ACTIVITY OF THE THORACIC MUSCLES DURING THERMAL HYPERPNEA

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UDC 612.217

Electrical activity of the thoracic muscles and the oxygen consumption of the animal were studied in unanesthetized cats with pre-implanted electrodes. Heating the cats in a climatic chamber led to a marked increase in the minute volume of respiration, accompanied by a reduction in the oxygen consumption of the animal and a decrease in the combined electrical activity in the serratus, external oblique abdominal, and internal and external intercostal muscles. Activity in the diaphragm and the interchondral muscles was unchanged during hyperpnea. The results point to a decrease in the heat production in the thoracic muscles during thermal hyperpnea as a result of a decrease in the electrical activity of most of the respiratory muscles.

KEY WORDS: thermal hyperpnea; electrical activity of muscles; thoracic muscles; oxygen consumption.

Thermal hyperpnea is an effective means of increasing the heat loss through evaporation [1]. However, a simultaneous increase in the heat production of the respiratory muscles accompanying intensification of their activity [5] could reduce the efficiency of thermal hyperpnea. Meanwhile, observations pointing to a decrease in the gas exchange under these conditions have also been made [3].

The object of this investigation was to study the role of the various thoracic muscles in thermal hyperpnea.

EXPERIMENTAL METHOD

Experiments were carried out on unanesthetized adult cats with electrodes measuring 1.3-1.5 mm^2 in area were previously implanted 3.5 mm apart in the thoracic muscles. Electrical activity of the diaphragm, the interchondral muscles (ICM), the external intercostal (EIC), internal intercostal (IIC), external oblique abdominal (EOA), and serratus (anterior and posterior) muscles was recorded with a 4EEG-1 electroencephalograph and N-105 multichannel loop oscillograph. The integral electrical activity was recorded in one channel with the aid of an integrator with cut-off [2]. The minute volume of respiration and the oxygen uptake of the animal also were determined [4]. During the tests the animals were placed in a frame of special design which limited their voluntary movements but did not prevent them from assuming a natural position of the body. Thermal hyperpnea was induced by heating the animals in a climatic chamber with an air temperature of 62-65°C.

EXPERIMENTAL RESULTS AND DISCUSSION

Thermal hyperpnea reached its maximal values 13.3 ± 0.96 min after the beginning of heating, when the cats' rectal temperature was raised by 0.5 ± 0.04°C. The respiration rate rose from 26.7 ± 0.8 to 240 ± 18.9/min and the minute volume of respiration from 950 ± 50.1 to 1773 ± 75.8 ml.

The decrease in heat production taking place in the cats during heating was reflected in a decrease in their oxygen consumption from 7.9 ± 0.35 to 5.1 ± 0.31 mg/kg/min. With a high minute volume of res-

Department of Human and Animal Physiology, O. V. Kuusinen Petrozavodsk University. (Presented by Academician of the Academy of Medical Sciences of the USSR, P. N. Veselkin.) Translated from Byulleten' Éksperimental'noi Biologii i Meditsiny, Vol. 78, No. 9, pp. 29-31, September, 1974. Original article submitted October 15, 1973.

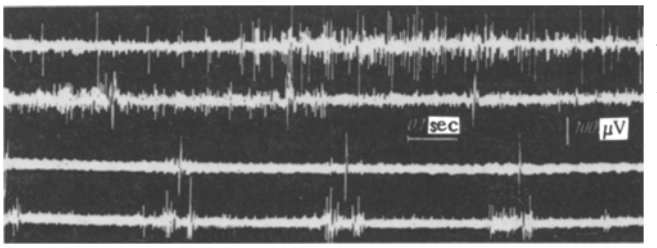

Fig. 1. Electrical activity of the internal intercostal muscle and diaphragm at a neutral temperature and during thermal hyperpnea: a) internal intercostal muscle; b) diaphragm; 1) activity at neutral ambient temperature (rectal temperature 38.3°C); 2) activity during thermal hyperpnea (rectal temperature 38.7°C).

piration during hyperpnea the effectiveness of heat loss by evaporation was ensured by the fact that most of the air breathed circulated in a short segment of the respiratory passages; a high respiration rate promotes a relative increase in the respiratory dead space [6].

With an increase in the respiration rate the duration of the inspiratory volley of the diaphragm and ICM was shortened (from 1.2 ± 0.1 to 0.1 ± 0.01 sec and from 1.0 ± 0.07 to 0.11 ± 0.01 sec, respectively). The amplitude and frequency of spikes within the volley remained practically unchanged in these muscles. Because of the increase in frequency and the simultaneous shortening of the duration of the inspiratory volleys, the integral electrical activity of the diaphragm and ICM, recorded by the integrator, was unchanged. A significant ($P < 0.01$) decrease in integral electrical activity was observed in EIC, IIC, EOA, and the serratus muscles. In 10 of the 17 EIC muscles tested during thermal hyperpnea very weak inspiratory volleys were recorded, whereas in the other muscles weak impulses of tonic activity were observed.

It must be pointed out that in the second half of the inspiratory phase weak phasic activity was recorded in the expiratory muscles, namely in 11 of the 22 EOA and 15 of the 20 IIC investigated (Fig. 1). This must restrict the expansion of the chest during the hyperpnea. The restriction of the depth of inspiration, however, leads to an increase in the frequency of respiration and thus to a more efficient evaporation.

The increase in the minute volume of respiration during thermal hyperpnea thus does not require increased activity of the respiratory muscles but, on the contrary, it is accompanied by a decrease in activity in the serratus muscles, EIC, IIC, EOA and, to a lesser degree, ICM. The decrease in tone of the postural and most of the respiratory muscles and the parallel decrease in gas exchange are evidence that, despite the increase in the minute volume of respiration in all the chest muscles except the diaphragm, the integral electrical activity decreases; this points to a decrease in heat production in the respiratory muscles during thermal hyperpnea.

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