EFFECT OF HIGH TRANSECTION OF THE SPINAL CORD ON HYPERVENTILATION APNEA (ROLE OF AFFERENTATION FROM MUSCLES IN THE FORMATION OF SENSATION OF RESPIRATORY FAILURE)

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UDC 616.24-008.444-092.9-092:616.
832-089.85-092.9

Transection of the spinal cord at the level C8-T1, causing paralysis of the respiratory muscles of the thorax, reduces sensitivity of the respiratory center to CO_2. This is explained, not by removal of afferentation from the respiratory muscles, but by inhibition of pulmonary ventilation and hypercapnea which results from paralysis and themselves lead to a diminution of sensitivity of the respiratory center to CO_2.

According to Campbell and Howell [5, 6] the sensation of respiratory insufficiency depends on afferent impulses from the stretch receptors of the respiratory muscles, and since there are very few muscle spindles in the diaphragm compared with their distribution in the respiratory muscles of the thorax these workers consider that a sensation of respiratory insufficiency is produced mainly through impulses arriving from the thoracic muscles. In this connection it is interesting to investigate the effect of afferent impulses from the thoracic respiratory muscles on the threshold sensitivity of the respiratory center to CO_2. This is most clearly determined with the aid of the phenomenon of the hyperventilation apnea.

The effect of high transection of the spinal cord (C8-T1)on hyperventilation apnea was investigated. Transection of the spinal cord at this level causes paralysis of the respiratory muscles of the thorax whereas activity of the diaphragm remains intact.

EXPERIMENTAL METHOD

Five cats weighing 3-4 kg were immunized by intraperitoneal injection of Nembutal (35-40 mg/kg). Tracheotomy was performed and after preliminary laminectomy under additional local procaine anesthesia the spinal cord was divided at the level C8-T1. The blood pressure in the carotid artery was recorded by a Barovar electromanometer. Neurograms from branches of the phrenic nerve and the EMG of the diaphram and intercostal muscles were recorded on a Disa electromyograph. Ventilation of the lungs was measured with a water spirometer with two-way valve. Blood was taken periodically from the femoral artery through preinserted cannulas and it pH and P_{aCO_2} determined with a micro-Astrup apparatus. Hyperventilation was carried out for 2-3 min until disappearance of spontaneous activity in the phrenic nerve and diaphragm.

EXPERIMENTAL RESULTS AND DISCUSSION

Before transection of the spinal cord the mean respiration rate was 24/min and the minute ventilation 540 ml; the pH of the arterial blood was 7.24 (7.28-7.22); P_{aCO_2} was 34 (39-30) mm Hg. Hyperventilation induced cessation of spontaneous respiration after 40-60 sec corresponding to values of pH 7.36 and P_{aCO_2} 34 mm Hg. The duration of hyperventilation apnea was 27-60 sec.

Institute of Normal and Pathological Physiology. Academy of Medical Sciences of the USSR. (Presented by Academician of the Academy of Medical Sciences of the USSR A. M. Chernukh.) Translated from Byulleten' Éksperimental'noi Biologii i Meditsiny, Vol. 74, No. 7, pp. 11-13, July, 1972. Original article submitted July 30, 1971.

After transection of the spinal cord (at the level C8-T1) activity of the intercostal muscles ceased but activity of the diaphragm continued. The blood pressure fell to 70-80 mm Hg. The pulmonary ventilation decreased to 360 ml/min, P_{aCO_2} rose to 37 (40-35) mm Hg, and pH shifted slightly to the acid side (to 7.22).

The spinal animals were subjected to hyperventilation 30-60 min after the operation when the blood pressure was raised although it had not reached its initial level. The first inspirations after hyperventilation apnea occurred when the value of P_{aCO_2} was 44 (51-42) mm Hg and pH was 7.20 (7.24-7.15), i.e., at a higher CO_2 tension. The total duration of hyperventilation apnea in the spinal animals was increased to 80 sec (70-90 sec). Division of the spinal cord, by blocking the activity of the thoracic respiratory muscles, thus actually causes a decrease in sensitivity of the respiratory center to CO_2: the first inspirations after hyperventilation apnea in the spinal animals occurred at a higher level of P_{aCO_2} (44 mm instead of 34 mm Hg before transection). This apparently concurs with the existing view that afferent impulses from stretch receptors of the thoracic respiratory muscles in the intact animal lead to an increase in sensitivity of the respiratory center [8]. However, the grounds for this conclusion are insufficient.

As these experiments show, transection of the spinal cord itself causes a decrease in ventilation and hypercapnea. Hypercapnea is known to reduce the sensitivity of the respiratory center to CO_2 [4, 13].

The results of these experiments were compared with those obtained by Camporesi et al. [7] and Sant' Ambrogio et al. [12], who investigated the sensitivity of the respiratory center to CO_2 in phrenicotomized rabbits and cats. In rabbits, because of weakness of the thoracic respiratory muscles, disturbances of respiration induced by paralysis of the diaphragm are not compensated and bilateral phrenicotomy causes a marked decrease in ventilation in these animals with a corresponding considerable increase in P_{aCO_2}. Inhalation of hypercapnic mixtures by prenicotomized rabbits did not cause an increase in pulmonary ventilation despite a persistent increase in P_{aCO_2}. In cats in which bilateral phrenicotomy is more easily compensated by the thoracic muscles and no marked changes in P_{aCO_2} arise, inhalation of CO_2 leads to a progressive increase in ventilation. In these experiments also a decrease in the sensitivity of the respiratory center to CO_2 was evidently due, not to removal of afferent impulses from the respiratory muscles, but to the increase in P_{aCO_2} induced by uncompensated paralysis of the diaphragm. These results have a bearing on clinical practice.

Rosomoff et al. [11] found that after anterolateral chordotomy the ventilation response of patients to inhalation of CO_2 is reduced. These workers explained this finding by interruption of special afferent pathways of the spinal cord responsible for the sensitivity of the respiratory system to CO_2. Nevertheless, as their observations show, high bilateral chordotomy itself causes a sharp decrease in pulmonary ventilation and also, consequently, hypercopnia; the latter is evidently the cause of the decreased sensitivity of the respiratory center to CO_2.

In patients with paralysis of the respiratory muscles and maintained on artificial respiration for a long time the sensitivity of the respiratory center to CO_2, on the contrary, is increased [2, 3]. The results of the present experiments suggest that the increase in sensitivity of the respiratory center to CO_2 is due, not to paralysis of the respiratory muscles (which itself leads to hypoventilation and hypercapnia), but to the hypocapnia developing during hyperventilation which, unlike hypercapnia, increases rather than decreases the sensitivity of the respiratory center to CO_2 [1, 9, 10].

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