

MECHANISM OF THE FAST NEUROGENIC COMPONENT OF THE RESPIRATORY  
RESPONSE TO MUSCULAR WORKI. S. Breslav, G. G. Isaev,  
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The dynamics of the pulmonary ventilation, electrical activity of the intercostal muscles, and composition of the alveolar gas were investigated in 12 healthy men performing measured muscular work and breathing mixtures of different composition. The respiratory response in the initial period of work during inhalation of a hypoxic, hypercapnic gas mixture was greater than when breathing air, but after hyperventilation with oxygen it virtually disappeared. The fast component of the ventilatory response to muscular work is evidently due largely to increased sensitivity of the respiratory center to impulses from chemoreceptors.

KEY WORDS: *Regulation of respiration; chemoreceptors; proprioceptors; respiratory center; muscular activity.*

The mechanisms determining the increased ventilation during muscular work are by no means fully understood despite a century of experimental study. A decisive role in the control of respiration is played by stimuli from chemoreceptors in response to changes in the partial pressures of the bases and the hydrogen ion concentration in the blood and cerebrospinal fluid. However, during muscular activity it often happens that the changes in the gas composition of the arterial blood are not in the same direction, and even if they are, they are so small that they cannot explain the considerable ventilation response [10]. It is important to note that hyperventilation develops after the first few seconds of work, before any chemical changes can have taken place in the arterial blood [5, 8, 9] and for that reason the initial response of the ventilation is called the fast neurogenic component. This component is evidently produced by certain nervous stimuli which play the role of disturbing factor, bringing the respiratory function into line with the current load [4]. Investigations by Soviet physiologists [1-3] have shown the role of receptors of working muscles and also of conditioned reflexes to signals linked with the beginning of work. The role of proprioceptive stimulation of respiration during muscle activity was confirmed later [6-8].

In this investigation the role of afferent impulsion from chemoreceptors in the mechanism of the initial ventilatory response to work was studied. Experiments were carried out in which the subjects performed work against the background of an altered intensity of chemoreceptive stimulation of respiration.

## EXPERIMENTAL METHOD

Twelve healthy men aged 22-35 years, accustomed to the experimental conditions and sitting in a comfortable armchair, turned the pedals of a bicycle ergometer at a steady speed of 60 rpm. The duration of each load was 5 min. The work was done while the subjects breathed either air, pure oxygen (with hyperventilation for 1 min before the work began), or a mixture of 4% CO<sub>2</sub> and 13% O<sub>2</sub> in nitrogen. The subject breathed through a mask. Pulmonary ventilation was recorded on a modified spiograph. The dynamics of the partial pressure of the bases in the alveolar air (P<sub>a</sub>CO<sub>2</sub>, P<sub>a</sub>O<sub>2</sub>), reflecting changes in the gas composition of

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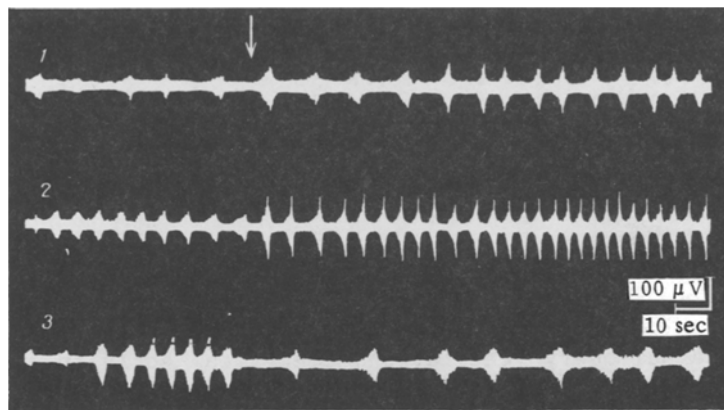


Fig. 1. Responses of electrical activity of external intercostal muscles of muscular exertion (300 kg·m/min) by subjects breathing air (1) or a mixture of 4% CO<sub>2</sub> and 13% O<sub>2</sub> in nitrogen (2) and after voluntary hyperventilation while breathing pure oxygen (3). Arrow indicates beginning of muscular work.

the arterial blood, was determined with the MKh 6202 mass spectrometer. Electrical activity of the external intercostal muscles (EMG), used as the indicator of the afferent signal to the respiratory center, was derived by surface electrodes and recorded after preamplification by the UBP 1-02 amplifier on an N-105 loop oscillograph.

#### EXPERIMENTAL RESULTS AND DISCUSSION

With an average muscular load (30 kg·m/min) and during inhalation of air a distinct increase in the respiratory volume was observed in nearly all experiments, starting from the first inspiration under the load, and this was accompanied by a sharp increase in the electrical activity of the respiratory muscles (Fig. 1). The respiration rate also rose. Ultimately the pulmonary ventilation during the first 10 sec of work was increased on the average by  $5.31 \pm 0.48$  liters/min. This took place despite relatively unchanged values of the partial pressure of O<sub>2</sub> and CO<sub>2</sub> in the alveolar gas, in which the changes began later (Fig. 2).

During the performance of work of the same intensity but against the background of increased chemoreceptor stimulation (inhalation of a mixture of 4% CO<sub>2</sub> and 13% O<sub>2</sub> in nitrogen) the increase in ventilation during the initial period of work was  $11.24 \pm 1.13$  liters/min, i.e., more than twice the increase obtained during the same phase of work but during inhalation of air ( $P < 0.01$ ). Under these conditions the initial component of the ventilatory response to work began before changes in P<sub>a</sub>CO<sub>2</sub> and P<sub>a</sub>O<sub>2</sub> (Fig. 2). Starting from the first inspiration during loading, high-amplitude spikes appeared on the EMG (Fig. 1).

In the experiments in which the subjects breathed oxygen after preliminary hyperventilation, which virtually abolished both chemoreceptor stimuli, the initial response of the ventilation to loading could not be observed (Fig. 2). Furthermore, the minute volume of respiration during the first 30 sec of work was only  $4.39 \pm 1.02$  liters, i.e., it was lower than the resting level ( $P < 0.01$ ). However, the decrease in the ventilatory response was due to slowing of respiration, whereas the depth of respiration, even of the first breath, was higher than initially. The EMG during this period was characterized by distinct intervals of electrical "silence" of the respiratory muscles between moderately well marked individual spikes (Fig. 1).

The manifestations of the fast component of the respiratory response followed the same general pattern in the case of light (when no weight was attached to the wheel of the bicycle ergometer) and relatively heavy (600 kg·m/min) work, although under steady-state conditions the pulmonary ventilation was dependent on the magnitude of the load.

This variability of the fast initial component of the ventilary response to physical exertion, depending on the humoral background against which the work is performed, suggests that impulses from chemoreceptors play a decisive role in the genesis of this component. Admittedly, this rule could not be observed in the response of the first "loaded" inspira-

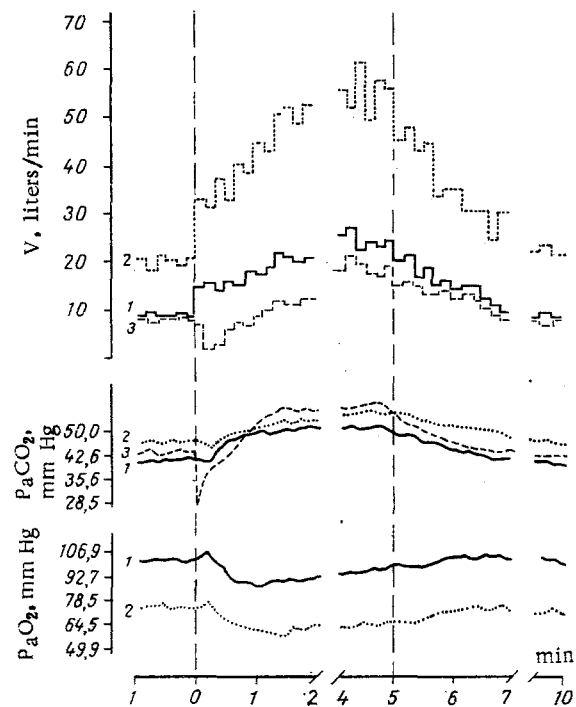


Fig. 2. Response of pulmonary ventilation ( $V$ ) and partial pressure of  $\text{CO}_2$  ( $P_a\text{CO}_2$ ) and oxygen ( $P_a\text{O}_2$ ) in alveolar air to muscular exertion ( $300 \text{ kg}\cdot\text{m}/\text{min}$ ) while subjects breathed air (1) or a mixture of 4%  $\text{CO}_2$  and 13%  $\text{O}_2$  in nitrogen (2) and after voluntary hyperventilation while breathing pure oxygen (3).

tion. It can tentatively be suggested that with the beginning of muscular exertion a powerful stimulus from the motor cortex spreads to the respiratory center and it is this which causes the increase in the first inspiration. However, at the same moment, the sensitivity of the respiratory center to chemoreceptor stimulation evidently increases [5], and against the background of an increase in this stimulation (breathing the hypercapnic or hypoxic mixture) the ventilatory response to work is intensified, whereas against the background of weakening (hyperventilation with oxygen) it disappears. The cause of the increased sensitivity to chemoreceptor stimulation is evidently afferent impulsion reaching the CNS from proprioceptors of the working muscles. The result of interaction between these stimuli is a rapid increase in pulmonary ventilation up to a level appropriate for the required intensity of muscular exertion.

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