insufficiency in old animals is more severe and is more often accompanied by the development of arrhythmia and disturbance of the hemodynamics. This difference can be considered to be due both to increased sensitivity of the coronary vessels in old animals to the action of vasopressin [3] and to increased sensitivity of their myocardium to restriction of its blood supply [2, 7].

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ARTIFICIAL VENTILATION OF THE LUNGS AFTER PARALYSIS OF THE DIAPHRAGM

T. I. Sergeeva, Z. N. Sergeeva, S. I. Frankshtein, and E. S. Ivanova

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To maintain a normal arterial pCO_2 in phrenicotomized rabbits artificial ventilation of the lungs must be increased to almost double the normal level. The reason is that phrenicotomy causes a decrease not only in pulmonary, but also in alveolar ventilation.

KEY WORDS: diaphragm, phrenicotomy, artificial ventilation of the lungs.

Division of the phrenic nerves leads to increased inspiratory activity of the respiratory center. The resulting paralysis of the diaphragm causes a decrease in the respiratory volume of the lungs, as a result of which the inhibitory reflex from the lungs on the respiratory center is reduced. However, the role of a reduction in the respiratory volume of the lungs in the increased activity of the respiratory center, demonstrated in experiments on rabbits with an open chest [1] or a closed chest (i.e., the conditions under which resuscitation is carried out on patients with paralysis of the respiratory muscles) may prove to be con-

Laboratory of Compensation of Disturbed Functions, Institute of Normal and Pathological Physiology, Academy of Medical Sciences of the USSR, Moscow.

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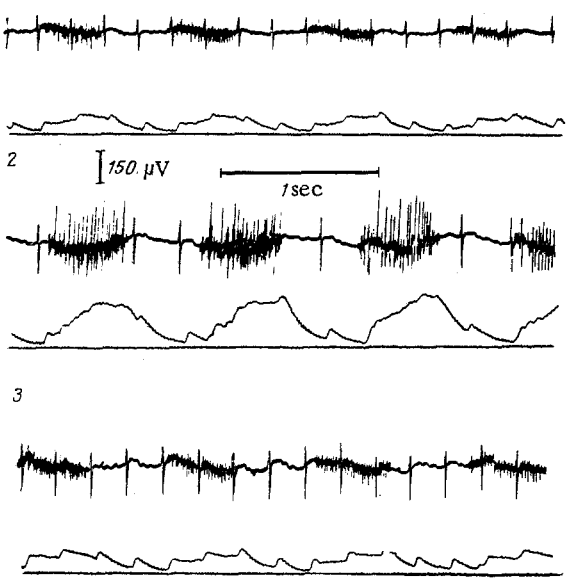

Fig. 1. Effect of phrenicotomy on the rabbit EMG. Above: EMG of inspiratory intercostal muscle; below: integrated EMG. 1) Before phrenicotomy: artificial ventilation corresponds in frequency and depth to animal's spontaneous respiration; 2) after phrenicotomy: artificial ventilation maintained at the same level as in the previous record, electrical activity sharply increased; 3) increase in volume of respiratory pump from 10 to 20 ml restores electrical activity.

siderably smaller. The increase in CO_2 partial pressure becomes of great importance in the strengthening of the activity of the respiratory center under those conditions.

EXPERIMENTAL METHOD

The respiration rate, the respiratory volume (by means of a syringe), and the values of pH and the arterial pCO_2 (p_aCO_2) (with a micro-Astrup apparatus) were determined in rabbits after tracheotomy. Both phrenic nerves of the animals were then divided at their point of entry into the thorax and the parameters listed above were again determined. Artificial respiration was then applied to the rabbits, during which the respiration rate and respiratory volume corresponded to those determined during spontaneous respiration. The electromyogram of the respiratory muscles of the chest was recorded on a Disa electromyograph. Altogether 10 experiments were performed.

EXPERIMENTAL RESULTS AND DISCUSSION

In the tracheotomized rabbits before division of the phrenic nerves the respiration rate was 46 ± 15.0 (M ± σ), the respiratory volume 12 ± 2.4 ml, pH of the arterial blood 7.32 ± 0.04, and p_aCO_2 38 ± 3.5. Phrenicotomy did not change the respiration rate but sharply reduced the respiratory volume (to 5 ± 0.5 ml; $P < 0.01$). The pH fell (to 7.20 ± 0.03; $P < 0.01$) and p_aCO_2 rose (to 51.0 ± 8.3; $P < 0.01$) respectively. Artificial normalization of the respiratory volume did not correct the disturbances of the blood gas composition, pH rose only to 7.24 ± 0.04, and p_aCO_2 fell to 48.5 ± 5.6. To restore the normal CO_2 partial pressure in the blood, the respiratory volume had to be increased from 12.0 to 22.5 ml. Under these circumstances the electrical activity of the thoracic muscles, sharply increased after phrenicotomy, was restored to normal (Fig. 1).

If the degree of artificial ventilation was not increased the animals usually died 0.5-1 h after phrenicotomy. The results described above suggest that the main cause of the increased activity of the extradiaphragmatic muscles after phrenicotomy is alveolar hypoventilation and hypercapnia rather than the reduction in lung volume.

In patients with paralysis of the respiratory muscles the lung ventilation has to be increased to much above the normal level. In the light of the facts now described the reason for this may be that, besides the pulmonary ventilation, their alveolar ventilation is also reduced.

Comparatively recently an attempt has been made to overcome dyspnea by blocking the phrenic nerves with lignocaine [2]. Such attempts can hardly be justified, for the paralysis of the diaphragm itself, as has been shown above, can lead to pulmonary insufficiency.

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EXPERIMENTAL PAIN AND ITCH SYNDROMES OF THALAMIC ORIGIN

G. N. Kryzhanovskii* and S. I. Igon'kina

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Syndromes of pain and itch of thalamic origin were produced in experiments on rats by inducing generators of pathologically increased excitation in the nucleus gelatinosus of the thalamus by means of a local disturbance of inhibitory processes in that nucleus caused by injection of tetanus toxin. The toxin was injected into the nucleus in microvolumes by a stereotaxic method. The results of the investigations agree with the concept of generator mechanisms of neuropathological syndromes which the authors have developed on the basis of the theory of the role of determinant dispatch stations in the activity of the nervous system.

KEY WORDS: pain and itch; thalamus; tetanus toxin; disturbance of inhibition; excitation generator; determinant dispatch station.

During a local disturbance of inhibition it has been found that generators of pathologically enhanced excitation can be formed in complex relay systems [1-3]. These generators lie at the basis of hyperactive central structures which form an increased functional volley [4]. This latter determines the character of activity of the parts of the central nervous system to which it is directed and, consequently, the behavior of the whole system which it activates. Hyperactive functional structures of this type have been called by the writers [5-7] hyperactive determinant dispatch stations (DDS). Hyperactive DDS generators transform physiological systems into pathological and induce the appearance of corresponding neuropathological syndromes [4-11]. On creation of a hyperactive DDS generator in the posterior horns of the spinal cord pain and itch of spinal origin appeared [12], whereas the creation of a DDS generator in the caudal nucleus of the trigeminal nerve induced a trigeminal syndrome of pain and itch [13].

The investigation described below showed that thalamic pain and itch syndromes can be produced by creating hyperactive DDS generators in the thalamic nuclei.

*Corresponding Member, Academy of Medical Sciences of the USSR.

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