To conclude, it must be pointed out that recording NWC between coordinates of volumes gives a clear picture of the process of gas mixing in the lungs during deep inhalation of oxygen. The volumes of DSP, AP, and LCV can be expressed quantitatively as percentages of VC. The gradient of the rise in the nitrogen concentration per unit volume in AP points to differences in the regional levels of ventilation of the lungs. The higher this gradient, the wider the range of possible ventilation of the lungs—from very high to sharply reduced, as occurs mainly in patients with lung diseases. The mechanism of closure of the lung zones, which is connected with gravitational aspects of ventilation, was clearly revealed in healthy subjects, much less so and less frequently in patients with heart diseases, and particularly badly in patients with lung diseases. In the last group disturbances of gas mixing were connected with regional changes in ventilation due to disease. An increase in the time of gas mixing by diffusion during breath holding at inspiration leads to a decrease in the volume of the dead space and to the entry of gas into the alveolar volume. In the presence of regional inequality, however, this does not improve the gas exchange in the worst ventilated zones.

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MECHANISM OF RHYTHMIC RESPIRATION DURING HYPOCAPNIA

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UDC 616.152.264-31-008.64-07:616.24-008.44

KEY WORDS: hyperventilation; hypocapnia; sensitivity of the respiratory center to CO_2; strychnine.

At the beginning of this century Haldane [5] established the stimulating action of CO_2 on the respiratory center. This was based on the fact that hyperventilation in anesthetized animals causes apnea. However, in later years attention was drawn to phenomena which, in the light of Haldane's discovery, seemed paradoxical. Not only hypercapnia, but also hypocapnia stimulates the respiratory center. In man, for instance, after voluntary hyperventilation hyperpnea often arises. In waking animals after passive hyperventilation, hypocapnic polypnea arises [3].

It was found comparatively recently that active hyperventilation in anesthetized animals due to crushing of the gastrocnemius muscle or stimulation of the nerve to the carotid sinus, by contrast with passive hyperventilation, likewise does not cause apnea. This led to the view that active respiration itself, as a result of reverberation of excitation in the network of respiratory neurons, can maintain rhythmic respiration [4].

The present investigation showed that passive hyperventilation also does not necessarily give rise to apnea in anesthetized animals if the excitability of the respiratory center is raised beforehand. Experiments were carried out on ten cats anesthetized with pentobarbital (30 mg/kg). The electromyogram of the diaphragm was recorded. In full agreement with published data artificial hyperventilation (60 breaths/min, volume 40 ml)

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(Presented by Academician of the Academy of Medical Sciences of the USSR A. M. Chernukh.) Translated from Byulleten' Éksperimental'noi Biologii i Meditsiny, Vol. 89, No. 7, pp. 6-7, July, 1980. Original article submitted August 31, 1979.

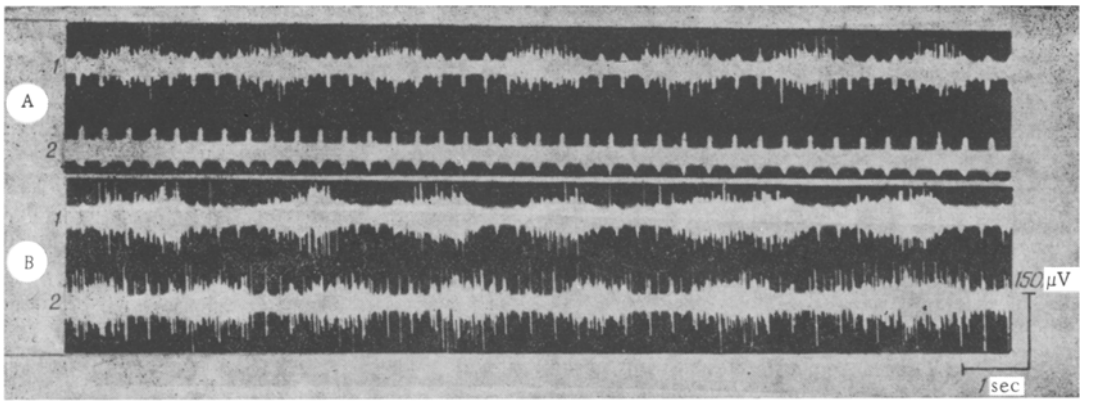

Fig. 1. A. Electromyogram of diaphragm before injection of strychnine. 1) Respiratory discharges under normal conditions; 2) apnea arises after the end of hyperventilation. B. Electromyogram of diaphragm after injection of strychnine. 1) Respiratory discharges intensified; 2) rhythmic respiration continues after the end of hyperventilation. Calibration: amplitude 150 μV, time 1 sec.

induced apnea which lasted for between 45 sec and 1 min. A solution of strychnine nitrate (0.1 mg/kg) was then injected intravenously into the animal and, 5 min later, hyperventilation with the same parameters was carried out. Apnea did not arise – the animal's rhythmic respiration was preserved (Fig. 1).

In a state of hypocapnea induced by elevation to a high altitude or artificial ventilation in patients with paralysis of the respiratory muscles the sensitivity of the respiratory center to CO_2 is increased [1, 2]. This evidently explains the fact that in waking subjects after hyperventilation hyperpnea often arises, whereas in waking animals hypocapnic polypnea arises. Rhythmic respiration in hyperventilation hypocapnea is evidently due to the fact that the sensitivity of the respiratory center to CO_2 is increased simultaneously with the development of hypocapnea.

Strychnine is known to increase the excitation of the respiratory center. With an increase in excitability of the respiratory center, a lower partial pressure of CO_2 is evidently sufficient to maintain rhythmic respiration. This is probably the explanation of the results of the experiments cited above [4]. Stimulation of the gastrocnemius muscle or of the nerve to the carotid sinus induces increased excitability of the respiratory center. Under these conditions rhythmic respiration can be maintained by a lower pCO_2.

Hence it can be concluded that Haldane's views on the stimulating role of CO_2 in the control of respiration are still completely valid.

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