Changes Related to Fatigue in Cardiorespiratory Response
Sensitivity to Hypoxic and Hypercapnic Stimulation during Strenuous Physical Load

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Results: The results showed an increase in respiratory response sensitivity to hypoxia and a decrease in sensitivity to acidosis stimulus of respiration (CO2–H+) at the end of a strenuous load in presence of fatigue. Ventilatory response to hypoxic stimulus increased reliably before the end of work. At high intensity of loads during increment hypercapnia even in highly trained athletes the inhibition of ventilatory response has been noted. Conclusions: The data provide additional grounds for correction of fatigue by means of regulating the character and the intensity of physical loads in the process of endurance training. It has been shown that changes in sensitivity of CRS responses and other aspects of reactive features are of importance for special work capacity manifestations.

Keywords
fatigue, cardiorespiratory response, hypoxia, hypercapnia, training load

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Introduction

In order to deeper understand the significance of control factors for the limits of endurance, related to fatigue, changes in cardiorespiratory system (CRS) reactive features during and after strenuous physical loads were analyzed [1, 2, 3, 4]. The resistance of the system response sensitivity to the fatigue constitutes one of the most important aspects of functional capacity changes in the process of endurance training [5, 6, 7, 8]. It is well known that fatigue induced by a strenuous physical load represents a complex phenomenon including peripheral, central (central and autonomic nervous system control) and metabolic factors [4, 5, 9]. We have proceeded from the fact that the changes in physiological reactivity of leading for the type of activity body system are one of the manifestations of control (informational) fatigue. In this regard the character of changes in CRS reactive features during fatigue being distinctively expressed in the process of athletic training has been estimated. The aim was to study changes in sensitivity of CRS responses of skilled athletes caused by fatigue during a strenuous physical load.

Material and Methods

Changes in response sensitivity to hypoxic and hypercapnic stimuli were analyzed in a group of high performance (national team) rowers (n=12) aged 18–22 years during exercising on “Concept-II” rowing ergometer. Two types of loads subjected to modeling were used: a sustained intensive load (of uniform character) at high oxygen consumption (78-83% of VO$_2$max), on the one hand, and a relatively easier load (49-62% of VO$_2$max) performed within 5 minutes, on the other hand. Besides, under standard conditions of progressively increased power of load (progressively increased every 5 min to power of VO$_2$max value) the bond of sensitivity level of CRS responses (according to major values) was determined. Ventilatory and heart rate (HR) responses to hypoxia were analyzed by a hyperbolic relationship. Hypercapnic normoxic ventilatory response and CO$_2$ threshold and sensitivity (by the modified Read rebreathing technique) were measured [10, 11, 12].

Results

Hypoxic drive (stimulus) changes were estimated at the first stage of studies. Studies have demonstrated an increase in sensitivity to a hypoxic stimulus (inspiration of hypoxic gas mixture) along with fatigue development during sustained heavy loads. Respective data are presented in Table1.

Tab. 1. The influence of fatigue on an increase (∆) in pulmonary ventilation (V$_E$) during short-term (30 s) inspiration of hypoxic gas mixture (14.0-14.2% of O$_2$ in nitrogen) under sustained strenuous load of uniform character (n=8), М±SD

<table>
<thead>
<tr>
<th>Duration of load performed to exhaustion (in % of its total individual duration)</th>
<th>9-17</th>
<th>24-32</th>
<th>42-51</th>
<th>65-74</th>
<th>86-98</th>
<th>P (t-test)</th>
</tr>
</thead>
<tbody>
<tr>
<td>∆ V$_E$, %</td>
<td>118.1±3.1</td>
<td>121.2±2.9</td>
<td>122.1±3.0</td>
<td>133.0±3.2</td>
<td>124.6±2.8</td>
<td>1,2,3-4, 4-&lt;0.05</td>
</tr>
<tr>
<td>P$_A$O$_2$, mm Hg</td>
<td>67.7±1.4</td>
<td>67.9±1.3</td>
<td>68.3±1.5</td>
<td>72.8±2.1</td>
<td>68.2±1.1</td>
<td>1-4&lt;0.1</td>
</tr>
<tr>
<td>∆P$_A$CO$_2$, mm Hg</td>
<td>-1.4</td>
<td>-2.1</td>
<td>-2.1</td>
<td>-2.8</td>
<td>-2.5</td>
<td>1-4&lt;0.1</td>
</tr>
</tbody>
</table>
As is obvious from the Table, ventilatory response to a hypoxic stimulus increased reliably before the end of work. In some athletes such an increase exceeded by 1/3 the values of pulmonary ventilation accretion to hypoxia at the beginning of load. Significant individual differences in changes of sensitivity to hypoxia were noted in the process of heavy load performance. The probability of a greater decrease in this sensitivity enhanced with an increase in the total duration of work to exhaustion. Besides, a direct relationship between the initial level of hypoxic sensitivity and the degree of its increase at the end of load performance was revealed ($r=0.61; p<0.05$).

Of special interest is the change in the sensitivity of response to the acidosis stimulus caused by fatigue. It may be estimated according to the response to hypercapnia. Measurements of this type demonstrated that according to mean values during the most part of sustained strenuous physical load inclination of the line of dependence $V_E-P_{A\text{CO}_2}$ (parameter S, $\Delta V_E/\Delta P_{A\text{CO}_2}$) did not change significantly (Table 2).

Tab. 2. Change in sensitivity and stability of ventilatory response to increasing $\text{CO}_2-\text{H}^+$-stimulus (according to hypercapnia stage) during sustained load (n=11), $\text{M}\pm\text{SD}$

<table>
<thead>
<tr>
<th>Variables</th>
<th>14-20</th>
<th>33-41</th>
<th>54-61</th>
<th>78-81</th>
<th>92-98</th>
<th>$p(t\text{-test})$</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sensitivity of response ($\Delta V_E/\Delta P_{A\text{CO}_2}$), L·min⁻¹mm Hg⁻¹</td>
<td>1.57±0.17</td>
<td>1.42±0.21</td>
<td>1.38±0.18</td>
<td>1.41±0.22</td>
<td>1.32±0.31</td>
<td>&lt;0.05</td>
</tr>
<tr>
<td>Response stability according to the level of $V_E$, onset of response sensitivity decline L·min⁻¹</td>
<td>73.3±2.8</td>
<td>88.2±3.7</td>
<td>84.5±3.5</td>
<td>79.1±3.2</td>
<td>71.5±1.9</td>
<td>2.3-5</td>
</tr>
<tr>
<td>Degree of $V_E$ decline from its highest value to that at $P_{A\text{CO}_2}$ 65-75 mm Hg, L·min⁻¹</td>
<td>-</td>
<td>6.1±3.1</td>
<td>2.4±2.9</td>
<td>6.4±3.0</td>
<td>11.2±3.1</td>
<td>3-5</td>
</tr>
</tbody>
</table>

As follows from the Table, the tendency of $\Delta V_E/\Delta P_{A\text{CO}_2}$ decrease at the end of the load relative to the initial part of load was simultaneously observed. In several cases during consideration of individual data either increase or decrease in response sensitivity was noted in the process of the sustained load.

The presented strategies of analysis allowed us to single out the evaluation of lung ventilation response stability. On the whole, the dependence of response stability and sensitivity during the load was negative. At the same time individual manifestations of ventilatory response stability during the sustained load should be noted. Their summarization indicated that the higher the value of response sensitivity in rest, the lower ventilatory response stability during recurrent $\text{CO}_2$ respiration at the end of the load ($r=-0.61$).

We have assumed that at certain regimes of physical loads effects of reactivity increase to the analyzed stimuli of CRS response could be obtained. In order to determine the intensity of such a load, sensitivity of ventilatory response was determined at various intensities of 5-min load of cyclic character (measurements were made at the end of the load). These data are presented in Table 3.
Tab. 3. Sensitivity of lung ventilation response of athletes to hypoxic and acidosis stimuli during different intensity of the physical load, M±SD

<table>
<thead>
<tr>
<th>Characteristics</th>
<th>Relative intensity of load, in % of VO₂max</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>35-39%</td>
</tr>
<tr>
<td>Sensitivity to hypercapnia – increase of lung ventilation by 1 mm Hg of PₐCO₂ (ΔVE/ΔPₐCO₂) increase, L·min⁻¹·mm Hg⁻¹</td>
<td>1.67±0.13*</td>
</tr>
<tr>
<td>Sensitivity to hypoxia – parameter of &quot;A&quot; - dependence VE-PₐO₂, c.u.</td>
<td>259±11*</td>
</tr>
</tbody>
</table>

* significant differences at p<0.05

The above presented data provide additional grounds for correction of fatigue by means of regulating the character and the intensity of physical loads in the process of athletic training. At high intensity of loads during hypercapnia (rebreathing during reaching PₐCO₂ equal to about 55-65 mm Hg) even in highly trained athletes the inhibition of ventilatory response – VE “plateau” or even decline – was noted. In less trained persons the above was observed at lower values of PₐCO₂ (46-56 mm Hg) which is indicative of their lower stability of ventilatory response to hypercapnia during fatigue induced by physical load. An analysis of individual data during heavy loads (VO₂ 67–72% and 82–86% VO₂max) showed a positive correlation between sensitivity to hypercapnia and the ratio of ventilation to CO₂ excretion (r=0.69; p<0.05).

A comparison of the ratio of CO₂ ventilatory equivalent (VE/VCO₂) to sensitivity to CO₂ during light and heavy loads demonstrated its increase during the latter one. At higher sensitivity to CO₂ at the end of the load a greater efficiency of “excessive” CO₂ excretion was maintained resulting in an increased level of metabolic acidosis respiratory compensation. These findings accentuate the importance of maintaining sensitivity to CO₂–H⁺ stimulus at the background of fatigue for improvement of work capacity. At the same time they indicate the possibility to determine the level of load intensity which can stimulate enhancement of sensitivity to hypercapnic stimulus and, perhaps, body reactivity of CRS.

Discussion

During an analysis of the impact of sustained heavy load of uniform character upon sensitivity of vegetative centers, one should take into account a possible inhibitory effect of hypoxia upon CNS and its inhibitory action on central structures of respiratory center [12, 13, 14]. Physical loads may enhance this effect of hypoxia [14, 15]. A combination of two factors peculiar for a competitive activity in sport, namely – high intensity and long duration of the load tends to increase the probability of the above. In endurance trained athletes the reduction of the ventilatory response to hypoxia at the end of load performance was rarely observed; according to average data the response in this period slightly increased. The reduction of the respiratory system response sensitivity to hypoxia and its greater stability are an important factor of work capacity under conditions of sustained, strenuous physical load [9, 16].

Determination of the initial (the first 10 s) and the total ventilatory response to hypoxia is an important additional factor of sensitivity to hypoxia analysis. Typical data of such an analysis in the process of sustained load demonstrated a lower initial part of ventilatory response to hypoxia in athletes as compared to the final part of the load [14]. During the whole load it was, as a rule, more stable in athletes with a higher level of fitness than in athletes with a lower level of preparedness for sustained activity. These data indicate that the initial ventilatory response to hypoxia (which is mainly provided by peripheral chemoreceptor) is more stable than its subsequent part which
depends on both central chemoreception and the stability of the integrative function of vegetative centers to hypoxia [17].

An analysis demonstrated that these changes have been largely connected with the dynamics of blood acidosis shifts [17, 18]. An increase in response sensitivity to CO₂–H⁺ stimulus at the background of acidosis is observed, as a rule, during a low level of acidosis as well as at the initial part of the load. Therefore, such an increase in response sensitivity is peculiar for a relatively short total duration of the load of the given intensity [19]. At a high level of acidosis expression as well as at the end of the load the sensitivity of response could decrease significantly [14]. The above mentioned decrease in sensitivity response occurred along with some increase in PₐCO₂. It should be noted that both the inclination of the line of dependence of PₐCO₂ and the level of Vₑ decreased during a physical load. At the beginning of the sensitivity decrease (or the total value of ventilatory response) to hypercapnia PₐCO₂ significantly decreased by the end of the load. During this part of the load individual maximum values of pulmonary ventilation in samples of rebreathing CO₂ were not attained.

These data contribute to a better understanding of important aspects of alteration of the respiratory system reactive features in the process of sustained physical loads which are closely connected with special work capacity manifestation under conditions of such loads. At the same time, they allow determining possible directions and means of correcting such changes related to fatigue. An example of such means may be relatively light physical loads [3, 14]. The grounds for the above are data showing that the initial part of the sustained load is characterized by a tendency to enhancement of sensitivity to CO₂–H⁺ stimulus.

**Conclusion**

Despite individual peculiarities of changing sensitivity of ventilatory response to CO₂ during a sustained load, the findings are indicative of a relative decrease in acidosis stimulus of respiration at the end of the load in the presence of fatigue. On this basis, the accumulation of reactive feature changes has been estimated as a factor of specific training effect formation.

There is every reason to believe that a modification of the respiratory system reactivity to the load, including CO₂–H⁺, determines, to a great extent, changes related to fatigue development. It has been supposed that changes in the sensitivity of CRS responses and other aspects of reactive features are of importance for special work capacity manifestations. Understanding of the essence and the significance of these changes may lay down the foundations for new opportunities of directed correction of fatigue and intensification of the training process.

**References**