HETEROGENEOUS CONTROL OF RESPIRATORY REACTIONS BY VENTRAL MEDULLA STRUCTURES IN APNEA MODELING

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The problem of sleep disordered breathing accompanied with snoring and breathing arrest always includes the analysis of central and peripheral pathological mechanisms [1, 2]. The sequential analysis of the problem will be carried out. Peripheral mechanisms provide air circulation in airways during respiratory cycle. The development of obstructive events in airways conditionally leads to apnea (for example after food aspiration). Such apnea always initiates protective reactions of the organism including forced expiration and cough being the signs of central mechanisms' switch. In most cases the initiation of these protective (central) mechanisms during wakefulness or sleep is sufficient for obstructive event elimination. Unfortunately, clinical practice shows that in certain cases, particularly in patients with obstructive sleep apnea syndrome, these protective (central) reactions are ineffective. An irreversible breathing arrest occurs. Given the complexity of central and peripheral breathing control organization, special attention was paid to the modeling of central mechanisms of ventilation embarrassment by blocking superficial structures of ventral medulla in the experimental study. These regions of brain stem include chemosensitive structures, which mainly react to hypercapnic stimulus, and neuron populations, which transmit signals from medullary chemoreceptors to respiratory centre [3].

Lidocaine (50-100µl, 0.01-1.0%) was applied on ventral medulla surface through polyethylene catheter introduced under dura mater by ventral access between first and second cervical vertebrae in acute experiments on anesthetized with nembutal and urethan (30 and 500 mg/kg) rats. The acceleration of diaphragm electrical activity was established in first tens of seconds after the application of lidocaine in small doses (0.01%) (Figure).

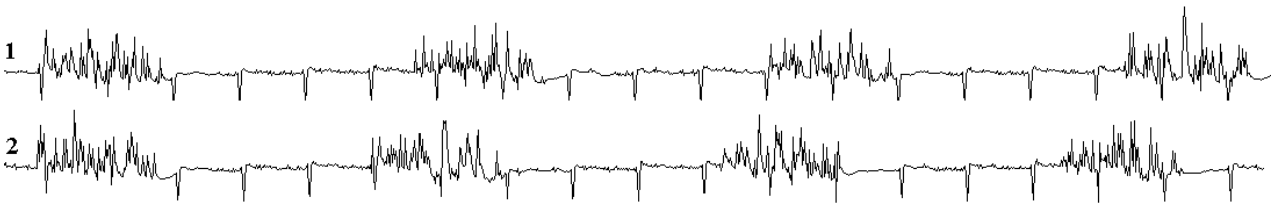

Fig. 1. The electrical activity of anesthetized rat diaphragm before (1) and in 10 seconds after the application of 0.01 % lidocaine on caudal regions of ventral medulla surface (2)

This experimental effect conflicted with the data obtained by other researchers [1, 2]. It was proved that the application of anesthetics (for example lidocaine) on ventral medulla surface of anesthetized animals leads to ventilation embarrassment up to apnea development. What is the reason for increased frequency and amplitude of diaphragm activity? The literature data [3-5] on the heterogeneous organization of neural networks in the ventral medulla may be the answer to this. Cell populations located in caudal regions of ventrolateral medulla tonically inhibit the activity of rostral cell groups which are responsible for the formation of signals' pattern to respiratory centre. Therefore, the injection of anesthetic in small volumes into caudal parts of brain stem is accompanied with lidocaine action primarily on the neurons of "caudal group". The blockade of "caudal group" cell functions is accompanied with disinhibition of

neurons in rostral regions of ventral medulla, leading to increase in frequency and amplitude of diaphragm electrical activity (Figure).

The fact established in acute experiments allows explaining the multidirectional changes of ventilation in patients with obstructive sleep apnea syndrome. The neurochemical interaction between cell populations in ventral medulla is presumably a reason for this phenomenon.

References

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