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COMPENSATORY REACTIONS OF THE RESPIRATORY CENTER AND THEIR PERIPHERAL MANIFESTATIONS IN ANIMALS WITH INJURIES OF THE RESPIRATORY TRACT AND LUNGS

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It is generally considered that electrical activity of the respiratory muscles under normal conditions reflects the functional state of the respiratory center. In pathology, however, when the flow of efferent impulses to the respiratory muscles and also, probably, the flow of afferent impulses to the respiratory center (from receptors of the affected respiratory passages and lung) is modified, electrical activity of the respiratory muscles can no longer be the chief criterion used to evaluate the state of function of the respiratory center. In such cases only a simultaneous study of the discharge patterns of bulbar respiratory neurons and of the electrical activity of various groups of respiratory muscles in a model of experimental inflammation of the respiratory tract and lung can adequately elucidate the functional state of the respiratory center in animals in different stages of the disease.

In the investigation described below the firing pattern of respiratory neurons and electrical activity of different groups of respiratory muscles and compensatory reactions of the respiratory center were studied in cats with an experimental lesion of the respiratory tract and lungs.

EXPERIMENTAL METHOD

A model of injury to the respiratory tract and lungs was created in cats (60 animals) by injecting 0.3 ml of wood turpentine into the trachea in the direction of the lungs [5]. Experiments were carried out 24-72 h after injection of the turpentine. Under pentobarbital anesthesia (40 mg/kg) action potentials of medullary respiratory neurons were derived by microelectrode and stereotaxic techniques (using Szentagothai's atlas). The metal microelectrode had a tip 1-3 μ in diameter. Parallel recordings were made of the pneumogram, EMG of the diaphragm, intercostal muscles, and abdominal muscles, and the composition of the blood gases. Gas mixtures of 10% in N_2 and 2% CO_2 in air and O_2 were used. After the investigation the animal was autopsied to establish the extent and severity of the lesion of the trachea and lungs.

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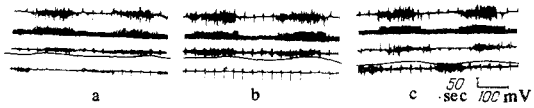

Fig. 1. Firing pattern of inspiratory bulbar neurons and EMG of diaphragm and intercoastal respiratory muscles in cats with tracheitis and breathing air (a) or hypoxic (b) and hypercapnic (c) gas mixtures. Legend (from top to bottom): EMG of diaphragm, discharges of inspiratory neurons, EMG of inspiratory intercostal muscles, pneumogram, EMG of expiratory intercostal muscles, EMG of oblique abdominal muscles.

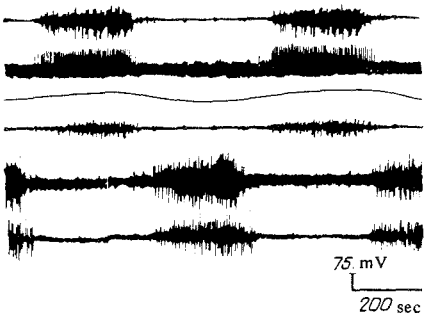

Fig. 2. Discharges of inspiratory bulbar neurons, EMG of diaphragm, EMG of intercostal respiratory muscles, and EMG of abdominal muscles in cats with unilateral lesion of lungs while breathing air.

EXPERIMENTAL RESULTS AND DISCUSSION

Depending on the location of the pathological focus, the animals were divided into three groups: 1) injury to the trachea; 2) injury to one lung (usually the right); 3) bilateral injuries of the lungs.

Animals with lesions of the trachea developed distinct hyperemia of the epithelium and edema of the trachea with the production of abundant mucus, mixed with blood and pus. The resistance to respiration was increased at inspiration and expiration, and this induced a reflex increase in spike and burst activity of both inspiratory and expiratory medullary neurons. The systematic reciprocal coordination between these neurons was disturbed, which indicates that it is adaptive in character, increasing the activity of the muscles of inspiration and expiration while maintaining normal values of the respiration rate; electrical activity of the diaphragm under these circumstances was so intensive that the breathing became diaphragmatic in character, with active expiration. Adequate reactions of the respiratory center to hypoxia and hypercapnia were preserved in the animals with tracheitis, indicating that the apparatus of external respiration has considerable reserve capacity for compensation (Fig. 1).

In animals with a unilateral lung lesion in some cases all lobes of the lung were involved and this was accompanied by bronchitis, whereas in others the lesions were found only near the root of the lung. The respiratory center of these cats also was in a state of enhanced excitation. The firing pattern of the inspiratory and expiratory neurons throughout the volley was extremely rapid and high in amplitude, because of mass activation of the neurons.

In animals with a unilateral lung lesion electrical activity of the expiratory intercostal muscles and abdominal muscles was intensified to the same degree as that of the diaphragm. Respiration became a little more rapid, of combined diaphragmatic and abdominal type. In most cases this respiration ensured a normal tidal volume and an adequate degree of oxygen saturation of the blood (Fig. 2). Compensatory reactions of the respiratory center to additional adequate stimuli were preserved in animals with a unilateral lung lesion. The main factors with an excitatory action on the respiratory center of such animals were the additional resistance to respiration at inspiration and expiration and the developing hypoxemia.

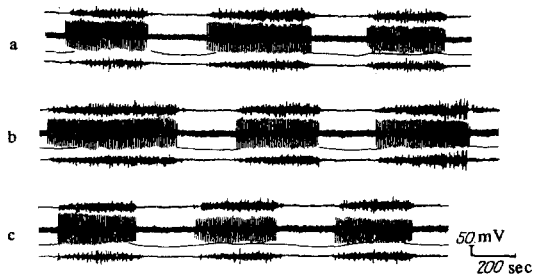

Fig. 3. Discharges of inspiratory bulbar neurons, EMG of diaphragm, and EMG of intercostal respiratory muscles in cats with bilateral lung lesions, breathing air (a) and hypoxic (b) and hypercapnic (c) gas mixtures.

Increased activity of the bulbar respiratory neurons was observed in animals with severe bilateral lesions of the lungs. Volleys of the inspiratory and expiratory neurons were distinguished not only by their extremely high frequency, but also by marked inequality as regards both duration and number of spikes of different amplitudes. Inequality also was manifested equally in volleys of electrical activity of the main groups of respiratory muscles. Breathing became highly pathological in character. It was a striking fact that with this increased discharge activity of the bulbar inspiratory and expiratory neurons, electrical activity of the principal respiratory muscles was much weaker than in cats with injury to the trachea or one lung. The decrease in electrical activity of the intercostal inspiratory muscles (Fig. 3) was particularly marked. At the same time electrical activity of the accessory muscles of respiration, especially the nasolabialis and posterior cricoarytenoid muscles, etc., was increased.

In the presence of severe injury to the lungs, when the animal was in a state of hypodynamia, the most marked compensatory reactions of external respiration were an increase in its frequency and an increase in electrical activity of the accessory muscles of respiration. Survival of the animals with such ineffective superficial respiration suggests that the process of diffusion of gases in these animals takes place in the dead space: oxygen from the dead space into the alveoli and carbon dioxide into the dead space. Such a gas exchange could correspond to the increase in electrical activity of the accessory muscles of respiration, taking part in the act of breathing under pathological conditions. The action of additional hypoxia on the respiratory center caused no appreciable adequate compensatory reactions in such animals; on the contrary, their unit activity was sharply disturbed. An increase in the inequality of volley activity of the bulbar neurons and of electrical activity of these respiratory muscles was noted. The animals soon died with a low level of oxygen saturation of the blood and with mixed acidosis. This suggests that the compensatory and adaptive powers of the respiratory center in the presence of such a degree of injury to the respiratory tract and lungs were exhausted in these animals. Analysis of the results thus showed that the earliest compensatory reaction in experimental injury to the respiratory tract and lungs was enhanced excitation of the respiratory center – of both inspiratory and expiratory neurons.

In animals with severe damage to the respiratory tract and lung there was a marked increase in the respiration rate and a redistribution of the efferent flow of impulses between the principal and accessory muscles of respiration. The main load fell under these circumstances on the diaphragm and on the abdominal and accessory muscles of respiration, contraction of which had no traumatic effect on the affected lungs and supported the gas exchange during shallow breathing.

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