
<td>$179 \pm 11$</td>
</tr>
</tbody>
</table>

Note: *$P<0.05$ compared with the first repetition. PP = passive recovery trial; AP = active recovery between Set A and B, but passive between the repetitions of set B; AA = active recovery trial.

Figure 2. Performance times during the 4 x 50-yards sprints ($n=8$, mean $\pm$ s). *$P<0.01$ denotes difference from the first sprint.

Figure 3. Blood lactate values during trials ($n=8$, mean $\pm$ s). *$P<0.05$ denotes difference from values before Set B. *$P<0.05$ AA vs. AP and PP trials.
recovery after Set B (Figure 3). The decline in blood pH values closely followed the increases in blood lactate. The lowest blood pH was observed 3 min after Set B both in the PP and AP trials (7.11 ± 0.10 and 7.12 ± 0.10 respectively) (Figure 4). Strong negative correlations were observed between blood lactate concentration and blood pH during all trials (r = −0.91 to −0.94, P = 0.01).

Heart rate responses

The heart rate response was similar during Set A and during the first and second minute of the recovery period between Set A and B across trials (main effect trials: P > 0.05). In the third, fourth, and fifth minute of recovery (between Set A and B), heart rate in the AP and AA trials was higher than in the PP trial (main effect trials: P = 0.01, interaction: P = 0.01; Table II). Peak heart rate after each 50-yards sprint in the AA trial was higher after all four sprints compared with the corresponding sprints in the PP trial (180 vs. 175 beats·min⁻¹; main effect trials: P = 0.01; Table II). Mean heart rate during the recovery period after each sprint was higher in the AA than in the PP and AP trials (165 vs. 152 beats·min⁻¹; main effect trials: P = 0.01; Table II).

Discussion

The main finding of this study was that performance during Set B was similar in all three conditions, with a tendency for poorer performance in the AA trial that was matched by a lower stroke rate. In the absence of statistical significance, the differences in the mean time over 4 × 50 yards between trials may still be important for performance in training for success in high-level swimming competition. Mean performance times in the AP trial were ~1% and ~2.4% faster (n.s.) than the PP and AA trials.

Table II. Heart rate (beats·min⁻¹) during the recovery period between Sets A and B, and during the repetitions and recovery of Set B (mean ± s).

<table>
<thead>
<tr>
<th>Trial</th>
<th>1st</th>
<th>2nd</th>
<th>3rd</th>
<th>4th</th>
<th>5th</th>
</tr>
</thead>
<tbody>
<tr>
<td>PP</td>
<td>167 ±11</td>
<td>138 ±11</td>
<td>124 ±14</td>
<td>117 ±11</td>
<td>114 ±11</td>
</tr>
<tr>
<td>AP</td>
<td>164 ±11</td>
<td>145 ±8</td>
<td>143 ±6</td>
<td>141 ±6</td>
<td>140 ±6</td>
</tr>
<tr>
<td>AA</td>
<td>163 ±11</td>
<td>143 ±6</td>
<td>141 ±6</td>
<td>140 ±6</td>
<td>138 ±6</td>
</tr>
<tr>
<td>Peak 1</td>
<td>162 ±14</td>
<td>148 ±11</td>
<td>141 ±6</td>
<td>140 ±6</td>
<td>138 ±6</td>
</tr>
<tr>
<td>Peak 2</td>
<td>170 ±14</td>
<td>152 ±8</td>
<td>152 ±8</td>
<td>150 ±6</td>
<td>148 ±6</td>
</tr>
<tr>
<td>Peak 3</td>
<td>172 ±14</td>
<td>153 ±14</td>
<td>153 ±14</td>
<td>148 ±11</td>
<td>141 ±11</td>
</tr>
<tr>
<td>Peak 4</td>
<td>174 ±14</td>
<td>156 ±14</td>
<td>156 ±14</td>
<td>152 ±8</td>
<td>148 ±8</td>
</tr>
<tr>
<td>Rec 1</td>
<td>169 ±14</td>
<td>155 ±14</td>
<td>153 ±14</td>
<td>150 ±6</td>
<td>148 ±6</td>
</tr>
<tr>
<td>Rec 2</td>
<td>180 ±14</td>
<td>177 ±14</td>
<td>172 ±14</td>
<td>160 ±6</td>
<td>152 ±8</td>
</tr>
<tr>
<td>Rec 3</td>
<td>181 ±14</td>
<td>174 ±14</td>
<td>171 ±14</td>
<td>162 ±14</td>
<td>156 ±14</td>
</tr>
<tr>
<td>Rec 4</td>
<td>181 ±14</td>
<td>174 ±14</td>
<td>171 ±14</td>
<td>162 ±14</td>
<td>156 ±14</td>
</tr>
</tbody>
</table>

Note: *P < 0.05 compared with the corresponding minute in the AP and AA trials; #P < 0.05 compared with the PP and AP trials.

Figure 4. Blood pH values during the trials (n = 8, mean ± s).

*P < 0.05 denotes difference from values before Set B in the PP trial. #P < 0.05 AA vs. AP and PP trials.
respectively, and declined less over the four repetitions (0.39 s) in the AP trial, compared with performance in the PP (1.07 s) and AA (0.85 s) trials.

Set A in the study protocol was designed to induce fatigue and similar metabolic responses in all three trials. Metabolic and performance parameters were successfully reproduced, since force output, stroke rate, oxygen uptake, and heart rate during, and blood lactate and pH after, Set A were similar between trials \((P > 0.05)\). Therefore, the impact of Set A was the same in all three trials and any effect on performance during Set B should be attributed to the different recovery conditions. It was expected that active recovery between Set A and Set B would enhance muscle blood flow, resulting in a faster phosphocreatine resynthesis, lactate removal, and restoration of acid balance. This in turn would improve performance, as shown during repeated 30-s cycling sprints following 4 min active recovery (Bogdanis et al., 1996; Spierer et al., 2004). However, no differences were observed in performance of the first 50-yards sprint of Set B, which was performed following active recovery in trials AP and AA. It has previously been shown that a 50-m sprint performance time was similar following active recovery in trials AP and AA. It has previously been shown that a 50-m sprint performance time was similar following repeated-sprint swims and 6 min of either active or passive recovery (Toubekis et al., 2005, 2006).

Efficient function of the anaerobic metabolic pathways is important for successful performance in 50-yards/metres sprinting (Ring, Mader, Wirtz, & Wilke, 1996). It is likely that following Set A \((4 \times 30\, \text{s at } \sim 156\% \dot{V}O_{2\text{max}})\), phosphocreatine stores would have been significantly depleted and the 5-min interval that was provided between sets was not enough for a complete restoration (Bogdanis et al., 1995). Additionally, phosphocreatine concentrations may be further reduced and the time-course of their restoration will be extended after repeated sprints (Dawson et al., 1997). Factors that may have affected the phosphocreatine resynthesis rate after active recovery \((\text{i.e. muscle blood flow, } O_2 \text{ availability})\) could have an impact on performance. However, performance of the first 50-yard sprint was not affected by the short duration of active recovery applied in the present study. It is likely that the high aerobic fitness of the swimmers \((\dot{V}O_{2\text{max}}: 4.2 \text{ litres} \cdot \text{min}^{-1})\), related to phosphocreatine resynthesis rate (Bogdanis et al., 1995), or the combined active (3-min) and passive (2-min) recovery, may have affected the recovery time. This would mask any positive or negative effect of active versus passive recovery on the phosphocreatine levels before the start of Set B.

Blood lactate after the completion of Set A was the same in all three trials and it was not changed by 5 min active recovery, consistent with other studies (Cazorla et al., 1983). The concentration of this metabolite has no direct effect on fatigue (Gladden, 2004) and is not related to subsequent performance in repeated sprints of short duration (Bodganis et al., 1995; Toubekis et al., 2005, 2006). The concomitant increase in \(H^+\) concentration and decreased muscle pH may have an impact on sprint performance through its direct effect on anaerobic glycolysis. Blood pH was the same after Set A in all three trials and was not changed after 5 min of active or passive recovery. However, similar blood pH values before the start of Set B does not mean that intracellular acidity was the same, as several factors may have affected the ion exchange between muscles and blood \((\text{i.e. blood flow, muscle fibre type, buffering capacity, muscle to blood gradient})\). Nevertheless, an increased blood flow (Bangsbo, Johansen, Graham, & Saltin, 1993) between sets in the AA and AP trials may have removed \(H^+\) faster, restoring the acid balance in the muscle (Sairyo et al., 2003). It is possible, therefore, that muscle \(H^+\) concentration was lower before Set B after active recovery, although any benefit could be minimal due to the short duration of active recovery and therefore not enough to affect performance on the first 50-yard sprint of Set B \((\text{similar in all three trials})\).

Following sprint 1 of Set B, performance declined less in the AP than in the PP or AA trials, although no differences were observed between trials. During repetitions of Set B in the AA trial, performance declined faster than in the AP trial (Figure 2), a similar pattern to that observed when active recovery was used between repetitions in a set of 8 × 25-m swimming sprints (Toubekis et al., 2005, 2006). Aerobic energy has been found to provide about 70% of the total energy by a fourth 30-s cycle sprint (Trump, Heigenhauser, Putman, & Spriet, 1996). It is likely that aerobic metabolism also contributed significantly to energy contribution in the present study. Furthermore, an increased oxygen consumption and contribution of aerobic metabolism after active compared with passive recovery may facilitate the maintenance of performance (Dorado et al., 2004), although this may not be beneficial when there is only a short interval between sprints. During active recovery between the sprints of Set B, heart rate was higher in the AA than in the AP and PP trials (Table II), indicating an increased metabolic rate and a higher aerobic metabolism. Increased metabolic rate during active recovery, despite an increase in oxygen uptake and aerobic energy system activation, may counteract phosphocreatine resynthesis, which is an oxygen-dependent process (Haseler, Hogan, & Richardson, 1999). For instance, a slower reoxygenation of oxyhaemoglobin during active recovery has been reported (Dupont, Moalla, Guinhouya, Ahmaidi, & Berthoin, 2004), and may reflect an
imbalance of oxygen supply and utilization. The aerobic energy contribution may have increased (and the anaerobic energy contribution decreased) in the AA trial because of the active recovery performed between repetitions, resulting in a marked decrease in performance times from sprint 1 to sprints 2, 3, and 4 during the AA trial (Figure 2).

Maintenance of performance in the AP trial is likely to be related to the effect of active recovery between Set A and Set B in combination with the passive recovery between repetitions of Set B. It is likely that any difference in intracellular acidity ($H^+$ removal) due to the increased muscle blood flow during the active recovery (Bangsbo et al., 1993) combined with adequate phosphocreatine resynthesis during the short (~1 min 30 s) passive recovery between sprints of Set B, is reflected in the maintenance of performance. Mean performance times in the AP trial were 2.4% (0.62 s) faster than in the AA trial (95% confidence intervals: 0.7 to 4.1% or 0.17 to 1.08 s), a percentage time change greater than the variation reported for national standard swimmers during competition (i.e. 1.4%) and greater than a performance change that may affect the placing of a swimmer in a competition (i.e. 0.5%; Stewart & Hopkins, 2000).

Performance times were not different between trials, although a reduction in stroke rate and similar stroke length were observed during repetitions of Set B in the AA trial. Decreased stroke rate in repeated-sprint swims caused by altered temporal parameters (increased time to start the catch), and unrelated to blood lactate concentration, has previously been reported (Aujouannet, Bonifazi, Hintzy, Vuillerme, & Rouand, 2006). A decreased stroke rate in swimming may be attributed to a slowing of the muscle relaxation rate. This may be related to an increased $H^+$ and its effects on $Ca^{2+}$ and myosin ATPase (Cady, Elshove, Jones, & Moll, 1989) or to impaired function of the sarcoplasmic reticulum (Allen, Westerblad, Lee, & Lannergren, 1992).

Due to the novel design of the present study, we found that active recovery (at 60% of 100-m pace), compared with passive recovery, does not offer any benefit between repetitions with recovery of short duration (~2 min). It is likely that a different intensity of active recovery may be more effective for subsequent performance in short-recovery, high-intensity repeated-sprint swims. Active recovery at an intensity corresponding to 28% of $VO_2_{max}$ has been reported to induce significant performance improvements during repeated cycling (Spierer et al., 2004). However, swimmers find it very difficult to swim at such slow speeds, as they have to maintain some element of technique efficiency. In addition, it appears that a lower intensity of active recovery (below 60% of 100-m pace) does not demonstrate any further benefit in repeated-sprint performance time (Toubekis et al., 2006). Indeed, an intensity corresponding to the “lactate threshold” has been shown to increase performance in a subsequent 200-yards race (Greenwood et al., 2008). The duration of recovery in previous studies (Greenwood et al., 2008; Spierer et al., 2004) was longer than that in the present study (i.e. 4–10 min vs. 1 min 30 s). It is likely that the intensity and duration of active recovery as well as the interval duration between maximal-intensity repetitions are important factors. These are all likely to have a different impact on the recovery of metabolites (phosphocreatine, $H^+$, lactate) and subsequent performance. Irrespective of the cause of poorer performances, the results of Set B in the AA trial seem to agree with previous studies that performed active recovery between repeated swims of short duration (Toubekis et al., 2005, 2006). Results from this study suggest that passive recovery should be recommended following maximal-intensity repeated-sprint swimming preceding a subsequent set of repetitions, especially when there is only a short rest interval (i.e. <2 min).

In conclusion, active recovery could be beneficial for swimming performance between sets of repetitions, but may compromise performance between sprint repetitions with relatively short rest intervals. An increase in stroke rate and the 2.4% faster (N.S.) performance times in the AP compared with the AA trial may indicate a beneficial effect of active recovery between Sets A and B. Probable benefits include a faster restoration of acid balance within the muscle cell, and adequate resynthesis of phosphocreatine during the passive recovery periods of Set B. It would appear that when active recovery is performed both between sets and between sprints, it may have a detrimental effect on swimming performance, probably because of a slower phosphocreatine resynthesis. This paradoxical effect may indicate that either a different intensity of active recovery is more appropriate, or that a combination of passive rest and active recovery is needed for optimal recovery of performance.

References


