INTRODUCTION

High intensity interval training is often used in sports practice. It can be defined as repeated exercise bouts performed at maximal or near maximal intensity, separated by brief periods of inactivity. The rest periods between exercises give the possibility to maintain a high work rate for a longer period of time [19]. Despite delay, fatigue also occurs, and there are various suggestions for its cause. During high intensity exercise, ATP is constantly resynthesized in both aerobic and anaerobic processes [20]. The anaerobic energy release, depending on lactate (LA) production, may be limited by hydrogen ion ($H^+$) accumulation in the working muscles [24]. Contraction and acidosis are associated with activation of potassium ($K^+$) channels and thus $K^+$ efflux from the muscles [13]. Accumulation of potassium in the muscle interstitium has also been suggested to be implicated in the fatigue process due to impaired membrane excitability [26].

Interval exercise protocols have been extensively studied in various, fixed work-to-rest ratios (from 5:1 to 1:11), and recommended for developing aerobic and anaerobic adaptations [3,18,31]. Therefore, this type of training is very popular in swimming practice for improving swimming velocity ($v$) [5,17]. The highest $v$ is achieved by combining stroke rate (SR) and stroke length (SL) [1]. Moreover, it has been shown that reduction in swimming speed (as a result of decrease in power output) is linearly related to the decrease in SR [29].

We hypothesised that swimming 8 x 25 m with short rest periods between efforts would easily provoke fatigue in collegiate swimmers, and therefore modify swimming kinematic, blood acid-base status and $K^+$ levels. The aim of the study was to examine $v$, SR and SL in that type of repeated swimming test and its relation to the changes in blood parameters.

MATERIALS AND METHODS

Subjects. Seven collegiate swimmers (4 women, 3 men), with at least 6 years of training experience, volunteered to participate in the study (age 21.4 ± 2.6 years, height 175 ± 9.5 cm, weight 69.4 ± 11.7 kg). Participants presented a similar level of sport proficiency; their swimming results achieved during inter-college meets were 300 ± 40 FINA points on average. The subjects were
asked to refrain from any physical activity or alcohol consumption for at least 24 h prior to testing. The study was approved by the Ethics Committee at the Medical University of Gdansk and all the subjects gave their informed consent before beginning the study.

Study procedure
All subjects were familiarized with the exercise protocol at least a week before starting the experiments. On the day of the experiment, the subjects had a standard lunch and were asked not to consume any other food until the end of the test. Four hours after lunch the exercise protocol was started. The warm-up consisted of mixed swimming drills: 300 m front crawl, 200 m as 50-m pull/swim, 100 backstroke and 2 x 25 front crawl with increasing speed. The test consisted of 8 x 25 m front crawl repetitions with maximum effort [28]. The rest period between repetitions was set to 5 seconds, as previously used by Siegler and Gleadall-Siddall [27]. We regarded its protocol as valid for the purpose of our study.

Blood analysis
At rest, after the warm-up, and then 3 minutes after completing the test, blood samples were taken from the swimmers’ fingertips into heparinized capillary tubes (Siemens Healthcare Diagnostics Inc.) and immediately analysed for blood gases, pH, haemoglobin (Hb), and electrolytes using a Rapidpoint 400/405 (Siemens Healthcare Diagnostics Inc.). In addition, bicarbonate (HCO₃⁻) was calculated from CO₂ and pH values according to the Henderson-Hasselbalch equation [11], and base excess (BE) was calculated according to the following equation:

\[
BE = (1 - 0.014 \times [Hb]) \times (HCO_3^-) - 24.8 + (1.43 \times [Hb] + 7.7) (pH - 7.4).
\]

Blood samples for LA determination were immediately deproteinized by adding ice cold 0.4 M perchloric acid. LA was determined using a standard test kit (Randox Laboratories Ltd.) and measurements were estimated spectrophotometrically using Super Aquarius CE9200 (Cecil Instruments Ltd.).

Swimming technique analysis
All swims were recorded using a Sony 8-mm Hi-8 (25 Hz) video camera for later analysis of time and technique. Time measurement was done using Adobe Premiere Pro v7.0 technology (Adobe Systems Incorporated, USA). The “pause” function was used to mark the sequence of video movie where the time had to be measured. The time of the marked sequence was measured automatically by Adobe software. Time measurement of each 25 m bout began when the swimmer’s feet came off the edge of the swimming pool. Measurement was stopped when the swimmer touched the opposite edge of the pool. The time of the “pure swimming zone”, i.e. between the 5th and 20th meter of the pool, was measured in the same manner, as well as the time of 3 swimming cycles and rest breaks.

SR, SL and stroke index (SI) were calculated using the following equations:

\[
SR = 60 \times 3 / tSR; \text{ where } tSR \text{ is the time of 3 cycles}
\]

\[
v = S / t; \text{ where } S \text{ is distance, } t \text{ is time}
\]

\[
SL = v \times 60 / SR
\]

\[
SI = v \times SL
\]

according to the method described previously [7].

Fatigue index
The fatigue index was calculated as the percentage difference between the velocity in the first and in the last swim repetition using the following equation [12]:

\[
FI = \min. v \times 100% / \max. v
\]

Swimming velocity threshold
The relationships between v and the swimming repetition for all subjects were analysed individually, by plotting log(v) vs. log(repetition) in the reverse order. The transition point was assumed to be the swimming velocity threshold (SVT).

Statistics
Statistical calculations were performed with Statistica 10 (StatSoft Inc., Tulsa, USA). Distributions of blood acid-base parameters and kinematic parameters were normal according to the Shapiro-Wilk test. Analysis of variance (ANOVA) with Fisher LSD post-hoc test was used to determine the statistical differences in the blood data and kinematic parameters. The Pearson product moment correlation coefficient was used to assess the relations between biochemical and kinematic variables. The level of statistical significance was established at p<0.05.

RESULTS
Muscular metabolism during the 8 x 25m front crawl test, performed with maximum effort, caused metabolic acidosis associated with changes in blood acid-base parameters, as well as an increase in blood LA concentration from 1.6 ± 0.1 to 7.2 ± 0.5 mmol·l⁻¹ (Table 1). Furthermore, three minutes after completion of the exercise, a decrease in blood K⁺ was observed (Table 1).

Average v during the test was 1.13 ± 0.08 m·s⁻¹, with the highest value during the first repetition: 1.32 ± 0.08 m·s⁻¹ (Table 2). Based on the decrease in swim velocity FI was calculated: 77.6 ± 1.8%.

**TABLE 1. BLOOD ACID-BASE PARAMETERS (pH, BE, HCO₃⁻), LACTATE AND POTASSIUM CONCENTRATIONS AT REST, BEFORE AND AFTER COMPLETION OF THE TEST**

<table>
<thead>
<tr>
<th></th>
<th>Rest</th>
<th>Before the test</th>
<th>After the test</th>
</tr>
</thead>
<tbody>
<tr>
<td>LA (mmol·l⁻¹)</td>
<td>1.6 ± 0.1</td>
<td>2.4 ± 0.1</td>
<td>7.2 ± 0.5 **</td>
</tr>
<tr>
<td>pH</td>
<td>7.42 ± 0.01</td>
<td>7.37 ± 0.02</td>
<td>7.23 ± 0.01 **</td>
</tr>
<tr>
<td>BE (meq·l⁻¹)</td>
<td>-1.2 ± 0.8</td>
<td>-3.9 ± 0.9</td>
<td>-12.9 ± 0.7 **</td>
</tr>
<tr>
<td>HCO₃⁻ (mmol·l⁻¹)</td>
<td>22.7 ± 0.9</td>
<td>20.7 ± 0.8</td>
<td>13.5 ± 0.7 **</td>
</tr>
<tr>
<td>K⁺ (mmol·l⁻¹)</td>
<td>4.4 ± 0.1</td>
<td>4.3 ± 0.1</td>
<td>4.0 ± 0.1 **</td>
</tr>
</tbody>
</table>

Note: values are means ± sem, ** P<0.005 as compared to rest values.
Kinematic variables and blood acid-base status in the analysis of collegiate swimmers’ anaerobic capacity

**TABLE 2. SWIMMING VELOCITY \((v)\), STROKE RATE (SR), STROKE LENGTH (SL), STROKE INDEX (SI) OF THE FIRST, THE LAST, AND ALL REPETITIONS**

<table>
<thead>
<tr>
<th></th>
<th>First repetition</th>
<th>Last repetition</th>
<th>Average of 8 repetitions</th>
</tr>
</thead>
<tbody>
<tr>
<td>(v) (m·s(^{-1}))</td>
<td>1.32 ± 0.08</td>
<td>1.05 ± 0.08</td>
<td>1.13 ± 0.08</td>
</tr>
<tr>
<td>SR (Hz)</td>
<td>0.66 ± 0.03</td>
<td>0.59 ± 0.03</td>
<td>0.61 ± 0.03</td>
</tr>
<tr>
<td>SL (m)</td>
<td>2.00 ± 0.12</td>
<td>1.77 ± 0.10</td>
<td>1.85 ± 0.12</td>
</tr>
<tr>
<td>SI (m·m·s(^{-1}))</td>
<td>2.67 ± 0.27</td>
<td>1.88 ± 0.21</td>
<td>2.13 ± 0.25</td>
</tr>
</tbody>
</table>

Note: values are means ± sem

The swimming velocity curve can be divided into a rapid decrease of velocity and relatively stable velocities. Analysis of the \(v\) curve, using log values, allow us to establish the SVT in all subjects individually (Fig. 1).

**FIG. 1. SWIMMING VELOCITY IN SEPARATE REPETITIONS FOR REPRESENTATIVE SUBJECT. PLOTTING LOG(\(v\)) VS. LOG(REPETITION) IN THE REVERSE ORDER INDICATED THE TRANSITION POINT, ASSUMED AS SWIMMING VELOCITY THRESHOLD (SVT)**

We found high correlations between the decrease of blood acid-base status parameters induced by exercise (pH, BE, HCO\(_3\)\(^{-}\)) and SVT, but not FI (Table 3).

**TABLE 3. CORRELATION COEFFICIENTS BETWEEN FATIGUE INDEX (FI), SWIMMING VELOCITY THRESHOLD (SVT) AND BLOOD PARAMETERS DETERMINED AFTER THE TEST AND EXPRESSED AS DIFFERENCES BETWEEN REST AND POST-EXERCISE VALUES**

<table>
<thead>
<tr>
<th></th>
<th>FI</th>
<th>SVT</th>
</tr>
</thead>
<tbody>
<tr>
<td>LA after the test</td>
<td>0.07</td>
<td>0.53</td>
</tr>
<tr>
<td>∆ LA (after the test – rest)</td>
<td>0.26</td>
<td>0.56</td>
</tr>
<tr>
<td>pH after the test</td>
<td>0.09</td>
<td>-0.54</td>
</tr>
<tr>
<td>∆ pH (after the test – rest)</td>
<td>-0.22</td>
<td>0.82 *</td>
</tr>
<tr>
<td>BE after the test</td>
<td>0.58</td>
<td>-0.27</td>
</tr>
<tr>
<td>∆ BE (after the test – rest)</td>
<td>-0.15</td>
<td>0.87 *</td>
</tr>
<tr>
<td>HCO(_3) after the test</td>
<td>0.69</td>
<td>-0.09</td>
</tr>
<tr>
<td>∆ HCO(_3) (after the test – rest)</td>
<td>-0.11</td>
<td>0.76 *</td>
</tr>
<tr>
<td>K(^+) after the test</td>
<td>0.37</td>
<td>0.23</td>
</tr>
<tr>
<td>∆ K(^+) (after the test – rest)</td>
<td>-0.32</td>
<td>-0.29</td>
</tr>
</tbody>
</table>

Note: * Presented correlations are statistically significant \((P<0.05)\)

A continuous decrease of SR and SL was also observed in our study (Fig. 2). The major difference in SR occurred in the second bout of the test \((4.4 ± 1.3\%)\), whereas SL decreased notably in the third bout of the test \((5.3 ± 1.4\%)\). In addition, in the last repetition a slight increase of SR and SL in comparison to the preceding bout was observed \((0.8 ± 0.9\% \text{ and } 0.7 ± 0.9\% \text{ respectively})\).

**FIG. 2. AVERAGE VALUES OF STROKE RATE (SR) AND STROKE LENGTH (SL) EXPRESSED AS PERCENTAGE OF THE FIRST REPETITION (± SEM)**

Note: * \(P<0.05\) as compared to the first repetition; ** \(P<0.005\) as compared to the first repetition

There was no correlation between mean \(v\), SR, SL, SI and blood parameters (data not shown).

**DISCUSSION**

This study provides additional information on kinematic and physiological data of collegiate swimmers who performed eight 25 m repetitions of a maximum effort with a 5-second rest between. It has been previously observed that the rest period between work-outs increases the total time of exercise performed at the same intensity [19]. However, when the rest period is not long enough for recovery, the relaxation time is longer and a reduction in force generation occurs [6]. Several mechanisms have been proposed as the cause of the fatigue [2], and the most likely is accumulation of muscle metabolites [32]. Muscle contractions are associated with anaerobic glycolysis, which leads to increased H\(^+\) production and a measurable decrease of intra- and extracellular pH [24]. The H\(^+\) accumulation may cause inhibition of the sarcoplasmic reticulum (SR) Ca\(^{2+}\) pump, and lower transport of Ca\(^{2+}\) back into the SR. Muscle relaxation therefore slows down, and the muscle contraction force is reduced [16].

In our study, we observed that front crawl swimming 8 x 25 m with maximum effort induced fatigue: about 20% decrease in \(v\), SR and SL. These changes were associated with increased LA and modifications in blood acid-base status. However, the direct correlation between these parameters was not statistically significant. This observation is in agreement with the previous study comparing different protocols of high-intensity intermittent exercises with different pause durations \((30, 60, \text{ and } 120 \text{ seconds})\) between efforts for a total of 15 sprints (running at maximum speed) [4].
As the 15 sprints progressed, a significant decrease in velocity was observed in the protocol with pauses of 30 seconds. In contrast, in the protocol with pauses of 120 seconds, maintenance of starting velocity was noted. However, both protocols presented a similar increase in blood LA concentrations. The authors suggested that LA production was not the explanation of the fatigue [4].

The analysis of intermittent swimming velocities in our study indicated that the curve can be divided into a rapid decrease in swimming velocities and swimming with relatively stable velocities. The breaking point, estimated as SVT in our study, highly correlates with the decrease of blood acid-base parameters. This may indicate an individual tolerance for muscle acidosis induced by exercise: higher tolerance for H\(^+\) accumulation, more 25 m repetitions until reaching SVT. As has previously been shown, in subjects performing ten 6-second maximal cycling sprints with a 30-second recovery, the inhibition of anaerobic glycolysis occurred before the end of the exercise [10]. In the study by Parolin et al. [23], the subjects exercised three times for 30 seconds (Wingate tests) separated by a 4-minute recovery, and during the last sprint the energy provision was mostly from oxidative phosphorylation. The authors also observed that increased concentration of H\(^+\) may completely inhibit glycogen phosphorylase and thus glycogenolytic flux, and maintained the high level of pyruvate dehydrogenase activity (the key enzyme of pyruvate oxidative metabolism). Therefore, this mechanism blocks further increase in H\(^+\) concentration. Despite the fact that the exercise protocol was different in our study, the obtained results confirm previous data. The repetitions following SVT are performed with relatively stable velocities, suggesting that energy for muscle contractions is produced mostly with aerobic metabolism.

Among the wide range of potential candidates involved in the fatigue processes, sarcolemmal depolarization due to extracellular K\(^+\) accumulation has been suggested to be of primary importance for fatigue development during high-intensity exercise [26]. This hypothesis is based on observations that, during high-intensity exercise, the contracting skeletal muscles release K\(^+\), causing elevation in venous plasma K\(^+\) concentration [14]. Accumulation of K\(^+\) in the extracellular space leads to depolarization of the membrane potential [8]. During recovery the kinetics of K\(^+\) reuptake by the muscle were described by a very fast (less than 1 min) and a slow component (more than 1 min) [14]. Moreover, recent studies indicate that training induces increase in muscle Na\(^+\)–K\(^+\) pump content [21]. Since in our study we measured K\(^+\) in the blood 3 minutes after completion of the exercise, we were unable to observe any relation between K\(^+\) and fatigue induced by exercise.

The kinematic values in our protocol were slightly lower than those observed in the previous study [15]. This difference may be due to the participants’ characteristics. Kapus et al. [15] studied only male subjects, whereas the group in our study was heterogeneous. Despite this point, as well as the fact that collegiate swimmers performed the exercise with short rest periods, the swimming velocities in our study decreased in a similar manner as during the 200 m test performed by recreational swimmers [15]. Moreover, in our study the decrease of SR occurred already in the second bout of the test, similarly to the previous results [9,15,29]. Touissant et al. [29] revealed a decrease in the velocity and SR already in the second repetition of 100 m arms-only crawl in a group of experienced male swimmers, while Chollet et al. [9] found a decrease of 6.5% in SR from the first to the second 50 m during 100 m front crawl with maximal intensity. Vorontsov and Binesevsky [30] noted even a 10.5% decrease in SR between the first and the last 25 m lap of 100 m front crawl distance.

Fluctuation in stroke length is different from that of stroke rate. According to some authors, in maximal intensity 100 m crawl swimming, a slight decrease in stroke length (ca. 2%) can be observed [9,22]. In contrast to these results, Seifert at al. [25] reported a 3.7% increase in SL. Diversity in the results for stroke length may be an effect of the swimmers’ strategy. Athletes who reached maximal velocity in the initial stage of the test usually decreased their stroke rate and stroke length in the final part of 100 m distance [9,22].

CONCLUSIONS

The analysis of swimmers’ kinematic variables together with blood parameters seems to be very informative in coaching practice. In the current study, the determination of swimming velocity threshold revealed the individual level of anaerobic capacity. The rest periods following this threshold should have been elongated, which would give the opportunity to improve recovery and delay fatigue. Individual training loads applied to the swimmers, especially at the non-competitive level, accelerate the training adaptation in order to achieve a higher performance level.

Acknowledgments

This work was supported by grant No. N RSA1 002851 from the Polish Ministry of Science and Higher Education.

Conflict of interest

Authors declare that there is no conflict of interest.

REFERENCES

Kinematic variables and blood acid-base status in the analysis of collegiate swimmers’ anaerobic capacity