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UNIT ACTIVITY IN THE GANGLION NODOSUM IN ACUTE CIRCULATORY AND RESPIRATORY DISTURBANCES

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KEY WORDS: pentobarbital; ganglion nodosum; neurons.

The writers showed previously [3] that during stimulation of reflexogenic zones of the heart and blood vessels, neurons of the bulbar cardiovascular center responded in different ways. Discharges of some neurons, synchronized with the cardiac rhythm, changed only after stimulation of certain reflexogenic zones. Neurons whose discharges were not strictly synchronized with the cardiac rhythm varied their firing pattern in response to excitation of receptors of many reflexogenic zones. It was not clear whether activity of neurons in the cardiovascular center changes directly under the influence of the flow of afferent information from receptors of the cardiovascular system or whether it can be integrated at the level of the ganglion nodosum, on account of influences of impulses from receptors of the respiratory system on its neurons. In setting out to study this problem the starting point was the fact there is clinical and experimental evidence of close interconnection between the activities of these two systems [5]. Furthermore, in the ganglion nodosum cardial and respiratory neurons are distributed diffusely [6], and histological studies have revealed different end structures and synaptic terminals [1, 2].

EXPERIMENTAL METHOD

Experiments were carried out on 23 cats of both sexes weighing 3-4 kg. The animals were anesthetized with pentobarbital (30-40 mg/kg, intraperitoneally) and artifically ventilated on the Vita-1 apparatus, by means of which the respiration rate and the volume of inspired air can be varied. After thoracotomy bilateral vagotomy was performed above the diaphragm and the pericardium was opened; ligatures were passed beneath the pulmonary trunk and aorta at the point where they leave the ventricles, beneath the descending aorta, and beneath the circumflex branch of the left coronary artery; PVC catheters were introduced into the right and left atria. The cat was placed in a stereotaxic apparatus in the supine position. The ganglion nodosum was exposed through a parasagittal incision passing through skin, cervical fascia, and connective tissue connecting m. sternohyoideus and m. mastoideo-humeralis. The ganglion was freed from its connective-tissue capsule and placed on an insulating support. Electrical activity of the neurons was recorded extracellularly with glass microelectrodes filled with 2.5M KCl solution (resistance 7-10 MΩ). Parallel recordings were made of the pressure in the femoral artery, the ECG in standard lead II and the pneumogram, using a carbon transducer. All processes were recorded on a four-channel Medicor (Hungary) myograph. Activity of 67 neurons was analyzed by the method described in [3].

EXPERIMENTAL RESULT

Activity of three grups of neurons was studied in the ganglion nodosum: cardiovascular, with discharges synchronized with the cardiac rhythm, cardiopulmonary, whose activity coincided both with phases of respiration and with the ECG, and respiratory, whose activity was

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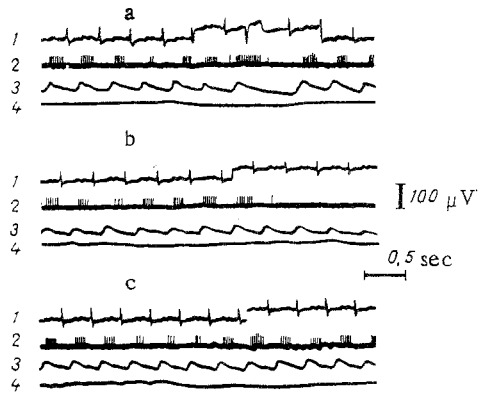

Fig. 1. Response of a cardiovascular neuron to stretching of the left atrium (a), compression of the pulmonary trunk at the point where it leaves the ventricle (b), and compression of the circumflex branch of the left coronar artery (c). 1) ECG (shift of isoelectric line corresponds to time of procedure); 2) unit activity; 3) arterial pressure; 4) pneumogram.

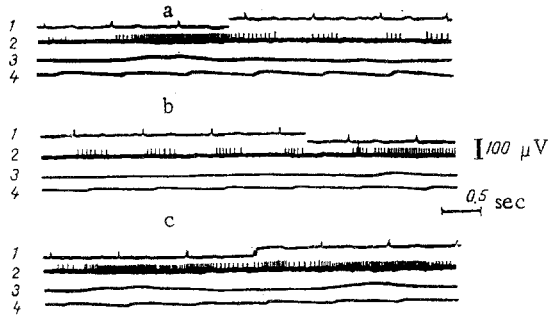

Fig. 2. Response of cardiopulmonary neuron to interruption (a) and activation (b) of respiration and compression of circumflex branch of left coronary artery (c). 1) ECG (shift of isoelectric line corresponds to time of procedure); 2) unit activity; 3) pneumogram; 4) arterial pressure.

synchronized with the phases of respiration [7]. Since changes in the hemodynamics are adequate stimuli for the afferent structures of the cardiovascular system and since changes in the filling of the lungs with air served the same purpose for the respiratory system [8], to study the polymodality of influences on neurons of the ganglion nodosum a combination of hemodynamic and respiratory tests was used. The first included dilation of the right and left atria with warm physiological saline, compression of the pulmonary trunk and aorta where they leave the ventricles, compression of the aorta above the diaphragm, and compression of the circumflex branch of the left coronary artery; the latter included blocking of artificial respiration or an increase in its frequency and depth. To exclude any effect of the hypoxia which arises during prolonged artifical limitation of the blood flow or interruption of artificial respiration, unit activity recorded during the first second after the procedure was recorded, i.e., at a time when side effects connected with the development of metabolic disturbances were absent during the use of the above-mentioned function tests, and analyzed.

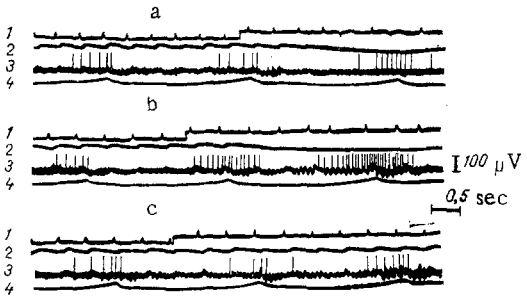

Fig. 3. Response of late inspiratory neuron to compression of aorta where it leaves the ventricle (a), above the diaphragm (b), and compression of circumflex branch of left coronary artery (c). 1) ECG (shift of isoelectric line corresponds to time of procedure); 2) arterial pressure; 3) unit activity; 4) pneumograms.

Cardiovascular neurons (12 cells), although not reacting to compression of the branch of the left coronary artery in the course of 1 sec, nevertheless modified their activity during the hemodynamic tests (Fig. 1). In this respect the responses of the cardiovascular neurons resembled those found previously in single cardiac and vascular afferent fibers of the vagus nerve [8].

In the group of cardiopulmonary neurons (16 cells) the effect of hemodynamic and respiratory tests on their spike discharges could be differentiated. For instance, compression of the great vessels was usually accompanied by a change in the cardiac component of activity of the neuron, whereas the respiratory tests mainly changed the respiratory component of its activity (Fig. 2a, b). Roughly half of the neurons of this group altered their activity immediately after compression of the coronary vessel (Fig. 2c) but the other half did not do so until 3-5 sec later, i.e., against the background of changes in the hemodynamics of the pulmonary circulation [4].

In the group of respiratory neurons (39 cells) all inspiratory neurons responded adequately to changes in the volume and frequency of artificial ventilation of the lungs and, as a rule, did not change their spike activity during hemodynamic tests. Inspiratory-expiratory and, in particular, late inspiratory neurons, while responding adequately to respiratory tests, often changed the character of their discharge in response to hemodynamic tests and, in more than half of all cases, did so during the first second in response to compression of the coronary artery (Fig. 3).

It can be concluded from these results that the ganglion nodosum of the cat consists not only of afferent neurons, which change their activity like the corresponding afferent fibers of the vagus nerve, during stimulation of the limited receptive field of the cardiovascular or respiratory system. These cells evidently transmit information to the bulbar cardiovascular center without any change in its character at the level of the ganglion nodosum. Other neurons contained in the ganglion, which respond by changes in their activity to stimulation not only of the different receptive zones of the heart and vessels, but also of receptors of the respiratory system, can change the character of their discharge at the ganglion cell level and thus transmit it transformed to the bulbar cardiovascular center when acute disturbances of the hemodynamics and respiration are present. This evidently ensures rapid activation of central nervous regulation in the mechanism of the compensatory reactions arising in these states.

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CHANGES IN THE HEART RATE DURING HYPERACTIVATION OF THE ANTERIOR AMYGDALOID NUCLEUS

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If a pathological process involves the limbic system it disturbs the cardiac rhythm [1-3, 5-11]. These disturbances may vary in character but their particular features and the conditions determining their origin are still largely unexplained. In the investigation described below this problem was studied in the light of views on the role of hyperactive determinant structures in disturbances of regulation of physiological systems and their conversion into pathological [4]. For this purpose a generator of pathologically enhanced excitation (GPEE) was created in the anterior amygdaloid nucleus (AAN), a formation belonging to the limbic system.

Stimulation of different zones of the amygdala is known to induce a predominantly bradycardic effect [15]. It must therefore be expected that hyperactivation of AAN as a result of the formation of a GPEE would cause changes in the cardiac rhythm.

EXPERIMENTAL METHOD

Experiments were carried out on 55 male rats weighing 150-200 g. Preparations for the experiment (tracheotomy, catheterization of the subclavian vein, fixation of the animal in a stereotaxic apparatus, and exposure of the cranium) were carried out under superficial pentobarbital sodium anesthesia (2.5 mg/100 g). After recovery from anesthesia the rats were immobilized with succinylcholine and artifically ventilated. The GPEE was created by microinjection of penicillin, which interferes with GABA-ergic control [12, 13, 16]. For this purpose penicillin in a dose of 100 i.u. in a volume of 0.2 µl was injected by means of a microinjector through a micropipet into one AAN of the animals (27 rats) taking coordinates from the stereotaxic atlas [14]: A = 6.5, L = 3.5, H = 7. In 10 animals of this group the ECG alone was recorded, in 17 rats the ECG together with electrical activity of AAN before and after injection of penicillin. Nichrome electrodes (150 µ), coated with enamel, with tips 50 µ in diameter uninsulated for a distance of 0.5 mm, were used. On the side of injection of penicillin the electrodes were inserted at an angle of 21° to the vertical axis (A = 3.5, L = 3, H = 7.7). In some experiments electrical activity also was recorded in the intact nucleus. In control experiments on seven animals physiological saline was injected into AAN in the same volumes as penicillin. To depress or eliminate the GPEE, cerebral hypothermia and electrical coagulation respectively were used. Cerebral hypothermia was created

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