their wide convergent properties they closely resemble neurons of the reticular type. For instance, in Burdach's nucleus – one of the relays to VPL – interaction between signals from the sensomotor cortex, nonspecific nuclei of the thalamus, cerebellum, and brain-stem reticular formation takes place on interneurons by means of which presynaptic inhibition of primary afferents is effected [14].

The thalamic relay nucleus carries out integration at a high level. Thus there is every reason to regard this nonspecific component as an intranuclear system which can determine the degree of tactile and pain perception and can regulate the modal spectrum of the sensory inflow to the cerebral cortex.

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CHANGES IN EFFERENT ACTIVITY IN THE RENAL AND SPLENIC NERVES DURING STIMULATION OF BULBAR RETICULAR NUCLEI

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KEY WORDS: paramedian and ventral reticular nuclei; acetylcholine; renal and splenic nerves.

The role of the medulla in the control of activity of the circulatory system is not in dispute. However, the problem of the functions of its various structures in this process is not yet clear. Since the distribution of the blood flow to the various vascular regions is controlled mainly by changes in the discharge frequency in the corresponding sympathetic nerves [1-3, 5-8], analysis of changes in efferent activity in the sympathetic nerve during stimulation of these structures is an essential preliminary to the assessment of the relative role of individual bulbar structures in the regulation of activity of the cardiovascular system.

Accordingly, the object of the investigation described below was to study efferent activity in the renal and splenic nerves during microinjection of acetylcholine (ACh) into the paramedian and ventral reticular nuclei.

EXPERIMENTAL METHOD

Cats were anesthetized with a mixture of chloralose (50 mg/kg) and pentobarbital (10 mg/kg), injected intraperitoneally. The stereotaxic coordinates of the paramedian and ventral reticular nuclei were determined

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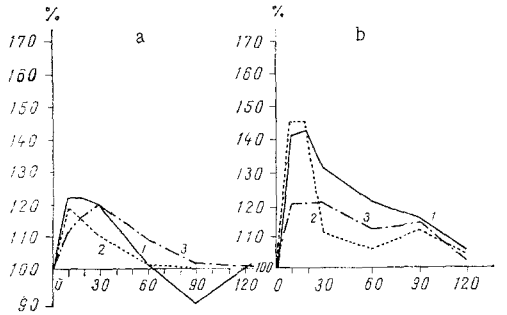

Fig. 1. Changes in efferent sympathetic activity and SAP in response to microinjection of ACh into paramedian reticular (a) and ventral reticular (b) nucleus. 1) Renal nerve; 2) splenic nerve; 3) SAP. Abscissa) time (in sec); ordinate) efferent sympathetic activity (in percent of control).

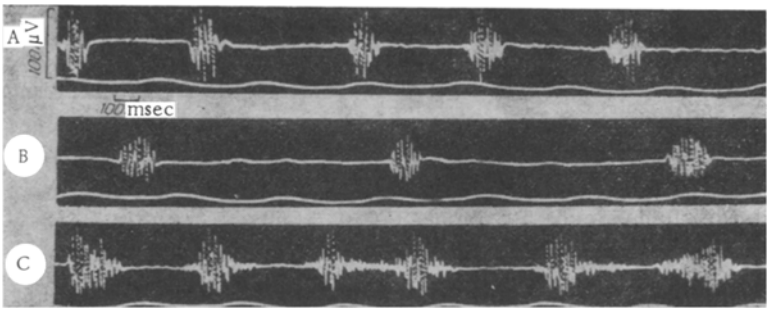

Fig. 2. Decrease in efferent sympathetic activity in renal nerve in response to stimulation of ventral reticular nucleus by ACh. A) Before stimulation, B) 30 sec after beginning of stimulation, C) 2 min after beginning of stimulation. Top trace shows activity in nerve, bottom trace SAP.

from the atlases [4, 9]. An injection of 2 μg ACh chloride in 0.0025-0.005 ml physiological saline was administered into the test nuclei. The location of the tip of the microinjector needle was verified in serial brain sections. The results were subjected to statistical analysis by Student's t-test in order to estimate the significance of differences.

EXPERIMENTAL RESULTS

The paramedian reticular nucleus was investigated in 38 experiments on 11 cats (Fig. 1a). Microinjections of ACh in 27 experiments were accompanied by a distinct increase in the frequency of volleys in the renal and splenic nerves on an average by 24% ($P < 0.05$) and 17% ($P < 0.05$), respectively, and also by a decrease in the duration of inhibition between volleys. Regular alternation of discharges with periods of inhibition were preserved under these circumstances. The maximal increase in frequency of the volleys was observed during the first 20 sec after the beginning of stimulation, and the level of activity returned to its initial value after 1 min. The systemic arterial pressure (SAP) rose from 100 ± 3.8 to 121 ± 2.5 mm Hg ($P < 0.01$), to reach a maximum on average 30 sec after the beginning of stimulation, and it was restored after 2 min. In 11 experiments stimulation of the nucleus was accompanied by a decrease in the frequency of volleys in the renal and splenic nerves by 17% ($P < 0.05$) and 14% ($P < 0.05$), respectively. The amplitude and duration of the volleys were not significantly changed. The level of SAP fell by 14% ($P < 0.05$).

The ventral reticular nucleus was investigated in 58 experiments on 22 cats (Fig. 1b). Stimulation of the nucleus in 47 experiments was accompanied by an increase in frequency of the volleys to both nerves on an average by 44% ($P < 0.01$) and 50% ($P < 0.01$), respectively. The most marked increase in the frequency of the volleys was observed during the first 20 sec after the beginning of stimulation. Later the discharge fre-

quency was reduced, and after 2 min it was the same as it was initially. Changes in the amplitude and duration of the discharges were not significant. Changes in SAP reached a maximum 10 sec after the beginning of stimulation. The initial level was restored after 2 min. In 11 experiments stimulation of the nucleus was accompanied (Fig. 2) by a reduction in the frequency of the volleys by 36% ($P < 0.01$) and 30% ($P < 0.05$). Under these circumstances the level of SAP fell from 121 ± 3.5 to 98 ± 6.3 mm Hg ($P < 0.05$).

Stimulation of the paramedian and ventral reticular nuclei thus leads to regular and marked changes in efferent activity of the renal and splenic nerves. The different character of the changes in efferent activity and SAP during stimulation of the nuclei by similar doses of ACh is evidence of the diffuse distribution of pressor and depressor neurons within these structures. Stimulation of the ventral reticular nucleus was accompanied by more marked changes in efferent activity in the splenic nerve, whereas microinjection of ACh into the paramedian reticular nucleus gave more marked changes in the renal nerve. Changes in efferent activity in the sympathetic nerve were much more marked after stimulation of the ventral reticular nucleus, possibly on account of the greater density of cholinergic neurons in this region than in the paramedian reticular nucleus.

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EFFECT OF LUTEINIZING HORMONE RELEASING HORMONE ON CALCIUM-ACCUMULATING CAPACITY OF RAT MYOCARDIAL MEMBRANES

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The writers showed previously that some releasing factors, notably luteinizing hormone releasing hormone (LRH), have a cardiotrophic as well as a hypophyseotrophic action [3]. Changes in the activity of Na,K-ATPase in the sarcolemma [4] and of NADH-oxidase activity of submitochondrial particles [1] under the influence of LRH are evidence that the mechanism of regulation of cardiac activity by this peptide evidently proceeds through its influence on the function of the various myocardial membranes. At the same time we know that LRH can alter the permeability of hypophyseal membranes for calcium [7]. During muscular contraction Ca^{++} is the link between excitation of the muscle and activation of myofibrillary ATPase.

Accordingly, the present investigation was undertaken to study the effect of LRH on the calcium-accumulating capacity of the sarcolemma, the sarcoplasmic reticulum, and the mitochondria of the rat myocardium.

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