Research article

The difference in respiratory and blood gas values during recovery after exercise with spontaneous versus reduced breathing frequency

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Abstract
Extrapolation from post-exercise measurements has been used to estimate respiratory and blood gas parameters during exercise. This may not be accurate in exercise with reduced breathing frequency (RBF), since spontaneous breathing usually follows exercise. This study was performed to ascertain whether measurement of oxygen saturation and blood gases immediately after exercise accurately reflected their values during exercise with RBF. Eight healthy male subjects performed an incremental cycling test with RBF at 10 breaths per minute. A constant load test with RBF (B10) was then performed to exhaustion at the peak power output obtained during the incremental test. Finally, the subjects repeated the constant load test with spontaneous breathing (SB) using the same protocol as B10. Pulmonary ventilation (V_E), end-tidal oxygen ($P_{ET}O_2$), and carbon dioxide pressures ($P_{ET}CO_2$) and oxygen saturation (SaO_2) were measured during both constant load tests. The partial pressures of oxygen (PO_2) and carbon dioxide (PCO_2) in capillary blood were measured during the last minute of exercise, immediately following exercise and during the third minute of recovery. At the end of exercise RBF resulted in lower $P_{ET}O_2$, SaO_2 and PO_2, and higher $P_{ET}CO_2$ and PCO_2 when compared to spontaneous breathing during exercise. Lower SaO_2 and $P_{ET}O_2$ were detected only for the first 16s and 20s of recovery after B10 compared to the corresponding period in SB. There were no significant differences in PO_2 between SB and B10 measured immediately after the exercise. During recovery from exercise, $P_{ET}CO_2$ remained elevated for the first 120s in the B10 trial. There were also significant differences between SB and B10 in PCO_2 immediately after exercise. We conclude that RBF during high intensity exercise results in hypoxia; however, due to post-exercise hyperpnoea, measurements of blood gas parameters taken 15s after cessation of exercise did not reflect the changes in PO_2 and SaO_2 seen during exercise.

Key words: Constant load test, reduced breathing frequency, recovery, respiratory parameters, oxygen saturation, blood gas.

Introduction

In some sports, the environment is inappropriate for direct measurement of respiratory and blood gas parameters during exercise. Moreover, the attachment of cumbersome measurement equipment may influence the motion technique and consequently increase energy cost. To overcome this problem, respiratory and blood gas parameters have been measured at the end of exercise to estimate physiological responses during the exercise. Backward extrapolation of the O_2 recovery curve has been used to calculate the peak oxygen uptake during swimming (Rodriguez et al., 2002) and synchronized swimming (Bante et

al., 2007). This method requires that measurements are made as soon as possible after the end of exercise. Data collection lasts a few minutes, and the recovery curve is extrapolated back to time zero, i.e., to the end of exercise.

Extrapolation from post-exercise measurements has also been used to estimate changes in partial pressures of blood gases induced by reduced breathing frequency (RBF), as observed in competitive swimming. However, Štrumbelj et al. (2006) found that measurements of respiratory and blood gas parameters taken after the end of a maximal front crawl swimming test did not reflect the conditions which appeared during the swimming test. RBF during swimming increased the alveolar CO_2 concentration (Dicker et al., 1980; Peyrebrune et al., 2002; Town and Vanness, 1990; West et al., 2005) and induced higher partial pressure of carbon dioxide in capillary blood after it (Kapus et al., 2002; Kapus et al., 2003). Nonetheless, these studies failed to demonstrate a reduction in oxygen saturation due to RBF either by analysing expired air during swimming (Holmér et al., 1980) or by sampling capillary blood after swimming (Kapus et al., 2002; 2003). On the contrary, RBF during cycle ergometry has been shown to cause a reduction in oxygen saturation and lower partial pressure of oxygen, measured in arterial (Yamamoto et al., 1987) and capillary (Kapus et al., 2007; Sharp et al., 1991) blood. Considering that hypoxia has been detected *during* cycling but not *after* swimming, the timing of measurement may be the reason for the apparent difference in response to RBF. The timing of measurement may be especially important when spontaneous breathing follows the exercise with RBF. The present study was designed to elucidate this problem. To date, no data of oxygen saturation and respiratory parameters during recovery after exercise with RBF have been presented. Therefore, the aim of the present study was to ascertain whether measurements of oxygen saturation and blood gases measured immediately after exercise could estimate their values during exercise with RBF.

Methods

Subjects

Eight healthy male subjects (age 25 ± 1 years, height 1.81 ± 0.03 m, weight 80 ± 7 kg, peak oxygen uptake (VO_2peak) 44.26 ± 2.93 ml·kg^{-1}·min^{-1}, forced vital capacity of 5.98 ± 0.58 l and forced expiratory volume of 4.76 ± 0.59 l in 1 s) volunteered to participate in this study. None of the subjects were smokers and were free of respiratory disease at the time of the study. The subjects were fully informed of the purpose and possible risks of the

study before giving their written consent to participate. The study was approved by the University's Research Ethics Committee.

Procedures

RBF was defined as 10 breaths per minute and was regulated by a breathing metronome. The breathing metronome was composed of a gas service solenoid valve 24 VDC (Jakša, Ljubljana, Slovenia) and a semaphore with red and green lights. Both were controlled by a micro automation Logo DC 12/24V (Siemens, Munich, Germany). The subjects were instructed to expire and inspire during a 2 s period of open solenoid valve (the green semaphore light was switched on) and to hold their breath, using almost all lung capacity (breath holding near total lung capacity), for 4 s when the solenoid valve was closed (the red semaphore light was switched on). Prior to the exercise testing, the subjects were familiarized with breathing through the breathing metronome. After familiarization, each subject performed 4 exercise tests on an electromagnetically braked cycle ergometer Ergometrics 900 (Ergoline, Windhagen, Germany) with pedal cadence at ~60 revolutions per minute (rpm). Tests were performed in a prescribed order, each of them on a different day.

Preliminary tests: The subjects initially performed an incremental exercise test (IT) to obtain VO_2peak. The test began at 30 W and increased by 30 W every 2 min until volitional exhaustion. VO_2peak was defined as the highest O_2 uptake averaged over 60-s interval. The subjects then performed an incremental exercise test with RBF (ITB10) to obtain peak power output. Except breathing, the protocol of this test was identical to the protocol of IT. The peak power output was defined as the highest work stage that each subject completed. From these results, the work rate for the constant load test with RBF was chosen for each subject.

Experimental protocol: After preliminary testing, a constant load test with RBF (B10) was performed to exhaustion at the peak power output obtained during ITB10. This test started with a 5 min warm-up at 50 W. After that, the resistance was increased to match the subject's peak power output and the subject continued to exhaustion. The constant load test was completed by 10 min of active recovery at 20 W with spontaneous breathing. Finally, the subjects repeated the constant load test, however, with spontaneous breathing (SB). The protocol (intensity and duration) of this test was otherwise identical to the protocol of B10.

Measurements

During the constant load tests (warm-up, exercise and 10 min recovery; SB and B10), the subjects breathed through a mouthpiece attached to a pneumotachograph. The subject's expired gas was sampled continuously by a V-MAX29 (SensorMedics Corporation, Yorba Linda, USA) metabolic cart for a breath-by-breath determination of respiratory parameters (pulmonary ventilation (V_E), end-tidal pressure of oxygen ($P_{ET}O_2$) and end-tidal pressure of carbon dioxide ($P_{ET}CO_2$)). The pneumotachograph and the O_2 and CO_2 analysers were calibrated prior to the test with a standard 3 L syringe and precision reference gases,

respectively. For further statistical analysis, breath-by-breath data were averaged for each 10-s interval. During the constant load tests (warm-up, exercise and 10 min recovery), oxygen saturation (SaO_2) was measured using a TruStat^{TM} Pulse Oximeter (Datex-Ohmeda, Madison, USA). The pulse oximeter is an indirect oximetry measuring instrument, which displays SaO_2 every 4 s. The ear probe was attached to the earlobe after cleaning the area with alcohol. The partial pressure of oxygen (PO_2) and carbon dioxide (PCO_2) in capillary blood were measured during the last minute of exercise, immediately following exercise (the delay between the subject's cessation of the exercise and the first measure did not exceed 15 s) and during the third minute of recovery. Capillary blood samples (60 – 80 μl) were taken by micro puncture from an earlobe. Earlobe capillary blood was arterialized by the application of hyperemic cream (Finalgon, Boehringer-Ingelheim, Reims, France) at least 20 min before the first capillary sample. Earlobe samples were collected in heparinized glass capillary tubes and introduced into a blood gas analyser ABL5 (Radiometer, Copenhagen, Denmark) for gas analysis at 37° C.

Statistical analysis

The results are presented as means and standard deviations (SD). One-way analysis of variance (ANOVA) for repeated measures was used to test the statistical differences between SB and B10 during entire recovery period. If significance was documented, post hoc, pair wise comparisons of the data of respiratory parameters and SaO_2 at the end of exercise and during initial 40 s of recovery were made using paired t test. In addition, the data of respiratory parameters measured every fortieth s from initial 40 s period of recovery were also included in these comparisons. Statistical significance was accepted at the p ≤ 0.05 level. All statistical parameters were calculated using the statistics package SPSS (version 15.0, SPSS Inc., Chicago, USA) and the graphical statistics package Sigma Plot (version 9.0, Jandel, Tübingen, Germany).

Results

Table 1 depicts VO_2 responses to incremental cycle ergometry in relation to VO_2 responses in subjects during the incremental test with RBF.

Data depicted in Table 2, describes the average intensity and time to exhaustion in B10 and yielded 191 W (32 W) and 566 s (332 s), respectively. By design, the intensity and duration of B10 and SB were identical. As expected, V_E was significantly lower (p ≤ 0.01) in B10 than in SB at the end of exercise (time zero of the recovery curve in Figure 1). However, in the B10 trial, V_E dramatically increased post-exercise and it was higher during the initial 200 s of recovery than it was during the corresponding period in the SB trial (p ≤ 0.01). $P_{ET}O_2$ was significantly lower at the end of exercise and during the initial 20 s of recovery in B10 than in SB (p ≤ 0.01; Figure 2). Similar results were shown for SaO_2 (Figure 3), which differed significantly (p ≤ 0.01) between the trials at the end of exercise and for the initial 16 s of recovery. $P_{ET}CO_2$ was significantly higher for the first 120 s of recovery after B10 than after SB (p ≤ 0.01; Figure 4).

Table 1. Individual VO$_2$ responses to incremental bicycle exercise in two different breathing conditions.

subject		power output (W)											
		0	**30**	**60**	**90**	**120**	**150**	**180**	**210**	**240**	**270**	**300**	**330**
1	IT	2,86	7,72	10,14	13,68	16,44	21,77	24,69	28,37	31,68	35,75		
	ITB10	5,00	9,26	12,11	13,80	17,13	20,75	23,65	27,12	29,81			
2	IT	3,63	8,44	12,03	15,30	19,97	23,36	27,37	31,67	36,19	41,90	47,35	
	ITB10	5,55	11,93	13,83	16,53	21,22	24,03	28,57					
3	IT	3,40	8,16	11,58	15,11	19,48	23,01	27,66	32,37	35,97	40,19	45,08	
	ITB10	4,70	8,27	13,71	16,48	21,03	24,56	28,26	31,64				
4	IT	3,79	8,26	11,83	15,06	17,84	20,30	23,99	28,33	32,55	36,25	39,97	
	ITB10	5,51	9,67	11,73	15,15	17,20	19,19						
5	IT	3,80	8,13	12,36	16,95	20,10	26,54	28,02	31,91	35,71	39,84	44,52	
	ITB10	4,97	9,31	13,07	15,31	18,92	21,24						
6	IT	3,80	8,68	11,50	15,68	17,68	22,08	24,78	27,98	32,68	34,69	40,10	44,63
	ITB10	5,22	10,28	15,25	16,54	20,06	23,54	26,49	28,94				
7	IT	4,04	8,26	11,84	15,77	18,06	22,62	25,60	29,01	33,01	36,68	42,56	46,73
	ITB10	4,01	8,84	12,50	14,77	18,18	18,13	22,98					
8	IT	3,84	9,24	12,06	15,60	19,75	23,60	27,78	31,75	36,75	43,12		
	ITB10	4,61	9,74	12,81	15,40	19,06	23,67	26,85	29,45				

IT and ITB10 denote incremental exercise test with spontaneous breathing and RBF, respectively.

Table 2. Individual values of power output and time to exhaustion during B10.

subject	power output (W)	duration (s)
1	240	420
2	180	317
3	210	358
4	150	1158
5	150	1032
6	210	354
7	180	408
8	210	479

Table 3 shows that PO$_2$ was significantly lower during the last minute of exercise in B10 than during SB (p $\leq$ 0.01). In addition there were significant differences between SB and B10 in PCO$_2$, measured during the last minute of exercise and immediately after it (p $\leq$ 0.01).

Discussion

In accordance with previous studies, RBF during exercise produced a marked reduction in V$_E$. V$_E$ measured at the end of exercise was 49% lower in B10 than SB (Figure 1). With a similar breathing frequency reduction during cycle ergometry, Yamamoto et al. (1987) and Sharp et al. (1991) obtained a smaller reduction of V$_E$. However, different testing protocols and intensities of exercise with RBF were used in these studies. Yamamoto et al. (1987) showed a reduction of 30% in V$_E$ during an interval test with RBF (30 s of exercise at 210 W with RBF alternating with 30 s rest intervals with spontaneous breathing). Sharp et al. (1991) measured a reduction of 25% in V$_E$ during 8 min of exercise at an intensity above the lactate threshold due to RBF. In the studies of RBF during front crawl swimming, a similar reduction in V$_E$, as it was obtained in the present study, was observed with taking a breath every sixth (Town and Vanness, 1990) or eighth (West et al., 2005) stroke cycle as compared to taking a breath every second stroke cycle. However, after cessa-

tion of B10, when spontaneous breathing was allowed, V$_E$ dramatically increased to a peak in the 20th s of recovery. Thereafter, it decreased to the resting values. In contrast, V$_E$ immediately began to decrease to the resting values after cessation of exercise with SB.

Table 3. Comparison of the PO$_2$ (kPa) and PCO$_2$ (kPa) values measured during the last minute of exercise, immediately following exercise, and during the third minute of recovery between the two different breathing conditions.

parameter		SB	B10
PO$_2$	last minute during the exercise	11.3 (0.6)	8.7 (1.2) **
	immediately following the exercise	11.8 (1.4)	10.9 (0.9)
	during the 3. minute of the recovery	12.4 (1.0)	12.5 (1.2)
PCO$_2$	last minute during the exercise	5.2 (0.8)	7.3 (0.5) **
	immediately following the exercise	5.1 (1.0)	5.9 (1.1) **
	during the 3. minute of the recovery	5.0 (0.5)	5.1 (0.5)

** denote p $\leq$ 0.01 between the exercises in two different breathing conditions.

During the initial 20 s of recovery after B10, the subjects breathed an average 26.4 liters of air compared to 20.5 liters after exercise with SB. This marked hyperventilation influenced changes in other respiratory and blood parameters. As expected, at the end of exercise, RBF resulted in lower P$_{ET}$O$_2$, SaO$_2$ and PO$_2$, when compared to spontaneous breathing during exercise (Figure 2 and 4, Table 3). These data were in accordance with previous studies, which measured these parameters during different cycling exercises with RBF (Kapus et al., 2007; Sharp et al., 1991; Yamamoto et al., 1987). Due to different testing protocols, Yamamoto et al. (1987) and Kapus et al. (2007) reported higher values of SaO$_2$ than that were measured at

the end of B10. The intensity (subject's peak power output) and duration (exercise to exhaustion) of B10 were maximal for each subject. Considering that, low values of SaO_2 (83% (8%)) at the end of exercise with RBF were expected in the present study. The results confirmed severe hypoxia during B10. However, hyperventilation after the cessation of B10 induced a rapid recovery of $P_{ET}O_2$, SaO_2, and PO_2. Lower values of O_2 were detected only during the initial 16 s and 20 s after B10 in comparison to SB, measured by SaO_2 (Figure 4) and $P_{ET}O_2$ (Figure 2) respectively. However, there were no significant differences in PO_2 between SB and B10 measured immediately after the exercise (Table 3). The delay between the subjects's cessation of the exercise and the first measurement did not exceed 15 s. Nonetheless, this delay was apparently too long to detect hypoxia with measurement of PO_2 after B10. According to our experience, this delay is longer for field testing such as swimming tests in the swimming pool. Considering that, the time of measurement may be the reason why previous studies failed to demonstrate a reduction in PO_2 due to RBF during swimming (Kapus et al., 2002; 2003).
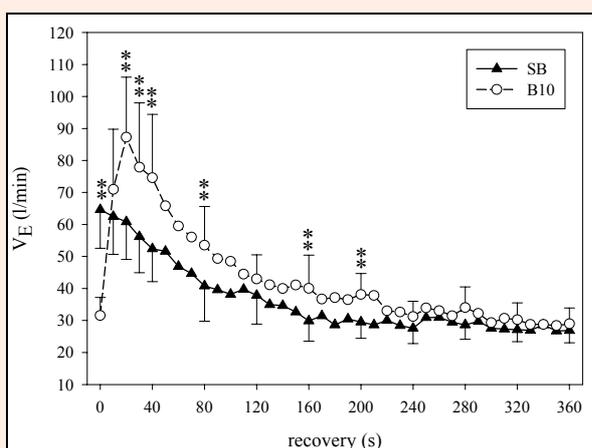
Figure 1. There were significant differences between SB and B10 in V_E measured at the end of exercise and during recovery (ANOVA for repeated measures, p ≤ 0.01). The statistically analysed results for comparison at defined time during recovery are marked with standard deviations (paired *t* test).
** denotes p ≤ 0.01 between the two different breathing conditions.

In accordance with previous studies (Dicker et al., 1980; Kapus et al., 2007; Peyrebrune et al., 2002; Sharp et al., 1991; Town and Vanness, 1990; West et al., 2005, Yamamoto et al., 1987), RBF produced hypercapnia, as evidenced by higher $P_{ET}CO_2$ and PCO_2 in B10 than during SB. Yamamoto et al. (1987) found that arterial partial pressure of carbon dioxide (P_aCO_2) and hydrogen ion concentration ($[H^+]$) continuously increased to the end of an interval test with RBF. Using 8 min of exercise with RBF at an intensity 10 % above lactate threshold workload, Sharp et al. (1991) reported similar results. They concluded that RBF during exercise caused respiratory acidosis at exercise intensities that were not associated with $[H^+]$ disturbance during unreduced V_E. Considering that, it was suggested that the combination of severe

hypercapnia, respiratory acidosis and metabolic acidosis was the possible reason for earlier fatigue during exercise at higher intensities, when RBF was used (Kapus et al., 2003). During recovery from exercise, $P_{ET}CO_2$ remained elevated in the B10 trial compared to the SB trial (Figure 4), even after $P_{ET}O_2$ had normalized (Figure 2). Lee et al. (1990) reported a reduction in VCO_2 during exercise with RBF, and a subsequent increase during recovery. They suggested that CO_2 was retained in muscle, plasma and erythrocytes during exercise with RBF and that it was released from these stores during recovery. It seemed that despite hyperventilation during recovery, hypercapnia could be detected by measuring blood gas parameters within 15 s after the exercise with RBF.
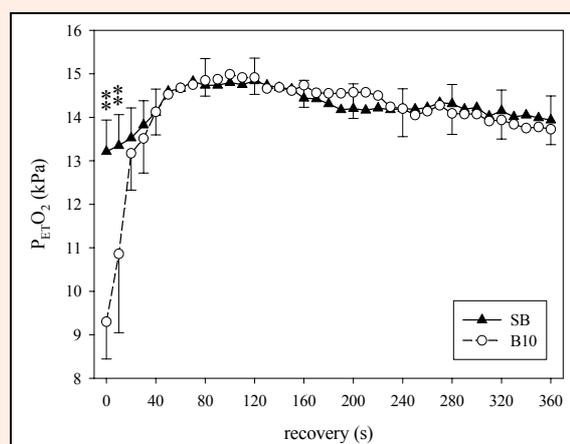
Figure 2. There were significant differences between SB and B10 in $P_{ET}O_2$ measured at the end of exercise and during recovery (ANOVA for repeated measures, p ≤ 0.01). The statistically analysed results for comparison at defined time during recovery are marked with standard deviations (paired *t* test).
** denotes p ≤ 0.01 between the two different breathing conditions.
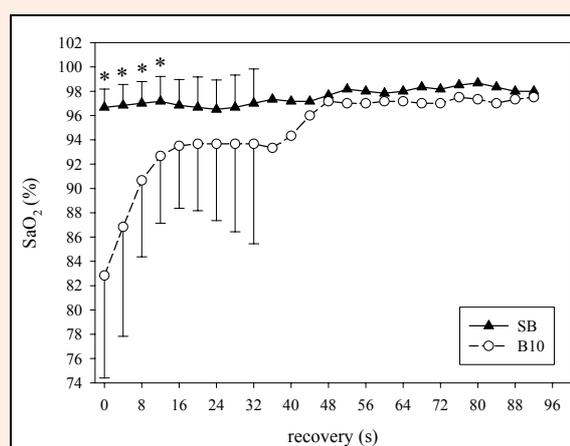
Figure 3. There were significant differences between SB and B10 in SaO_2 measured at the end of exercise and during recovery (ANOVA for repeated measures, p ≤ 0.01). The statistically analysed results for comparison at defined time during recovery are marked with standard deviations (paired *t* test).
* denote p ≤ 0.05 between the two different breathing conditions.
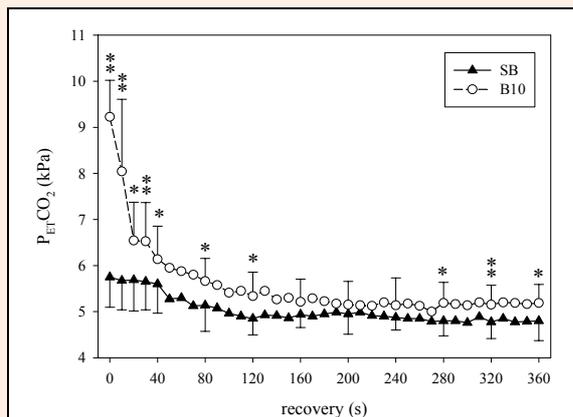
Figure 4. **There were significant differences between SB and B10 in $P_{ET}CO_2$ measured at the end of exercise and during recovery (ANOVA for repeated measures, $p \leq 0.01$). The statistically analysed results for comparison at defined time during recovery are marked with standard deviations (paired t test).**

* and ** denote $p \leq 0.05$ and 0.01 respectively between the two different breathing conditions.

Possible study limitations: Ideally blood gases should be obtained in arterial blood. However, indwelling arterial catheters for sampling arterial blood are not always feasible and desirable. Considering that, some indirect methods were used to assess blood gases in the present study. Therefore, the degree to which the actual measurements provide an accurate proxy for arterial measures should be considered. Arterial blood gases (P_aO_2 and P_aCO_2) during exercise could be estimated by using arterialized earlobe blood samples (PO_2 and PCO_2). Some previous studies found that arterialized earlobe blood samples are in good agreement with arterial blood samples for partial pressure of carbon dioxide, but not for partial pressure of oxygen (Dall´Ava-Santucci, 1996; Fajac et al., 1998; McEvoy and Jones, 1975). During exercise, PO_2 was lower than P_aO_2 on average 0.23 kPa (McEvoy and Jones, 1975), 0.63 kPa (Fajac et al., 1998) and 1.2 kPa (Dall´Ava-Santucci, 1996). The main cause of underestimation of P_aO_2 in earlobe samples could be insufficient arterialization of blood due to venus admixture. The earlobe method requires adequate blood flow in the earlobe to enable a sufficient volume of blood to be sampled without additional external pressure during sampling. This was the reason for the delay of up to 15 s between the subject's cessation of the exercise and the first measure in the present study. In addition, measurement of end-tidal pressure of carbon dioxide ($P_{ET}CO_2$) has been used to estimate P_aCO_2 at rest and during exercise. Most comparative studies have concluded that $P_{ET}CO_2$ provides good index of P_aCO_2 at rest (Jones et al., 1979, Williams and Babb, 1997). However, during exercise, the differences between $P_{ET}CO_2$ and P_aCO_2 were 0.3 kPa (0.3 kPa) (Williams and Babb, 1997) and 0.4 kPa (0.3 kPa) (Robbins et al., 1990). These differences increased at a higher workload and with increasing tidal volume (Jones et al., 1979). Ear pulse oximeters are often used to provide a non-invasive, continuous estimate of the oxyhemoglobin saturation of arterial blood (SaO_2). In most previ-

ous validation studies, ear pulse oximeter estimates during exercise have been shown to be accurate predictors of SaO_2 at least when saturation is above 85% in non-smoking subjects (Mengelkoch et al., 1994, Smyth et al., 1986, Powers et al., 1989, Martin et al., 1992). Considering ear pulse oximeters of Datex-Ohmeda, differences between estimated and measured (via blood sampling) SaO_2 values were 0.87 % (2.6 %), 0.59 % (2.4 %) (Martin et al., 1992) and -0.57 % (1.78 %) (Powers et al., 1989). Thus, the error in the pulse oximeter is not likely greater than 1 %, while significant differences between SB and B10 in SaO_2 were between 5 and 10 % during the initial 16 s of recovery.

Conclusion

In conclusion, reduced breathing frequency during high intensity exercise results in hypoxia, however, due to marked post-exercise hyperventilation, measurements of blood gas parameters analyzed 15 s after the cessation of exercise did not accurately reflect the condition during exercise.

References

Bante, S., Bogdanis, G.C., Chairpoulou, C. and Maridaki, M. (2007) Cardiorespiratory and metabolic responses to a simulated synchronized swimming routine in senior (>18 years) and comen (13-15 years) national level athletes. *Journal of Sports Medicine and Physical Fitness* **47,** 291-299.

Dall Ava-Santucci, J., Dessanges, J.F., Dinh Xuan, A.T. and Lockhart, A. (1996) Is arterialized earlobe blood PO_2 an acceptable substitute for arterial blood PO_2? *European Respiratory Journal* **9,** 1329-1330.

Dicker, S. G., Lofthus, G. K., Thornton, N. W. and Brooks, G. A. (1980) Respiratory and heart rate responses to tethered controlled frequency breathing swimming. *Medicine and Science in Sport and Exercise* **12,** 20-23.

Fajac, I., Texereau, J., Rivoal, V., Dessanges, J.F., Dihn-xuan, A.T. and Dall Ava-Santucci, J. (1998) Blood gas measurement during exercise: a comparative study between arterialized earlobe sampling and direct arterial puncture in adults. *European Respiratory Journal* **11,** 712 - 715.

Holmér, I. and Gullstrand, L. (1980) Physiological responses to swimming with a controlled frequency of breathing. *Scandinavian Journal of Sports Science* **2,** 1-6.

Jones, N.L., Robertson, D.G. and Kane, J.W. (1979) Difference between end-tidal and arterial PCO_2 in exercise. *Journal of Applied Physiology* **47,** 954-960.

Kapus, J., Ušaj, A., Kapus, V. and Štrumbelj, B. (2002) The influence of reduced breathing during swimming on some respiratory and metabolic values in blood. *Kinesiologia Slovenica* **8,** 14-18.

Kapus, J., Ušaj, A., Kapus, V. and Štrumbelj, B. (2003) The influence of reduced breathing during intense front crawl swimming on some respiratory and metabolic values in blood. *Kinesiologia Slovenica* **9,** 12-17.

Kapus, J., Ušaj, A., Kapus, V. and Štrumbelj, B. (2007) The influence of reduced breathing during incremental bicycle exercise on some ventilatory and gas exchange parameters. In: *Proceedings of 11th Mediterranean Conference on Medical and Biological Engineering and Computing*. Eds: Jarm, T., Kramar, P. and Zupanic, A. New York: Springer.

Lee, C., Cordain, L., Sockler, J. and Tucker, A. (1990) Metabolic consequences of reduced frequency breathing during submaximal exercise at moderate altitude. *European Journal of Applied Physiology* **61,** 289-293.

Martin, D., Powers, S., Cicale, M., Collop, N., Huang, D. and Criswell, D. (1992) Validity of pulse oximetry during exercise in elite endurance athletes. *Journal of Applied Physiology* **72,** 455-458.

McEvoy, J.D.S., Jones, N.L. (1975) Arterialized capillary blood gases in exercise studies. *Medicine and Science in Sports* **7,** 312-315.

Mengelkoch, L.J., Martin, D. and Lawler, J. (1994) A review of the principles of pulse oximetry and accuracy of pulse oximeter estimates during exercise. *Physical Therapy* **74,** 40-49.

Peyrebrune, M., Robinson, J., Lakomy, H. and Nevill, M. (2002) Effects of controlled frequency breathing on maximal tethered swimming performance. In: *Biomechanics and Medicine in Swimming 9*. Ed: Chatard, J.C. Saint-Etienne: Laboratoire de Physiologie and Service de Exploration Fonctionnelle Cardio-respiratorie et de Medecine du Sport. 289-294.

Powers, S.K., Dodd, S., Freeman, J., Ayers, G.D., Samson, H. and McKnight, T. (1989) Accuracy of pulse oximetry to estimate HbO_2 fraction of total Hb during exercise. *Journal of Applied Physiology* **67,** 300-304.

Robbins, P.A., Conway, J., Cunningham, D.A., Khamnei, S. and Paterson, D.J. (1990) A comparison of indirect methods for continuous estimation of arterial Pco_2 in men. *Journal of Applied Physiology* **68,** 1727-1731.

Rodriguez, F., Keskinen, K., Malvela, M. and Keskinen, O. (2002) Oxygen uptake kinetics during free swimming: A pilot study. In: *Biomechanics and Medicine in Swimming 9*. Ed: Chatard, J.C. Saint-Etienne: Laboratoire de Physiologie and Service de Exploration Fonctionnelle Cardio-respiratorie et de Medecine du Sport. 379-384.

Sharp, R.L., Williams, D.J. and Bevan, L. (1991) Effects of controlled frequency breathing during exercise on blood gases and acid-base balance. *International Journal of Sports Medicine* **12,** 62-65.

Smyth, R.J., D`Urzo, A.D., Slutsky, A.S., Galko, B.M. and Rebuck, A.S. (1986) Ear oximetry during combined hypoxia and exercise. *Journal of Applied Physiology* **60,** 716-719.

Štrumbelj, B., Ušaj, A., Kapus, J. and Bednarik, J. (2006) Evidence of insufficient pulmonary ventilation during crawl swimming with maximal and supramaximal intensities. In: *Biomechanics and Medicine in Swimming 10*. Eds: Vilas-Boas, J.P., Alves, F., Marques, A. 1 th edition. Porto: Portuguese Journal of Sport Sciences. 175-177.

Town, G. P. and Vanness, J. M. (1990) Metabolic responses to controlled frequency breathing in competitive swimmers. *Medicine and Science in Sport and Exercise* **22,** 112-116.

Yamamoto, Y., Mutoh, Y., Kobayashi, H. and Miyashita, M. (1987) Effects of reduced frequency breathing on arterial hypoxemia during exercise. *European Journal of Applied Physiology* **56,** 522-527.

West, S.A, Drummond, M.J., VanNess, J.M. and Ciccolella, M.E. (2005) Blood lactate and metabolic responses to controlled frequency breathing during graded swimming. *Journal of Strength and Conditioning Research* **19,** 772-776.

Williams, J.S. and Babb, T.G. (1997) Differences between estimates and measured Pa_{co2} during rest and exercise in older subjects. *Journal of Applied Physiology* **83,** 312-316.

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Key points

- In some sports, the environment is inappropriate for direct measurement of respiratory and blood gas parameters during exercise. To overcome this problem, extrapolation from post-exercise measurements has often been used to estimate changes in respiratory and blood gas parameters during exercise.
- The possibility of hypoxia and hypercapnia during exercise with reduced breathing frequency has been tested by measuring capillary blood sampled after the exercise.
- Reduced breathing frequency during high intensity exercise results in hypoxia; however, due to marked post-exercise hyperventilation, measurements of blood gas parameters taken 15 s after the cessation of exercise did not yield any changes in these parameters.
- Despite hyperventilation during recovery, hypercapnia could be detected by measuring blood gas parameters within 15 s after the exercise with reduced breathing frequency.

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