REFLEX DRIVING OF THE RESPIRATORY CENTER

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Investigations carried out on man and animals have shown that passive movements increase the pulmonary ventilation [2, 3, 7, 9]. This is explained by assuming that when the skeletal muscles are stretched, the mechanoreceptors present in muscles and tendons are stimulated, causing reflex excitation of the respiratory center and an increase in the intensity of respiration. It is considered that these reflexes, commonly called proprioceptive respiratory reflexes, play an important role in increasing the ventilation of the lungs during muscular work.

The question has been raised whether the influence of proprioceptive respiratory reflexes (like other reflex influences acting on the respiratory center) is limited to a change in the magnitude of pulmonary ventilation or whether they can force a new rhythm of respiration on the respiratory center, coinciding with the rhythm of the reflex influences.

According to the very limited information in the literature, during artificial respiration the rhythm of natural respiration is modified, coming close to the rhythm of artificial inspiration and expiration [4,8]. It has also been noted that during rhythmic stimulation of the mucous membrane of the respiratory tract the respiration rate sometimes coincides with the frequency of stimulation of the mucous membrane [1]. Muscular work in man has also been shown to affect the respiration rhythm [2].

The object of the present investigation, carried out on animals, was to determine whether during reflex action on the respiratory center, it is driven by the rhythm of stimulation, i. e., whether "a hitherto alien rhythm is adopted as its own" [5].

EXPERIMENTAL METHOD

Experiments were carried out on 70 cats anesthetized with ether and urethane. Artificial respiration was carried out with an apparatus making it possible to change the volume of air pumped into the lungs and the frequency of inhalation. The artificial respiration apparatus was connected through a three-way tube to the trachea and to a Marey's capsule attached to a "Diza" electromyograph, which was used to record the electrical activity of the respiratory muscles and of the nerves supplying them.

In one series of experiments the conditions of artificial respiration, mainly the frequency of expansion of the lungs, were modified within wide limits. To prevent the development of hypocapnia, influencing the respiratory rhythm, 3-4% CO_2 was added to the air blown into the lungs. In another series of experiments the effect of passive limb movements on electrical activity of the respiratory muscles was studied. These movements were produced by electrical stimulation of the limb muscles or they were carried out by the experimenter, who modified the rhythm of flexion and extension of the limb (using a metronome).

In the 3rd series of experiments the changes produced in the rhythm of respiration by stimulation of the mechanoreceptors of the tracheal mucous membrane by a stream of air supplied at different frequencies from the artificial circulation apparatus were studied.

EXPERIMENTAL RESULTS AND DISCUSSION

Usually a few seconds after changing to artificial respiration, with a frequency of lung expansion widely different from the natural respiration rate, changes were observed in the electrical activity of the respiratory muscles. It is clear from Fig. 1 that 1 min after the beginning of artificial respiration the duration of individual volleys became uneven and respiration was slightly slowed. After artificial respira-

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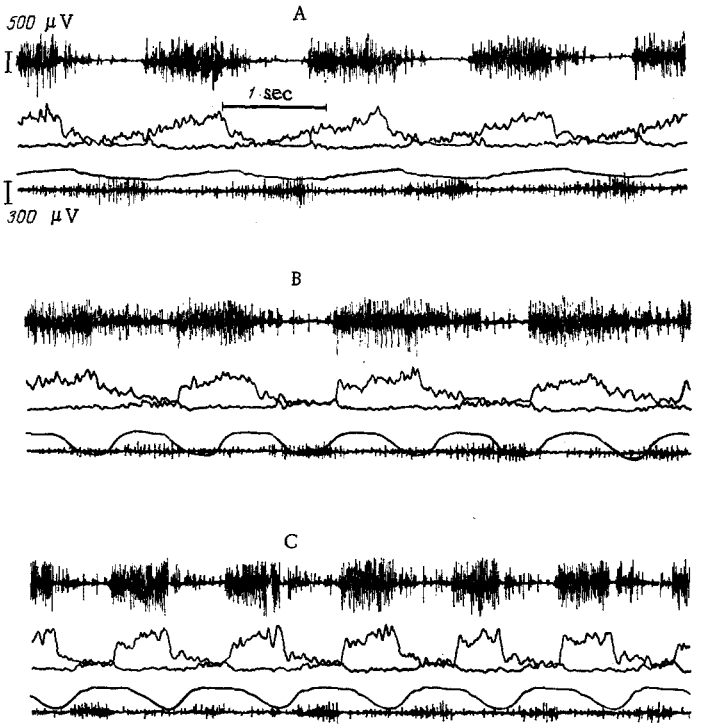

Fig. 1. Dynamic driving of the new respiratory rhythm during artificial ventilation of the lungs. A—electrical activity (EA) of inspiratory and expiratory muscles during natural respiration. From top to bottom: EA of diaphragm; integration curves of EA of inspiratory and expiratory muscles; pneumogram; EA of internal intercostal muscle. B—EA of respiratory muscles 1 min after beginning of artificial respiration. From top to bottom: EA of diaphragm; integration curves of EA of inspiratory and expiratory muscles; curve of artificial ventialtion of lungs; EA of internal intercostal muscle. C—EA of respiratory muscles after artificial respiration for 4 min. Legend as in B.

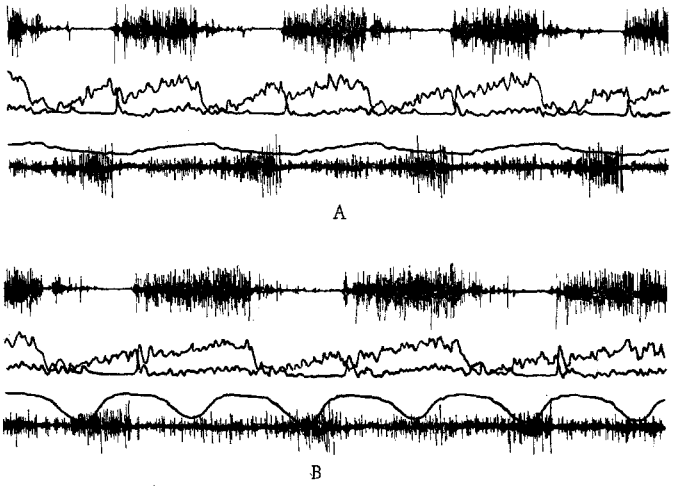

Fig. 2. Effect of division of the vagus nerves in the neck on driving by the rhythm of artificial respiration. A—before division of vagus nerves; natural respiration. From top to bottom: EA of diaphragm; integration curves of EA of respiratory muscles; pneumogram; EA of internal intercostal muscles. B—58 min after division of vagus nerves; artificial respiration (10 min). From top to bottom: EA of diaphragm; integration curves of EA of respiratory muscles; curve of artificial ventilation of lungs; EA of internal intercostal muscle.

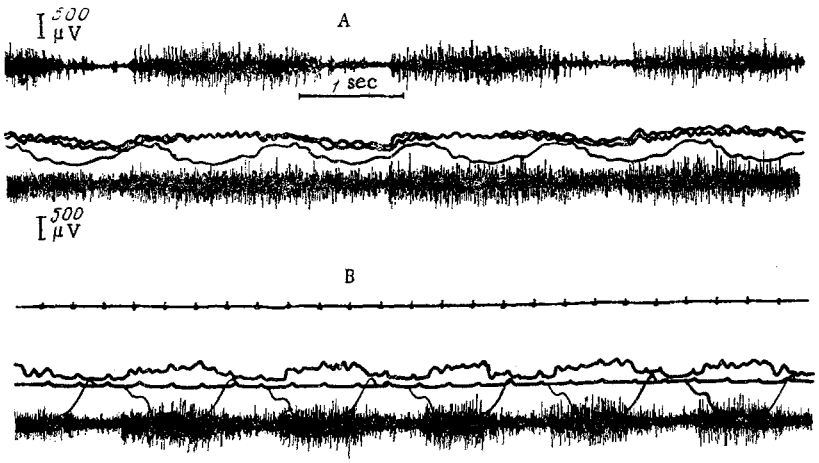

Fig. 3. Driving of respiratory rhythm during artificial ventilation of the lungs after administration of relaxants. A—Natural respiration. From top to bottom: EA of diaphragm; integration curves; recording of work of artificial respiration apparatus (stage not connected to animal); EA of phrenic nerve. B—Artificial ventilation of lungs after injection of relaxant. Legend as in A.

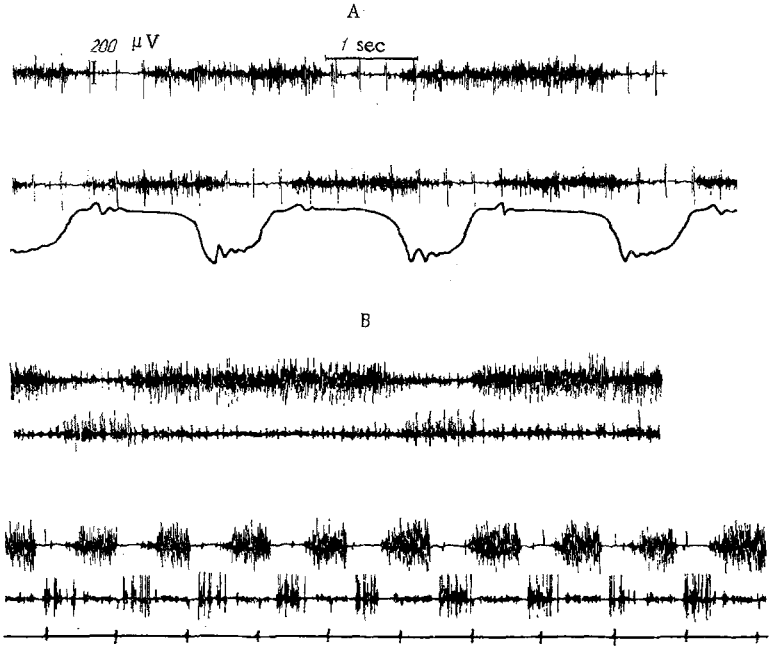

Fig. 4. Driving of respiratory rhythm by passive limb movements. A—EA of diaphragm during repeated flection and extension of limb. From top to bottom: EA of diaphragm before passive movements; EA of diaphragm after passive flections and extension of the limb for 3 min; mechanogram of flection and extension of limb. B—EA of respiratory muscles during passive limb movements caused by repeated electrical stimulation of the limb muscles. From top to bottom: EA of diaphragm before passive movements; EA of internal intercostal muscle before passive movements; EA of diaphragm after passive limb movements for 4 min; EA of internal intercostal muscle after passive limb movements for 4 min; marker of electrical stimulation.

tion for 4 min (Fig. 1, C), the frequency of inspiratory and expiratory volleys increased considerably and individual volleys coincided to a large measure in time with the phase of expansion of the lungs. In this case the natural rhythm of the respiratory center was replaced by the driving rhythm of respiration.

The disturbance of the normal respiratory rhythm observed after changing to artificial respiration may be regarded as reflecting a "conflict" between the natural rhythm of respiration and the reflex influences on the respiratory center arising during artificial ventilation of the lungs. During repeated stimulation of the respiratory center, the normal rhythm of respiration is suppressed by the driving rhythm. In most experiments this adoption of the new respiratory rhythm took place after 1-6 min of artificial respiration. If anesthesia was very deep, driving of the new rhythm of respiration was slight or absent altogether. After vagotomy, the ability of the rhythm of artificial ventilation of the lungs to drive the respiratory center disappeared.

It is clear from Fig. 2, B that after vagotomy, as might be expected, respiration became slower and remained the same despite the fact that the frequency of lung ventilation was twice as fast as the natural respiration rate. The blocking of proprioceptive respiratory reflexes observed after intravenous injection of a relaxant, while the vagus nerves remained intact, did not prevent enforcement of the driving rhythm. It may be seen from Fig. 3, B that after injection of relaxant, artificial respiration for 3 min caused definite synchronization of the phases of the inspiratory volleys in the phrenic nerve with the phases of lung ventilation.

During intermittent stimulation of the tracheal mucous membrane by a jet of air, discoordination of the respiratory movements took place, but the respiratory rhythm was not driven by the rhythm of stimulation. Usually after a few minutes of stimulation of the tracheal mucous membrane the respiratory rhythm reverted to normal. This suggests that in this particular case the mechanoreceptors of the tracheal mucous membrane became adapted to the action of the mechanical stimuli.

To determine the role of the proprioceptive respiratory reflexes in enforcement of the new respiratory rhythm, the effect of passive limb movements of the experimental animal on the respiratory rhythm was investigated. Within a few seconds after the beginning of passive movements, the respiratory rhythm changed and discoordination of respiration developed as was observed during artificial respiration; later the respiratory rhythm gradually began to approach the rhythm of passive movements more closely (Fig. 4). This driving of the respiratory center by the rhythm of these movements persisted after division of the vagus nerves in the neck, and this may be understood because the afferent fibers of the proprioceptive reflexes enter the central nervous system through the spinal cord. It remained to be discovered whether only the bulbar respiratory center can be driven by the new rhythm of respiration or whether this driving was due to the influence of higher levels of the brain in close association with the reticular formation of the medulla. To investigate this problem, cats were decerebrated and investigations were carried out to discover if the inspiratory and expiratory electrical activity of their respiratory muscles changed in response to changes in the frequency of artificial lung ventilation. It was found that after decerebration, driving of the respiratory rhythm could still take place.

The experimental results described in this paper afford proof that the respiratory center can synchronize its activity with the rhythm of incoming stimuli. Although enforcement of a new respiratory rhythm can still take place after decerebration, when influences of higher levels of the brain on the bulbar respiratory center are removed in natural conditions, as investigations on man have shown [2, 6], the higher levels of the central nervous system play an important role in the formation and enforcement of the new respiratory rhythm.

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