

Research article

## Neuromuscular Fatigue during 200 M Breaststroke

Ana Conceição<sup>1,3</sup>✉, António J. Silva<sup>2,3</sup>, Tiago Barbosa<sup>4,3</sup>, István Karsai<sup>5</sup> and Hugo Louro<sup>1,3</sup>

<sup>1</sup> Sport Sciences School of Rio Maior, Rio Maior, Portugal; <sup>2</sup> University of Trás-os-Montes and Alto Douro, Vila Real, Portugal; <sup>3</sup> Research Center in Sports Science, Health and Human Development, Vila Real, Portugal; <sup>4</sup> Nanyang Technological University, Singapore; <sup>5</sup> University of Pécs, Pécs, Hungary

### Abstract

The aims of this study were: i) to analyze activation patterns of four upper limb muscles (duration of the active and non-active phase) in each lap of 200m breaststroke, ii) quantify neuromuscular fatigue, with kinematics and physiologic assessment. Surface electromyogram was collected for the biceps brachii, deltoid anterior, pectoralis major and triceps brachii of nine male swimmers performing a maximal 200m breaststroke trial. Swimming speed, SL, SR, SI decreased from the 1<sup>st</sup> to the 3<sup>rd</sup> lap. SR increased on the 4<sup>th</sup> lap ( $35.91 \pm 2.99$  stroke·min<sup>-1</sup>). Peak blood lactate was  $13.02 \pm 1.72$  mmol·l<sup>-1</sup> three minutes after the maximal trial. The EMG average rectified value (ARV) increased at the end of the race for all selected muscles, but the deltoid anterior and pectoralis major in the 1<sup>st</sup> lap and for biceps brachii, deltoid anterior and triceps brachii in the 4<sup>th</sup> lap. The mean frequency of the power spectral density (MNF) decreased at the 4<sup>th</sup> lap for all muscles. These findings suggest the occurrence of fatigue at the beginning of the 2<sup>nd</sup> lap in the 200m breaststroke trial, characterized by changes in kinematic parameters and selective changes in upper limb muscle action. There was a trend towards a non-linear fatigue state.

**Key words:** Swimming; Kinematics; EMG; Mean frequency.

### Introduction

The breaststroke technique is considered one of the least economic of the four swimming styles (Barbosa et al., 2006), which can lead to early fatigue while swimming. Neuromuscular fatigue can be defined as: (i) the failure to maintain the required or expected force, accompanied by changes in muscular activity (Dimitrova et al., 2003); and (ii) the inability of skeletal muscle to generate high levels of muscular strength or maintain these levels over time (Enoka and Stuart, 1992). Additionally, the manifestations of fatigue have been associated with (Allen et al., 1995; Pagala et al., 1994): (i) a decline in muscle tension produced during and after submaximal and maximal exercise; (ii) an inability to maintain a given exercise intensity over time, reducing the speed of contraction and increased muscle relaxation time, as well as; (iii) the variation of intra and extracellular concentrations of certain metabolites and ions.

Neuromuscular fatigue can be divided into central and peripheral fatigue. Central fatigue comprises of decreases in the voluntary activation of the muscle, which is due to decreases in the number of recruited motor units and their discharge rate (González et al., 2012). Peripheral fatigue involves changes in neuromuscular transmission, muscle action potential propagation and decreases in the

contractile tension of the muscle fibers (Boyas and Guevel, 2011). Peripheral fatigue during exercise is considered an impairment of the peripheral mechanisms from excitation to muscle contraction. Peripheral regulation is, therefore, related to a perturbation of calcium ion movements, an accumulation of phosphate, and/or a decrease of adenosine triphosphate stores (Boyas and Guevel, 2011).

Some studies attempted to relate the onset of fatigue with the execution of various sport techniques (Camata et al., 2011; Giangarra et al., 1993; Pink et al., 1993; Robineau et al., 2012). While swimming, neuromuscular mechanisms related to fatigue remain unclear, and the few studies which carried out research, focused mainly on the front-crawl technique (Coty et al., 2006; Figueiredo et al., 2011; Ikuta et al., 2012; Stirn et al., 2011).

Coty et al. (2006) observed a decrease in the instantaneous mean frequency in the extensor carpi ulnaris muscles (11.4% and 8.5%, respectively) after a 4x50m high intensity front crawl. Stirn et al. (2011) reported that at the end of a 100m front crawl at a maximal effort, the mean power frequency decreased by 20-25%. Ikuta et al. (2012) suggested that the decrease in swimming velocity was related to a decrease in the activity of several muscles coordinated with each other, and that a compensating strategy was involved between the pectoralis major and other muscles during the last lap of a 4x50m front crawl test.

Important muscles activated in breaststroke swimming seem to be the biceps brachii, triceps brachii, subscapularis, latissimus dorsi, pectoralis major, supraspinatus, infraspinatus, serratus anterior, and deltoid anterior, teres minor and trapezio ) for the upper limbs (Conceição et al., 2010; Nuber et al., 1986; Ruwe et al., 1994; Yoshizawa et al., 1976). While gluteus maximus, vastus medialis, rectus femoris, biceps femoris, abductor magnus, quadriceps, gastrocnemius, tibialis anterior, abductor hallucis, abductor digiti minimi, flexor digitorum brevis are for the lower limbs (McLeod, 2010; Yoshizawa et al., 1976).

It is clear that EMG can be useful in tracking muscle fatigue for many reasons (the relationships found between sEMG features and muscle fatigue, and the possibility of recording them in almost any type of situation). Therefore, some studies have examined the relationship between muscle fatigue and EMG variables and, consequently, the possibility of using EMG models to accurately track muscle fatigue (González-Izal et al., 2012).

The linear techniques that are used to estimate muscle fatigue are based on linear regression, which relates changes in EMG parameters to changes in power

loss (as a direct measurement of muscle fatigue). The non-linear techniques that are used to estimate muscle fatigue are based on neural networks to relate EMG parameters to muscle fatigue. Both linear and non-linear techniques provided good results for mapping changes in power or force loss during dynamic exercises based on sets of EMG parameters (González-Izal et al., 2012).

Apart from this, however, we have no idea of the appearance of fatigue of the upper limbs in breaststroke. Since there is a lack of studies to quantify the neuromuscular fatigue in the upper limbs, using spectral parameters, we believe that this research would have the potential to make a contribution to the limited body of knowledge in this field. The main aim for swimming coaches and researchers relies on the identification of the factors that might predict performance with higher validity and accuracy.

The aims of this study were: i) to analyze the activation patterns (duration of active and non-active phase) of the upper limbs' four muscles during each lap for 200m breaststroke, ii) quantify neuromuscular fatigue, through kinematics and physiologic assessment. It was hypothesized that an increase in signal amplitude and a decrease in spectral parameters due to repetitive sub-maximal contractions, characterized a non-linear fatigue process. Also considered was that the fatigue process occurs differently for each of the muscles studied.

## Methods

### Subjects

Nine male swimmers (age:  $22.3 \pm 2.9$  years; height:  $1.81 \pm 0.05$  m; body mass:  $73.60 \pm 3.82$  kg; mean  $\pm$ SD) volunteered to participate in this study and provided written consent. They were all swimmers competing at the national level with an average personal best result for 200-m breaststroke ( $149.44 \pm 6.59$  s, corresponding respectively to  $643.75 \pm 53.77$  FINA ranking points). All the procedures were approved by the institutional Ethics Committee and carried out according to the Helsinki Declaration.

### Testing procedure

The experiments were performed in a 50m indoor swimming pool at a water temperature of  $27.5^\circ\text{C}$  and 75% humidity. Subjects performed a standard warm-up of 800m front crawl, and a specific warm-up of 200m breaststroke at a medium level of effort. After a twenty minute rest subjects performed a maximal 200m breaststroke trial with a push off start.

### Data acquisition

#### EMG data collection

Surface EMG signals from the biceps brachii (BB), deltoid anterior (DA), pectoralis major (PM) and triceps brachii (TB) on the right side of the body were collected. These muscles were selected according to their importance in breaststroke (Conceição et al., 2010; Nuber et al., 1986; Ruwe et al., 1994; Yoshizawa et al., 1976).

Bipolar surface electrodes were used (10-mm diameter discs, Plux, Lisbon, Portugal) with an inter-electrode distance of 20mm. Electrodes on the upper part of the PM were placed in the middle of the line that con-

nects the acromion process and the manubrium (sternum) two fingers below the clavicle (Stirn et al., 2011). The electrodes on the long head of the BB, DA and TB were placed in accordance with SENIAM recommendations (Herrmens and Freriks, 1999).

The skin was shaved, rubbed with sandpaper and cleaned with alcohol so that the inter-electrode resistance did not exceed 5 KOhm (Basmajian and De Luca, 1985). The ground electrode was positioned over the cervical vertebrae. Transparent dressings with labels (Hydrofilm®, 10 cm x 12,5 cm, USA) were used to cover the electrodes to isolate them from the water (Hohmann et al., 2006). All cables were fixed to the skin by adhesive tape in several places to minimize their movement and any signal interference. Swimmers wore a full body swimming suit to further immobilize the cables (Fastskin Speedo®, Speedo Aqualab, USA).

The wireless EMG device (BioPLUX.research, Lisbon, Portugal) with 8 analog channels (12-bit), sampling rate at 1 kHz, was put in a waterproof bag (84x53x18mm) and placed inside the swimmer's cap. Data was transmitted to the PC in real time via Bluetooth.

The EMG and video data were recorded simultaneously and were not synchronized with video recording, because the aim of this study was to understand the appearance of fatigue in relation to race parameters rather than detailed within cycle swimming kinematics.

All EMG analysis was conducted with a MATLAB routine (Mathworks, Inc., Natick MA, USA). The process of determining the muscle activity boundaries consists of finding the neighborhood points, where the energy was 30% of muscle activation maximum peak within a stroke (Stirn et al., 2011). These were calculated by segmenting the muscle input signal energy according to the same criteria described in Stirn et al. (2011). Starting from the raw signal, DC components were removed and thereafter filtered with a fifth-order Butterworth band pass filter where the lower and upper cut-off frequencies were set to 10 and 500Hz respectively. The signal energy was then determined with a 250 ms sliding window (Stirn et al., 2011) and according to:

$$E(t_0) = \int_{t_0+125ms}^{t_0+125ms} x^2(t) dt \quad (1)$$

Even though the high frequencies of the input signal were filtered with a Butterworth filter, muscle energy is very noisy and presents several local maximum peaks that didn't correspond to the muscle active window center, as shown in Figure 2. To overcome this difficulty, a strategy to determine the muscle's "true" maximum energy peaks was devised. Each stroke taken by a swimmer produces patterns in the signal, these patterns are mainly translated by a periodicity in EMG energy, see Figure 1.

By defining the signal mean period, one can use this information to determine the maximum peak candidates with the highest and with minimal differences between two maximum candidates and the expected period.

Once the maximum candidates have been determined, the muscle activity boundaries were then selected by determining the neighborhood points where the energy

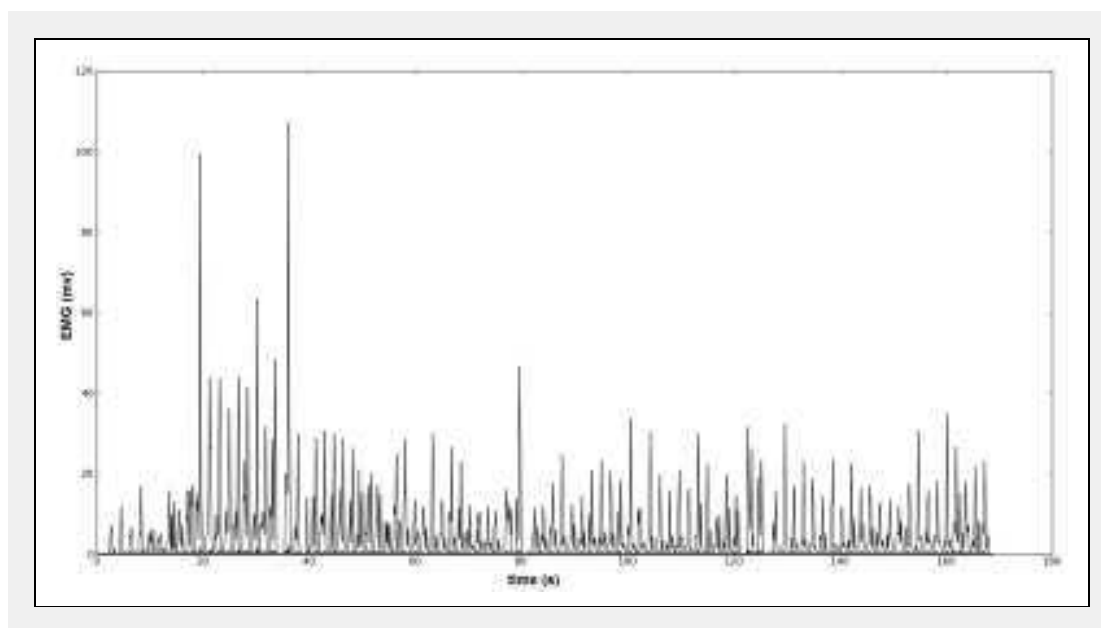


Figure 1. Energy envelope of a EMG signal segment.

is 30% of the determined maximum peaks. For each muscle activations, the active phase was defined as the part of the EMG signal for which the energy was at least 30% of the local maximum energy value, for each particular muscle activation. The raw EMG segments belonging to the active phases were extracted and used in the calculation of the active phase duration and the amplitude frequency analysis. The non-active phase was defined as the time interval between the two consecutive active phases as shown in Figure 2.

The temporal evolution of the active and non-active phases average durations during stroke were calculated for each muscle for the entire 200m. Linear regression curves were fitted to the data and the durations of the fitted curves at the beginning and completion of the swim were compared. The comparisons of the durations of the active and non-active phases of each muscle during the

stroke were made to detect changes that might occur due to fatigue.

The average amplitude of EMG of each active phase was estimated using the average rectified value (ARV) of the EMG. ARV was calculated in accordance with SENIAM recommendations (Herrmens and Freriks 1999) and plotted as a function of time. A linear regression curve was fitted to the data and the ARV values of the curve at the time of commencement and the end of the swimming bout were compared (Figure 3).

For the frequency analysis, each extracted segment was zero-padded to the total length of 1s (i.e., 1,000 samples). In this way, a uniform frequency resolution (the frequency sampling interval) was used for all signal segments. The power spectral density (PSD) for each segment was estimated using the periodogram method (Proakis and Manolakis, 1996). The periodogram of a

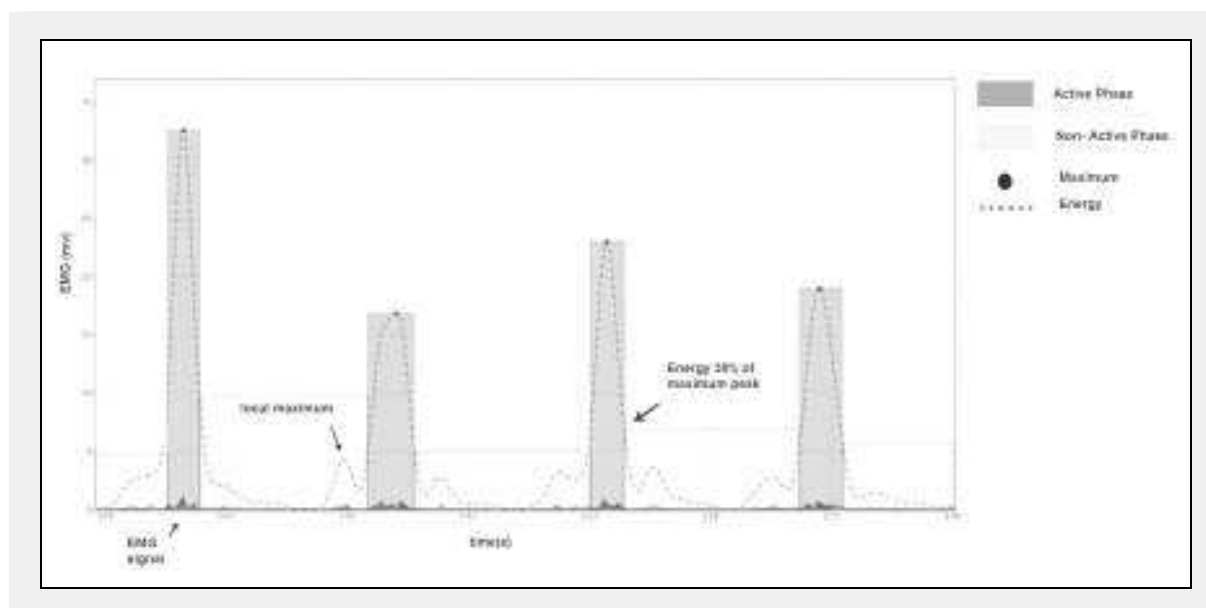


Figure 2. Segment of non-active phase and active phase of the EMG signal.

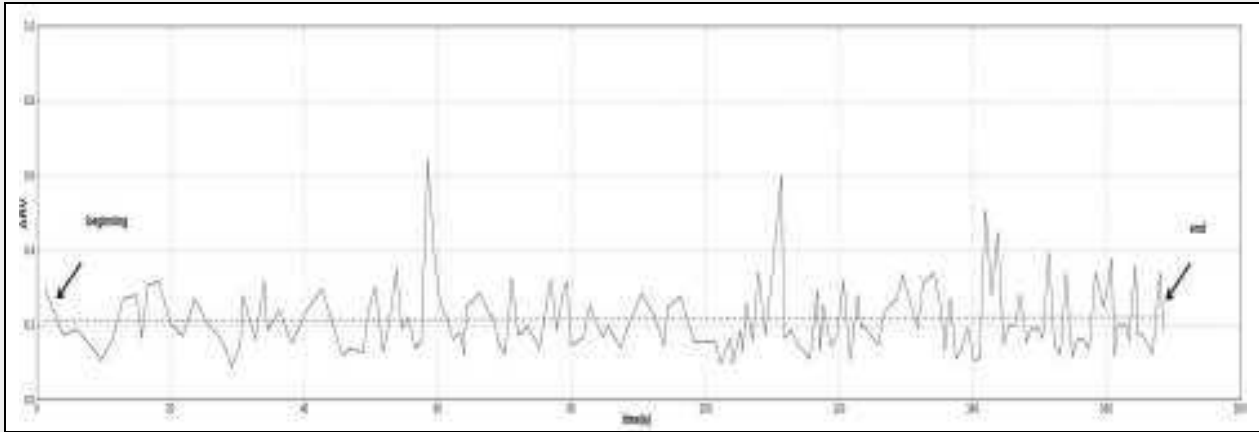


Figure 3. Example of a linear regression of the triceps brachii of one swimmer, of the ARV values in the beginning and the end of the swimming bout.

continuous signal segment  $x(t)$  of length  $T$  is defined as:

$$x(f) = \frac{1}{T} \quad (2)$$

Even though the periodogram is a non-consistent estimate of PSD (its variance is large and does not become zero with increasing length of the signal), it was demonstrated that using more sophisticated methods for PSD estimation, does not significantly improve the estimation of power spectrum central frequency measures (the mean or the median frequency) (Farina and Merletti, 2000).

Since the differences were insignificant, we decided to use the periodogram estimate in our study. As a measure of the central tendency of PSD we used the mean frequency of the PSD (MNF). MNF is defined as the first moment of the PSD and was developed by Kwatny et al., (1970). For the continuous spectrum spanning the frequencies between 0 and  $f_{MAX}$  is defined as:

$$MNF = \frac{\int_0^{f_{MAX}} f \cdot P_x(f) df}{\int_0^{f_{MAX}} P_x(f) df} \quad (3)$$

The value of MNF was calculated for each segment and used as a frequency parameter of muscle fatigue.

The values of MNF belonging to each muscle were plotted as a function of time. Since all data exhibited a trend toward linear decrease with time, a linear model was fitted to all MNF data sets to the initial MNF value (the value of MNF at the time of the first stroke) and the final MNF value (the value of MNF at the time of the last stroke) and labeled as  $MNF_{beg}$  and  $MNF_{end}$ , respectively. To normalize results between subjects, the final MNF values were expressed as a percentage of the initial values and labeled  $MNF_n$  as:

$$MNF_n = \frac{MNF_{end}}{MNF_{beg}} \times 100 \quad (4)$$

The slope of the regression line was also calculated as an estimate of the rate of change over time.

### Physiological data collection

Blood samples were collected from the earlobe at 3, 5, 7 minutes after the bout. Post exercise blood lactate concentrations were measured using a hand-held portable apparatus (Lactate Pro Analyser™, Arkay, Kyoto, Japan). According Pyne et al., (2001) this equipment exhibits a high accuracy compared with other lactate analyzers and high degree of versatility under a variety of testing conditions.

### Kinematic data collection

The swims were videotaped simultaneously on sagittal plane with a pair of cameras providing a dual projection from both underwater (Sony Mini Dv DCR-HC42E, EUA) and above (Sony Mini Dv DCR-HC42E, EUA) the water surface. The cameras were placed stationary at 25-m of the headwall, on a lateral wall, perpendicular to the swimmer's trajectory and 10-m away. The study comprised the kinematic analysis of stroke cycles (Ariel Performance Analysis System, Ariel Dynamics Inc., USA) at a sample rate of 50Hz. Zatsiorsky's model with an adaptation by de Leva (1996) was used with 8 anatomical landmarks and a trunk division in two articulated parts (Barbosa et al., 2010; Hirata and Duarte, 2007; Lafond et al., 2004; Pavol et al., 2002). To create a single image of dual projection as described previously (Barbosa et al. 2006; 2010), the independent digitalization from both cameras was reconstructed with the support of a calibration volume (16 point) and a 2D-DLT algorithm (Abdel-Aziz and Karara, 1971).

The stroke cycle was measured between the 18<sup>th</sup> m and 22<sup>th</sup> m, and identified at the end of the leg's recovery (i.e., when the knees were in full flexion and feet on eversion).

Stroke mechanics were measured by the stroke cycle period ( $P$ , s), the stroke rate ( $SR = 1/P$ , Hz), the stroke length ( $SL$ , m) and the mean swimming speed of the full stroke ( $v$ ,  $m \cdot s^{-1}$ ). Finally, the swimming efficiency was estimated using the stroke index ( $SI = v \cdot SL$ ,  $m^2 \cdot c^{-1} \cdot s^{-1}$ ) as suggested by Costill et al. (1985). This index assumes that, at a given  $v$ , the swimmer with greater  $SL$  has the most efficient swimming technique.

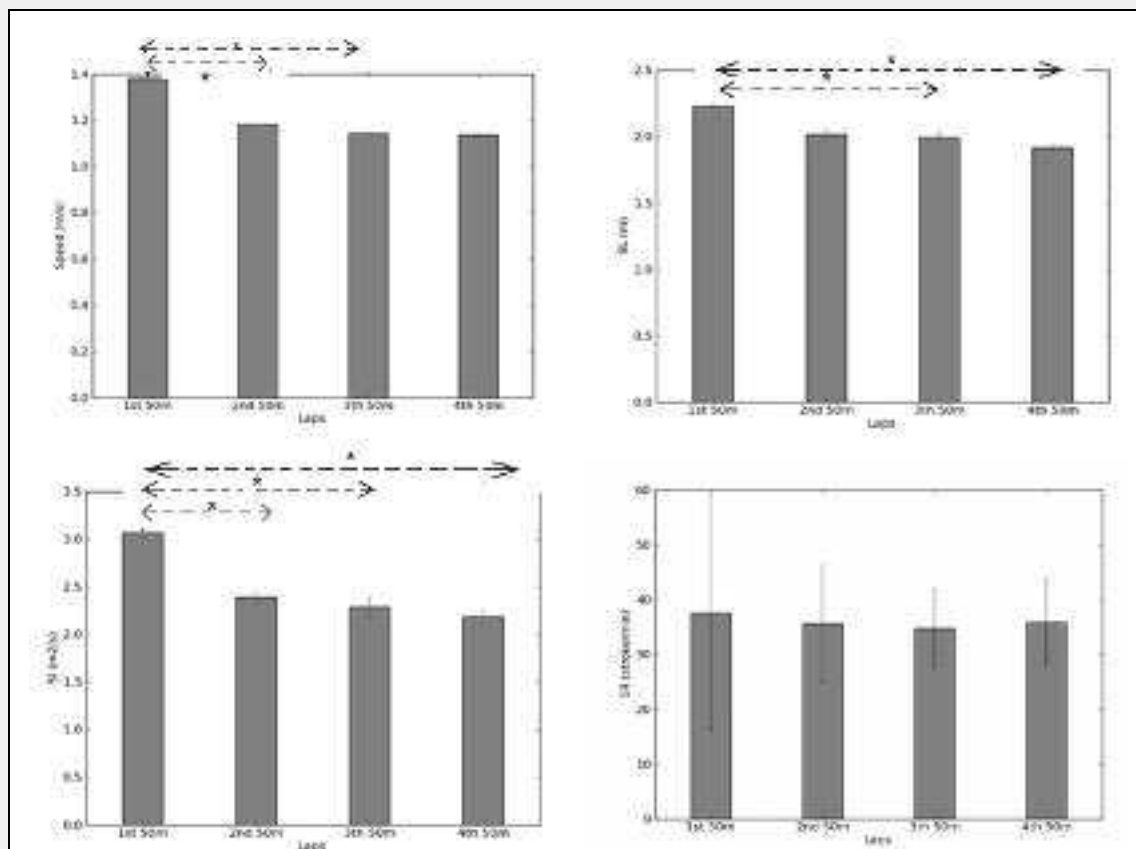


Figure 4. Mean and SD of Swimming Speed ( $\text{m}\cdot\text{s}^{-1}$ ); Stroke length (SL) m; Stroke index (SI)  $\text{m}^2\cdot\text{c}^{-1}\cdot\text{s}^{-1}$  and Stroke rate (SR)  $\text{stroke min}^{-1}$  in the four 50m lap of a 200-m breaststroke. \*  $p < 0.05$ .

### Statistical procedures

The assumptions of normality of data (Kolmogorov–Smirnov test) and homogeneity of variance (Levene's test) were both confirmed for all parameters. All results are reported as the mean values along with standard deviation values. The repeated measures ANOVA, with subsequent Tukey's test as post hoc analysis where applicable, were used for multiple comparisons between the laps ( $p < 0.05$ ).

## Results

### Kinematics data

Figure 4 presents the mean and SD of the kinematics parameters (Swimming Speed, SL, SR, SI) in the 4<sup>th</sup> lap of the 200m breaststroke. Swimming speed decreased from  $1.38 \pm 0.09 \text{ m}\cdot\text{s}^{-1}$  in the 1<sup>st</sup> lap to  $1.14 \pm 0.08 \text{ m}\cdot\text{s}^{-1}$  in the 4<sup>th</sup> lap. Significant variations were observed on swimming speed between the 1<sup>st</sup> lap and 2<sup>nd</sup> lap, and between the 1<sup>st</sup> lap and 3<sup>rd</sup> lap [ $F = 21.27$ ,  $p < 0.05$ ].

SL decreased from  $2.23 \pm 0.18 \text{ m}$  in the 1<sup>st</sup> lap to  $1.92 \pm 0.15 \text{ m}$  in 4<sup>th</sup> lap. Significant variations were observed on the SL between the 1<sup>st</sup> lap and 3<sup>rd</sup> lap, and between the 1<sup>st</sup> lap and 4<sup>th</sup> lap [ $F = 4.41$ ,  $p < 0.05$ ].

SR decreased from  $37.58 \pm 4.90 \text{ stroke min}^{-1}$  in the 1<sup>st</sup> lap to  $34.80 \pm 2.83 \text{ stroke min}^{-1}$  in 3<sup>rd</sup> lap and then increased in the 4<sup>th</sup> lap ( $35.91 \pm 2.99 \text{ stroke}\cdot\text{min}^{-1}$ ). No significant variations were observed between laps [ $F = 0.92$ ,  $p > 0.05$ ]. Lastly, the SI was higher in the 1<sup>st</sup> lap, and

decreasing from the 1<sup>st</sup> ( $3.07 \pm 0.25 \text{ m}^2\cdot\text{s}^{-1}$ ) to the last lap ( $2.19 \pm 0.29 \text{ m}^2\cdot\text{s}^{-1}$ ). Significant variations were observed on the SI between the 1<sup>st</sup> lap and 2<sup>nd</sup> lap, between the 1<sup>st</sup> lap and 3<sup>rd</sup> lap and between the 1<sup>st</sup> lap and 4<sup>th</sup> lap, [ $F = 16.94$ ,  $p < 0.05$ ].

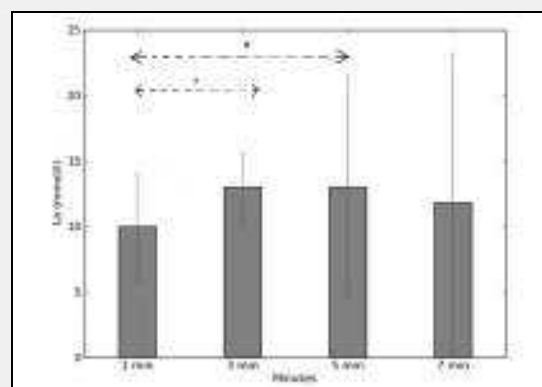


Figure 5. Blood lactate concentration after swim 1, 3, 5 and 7 min. \*  $p < 0.05$ .

### Physiology

The means and standard deviations in blood lactate concentration (La) measured after the 200 m breaststroke are shown in the Figure 5. The highest value ( $13.02 \pm 1.72 \text{ mmol}\cdot\text{l}^{-1}$ ) was observed at the 3<sup>rd</sup> minute after the trial. Significant variations were observed in La between the

**Table 1. Mean ( $\pm$ SD) duration of active phase for active phase<sub>beg</sub> and active phase<sub>end</sub> for all the muscles (BB- biceps brachii; DA- deltoid anterior; PM- pectoralis major and TB- triceps brachii), over the four laps of the 200 m breaststroke maximal effort.**

Laps	1 <sup>st</sup> lap		2 <sup>nd</sup> lap		3 <sup>rd</sup> Lap		4 <sup>th</sup> Lap	
	Active phase <sub>beg</sub> (s)	Active phase <sub>end</sub> (s)	Active phase <sub>beg</sub> (s)	Active phase <sub>end</sub> (s)	Active phase <sub>beg</sub> (s)	Active phase <sub>end</sub> (s)	Active phase <sub>beg</sub> (s)	Active phase <sub>end</sub> (s)
BB	.99 (.78)	.98 (.90)	.73 (.26)	.58 (.14)	.68 (.27)	.56 (.20)	.70 (.24)	.63 (.10)
DA	.78 (.30)	.64 (.17)	.63 (.14)	.72 (.14)	.87 (.67)	.58 (.25)	.82 (.38)	.93 (.58)
PM	.61 (.18)	.71 (.32)	.64 (.17)	.88 (.48)	.69 (.62)	.55 (.33)	.62 (.35)	.79 (.63)
TB	.71 (.21)	.69 (.25)	.54 (.14)	.58 (.14)	.54 (.22)	.52 (.15)	.84 (.74)	.85 (.76)

1<sup>st</sup> minute and 3<sup>rd</sup> minute, and between the 1<sup>st</sup> minute and the 5<sup>th</sup> minute [ $F = 2.47$ ,  $p < 0.05$ ].

### Neuromuscular

The duration of the active phase (Table 1) was, for the BB muscle, higher in the beginning phase compared to the end phase of the swim for all four laps. The DA muscle presented a higher value in the beginning phase in the 1<sup>st</sup> lap and 3<sup>rd</sup> lap, but exhibited a higher value in the end phase for the 2<sup>nd</sup> and 4<sup>th</sup> lap. The PM muscle demonstrated a higher duration of active phase at the end for all laps with the exception of the 3<sup>rd</sup> lap (PM<sub>beg</sub>:  $0.69 \pm 0.62$  s and PM<sub>end</sub>:  $0.55 \pm 0.33$  s) where the active phase was higher at the beginning than the end of the session. The TB muscle showed a higher active phase at the beginning in the 1<sup>st</sup> and 3<sup>rd</sup> lap, and higher values in the end in the 2<sup>nd</sup> and 4<sup>th</sup> lap.

The long duration of active phase was shown in BB at the beginning of the swim in the 1<sup>st</sup> lap ( $0.99 \pm 0.78$  s) and the shorter for the TB at the end of swimming the 3<sup>rd</sup> lap ( $0.52 \pm 0.15$  s). Moreover, for all the muscles, the active phase showed the same trend in the 3<sup>rd</sup> lap. The duration was higher in the beginning compared to the end of the swim. The differences between beginning and the end for each muscle were not statistically significant for  $p < 0.05$ .

The non-active phase in Table 2, showed an increase in the end of the test for all the muscles in the 3<sup>rd</sup> lap and a decrease in the 4<sup>th</sup> lap. The muscles BB, DA and PM demonstrated the same behavior in the relative duration of the non-active phase for each lap of the 200m breaststroke, that is, in the 1<sup>st</sup> and 3<sup>rd</sup> lap they had a longer duration at the end compared to the beginning of the swim, unlike in the 2<sup>nd</sup> and 4<sup>th</sup> lap where the higher duration was observed in the beginning of the swim.

The TB showed a different behavior compared to the other muscles studied, which presented a higher duration in the non-active phase in the beginning compared to the end of the swimming the 1<sup>st</sup> and 4<sup>th</sup> lap, and in the end for the 2<sup>nd</sup> and 3<sup>rd</sup> laps, moreover for all the muscles under study the non-active phase showed the same behav-

ior in the 3<sup>rd</sup> lap, where the duration was higher in the beginning which also happened in the active phase. The differences between the beginning and the end for each muscle were not statistically significant for  $p < 0.05$ .

The relative duration of activation (RAF) in Figure 6, decreases at the beginning phase in the 1<sup>st</sup> and 3<sup>rd</sup> lap for the DA and PM, whereas for the 2<sup>nd</sup> and 4<sup>th</sup> lap it was higher in the end for both the DA and PM. The BB demonstrated a RAF higher in the end phase in the 1<sup>st</sup> and 2<sup>nd</sup> lap, and the reverse in the 3<sup>rd</sup> and 4<sup>th</sup> lap. The TB decreased at the beginning phase over the first three laps, in contrast to the 4<sup>th</sup> lap where the value was higher in the end. The differences between the beginning and the end for each muscle were not statistically significant for  $p < 0.05$ .

Figure 7 demonstrated that the average rectified value increases at the end phase compared to the beginning phase for all muscles comparing the 1<sup>st</sup> lap with the 4<sup>th</sup> lap, except for the DA (DA<sub>beg</sub>:  $0.42 \pm 0.13$ ; DA<sub>end</sub>:  $0.42 \pm 0.10$ ) that demonstrated higher values in the beginning phase. The BB and TB muscles have similarities throughout the laps, that is, in the first three laps (1<sup>st</sup>, 2<sup>nd</sup> and 3<sup>rd</sup> laps) the ARV was higher in the end compared to the beginning and in the 4<sup>th</sup> lap the reverse was observed. The PM showed similarities in the 1<sup>st</sup> and 3<sup>rd</sup> lap which had higher values in the beginning, and in the 2<sup>nd</sup> and 4<sup>th</sup> lap had higher values at the end.

In Figure 8, the mean MNF value for the BB increased in all the laps at the end except the 3<sup>rd</sup> lap (BB<sub>end</sub>:  $82.47 \pm 16.20$  Hz; BB<sub>beg</sub>:  $86.15 \pm 11.72$  Hz). DA decreases in all the laps at the beginning except in the 3<sup>rd</sup> lap (DA<sub>end</sub>:  $77.38 \pm 13.68$  Hz; DA<sub>beg</sub>:  $74.54 \pm 15.52$  Hz). The PM decreases at the beginning in the 1<sup>st</sup> lap (PM<sub>beg</sub>:  $89.65 \pm 23.61$  Hz; PM<sub>end</sub>:  $89.53 \pm 17.74$  Hz) and 3<sup>rd</sup> lap (PM<sub>beg</sub>:  $79.37 \pm 10.58$  Hz; PM<sub>end</sub>:  $76.16 \pm 16.92$  Hz), and increases at the beginning in the 2<sup>nd</sup> lap (PM<sub>beg</sub>:  $82.13 \pm 15.33$  Hz; PM<sub>end</sub>:  $86.11 \pm 18.12$  Hz) and 4<sup>th</sup> lap (PM<sub>beg</sub>:  $74.33 \pm 14.61$  Hz; PM<sub>end</sub>:  $75.18 \pm 13.92$  Hz). TB decreases at the beginning in the 1<sup>st</sup> lap (TB<sub>beg</sub>:  $78.17 \pm 10.83$  Hz; TB<sub>end</sub>:  $74.79 \pm 16.37$  Hz) and in the 4<sup>th</sup> lap (TB<sub>beg</sub>:  $72.45 \pm 11.83$  Hz; TB<sub>end</sub>:  $65.72 \pm 12.94$  Hz),

**Table 2. Mean ( $\pm$ SD) duration of non-active phase for non- active phase<sub>beg</sub> and non- active phase<sub>end</sub> for all the muscles (BB- biceps brachii; DA- deltoid anterior; PM- pectoralis major and TB- triceps brachii), over the four laps of the 200 m breaststroke maximal effort.**

Laps	1 <sup>st</sup> lap		2 <sup>nd</sup> lap		3 <sup>rd</sup> Lap		4 <sup>th</sup> Lap	
	Non-Active phase <sub>beg</sub> (s)	Non-Active phase <sub>end</sub> (s)	Non-Active phase <sub>beg</sub> (s)	Non-Active phase <sub>end</sub> (s)	Non-Active phase <sub>beg</sub> (s)	Non-Active phase <sub>end</sub> (s)	Non-Active phase <sub>beg</sub> (s)	Non-Active phase <sub>end</sub> (s)
BB	.76 (.24)	.80 (.24)	.81 (.21)	.72 (.22)	.77 (.20)	.81 (.28)	.79 (.26)	.78 (.17)
DA	.66 (.16)	.71 (.14)	.79 (.15)	.69 (.17)	.70 (.16)	.75 (.19)	.70 (.24)	.68 (.26)
PM	.60 (.18)	.60 (.13)	.70 (.24)	.68 (.32)	.63 (.21)	.65 (.17)	.72 (.20)	.68 (.16)
TB	.63 (.27)	.62 (.18)	.55 (.17)	.67 (.26)	.59 (.17)	.64 (.29)	.61 (.18)	.53 (.22)

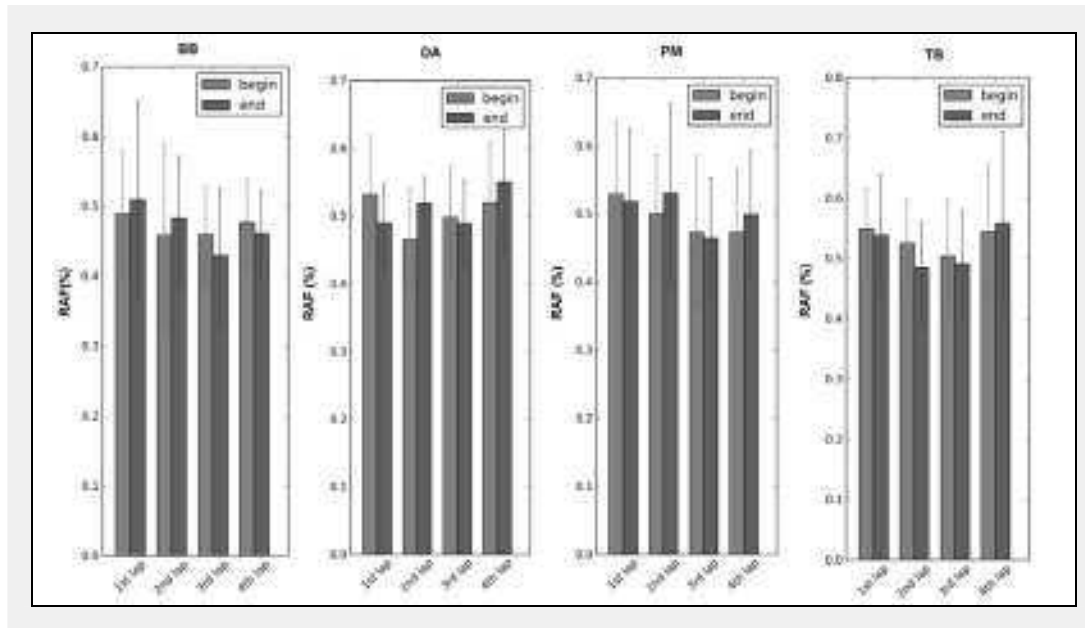


Figure 6. Comparison of the mean relative duration of activation value with SD for  $RAF_{beg}$  (gray bars) and  $RAF_{end}$  (black bars) for all the muscles (BB- biceps brachii; DA- deltoid anterior; PM- pectoralis major and TB- triceps brachii), over the four laps of the 200 m breaststroke maximal effort.

whereas in the 2<sup>nd</sup> ( $TB_{end}$ :  $73.72 \pm 13.68$  Hz;  $TB_{beg}$ :  $72.41 \pm 12.65$  Hz) and 3<sup>rd</sup> ( $TB_{end}$ :  $71.32 \pm 11.26$  Hz;  $TB_{beg}$ :  $68.78 \pm 11.03$  Hz) we can observe an increase at the end phase compared to the beginning. Moreover, the MNF decreases at the end of swimming the 4<sup>th</sup> lap relative to the 1<sup>st</sup> lap for all muscles under observation for the 200m breaststroke. Note that the differences between the beginning and the end for each muscle were not statistically significant for  $p < 0.05$ .

## Discussion

The aims of this study were : i) to analyze four muscle

activation patterns (duration of active and non-active phases) of the upper limbs during each lap for the 200m breaststroke, ii) quantify neuromuscular fatigue, through kinematics and physiologic assessment. The main findings were that some indicators of neuromuscular fatigue begin to appear shortly into the 2<sup>nd</sup> lap, but are more evident in the 3<sup>rd</sup> lap (i.e amplitudes increase and spectral variables decrease) in a non-linear fashion.

## Kinematics

The average decrease in the swimming speed from the 1<sup>st</sup> lap to the 4<sup>th</sup> lap shows a much greater decline in speed compared to the results of similar studies (Takagi et al.,

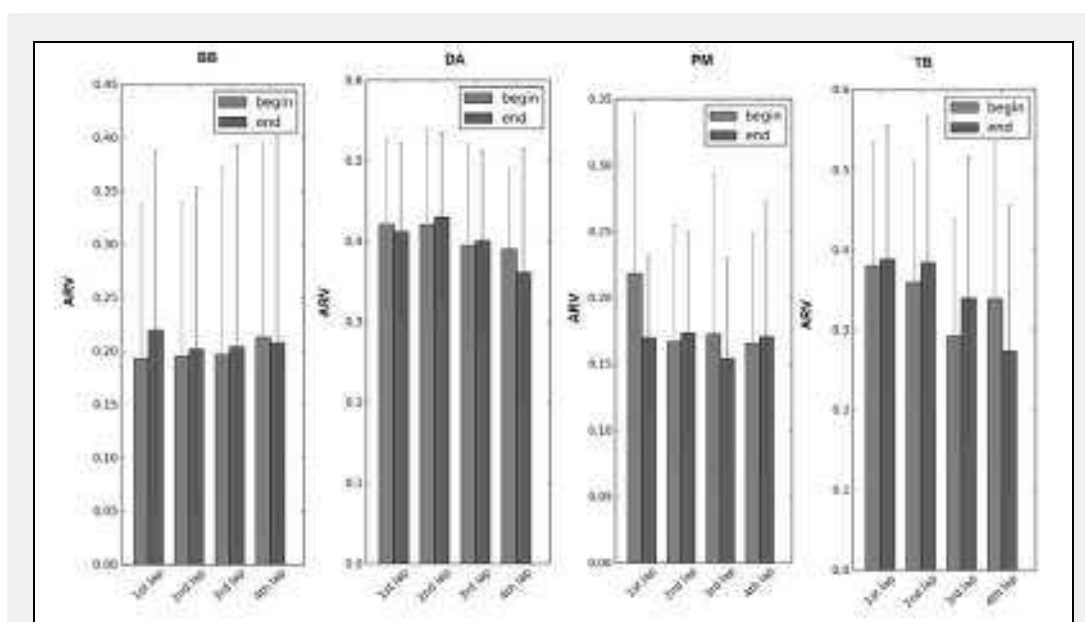


Figure 7. Mean ARV value with SD for  $ARV_{beg}$  (gray bars) and  $ARV_{end}$  (black bars) for all the muscles (BB- biceps brachii; DA- deltoid anterior; PM- pectoralis major and TB- triceps brachii), over the four laps of the 200 m breaststroke maximal effort.

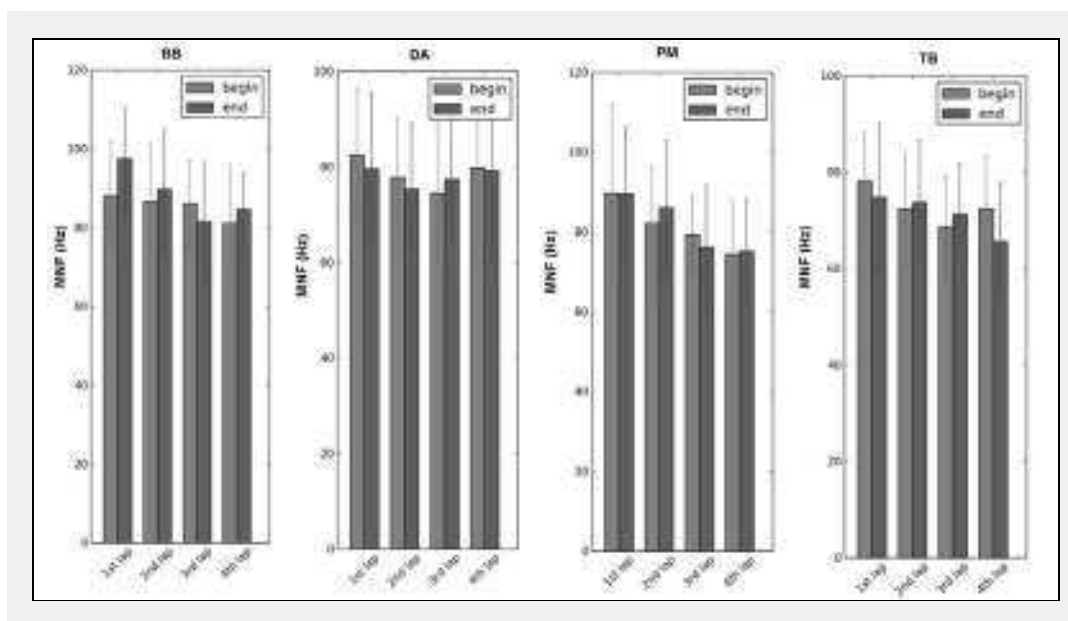


Figure 8. Mean MNF value with SD for MNF<sub>beg</sub> (black bars) and MNF<sub>end</sub> (gray bars) for all the muscles (BB- biceps brachii; DA- deltoid anterior; PM- pectoralis major and TB- triceps brachii) and swimming bouts, over the four laps of the 200 m breaststroke maximal effort.

2004; Thompson et al., 2000; 2004). In breaststroke, an increase in swimming speed is associated with an increase in SR, but has a decline more in SL relative to the other swimming strokes (Thompson et al., 2004), supporting the changes in the swimming technique under the 200m breaststroke (Takagi et al., 2004). In this study SL suffered a decrease in the 1<sup>st</sup> lap to the 4<sup>th</sup> lap, whereas the SR decreased from the 1<sup>st</sup> lap to the 3<sup>rd</sup> lap and then increased in the 4<sup>th</sup> lap.

As swimming speed decreased, different SR and SL combinations were observed, giving the best performance in the face of the fatigue task constraints, and decreasing the swimmer's ability to maintain a constant SL. Increasing SR in the last 50m lap is due to the inability to generate sufficient extra effort to overcome high drag. The decrease in the swimming speed SL, SI and the increase of SR in the last lap suggested a trend for the inability to generate sufficient extra effort needed to overcome high drag throughout the test (Craig and Pendergast, 1979; Chatard et al., 2003; Thompson et al., 2004). This change in the stroke kinematics due to a decrease in the power output can be a consequence of peripheral fatigue phenomena (Ikuta et al., 2012; Stirn et al., 2011).

### Physiology

The blood lactate concentration levels after the test reached values that would be expected after a 200m breaststroke race and are in agreement with the results in other studies in which similar tests were performed with swimmers of the same or similar competitive level (e.g., Capelli et al., 1998; Lomax and Castle, 2011). These results suggest that the swimmers performed the swimming test very close to their maximum effort level. The average blood lactate concentration measured after 3 minutes was  $13.2 \pm 1.72 \text{ mmol} \cdot \text{l}^{-1}$ , representing the highest values. Increases of lactate concentration are responsible for fatigue by changes in the intracellular pH. As a

result, muscle fiber conduction velocity decreases and changes the shape of the motor unit action potential waveform (Cifrek et al., 2009).

### Neuromuscular

The amplitude of EMG signals is influenced by the number of active motor units (Moritani et al., 1986), their discharge rates, and the shape and propagation velocity of the intracellular action potentials (Dimitrova and Dimitrov, 2002).

The amplitude signal of EMG provided by the ARV demonstrated an increase and maintenance at the end phase compared to the beginning for BB and PM, comparing the 1<sup>st</sup> lap with the 4<sup>th</sup> lap, and a small decrease for DA and TB that demonstrated higher values in the beginning phase compared to the end phase. The swimmers, during the end of the swim, increase the period in the recovery phase, decreasing the amplitude of the EMG signal for DA and TB. These muscles are responsible for the movement of antepulsion of the arms up to the point where it is stretched in front.

EMG amplitude has been observed to increase during submaximal dynamic exercise (Tesch et al., 1990) and to decrease during exercises at maximal levels of voluntary contraction (Komi and Tesch, 1979). An increase of the EMG amplitude during swimming has already been observed in previous studies (Rouard and Clarys, 1995; Rouard et al., 1997; Stirn et al., 2011; Wakayoshi et al., 1994). Ikuta et al., (2012) noted that the mean amplitude value of the PM was significantly higher for the 4<sup>th</sup> lap than for the 2<sup>nd</sup> and 3<sup>rd</sup> laps in 4x50 m front crawl. The authors noted that the increase activity of the PM may have been a compensatory strategy to maintain swimming speed during the test swim. Moreover, Nuber et al. (1986) verified that the PM continued to activate through recovery in the breaststroke.

So, it seems that a decrease in the active phase du-



ration and the increase in the non-active phase on the 3<sup>rd</sup> lap, with all the muscles monitored, reveal that in this lap muscles start to display the onset of neuromuscular fatigue indicating that this is a milestone of the 200m breaststroke race.

The MNF decreases at the 4<sup>th</sup> lap in comparison to the 1<sup>st</sup> lap for all muscles. Dynamic fatiguing tasks have been found to show a decrease in the mean power frequency (Tesch et al., 1990). A number of authors reported similar data in several other human muscles (Basmajian and De Luca, 1985; De Luca, 1984). The same is also apparent for swimming, or at least for front-crawl (Figueiredo et al., 2011; Stirn et al., 2011). The reduction of muscle fiber conduction velocity is one of the causes of signal power spectrum shift toward lower frequencies, and also of the increase in the EMG signal amplitude because of a spatial low-pass filtering effect of tissue as a volume conductor (De Luca, 1984).

However, during a fatiguing exercise, two opposing effects might occur (Petrofsky and Lind, 1980): (i) a decrease in the mean frequency as discussed above and; (ii) increase in intramuscular temperature due to the exercise, which causes an increase in the mean frequency. So it is possible that during some types of exercise, the two effects compensate each other, and the decrements found in the mean frequency can be small or non-significant.

### Associations

A decrease in swimming speed, SL, SI and an increase in the SR in the last lap, can be associated to the higher La after the test, to a non-significant increase of the amplitude and a non-significant decrease in the spectral variables. These suggest that the presence of fatigue in the upper limbs occurs in a different way for the different muscles studied, by a non-linear process. However, more investigation in order to better understand this phenomenon is required in the near future.

As blood flow is restricted, at a certain level of contraction, by intramuscular pressure, muscle becomes ischemic. Myoelectric manifestations of muscle fatigue might be affected by this event (Merletti et al., 1984). However, these are evidences for land-based exercises, and to the best of our knowledge none of this data has been collected during swimming.

The shape of the ARV during the four laps represent the increased activation patterns in the 2<sup>nd</sup> and 3<sup>th</sup> lap in accordance with the normal adaptation and the tactic to use the remaining capacity in the final lap to achieve the best result. The higher activation by DA and TB showed that these muscles play a significant role in this event (Yoshizawa et al., 1976; Conceição et al., 2010).

With this research we can provide quick practical information for swimmers and coaches to adjust training methods.

### Conclusion

This study showed a trend for a neuromuscular fatigue state in a non-linear fashion, with the 2<sup>nd</sup> and 3<sup>rd</sup> lap being the critical moments of a 200m breaststroke race. Decreases in swimming speed are related to SL, SI, and

increases of the SR in the last lap, increases in the blood lactate concentration after the test, and a non-significant increase of the amplitude and non-significant decrease in the spectral parameters.

Both the technical and physiologic training in breaststroke swimmers should take into account the onset of fatigue in a non-linear fashion, including specific training to prevent sharp falls in the 2<sup>nd</sup> and 3<sup>rd</sup> laps of competitive racing.

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### Key points

- Fatigue in the upper limbs occurs in different way as it described by 100m swimming events.
- Neuromuscular fatigue was estimated by analyzing the physiological changes (high blood lactate concentrations), biomechanical changes in the swimming stroke characteristics (decreased in swimming velocity), and by the changes in the EMG amplitude and frequency parameters at the end of the swimming bout.
- The amplitude signal of EMG provided by the ARV demonstrated an increase at the end with the respect to the beginning for all muscles under study, excepted for the muscle deltoid anterior.
- The mean frequency (MNF) in our study decrease at the end of the swimming in the 4th lap relative to the 1st lap for all muscles under observation, along the 200m breaststroke.

### AUTHORS BIOGRAPHY



#### Ana CONCEIÇÃO

##### Employment

Assistant Professor at the Sports Science Institute of Rio Maior, Rio Maior, Portugal.

##### Degree

PhD

##### Research interests

Swimming biomechanics and muscle mechanics.

E-mail: [anaconceicao@esdrm.ipsantarem.pt](mailto:anaconceicao@esdrm.ipsantarem.pt)



#### António José SILVA

##### Employment

Full Professor at the Sport Sciences Department of the University of Trás-os-Montes and Alto Douro, UTAD - Portugal.

##### Degree

PhD

##### Research interests

The physiological and biomechanical indicators of energy cost during physical activities, namely in swimming.

E-mail: [ajsilva@utad.pt](mailto:ajsilva@utad.pt)



#### Tiago BARBOSA

##### Employment

Professor at the Nanyang Technological University, and Member of the Research Centre in Sports, Health and Human Development, Portugal.

##### Degree

PhD

##### Research interests

The biomechanical and physiological determinant factors of swimming performance.

E-mail: [tiago.barbosa@nie.edu.sg](mailto:tiago.barbosa@nie.edu.sg)



#### István KARSAI

##### Employment

Assistant Professor at the University of Pécs, Pécs, Hungary.

##### Degree

PhD

##### Research interests

Swimming biomechanics and muscle mechanics.

E-mail: [karsai@gamma.ttk.pte.hu](mailto:karsai@gamma.ttk.pte.hu)



#### Hugo LOURO

##### Employment

Professor at the Sports Science Institute of Rio Maior, Rio Maior, Portugal.

##### Degree

PhD

##### Research interests

Methodological observation in swimming.

E-mail: [hlouro@esdrm.ipsantarem.pt](mailto:hlouro@esdrm.ipsantarem.pt)

#### ✉ Ana Conceição

Sports Sciences School of Rio Maior, Polytechnic Institute of Santarém, Av. Dr. Mário Soares, 2040-413 Rio Maior, Portugal