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ORIGINAL ARTICLE

Evaluation of muscle fatigue during 100-m front crawl

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Abstract The aim of this study was to evaluate muscle fatigue in upper body muscles during 100-m all-out front crawl. Surface electromyogram (EMG) was collected from the pectoralis major, latissimus dorsi and triceps brachii muscles of 11 experienced swimmers. Blood lactate concentration level increased to 14.1 ± 2.9 mmol l^{-1} 5 min after the swim. The velocity, stroke length and stroke rate calculated based on video analysis decreased by 15.0, 5.8 and 7.4%, respectively, during the swim. EMG amplitude of the triceps and the lower part of the latissimus muscles increased, whilst the mean power frequency (MNF) of all muscles significantly decreased by 20–25%. No significant differences in the relative MNF decrease were observed amongst the muscles; however, the differences in the rate of the MNF decrease between the lower part of the latissimus and the triceps brachii muscles were found ($P < 0.05$). The time of rest between the muscle activation of the two consecutive arm strokes at the end of swimming was extended ($P < 0.05$). It was concluded that 100-m all-out crawl induced significant fatigue with no evident differences amongst the analysed muscles.

Keywords Swimming · Stroke parameters · EMG · Power spectrum · Mean frequency

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Introduction

Fatigue during swimming has been widely examined by monitoring different physiological, kinematic and biomechanical parameters such as swimming velocity, acceleration, stroke length, stroke rate, arm coordination, spatiotemporal parameters (such as 3-D fingertip pattern), mechanical power output, (net) energy cost, heart rate, oxygen consumption, blood lactate concentration and the isometric force (Alberty et al. 2008; Aujouannet et al. 2006; Billat et al. 1996; Bonifazi et al.1993; Dekerle et al. 2005; Fernandes et al. 2006; Rouard et al. 1997; Seifert et al. 2005, 2007; Tella et al. 2008; Toussaint et al. 2006). All these studies demonstrate that development of muscle fatigue during swimming is an interesting topic, which can be studied using various methods and parameters. However, there were fewer attempts to establish differences amongst the muscles related to swimming.

A significant part of energy consumed during 100-m all-out swimming is derived from anaerobic metabolism, which causes lactate concentration in blood to reach values of 10–20 mmol l^{-1} during swimming sprint events (Bonifazi et al. 1993). The accumulated hydrogen ions interfere with the muscle contraction mechanisms, thus causing a decrease in the generated muscle force (Fitts 1994). This was shown by comparing isometric forces measured before and after a four 50-m all-out effort laps (Aujouannet et al. 2006) and by the decrease in the mechanical power output measured during a 100-m all-out front crawl swimming test using arms only on a MAD system, a system for active drag measurement (Toussaint et al. 2006). Due to reduced muscle force produced by the fatiguing muscles, the swimmer is unable to sustain the initial stroke length (SL), which has been used as one of the first indicators of fatigue (Dekerle et al. 2005; Keskinen

and Komi 1993; Weiss et al. 1988). Swimmers often compensate for the decrease in SL with a higher stroke rate (SR) to maintain the velocity (Alberty et al. 2008), but as the fatigue progresses the stroke rate and, consequently, the swimming velocity both diminish as well. Some authors have found that the relative duration of the non-propulsive phase within a stroke is reduced with respect to the propulsive phase with progressive fatigue, which reflects in changed arm coordination (Alberty et al. 2008; Seifert et al. 2007).

Important muscles engaged in front crawl swimming are the gluteus maximus, the abdominal muscles and some upper trunk and arm muscles: *latissimus dorsi*, *pectoralis major* and *triceps brachii* (Piette and Clarys 1979; Clarys et al. 1983), *flexor carpi ulnaris* (Clarys et al. 1983; Rouard et al. 1997; Caty et al. 2006) and *extensor carpi ulnaris* (Caty et al. 2006). It is generally accepted that the muscles involved in arm and shoulder rotation contribute most significantly to the propulsion in crawl and are responsible for approximately 85% of the propulsion (Hollander et al. 1988; Deschodt et al. 1999). The *pectoralis major* and *latissimus dorsi* muscles propel the body (Nuber et al. 1986; Scovazzo et al. 1991), whilst the triceps brachii plays an important role by extending the elbow at the end of the underwater part of the arm stroke (Maglischo 2003).

To better understand the fatigue process during front crawl, the role of individual muscles should be considered. The use of surface electromyography enables monitoring of the fatigue process in different muscles simultaneously (De Luca 1984; Masuda et al. 1999). The amplitude of the surface EMG during sustained muscle contraction increases due to synchronisation of the recruited motor units (MU) and activation of new ones (Lowery et al. 2002; Masuda et al. 1999; Merletti et al. 1991, 2004). During swimming, this has been shown by Wakayoshi et al. (1994), who estimated the degree of muscle fatigue of the flexor carpi radialis, biceps brachii, triceps brachii and deltoid muscles at different swimming velocities. Only the deltoid muscle showed an increased integrated EMG (iEMG) at swimming velocities between 1.3 and 1.4 ms^{-1}, whilst other muscles showed no significant changes. In another study involving a 4 × 100-m front crawl at approximately 85% of swimmer's best performance, only *flexor carpi ulnaris* muscle showed an increased iEMG regardless of the level of exhaustion observed in the subjects at the end of the swim (Rouard and Clarys 1995; Rouard et al. 1997).

For evaluation of muscle fatigue, the EMG signal is commonly analysed in the frequency domain (Gerdle et al. 1988; 2000; Komi and Tesch 1979; Masuda et al. 1999; Merletti and Roy 1996). A power spectral density (PSD) estimate is usually calculated from the raw EMG signal. The mean (MNF) or median (MDF) frequency of the PSD was shown to shift to lower frequencies during increasing fatigue (De Luca 1979; Lindstrom and Magnusson 1977; Viitasalo and Komi 1977; Merletti et al. 1990; Merletti and Lo Conte 1997). MNF and MDF decrease has been largely attributed to the diminished muscular fibre conduction velocity (MFCV) as a consequence of local metabolic changes in the working muscle (Bigland-Ritchie 1981; Masuda et al. 1983). However, the modifications of the motor unit (MU) action potential shape, MU firing rate and synchronisation of MUs may contribute to MNF and MDF changes as well (Brody et al. 1991; Bigland-Ritchie and Woods 1984; Dimitrova and Dimitrov 2003; Gabriel and Kamen 2009).

Due to the high level of nonstationarity of EMG signals during dynamic muscle contractions (the signal properties change rapidly in time), the spectral properties of such signals should in general be analysed using methods capable of simultaneous presentation of signal properties in time and frequency domains. Various advanced methods have been utilised successfully for time–frequency analysis of EMG, such as Cohen-class transformations (Bonato et al. 1996; Knaflitz and Bonato 1999; Bonato et al. 2001) and continuous wavelet transform (e.g. Karlsson et al. 2000). However, even the simpler method of short-time Fourier transform can be applied effectively in the analysis of dynamic muscle contractions for detection of decrease in MNF and MDF commonly associated with the development of fatigue (Mac Isaac et al. 2001; Gerdle et al. 2000; Christensen et al. 1995).

Only few studies concerning swimming took interest in the analysis of EMG in the frequency domain. Aujouannet et al. (2006) evaluated muscle fatigue by comparing EMG measured during isometric contraction of arm flexors before and after swimming. MNF of the EMG power spectrum for the biceps brachii and triceps brachii muscles decreased after a 4 × 50 m front crawl at maximum intensity with respect to the values obtained before the swim. But to the best of our knowledge, only one study analysed changes in MNF during the swim itself (Caty et al. 2006). Two forearm muscles were examined and a decrease of instantaneous MNF in the *extensor carpi ulnaris* and *flexor carpi ulnaris* muscles was observed (11.41 and 8.55%, respectively) during the 4 × 50 m front crawl swimming and was attributed to fatiguing of the muscles due to their wrist stabilisation role during swimming.

Besides Caty et al. (2006), all other studies on development of muscle fatigue during swimming were based on the analysis of EMG amplitude in the time domain. However, the diagnostic value of time domain methods in muscle fatigue evaluation is considered to be more limited than that of the frequency domain methods (Merletti et al. 2004). The aim of the present study was therefore to evaluate peripheral muscle fatigue during 100-m maximum

effort front crawl swimming in selected upper trunk and arm muscles by means of the amplitude and frequency parameters of EMG, supported by some kinematic and physiological data.

Methods

Subjects

Eleven male swimmers (age 22.0 ± 2.9 years; height 184.8 ± 8.2 cm; weight 77.2 ± 4.9 kg, mean ± SD) were included in the study. They were all experienced competitive swimmers, who had been involved in swimming for 13.6 ± 3.1 years on average with an average personal best result in 100-m front crawl of 53.05 ± 1.72 s. However, not all of them were front crawl specialists. In accordance with other similar studies (Toussaint et al. 2006; Seifert et al. 2007), the swimmers who participated in our study may be considered to be experienced elite swimmers. Their regular weekly training schedule consisted of approximately 12–20 h of swimming and additionally of approximately 4–5 h of dryland training. Swimmers were asked to refrain from intensive strength or swimming training 2–3 days prior to the study. They were fully explained about the procedures and the purpose of the study, and they all signed the informed consent form. The study was approved by the Slovenian National Medical Ethics Committee.

Testing procedure

Measurements were performed in a 25-m indoor swimming pool. After putting on all the equipment, the subjects performed a warm-up series of ten 50-m front crawl laps at a medium level of effort. After the warm-up, the subjects were instructed to perform a 100-m front crawl swim at a maximum perceived effort level. Due to the measurement equipment that was attached to the swimmers, they started with pushing off from the side of the pool and were not allowed to perform underwater turns.

Data acquisition

Blood samples from the earlobe were taken before swimming, immediately after swimming, and 3 and 5 min after swimming. Blood lactate concentrations were measured post-exercise using an Eppendorf (Germany) lactometer.

Each swimmer was filmed with a video camera at 50 frames per second acquisition rate. The camera was fixed to a pushcart and pushed along the pool in parallel with the swimmer's head. The pushcart was controlled by an operator who moved it at the swimmer's velocity to minimise parallax error. Equidistant markers placed along the poolside were used to measure the distance of the head with respect to the starting wall at any moment. This position was used to calculate the clean swimming velocity (V), the stroke length (SL) and the stroke rate (SR). All three parameters were calculated only for the middle 15 m of the length of the swimming pool to avoid the influence of the turn and push-off at the swimming pool wall on calculation of these parameters.

The average stroke rate for every 25-m lap was calculated from the number of full strokes executed between the marks for 5 and 20 m (of a 25-m swimming pool) and the number of video frames (at 50 fps resolution) needed for completion of these full strokes. The average stroke length was calculated from the total distance covered during these full strokes divided by the number of full strokes. The clean swimming velocity was calculated from the total distance covered during these full strokes divided by the corresponding time obtained from the number of video frames for this distance.

Collection of the EMG data

Surface EMG signals from the *pectoralis major* (PM), *latissimus dorsi* (LD) and *triceps brachii* (TB) muscles on the right side of the body were measured. These muscles were chosen because of their importance in front crawl as mentioned in the introduction (Clarys et al. 1983; Nuber et al. 1986; Rouard et al. 1997; Maglischo 2003). *Pectoralis major* and *latissimus dorsi* are large muscles and therefore separate muscle compartments may be involved in their contractions. Therefore, EMG was collected separately from the *upper* and *lower* parts of these muscles (marked as PM1, LD1 and PM2, LD2, respectively). Bipolar Ag–AgCl surface electrodes were used (9-mm diameter discs, Hellige, Freiburg, Germany) with the inter-electrode distance of 20 mm.

The electrodes on the long head of the TB muscle were placed in accordance with SENIAM recommendations (Herrmens and Freriks 1999). Electrodes for the upper part of PM (PM1) were placed in the middle of the line that connects the acromion process and the manubrium (sternum) two fingers below the clavicle. Electrodes for the lower part of PM (PM2) were placed two fingers above the areola. Electrodes for the upper part of LD (LD1) were placed two fingers below the inferior angle of the scapula, and for the lower part of LD (LD2) at the lateral side of the muscle at the height of the L3 vertebra.

The skin under the electrodes was shaved, rubbed gently with sandpaper and cleaned with alcohol so that the inter-electrode resistance did not exceed 5 kOhm. The ground electrode was positioned over the cervical vertebrae. Transparent dressings with label (Tegaderm™, 6 cm × 7 cm,

3 M, USA) were used to cover the electrodes to isolate them from the water.

All cables were fixed to the skin by adhesive tape in several places to minimise their movement and consequently their interference with the signal. To additionally immobilise the cables, the swimmers wore a thin long-sleeved custom-made swimming suit.

The EMG equipment carried by the swimmer was very light consisting of only the electrodes, the corresponding cables and the transparent dressings used to protect them from the water. The telemetric EMG device (Biotel 88, Glonner, Munchen, Germany; differential amplifier, input impedance 16 Mohm, CMRR 85 dB, input noise <1 μV rms, gain 1000, input filter 6-pole Butterworth type, bandwidth 16–500 Hz) was fixed to a rod and carried alongside the pool above the swimmer by an assistant. The data were recorded using Dasy Lab 7.0 software (2002, National Instruments) at a sampling frequency of 2,000 Hz.

For detection of water immersion of the hand at each stroke, a conductance sensor (Furlan EMF, Ljubljana, Slovenia) was placed on the middle finger of the arm under observation. Whilst the sensor was immersed in the water, the electrical circuit was closed, and whilst it was outside the water, the circuit was open. This resulted in a binary signal at the sensor's output, which was used as an indicator of the above- and underwater phases of each swimming stroke. EMG and water immersion sensor signals were recorded simultaneously and were not synchronised with video recording. The water immersion sensor was used to determine the activation of muscles in the underwater and the above-water phase of the entire arm stroke.

EMG signal processing

MATLAB software (MathWorks, Inc., Natick MA, USA) was used for signal processing. Raw EMG signals were filtered using the fifth-order Butterworth band-pass filter with the lower and upper cutoff frequencies set to 10 and 500 Hz, respectively.

To obtain the amplitude and frequency description of EMG signal for each swimming stroke, the active phase of a muscle was determined individually for every stroke and muscle. The procedure is illustrated in Fig. 1. First, the energy envelope $E(t)$ of the rectified EMG signal. $x(t)$ was calculated using a sliding data window of length 250 ms and according to Eq. 1:

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

Muscle activation within each stroke resulted in a local maximum in the energy envelope.

For each muscle activation, we defined its "active" phase (AF) as the part of the EMG signal for which the energy of the EMG was at least 30% of the local maximum energy value for the particular muscle activation. The raw EMG segments belonging to the active phases were extracted and used for calculation of the active phase duration and for amplitude frequency analysis of the EMG. The non-active phase (NAF) was defined as the time interval between the two successive active phases as shown in Fig. 1.

It should be noted here that our use of the term "active phase" is somewhat relaxed with respect to the usual use of this term in muscle activation studies. Our aim was simply to extract from each stroke a period of relatively high muscle activity (high power) and to discard regions of relatively low activity in both "tails" left and right from the peak activity, which would be considered a part of the activation phase in a strict sense. For this reason, we used a relatively high level of threshold for the boundaries of the active phase, which worked well for all muscles under

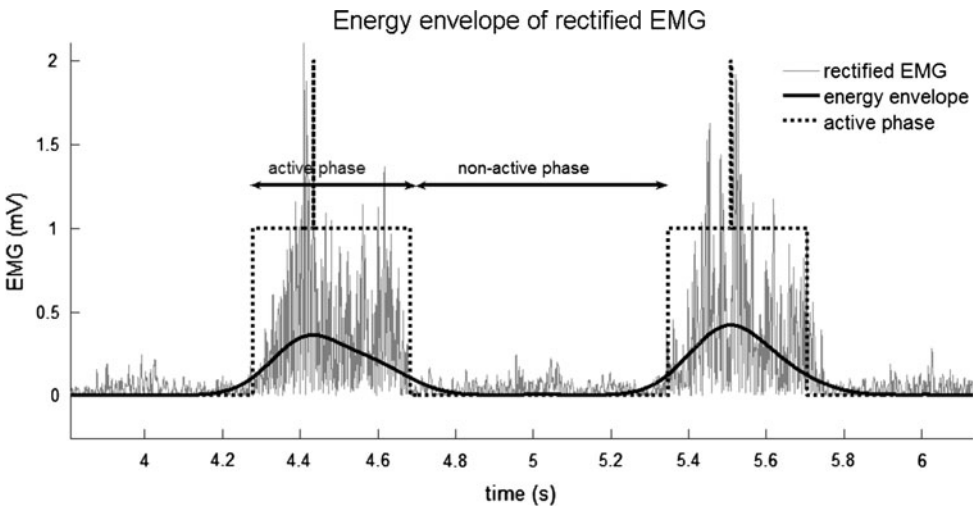

Fig. 1 Energy envelope of the rectified EMG signal of LD1 of three consecutive muscle contractions during two consecutive arm strokes. Local maximums in the energy envelope and the extracted parts of the signal used for the analysis are shown

observation (30% of the peak activity, see Fig. 1 for an example), and also the peak activity was determined for each stroke individually. The same criteria were applied to all EMG signals analysed in this study.

The average duration of five active and non-active phases of the stroke were calculated for each muscle at the beginning (from the second to the sixth stroke of the first 25-m lap) and at the end of swimming (five consecutive strokes without the last stroke in the fourth 25-m lap). First and last strokes of the 100-m swim were excluded from the analysis. The comparisons of the average duration 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 relative duration of muscle activation was also computed by Eq. 2:

$$\mathrm{RAF} = \frac{\mathrm{AF}}{\mathrm{AF} + \mathrm{NAF}} \tag{2}$$

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. Linear regression curve was fitted to the data and the ARV values of the fitted curve at the time of the first and last stroke were compared.

For the frequency analysis, each extracted segment was additionally detrended and zero-padded to the total length of 1 s (2,000 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 continuous signal segment $x(t)$ of length T is defined as:

$$P_X(f) = \frac{1}{T}|X(f)|^2 \tag{2a}$$

Even though the periodogram is a nonconsistent 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 improve significantly the estimation of power spectrum central frequency measures (the mean or the median frequency (Farina and Merletti 2000). We have also verified this for our own signals by comparing the Welch estimate (calculated as an average of modified periodograms), the estimate based on a 12th-order autoregressive model of EMG, and the periodogram estimate. 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. For the continuous spectrum spanning, the frequencies between 0 and f_{MAX} is defined as:

$$\mathrm{MNF} = \frac{\int_0^{f_{\mathrm{MAX}}} f \cdot P_X(f)df}{\int_0^{f_{\mathrm{MAX}}} P_X(f)df} \tag{2b}$$

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), labelled as $\mathrm{MNF}_{\mathrm{beg}}$ and $\mathrm{MNF}_{\mathrm{end}}$, respectively. To normalise results between subjects, the final MNF values were expressed as a percentage of the initial values and labelled $\mathrm{MNF}_{\mathrm{n}}$ as shown in Eq. 3:

$$\mathrm{MNF}_{\mathrm{n}} = \frac{\mathrm{MNF}_{\mathrm{end}}}{\mathrm{MNF}_{\mathrm{beg}}} \times 100 \tag{3}$$

The slope of the regression line was also calculated as an estimate of the rate of change over time. The assumptions of normality of data (Kolmogorov–Smirnov test) and of homogeneity of variance (Levene's test) were both confirmed for all parameters under investigation prior to the use of parametric statistical tests. The repeated measures ANOVA, with subsequent Tukey's test for post hoc analysis where applicable, were used for multiple comparisons between the groups. The differences were considered statistically significant for $P < 0.05$. All results are reported as the mean values along with standard deviation values.

Results

The average blood lactate values collected before and after the all-out 100-m swim are shown in Fig. 2. The lowest average values were measured in resting condition before the swim (1.8 ± 0.6 mmol l^{-1}), and the highest values (14.1 ± 2.9 mmol l^{-1}) were found 5 min after swimming. The absolute highest lactate value measured was 17.7 mmol l^{-1}.

The average swimming time achieved by the subjects in this study for the 100-m front crawl was 62.7 ± 2.4 s. Swimming velocity significantly decreased in every lap [$F(3,30) = 57.307$, $P < 0.05$] as shown in Fig. 3: by 6.18, 5.4 and 4.2% from the first to second, second to third and third to final lap, respectively. The total average decrease in velocity from 1.68 ± 0.08 ms^{-1} in the first 25-m lap to 1.43 ± 0.08 ms^{-1} in the last 25-m lap was 15.0%.

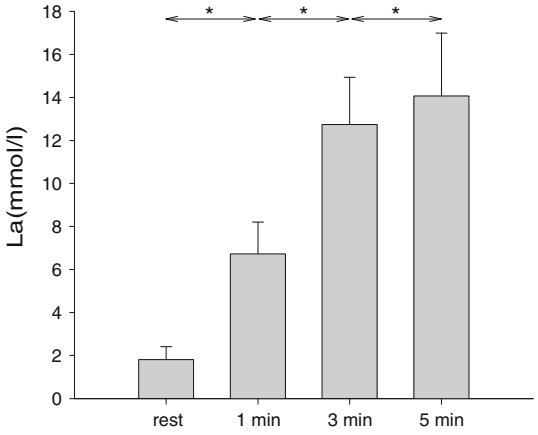

Fig. 2 Blood lactate concentration before swim and 1, 3 and 5 min after the swim. Significant differences are marked with *arrows* (*$P < 0.05$)

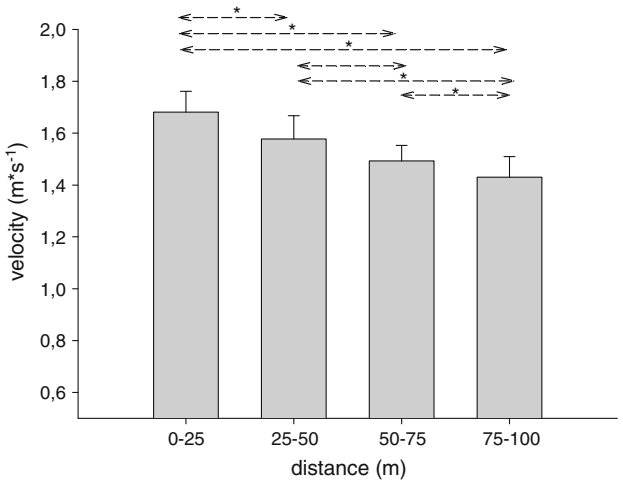

Fig. 3 The average swimming velocity in four 25-m laps of a 100-m all-out front crawl. Significant differences between laps are marked with *arrows* (*$P < 0.05$)

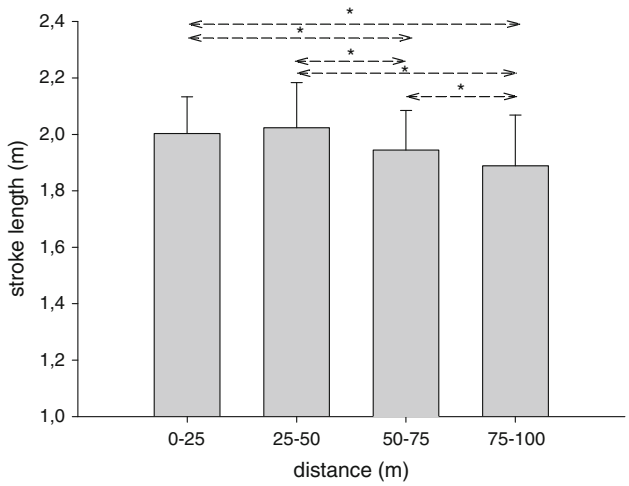

Fig. 4 The average swimming stroke length of four 25-m laps of 100-m all-out front crawl. Significant differences between laps are marked with *arrows* (*$P < 0.05$)

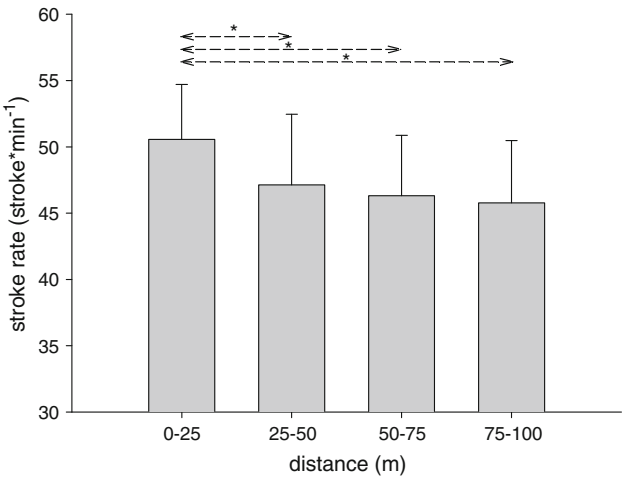

Fig. 5 The average swimming stroke rate of four 25-m laps of 100-m all-out front crawl. Significant differences between laps are marked with *arrows* (*$P < 0.05$)

The average stroke length remained unchanged for the first two laps (2.00 ± 0.13 and 2.02 ± 0.16 m, respectively) and then decreased in the third and fourth laps to 1.94 ± 0.14 and 1.89 ± 0.18 m [$F(3,30) = 8.057$, $P < 0.05$], respectively (Fig. 4).

The stroke rate decreased from the 50.56 ± 4.15 stroke min^{-1} in the first lap to the 47.12 ± 5.33 stroke min^{-1} in the second one [$F(3,30) = 15.92$, $P < 0.05$]. Further decrease in the stroke rate in the remaining two laps was not significant (Fig. 5).

Figure 6 shows the comparison of an average increase in ARV at the end with respect to the beginning of the 100-m swim. The initial and final ARV values used for calculation were estimated from the linear regression lines fitted to individual ARV values of all swimming strokes for each muscle and each swimmer separately. The ARV increased significantly at the end with respect to the beginning of swimming only in LD2 by 17.6% ($P < 0.05$) and TB by 13.5% ($P < 0.05$) muscles (Fig. 6), while in LD1 and PM2 the P values were close to significance (0.087 and 0.063, respectively).

MNF decreased linearly during swimming in all muscles of all subjects. An example is presented in Fig. 7.

The differences between the MNF_{beg} and MNF_{end} (calculated from the fitted line at the time of the first and the last stroke, respectively) were statistically significant for all muscles ($P < 0.05$, Fig. 8). The greatest change of MNF was observed in LD1 and TB muscles. MNF decreased by $24.6 \pm 8.4\%$ (from 94.9 ± 7.3 to 69.4 ± 10.2 Hz) and by $24.3 \pm 7.8\%$ (from 102.6 ± 8.1 to 77.8 ± 10.9 Hz) in LD1 and TB muscles, respectively. The least relative decrease by $20.5 \pm 9.1\%$ was observed in LD2 muscle

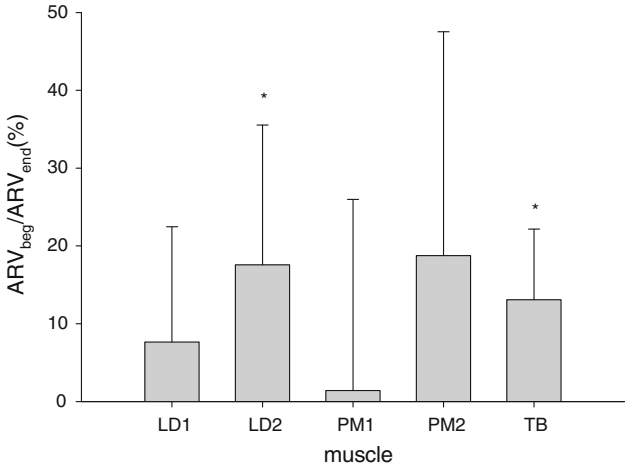

Fig. 6 The comparison between the muscles of an average increase in ARV at the end with respect to the beginning of swimming in percentages. Note high SD values. Note significant differences for LD2 and TB. $*P < 0.05$

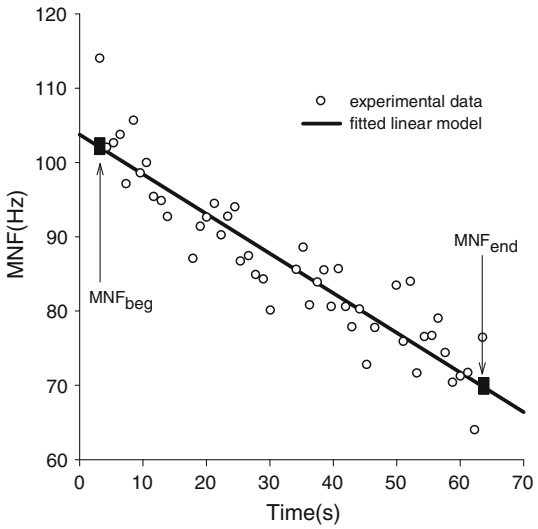

Fig. 7 MNF of muscle triceps brachii during a 100-m crawl swim. Each *symbol* presents the MNF value of one stroke within the active phase. MNF_{beg} and MNF_{end} are marked with a *black box symbol*

(from 79.7 ± 8.9 to 63.3 ± 9.4 Hz). However, the differences between the muscles in the relative decrease of MNF were not statistically significant [$F(4,36) = 0.51$, $P > 0.05$, Fig. 9].

The comparison of the average values of the slopes of the MNF decrease is shown in Fig. 10. Differences between LD2 and TB were found using Tukey's post hoc test ($P < 0.05$). In our study, we defined the active phase of each stroke based on a power envelope of the raw EMG signal. Only the part of the EMG signal contained within the active phases was further analysed. However, we also examined the duration of active phase (and the non-active phase) within the entire stroke cycle. Figure 11 shows in

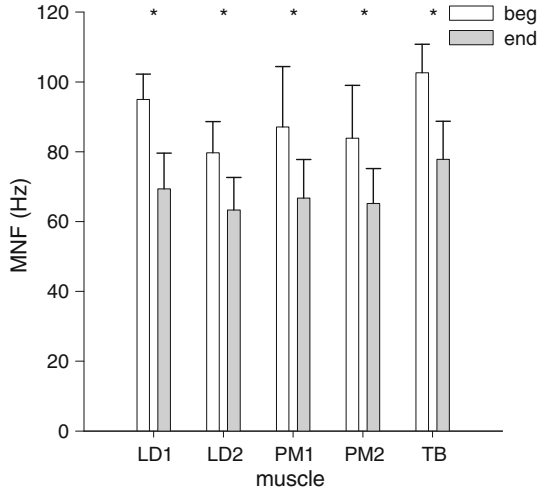

Fig. 8 The mean MNF value with SD for MNF_{beg} and MNF_{end} (*empty* and *filled bars*, respectively) shown for all muscles. *TB*, *triceps brachii*; *PM1*, *pectoralis major*, upper part; *PM2*, *pectoralis major*, lower part, *LD1*, *latissimus dorsi*, upper part; *LD2*, *latissimus dorsi*, lower part. Significant differences are marked with *asterisk*, $*P < 0.05$

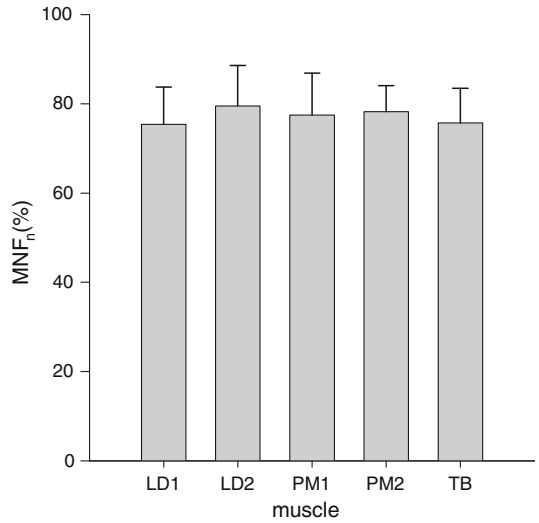

Fig. 9 Normalised MNF_n (%) at the end of swimming. Note no statistically significant differences. *TB*, *triceps brachii*; *PM1*, *pectoralis major*, upper part; *PM2*, *pectoralis major*, lower part; *LD1*, *latissimus dorsi*, upper part; *LD2*, *latissimus dorsi*, lower part

absolute time units the duration of active (left) and non-active (right) phases at the beginning (empty bars) and at the end (filled bars) of swimming for the five muscles under observation. The PM1 muscle was activated for a significantly shorter period within each stroke than other muscles [$F(2.4,23.8) = 12.729$, $P < 0.05$, Fig. 10a], and the non-active phase of PM1 was significantly longer then the non-active phase of PM2 and TB muscles [$F(4,40) = 13.825$, $P < 0.05$, Fig. 10b]. Comparison of the absolute duration of active phase at the end of

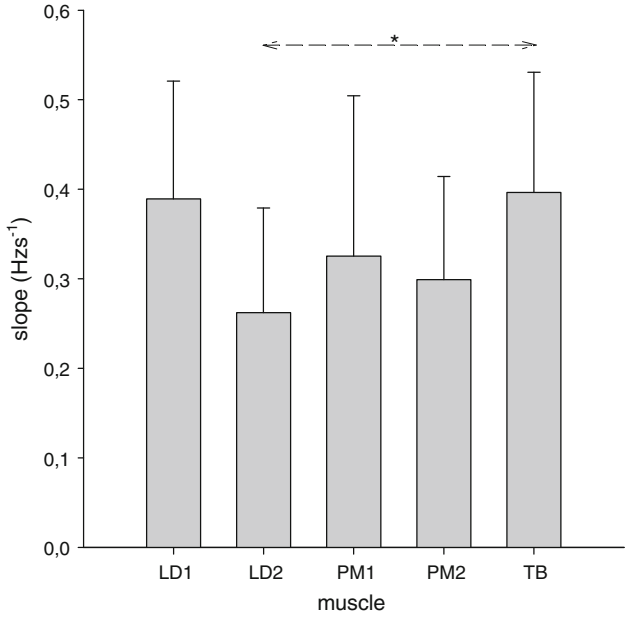

Fig. 10 Comparison of the average slopes of the MNF decrease between the muscles. Note the differences between the LD2 and TB muscle (*$P < 0.05$). *TB*, *triceps brachii*; *PM1*, *pectoralis major*, upper part; *PM2*, *pectoralis major*, lower part; *LD1*, *latissimus dorsi*, upper part; *LD2*, *latissimus dorsi*, lower part

swimming, with respect to the beginning, showed significant differences for the LD2 muscle (Fig. 11a). It increased from 386.9 ± 49.2 to 424.5 ± 72.0 ms. The TB muscle also showed a tendency of an increasing duration of the active phase, but the difference was not significant ($P = 0.11$). The absolute duration of the non-active phases of all muscles was significantly increased at the end of the swimming with respect to the beginning (Fig. 11b).

The relative duration of the active phase defined in Eq. 2 decreased significantly at the end of swimming for PM1 and PM2 muscles ($P < 0.05$), whilst no changes were observed in other analysed muscles (Fig. 12).

Discussion

In the present study, we evaluated changes in some physiological, kinematic and the EMG-derived signal parameters during 100-m front crawl all-out swimming. High blood lactate concentrations, changes in the swimming stroke characteristics, decreased swimming velocity, and changes in EMG amplitude and frequency parameters at the end of the swimming with respect to the beginning of swimming indicated the presence of muscle fatigue.

Although the swimming times achieved by the swimmers during the 100-m all-out front crawl swim test were significantly above their personal best results, it is possible to conclude that they performed at or close to their maximum effort level. This conclusion is supported by the blood lactate concentration levels after the test, which reached values that would be expected after an all-out 100-m front crawl and were comparable to the results of other studies in which similar tests or races had been performed (Bonifazi et al. 1993; Maglischo 2003; Kapus et al. 2005). The average blood lactate concentration measured 5 min after the swimming test was 14.1 ± 2.9 mmol l^{-1}. The lack of competitive dive start, the execution of the above-water turns instead of the regular underwater flip turns, the restrictions resulting from the equipment attached to the swimmers together with the swimming suit, and the lack of competitive conditions during the testing are the factors that contributed to poorer swimming time results than would be expected from the swimmers' personal best results.

The average decrease of velocity of 15% from the first to the last 25-m lap are in agreement with the results of similar studies. Seifert et al. (2007) reported 16.1, 18 and 18.9% decrease of velocity during 100-m front crawl all-out test for the high-velocity male swimmers, medium velocity male swimmers and high-velocity female swimmers, respectively, whilst Toussaint et al. (2006) reported a 12.4% decrease in clean swimming velocity.

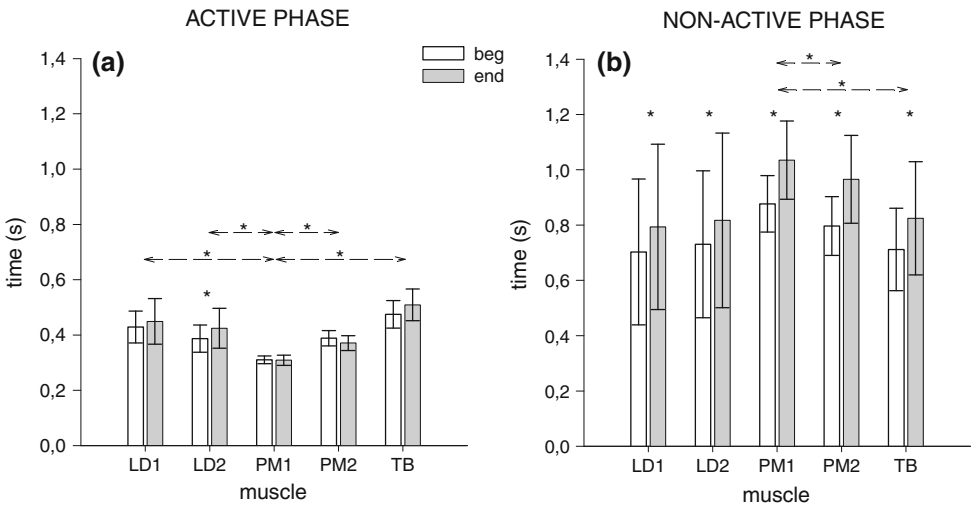

Fig. 11 Comparison of duration of active (**a**) and non-active (**b**) phases at the beginning (*white*) and end (*grey*) of swimming. Differences amongst the muscles in duration of activation (**a**) and non-activation (**b**) of the muscle considering all strokes are marked with *arrows*. Differences between the duration of the activation and non-activation at the beginning and end of swimming are marked with *asterisk* (*$P < 0.05$)

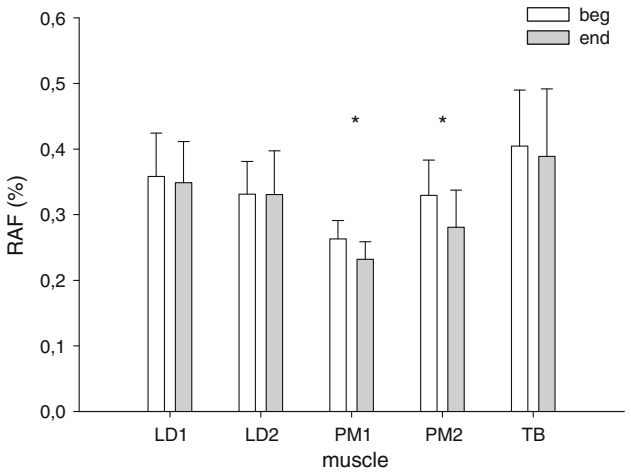

Fig. 12 Comparison of the relative duration of activation of muscles at the beginning and the end of swimming (*$P < 0.05$)

Swimmers in our study sustained a stable stroke length (SL) in the first 50 m; however, the stroke rate (SR) was reduced from the first to the second 25-m lap by 6.8% and, as a result, the velocity decreased. In the second half of the 100-m distance, SR remained unchanged but the SL was then reduced from 2.02 ± 0.16 m in the second lap to 1.89 ± 0.18 in the final lap ($P = 0.007$). As a result of combined changes in the stroke rate and stroke length, the velocity was significantly reduced in every lap. The dynamics and the amount of the decrease of SR match perfectly with the results of Toussaint et al. (2006), where SR also decreased in the second lap with respect to the first and then remained unchanged to the fourth lap, and with the study of Vorontsov and Binevsky (2003). The decreases from the first to the final lap reported were 10.6, 10.5 and 10.5% in the study of Toussaint et al. (2006), Vorontsov and Binevsky (2003) and our study, respectively. Seifert et al. (2007) found SR decreases as follows: 15.1, 11.9, 11.4 and 15.1% for high-velocity male, middle-velocity male, low-velocity male and high-velocity female swimmers, respectively. The decrease of SL has been found to be a characteristic for the middle- and low-velocity swimmers, whilst the elite swimmers could sustain stable stroke length to the end of the race (Seifert et al. 2007). However, these characteristics were obtained during free competitive swimming, whilst our subjects performed a test in specific conditions due to the research protocol demands discussed earlier in the text. It is possible that these experimental requirements demanded some extra effort (causing additional fatigue), which could account for the observed SL decrease, even though the subjects were experienced elite swimmers.

The lack of capability in maintaining a constant SL and swimming velocity in previous reported studies was attributed to the inability of generating sufficient power output needed to overcome high drag throughout the test (Toussaint et al. 2006). Namely, 13% decrease of velocity measured whilst all-out swimming 100 m with arms only on a MAD system reflected on the 25% decrease of the mechanical power output, which was measured at the same time. It was concluded by the authors that the fatigue accumulated in the arm muscles was the cause of the observed decrease in velocity and mechanical power.

In our study, EMG signals of LD, PM and TB muscles were recorded and analysed in the time and frequency domains. The average ARV calculated at the end of the all-out swim test increased with respect to the values at the beginning of the test for the LD2 and TB muscles. For LD1 and PM2, only tendencies towards such changes could be observed. The increased amplitude was most likely due to recruitment of additional motor units during swimming and/or their increased synchronisation. This implies that in spite of swimming at maximum effort level, the muscles under observation were engaged at a sub-maximum level at the beginning of the swimming. Indeed, past research shows that forces generated by the arm muscles during swimming do not present the maximum load for the muscle. The forces (one hand) measured during swimming at velocities similar to the velocities measured in our study were 66.3 N at the velocity 1.55 ms^{-1} (Hollander et al. 1988), 53.2 ± 5.8 N at 1.48 ms^{-1} (Van der Vaart et al. 1987) and 110 N at approximately 1.6 ms^{-1} (Havriluk 2004), whilst the maximal force of 133.5 ± 21.9 N was measured during tethered swimming (Aspenes et al. 2009). On the other hand, isometric arm flexion forces for highly trained swimmers lying prone on the bench were measured to be approximately between 150 and 240 N (Miyashita 1975). Approximately, double values (383 ± 89.3 N) were reported for bilateral shoulder extension in a Techogym crossover apparatus from a starting position of 170° shoulder flexion (Aspenes et al. 2009). Forces produced during front crawl can therefore be roughly estimated to be sub-maximal at about 50% of the MVC.

An increase of the EMG amplitude parameters during swimming has already been observed in the past: an increase in the integrated EMG (iEMG) of the deltoid muscle was found when swimming at a velocity of 1.3 and 1.4 ms^{-1} (Wakayoshi et al. 1994), and an increase in iEMG of a *flexor carpi ulnaris* muscle was found during a 4×100-m front crawl test at the 85% of maximum intensity (Rouard and Clarys 1995; Rouard et al. 1997). It should be noted that contrary to our study, the intensity of swimming was not maximal in the mentioned studies and therefore the stimulus to the working muscles might have been insufficient to evoke significant changes of the EMG amplitude parameters in the muscles under observation, except for the deltoid (Wakayoshi et al. 1994) and the flexor carpi ulnaris muscle (Rouard and Clarys 1995; Rouard et al. 1997).

The mean frequency of the power spectrum (MNF) at the end of swimming significantly decreased by 20.5–24.6% with respect to the value at the beginning of swimming in all muscles under observation. The decrease in MNF is generally (although not exclusively) attributed to the reduced muscular fibre conduction velocity (MFCV) (Lowery et al. 2002; Linssen et al. 1990), which is causally related to a decrease in the pH (Allen et al. 2008), Although pH was not directly measured in the present study, high values of blood lactate concentration collected after the swim implied significant pH decrease during swimming. However, other factors besides MFCV decrease such as changes in the action potential (AP) duration and afterpotential magnitude (Merletti et al. 1990; Dimitrova and Dimitrov 2003), motor unit recruitment (Gazzoni et al. 2004) and short-term motor unit synchronisation (Farina et al. 2002; Hermens et al. 1992) can contribute to the MNF decrease. For these reasons, Kallenberg and Hermens (2008) suggested that both central motor control properties (i.e. changes in recruitment) as well as peripheral muscle properties (i.e. changes in the AP shape and duration, MFCV and motor unit size) should be considered when interpreting changes in EMG amplitude and in MNF, and not solely the specific physiological mechanisms related to fatigue. Therefore, the alteration in motor control, such as an adaptation and optimisation to periodic motion as a consequence of repetitive crawl arm strokes, might have importantly contributed to the MNF decrease. In other words, it is possible that during high-intensity exercise, such as 100-m all-out crawl, the processes of adaptation in motor control and of fatigue occur in parallel. Further on, it is not likely that the muscles under observation would fatigue to the same extent during swimming. Equal levels of fatigue in all muscles would be unexpected for two reasons. First, significant differences in the duration of the activation amongst the muscles were found. For example, the TB and PM1 muscles were activated for the relatively longest and shortest periods of time, respectively, within one arm cycle (Fig. 12), and therefore a greater level of fatigue could be expected at the end of swimming for the TB than for the PM1 muscle. The second reason was that the greatest fatigue during maximal front crawl was expected for the LD and TB muscles due to their dominant roles in the crawl arm stroke. Contraction of LD produces internal rotation, extension and adduction of the shoulder joint, which is a description of nearly the entire crawl arm stroke and which explains why the *latissimus dorsi* (LD) has been labelled as "the swimming muscle" (Behnke 2001). The TB muscle is activated during downsweep and insweep enabling arm stabilisation when co-contracting with biceps, and to the greatest extent during the elbow extension, which is executed during the upsweep. The upsweep is the part of the stroke where the maximal propulsive forces (Schleihauf 1979; Rouard et al. 1996) and the greatest forward velocities of the swimmers are measured (Maglischo 2003). Girold et al. (2006) reported the greatest improvement in strength for elbow extensors (basically for TB) as a result of resisted swimming training, which also supports the importance of TB during crawl.

Much lower decrease of MNF of 11.41 and 8.55% for the *extensor carpi ulnaris* and *flexor carpi ulnaris*, respectively, was reported after the 4 × 50-m front crawl (Caty et al. 2006). The reason for the smaller decline of MNF may be that unlike the muscles chosen in our study, these two muscles are not propulsive and their work of wrist stabilisation during swimming could be less fatiguing than the work of the muscles that are responsible for propelling the body.

The cause of the different rate of decrease in MNF between the LD2 and TB muscles might be the differences in muscle morphology. Changes in MNF occur more rapidly in muscles composed of a high proportion of fast twitch muscle fibres than in muscles composed of a high proportion of slow twitch fibres (Komi and Tesch 1979; Larsson et al. 2003). The structure of motor units (muscle fibres) influences the amount of decrease in the muscular fibre conduction velocity (MFCV) and consequently the decrease in MNF. Slow motor units activate muscle fibres with lower MFCV, and fast motor units activate fibres with higher MFCV. Consequently, the decrease of MFCV is related to the proportion of FT muscle fibres. The TB muscle is composed of 65–75% of fast twitch fibres (Larsson et al. 2003), which is the highest proportion in comparison to other analysed muscles.

By analysing the rectified EMG signal, it is possible to observe very clearly the differences between the activation and resting periods of the muscles as shown in Fig. 1. The observed muscles were activated during the underwater part of the stroke and almost totally relaxed during the recovery, which provided a period of reduced effort for these muscles. The fatigue accumulated in the muscles might be the cause for changes in muscle coordination.

The average absolute duration of the activity of the LD2 muscle increased at the end of the swim, whilst for the TB muscle only tendency towards such change was observed; however, it was not found to be significant ($P > 0.05$). This is in agreement with previous studies, which showed that hand velocity decreased during the propulsive part of the arm stroke probably due to fatigue and, therefore, the time needed to complete this part of the stroke was prolonged (Seifert et al. 2007). The average absolute duration of the non-active phase significantly increased in all observed muscles as shown in Fig. 11b. This clearly shows the mechanism that the muscles used to adapt to the fatigue: by extending the time of rest. Consequently, the time to complete one cycle (stroke) was extended and manifested

in the decreased stroke rate (Fig. 5). The decreased stroke rate was therefore mostly due to the extension of the non-active (rest) phase, since the duration of the active phase did not change significantly in all muscles except the LD2. Because the durations of the active and non-active phases prolonged to approximately the same extent, the relative durations of the active phases did not change for LD1, LD2 and TB. For the PM1 and PM2, the duration of the active phase did not change; however, the non-active phase was prolonged as well as for the other muscles and, as a result, the relative duration of the active phase for these two muscles decreased. It is possible that as the duration of the active phase of LD2 (significantly) and TB (only tendency towards the change, $P = 0.11$) prolonged inside one cycle, it presented the prolongation of non-activation time for PM1 and PM2. Other authors (Seifert et al. 2004, 2007; Seifert and Chollet 2008; Alberty et al. 2008) used the kinematics to obtain data on arm coordination, so our results are not directly comparable to theirs. However, some common points can be outlined. Seifert et al. (2007) obtained similar results for the high-velocity male swimmers: the relative duration of the propulsive phase of the stroke was longer in the first lap with respect to the last lap, and the opposite results (the relative duration of the propulsive phase of the stroke was shorter) for the middle- and low-velocity swimmers. The increase of the relative propulsive (push and pull) phase occurring during fatiguing swim was also reported by Alberty et al. (2008, 2005). It is possible that to maintain a stable stroke length in spite of accumulating fatigue, the swimmers extended the gliding phase of the stroke, which led to extension of the non-active phase for the muscles and possibly also induced some changes in the arm coordination.

Our results showed that the muscles needed to maintain an absolute duration of activation to complete their task (propulsion), but they also had to extend the resting period due to accumulating fatigue. Perhaps, maintaining constant duration of the resting period of the individual muscle within one arm stroke would enable the swimmers to maintain the initial SL and, therefore, initial swimming velocity. However, this obviously was not possible due to fatigue accumulated in the muscles.

Conclusion

Changes in velocity and stroke parameters as well as high lactate accumulation confirmed the presence of muscular fatigue after the 100-m all-out crawl. In addition, the changes of amplitude and frequency EMG parameters (ARV and MNF) were in agreement with fatigue appearance. However, EMG parameters did not indicate significant differences amongst analysed muscles after swimming, because changes in EMG signal amplitude and MNF could not be attributed solely to fatigue process, since EMG parameters can also be affected by alterations in motor control. Intramuscular coordination was affected as well, but for more detailed analysis it should be further investigated combining EMG signals with the underwater kinematic recordings.

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