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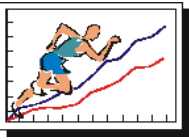
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The Oxygen Uptake Slow Component at Submaximal Intensities in Breaststroke Swimming

by

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The present work proposed to study the oxygen uptake slow component (VO₂ SC) of breaststroke swimmers at four different intensities of submaximal exercise, via mathematical modeling of a multi-exponential function. The slow component (SC) was also assessed with two different fixed interval methods and the three methods were compared. Twelve male swimmers performed a test comprising four submaximal 300 m bouts at different intensities where all expired gases were collected breath by breath. Multi-exponential modeling showed values above 450 ml·min⁻¹ of the SC in the two last bouts of exercise (those with intensities above the lactate threshold). A significant effect of the method that was used to calculate the VO₂ SC was revealed. Higher mean values were observed when using mathematical modeling compared with the fixed interval 3rd min method ($F=7.111$; $p=0.012$; $\eta^2=0.587$); furthermore, differences were detected among the two fixed interval methods. No significant relationship was found between the SC determined by any method and the blood lactate measured at each of the four exercise intensities. In addition, no significant association between the SC and peak oxygen uptake was found. It was concluded that in trained breaststroke swimmers, the presence of the VO₂ SC may be observed at intensities above that corresponding to the 3.5 mM⁻¹ threshold. Moreover, mathematical modeling of the oxygen uptake on-kinetics tended to show a higher slow component as compared to fixed interval methods.

Key words: VO₂ on-kinetics, mathematical modeling, breaststroke swimming.

Introduction

During the step transition from rest to constant-load exercise, oxygen uptake (VO₂) has shown varied responses to different workloads, being directly dependent on the type of exercise (Billat et al., 1998) and the level of endurance (Carter et al., 2000; Casaburi et al., 1987). In 1982, Whipp et al. (1982) theorized a model of VO₂ on-kinetics describing three phases. The first one, the cardio-dynamic component, corresponds to a fast increase in alveolar O₂, enabling a 15-20 s

transient plateau after on-transition (Demarle et al., 2005). The second phase, called the primary component, is characterized by an exponential increase of VO₂ due to muscle oxygen demand, lasting about 2-3 min in healthy subjects (Barstow and Mole, 1991). The third phase is described by an additional slow rise in VO₂, superimposing the primary component initiated at exercise onset. This slow phase, commonly called the slow component (SC), continues until either a delayed

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steady state is attained or maximal VO₂ is reached (Whipp, 1994). The magnitude of the SC is considered to have a physiological meaning only when it is higher than 200 ml·min⁻¹ (Billat et al., 2000).

The type of muscle contraction (Pringle et al., 2002), the pattern of motor unit recruitment (Carter et al., 2000) and total muscle mass involved in exercise (Ryschon et al., 1997) have direct influence on the SC. Indeed, the SC has been reported to be sensitive to different types of exercise, being larger for running when compared to cycling exercise (Billat et al., 1998). The SC, which was first observed by Astrand and Saltin (1961), has been assessed through a number of methods including: the difference between VO₂ at the 6th and the 3rd min of exercise (Koppo and Bouckaert, 2001), the difference between VO₂ at the last and the 3rd min of exercise (Patterson and Whipp, 1991), the difference between VO₂ at the last and the 2nd min of exercise (Koppo and Bouckaert, 2002), a single exponential function (Wasserman and Whipp, 1983), a linear function superimposing the exponential component (Casaburi et al., 1987) and the amplitude of a multi exponential function (Santos and Gianella-Neto, 2006). Green and Dawson (1996) observed that the SC was more pronounced between the 3rd and 6th min of exercise, which may explain the popularity of this method among researchers. Notwithstanding, Bearden and Moffat (2001) suggested that this method tended to underestimate the SC when compared with mathematical modeling.

Studies about the on-kinetics of VO₂ during swimming are scarce. The SC has already been reported in front crawl swimming (Demarie et al., 2001; Fernandes et al., 2003, 2008; Machado et al., 2006; Reis et al., 2010), but only one study has attempted to evaluate the SC during breaststroke swimming (Reis et al., 2010). As they are, alternated and simultaneously swimming techniques, respectively, their kinematics (Thompson et al., 2004) and energetics (Barbosa et al., 2008) differ. Reis et al. (2010) concluded, by observing the difference between VO₂ at the end and at the 3rd min of exercise, that a SC seemed to be present only at swimming intensities above that corresponding to the lactic threshold, as occurs in land-based activities (Billat et al., 1998; Machado et al., 2006). However, to date, no study

has modeled with a multi exponential function the kinetics of VO₂ in breaststroke swimming.

Therefore, the aim of the present study was to analyze the VO₂ slow component of trained breaststroke swimmers during submaximal incremental exercise, using a multi exponential function. It was hypothesized that the SC would only appear at swimming intensities higher than the 3.5 mM⁻¹ lactate threshold. In addition, a comparison of the multi exponential function with the method of fixed intervals was carried out. Potential relationships between the SC and blood lactate concentration (LA⁻) as well as peak VO₂ were also investigated.

Material and Methods

Participants

Twelve male high-level swimmers volunteered to participate in the study. Mean (±SD) values for age, body mass and height, the arm span as well as the estimated body fat percentage were 16.9 ±2.8 yrs, 69.7 ±9.0 kg, 178 ±5.0 cm, 186 ±6.0 cm and 11.6 ±2.3%, respectively. All subjects were involved in systematic training (from 9 to 12 training sessions a week) and participated regularly in swimming breaststroke events. The swimmers' mean personal best performance at the time of the measurements in the 100 m and 200 m breaststroke short course were 70.20 ±5.02 and 153.67 ±10.40 s, respectively. All participants signed a written informed consent form and those under 18 years of age had their parent's written consent to participate in the study. All ethical standards for research with human subjects were in accordance with the Declaration of Helsinki and were approved by the Review Board of the University of Trás-os-Montes & Alto Douro.

Measures

Measurement of pulmonary gas exchange

Pulmonary gas exchange variables, particularly VO₂, were calculated from the gas concentration and flow using a telemetric metabolic cart (K4b2, Cosmed, Italy). Subjects breathed through a low-resistance attached snorkel valve (AquaTrainer, Cosmed, Italy). Before each test, a reference air calibration of the device was performed using a gas sample with 16% O₂ and 5% CO₂ concentrations, according to the manufactured instructions, as described by Reis et al. (2010). The data was telemetrically

displayed and analyzed breath-by-breath.

VO₂ on-kinetics model

Prior to VO₂ kinetic modeling, a linear interpolation was applied to all breath-by-breath collected data to fit the time response to 1 s intervals. Thereon, an average filter with 11 samples (data ± 5 values) was used to smooth the data. According to the short-term fluctuations observed in the data, the number of samples used before and after, the central value was chosen to eliminate the high-frequency oscillations and simultaneously to preserve the information from the original data.

The kinetics of VO₂ was modeled by the following exponential function:

$$\dot{V}O_2(t) = A_0 + A_1 \times \left(1 - e^{-\left(\frac{t}{\tau_{u1}}\right)} \right) + A_2 \times \left(1 - e^{-\left(\frac{t-TD}{\tau_{u2}}\right)} \right)$$

(Equation 1)

where $\dot{V}O_2(t)$ represents oxygen uptake per unit of time (ml·min⁻¹); A₀ is the baseline value for VO₂ (ml·min⁻¹), A₁ and A₂ (ml·min⁻¹) are the amplitudes of the primary and slow component phases, respectively, TD is the time delay from the first exponential phase until the beginning of the second exponential (s), and tau₁ and tau₂ are time constants (s).

A non-linear regression analysis was applied to fit the time responses of VO₂, in which coefficients were obtained by the least squares method, being the oxygen uptake slow component determined by mathematical modeling using the magnitude of the second exponential amplitude (MM_{SC}).

The calculation of the VO₂ SC through the fixed interval methods consisted of subtracting average VO₂ observed in the last min of each bout by:

- average VO₂ observed in the 3rd min of exercise (Δ_{SC3min});
- average VO₂ observed in the 2nd min of exercise (Δ_{SC2min}).

The second exponential rise was considered to represent a SC when it surpassed 200 ml·min⁻¹ (Billat et al., 2000).

All mathematical procedures and modeling

were done using MATLAB R2010b (Mathworks, USA) for Windows®.

Measurement of blood lactate

Immediately after the completion of each bout, capillary blood samples were collected from the fingertip to determine LA⁻ values (Accusport, Boehringer, Germany). Before each test a calibration of the device was performed with several YSI 1530 Standard Lactate Solutions (2, 4, 8 and 16 mM⁻¹). The lactate threshold was accepted to be the value corresponding to 3.5 mM⁻¹ of LA⁻ (Fernandes et al., 2003).

Procedures

Experimental testing was conducted in an indoor 25 m swimming pool, with water temperature of 28.5° C and air humidity of 55%. During the five days prior to the experiment subjects were not involved in high-intensity training and limited their training program to a single daily low-intensity swimming session. The experimental procedures were conducted in the morning between 10 and 12 am.

All subjects performed a graded individualized swimming test, with four 300 m exercise bouts, each one performed at a controlled constant speed (ensuring that all subjects performed, at least, 5 min of exercise in each bout); a recovery period of 15 min was introduced between successive bouts. Initial speed corresponded to 50% of the swimmer's estimated best performance at the 300 m breaststroke event and subsequent increments were equal to 10% of the 300 m best performance speed, controlled by an electronic pacer (TAR. 1.1, GBK-electronics, Aveiro, Portugal), with successive flashing lights placed at the bottom of the pool. In-water starts and open turns were used, and only a dry land warm-up was performed before the test. The performance in the 300 m breaststroke was estimated taking into account the performance at the 100 m and 200 m breaststroke (Reis et al., 2010):

$$v_{300} = v_{200} - (v_{200} - v_{400}) / 2$$

(Equation 2)

$$v_{400} = (7 * v_{200} / 4) - (3 * v_{100} / 4)$$

(Equation 3)

where v₁₀₀ and v₂₀₀ represent the velocity of the 100 m and 200 m breaststroke event

and v300 and v400 represent the estimation of the velocity of the 300 m and 400 m breaststroke event.

Statistical analysis

Normal distribution of data was confirmed by the Shapiro-Wilk test and sphericity by the Mauchly's test. To compare the SC according to the different intensities and calculation methods, a two factor repeated measure analysis of variance (ANOVA) with a post-hoc Bonferroni test was performed. With the aim of identifying potential relationships between the SC, LA and peak oxygen uptake (VO_{2peak}), Pearson or Spearman correlations were applied, whenever appropriate. The level of significance was set at 5% ($p \leq 0.05$). Results are presented as means and standard deviations. All data analysis was performed using Statistical Package for Social Sciences (SPSS 17.0 - Science, Chicago, USA) for Windows®.

Results

In Figure 1, the first bout of a representative subject is shown where, as expected, no slow component was observed in VO_2 on-kinetics.

In Figure 2, VO_2 kinetics of another swimmer is shown during the fourth bout of the experimental protocol, being evident the appearance of the slow component.

Blood lactate in the four bouts of exercise was 2.33 ± 0.53 , 2.28 ± 0.52 , 4.03 ± 2.45 and 5.72 ± 1.73

mM^{-1} . Mean peak VO_2 value obtained for the total sample during the incremental protocol was 4.4 ± 0.90 $l \cdot min^{-1}$. Table 1 presents the variables extracted from mathematical modeling of the VO_2 on-kinetics and Table 2 compares the magnitude of the slow component reached by the three methods. Table 3 presents the descriptive data of absolute VO_2 , relative VO_2 and the number of subjects that surpassed the 200 $ml \cdot min^{-1}$ as to the slow component in the four bouts of exercise.

The repeated measures analysis of variance revealed a significant effect of the method that was used to calculate the VO_2 SC. Higher mean values were observed when using mathematical modeling compared with the Δ_{SC3min} method ($F=7.111$; $p=0.012$; $\eta^2=0.587$); furthermore, differences were detected among the two interval methods. The effect of exercise intensity was also evident ($F=6.680$; $p=0.011$; $\eta^2=0.690$), and significant differences were observed between the second and the third bout, and between the second and the fourth bout of the exercise. Both the method and intensity effects presented a strong statistical power (0.830 and 0.865, respectively). However, there was no significant effect of the interaction method vs. intensity on the VO_2 SC ($F=2.014$; $p=0.012$; $\eta^2=0.668$).

No significant relationship was found between the SC determined by whichever method and blood lactate measured at each of the four exercise intensities. In addition, no significant relationship between the SC and peak VO_2 was found.

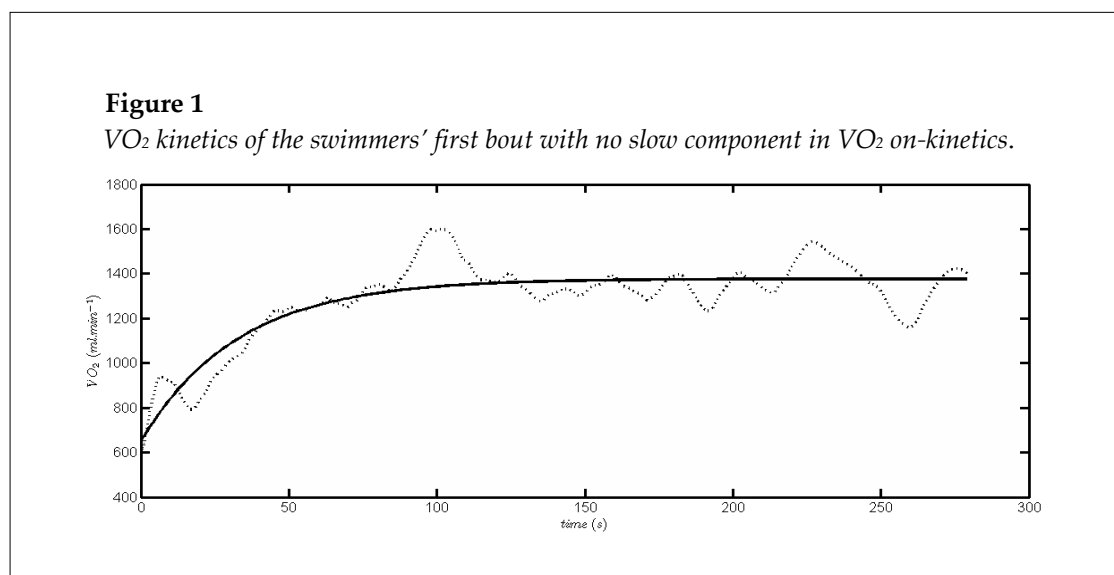


Figure 2

VO₂ kinetics of the swimmers' fourth bout with the appearance of the VO₂ SC

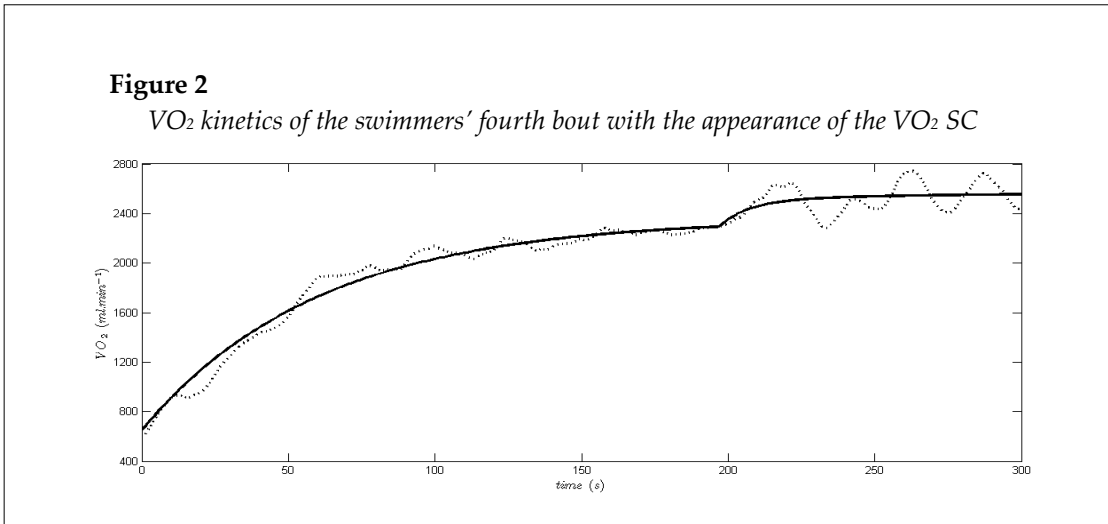


Table 1

Mean values (±SD) of the parameters extracted from the bi-exponential modeling of oxygen uptake.

	1 st stage	2 nd stage	3 rd stage	4 th stage
A ₀ , ml·min ⁻¹	278.71 ±67.21	270.56 ±59.39	270.79 ±57.74	269.48 ±45.74
A ₁ , ml·min ⁻¹	708.69 ±285.96	1035.71 ± 386.18	1571.68 ±739.26	1988.16 ±565.06
A ₂ , ml·min ⁻¹	113.08 ±162.51	227.00 ±269.93	497.76 ±276.07	473.36 ±404.22
tau ₁ ,s	48.07 ±31.69	60.83 ±27.23	71.09 ±22.36	72.77 ±19.86
tau ₂ ,s	13.03 ±16.14	14.30 ±13.45	17.83 ±10.04	22.39 ±13.73
TD,s	94.82 ±109.14	131.45 ±90.61	147.40 ±34.42	148.78±56.17

A₀ = oxygen uptake value of the cardio-dynamic phase, A₁ and A₂ = amplitude of each exponential component; tau₁ and tau₂ = time constants of the equation; TD = time delay from the slow phase to the primary phase.

Table 2

Mean values (±SD) of the slow component calculated by the three methods in each of the four bouts (with indication of mean blood lactate values at each stage)

	1 st stage	2 nd stage	3 rd stage	4 th stage
	2.33 ±0.53	2.28 ±0.52	4.03 ±2.45	5.72 ±1.73
MM _{sc} , ml·min ⁻¹	113.08 ±162.51	227.00 ±269.93	497.76 ±276.07	473.36 ±404.22
ΔSC _{3min} , ml·min ⁻¹	107.9 ±54.9	88.3 ±73.6	311.0 ±153.4	325.1 ±160.5
ΔSC _{2min} , ml·min ⁻¹	121.6 ±66.2	109.1 ±90.8	373.2 ±185.7	384.8 ±197.9

*MM_{sc} = slow component values obtained by mathematical modeling;
 ΔSC_{3min} = slow component values obtained by the fixed interval method last minute mean minus third minute mean;
 ΔSC_{2min} = slow component values obtained by the fixed interval method last minute mean minus second minute mean.*

Table 3

Mean values (\pm SD) of absolute VO_2 , relative VO_2 and the number of subjects with an evident slow component in each of the four bouts (with indication of mean blood lactate values at each stage)

	1 st stage 2.33 \pm 0.53	2 nd stage 2.28 \pm 0.52	3 rd stage 4.03 \pm 2.45	4 th stage 5.72 \pm 1.73
VO_2 , ml·min ⁻¹	1958.05 \pm 191.74	1970.38 \pm 251.79	2547.74 \pm 383.90	4167.85 \pm 428.67
VO_2 , ml·min ⁻¹ ·kg ⁻¹	27.91 \pm 2.73	28.09 \pm 3.89	36.32 \pm 5.47	59.41 \pm 6.11
$n > 200$ ml·min ⁻¹	3	4	9	8

MM_{sc} = slow component values obtained by mathematical modeling;

Δ_{sc3min} = slow component values obtained by the fixed interval method
last minute mean minus third minute mean;

Δ_{sc2min} = slow component values obtained by the fixed interval method
last minute mean minus second minute mean.

Discussion

The aim of the present study was to analyze the SC of trained breaststroke swimmers during submaximal exercise using a multi exponential function. Testing procedures were conducted in ecological swimming conditions, using a modern telemetric procedure for collecting and measuring the breath-by-breath expired gases, and a suitable modified snorkel and valve system (Keskinen et al., 2003) specific for breath-by-breath data analysis. It was hypothesized that the SC would only appear during swimming at intensities above 3.5 mM⁻¹ of the LA- threshold, i.e., at the heavy intensity domain.

The main finding of the present study was that the SC determined by mathematical modeling was evident in most subjects at intensities higher than that corresponding to 3.5 mM⁻¹. The results of both the interval methods indicated the presence of the SC in the last two bouts of exercise, both with exercise intensity above the 3.5 mM⁻¹ lactate threshold.

The decision to invite highly-trained individuals to participate in the present study

relied on the fact that top level swimmers were expected to be characterized by maximal physical fitness, very close to human genetic limits (Fernandes et al., 2008). Another very important reason was the fact that athletes were the main target population in terms of the possible applicability of this type of research.

The protocol was designed to investigate VO_2 kinetics in different intensity domains. The SC is commonly described in studies where the intensity corresponds to the 3.5 mM⁻¹ lactate threshold (Fernandes et al., 2003, 2008; Reis et al., 2010, 2012). In the protocol of Demarie et al. (2001) constant-velocity front crawl swimming in a flume above critical velocity was used. Fernandes et al. (2003) used a test protocol where subjects swam at a pace corresponding to their $\text{VO}_{2\max}$ velocity until volitional exhaustion. This was repeated in 2006 (Machado et al., 2006) and 2008 (Fernandes et al., 2008) in tests where swimmers swam until exhaustion at the previously determined $\text{VO}_{2\max}$ velocity. However, to the best of our knowledge, the SC has not been investigated yet using mathematical modeling of the moderate-domain in breaststroke swimming.

In the present study, the SC was assessed

through mathematical modeling (Santos and Gianella-Neto, 2006), a more precise and accurate method (Barstow and Mole, 1991) than rather simplistic calculation of the increase in VO_2 between the second or third minute and the last minute of exercise (Bearden and Moffatt, 2001; Machado et al., 2006). Moreover, the mathematical model used in the present study allows the discrimination of different components of VO_2 kinetics. The obtained results are markedly different from the findings of all other studies published about the SC in swimming. As an example we may indicate the single work found in literature with the same methodological characteristics (Fernandes et al., 2008), however, performed with front crawl swimmers, where the SC values were positive in all subjects and the mean of the sample was equal to 356.27 ± 168.16 ml·min⁻¹.

To our knowledge, the study by Reis et al. (2010) is the only report in the literature with data for the VO_2SC in breaststroke swimming. However, their SC was calculated by a fixed interval method (difference between mean VO_2 at the last and third minute of exercise). A SC above 350 ml·min⁻¹ was verified at all four exercise intensities of testing, which comprehended different domains of intensity. In fact, according to the literature, we expected to observe the SC in the last two stages of effort, stages related with the heavy and maximal domain of intensity (Denadai et al., 2000). On the other hand, the moderate domain, where the first two bouts of the present study are included, is typically reported

in the literature as of an exercise intensity which does not enable the presence of the SC (Gaesser and Poole, 1996). However, our results do not confirm such an assumption for breaststroke swimming, as the 2nd bout of exercise showed a mean value higher than 200 ml·min⁻¹, a limit often referred as being the lower threshold for an evident SC (Billat et al., 2000).

The absence of a relationships between post-exercise LA- and the SC, irrespective of the method used for its assessment, is in agreement with some authors (Mole and Hoffmann, 1999; Ogita and Tabata, 1992) who stated it was unlikely that blood lactate per se could be responsible for the SC phenomenon, but rather the concurrent acidosis. Hence, our SC observations could alternatively be explained by larger rates of type II fiber recruitment and additional energy cost of contraction (Whipp, 1994) at higher exercise intensities. However, Casaburi et al. (1987) and Billat et al. (1998) found a significant correlation between the SC and LA- obtained after high intensity exercise.

Conclusion

In summary, our results indicated that mathematical modeling of VO_2 on-kinetics during submaximal breaststroke swimming revealed, in most cases, a higher SC as compared to fixed interval methods. The SC values observed with the aid of mathematical modeling confirmed the hypothesis of a presence of the SC at intensities above the 3.5 mM⁻¹ lactate threshold.

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