

## Effect of moderate and high intensity training sessions on cardiopulmonary chemosensitivity and time-based characteristics of response in high performance rowers

### Authors' Contribution:

A – Study Design  
 B – Data Collection  
 C – Statistical Analysis  
 D – Data Interpretation  
 E – Manuscript Preparation  
 F – Literature Search  
 G – Funds Collection

Tomasz Tomiak<sup>1 ABCD</sup>, Viktor Mishchenko<sup>1 ABDE</sup>,  
 Elena Lusenko<sup>2 BD</sup>, Andrej Diachenko<sup>2 BD</sup>, Adam Korol<sup>1 BC</sup>

<sup>1</sup> Gdansk University of Physical Education and Sport in Gdansk, Poland

<sup>2</sup> National University of Physical Education of Ukraine, Kiev, Ukraine

**Key words:** sport training, recovery phase, hypercapnic chemosensitivity, hypoxic chemosensitivity; fast kinetics.

### Abstract

**Background:** The present study was performed to clarify fatigue-induced effects of a strenuous and moderate intensity endurance training session on temporary changes of cardiopulmonary (CP) chemosensitivity and fast kinetics response.

**Material/Methods:** Eleven high performance (national level) male rowers participated in this study [age 21.8 ± 1.7 (range 18-25 years), 89.3 ± 2.0 kg, 190.1 ± 1.7 cm, VO<sub>2</sub> max 67.9 ± 1.1 ml·kg<sup>-1</sup>·min<sup>-1</sup>]. The studies involved three steps: 1) a study of effects related to a training session of moderate intensity, 2) effects of a high intensity session, and 3) an impact of a high intensity session on values of peak response. The high intensity session consisted of intermittent training loads made up of five sets of four repetitions of sixty-second work intervals (HR of 149-186 bt·min<sup>-1</sup>). The moderate intensity session consisted of unvarying type of exercise (HR of 138-167 bt·min<sup>-1</sup>). Measurements were made at rest before, 13-15, and 37-39 hours after the training session. In rebreathing tests ventilatory sensitivity to CO<sub>2</sub> and HR response sensitivity to normocapnic hypoxia were measured. Fast kinetics of ventilation, oxygen uptake, CO<sub>2</sub> production and the heart rate were measured in a 5-min standard power test (0.7 VO<sub>2</sub> max, 5 min, transition from 25 w) and in a 6-min test (1.12 ± 0.11 VO<sub>2</sub>max).

**Results:** We found that a training session of high intensity resulted in a significant decrease in sensitivity to hypercapnia, an increase in CP sensitivity to hypoxia, a decrease in CP fast kinetics and stability of peak response 13-15 hours after the session vs. baseline. Mean power in a 6-min maximum test decreased, which was mainly determined by a decrease in mean power during the first 3 min and utilization of VO<sub>2</sub>max for a 6-min test. Moderate intensity of a training session resulted in an increase in ventilatory sensitivity to hypercapnia whereas sensitivity CP to hypoxia and fast kinetics remained unaffected.

**Conclusions:** These results suggest that not only CP chemosensitivity to hypoxia but also CP chemosensitivity to hypercapnia are variable in high intensity endurance training. The variability related to the effect of fatigue in the recovery phase (up to 15-15 hours) after strenuous training sessions.

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### Corresponding author:

Prof. dr hab. Viktor Mishchenko  
 Gdansk University of Physical Education and Sport  
 Kazimierza Gorskiego Str. 1, 80-336 Gdansk  
 Phone: +48 (58) 5547459  
 E-mail: vmishch@awf.gda.pl

## Introduction

It observed that humans have several physiological adaptations to high physical activity in high performance athletes [1, 2, 3]. Some of these physiological responses include a change in ventilatory and heart rate responses to hypoxia and hypercapnia [4-12]. A few studies investigated prolonged effects of exercise training on cardiopulmonary (CP) chemosensitivity and fast kinetics [13-19]. These results suggest that ventilatory chemosensitivity to hypoxia is more variable by endurance training and detraining than that to hypercapnia [5, 12, 15, 20, 21, 22]. Although there appears to be consensus in the literature that endurance-trained individuals are more prone to have lower resting responses to hypercapnia, some previous studies have found little difference between athletic and untrained subjects. Recently, we have found that fatigue at the end part of prolonged high intensity cyclic exercise is connected with a decrease in CP fast kinetics and hypercapnic sensitivity [23, 24]. It is well recognized that fatigue from strenuous physical exercise is a complex phenomenon in which peripheral, metabolic and central components may be observed [2, 3, 25, 26]. We suppose that fatigue accumulation for one or several consecutive days of high intensity exercises (typical of endurance sports training) may be connected with delayed effects on CP responsiveness in the recovery phase. CP responsiveness changes that result from high intensity of endurance training sessions might be an integral reflection of overall fatigue of high performance athletes [23]. Sports specific CP responsiveness changes are accumulated through a proper influence of metabolic and cardiovascular stress [7]. The lack of agreement in the literature on the influence of exercise upon CO<sub>2</sub> sensitivity can probably be accounted for by inter subject variability, together with differences in methodology, protocol, selection of subjects and the stage of recovery in training. We believe that our data have rationalized some of these inconsistencies by identifying an important factor in determining the influence of endurance sport training upon hypercapnic and hypoxic sensitivity. It is also related to fast kinetics changes in recovery stages. Oxygen uptake fast kinetics and CP peak of response during high intensity exercise is critical to endurance capacity and increase in sports specific training [27-31]. There are no investigations of CR responsiveness induced by fatigue after strenuous training sessions. In this study, it is hypothesized that a high performance athlete's fatigue, induced by a vigorous training session, would be related to a decrease in CP response hypercapnic sensitivity and primary kinetics. In addition, we assumed that CP response kinetics plays a major role in the variation of high performance athletes in power events such as 2000 meter rowing. Understanding CP response to intense exercise related to postponed fatigue accumulation may be beneficial in designing training regimes for elite athletes competing in such events. The purpose of this study was to determine the fatiguing effect of a strenuous training session and a moderate intensity session on the CP primary kinetics, peak of response and chemosensitivity to CO<sub>2</sub> and hypoxia.

## Material and methods

*Subjects.* Eleven high performance (national level) male rowers participated in this study [age 21.8 ±1.7 (range 18-25 years), 89.3 ±2.0 kg, 190.1 ±1.7 cm, VO<sub>2</sub> max 67.9 ±1.1 ml·kg<sup>-1</sup>·min<sup>-1</sup>]. All subjects provided informed written consent prior to participation in the research protocol, which was in accordance with legal requirements. The subjects refrained from ingesting nutritional supplements, ergogenic aids, and medications during the course of the study. The training loads of the research protocol were similar to the usual training program for these athletes. The exercise tests were conducted on a rowing ergometer (Concept II, Morrisville, Vermont, USA) with which the subjects were familiar.

*Procedures.* The study was performed during the preparatory stage of year's cycle of training and involved of three steps: the first one – a study of effects related to a training session of moderate intensity (last day of a week's training microcycle); the second step – effects of a high intensity session (the first day of a week's microcycle); and the third step – an impact of a high intensity session on values of peak response measured in the first day of the following week. The subjects refrained from strenuous training for 72 hours prior to testing. Primary measures in rest (sitting position) and in exercise tests were made after a 24-hour recovery period prior to testing. In

addition, subjects were instructed to consume a carbohydrate rich diet in the days preceding the testing sessions. To obtain the informed consent, the subjects were instructed on the experimental protocol. The training session of moderate intensity (subjectively light) consisted of an unvarying type of exercise intended to improve the rowing technique (motor coordination) (85-90 min at HR of 138-167  $\text{bt}\cdot\text{min}^{-1}$ ). Measurements were made at rest (sitting position) before and 13-15 hours after the training session finishing point. The high intensity session (*subjectively* exhaustive) consisted of intermittent training loads, which consisted of five sets of four repetitions of sixty-second work intervals (110-118 min at HR of 149-186  $\text{bt}\cdot\text{min}^{-1}$ ). Recovery between repetitions was 2.5 minutes; the break between sets was 6 minutes. Data were collected in rest (sitting position) and in exercise tests at three points: prior to a strenuous exercise session, 13-15 hours and 37-39 hours after the high intensity session. All data were collected under identical conditions relative to ambient temperature (20-23°C) and relative humidity (45-55%).

*Measurements and exercise tests.*  $\text{VO}_{2\text{max}}$  in each subject was determined 5-7 days earlier by using a rowing ergometer with incremental loading.  $\text{VO}_2$  derived during maximal exhaustive exercise was considered  $\text{VO}_{2\text{max}}$  when two of the following criteria were met: identification of a plateau in  $\text{VO}_2$  with an increase in power output (150 ml  $\text{VO}_2$  increase) and respiratory exchange ratio near to 1.0. In rebreathing tests ventilatory sensitivity to  $\text{CO}_2$  ( $\Delta V_E/\Delta \text{PACO}_2$ ) and HR response sensitivity to normocapnic hypoxia ( $\Delta \text{HR}/\Delta \text{SaO}_2$ ) were measured at standard rest. The subjects were sitting comfortably in chairs. Subjects rebreathed a gas mixture of 7%  $\text{CO}_2$  in 50-70%  $\text{O}_2$  from a bag (5–6 liters) in a box for 3–4 min [32]. Hypercapnic sensitivity was assessed as the slope of the line (S) determined by linear regression relating  $V_{\text{ETCO}_2}$  to  $V_E$ . Resting hypoxic sensitivity was measured by using a progressive isocapnic hypoxic test [33]. A rebreathing system similar to that in hypercapnic test was used. During rebreathing tidal volume ( $V_T$ ), minute lung ventilation ( $V_E$ ), heart rate (HR) and arterial oxygen saturation ( $\text{SaO}_2$ ) were continuously determined.  $\text{SaO}_2$  was measured on the tip of the left forefinger during rebreathing by a pulse oximeter (OLV-1200). Hypoxic sensitivity was evaluated by a rise in lung ventilation and HR in progressive blood deoxygenating. Changes in the speed of CP response Pre and Post training sessions were measure by fast kinetics of ventilation ( $T_{50}V_E$ ), oxygen uptake ( $T_{50}\text{VO}_2$ ), carbon dioxide production ( $T_{50}\text{VCO}_2$ ) and heart rate ( $T_{50}\text{HR}$ ) in a 5-min standard power test (0.7  $\text{VO}_2$  max, 5 min, transition from 25 w) and in a 6-min test ( $1.12 \pm 0.11 \text{VO}_2$  max). Monoexponential function, breath by breath with 5 s stationary averages throughout the test protocol were used (Jaeger Oxycon Alfa, Germany; Polar Accurex Plus).

The values of peak response were measured at additional loads in the form of 30 s increase of load power (in 1/3) by increasing the pace of rowing at the end of sustained exercise on a rowing ergometer (30 min, 79-85%) before high intensity training and immediately (30-60 min) after it. The peak of response was determined according to maximum  $V_E$ ,  $\text{VO}_2$  and HR in one of 10-s fragments of 30-s "speeding up". Effects of high intensity session were evaluated in the second step of studies by working out capacity changes (Post vs. Pre) as demonstrated during a 6-min maximal work rowing ergometer test (mean value for 6 min, for first 3 min and for second 3 min of the test).

*Statistical analysis.* The values were expressed as means and SD. The differential changes in the parameters between Pre and Post training sessions were compared by using two-way repeated-measures ANOVA. Differences in the parameters (Pre, Post) within each group were determined by using the Wilcoxon test. The level of significance was set at 0.05. Pearson's correlation coefficient was used to evaluate the relationship between characteristics of physical capacities during the 6-minute rowing test.

## Results

In studies of trace changes (after-action) provoked by moderate intensity (subjectively light) training loads (85-100 min at HR of 138-156  $\text{bt}\cdot\text{min}^{-1}$ ) measurements were made at rest (sitting position) 13-15 hours after the training session' finishing point (Table 1).

As follows from Table 1, the level of lung ventilation 13-15 hours after the training session was higher (13.8%) than before the session.

Changes in hypercapnic and hypoxic CP sensitivity 13-15 hours after training load of moderate intensity are presented in Table 2.

As is obvious from Table 2, sensitivity to hypercapnia in the recovery phase after (13-15 hours) the training session of moderate intensity increased. One should take notice of a significant expression of the decrease in the threshold response to a hypercapnic stimulus rather than the change in the rate of response intensification. A decrease in the response threshold resulted in an increase in lung ventilation at standard PA CO<sub>2</sub> = 50 mm Hg from 24.6 ± 1.3 L·min<sup>-1</sup> before the training session to 71.5 ± 3.7 L·min<sup>-1</sup> 13-15 hours after session (p < 0.01). There were no changes in time-related CP response characteristics – T<sub>50</sub> VO<sub>2</sub>, T<sub>50</sub> V<sub>E</sub> and T<sub>50</sub> HR.

Hypoxic sensitivity of rowers' CP responses did not change after a moderate training session. At the same time, the value of rest oxygen uptake, lung ventilation and resting HR after (13-15 hours) the training session significantly increased.

Fast kinetics after a high intensity training session was analyzed by evaluation of changes in peak CP response to 30 s increase in power during sustained exercising on a rowing ergometer (30 min) and T<sub>50</sub> CP response in the initial part of the test exercises.

Table 1. Characteristics of oxygen uptake, lung ventilation and HR 13-15 hours after moderate intensity training session, n = 9, M ±SD

Characteristics	Before training session	After training session
Pulmonary ventilation, L·min <sup>-1</sup>	8.9 ± 0.8*	12.2 ± 1.0*
HR, bt·min <sup>-1</sup>	51.5 ± 1.0	54.0 ± 1.2
Oxygen uptake level, ml·min <sup>-1</sup> ·kg <sup>-1</sup>	4.9 ± 0.2	5.9 ± 0.7
Respiratory exchange ratio	0.85 ± 0.02	0.89 ± 0.04

\* Significant at p < 0.05

Table 2. Change in CP hypercapnic and hypoxic response sensitivity in rowers (n = 9) in the recovery phase (13-15 hours) training session of moderate intensity, M ±SD

Characteristics	Before training session	After training session
Sensitivity of lung ventilation to hypercapnia (ΔV <sub>E</sub> /ΔP <sub>A</sub> CO <sub>2</sub> ), L·min <sup>-1</sup> ·mm Hg <sup>-1</sup>	1.39 ± 0.12*	1.86 ± 0.19*
Threshold of lung ventilation to hypercapnia (point of apnea - „B”), mm Hg	33.9 ± 1.6*	21.8 ± 1.9*
Sensitivity of lung ventilation to hypoxia – (ΔV <sub>E</sub> /ΔSaO <sub>2</sub> ), L·min <sup>-1</sup> ·% <sup>-1</sup>	0.231 ± 0.08	0.265 ± 0.09
Sensitivity of heart rate to 1 % of SaO <sub>2</sub> decline (ΔHR/ΔSaO <sub>2</sub> ), bt·min <sup>-1</sup> ·% <sup>-1</sup>	0.89 ± 0.08	0.81 ± 0.07
T <sub>50</sub> VO <sub>2</sub> , s	28.8 ± 1.8	28.1 ± 2.0
T <sub>50</sub> HR, s	19.7 ± 1.4	20.8 ± 1.4
T <sub>50</sub> V <sub>E</sub> , s	42.9 ± 3.8	40.9 ± 3.9

\* Significant at p < 0.05

An analysis of percentage changes of the peak of response to short-term (30 s) increases in load power between the 4<sup>th</sup> min and 30<sup>th</sup> min indicated a decrease in V<sub>E</sub>, VO<sub>2</sub> and HR responses at the end of the 30 min test exercise (Table 3).

Study of changes of the peak of response to short-term (30 s) increases of load power (Table 3) indicated a reduction in V<sub>E</sub>, VO<sub>2</sub> and HR responses from the beginning to the latest part of sustained load to exhaustion. This reduction coincided, as a rule, with a decrease in the speed of 30 s increases in load power.

The results demonstrated a reduction of fast kinetics of CP and gas exchange responses according to characteristics of half-period (T<sub>50</sub>) response concerned of high intensity training session of an interval type (13-15 hours after it) (Table 4).

Table 3. Changes in the peaks of lung ventilation, oxygen uptake and HR response (in percentage) induced by 30 s increase in power after a high intensity training session (25-55 min after it) vs. before the training session. Increasing consisted in 1/3 by rising of pace of rowing between the 4<sup>th</sup> min and 30<sup>th</sup> min of sustained exercising on a rowing ergometer (30 min, 79-85%), n = 9, M ±SD.

Characteristics	Time period of measurements	
	before training session	after training session
Lung ventilation, %	4.9±0.6*	0.1±0.6*
Oxygen uptake, %	6.9±1.0*	1.5±0.8*
Heart rate, %	5.5±0.4*	0.2±0.5*

\* Significant at  $p < 0.05$

Table 4. The effects of a high intensity training session on fast kinetics of VO<sub>2</sub>, VCO<sub>2</sub>, V<sub>E</sub> and HR responses (T<sub>50</sub> at exercise 0, 7 VO<sub>2max</sub>) in high performance rowers (n = 11), M ±SD

Characteristics	Time period of measurements	
	before training session	after training session
T <sub>50</sub> VO <sub>2</sub> , s	28.3±1.7	31.9±1.9
T <sub>50</sub> VCO <sub>2</sub> , s	46.2±3.8	53.1±4.2*
T <sub>50</sub> HR, s	19.5±1.2	22.9±1.3
T <sub>50</sub> V <sub>E</sub> , s	43.7±3.7	50.3±3.9*

\* Significant differences at  $p < 0.05$

As follows from Table 4, the fast part of lung ventilation response showed the most distinct decline compared fatigue related to a high intensity training session.

The fast kinetics of gas exchange and CP responses in about 2000m race simulation (6 min rowing test) decreased after a high intensity training session (measurements at 30-45 min after its end) (Table 5).

As follows from Table 5, the fast part of lung ventilation and VCO<sub>2</sub> response showed the most distinct decline (T<sub>50</sub> increase) after the training session. T<sub>50</sub> for VO<sub>2</sub> also significantly increased.

Table 5. Change of fast kinetics (half-period of response – T<sub>50</sub>) of gas exchange and CP responses in simulated rowing distance (6 min rowing test, 1.12 ±0.11 VO<sub>2max</sub>) as a result of a high intensity training session (30-50 min before and 30-45 min after) in highly skilled rowers (n = 9), M±SD

Characteristics	Six min rowing test	
	before training session (1.15±0.10 VO <sub>2max</sub> )	after training session (1.10±0.13 VO <sub>2max</sub> )
T <sub>50</sub> VO <sub>2</sub> , s	24.1 ±1.3	29.6 ±1.4*
T <sub>50</sub> VCO <sub>2</sub> , s	33.0 ±2.7	41.7 ±3.7*
T <sub>50</sub> HR, s	16.1 ±1.3	19.9 ±1.2
T <sub>50</sub> V <sub>E</sub> , s	33.7 ±2.7	39.6 ±2.8*

\* Significant differences at  $p < 0.05$

Results showed that a reduction in the mean power in a 6-min maximum test related to fatigue of hard training session (after 13-15 hours) was also observed (Fig. 1).

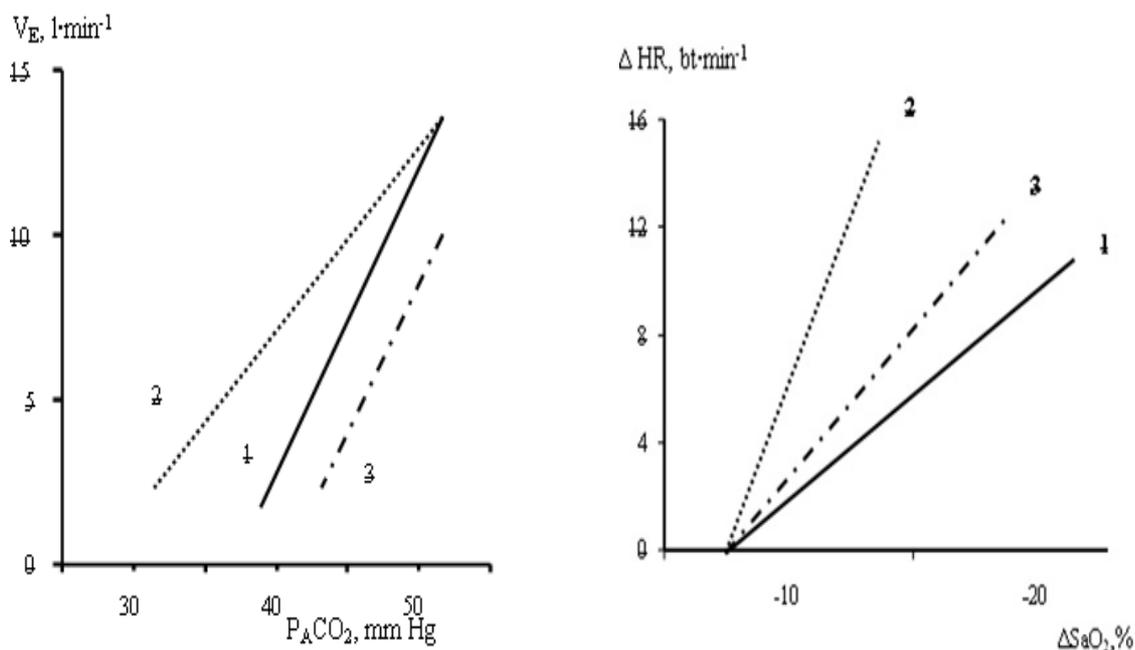


Fig. 2. Changes of lung ventilation response sensitivity to increasing  $P_A\text{CO}_2$  (rebreathing) and HR sensitivity to decreasing blood oxygenation in skilled athletes 13-15 and 37-39 hours after high intensity training session. 1 – Initial value; 2 – after 13-15 hours; 3 – after 37-39 hours.

This decline of the mean power in a 6-min maximum test was mainly determined by a decrease in the mean power during the first 3 minutes of a 6-min test. 37-39 hours later characteristics of work capacity were close to their complete recovery, whereas utilization of  $\text{VO}_{2\text{max}}$  remained decreased. The decline of the mean power during a 6-min test was related to the degree of  $\text{VO}_{2\text{max}}$  utilization ( $r = 0.51$ ,  $p < 0.05$ ) and correlated with the value of blood lactate concentration increase after the test ( $r = 0.49$ ;  $p < 0.05$ ). After a high intensity training session the peak of lung ventilation response during a 6-min test decreased by  $6.7 \pm 1.1\%$  and was reached  $0.71 \pm 0.10$  min later ( $p < 0.05$ ). The peak of HR response was also reached later (by  $0.31 \pm 0.07$  min).

Changes of hypercapnic and hypoxic sensitivity characteristics of CP responses related to trace effects of high intensity training session are demonstrate in Table 6.

Table 6. Changes of hypercapnic and hypoxic sensitivity characteristics of CP responses of rowers 13-15 hours after a high intensity training session,  $n = 11$ ,  $M \pm \text{SD}$

Characteristics	Period of measurements	
	before training session	after training session
Lung ventilation level at $P_A\text{CO}_2$ 50 mm Hg ( $V_{E50}$ ), $\text{L} \cdot \text{min}^{-1}$	$13.7 \pm 0.4$	$14.7 \pm 0.3$
Ventilatory response threshold (point of apnea, "B" parameter), mm Hg	$36.4 \pm 0.3^*$	$31.8 \pm 0.4^*$
Sensitivity ventilatory response to hypercapnic - $\Delta V_E / \Delta P_A\text{CO}_2$ , $\text{L} \cdot \text{min}^{-1} \cdot \text{mm Hg}^{-1}$	$1.43 \pm 0.11$	$1.34 \pm 0.10$
Sensitivity ventilatory response to hypoxic stimulus - $\Delta V_E / \Delta \text{SaO}_2$ , $\text{L} \cdot \text{min}^{-1}$ by 1 %	$0.180 \pm 0.05^*$	$0.298 \pm 0.06^*$
Increase in heart rate by 1 % of $\text{SaO}_2$ ( $\Delta \text{HR} / \Delta \text{SaO}_2$ ) decline, $\text{bt} \cdot \text{min}^{-1} \cdot \%^{-1}$	$0.72 \pm 0.12^*$	$1.44 \pm 0.14^*$
Increase (delta) in lung ventilation subsequently to standard decrease in blood oxygenation (-12%) ( $\Delta V_{E84}$ ), $\text{L} \cdot \text{min}^{-1}$	$7.80 \pm 0.40^*$	$10.20 \pm 0.35^*$

\* Significant differences at  $p < 0.05$

As is clear from Table 6, a training session of high intensity (subjectively heavy) resulted in a significant increase in the reactivity to hypoxia reflecting enhancement of sensitivity to it 13-15 hours after the session.

Differences in relation of lung ventilation sensitivity to hypercapnia after one (13-15 hours) and two (37-39 hours) days from the ending point of training sessions are illustrated by Figure 2 (the left part).

Results showed that disposition of the line returns to the initial level in case of an increase in threshold response to CO<sub>2</sub>. Complete recovery of HR response sensitivity to hypoxia did not occur even 37-39 hours after the session (Fig. 2, the right part).

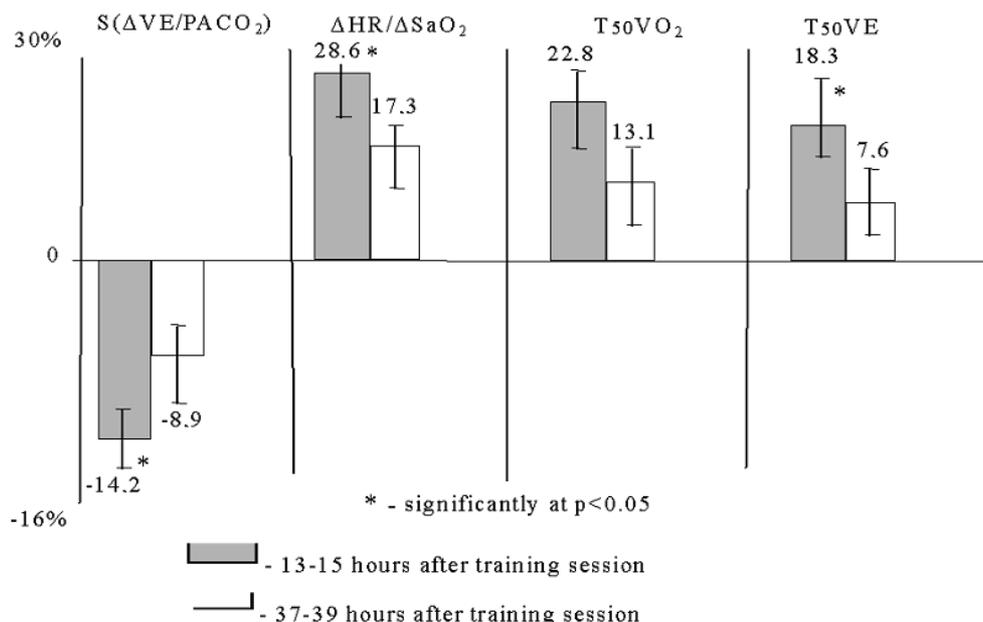


Fig. 2. Changes (percentage to baseline) in ventilatory response to CO<sub>2</sub>, heart rate response to hypoxia, primary kinetics (0.7 VO<sub>2</sub>max) 13-15 hours and 27-39 hours after strenuous training sessions in high performance rowers (M ±SD)

## Discussion

In the present study, we found that 1) sensitivity to hypercapnia in the recovery phase after (13-15 hours) an endurance training session of moderate (subjectively light) intensity increased, whereas sensitivity to hypoxia and CP fast kinetics remained unaffected in comparison to the baseline (before the training session); 2) a training session of high intensity (subjectively exhaustive) resulted in a significant decrease in sensitivity to hypercapnia, an increase in CP sensitivity to hypoxia, a decrease in CP fast kinetics and stability of peak response 13-15 hours after the session. Complete recovery of HR response sensitivity to hypoxia did not occur even 37-39 hours after the session; 3) mean power in a 6-min maximum test after 13-15 hours of a hard training session decreased. It was mainly determined by a decrease in the mean power during the first 3 minutes of a 6-min test. 37-39 hours later characteristics of work capacity were close to their complete recovery, whereas utilization of VO<sub>2</sub>max remained decreased.

Most studies have shown that a decrease in the ability to produce power is identified as a manifestation of fatigue, and a trace of fatigue may be observed for extended periods [2, 3, 23, 26]. In sports with high-energy demands, an understood limitation of endurance capacity is fatigue-induced exhaustion of metabolic reserves [2, 3]. It is known that stability of CP response in high intensity exercises is higher in athletes [2]. It may be related not only metabolic resources but CP responsiveness stability, too. The results showed that CP responsiveness trends to dissimilar changes in the recovery phase depend on the intensity of endurance training sessions. Previous

studies showed some chronic effects of ventilatory responses to hypoxia and hypercapnia related to enduring physical training [4, 5, 7, 8, 9, 10, 11, 12]. A training session of high intensity resulted in a decrease of sensitivity to hypercapnia and fast kinetics, whereas a moderate intensity session resulted in an increase in CO<sub>2</sub> sensitivity, that is a training session of moderate intensity can have a stimulating effect on ventilatory sensitivity to carbon dioxide. This may be due to the previously shown increased sensitivity to CO<sub>2</sub> during mild physical activity [31, 34]. CP fast kinetics and hypoxic sensitivity after a moderate intensity session remained unaffected vs. baseline. Some physiological responses include an increase in ventilatory responses to hypoxia, a decreased or increased response to hypercapnia in athletes. The data concerning this issue are contradictory [4, 7, 8, 9, 10, 11]. In some cases, a decrease of ventilatory chemosensitivity to hypercapnia was found in athletes, whereas in others only a decrease of hypoxic sensitivity takes place. The discrepancy in observations may be attributable to the training intensity, prolonging of training, age involvement in training and athletes' performance. A few studies investigated the effects of intermittent hypoxic exposure combined with exercise training on ventilatory chemosensitivity in humans, for example studies by Katayama et al. [17, 21]. An increase in hypoxic ventilatory response showed at intermittent exposure to altitude combined with endurance training for several weeks [17]. In another study, intermittent hypoxic exposure for six consecutive days with exercise training did not significantly increase the hypoxic and hypercapnic ventilatory response, whereas without exercise training the hypoxic ventilatory sensitivity did increase [21]. Oxygen uptake fast kinetics increases in long-lasting sports specific training [7, 28, 30, 31], whereas it changes related to fatigue are insufficiently researched. For example, a decrease in CP fast kinetics showed after exhaustive ergometric exercise [23]. So a decline of both hypoxic and hypercapnic drives of breathing as well as the response of carotid sinus baroreceptors at rest has been typical of persons training endurance in many years. The common character of changes of the described physiological responses to various irritants under the influence of sports training may be explained by the fact that the role of chemo- and baroreceptor impulse is interrelated by the general breathing function and hemodynamics of body oxygen delivery [2, 22, 35, 36, 37]. During analysis of response sensitivity changes in the process of training one should bear in mind general regularity of control improvement in the system associated with afference "narrowing" in the process of the functional system adaptation to a specific factor [38]. The total amount of afferent impulse necessary for efficient system functioning is reduced.

One may suppose that in the case of athletes involved in intensive training the greatest changes of CP responsiveness should take place during strenuous sports exercises and after them. Athlete's working capacities largely depend upon how long and how much of body oxygen demand above its current uptake it may tolerate. Taking into account that athletes are under stress respiratory homeostasis for a long time, can be assumed that the shifts of homeostatic parameters (as well as the level of strain in regulatory mechanisms for their maintenance) tend to become habitual along with fitness development. That is, sensitivity to shifts of pCO<sub>2</sub>, pH and PaO<sub>2</sub> may be transformed to some extent. This determines enhancement of reserve power of the homeostasis maintenance in endurance athletes. There is a limited data about the mechanism of changes in chemosensitivity for enhancement of respiratory homeostasis reserve during training. This data yields a conclusion that the efficiency of the oxygen delivery system during intensity endurance training may well be evaluated according to the degree of homeostatic control mechanism strain. The latter after training sessions may be regarded as chemosensitivity and fast kinetics changes and considered as related to the assessment of fatigue. An increase in CP hypoxic sensitivity after a high intensity training session tends to more reflect the state of peripheral chemosensitive formations, whereas a decrease in sensitivity to hypercapnia – central medullar chemoreceptors [37,39]. At the same time, sensitivity to hypoxia during a physical load also reflects the degree of cerebral anaerobic metabolism, which induces an increase in H<sup>+</sup> concentration in the area of the respiratory center [40]. Due to the above changes of sensitivity to hypoxia largely reflect the long-term accumulation of impacts. That is why they are more associated with aerobic output than other factors, whereas the changing of sensitivity to hypercapnia reflects other characteristics of the body functional state. It is known that sensitivity of CP responses to CO<sub>2</sub> is the fastest and the

most efficient mechanism of homeostatic regulation. Therefore, it may vary in high intensity physical training. It is clear if one takes into account the dynamic role of regulation of fast CO<sub>2</sub> removal at high intensity exercises and fast CO<sub>2</sub> accumulation after exercises [41-45]. To explain the regulation of O<sub>2</sub> delivery and metabolites (CO<sub>2</sub>) removal during a physical load, characteristics of the apparatus of this process management as the “regulator of variable enhancement” are sometimes used [46]. In this way, an attempt is made to explain a seeming conflict between fast and considerable changes of CP responses and stability of chemoreceptor stimuli during transition from rest to muscular activity. It may also be related to chemosensitivity and fast kinetics changes in the recovery phase of a training session [41, 47, 48].

## Conclusion

These results suggest that not only CP chemosensitivity to hypoxia but also CP chemosensitivity to hypercapnia are variable in high intensity endurance training. Temporary changes of CP chemosensitivity and fast kinetics in the recovery phase of different intensity of training sessions may change the training effect of the next training session. Trainability of athletes is strictly connected with value, specific and prolongation of characteristics of fatigue after a training session. Currently it is difficult to answer whether the above-described features of CP responsiveness are preferentially associated with trainability endurance athletes in the process of long-term impact of strenuous sports training. Checking of chemosensitivity and fast kinetics in the recovery phase (13-15 hours) after a high intensity endurance training session may be useful for evaluation of temporary changes in CP responsiveness related to fatigue.

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