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THE INFLUENCE OF REDUCED BREATHING DURING SWIMMING ON SOME RESPIRATORY AND METABOLIC VALUES IN BLOOD

VPLIVI ZMANJŠANE FREKVENCE DIHANJA MED PLAVANJEM NA DIHALNE IN METABOLNE KAZALCE V KRVI

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Abstract

The purpose of the present study was to ascertain the influence of reduced pulmonary ventilation on blood acid-base balance during swimming. Five trained swimmers (age 21±2 years, height 187±5 cm and weight 83±6 kg) volunteered to participate in this study. They had to swim 400 m front crawl at velocity V_{OBLA} two times. Firstly, they were taking breath every two strokes (B2). During the second trial they swam the same distance at a similar velocity, however with reduced breathing frequency, taking breath every four strokes (B4). Measures included lactate concentration ([LA]) and parameters of blood acid-base status (pH, Po_2, Pco_2) before and during the third minute after the exercise. Only Pco_2 significantly increased after B4 than after B2 ($p<0.05$). After the exercise other parameters ([LA], pH, HCO_3 and Po_2) did not change significantly in response to reduced breathing frequency during swimming. It may be concluded that the reduced breathing frequency during front crawl swimming at V_{OBLA} velocity did not cause hypoxia nor increased [LA]. However, it increased the Pco_2 to the range of hypercapnia.

Keywords: swimming, reduced breathing frequency, blood acid base status, blood lactate

Izvleček

Namen raziskave je bil ugotoviti učinke zmanjšane frekvence dihanja na kazalce acidobaznega statusa krvi med plavanjem s hitrostjo, ki jo določa kriterij Onset of Blood Lactat Accumulation (V_{OBLA}). V raziskavo je bilo vključenih pet zdravih plavalcev, starih od 18 do 23 let (višina 187 ± 6 cm in teža 83 kg ± 9 kg), ki so odplavali 400 m kravl z dihanjem na vsak drugi zavesljaj (D2) in 400 m kravl z dihanjem na vsak četrti zavesljaj (D4). Pri tem smo merili: vsebnost laktata ([LA]) in kazalce acido-baznega statusa v krvi (pH, Po_2, Pco_2). Vzorce smo jemali pred naporom in v tretji minuti po naporu. Le vrednosti Pco_2 po D4 so značilno višje od vrednosti Pco_2 po D2 ($p<0.05$). Preostale vrednosti drugih kazalcev acido-baznega statusa po D4 so podobne vrednostim po D2. Na osnovi teh rezultatov smo zaključili, da zmanjšana frekvenca dihanja med plavanjem pri hitrosti V_{OBLA} ne vpliva na znižanje Po_2 ali povečanje [LA], temveč zviša Pco_2 v področje hiperkapnije.

Ključne besede: plavanje, zmanjšana frekvenca dihanja, acido-bazni status krvi, krvni laktat

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INTRODUCTION

Pulmonary ventilation is limited by the swimming technique. Breathing frequency has to be in accordance with the stroke frequency. It may be assumed that the front crawl swimming velocity should also be regulated in a way, which ensures maintaining needs of increased pulmonary ventilation. If the breathing pattern is changed at a similar velocity, this may dramatically influence blood oxygenation and acid-base status and the swimmer's performance. Hypoxia and/or hypercapnia with additional respiratory acidosis may hypothetically be effects of reduced pulmonary ventilation. During front crawl swimming with restricted breathing (a breath every four, six or eight strokes), the swimming technique may not permit an adequate increase of pulmonary ventilation to match it's needs. It may have a significant influence on the swimmer's exertion (Dicker, Lofthus, Thorton, & Brooks, 1980; Town, & Vanness, 1990).

Swimmers, throughout their training learn to maintain the highest swimming velocity (»critical« velocity) where ventilation generally still matches its needs. However, it may be expected that swimming velocity may exceed this »critical« velocity for a while, especially during shorter swimming distances. On the other hand, the similar phenomenon may be caused during swimming with reduced breathing frequency or during swimming of certain distances with breath holding. This training technique is often referred to as »hypoxic training« (Maglischo, 1990). It has been thought that reduced breathing frequency can be used to induce arterial hypoxemia, and to enhance lactate production in working muscles (Counsilman, 1977). It is believed that reduced breathing frequency during swimming permits swimmers to derive both »aerobic« and »anaerobic« conditioning from exercise of sub maximal intensity (Dicker et al., 1980).

Some previous studies focused on reduced breathing frequency during swimming, running and cycling. Swimmers restricted the breathing frequency (a breath every four, six and eight strokes) during tethered flume swimming (Dicker et al., 1980; Town et al., 1990) and during interval training (Holmer, & Gulstrand, 1980). Studies on the bicycle ergometer used cycling bouts at different intensities and different breathing patterns: normal breathing, breathing every 4 s, breathing every 8 s and reduced frequency as low as possible. Subjects held their breath at functional residual capacity (Yamamoto, Mutoh, Kobayashi, & Miyashita, 1987; Yamamoto, Takei, Mutoh, & Miyashita, 1988) and at total lung capacity (Lee, Cordain, Sockler, & Tucker, 1990). Runners performed intervals at 125% Vo2max with breath holding (Matheson, & Mckenzie, 1988).

Although reduced breathing frequency during swimming was associated with reduced ventilation (VE), increased alveolar partial pressure of CO_2 (P_Aco_2) and in decreased alveolar partial pressure of O_2 (P_Ao_2) (Town et al., 1990), compensatory responses resulted in increased tidal volume (VT). But this reduction of P_Ao_2 was insufficient to cause a marked reduction in arterial oxygen saturation (Dicker et al., 1980). The arterial oxygen desaturation during exercise with reduced breathing frequency appears to depend on lung volume at which breath holding occurs. Alveolar hypoxia was accentuated more during breath holding at functional residual capacity than at total lung capacity (Yamamoto et al., 1987; Lee et al., 1990). Reduced breathing frequency during exercise increased lactate concentration (Lee et al., 1990) or did not influence it (Yamamoto et al., 1987; Matheson et al., 1988; Town et al., 1990). Holmer et al. (1980) noted a significantly lower Vo_2 during swimming with reduced breathing frequency and a slight decrease in blood lactate concentration. This seemed contradictory because reduced oxidative metabolism should be compensated for by an increased anaerobic yield. Reduced breathing frequency during swimming could also impede stroke rate. Especially when the need to breathe becomes critical, swimmers have to increase their stroke rate, which helps to increase ventilation (Town et al., 1990).

It has been assumed that maximal lactate steady state occurs during swimming at constant velocity, which corresponded to Onset of blood lactate accumulation (OBLA) (Sjodin, Schele, Karlsson, Linarsson, & Care, 1982). Therefore the volume of CO_2, which comes from the bicarbonate buffer system, should be also constant together with additional volume of CO_2 from the aerobic metabolism. Oxygen delivery to muscles seems not to be limited due to constant Po_2. If pulmonary ventilation is reduced substantially during this velocity, then it may be expected that lower volume of CO_2 will be expired by the lungs, which may cause hypercapnia and respiratory acidosis. According to data of Yamamoto et al. (1986) an influence on increased Pco_2 and no effect on [LA] may be expected. Unfortunately, these data were not obtained at velocity V_{OBLA} and/or during swimming. It may be

hypothesised that V_{OBLA} is the lowest velocity, which can be swum for a longer distance (400 m) also with restricted breathing. This fact is necessary for studying phenomena related to restricted breathing. The purpose of the present study was therefore to ascertain the influence of reduced breathing frequency on the blood acid-base status during swimming at Onset of Blood Lactate Accumulation velocity (V_{OBLA}).

METHODS

Subjects

Five trained swimmers (age 22 ± 2 years, height 184 ± 8 cm and weight 80 ± 6 kg) volunteered to participate in this study.

Procedures

Swimmers swam a 5 × 200 m front crawl progressive step test. The swimming velocity of the first step was 1.09 m/s. From step to step the swimming velocity was increased by 0.1 m/s. Breaks between each exercise step were 3 minutes.

After the progressive step test swimmers had to swim 400 m front crawl at V_{OBLA} twice: first, by taking breath every two strokes (B2) and the second, by taking breath every four strokes (B4). Velocity and stroke rate of 400 m front craw with B4 were defined with 400 m front crawl with B2, since we knew that swimmers reduced swimming velocity and/or increased stroke rate, when the need to breathe become critical during swimming with reduced breathing frequency (Town et al., 1990).

Blood collection and breathing measurements

During the break between each exercise in the front crawl progressive step test a blood sample (10 µl) is taken in order to determine the lactate concentration ([LA]). [LA] was analysed with the use of MINI8 (dr. LANGE, Germany) photometer.

Measures before and after 400 m front crawl with different breathing frequency included parameters of blood acid-base status (pH, Po_2, Pco_2, $[HCO_3^-]$) and [LA]. Capillary blood samples (60 – 80 µl) were collected in the third minute after each swim from a hyperemied ear lobe for pH, Po_2, Pco_2, $[HCO_3^-]$ analysis using an ABL5 (Radiometer Copenhagen) instrument. Stroke rate, inspiratory time (T_I), expiratory time (T_E), number of breaths per 25 meters (Ni) and breathing frequency during swimming (BF) were obtained by analysing film shots.

Data processing and analysis

OBLA (Onset of Blood Lactate Accumulation) is defined by [LA] 4 mmol/l (Maglischo, 1990) and it is one of the possible criteria for anaerobic threshold (Ušaj, & Starc, 1990). It was evaluated on the basis of lactate concentration in dependence to swimming velocity curves.

Statistics

The values were presented as means ± standard deviations (SD). The paired t-test was used to compare the data between swimming in two different conditions. All statistical parameters were calculated using the graphical statistics package Sigma Plot (Jandel, Germany).

RESULTS

Table 1: OBLA velocity, measured B2 and B4 velocity and measured B2 and B4 stroke frequency.

SUBJECT	V_{OBLA} (m/s)	V_{B2} (m/s)	SF_{B2} (min^{-1})	V_{B4} (m/s)	SF_{B4} (min^{-1})
1.	1.21	1.19	29	1.20	29
2.	1.46	1.44	31	1.44	31
3.	1.20	1.21	24	1.21	24
4.	1.34	1.33	31	1.33	32
5.	1.43	1.43	32	1.43	31
MV	1.33	1.32	/	1.32	/
SD	0.12	0.12	/	0.11	/

Legend:
V_{OBLA} – OBLA velocity;
V_{B2} – measured B2 velocity
SF_{B2} – measured B2 stroke frequency
V_{B4} – measured B4 velocity
SF_{B4} – measured B4 stroke frequency
MV – mean values;
SD – standard deviation

As expected, swimming speeds and stroke frequencies did not change between two different breathing conditions (Table 1). Only one subject could not finish the 400 m front crawl taking breath every four strokes. Because of fatigue he could swim only 300 m.

Table 2: Respiratory parameters of swimming with two different breathing conditions (B2 and B4).

	B2		B4	
	MV	SD	MV	SD
T_I (s)	0.72	0.1	0.72	0.1
T_E (s)	1.29	0.2	3.30	0.5
Nb (per whole distance)	124	9	74	9
BF (min^{-1})	24	2.7	15	2

Legend:
T_I – inspiratory time;
T_E – expiratory time;
Nb – number of breaths per whole distance;
BF – breathing frequency

Reduced Nb and breathing frequency during swimming (BF) was accompanied by almost the same inspiratory time (T_i) in both breathing conditions.

Table 3: Comparisons [LA], pH, Po_2, Pco_2 values before and after swimming between two different breathing conditions (B2 and B4).

	B2				B4			
	Before		After		Before		After	
	MV	SD	MV	SD	MV	SD	MV	SD
[LA] (mmol/l)	1.2	0.4	7.9	1.1	1.6	0.3	8.1	1.9
pH	7.42	0.01	7.27	0.03	7.41	0.001	7.26	0.05
Pco2 (kPa)	4.8	0.3	4.8*	0.3	5	0.2	5.4*	0.2
Po2 (kPa)	11.4	1.3	12.4	1	10.5	0.5	11.3	0.4
[HCO_3^-] (mmol/l)	23	1	17	2	23	1	18	3

Legend:
Paired t-test (* - $p<0.05$)

Table 3 shows that Pco2 was significantly higher after B4 than after B2 ($p<0.05$). Other parameters ([LA], pH, Po2) did not change significantly in response to reduced breathing frequency during swimming.

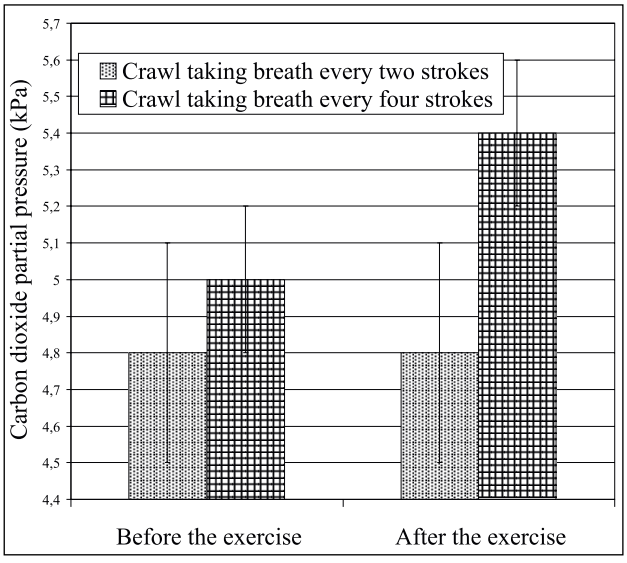

Figure 1: Comparisons the Pco2 values before and after swimming between two different breathing conditions.

DISCUSSION

The results of this study indicate that reduced breathing frequency during swimming 400 meters front crawl at velocity V_{OBLA} increased Pco_2 in capillary blood (Figure 1), which was different than during non-restricted breathing.
The average Pco_2 (5.4 kPa) measured 3 min after the swimming under B4 conditions (B4 (reduced pulmonary ventilation) was within the upper limit of

Pco_2 in the resting healthy subjects. However it was still significantly higher than Pco_2 after the B2 (normal breathing during front crawl swimming). Someone may assume that such differences within resting interval may not lead to a conclusion that B4 breathing influences hypercapnia. However, when fast exchange of CO_2 between blood and alveolar air was taken into account and resting interval of 3 minutes is also considered, then it may be concluded that the actual Pco_2 at the end of swimming is higher then measured Pco_2 after 3 minutes of rest. The inadequate pulmonary ventilation could also cause respiratory acidosis. Using trained female runners, Matheson et al. (1988) determined that breath holding during intermittent intense (125% Vo_2max) exercise induced a measurable rapidly reversible respiratory acidosis. The reduced breathing frequency during swimming did not influence pH in the present study. It might be presumed that respiratory acidosis did not occur at all, or it occurs at swimming and during very early recovery phase as an effect of hypercapnia. However it may rapidly disappear during later phase of recovery. Yamamoto et al. (1988) and Matheson et al. (1988) reported that blood lactate did not rise during exercise with reduced breathing frequency, but there were greater levels of blood lactate during recovery. They concluded that respiratory acidosis due to reduced pulmonary ventilation inhibited lactate transport from working muscles during activity. Our results did not show any change of [LA] and pH due to decreased ventilation at the third minute after the exercise. Therefore, the presented conclusions cannot be verified by present data and the problem remains to be solved in the future.

There was no statistical difference in Po_2 after swimming between B2 in B4 in the present study. According to Dicker et al. (1980) P_AO_2 in B2 decreased by 14% in comparison with B4. But estimated arterial oxygen saturation was essentially undiminished during swimming with reduced breathing frequency. Results of their study and our findings argue against the hypothesis of systemic hypoxia during sub maximal swimming with reduced breathing frequency. Different studies ascertained the hypothesis of systemic hypoxia during cycling exercise or running with reduced breathing frequency. Lee et al. (1990) concluded that the arterial oxygen desaturation during exercise with reduced breathing frequency appears to depend on: lung volume at which breath holding occurs, the barometric pressure of O_2 and exercise intensity. Alveolar hypoxia was accentuated more during breath holding at functional residual capacity than at total lung capacity at sea level and at low altitudes (Yamamoto et al., 1987). Breath holding at total lung capacity during sub maximal cycling exercise at moderate altitude (1520 m) caused arterial hypoxemia, tissue hypoxia, systemic hypercapnia and acidosis (Lee et al., 1990).

It may be concluded that the reduced breathing frequency from every second to every fourth stroke during front crawl swimming at V_{OBLA} velocity did not cause hypoxia. However, it increased the hypercapnia without substantial influence on acidosis.

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