ON THE REFLEX MECHANISM OF DISTURBANCES IN RESPIRATORY MOTIONS WITH LESIONS OF THE UPPER RESPIRATORY TRACT (IN THE PHENOMENON OF "SIMULATED ASPHYXIA")

ELECTROMYOGRAPHIC STUDY

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Studying the reflex mechanisms of respiratory disorders in lesions of the lungs and upper respiratory tract, we, like many other researchers [7], have often observed the complete conformity of the nose and diaphragm respiratory motions in tracheotomized animals.

We called these respiratory motions of the facial muscles in tracheotomized animals "simulated respiration" [8], as the phenomenon is similar to the "simulated feeding" of esophagotomized animals. "Simulated feeding" greatly affects the function of the gastro-intestinal tract, and "simulated respiration" has a similar effect on the act of respiration. If the nostrils of a tracheotomized animal are pinched, the animal's breathing will become slower, even though there is nothing to hinder the intake of air through the trachea. We named this phenomenon "simulated asphyxia." Klod Bernar [1] has already shown that constriction of the larynx causes similar respiratory changes.

From these observations, it follows that the inhibition of respiration up to prolonged pauses which occurs from the very first moments of mechanical asphyxia is actually of reflex origin.

In the experiments described above, we noticed the following fact. When simulated asphyxia was caused by pinching the laryx of a tracheotomized animal, the respiratory motions of the nose and thorax muscles differed considerably as to force, although they became equally retarded. The amplitude of the thorax motions remained about the same or even decreased, while the motions of the nose involved the greatest possible exertion of the muscles.

The difference in the reactions of the different groups of respiratory muscles in the phenomenon mentioned is important to the study of the reflex mechanisms of true asphyxia, and therefore these observations were made the subject of special investigations. We used the electromyographical method for this purpose, as by that method one can record the activity of separate muscle groups.

In this work, we made comparative studies of the electric activity of two respiratory muscle groups – the muscles of the alae nasi and the main respiratory muscle of the diaphragm which principally determines the volume of respiratory movements.

EXPERIMENTAL METHODS

The experiments were done on 14 rabbits weighing 2-3 kg each. The animals were fixed on their backs and not anesthetized. We recorded the respiratory movements of the nose and diaphragm with an amplifier and with a Sceife oscillograph manufactured in the precision tool shop of the Lensoviet Leningrad Technological Institute. The usual amplification was used, with which the calibration impulse amplitude was 10 mm with a voltage of 1 mv.

Electrodes from a thin, steel needle covered with an insulating layer of shellac (except for the last 6-8 mm at the tip of the needle) were inserted into the muscular part of the diaphragm through the abdominal or thoracic wall (from the right) at least 1 cm apart. The entry of the needle was controlled according to the appearance of rhythmic group discharges, synchronized to the fluctuations of a pneumograph (the fluctuations of a ray from a small mirror attached to a Marey's capsule were recorded at the same time as the biocurrents of the respiratory muscles). The electrodes were fixed by "piercing" the diaphragm, i.e., by pricking it repeatedly near the place where the needle was inserted.

The nose or larynx was constricted with pincers wrapped in cotton or by the experimenter's hand in a rubber glove (the latter method was sufficiently effective and less traumatic) until the walls of the nostril or larynx were pressed together. In most of the experiments with nose constriction, a definite reaction could be obtained by pinching only one nostril. This enabled us to use the other nostril to record electromyograms of the nose muscles. A second pair of needles was inserted into the nose muscles, into the upper and lower portions of the right nostril – the sections expanding most with inhalation (the largest muscles are the m. levator alae nasi and the m. orbicularis oris).

EXPERIMENTAL RESULTS

The electric activity of the nose muscles with calm respiration was characterized by continuous impulsation, the rate of the predominant rhythm being about 150-200 impulses per second, according to the maintenance of constant tonicity of the open nostrils. The average discharge amplitude ranged from 0.3-0.5 to 2 mv, increasing with inhalation.

There was a greater increase in the discharge amplitude of the diaphragm during inhalation, although continuous activity is maintained between inspirations in the rabbit [13, 15, 16, 18, 20].

In true asphyxia, the electric activity of the alar nose muscles and the diaphragm muscles is considerably intensified. As soon as the reflexogenic zone of the upper respiratory tract is contacted, ordinarily, a discharge of high-amplitude impulses arises in the muscles of both the nose and diaphragm. This reaction lasts $\frac{1}{2}$-1 second and is nonspecific, since it is also observed with pressure on any other part of the animal's body. Typical respiration retardation occurs immediately after it, and the amplitude of the nose muscle action currents with inspiration increases sharply (to 4.5-5 mv, sometimes more). There are more sharply defined pauses with expiration, sometimes a complete cessation of activity. Then the same type of high-amplitude discharges also appear with expiration, corresponding to the expiratory efforts of the alar nose muscles.

In mechanical asphyxia, the changes in the electric activity of the diaphragm (as already described by Dittler [13], Wachholder, McKinley [20], Gesell [14] and others) completely correspond to the changes in the electric activity of the nose muscles (Fig. 1).

The same conformity has been observed in the case where asphyxia is caused by closing the opening of the tracheal tube, i.e., with direct stimulation of the upper respiratory tract reflexogenic zone excluded. The electric activity of the diaphragm does not change immediately, but gradually, over a period of 3-5 seconds, [14] as does the electric activity of the nose muscles.

With simulated asphyxia, the electric activity of the diaphragm and nose muscles differed from that observed with true asphyxia. Although the impulsation from the alae nasi retained signs typical of true asphyxia (sharp increase in amplitude – up to 5 mv – more definite intervals between inspiration and exspiration discharges), the impulsation from the diaphragm was completely different.

The inspiration discharges of the diaphragm, maintaining their gradually increasing character, became longer and longer and slower and slower; their amplitude usually remained about the same, increasing not more than $1\frac{1}{2}$-2 times if at all, never even approaching the sharp increase in amplitude characteristic of the nose

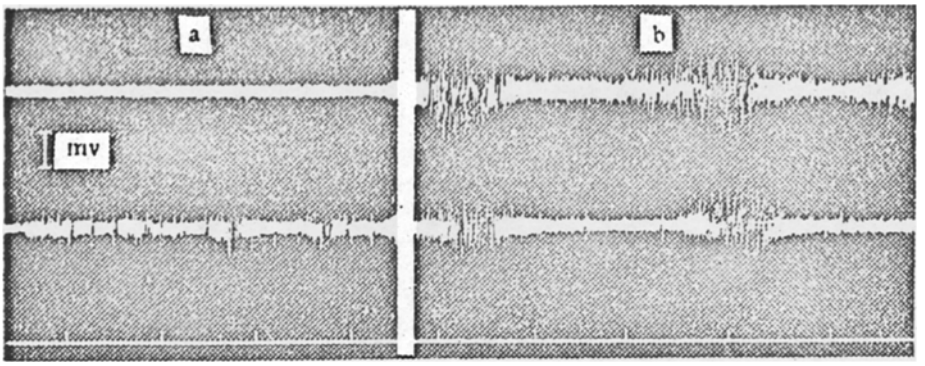

Fig. 1. Electromyograms of the respiratory muscles in true asphyxia.
Curves from top to bottom show: action currents of nose muscles, action currents of diaphragm, indication of time (in one second marks); a) original background; b) two seconds after constriction of the larynx – retarded respiration, simultaneous increase in discharge amplitudes of diaphragm and nose muscles.

muscles. We also noted that the amplitude changes with inspiration and expiration were less distinct (Fig. 2), again in contrast to the nose muscles.

Now why did the action currents of the nose increase in amplitude with simulated asphyxia, while those of the diaphragm did not? It would seem that impulsation from the respiratory center, changing due to the effect of extraordinary afferent stimulation from the constricted larynx or nose, would affect the constrictions of both the diaphragm facial muscles equally.

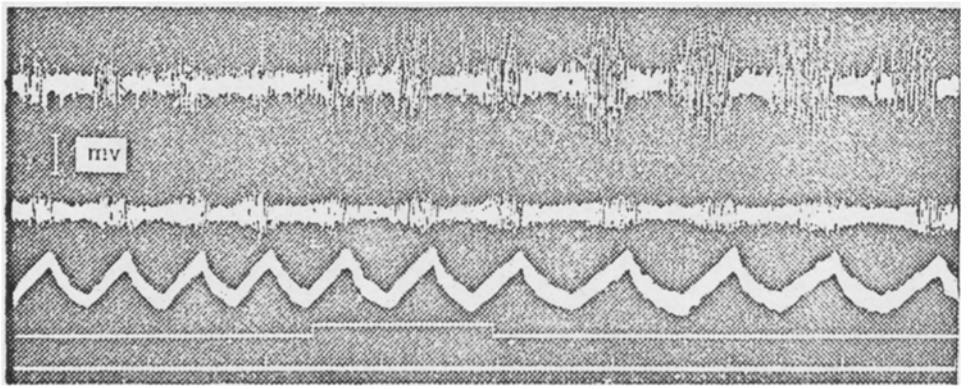

Fig. 2. Electromyograms of respiratory muscles in simulated asphyxia caused by closing one nostril of a tracheotomized animal.
Curves from top to bottom show: action currents of nose muscles, action currents of diaphragm muscles, pneumogram, indication of stimulation (closing nostril), indication of time (in 1 second marks). Respiration retarded as in true asphyxia, but character of changes in discharge amplitude is different – action current discharges of nose muscles are greater, those of the diaphragm muscles – smaller.

The mechanism of this phenomenon seems to be associated with peculiarities in the nervous regulation of the respiratory muscles. The impulses from the respiration center, before reaching the diaphragm and nose muscles, enter their motoneurons, especially the nuclei of the facial nerve (in the brain stem) and the nuclei of the phrenic nerve in the cervical part of the spinal cord (C_4).

The reaction of these motoneurons to impulses from the respiration center depends on their excitability, which is regulated by afferent influences from the periphery. These latter are different in true and simulated asphyxia.

In true asphyxia, both the motoneurons of the upper respiratory tract muscles and motoneurons of the diaphragm undergo sharp afferentation disturbances: in the first, this is due to the direct stimulation of the upper respiratory tract receptors caused by pressure on them, in the second, to the heightening of intrapulmonic pressure. This last factor is lacking in simulated asphyxia since, in tracheotomized animals, the lungs have free access to the outside air through the opening in the trachea.

As a result, the excitability of the phrenic nerve center is lower than that of the facial nerve center, which causes the impulsation from the respiration center to be manifested especially strongly in the nose muscles, and sometimes earlier than in the diaphragm.

Therefore, it follows that in true asphyxia, the intensified operation of the diaphragm is largely caused by the heightened excitability of its spinal center which, in turn, depends on the local, peripheral influences caused by extraordinary impulsation from the pleura, lungs and actual diaphragm.

The excitability of the respiratory muscle motoneurons can be raised by the influence of impulsation from the brain stem centers regulating muscular tonicity, as well as from the periphery due to impulsation from the receptors of the respiratory tract. In this way, serious respiratory disturbances, associated with a sharp increase in muscular tonicity, are known to occur both clinically [2, 9] and experimentally [3, 4, 10, 11, 12, 17] in decerebrate rigidity. These disturbances are especially pronounced during attacks of raised muscular tonicity [6]. Mislavsky [5] has already shown that these disturbances depend on disturbed motoneuron excitability, and not on disturbances in the function of the respiration center in the medulla oblongata. It has recently been established that these respiratory disorders disappear when the stem is transected at the level of the VIII pair of cranial nerves (below Deiter's nucleus), i.e., after a transection which also removes the phenomenon of decerebrate rigidity [10, 12, 19].

Therefore, both our experiments and the literature data show that, when analyzing respiratory disturbances, one must consider the condition of the peripheral neurons as well as that of the respiration center. The qualitative and quantitative characteristics of the respiration type in various pathological processes can evidently depend on the excitability of these motoneurons.

SUMMARY

Electric activity of the muscles of the nose and of the diaphragm was investigated in rabbits. It was revealed that the amplitudes of action currents of these muscles grow simultaneously in true asphyxia. In "simulated asphyxia," i.e., in disturbances of respiration caused by obstruction of nostrils or trachea in tracheotomized animals, the electric activity of the diaphragm is changed much less than in that of nose muscles. Analysis of this phenomenon suggests that the character of the movements of respiratory muscles is determined not only by the character of impulses from the respiratory center, but also by the change in excitability of peripheral motoneurons.

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