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ROLE OF THE PYRAMIDAL TRACT IN THE MECHANISM OF DYSPNEA AND HYPERVENTILATION

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KEY WORDS: pyramidal tract; hypoxia; mechanical asphyxia; hypoxic work; hyperventilation; dyspnea.

Hypoxemia by itself causes neither respiratory discomfort nor excessive hyperventilation in healthy subjects. However, both respiratory discomfort and excessive ventilation soon arise during hypoxemia if the subject attempts to carry out physical work [1]. Attempts to explain these phenomena by a change in afferentation from vascular chemoreceptors have not proved successful. Hypoxic work does not affect impulsation from vascular chemoreceptors [4]. Removal of the carotid sinuses (for bronchial asthma) does not prevent the onset of dyspnea and excessive hyperventilation during hypoxic work [5].

Physical exertion is induced by impulses from the sensomotor cortex which spread to the somatic muscles via the pyramidal tract. It is shown in this investigation that hypoxemia, induced by mechanical asphyxia, disturbs initially the generation of discharges in pyramidal tract neurons. This fact is of definite interest for the analysis of how both respiratory discomfort and excessive ventilation during hypoxemia are provoked by physical exertion.

EXPERIMENTAL METHOD

Experiments were carried out on 11 cats anesthetized with pentobarbital (40 mg/kg, intraperitoneally). N. saphenus and n. tibialis were divided in the region of the groin and knee, respectively, and their peripheral ends were placed on bipolar platinum electrodes with an interelectrode distance of 5 mm. These electrodes served for stimulation. Peripheral branches of n. saphenus were placed on similar electrodes for recording antidromic action potentials. Responses of the gastrocnemius muscle to stimulation of n. tibialis were recorded by bipolar wire electrodes, inserted into the thickness of the muscle by means of a surgical needle. The distance between the electrodes was 7-10 mm. Primary responses in the first somatosensory region of the cortex were recorded by monopolar silver ball electrodes. The indifferent electrode (steel needle) was fixed in the nasal bones. Discharges of the bulbar pyramidal tract and bulbar medial lemniscus were recorded by monopolar needle electrodes, inserted from the dorsal surface of the medulla at the level of the obex. Primary cortical responses, and discharges of the pyramidal tract and medial lemniscus were induced by stimulation of the contralateral forelimb. For this purpose, needle electrodes with an interelectrode distance of 10 mm were inserted beneath the skin of the dorsal surface of the foot. The exposed surfaces of the brain, nerves, and muscles were covered with warm mineral oil. Potentials were recorded before the tracheotomy tube, introduced beforehand into the animal, was covered and after covering for 1, 2, and 3-4 min.

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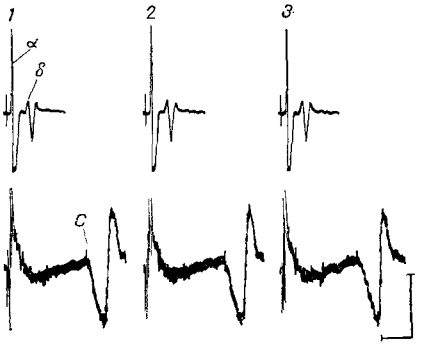

Fig. 1

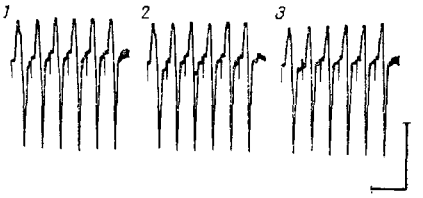

Fig. 2

Fig. 1. Action of asphyxia on skin receptors and afferent fibers connected to them. Top tracings: α and δ components; bottom tracings: C component of antidromic complex action potential before asphyxia (1) and during asphyxia for 3 min (2) and 4 min (3). Antidromic complex action potential of cutaneous nerve unchanged after asphyxia for 4 min. Calibration: time, 10 msec for top and 40 msec for bottom tracings; voltage, 500 μV for top and 50 μV for bottom tracings.

Fig. 2. Action of asphyxia on neuromuscular transmission. 1-3) Responses of gastrocnemius muscle to stimulation of peripheral end of divided n. tibialis by volley of six pulses with frequency of 93 Hz before asphyxia (1) and during asphyxia lasting 3 min (2) and 3.5 min (3). Neuromuscular transmission unchanged throughout the period of asphyxia. Calibration: time, 20 msec, voltage 5 mV.

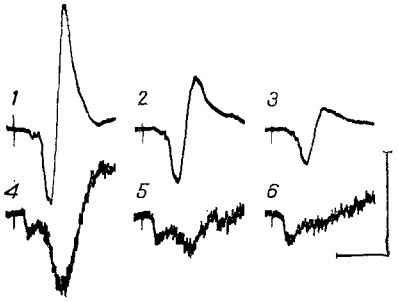

Fig. 3. Action of asphyxia on primary cortical responses, discharges of medial lemniscus, and corticofugal reflex of pyramidal tract. 1-3) Primary responses in somatosensory area I to stimulation of contralateral forelimb. 4-6) Discharges of bulbar medial lemniscus (first positive wave with latent period of 4.5 msec) and of pyramidal tract (second positive wave with latent period of 12 msec) to stimulation of contralateral forelimb before asphyxia (1, 4) and during asphyxia lasting 2 min 45 sec (2, 5) and 3 min 10 sec (3, 6). Responses of medial lemniscus unchanged throughout the period of asphyxia; primary cortical responses reduced, corticofugal reflex in pyramidal tract disappears almost completely. Calibration: time 20 msec, voltage 200 μV for top tracings and 150 μV for bottom tracings.

EXPERIMENTAL RESULTS

The peripheral receptors in asphyxia possess exceptional stability: Even after covering of the tracheotomy tube for 3 min.the complex action potential of n. saphenus and potentials of the medial lemniscus were

unchanged (Figs. 1 and 3: 4-6). Neuromuscular transmission also possesses stability: Responses of the gastrocnemius muscle to stimulation of n. tibialis likewise were unchanged after this same period of time (Fig. 2). Primary cortical responses to stimulation of the contralateral forelimb were much more sensitive to asphyxia: After covering of the tracheotomy tube for 3 min they were significantly reduced. However, most sensitive of all was the corticofugal reflex – discharges of the pyramidal tract to peripheral stimulation completely disappeared by this time (Fig. 3).

The results are evidence that in hypoxemia induced by mechanical asphyxia, generation of potentials is disturbed primarily in neurons of the pyramidal tract.

Unlike other autonomic functions, respiration is controlled not only automatically, but also voluntarily. Voluntary regulation of respiration is effected by impulses from the sensomotor cortex to the respiratory center in the brain stem, and directly via the pyramidal tracts to spinal motoneurons of respiratory muscles. In cases of cerebral paralysis caused by a lesion of the pyramidal tract, voluntary regulation of respiration in the half of the chest that is opposite to the pathological focus becomes impossible, whereas automatic rhythmic respiration on this side is intensified.

The disturbance of potential generation in pyramidal tract neurons due to hypoxemia evidently is responsible for the inadequate contractions of the muscles during hypoxic work, with the result that the work is performed with difficulty and a sensation of respiratory discomfort arises [2, 3].

In the light of these data the therapeutic effect of oxygen inhalation during dyspnea can be explained. Dyspnea is accompanied by an increase in respiratory work. The effect of oxygen therapy evidently likewise cannot be explained by the action of oxygen on the chemoreceptors, but by restoration of the function of the CNS and, in particular, the function of the pyramidal tract, so that the performance of respiratory work is facilitated and the sensation of respiratory discomfort is abolished.

It was mentioned above that in central paralyses caused by a lesion of the pyramidal tract, besides the loss of voluntary regulation of respiration, automatic breathing by half of the chest opposite to the pathological focus is intensified – the bulbar respiratory center is disinhibited by a lesion of the pyramidal tract. As already mentioned, hypoxic work causes not only respiratory discomfort, but also considerable hyperventilation, and attempts to explain it by the action of hypoxemia on chemoreceptors have likewise proved unsuccessful [4]. In the light of the data given above, it can be explained by the disinhibition of the respiratory center in the brain stem arising through blocking of the pyramidal tract.

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