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ORIGINAL RESEARCH

Effect of self-paced active recovery and passive recovery on blood lactate removal following a 200 m freestyle swimming trial

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Purpose: The aim of this study was to investigate the effect of self-paced active recovery (AR) and passive recovery (PR) on blood lactate removal following a 200 m freestyle swimming trial.
Patients and methods: Fourteen young swimmers (with a training frequency of 6–8 sessions per week) performed two maximal 200 m freestyle trials followed by 15 minutes of different recovery methods, on separate days. Recovery was performed with 15 minutes of passive rest or 5 minutes of passive rest and 10 minutes of self-paced AR. Performance variables (trial velocity and time), recovery variables (distance covered and AR velocity), and physiological variables (blood lactate production, blood lactate removal, and removal velocity) were assessed and compared.
Results: There was no difference between trial times in both conditions (PR: 125.86±7.92 s; AR: 125.71±8.21 s; p=0.752). AR velocity was 69.10±3.02% of 200 m freestyle trial velocity in AR. Blood lactate production was not different between conditions (PR: 8.82±2.47 mmol L^{-1}; AR: 7.85±2.05 mmol L^{-1}; p=0.069). However, blood lactate removal was higher in AR (PR: 1.76±1.70 mmol L^{-1}; AR: 4.30±1.74 mmol L^{-1}; p<0.001). The velocity of blood lactate removal was significantly higher in AR (PR: 0.18±0.17 mmol L^{-1} min^{-1}; AR: 0.43±0.17 mmol L^{-1} min^{-1}; p<0.001).
Conclusion: Self-paced AR shows a higher velocity of blood lactate removal than PR. These data suggest that athletes may be able to choose the best recovery intensity themselves.
Keywords: athletic performance, fatigue, acidosis, anaerobic, metabolic response, sports

Introduction

In the beginning of any moderate exercise, the blood lactate concentration ([Lac]) rises. However, after a while, depending on the intensity of the activity, [Lac] decreases due to the variable relation between production and removal of lactate.[1] The lactate threshold represents the intensity in which [Lac] remains constant, due to a balance between glycolytic production rate and pyruvate conversion rate, and corresponds to a level of 4 mmol L^{-1}.[2] From then on, during an exercise more intense than the lactate threshold, [Lac] increases exponentially, due to a rise in lactate production and liberation by the active skeletal muscle tissue, a reduction in blood lactate uptake and removal, and an imbalance between the rate of lactate production and removal.[2,3]

Lactate is released from the dissociation of lactic acid, produced through the anaerobic reaction of glucose. This dissociation produces, as a subproduct, hydrogen ions (H^+).[4,5] Although increased [Lac] has little or no effect on muscular contraction, the increase of H^+ induces decrease of pH, metabolic acidosis, and insufficiency in the glycolytic metabolism due to the suppression of glycogen phosphorylase and

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phosphofructokinase enzymes. Additionally, changes in systemic acidity promote deficiency in muscle contraction inhibiting myofibrillar ATPase and calcium reabsorption.[4,6] This decrease of muscular function, although limited, might negatively affect work capacity. Moreover, acidosis indirectly affects the feeling of pain and discomfort, through the activation of nerves from groups III and IV of the afferent fiber.[6,7]

Despite the little influence in muscular activity, studies analyzing [Lac] are frequent and important, for this metabolite presents, even if indirectly, a precise estimation of acidosis, besides being involved in the metabolic process of energy conversion in the skeletal muscle, heart, and brain.[8] According to Boning et al,[9] the lactate production rate during and after exercise is in a proportion of 1:1 with the H^+ release rate. Therefore, lactate is related to the reduction in muscle work and is an efficient fatigue indicator, even though it is not responsible for the decrease in force production capacity.[10]

In virtue of the search for the best performance and the physical and physiological exigence in which athletes are put through, the search for the most effective recovery strategy becomes crucial.[11] Studies assessing the difference between active recovery (AR) and passive recovery (PR) initiated over two decades ago.[12] However, the existence of a consensus about the best type of recovery seems quite distant. AR is suggested in some cases after prolonged exercises for it provides reduction in acidity and acceleration of lactate removal.[13] In events <3 minutes, on the other hand, AR may not affect [Lac], although it provides increases in peak power output.[14]

In this sense, the choice of the appropriate recovery modality depends on the activity performed and on the protocol that was adopted.[14] PR seems to be more suitable for the maintenance of performance in repetitive trials, which comprise high intensity and short duration.[15] This occurs because AR promotes a significant reduction on the phosphocreatine (PCr) levels, changing the capacity for force production in high velocity.[16] On the other hand, AR seems to potentiate pH restoration and lactate removal by activating oxidative metabolism.[12,17–19] Previous studies affirm that the accelerated decrease of [Lac] in AR is due to an increase of the blood flow promoted by activity, carrying lactate to the liver, heart, and skeletal muscle in activity, activating oxidative metabolism and favoring gluconeogenesis.[13,17–21] Nevertheless, Toubekis et al[22] showed that PR has the same removal velocity and demonstrated the benefits achieved by AR performed during 5 minutes at 60% of the maximum effort.

Another relevant variable that defines the effectiveness of AR is the intensity to be adopted. Several studies investigated the best intensity to accelerate the restoration of pH, potentiate blood lactate removal, and improve performance in several modalities,[23–26] not reaching a consensus. Toubekis et al[26] investigated in swimmers the difference in performance and lactate removal between two AR intensities after a swimming trial. Its results neither did show any difference in lactate removal nor in the performance between ARs carried out at 50% and 60% of the 100 m trial velocity, probably due to the proximity between the two intensities. In fact, the performance at 50% might have been considerably slow, to the point of altering the swimming technique. Greenwood et al[23] analyzed the effect of AR at 50%, 100%, and 150% of the lactate threshold on performance and lactate removal. The main findings showed the superiority of AR performed at lactate threshold both in the decrease of [Lac] and in the performance in subsequent events. This intensity is justified for it is the moment when oxygenation, blood flow, and lactate conversion metabolism are high, without an imbalance between production and removal of this metabolite. However, AR performed at high intensities might promote reduction in the PCr stocks, increase in muscle damage, and reduction in performance.[16,25] Thus, self-paced AR seems to be effective in lactate removal after submaximal exercise without compromising energetic stocks.[24]

Despite contradictory propositions about the modality and intensity of the ideal recovery, a higher lactate removal rate seems to be related to an improvement in performance.[17] Therefore, this study is justified in the uncertainties about the most effective type of recovery and in the influence of the specificity of the trial in choosing the ideal intensity and type. The aim of this research was to analyze the blood lactate removal velocity in self-paced AR and PR after a 200 m freestyle trial.

Materials and methods

Subjects

Fourteen high-performance swimmers (7 men, age: 17.71±1.25 years; body mass: 69.13±5.85 kg; height: 1.79±0.05 m; and 7 women, age: 18.29±1.60 years; body mass: 60.66±4.84 kg; height 1.65±0.04 m) participated in this study. All athletes had at least 4 years of experience with competitions, a training frequency of 6–8 sessions per week, with a training volume of 35,428.57±3,715.13 m per week. Prior to the experiments, the swimmers and their parents were informed about the experimental design and its procedures, and signed an informed consent form. All procedures had the approval of the University Center of Brasilia's Ethics

Committee in Research and were conducted according to the Helsinki declaration.

Experimental design

This study was performed in a randomized crossover design. Each participant completed two 200 m freestyle trials at maximum speed followed by a 15-minute rest under two experimental conditions: 15 minutes of PR and 5 minutes of PR followed by 10 minutes of self-paced AR. The sessions were performed in a counterbalanced order, 72 hours apart. During PR, the athletes remained lying down covered by a towel. During AR, the athletes swam in a self-selected velocity, which was recorded by the researcher. Each athlete performed the 200 m freestyle trial at maximum speed individually, in order to avoid any influence of rhythm and/or strategy.

In the PR protocol, the swimmers initiated PR immediately after the end of the test and remained in the same position for 15 minutes. In the AR protocol, the swimmers performed a 5-minute PR initiated immediately after the end of the trial, followed by a 10-minute self-paced AR. In this protocol, 5 minutes of PR were used to provide identification of the peak blood [Lac], since the production of this metabolite remains higher than its removal 3–5 minutes after the end of the trial.[3,22,27,28]

Before the 200 m freestyle trial, on both days, the swimmers performed a 1,400-m warm-up, similar to the one performed in competitions, viz 400 m of light-intensity swimming, 200 m leg kick, 200 m stroke, 4×50-m swimming drills, 4×50 m with progressively increasing speed, and 200 m of light-intensity swimming. The 200 m freestyle trials at maximum velocity started 10 minutes after the warm-up, with commands and procedures identical to the ones used in official competitions, using the front-crawl swimming style.

Time was recorded by two experienced coaches using digital chronometers (Seiko S141; Seiko Holdings Corporation, Tokyo, Japan). The mean value was used for analysis. The mean velocity for the 200 m freestyle trial was calculated by the ratio between the distance covered (200 m) and the time recorded. Likewise, the swimming velocity in AR was calculated by the ratio between the distance covered and the swimming time (10 minutes). All procedures were conducted in a 25-m outdoor swimming pool with water temperature between 26°C and 28°C, during the same period of the day (4:00–6:00 PM).

Blood lactate analysis

Capillary blood samples (5 μL) were collected in order to measure the blood [Lac] through a puncture of the ring finger's distal phalanx of the nondominant hand. Before collection, in the local region of the puncture, asepsis was ensured, using alcohol 70°. The first drop was discarded; then, the sample was analyzed in an Accutrend Plus lactate monitor (Roche Brasil, São Paulo, Brazil).[29] Blood was collected immediately before the test (Pre), and 5 minutes (5Post) and 15 minutes (15Post) after the end of the 200 m freestyle trial. The production range (Δprod) was calculated by subtracting [Lac] 5 minutes after the trial from the [Lac] before the trial, in both conditions. The removal range (Δrem) was calculated by subtracting the [Lac] 15 minutes after the trial from the [Lac] 5 minutes after the trial, in both conditions. The blood lactate removal velocity was calculated by the ratio between Δrem and the elapsed time (10 minutes), in both conditions.

Statistical analysis

Descriptive analysis was used to calculate mean and standard deviation of all variables. Normality of the data was verified by the Shapiro–Wilk test and parametric statistic was adopted. [Lac] in both conditions (PR and AR) and at the three moments (Pre, 5Post, and 15Post) was assessed by analysis of variance for repeated measures on two factors (condition × time) with Bonferroni's method in order to identify any difference between the means. The durations of the two 200 m freestyle trials were compared by the paired *t*-test. Δprod, Δrem, and removal velocity in both conditions (PR and AR) were compared by the paired *t*-test. All statistical analyses were performed using SPSS version 21.0 (IBM Corporation, Armonk, NY, USA). The effect size and Cohen's coefficient *d* were calculated by the software G-Power 3.1.9.2 for Mac OS X (University of Kiel, Germany). For all analysis, $p \leq 0.05$ was adopted as the significance level.

Results

Performances in the 200 m freestyle trial in the PR and AR conditions clocked 125.86±7.92 and 125.71±8.21 seconds, respectively. There was no difference in the time of performance of the trials in the two conditions ($p=0.752$; Table 1). The swimming velocity for the two 200 m freestyle trials did not present any significant difference between the two conditions ($p=0.686$; Table 1). During AR, the athletes swam 661.43±39.78 m and the swimming velocity was 1.10±0.07 m s^{-1}. The swimming velocity during AR corresponded to 69.10±3.02% of the swimming velocity in the 200 m freestyle trial in the AR condition.

There was no difference in [Lac] in the Pre moment between both conditions (PR: 3.45±0.62 mmol L^{-1}; AR: 3.79±0.89 mmol L^{-1}; $p=1.000$). [Lac] significantly increased

Table 1 Performance and recovery variables in 200 m freestyle trials and recovery in passive and active recovery conditions (n=14)

Variables	Condition (mean ± SD)				
	Passive recovery	Active recovery	*p*-value	Cohen's *d*	ES (r)
200 m time (s)	125.86±7.92	125.71±8.21	0.752	0.019	0.009
200 m velocity (m s^{-1})	1.59±0.10	1.60±0.10	0.686	0.100	0.050
Δprod (mmol L^{-1})	8.82±2.47	7.85±2.05	0.069	0.209	0.427
Δrem (mmol L^{-1})*	1.76±1.70	4.30±1.74	<0.001	1.477	0.594
Removal velocity (mmol L^{-1} min^{-1})*	0.18±0.17	0.43±0.17	<0.001	1.471	0.592

Notes: Δprod: blood lactate production; Δrem: blood lactate removal. *$p<0.05$.
Abbreviations: SD, standard deviation; ES, effect size.

after the trial, both in the PR condition (5Post: 12.27±2.20 mmol L^{-1}; Pre: 3.45±0.62 mmol L^{-1}; $p<0.001$) and AR condition (5Post: 11.64±2.32 mmol L^{-1}; Pre: 3.79±0.89 mmol L^{-1}; $p<0.001$). Differences in [Lac] between the PR and AR conditions at the 5Post moment ($p=1.000$) were not observed. [Lac] decreased significantly at the 15Post moment in relation to the 5Post moment, both in PR (15Post: 10.51±1.52 mmol L^{-1}; 5Post: 12.27±2.20 mmol L^{-1}; $p=0.038$) and in AR (15Post: 7.34±2.27 mmol L^{-1}; 5Post: 11.64±2.32 mmol L^{-1}; $p<0.001$) conditions. [Lac] at the 15Post moment was significantly lower in the AR condition compared to the PR condition ($p=0.001$) (Figure 1).

Blood lactate production (Δprod) did not present a significant difference between the two conditions ($p=0.069$; Table 1). However, the blood lactate removal (Δrem) was greater and removal velocity significantly greater in AR ($p<0.001$; Table 1).

Discussion

This study aimed to analyze the blood lactate removal velocity in self-paced AR and PR after a 200 m freestyle trial, in order to propose the most adequate recovery modality for the reduction of this metabolite. The results suggest a higher lactate removal velocity during the self-paced AR performed for 10 minutes. Data were obtained in highly trained athletes and suggest a possible application aiming to improve the performance in subsequent trials, since a higher lactate removal rate seems to relate to an enhancement in the performance.[17]

These results corroborate other studies which had investigated the effects of AR and PR on lactate removal by suggesting greater effectiveness of AR.[17,20,22,23,26] Greenwood et al,[23] for example, showed that the lactate removal occurred in a potentiated way in the AR performed at lactate threshold in relation to PR. However, they failed to identify the effect of self-paced recovery on [Lac] reduction. Additionally, the recovery intensity at lactate threshold seems to influence negatively the performance in subsequent trials, for it reduces the maximum force production capacity.[16] The reduction of the force production capacity is related to the decrease in PCr and to the consequent increase in Pi, also promoting a reduction of the pH and a reduction in the performance at

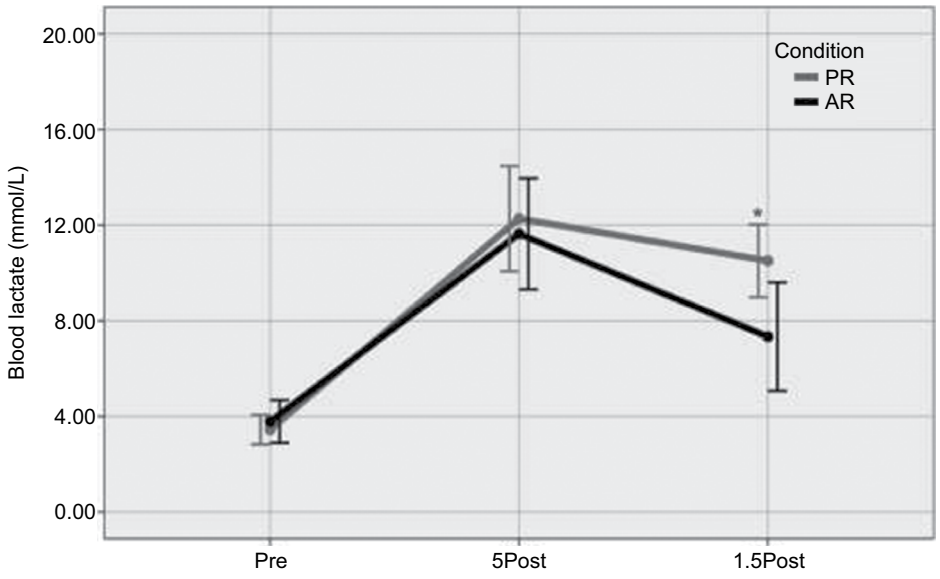

Figure 1 Blood lactate responses during passive recovery (PR) and active recovery (AR).
Note: *$p=0.001$, PR versus AR.

the end of the trials.[6,25] Indeed, determination of the ideal intensity for AR must consider the interval and the duration of the trials.[25] Thus, the intensity must be controlled, and the duration must be enough to promote PCr resynthesis, essential for the 100 m and 200 m trials.[27]

The results of the present study showed that self-paced AR, performed at an intensity of 69.10% of the maximum velocity at the 200 m freestyle trial, was able to remove blood lactate faster than PR. In this sense, Menzies et al[24] affirm that the recovery intensity does not need to be fixed in treadmill exercises, because athletes tend to perform self-paced AR at an intensity close to 80% of the lactate threshold. In some way still unknown, the biological feedback system stimulates the athletes to perform recovery in the most effective intensity for lactate removal and rebalance of the pH.

The higher blood lactate removal velocity in AR is related to the reduction of [Lac] itself, to the removal of H^+, and to the restoration of pH, which propitiates the recovery of the maximal capacity of glycolysis, fundamental for providing energy for the 100 m and 200 m trials.[27,30] The decrease of lactate is related to oxidation in skeletal muscle tissue due to increased blood flow, through the metabolic oxidation pathway at the tricarboxylic acid cycle, transforming it into CO_2 and H_2O products, favoring the removal of H^+ and the reinstatement of the adequate acid–base balance.[13,22] Additionally, AR stimulates the conversion of ~13%–27% of the lactate in glycogen, by the Cori cycle, through gluconeogenesis.[13]

The reduction of [Lac] in self-paced AR in this study was evident and markedly significant. Nevertheless, the values found after recovery are still superior to the resting levels. When considering the concentration related to lactate threshold 4 mmol L^{-1},[2,3] it is understood that [Lac] in 7.34±2.27 still represents a high acidosis level. In this way, Vescovi et al[28] propose that the ideal AR after the 200 m freestyle trial has a distance of 1,300–1,500 m for men and 800–1,000 m for women, in order to return [Lac] to values inferior to 3 mmol L^{-1}.

The present study has several limitations. The investigation of athletes of both sexes in the same analysis neglects the existence of individual factors that alter the behavior of metabolic variables, such as hormone release. However, this limitation is also present in the daily life of coaches and athletic trainers, making the methods raise the external validity of the research. Furthermore, the effect of self-paced AR on performance in a subsequent trial still needs to be investigated, aside from the relationship between the ideal intensity and the different trials present in a competition. It is also necessary to understand the biological feedback systems that provide execution at the best removal intensity. Notwithstanding, this study is justified for being the first to investigate the effects of self-paced intensity on lactate removal after a 200 m freestyle trial.

Conclusion

In summary, self-paced AR presented a higher lactate removal velocity than PR. Possibly, this higher removal velocity occurs due to the increase in blood flow, promoting the buffering of H^+ and of the pH restoration. These factors might largely contribute to the performance in a subsequent trial. Nevertheless, despite having been relevant to the decrease of [Lac], 10 minutes of self-paced AR was not enough to reestablish the resting [Lac].

Disclosure

The authors report no conflicts of interest in this work.

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