Intensity of exercise recovery, blood lactate disappearance, and subsequent swimming performance

JAMES D. GREENWOOD, G. EDWARD MOSES, F. MARK BERNARDINO, GLENN A. GAESSER, & ARTHUR WELTMAN

Exercise Physiology Laboratory, Curry School of Education, University of Virginia, Charlottesville, VA, USA

(Accepted 5 February 2007)

Abstract

The aim of this study was to examine the effects of active versus passive recovery on blood lactate disappearance and subsequent maximal performance in competitive swimmers. Fourteen male swimmers from the University of Virginia swim team (mean age 20.3 years, $s = 4.1$; stature 1.85 m, $s = 2.2$; body mass 81.1 kg, $s = 5.6$) completed a lactate profiling session during which the speed at the lactate threshold ($V_{LT}$), the speed at 50% of the lactate threshold ($V_{LT.5}$), and the speed at 150% of the lactate threshold ($V_{LT1.5}$) were determined. Participants also completed four randomly assigned experimental sessions that consisted of a 200-yard maximal-effort swim followed by 10 min of recovery (passive, $V_{LT.5}$, $V_{LT}$, $V_{LT1.5}$) and a subsequent 200-yard maximal effort swim. All active recovery sessions resulted in greater lactate disappearance than passive recovery ($P < 0.0001$ for all comparisons), with the greatest lactate disappearance associated with recovery at $V_{LT}$ ($P = 0.006$ and 0.007 vs. $V_{LT.5}$ and $V_{LT1.5}$ respectively) [blood lactate disappearance was 2.1 mmol/L ($s = 2.0$), 6.0 mmol/L ($s = 2.6$), 8.5 mmol/L ($s = 2.8$), and 6.1 mmol/L ($s = 2.5$) for passive, $V_{LT.5}$, $V_{LT}$, and $V_{LT1.5}$ respectively]. Active recovery at $V_{LT}$ and $V_{LT1.5}$ resulted in faster performance on time trial 2 than passive recovery ($P = 0.005$ and 0.03 respectively); however, only active recovery at $V_{LT}$ resulted in improved performance on time trial 2 ($TT_2$) relative to time trial 1 ($TT_1$) ($TT_2 - TT_1$: passive +1.32 s ($s = 0.64$), $V_{LT.5}$ +1.01 s ($s = 0.53$), $V_{LT}$ −1.67 s ($s = 0.26$), $V_{LT1.5}$ −0.07 s ($s = 0.51$); $P < 0.0001$ for $V_{LT}$). In conclusion, active recovery at the speed associated with the lactate threshold resulted in the greatest lactate disappearance and in improved subsequent performance in all 14 swimmers. Our results suggest that coaches should consider incorporating recovery at the speed at the lactate threshold during competition and perhaps during hard training sessions.

Keywords: Lactate threshold, aerobic capacity, athletes, post exercise, endurance

Introduction

During competition, swimmers often face a preliminary and final race format that results in multiple races in a day. This might require swimmers to swim multiple events within short periods of time, sometimes with only minutes separating heats. It has become common practice during competitions for swimmers to use active recovery between events to facilitate better performance. However, recovery intensities are generally self-selected and range from slow pace to near maximal pace.

Previous research has indicated that active recovery is better than passive recovery for blood lactate disappearance (McMaster, Stoddard, & Duncan, 1989a; Reaburn & Mackinnon, 1990; Stamford, Weltman, Moffatt, & Sady, 1981; Weltman, Stamford, & Fulco, 1979; Weltman, Stamford, Moffatt, & Katch, 1977). However, lower concentrations of blood lactate have not necessarily been associated with improved subsequent performance in laboratory settings (Weltman et al., 1977, 1979). Although few studies have examined active versus passive recovery after an all-out bout of swimming, these studies examined lactate disappearance relative to intensities based on maximum speed, with no consideration of the blood lactate response to exercise (McMaster et al., 1989a; Reaburn & Mackinnon, 1990). Previous research has suggested that the optimal recovery intensity for blood lactate disappearance should be based on intensities that are prescribed relative to the blood lactate response to exercise (Weltman, 1995; Weltman et al., 1990). The intensity associated with the lactate threshold is thought to be the optimal recovery intensity, since it is associated with a speed that should promote...
maximal lactate disappearance without additional lactate accumulation. This recovery strategy is better than the use of a percentage of maximum speed, as there is considerable variability in the lactate threshold among swimmers, and thus the use of percent maximum speed cannot be used as a surrogate measure of blood lactate concentration. In addition, to our knowledge, none of the studies of swimmers examined subsequent swimming performance.

Accordingly, the aims of this study were: (1) to examine blood lactate disappearance rates following all-out swim performance using the blood lactate response to exercise when prescribing exercise recovery intensity, and (2) to determine whether enhanced lactate disappearance would result in improved subsequent performance. We hypothesized that active recovery at the lactate threshold would result in greater lactate disappearance relative to passive recovery and recovery above and below the lactate threshold. Furthermore, because most competitive swimming performances are between 50 and 200 m, corresponding to approximately 0.33–2.5 min, we also hypothesized that lower pre-exercise blood lactate concentration would be associated with improved subsequent performance.

**Methods**

Fourteen males (mean age 20.3 years, SD = 4.1; stature 1.85 m, SD = 2.2; body mass 81.1 kg, SD = 5.6) completed the study. All participants were members of the varsity swim team at the University of Virginia. Before the study began, all the experimental procedures, benefits and risks of the study were explained to the participants, and they each provided written informed consent. The study was approved by the Human Investigation Committee of the University of Virginia. Each participant completed an initial lactate profiling session and four randomly assigned experimental sessions. The four experimental sessions were separated by ~1 week. All testing took place at the Aquatics and Fitness Center at the University of Virginia.

The lactate profiling protocol has been described previously by Pyne, Lee, and Swanwick (2001). Briefly, the protocol involves seven graded incremental 200-m freestyle swims with the first (baseline) swim target time being 30 s slower than the individual’s best 200-m time for the present participants, 150.79 s (SD = 8.77). The subsequent swims all had a target time 5 s faster than the swim before it. Before the initial swim and following each swim, a blood sample (1–2 drops of blood) was drawn from the ear or finger and lactate concentration analysed with an Accutrend portable lactate analyser (Roche Diagnostics, Mannheim, Germany). The total time for each stage was 5 min. All swims began from a push start and an emphasis was placed on even pacing for the entire 200 m. Although pacing was the responsibility of the participant, pace clocks were in use at several locations lining the pool deck. A 50-m long-course set up was used to minimize the number of turns during the lactate profiling session. From these profiling data, the speed–blood lactate relationship was determined. The speed at the lactate threshold (VLT) was defined as the highest speed attained before the curvilinear increase in blood lactate with increasing speed (Weltman, 1995). Speed at 50% of the lactate threshold (VLT.5) was defined as 50% of the difference between baseline speed and VLT. Speed at 150% of the lactate threshold (VLT1.5) was defined as 50% of the difference between maximum speed and VLT.

Each experimental trial began with a warm-up session equivalent to what each participant would use to warm up for a competition. To simulate college swim competition, a short course (25-yard) was employed. The warm-ups were designed by the coaches and varied from 1000 to 3000 yards with mostly slow aerobic swimming but with brief bouts of sprinting included. Not all swimmers had the same warm-up, as distance swimmers swim further than sprinters, as is common practice before competitions. Each individual in any particular group (sprint, stroke, distance) completed the identical warm up prior to each of their experimental trials. Each participant then completed a 200-yard maximal-effort swim in their primary stroke (freestyle, n = 7; individual medley, n = 3; butterfly, n = 2; backstroke, n = 2) and time was recorded manually with a stopwatch. The participant then completed 10 min of recovery at one of four intensities (passive, VLT.5, VLT, VLT1.5), the order of which was randomly assigned. Recovery swims were all freestyle, as the lactate profiling session was freestyle and because swimmers, regardless of primary stroke, tend to use freestyle for recovery. Passive recovery involved sitting in a chair or on the pool deck. Following recovery, each participant completed a second 200-yard maximal-effort swim. Blood lactate concentrations were measured before and immediately after swim 1, after recovery just before swim 2, and immediately after swim 2.

A 4 x 4 analysis of variance (ANOVA) with repeated measures was used to test for significant differences among blood lactate concentrations (P < 0.05). A separate 2 x 4 ANOVA was used to test for differences between time trials. Where an interaction between swim performance and recovery was observed, separate one-way ANOVAs with repeated measures were performed on the difference scores. All data are presented as means and standard deviations (SD).
Results

Figure 1 presents a representative blood lactate–speed relationship during the lactate profiling session. For this participant, his best 200-m freestyle time was 115 s. Therefore, the initial target time was set at 145 s and subsequent 200-m swims were reduced by 5 s. For this participant, $V_{LT}$ was chosen to be 85.7 m·min$^{-1}$ (70.0 s·100 m$^{-1}$), since this was the highest speed achieved before the curvilinear increase in blood lactate concentration. The speed at 50% of the lactate threshold was 84.3 m·min$^{-1}$ (64.3 s·100 m$^{-1}$). The speed at 150% of the lactate threshold was 93.3 m·min$^{-1}$, since this was the highest speed achieved before the curvilinear increase in blood lactate concentration. The mean recovery speeds for the entire group were as follows: 75.8 m·min$^{-1}$ ($s = 6.7$), 78.3 m·min$^{-1}$ ($s = 6.0$), and 85.2 m·min$^{-1}$ ($s = 4.5$) for $V_{LT.5}$, $V_{LT}$, and $V_{LT1.5}$ respectively.

Figure 2 presents the blood lactate responses for the four exercise recovery conditions. Analysis of variance with repeated measures revealed no differences between conditions for pre exercise, post time trial 1, and post time trial 2. Blood lactate concentrations ranged from 1.8 to 2.2, 9.2 to 10.5, and 9.5 to 10.6 mmol·l$^{-1}$ for pre exercise, post time trial 1, and post time trial 2 respectively. The mode of recovery between time trials affected lactate disappearance, as lower blood lactate concentrations were observed just before time trial 2 in all three active recovery conditions compared with passive recovery ($P < 0.0001$) [passive 7.1 mmol·l$^{-1}$ ($s = 2.6$), $V_{LT.5}$ 4.0 mmol·l$^{-1}$ ($s = 1.5$), $V_{LT}$ 3.1 mmol·l$^{-1}$ ($s = 1.1$), $V_{LT1.5}$ 3.8 mmol·l$^{-1}$ ($s = 1.9$)]. The blood lactate concentration following recovery at $V_{LT}$ was lower than for the other two active recovery conditions ($P = 0.02$ and 0.14 vs. $V_{LT.5}$ and $V_{LT1.5}$ respectively). Analysis of difference scores (pre and post recovery) revealed significantly greater lactate disappearance for $V_{LT}$ relative to all other conditions ($P < 0.0001$ vs. passive recovery, $P = 0.006$ and 0.007 vs. $V_{LT.5}$ and $V_{LT1.5}$ respectively), while passive recovery had significantly less lactate disappearance relative to all other conditions ($P < 0.001$ for all comparisons) [blood lactate disappearance: passive 2.1 mmol·l$^{-1}$ ($s = 2.0$), $V_{LT.5}$ 6.0 mmol·l$^{-1}$ ($s = 2.6$), $V_{LT}$ 8.5 mmol·l$^{-1}$ ($s = 1.8$), $V_{LT1.5}$ 6.1 mmol·l$^{-1}$ ($s = 2.5$)].

Figure 3 shows the results of the time trial performance. No differences were observed for performance times for time trial 1 across conditions [passive 114.7 s ($s = 5.8$), $V_{LT.5}$ 113.6 s ($s = 4.9$), $V_{LT}$ 114.1 s ($s = 4.8$), $V_{LT1.5}$ 114.4 s ($s = 5.5$)]. Recovery intensity affected performance on the second time trial. Performance during time trial 2 following recovery at $V_{LT}$ and $V_{LT1.5}$ was significantly faster than performance following passive recovery ($P = 0.0005$ and 0.03 respectively). Performance following recovery at $V_{LT.5}$ was not different from passive recovery. In addition, performance following recovery at $V_{LT}$ was faster than
performance following all other recovery conditions ($P = 0.0005$ for passive and $P = 0.02$ for $V_{LT.5}$ and $V_{LT1.5}$) [passive 116.0 s ($s = 5.6$), $V_{LT.5}$ 114.6 s ($s = 3.8$), $V_{LT}$ 112.4 s ($s = 4.6$), $V_{LT1.5}$ 114.3 s ($s = 5.4$)]. Additional analyses revealed that performance on time trial 2 ($T_2$) was faster than on time trial 1 ($TT_1$) following recovery at $V_{LT}$ ($P < 0.0001$) [$TT_2 - TT_1$: passive $+1.32$ s ($s = 2.39$), $V_{LT.5}$ $+1.01$ s ($s = 1.98$), $V_{LT}$ $-1.67$ s ($s = 0.97$), $V_{LT1.5}$ $-0.07$ s ($s = 1.91$)].

Figure 4 shows the individual and mean responses for the differences between performance for time trial 1 and time trial 2 for each of the recovery conditions. The individual primary strokes are represented by different symbols (see figure legend). Recovery at $V_{LT}$ resulted in improved performance for all 14 swimmers on time trial 2 ($TT_2 - TT_1$, negative difference indicates faster performance on time trial 2). For the other recovery conditions, individual responses varied. Inspection of the standard deviations of the change scores revealed that recovery at $V_{LT}$ resulted in the least variation in individual response ($s = 0.97$ s) compared with the other recovery conditions ($s = 1.91 - 2.39$ s). Recovery appeared to impact all primary strokes in a similar manner. There was a correlation between lactate disappearance (post $TT_1$ - pre $TT_2$) and improved performance ($TT_2 - TT_1$) ($r = 0.49$, $P = 0.0001$).

**Discussion**

The main findings of the present study were that lactate disappearance was facilitated and that subsequent 200-yard swimming performance was enhanced by active recovery at the speed associated with the lactate threshold (Figures 3 and 4).

The results of the present study are in line with those of previous ones that reported that active recovery is better than passive recovery for lactate disappearance from the blood (Bangsbo, Graham, Johansen, & Saltin, 1994; Belcastro & Bonen, 1975; Bonen & Belcastro, 1976; McMaster, Stoddard, & Duncan, 1989b; Monedero & Donne, 2001; Reaburn & Mackinnon, 1990; Stamford et al., 1981; Weltman et al., 1977). For example, McMaster et al. (1989b) showed that active recovery at 65% of maximum speed enhanced lactate clearance following all-out swimming compared with passive recovery, but failed to show any difference in lactate clearance during active recovery at intensities of 55%, 65%, and 75% of maximum speed (McMaster et al., 1989a). The results of the present study revealed that maximum lactate disappearance occurred at the speed associated with the lactate threshold. For the participants in the present study this represented 86% of maximum speed, which is higher than the recovery intensities used by McMaster et al. (1989a). The standard of the present swimmers was comparable to that of those recruited by McMaster et al. (1989a, 1989b), who were senior national swimmers; all participants in the present study had senior national qualifying times. The likely reason for the discrepancy between the results of the present study and those of McMaster et al. (1989b) is that they did not base their recovery patterns on the blood lactate response to exercise.
recovery is that this percentage concept does not adequately discriminate across participants when the blood lactate response to exercise is used as a criterion (Weltman et al., 1990). Therefore, some of their participants could have exceeded the lactate threshold at the 75% maximum speed recovery pattern, and had they chosen 85% maximum recovery speed variability would likely have increased.

Blood lactate concentration during exercise and recovery is a complex phenomenon influenced by a combination of lactate production by the exercising muscle, blood flow, and lactate uptake and oxidation by the liver, heart, and skeletal muscle (Bangsbo et al., 1994; Brooks, Brauner, & Cassens, 1975; Jorfeldt, 1970; Rowell, 1974; Rowell et al., 1966). As exercise intensity increases, blood flow to tissues that can oxidize lactate (e.g. heart and skeletal muscle) is increased (Weltman et al., 1990). If, however, the intensity of exercise exceeds the lactate threshold, then lactate might begin to accumulate in the blood and counter the effects of lactate disappearance. Bangsbo et al. (1994) reported that total blood and muscle lactate concentration following active recovery was lower compared with passive recovery. Furthermore, they determined that this difference in muscle lactate was not explained by a larger release of lactate into the blood but rather due to an increase in lactate metabolism in the active muscle. This could translate to improved subsequent performance.

The results of the present study support this notion in that subsequent performance was enhanced by active recovery immediately following all-out performance. To our knowledge, there are no comparable data on swimmers. However, the present data support those previously reported for cycling and weightlifting. Corder and colleagues (Corder, Potteiger, Nau, Figoni, & Hershberger, 2000) found that active recovery between sets of parallel squats (pedalling on a cycle ergometer at 25% of the power output associated with the onset of blood lactate accumulation, defined as 4.0 mmol·l⁻¹) enhanced the total number of repetitions performed in a test set following six sets of 10 repetitions at 85% of 10 repetitions maximum. Weltman et al. (1977) showed that, compared with passive recovery, active recovery following 1 min of cycling improved post recovery pedal revolutions during a second 1-min cycling bout. In contrast, in a second study Weltman et al. (1979) revealed that active recovery did not enhance performance during a 5-min bout of maximal-effort cycling. However, inspection of their figure 2 shows that in the first few minutes of the second exercise bout, more exercise was performed following active recovery than passive recovery. These data support the present findings, as participants were not allowed to pace themselves during the all-out maximal-effort cycling bouts (Weltman et al., 1979). The results of Monedero and Donne (2001) were consistent with the latter findings. They found that both 15 min of active recovery alone at 50% of maximal oxygen uptake ($\dot{V}O_{2max}$) and a combined programme of 7.5 min of massage and 7.5 min of recovery at 50% $\dot{V}O_{2max}$ lowered lactate compared with passive recovery. However, neither routine improved subsequent performance, although the combined programme was better at sustaining performance.

It is possible that active recovery that enhances lactate disappearance is beneficial to performance on short, intense activities such as competitive swimming and that it becomes less important in longer activities where a larger percentage of energy is derived from aerobic pathways. Intense physical activity leads to an accumulation of muscle lactate. The resulting pH change inhibits glycolytic enzymes such as lactate dehydrogenase and phosphofructokinase and muscular contraction might be impaired (Karlsson, Hulten, & Sjödin, 1974; Trivedi & Danforth, 1966). Therefore, the ability effectively to remove lactate from the muscle and blood should translate to improved performance in short-duration high-intensity activities. However, modest elevations in blood (and presumably muscle) lactate concentrations do not appear to impair subsequent performance because swim performance on the second time trial was faster following recovery at $V_{LT}$ than it was on the first time trial. It is unlikely that the participants, knowing that they had two all-out swims to complete, did not give maximal effort on the first time trial because only one condition ($V_{LT}$) resulted in a faster performance on the second time trial. Although we did not measure muscle lactate concentration or acid–base status, it could be that recovery at $V_{LT}$ results in the lowest muscle lactate concentrations before time trial 2 (Bangsbo et al., 1994).

The results of the present study have implications for swimming performance (we also speculate that similar results would be observed for other disciplines that involve repeated high-intensity short-term performance, such as sprinting). Swimming is one of several sports where multiple races might be completed in short periods of time during competition, with sometimes only minutes separating heats. As improved lactate disappearance is associated with improved subsequent performance following active recovery compared with passive recovery, with recovery at the speed associated with the lactate threshold resulting in the greatest lactate disappearance and the fastest time trial performance, we suggest that a recovery swim should be performed at $V_{LT}$. In the past, coaches and athletes have had to use subjective means to determine what speed would
best serve them. Self-selected recovery paces have been commonplace. When we questioned the athletes involved in the present study, they revealed that they normally recovered at a considerably slower speed than the speed that was associated with their individual lactate threshold. Although we recognize that in some situations swimmers have longer than 10 min to recover, 10 min of passive recovery only lowered blood lactate from 9.2 to 7.1 mmol·l⁻¹ (suggesting that a considerable recovery time would be required for complete lactate disappearance). With the availability of inexpensive, efficient, portable lactate testing, it is possible for coaches to employ a more structured approach to post-competition recovery. A simple lactate profiling on each athlete should allow the athlete to recover at his or her lactate threshold and better facilitate post race recovery. Furthermore, using active recovery at the LT during recovery sets following very hard practice swims might translate into more productive training. This notion needs to be examined in future studies.

References


