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INFLUENCE OF REDUCED BREATHING DURING INTENSE FRONT CRAWL SWIMMING ON SOME RESPIRATORY AND METABOLIC VALUES IN BLOOD

VPLIVI ZMANJŠANEGA DIHANJA MED INTENZIVNIM PLAVANJEM KRAVLA NA NEKATERE DIHALNE IN METABOLNE KAZALCE V KRVI

Abstract

The purpose of the present study was to ascertain the influence of reduced breathing on the blood acid-base status during swimming at 90% velocity of maximal 200-m front crawl. Ten swimmers (age 16.6 ± 1.8 years, height 180 ± 7 cm and weight 70 ± 7 kg) volunteered to participate in this study. They performed maximal 200-m front crawl swim. Then they performed a sub-maximal front crawl swim twice to exhaustion: first, by taking breath every two strokes (B2) and second, by taking breath every four strokes (B4). The swimming velocity was determined as 90% of maximal velocity at 200-m front crawl swim. Measures included lactate concentration ([LA]) and parameters of blood acid-base status (pH, Po_2, Pco_2, [HCO_3-]) before and during the first and the third minute after the exercise. Swimmers swam with B2 significantly longer as they did with B4 ($p < 0.05$). [LA] was significantly lower after swimming with B4 than after swimming with B2 ($p < 0.05$). Pco_2 and [HCO_3-] were significantly higher after swimming with B4 than after swimming with B2 ($p < 0.05$). Po_2 and pH did not change significantly in response to reduced breathing during swimming. It may be concluded that the combination of severe hypercapnia, respiratory ac idosis and metabolic acidosis was the possible reason why swimmers had to stop earlier due to fatigue, when taking breath every four strokes.

Key words: swimming, reduced breathing, blood acid base status, blood lactate

Izvleček

Namen raziskave je bil ugotoviti učinke zmanjšanega dihanja na kazalce acido baznega statusa krvi med plavanjem z 90 % hitrostjo pri 200 m kravl maksimalno. V raziskavo je bilo vključenih 10 zdravih rekreativnih plavalcev (starost 16,6 ± 1,8 let, višina 180 ± 7 cm in teža 70 kg ± 7 kg), ki so najprej kar najhitreje odplavali 200 m kravl. Nato so odplavali še dve submaksimalni plavanji kravla, prvič z vdihom na vsak drugi zaveslaj (B2) in drugič z vdihom na vsak četrti zaveslaj (B4). Plavali so tako dolgo, dokler so lahko ohranjali 90 % hitrosti od 200 m kravl maksimalno. Pri tem smo merili: vsebnost laktata ([LA]) in kazalce acido baznega statusa v krvi (pH, Po_2, Pco_2 in [HCO_3-]). Vzorce smo jemali pred naporom in v prvi ter tretji minuti po naporu. Plavalci so plavali z B2 statistično značilno dlje kakor z B4 ($p < 0,05$). Pri tem je bila vsebnost laktata statistično značilno nižja, vrednosti Pco_2 in [HCO_3-] pa statistično značilno višji po plavanju z B4 v primerjavi s plavanjem z B2 ($p < 0.05$). Na osnovi teh rezultatov smo zaključili, da je kombinacija hiperkapnije in respiratorne ter metabolične acidoze možen razlog za krajše plavanje ob vdihih na vsak četrti zaveslaj.

Ključne besede: plavanje, zmanjšano dihanje, acido bazni status krvi, krvni laktat

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INTRODUCTION

The pulmonary ventilation during swimming is synchronised with swimming strokes. Therefore the breathing frequency is in accordance with the stroke frequency. It may be assumed that the swimming velocity and stroke frequency should also be regulated, so that the needs of increased pulmonary ventilation are met. If the breathing pattern is changed at a similar velocity, this may influence blood oxygenation and acid-base status as well as the swimmer's performance. It has been believed that reduced breathing (taking breath every four, six or eight strokes) during front crawl swimming induced arterial hypoxemia, and enhanced lactate production in working muscles (Counsilman, 1977). Therefore this training technique is often referred to as "hypoxic training" (Maglischo, 1990). In some previous studies swimmers reduced the breathing frequency (taking breath every four, six and eight strokes) during tethered flume front crawl swimming (Dicker, Lofthus, Thornton, & Brooks, 1980; Town, & Vanness, 1990), in interval training (Holmer, & Gullstrand, 1980) and during front crawl swimming at OBLA velocity (Kapus, Ušaj, Kapus, & Štrumbelj, 2002). These studies were unable to demonstrate reduced arterial oxygen saturation with this training technique, but did show a systematic hypercapnia. Kapus, Ušaj, Kapus and Štrumbelj (2002) also presumed that influences of reduced breathing during front crawl swimming would be more evident at higher velocity. The question is whether reduced breathing during swimming (taking breath every four strokes, which is often used during front crawl swimming), which induces respiratory acidosis, is sufficient to produce greater impacts on acid-base status and a swimmer's performance, given the impacts of the already present metabolic acidosis of heavy exercise. Matheson and McKenzie (1988) demonstrated that breath holding during intermittent intense exercise induced rapidly reversible respiratory acidosis superimposed on the metabolic acidosis of maximal exercise. Unfortunately, these data were not obtained during swimming. They also used 15-second breath holds, which is much longer that 3.3 seconds of expiratory time measured during front crawl swimming when taking breath every four strokes (Kapus, & Ušaj, 2002). Therefore the purpose of the present study was to ascertain the influence of reduced breathing on the blood acid-base status during swimming at 90% velocity of maximal 200-m front crawl.

METHOD

Participants

Ten recreational swimmers (age: M = 16.6 years, SD = 1.8 years; height: M = 180 cm, SD = 7 cm; weight: M = 70 kg, SD = 7 kg) volunteered to participate in this study.

Instruments

Blood collection and breathing measurements

Blood gas and acid-base parameters (pH, Po_2, Pco_2, [HCO_3-]) and [LA] were measured before and after sub-maximal swimming with different breathing frequency. Capillary blood samples (60 – 80 ml) were collected in the first and the third minute after each swim from a hyperemied earlobe for pH, Po_2, Pco_2, [HCO_3-] analysis using an ABL5 (Radiometer Copenhagen) instrument. [LA] was analysed with the use of MINI8 (LANGE, Germany) photometer.

Procedure

First, swimmers performed maximal 200-m front crawl swim. Then they performed sub-maximal

front crawl swimming twice: first, by taking breath every two strokes (B2), and second, by taking breath every four strokes (B4). They swam as long as possible at fixed, pre-determined velocity, which equalled 90% of velocity achieved during a 200-m front crawl. Stroke rate of sub-maximal swimming with B4 was the same as with B2, since we knew that swimmers reduced swimming velocity and/or increased stroke rate, when the need to breathe became critical during swimming with reduced breathing (Town, & Vanness, 1990).

The values were presented as means ± standard deviations (SD). The paired t-test was used to compare the data obtained during submaximal front crawl swimming (the term swimming will be used in the sense of front crawl swimming in the following text) under two different sets of breathing conditions.

RESULTS

Measurements of the swimming distance and breathing frequency during swimming under two different sets of breathing conditions are given in Table 1.

Table 1: Comparisons of the swimming distance and breathing frequency (fb) during swimming under two different sets of breathing conditions (B2 and B4).

Subject	Swimming with B2		Swimming with B4	
	Distance (m)	Fb (min^{1-})	Distance (m)	Fb (min^{1-})
1.	650.0	29.31	425.0	16.27
2.	400.0	32.37	350.0	16.86
3.	400.0	26.09	300.0	15.11
4.	350.0	24.95	350.0	13.70
5.	400.0	28.58	450.0	14.69
6.	450.0	29.36	350.0	16.37
7.	650.0	28.80	400.0	16.06
8.	450.0	28.82	250.0	15.52
9.	800.0	33.69	500.0	18.50
10.	550.0	35.30	400.0	19.90
M	**510.0	29.72	**377.5	16.29
SD	146.8	3.21	73.1	1.81

Legend: *M – mean values; SD – standard deviation; Paired t-test (** $p < 0.01$)*

Swimmers swam with B2 significantly longer as they did with B4 ($p < 0.01$). Comparisons of [LA] and pH values before and after swimming under two different sets of breathing conditions are given in Figure 1.

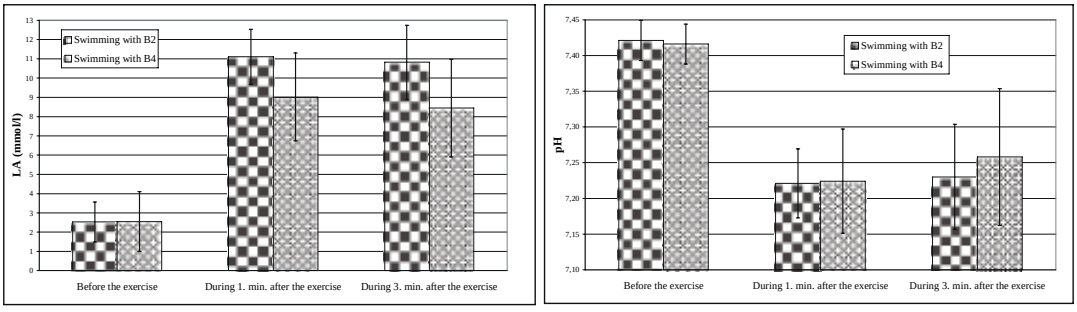

Figure 1: Comparisons of [LA] and pH values before and after swimming under two different sets of breathing conditions (p < 0.05, ** p < 0.01).*

[LA] was significantly lower after swimming with B4 than after swimming with B2 ($p < 0.01$). As expected, pH values measured in the first minute after the exercise did not change under two different sets of breathing conditions. The swimmers were told to swim as long as they were able to. Therefore it seemed that 7.22 is the limit pH value. But there were significant differences in pH measured in the third minute of recovery ($p < 0.05$). Pco2 and [HCO3-] values before and after swimming under two different sets of breathing conditions are stated in Figure 2.

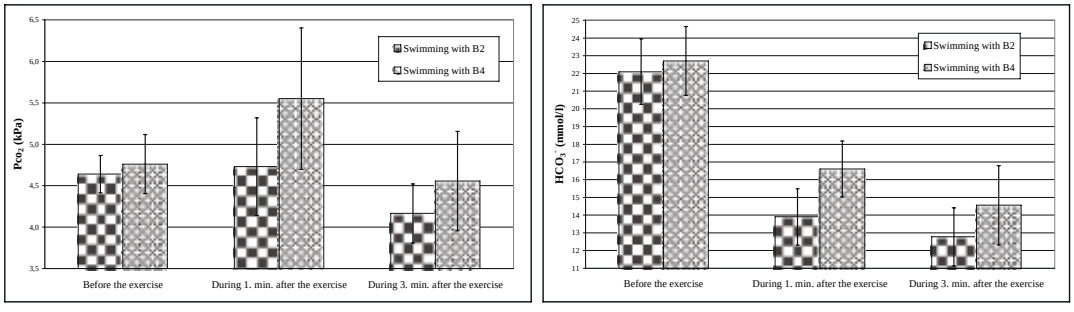

*Figure 2: Comparisons of Pco2 and [HCO3-] values before and after swimming under two different sets of breathing conditions (** p < 0.01).*

Pco_2 and [HCO_3-] were significantly higher after swimming with B4 than after swimming with B2 ($p < 0.01$).

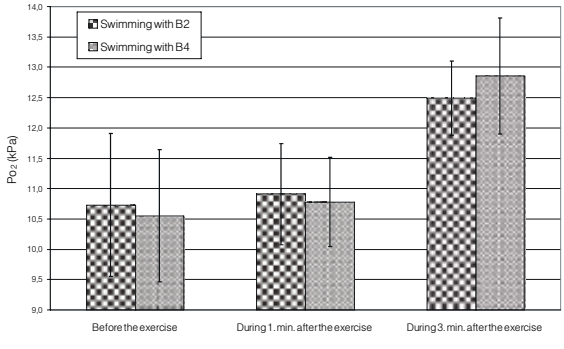

Figure 3: Comparisons of Po_2 values before and after swimming under two different sets of breathing conditions.

Figure 3 shows that Po_2 did not change significantly in response to reduced breathing during swimming.

DISCUSSION

In the present study both experimental conditions (normal and reduced breathing) applied the same relative work load. The swimmers were told to swim as long as they were able to maintain steady velocity. According to this instruction the swimming intensities were maximal for selected velocity and different durations for specific breathing conditions. This was different from the previous studies (Holmer, & Gullstrand, 1980; Dicker, Lofthus, Thornton, & Brooks, 1980; Town, & Vanness, 1990; Kapus, Ušaj, Kapus, & Štrumbelj, 2002), which concentrated on reduced breathing during exercise. In these studies the experimental exercises were restricted by time and/or by distance. According to this, it was expected that pH would be almost the same after both swims with different breathing frequency (Figure 1). But higher Pco_2 and [HCO_3-] (Figure 2) and lower [LA] (Figure 1) after swimming with B4 indicated that reduced breathing during intense swimming induced respiratory acidosis in capillary blood, which also persisted during recovery. Stanford, Williams, Sharp and Bevan (1985) concluded that reduced breathing during the exercise resulted primarily in an inhibition of the normal respiratory compensation that occurred during the exercise with normal breathing. In the present study swimmers swam with B2 for about 132 meters more than with B4. It may be concluded that respiratory acidosis superimposed on the metabolic acidosis of maximal swimming was the reason why swimmers were not able to swim longer while taking breath every four strokes.

In the present study the average Pco_2 (M = 5.5 kPa, SD = 0.8 kPa; Figure 2) measured in the first minute after swimming under B4 conditions was within the upper limit of Pco_2 with a resting healthy subject. Dicker, Lofthus, Thornton, & Brooks (1980) and Town & Vanness (1990) measured $Paco_2$ to be about 5.9 kPa (SD = 0.13 kPa) during swimming with B4. Considering the fast exchange of CO_2 between blood and alveolar air, it may be concluded that the actual Pco_2 at the end of swimming is higher than Pco_2 measured after 1-minute rest. Higher Pco_2 may have constituted a major stress during swimming with reduced breathing (Dicker, Lofthus, Thornton, & Brooks, 1980). In the third minute after swimming with B4, pH was significantly higher than the pH measured in the third minute after swimming with B2 (Figure 1). Kapus and Ušaj (2002) measured V_E during recovery after swimming with and without reduced breathing. Their measurements showed that V_E measured immediately after swimming was about 5% higher after swimming with B4 than after swimming with normal breathing. It seemed that elevated Pco_2 after swimming with reduced breathing presented the main stimulus for faster respiratory compensation of the acidosis during recovery.

Yamamoto, Takei, Mutoh and Miyashita (1988), measuring the exercise on a bicycle ergometer, failed to support the previous idea of enhanced lactic acidosis as a product of reduced breathing during exercise. Holmer & Gullstrand (1980) and Matheson & McKenzie (1988) measured [LA] in rest intervals during intermittent exercise with reduced breathing. They obtained significantly lower or unchanged lactate levels during reduced breathing conditions. After the exercise with reduced breathing unchanged (Town, & Vanness, 1990; Stanford, Williams, Sharp, & Bevan, 1985; Kapus, Ušaj, Kapus, & Štrumbelj, 2002) or significantly higher (Matheson, & McKenzie, 1988; Yamamoto, Takei, Mutoh, & Miyashita, 1988) lactate levels were reported in comparison with normal breathing exercise. Yamamoto, Takei, Mutoh and Miyashita (1988) concluded that inhibition of lactate efflux from working muscles due to hypercapnia occurred during the exercise with reduced breathing. In the present study the [LA] in the first and the third minute after swimming with B4 decreased by 19% and 23%, respectively, in comparison to swimming with B2 (Figure 1). It may be presumed that [LA] was lower during swimming with B4 first because of shorter swimming exercise. However, it maintained in working muscles as an effect of hypercapnia may also be possible.

In the present study there was no statistical difference in Po_2 after swimming between B2 and B4 (Figure 3). To our knowledge, hypoxia has not yet been proved a result of reduced breathing during swimming (Holmer, & Gullstrand, 1980; Dicker, Lofthus, Thornton, & Brooks, 1980; Town, & Vanness, 1990; Kapus, Ušaj, Kapus, & Štrumbelj, 2002). According to Dicker, Lofthus, Thornton and Brooks (1980) PAo_2 with B2 decreased by 14% in comparison with B4. But estimated arterial oxygen saturation was essentially undiminished during swimming with reduced breathing. The reason for this phenomenon could be increased V_T during swimming with reduced breathing. Therefore it could be assumed that swimmers hold their breath closer to total lung capacity than to functional residual capacity, since we know that alveolar hypoxia was accentuated more during breath holding at functional residual capacity than at total lung capacity (Yamamoto, Mutoh, Kobayashi, & Miyashita, 1987).

It may be concluded that the combination of severe hypercapnia, respiratory acidosis and metabolic acidosis was the possible reason why swimmers had to stop earlier due to fatigue, when taking breath every four strokes.

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