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HYPERVENTILATION AND INHIBITORY SYNAPSES

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Injection of a subconvulsive dose of strychnine, which blocks inhibitory synapses, considerably potentiates the reflex activity of the respiratory muscles in response to sciatic nerve stimulation and inhalation of a hypercapnic mixture. Inhibitory synapses thus prevent excessive hypocapnia induced by hyperventilation.

KEY WORDS: strychnine; hyperventilation; hypercapnia.

Strychnine is a stimulator of respiration. Its action is due to blocking of inhibitory synapses [4]. However, it does not affect the vagal inhibitory reflex from the lungs [1-3]. It can be postulated that the mechanism of action of strychnine on the respiratory system depends on its disinhibitory action on motoneurons innervating the respiratory muscles. To analyze this question the effect of subconvulsive doses of strychnine (0.07 mg/kg) was studied on: 1) respiratory reflexes induced by stimulation of the central end of the divided sciatic nerve, and 2) activity of respiratory muscles induced by inhalation of a hypercapnic mixture.

EXPERIMENTAL METHOD

Experiments were carried out on 12 cats anesthetized with urethane (1.5-2.0 g/kg, intravenously). Recording electrodes were sutured to the sternal part of the diaphragm. The central ends of the divided sciatic nerve were stimulated with square pulses 0.5 msec in duration and with a frequency of 10 Hz. A gas mixture consisting of 6% CO_2 in air was used.

EXPERIMENTAL RESULTS

Before injection of strychnine the respiration rate was 24.5 ± 1.4 per minute. The threshold of the respiratory reflex (quickening of respiratory discharges of the diaphragm) was 20-30 V. When stimulation of the nerves ceased, respiration was immediately restored. Subconvulsive doses of strychnine caused a very slight increase in respiration rate (27.5 ± 2.1), and sometimes also an increase in the respiratory discharges of the diaphragm.

After injection of the subconsulsive dose of strychnine the threshold of the respiratory reflex was reduced by 75-80%. The respiration rate reached 36.0 ± 1.7 and remained at this level for 15 sec to 1 min after the end of nerve stimulation.

Inhalation of the hypercapnic mixture caused an increase in the depth and frequency of the respiratory discharges. After injection of strychnine, inhalation of the same gas mixture caused a sharper increase in the

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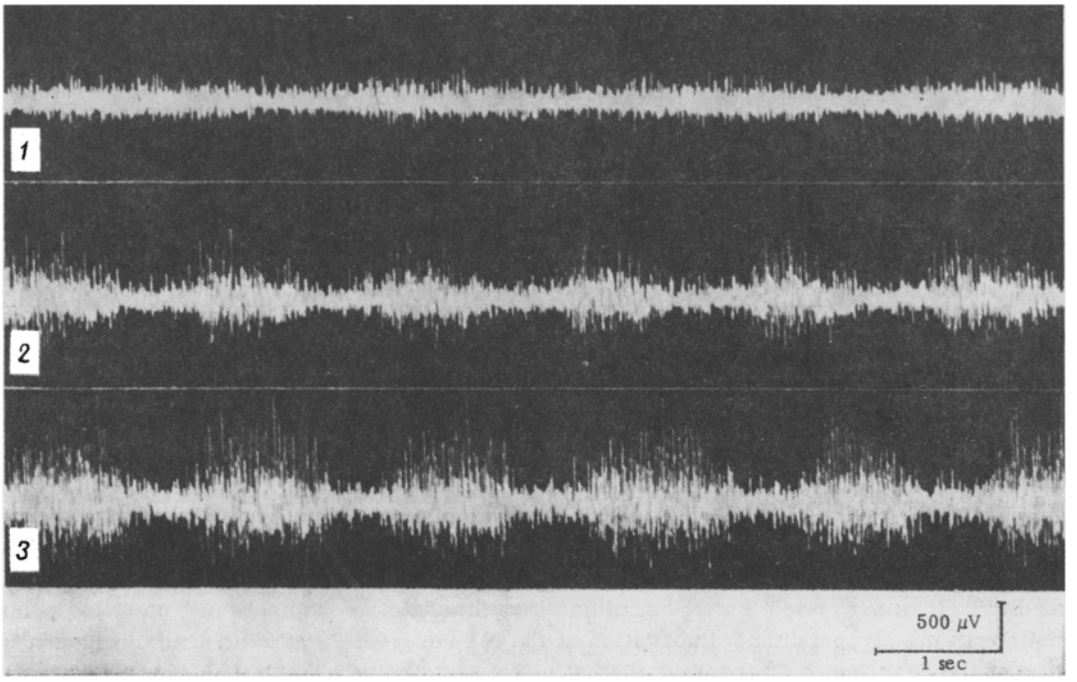

Fig. 1. EMG of the diaphragm. 1) Respiratory discharges under normal conditions, 2) before injection of subconvulsive doses of strychnine (inhalation of a hypercapnic mixture increases the frequency and depth of respiration), 3) after injection of subconvulsive dose of strychnine (inhalation of a hypercapnic mixture increases respiratory discharges much more strongly).

intensity of respiratory volleys and the appearance of activity in new motor units (Fig. 1).

Inhibitory synapses thus play an important role in the prevention of excessive hyperventilation, which gives rise to severe hypocapnia and possible disturbance of the function of the nervous system and regulation of respiration.

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