Effect of different intensities of active recovery on sprint swimming performance

Argyris G. Toubekis, Ilios Smilios, Gregory C. Bogdanis, Georgios Mavridis, and Savvas P. Tokmakidis

Abstract: Active recovery reduces blood lactate concentration faster than passive recovery and, when the proper intensity is applied, a positive effect on performance is expected. The purpose of the study was to investigate the effect of different intensities of active recovery on performance during repeated sprint swimming. Nine male well-trained swimmers performed 8 repetitions of 25 m sprints (8 × 25 m) interspersed with 45 s intervals, followed by a 50 m sprint test 6 min later. During the 45 s and 6 min interval periods, swimmers either rested passively (PAS) or swam at an intensity corresponding to 50% (ACT50) and 60% (ACT60) of their individual 100 m velocity. Blood lactate was higher during PAS compared with ACT50 and ACT60 trials (p < 0.05), whereas plasma ammonia and glycerol concentration were not different between trials (p > 0.05). Mean performance time for the 8 × 25 m sprints was better in the PAS compared with the ACT50 and ACT60 trials (PAS: 13.10 ± 0.07 vs. ACT50: 13.43 ± 0.10 and ACT60: 13.47 ± 0.10, p < 0.05). The first 25 m sprint was not different across trials (p > 0.05), but performance decreased after sprint 2 during active recovery trials (ACT50 and ACT60) compared with the passive recovery (PAS) trial (p < 0.05). Performance time for the 50 m sprint performed 6 min after the 8 × 25 m sprints was no different between trials (p > 0.05). These results indicate that active recovery at intensities corresponding to 50% and 60% of the 100 m velocity during repeated swimming sprints decreases performance. Active recovery reduces blood lactate concentration, but does not affect performance on a 50 m sprint when 6 min recovery is provided. Passive recovery is advised during short-interval repeated sprint training in well-trained swimmers.

Key words: swimmers, lactate removal, sprint training.

Résumé : La récupération active diminue plus rapidement la concentration sanguine de lactate que ne le fait la récupération passive. Si l’intensité de la récupération active est bien ajustée, on peut s’attendre à une amélioration de la performance. Le but de cette étude est d’analyser l’effet de différentes intensités de récupération active sur la performance après des épreuves répétées de sprint à la nage. Neuf nageurs bien entraînés participent à une séance de 8 épreuves de sprint sur 25 m à la nage (8 × 25 m) intercalées de périodes de récupération d’une durée de 45 s et suivies, 6 min plus tard, d’un test de performance sur 50 m. Durant les périodes de récupération entre chaque épreuve de sprint sur 25 m et avant le sprint sur 50 m, les nageurs récupèrent au repos (PAS) ou en nageant à 50 % (ACT50) ou à 60 % (ACT60) de leur vitesse maximale sur 100 m. On observe une plus grande concentration de lactate sanguin durant la récupération passive que durant la récupération active (p < 0.05); on n’observe cependant aucune différence entre les concentrations d’ammoniac et de glycérol plasmatiques (p > 0.05). On enregistre un meilleur temps de performance au cours de la séance avec récupération passive comparativement aux séances avec récupération active : PAS, 13.10 ± 0.07 vs. ACT50, 13.43 ± 0.10 et ACT60, 13.47 ± 0.10, p < 0.05. La performance au premier sprint ne varie pas d’une séance à l’autre (p > 0.05), mais comparativement aux séances avec récupération passive, les performances au sprint diminuent après le deuxième dans les séances avec récupération active (p < 0.05). Le temps de performance au sprint sur 50 m accompli après les huit sprints sur 25 m ne varie pas d’une séance à l’autre (p > 0.05). D’après ces observations, la récupération active à des intensités correspondant à 50 % et 60 % de la vitesse sur 100 m au cours de sprints répétés diminue la performance. La récupération active diminue la concentration de lactate sanguin, mais ne modifie pas la performance au cours d’un sprint de 50 m après 6 min de récupération. On recommande donc aux nageurs bien entraînés la récupération passive au cours de séances d’entraînement au sprint répété en intervalles brefs.

Mots clés : nageurs, évacuation du lactate, entraînement au sprint.

[Traduit par la Rédaction]
Introduction

During swimming training, repeated short-duration sprints with maximal or near-maximal effort are widely used to increase anaerobic power (Maglischo 2003). In cases where the resting interval is short, swimming speed cannot be sustained for more than a few repetitions (Toubekis et al. 2005). Previous research has shown that low-intensity active recovery (up to 40% of the VO$_2$$_{max}$) can facilitate the restoration of performance during repeated cycling sprints (Bogdanis et al. 1996b; Signorile et al. 1993; Spierer et al. 2004).

However, other studies showed that active recovery of similar intensity (i.e., 40% of the VO$_2$$_{max}$) can have a detrimental effect on performance during intermittent high-intensity cycling (Dupont et al. 2004) and repeated swimming sprints (Toubekis et al. 2005). The negative effect of active recovery observed in the above studies may possibly be explained by the short duration of the rest interval and the intensity of active recovery. A short recovery period may not be sufficient for removal of metabolic byproduct (i.e., inorganic phosphate (Pi), H$^+$) and for resynthesis of adequate phosphocreatine (PCr) (Bogdanis et al. 1995; Westerblad and Allen 2003). PCr is the most important source for ATP resynthesis during repeated sprints of short duration (Gaitanos et al. 1993; Bogdanis et al. 1998). Since PCr resynthesis is dependent on O$_2$ availability (Haseler et al. 1999) and active recovery may decrease haemoglobin reoxygenation (Dupont et al. 2004), it is possible that a lower intensity of active recovery may decrease its energetic cost and make more O$_2$ available for PCr resynthesis. It is possible that only a very low intensity of active recovery would make more O$_2$ available for PCr resynthesis. This may explain why significant improvements of performance (6%–27%) were observed only when the intensity of active recovery was very low (i.e., 28% of VO$_2$$_{max}$ in Spierer et al. (2004) and 60 W in Signorile et al. (1993)). When the intensity of active recovery was higher (i.e., 40% of VO$_2$$_{max}$ in Bogdanis et al. 1996b) the improvement in performance was marginal (about 2%). The different rates of blood lactate removal observed during recovery after exercise (Baltari et al. 2004; McLellan and Skinner 1982; Stamford et al. 1981; Cazorla et al. 1983) may not be important for performance during repeated sprint exercise (Toubekis et al. 2005; Bogdanis et al. 1996b).

To our knowledge, only one study has examined the effects of different active recovery intensities on performance, by measuring peak torque restoration (McEniery et al. 1997). That study supported the hypothesis that active recovery at a lower intensity (i.e., 30% VO$_2$$_{max}$) is preferable than a higher intensity (i.e., 60% VO$_2$$_{max}$) or a passive recovery for a faster restoration of performance. However, the duration of active recovery in that study was long (16 min; McEniery et al. 1997) and thus the results cannot be directly applied to sports such as swimming where the rest interval during high-intensity training is very short. Therefore, there is a lack of information on the effects of different intensities of active recovery during repeated sprints with a short recovery interval (5–90 s), such as those used during anaerobic swimming training for well-trained swimmers (Maglischo 2003). The purpose of the present study was to examine and compare the effect of different intensities of active recovery and passive recovery (rest) on performance during repeated sprints with short intervals in well-trained swimmers. In the present study, a 45 s resting interval between sprints was chosen to simulate high-intensity training of elite swimmers and to maximize performance impairments during repeated sprints, while allowing sufficient time for a possible benefit during the active recovery intervals between sprints. We hypothesized that the lower intensity of active recovery would be more beneficial for performance maintenance.

Materials and methods

Subjects

Nine male well-trained swimmers (n = 9, age, 18.8 ± 0.7 y; height, 179 ± 2 cm; body mass, 72.3 ± 1.6 kg; VO$_2$$_{max}$, 65.1 ± 1.1 mL·kg$^{-1}$·min$^{-1}$; 100 m time, 55.9 ± 0.6 s) participated in the study, which had the approval of the Democritus University Ethical Committee. The swimmers were informed for the experimental procedure and signed an informed consent statement. All swimmers had qualified and participated at national senior-level competition, 4 of them had competitive experience at international level, and all had at least 8 years of competitive training background. Two of the swimmers were national record holders. During the testing period swimmers trained daily and covered distances of 40–45 km/week.

Preliminary testing

Initially all swimmers performed a maximum oxygen uptake test using tethered swimming. During the tethered swimming test, the load was increased every 3 min up to exhaustion (4–6 stages were completed by each swimmer). Expired air was continuously collected in a mixing chamber (Oxycon Jaeger, Germany) using a special mask (Toussaint et al. 1987) and a low-resistance tube. The oxygen uptake of the last minute of each stage was used to obtain the linear relationship of oxygen uptake vs. heart rate (HR).

Main trials

During 3 separate trials, all swimmers completed 8 repetitions of 25 m sprints (8 × 25 m) interspersed with 45 s intervals, followed by a 50 m sprint test 6 min later. During the interval period separating the 25 m sprint repetitions and 6 min before the 50 m sprint test, swimmers rested passively (PAS) or swam at an intensity corresponding to 50% or 60% of the 100 m velocity (ACT50 and ACT60, respectively). Six to 8 s after each sprint, swimmers were allowed to get ready and take the starting position during the active recovery trials. During the 6 min recovery period a total time of about 2.5 min was used for blood sampling after the 8 × 25 m sprints and before the 50 m sprint test. Therefore this time period (2.5 min) was considered as passive recovery in all 3 trials and the actual active recovery duration was 3.5 min.

The 100 m velocity was recorded during competition within 2–4 weeks preceding the first experimental trial. It has been previously shown that for recreationally trained swimmers the velocity corresponding to 60% of their 100 m velocity elicits 40%–45% of their VO$_2$$_{max}$ (unpublished...
observations) and does not induce a significant increase in blood lactate concentration (Toubekis et al. 2005). The other intensity used in this study (50% of the 100 m velocity) corresponded to the slowest velocity that the swimmers could adopt with a proper technique. A further reduction in the active recovery velocity (i.e., below 0.8 m·s⁻¹) will not cause a significant change in the energetic cost, since the energy cost vs. swimming velocity relationship does not follow the linear pattern at very slow swimming velocities (Holmér 1979). The intensity of active recovery was expressed as a percentage of the 100 m velocity, since during swimming sessions it is easier to refer to an exercise intensity related to a performance time than to a physiological parameter such as the VO₂ max.

HR was continuously recorded in 5 s intervals using short range telemetry (Polar XTrainer plus). An official electronic timing system with an accuracy of 1/1000 s (TanCo timing system) was used for time recording during each sprint. The active recovery swim after each sprint was also timed. The swimmers were familiarized with this type of sprint training and the active recovery pace during their training sessions and had no difficulty following the prescribed pace. However, to avoid any inconsistency of the swimming pace, one of the investigators was walking alongside the pool deck giving pacing instruction when necessary. The procedures have been previously described in detail (Toubekis et al. 2005). All tests took place at the same time of the day in a 25 m indoor swimming pool with a water temperature of 25–26 °C and started 15 min after a controlled warm-up of 1000 m (600 m swim, 200 m kick, 200 m pull, 4 × 10 m sprints). The order of trials was counterbalanced and the front crawl swimming style was used starting with a push-off from within the water in all trials.

**Estimation of energetic cost**

Using a least-square analysis and the obtained individual linear relationship of oxygen uptake and HR during the incremental tethered swimming test (r = 0.97 to 0.99, SEE:2.04 mL·kg⁻¹·min⁻¹) the corresponding mean oxygen uptake during the third, fourth, and fifth minute of the recovery period after the 8 × 25 m sprints was estimated. This time period was the actual duration spent for active recovery (6 min minus the sampling time, as described in Materials and methods). The oxygen uptake estimated from this relationship reflects the energy cost of the active recovery plus the excess post-exercise oxygen uptake resulting from the previously performed sprints. Therefore, no absolute value of oxygen uptake is reported in the results section (the difference in the estimated energetic cost between trials is reported instead).

**Diet and training restrictions**

Two days before the first trial, all participants recorded their diet and were instructed to perform only aerobic sessions during training. They were then asked to repeat the same diet and exercise before each trial. Swimmers were advised to avoid any alcohol or caffeine consumption 24 h before each trial.

**Stroke rate (SR) and stroke length (SL) calculation**

Three complete stroke cycles were timed in the middle of each 25 m sprint distance. The mean SR was calculated by dividing the number of 3 stroke cycles by the time to complete them then multiplying the result by 60. The test–retest reliability of this procedure was tested during sprint swimming (intraclass correlation coefficient r = 0.894, p = 0.001, n = 18). SL was calculated by the quotient of mean velocity during each sprint with the mean SR.

**Blood sampling and analysis**

The swimmers appeared in the pool 30 min before the start of each main trial. They rested for 20 min and then a resting blood sample (5 mL) was collected from an antecubital vein using a venous puncture. One more venous sample was collected during the second minute of recovery after the last 25 m sprint. The venous sample was placed into tubes containing lithium heparin and centrifuged immediately at 4000 r/min (2361 g) for 10 min. Immediately after centrifuging, a part of the supernatant was placed into an ice bath and ammonia was determined within 3 h after sampling using the procedures described by the manufacturer (kit No. 171-UV, Sigma, St. Louis, Mo.; coefficient of variation 4.2%). The remaining plasma was stored at −80 °C and later analyzed for glycerol concentration (Sigma kit No. 337, coefficient of variation 2.2%).

Blood samples (50 μL) were taken from a finger tip after warm-up, at the second minute of recovery after the 8 × 25 m sprints, as well as before and 5 min after the 50 m sprint test. Capillary samples (50 μL) were deproteinized in tubes containing 100 μL trichloroacetic acid. The tubes were centrifuged at 3200 r/min (1522 g) for 10 min, stored at −80 °C, and later analyzed enzymically for blood lactate concentration using a spectrophotometer (Hitachi U-2001) and the procedure described by Sigma (kit No. 826-UV).

Triplicate samples of 75 μL were collected at rest and after the 8 × 25 m into heparinized capillaries. After centrifuging for 15 min at 13 000 r/min (15 682 g) haematocrit was determined. Haemoglobin was measured by the Cyanmethemoglobin method (Sigma kit No. 525). Changes in plasma volume were estimated as described by Dill and Costill (1974).

**Statistical analysis**

Normal distribution of the data was tested using the Kolmogorov–Smirnov test and sphericity was verified by the Mauchly’s test. A 2-way analysis of variance for repeated measures was used to examine differences between means (3 trials × 8 sprints). One-way analysis of variance was used to compare the 50 m sprint test performance and percentage differences of the estimated energetic cost across trials during the third, fourth, and fifth minute of the recovery after the 8 × 25 m sprints. A Tukey’s post-hoc test was used to locate the observed differences and a Pearson product–moment correlation coefficient was used to examine any association between variables. The accepted level of significance was set at p < 0.05. The results are presented as mean ± SEM.

**Results**

**Performance for 8 × 25 m sprints and 50 m sprint test**

Mean performance time for the 8 × 25 m sprints was better.
in the PAS than in the ACT50 and ACT60 trials (PAS: 13.10 ± 0.07; ACT50: 13.43 ± 0.10; ACT60: 13.47 ± 0.10 s, p < 0.05). As can be seen in Fig. 1A, the time for the first 25 m sprint was not different between trials (p > 0.05). However, in the second 25 m bout, a significant deterioration in sprinting performance compared with the first sprint was observed in the ACT50 and ACT60 trials (p < 0.05). No further reduction in performance was seen after the second sprint in these trials. In the PAS trial, performance was maintained until the 6th sprint and a significant deterioration in performance was observed only in the last 2 bouts (Fig. 1A).

Starting from the second sprint, swimmers were swimming slower in the ACT50 and ACT60 trials than in the corresponding sprint in the PAS trial (p < 0.05). The time to complete the 50 m sprint test, 6 min after the 8 × 25 m sprints, was not different between trials (PAS: 27.29 ± 0.20; ACT50: 27.30 ± 0.18; ACT60: 27.45 ± 0.22 s, p > 0.05, Fig. 1A).

**SR and SL for the 8 × 25 m sprints and 50 m sprint test**

The mean SR during the 8 × 25 m sprints decreased after the first sprint, but was similar between trials (PAS: 59.9 ± 2.8; ACT50: 60.5 ± 3.1; ACT60: 58.7 ± 2.9 cycles-min⁻¹; main effect sprints, p < 0.05; main effect trials, p < 0.05; interaction, p > 0.05). The SR during the 50 m sprint test was not different between trials (PAS: 59.6 ± 2.7; ACT50: 62.07 ± 3.4; ACT60: 60.6 ± 2.9 cycles-min⁻¹, p > 0.05). SL was comparable between trials during the 8 × 25 m sprints (PAS: 1.94 ± 0.08; ACT50: 1.89 ± 0.09; ACT60: 1.93 ± 0.08 m-cycle⁻¹, p > 0.05). During the 50 m sprint test, SL was also not significantly different between trials (PAS: 1.87 ± 0.08; ACT50: 1.81 ± 0.09; ACT60: 1.83 ± 0.07 m-cycle⁻¹, p > 0.05)

**HR and differences in the energetic cost during trials**

Peak HR after each 25 m sprint was not different between trials (HR range, PAS: 157–175; ACT50: 155–176; ACT60: 152–176 beats-min⁻¹, p > 0.05). During the 45 s interval period separating the 8 × 25 m sprints from the second and third sprint, HR was increased in the ACT50 and ACT60 compared with the PAS trial (p < 0.05). HR was increased during the ACT60 compared with PAS and ACT50 trials on the third and fourth minutes after the 8 × 25 m sprints (p < 0.05, Fig. 1B). The mean HR during the third, fourth, and fifth minutes of recovery after the 8 × 25 m sprints corresponded to 59% ± 2%, 64% ± 2%, and 69% ± 2% of the maximum HR attained during the VO₂max test for the PAS, ACT50, and ACT60 trials, respectively (p < 0.05). The estimated differences in the energetic cost obtained from the HR vs. VO₂ relationship was 16% ± 6%, 11% ± 3%, 26% ± 4% between PAS vs. ACT50, ACT50 vs. ACT 60, and PAS vs. ACT60 trials, respectively (p < 0.05).

**Blood metabolites**

Blood lactate at the second minute after the 8 × 25 m sprints was higher in the PAS than in the ACT50 and ACT60 trials (p < 0.05). Just before the 50 m sprint test the blood lactate concentration was unchanged in the PAS (p > 0.05), but was reduced in both active recovery trials compared with the value of the post 8 × 25 m sprints (p < 0.05). The percent change of blood lactate concentration during the 6 min recovery period was 6.1% ± 2.7%, 16.0% ± 2.5%, and 15.5% ± 3.7% for PAS, ACT50, and ACT60 trials, respectively (between trials, p < 0.05). Blood lactate concentration 5 min after the 50 m sprint test was still higher in the PAS than in either the ACT50 or ACT60 trial (main effect trials and interaction p < 0.05, Fig. 1C). No relationship was found between blood lactate at the start of the 50 m sprint test and performance during that sprint (r = 0.22, p > 0.05). Plasma ammonia was related to performance time of sprints 6, 7, and 8 (r = 0.41–0.47, p < 0.05), but not with the 50 m sprint test (r = 0.35, p > 0.05).

Plasma ammonia and glycerol concentrations were increased significantly after the 8 × 25 m sprints compared with concentrations at rest (p < 0.05), but no difference was observed between trials (p > 0.05, Table 1). Haemoglobin and haematocrit were increased after the 8 × 25 m trial sprints without any significant difference between trials. The decrease in plasma volume was not different between trials (p > 0.05, Table 1). Thus, correction of blood metabolite concentrations for changes in plasma volume did not affect ammonia and glycerol concentration, since they were similarly affected in between trials (between p > 0.05).

**Discussion**

The results of the present study clearly demonstrated that active recovery at an intensity corresponding to 50% or 60% of the 100 m velocity during the 45 s interval period separating 8 × 25 m sprints resulted in a greater decrease of repeated sprint performance than passive rest. Swiming time of a 50 m sprint test performed 6 min after the series of 8 × 25 m repetitions was not different between trials.

The decreased performance during active recovery in the present study was demonstrated by a significant drop in swimming speed from sprint 1 to sprint 2 during the 8 × 25 m sprint repetitions. Thereafter, no changes in swimming speed occurred in active recovery trials. The percent increase in sprint time from sprint 1 to sprint 8 during both active recovery trials (ACT50 and ACT60) in the present study was much lower (approximately 3%) compared with that observed in a previous study in our laboratory using the same test protocol (12.5%; Toubekis and Tokmakidis 2003). This may be attributed to the fact that swimmers in the present study were well trained and had a high level of aerobic fitness as reflected by their high VO₂max values (65.1 ± 1.1 mL·kg⁻¹·min⁻¹) compared with untrained swimmers (49.4 ± 1.9 mL·kg⁻¹·min⁻¹; unpublished, n = 6). It is well documented that aerobic fitness is related to metabolic recovery and performance restoration during repeated maximal exercise (Bogdanis et al. 1996a; Tomlin and Wenger 2002).

Similarly, the high aerobic fitness of the swimmers in the present study may explain the almost unaffected performance during the passive recovery trial. Maintenance of performance from sprint 1 to 6 in the PAS trial was observed despite the expected large increases in muscle lactate, H⁺, and Pi, and a considerable decrease in PCr, all of which occur during repeated cycling sprints (Bogdanis et al. 1998; Gaitanos et al. 1993). Increased aerobic contribution may have supported performance maintenance during the last sprints (Bogdanis et al. 1996a; Gaitanos et al. 1993). The increased plasma glycerol levels (Table 1) would suggest an
Fig. 1. Sprint time for the 25 and 50 m sprints (A), peak HR after each sprint and during recovery (B), and blood lactate responses (C) during passive (PAS) and active recovery (ACT50 and ACT60). a, $p < 0.05$ compared with the first sprint; b, $p < 0.05$ PAS vs. ACT50 and ACT60 for the corresponding sprint; c, $p < 0.05$ ACT60 vs. PAS; d, $p < 0.05$ ACT60 vs. ACT50; e, $p < 0.05$ compared with the previous sampling time point (mean ± SEM).
is interesting to note that most of the swimmers reported a recovery after a sprint (Bogdanis et al. 1995). However, it has caused similar metabolic perturbations in all conditions. Therefore, the decreased performance on sprint 2 on sprint 1 was similar across trials, thus it can be assumed that the intensity of 60% of the 100 m velocity was selected based on the efficiency of lactate removal (Cazorla et al. 1983), whereas the intensity of 50% was chosen for its lower energetic cost. However, it is possible that the slower swimming velocity of ACT50 may have altered stroke mechanics. We have seen that swimmers use an intensity corresponding to 65%–68% of the 100 m velocity during self-selected active recovery (Toubekis et al. 2005) and that they found it difficult to keep the proper stroke mechanics when swimming at a very slow intensity. Similar observations were reported by Cazorla et al. (1983). A very slow velocity may result in a change of body position (Kjendlie et al. 2004) and this may alter underwater arm movements (Kjendlie et al. 2004) and this may alter underwater arm movements in the ACT60 compared with the ACT50. It has been shown that during slow arm-cranking rate the gross efficiency is decreased at the lower workloads (Smith et al. 2006). Thus, increased efficiency of arm movements in the ACT60 compared with the ACT50 trial may counteract the increased energy cost. It is also likely that the oxygen uptake to velocity relationship is not linear during very slow swimming speeds (Holmér 1979) yielding similar energetic cost, despite different swimming velocities during active recovery in our study (0.89 ± 0.01 m s⁻¹ vs. 1.06 ± 0.02 m s⁻¹).

Increased plasma ammonia levels after maximum-intensity exercise may be an index of energy deficit in the cell (Sahlin 1996). The elevation of plasma ammonia following the 8 × 25 m repetitions in the present study shows the stressful nature of our protocol. Despite a tendency for a higher increase of ammonia concentration in the ACT60 trial, there was no significant difference between trials (p = 0.09, Table 1). Higher plasma ammonia values after active compared with passive recovery have been reported by Bogdanis et al. (1996b), and this may be attributed to increased trials. The use of active recovery during the short rest periods of maximum intensity repetitions may lead to a decreased performance, but could also enhance the muscle’s ability to oxidize lactate, thus leading to the use of other metabolic sources (i.e., aerobic) for performance maintenance.

It would be expected that an increased energetic cost during the ACT60 compared with the ACT50 trial will lead to a greater mismatch between oxygen supply and availability and further delay PCR resynthesis rate. However, performance time for the 8 × 25 m sprints was similar in both active recovery trials. The velocity of active recovery during ACT50 and ACT60 trials was 0.89 ± 0.01 m s⁻¹ and 1.06 ± 0.02 m s⁻¹ (p = 0.00), respectively, and was expected to differentiate the energetic cost. One possible explanation for the similar effects of both active recovery intensities may be that the difference between them was not high enough to affect performance. The intensity of 60% of the 100 m velocity was selected based on the efficiency of lactate removal (Cazorla et al. 1983), whereas the intensity of 50% was chosen for its lower energetic cost. However, it is possible that the slower swimming velocity of ACT50 may have altered stroke mechanics. We have seen that swimmers use an intensity corresponding to 65%–68% of the 100 m velocity during self-selected active recovery (Toubekis et al. 2005) and that they found it difficult to keep the proper stroke mechanics when swimming at a very slow intensity. Similar observations were reported by Cazorla et al. (1983). A very slow velocity may result in a change of body position (Kjendlie et al. 2004) and this may alter underwater arm movements, all of which are related to energy cost (Zamparo et al. 1996). It has been shown that during slow arm-cranking rate the gross efficiency is decreased at the lower workloads (Smith et al. 2006). Thus, increased efficiency of arm movements in the ACT60 compared with the ACT50 trial may counteract the increased energy cost. It is also likely that the oxygen uptake to velocity relationship is not linear during very slow swimming speeds (Holmér 1979) yielding similar energetic cost, despite different swimming velocities during active recovery in our study (0.89 ± 0.01 m s⁻¹ vs. 1.06 ± 0.02 m s⁻¹).

Increased plasma ammonia levels after maximum-intensity exercise may be an index of energy deficit in the cell (Sahlin 1996). The elevation of plasma ammonia following the 8 × 25 m repetitions in the present study shows the stressful nature of our protocol. Despite a tendency for a higher increase of ammonia concentration in the ACT60 trial, there was no significant difference between trials (p = 0.09, Table 1). Higher plasma ammonia values after active compared with passive recovery have been reported by Bogdanis et al. (1996b), and this may be attributed to increased

| Table 1. Haematocrit, haemoglobin, plasma volume changes (PVC), plasma ammonia, and glycerol at rest and after 8 × 25 m sprints. |
|-----------------|-----------------|-----------------|-----------------|-----------------|-----------------|
| Trial | Hematocrit (%) | Hemoglobin (g dL⁻¹) | Plasma ammonia (μmol L⁻¹) | Plasma glycerol (mmol L⁻¹) |
|-------|-----------------|-----------------|-----------------|-----------------|-----------------|
|       | Rest | Post 8×25 m | Rest | Post 8×25 m | Rest | Post 8×25 m | Rest | Post 8×25 m |
| PAS   | 48.4±1.0  | 51.7±0.8  | 15.5±0.3  | 16.2±0.3  | -10.7±1.2  | 32.3±4.9  | 93.5±10.4 | 0.100±0.034 | 0.164±0.018 |
| ACT50 | 48.1±1.1  | 51.2±0.9  | 15.4±0.4  | 16.0±0.4  | -9.1±1.5  | 34.6±6.9  | 111.4±22.2 | 0.095±0.022 | 0.174±0.027 |
| ACT60 | 48.2±0.8  | 51.3±1.1  | 15.4±0.5  | 16.2±0.5  | -10.8±1.0  | 35.3±6.1  | 134.3±16.4 | 0.095±0.017 | 0.149±0.024 |

Note: Values are mean ± SEM, n = 9, p < 0.05 rest compared to post 8 × 25 m for all variables.
efflux from the muscle because of the higher muscle blood flow during active recovery (Bangsbo et al. 1994). Alternatively, higher plasma ammonia levels may indicate an increased energy deficit during active compared with passive recovery, and would further indicate the detrimental effect of active recovery during repeated swimming sprints with short intervals.

Swimming velocity on the 50 m sprint test was not different between trials, despite different blood lactate concentrations during the 6 min recovery period preceding the sprint. In addition, blood lactate concentration at the start of the 50 m sprint test was not related to performance time, as has been also seen in previous studies (Bogdanis et al. 1995; Bogdanis et al. 1996b; Toubekis et al. 2005). This suggests that blood lactate may not be the most important factor for performance recovery during this type of exercise. Furthermore, blood lactate may not correspond to muscle lactate and intracellular acidosis, which are more directly related to reduced performance. A possible explanation for the similar swimming times of the 50 m sprint test after all types of recovery may be that the 6 min rest interval appears to be long enough for complete performance recovery in those athletes. Although the restoration of intramuscular acidity after repeated sprints is a slow process (Bogdanis et al. 1995), resynthesis of PCr is faster and may be complete in that time frame for aerobically fit athletes (Bogdanis et al. 1995; Bogdanis et al. 1996a; Nevill et al. 1997) such as the swimmers of our study. Even further, energy supply from aerobic metabolism is expected to be higher during the 50 m sprint test owing to the effect of the preceding sprints (Bogdanis et al. 1996a). Thus, a possibly decreased anaerobic glycolysis may have been compensated for by an increase in aerobic metabolism and a high PCr contribution.

In conclusion, active recovery at intensities of 50% and 60% of the 100 m velocity, applied between swimming sprints interspersed with a short interval (i.e., 45s), resulted in a lower blood lactate concentration compared with passive recovery, but caused a negative effect on performance. Active recovery during longer intervals (i.e., 6 min) had a similar effect to passive recovery on repeated sprint performance. Repeated 25 m sprints are useful for “lactate tolerance” training when passive recovery is applied. Active recovery can be used during long resting periods (i.e., 6 min) without any impact on performance. The physiological adaptations of training with either active or passive recovery and their effects on competitive swimming performance remain to be examined.

References


