When the mechanisms of this phenomenon are analyzed at least two possibilities must be considered. The first is that the powerful adrenergic effect associated with stress induces equal activation of lipid peroxidation, lipases, and phospholipases, i.e., it results in equal realization of the lipid triad of injury to biomembranes characteristic of stress [7]; correspondingly, an equal disturbance of the process of coupling of excitation with contraction leads to an equal decrease in the parameters of contractility of the heart muscle. However, this possibility is not in fact realistic, for equal injury to the sarcolemmal membranes of the cardiomyocytes, affecting a small and large relative area, must inevitably lead to equal (in relative values) disturbances of coupling processes and of myocardial contractile function. A different result was obtained in the present experiments.

The second possibility stems from the fact that prolonged hypokinesia is a chronic stress state which persists 2 months after the animals are placed in restraining cages [4, 5]. There is evidence that during repeated exposure to moderate stress, the resistance of animals [5] and, in particular, of their heart muscle [3], to the harmful action of stress rises significantly. Accordingly prolonged hypokinesia could be the cause of adaptation of this type and could lead to increased resistance of the animals' heart to stress injury.

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UNIT ACTIVITY OF THE GANGLION NODOSUM IN MYOCARDIAL ISCHEMIA

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The writers showed previously [1] that the discharge frequency of afferent neurons and interneurons in the bulbar cardiovascular center increases as a rule in myocardial ischemia (MI). These changes may evidently be linked not only with the increased flow of afferent impulses from the ischemic myocardium [3, 5-7], but also with changes in the firing pattern of different receptors and, in particular, from organs of the vascular and respiratory systems. The latter is particularly important because afferent impulses are sent to the bulbar cardiovascular center both from cardiovascular neurons and also from certain types of respiratory neurons of the ganglion nodosum [2].

The aim of this investigation was to study changes in the firing pattern of different types of ganglion nodosum neurons during the development of MI.

EXPERIMENTAL METHOD

Experiments were carried out on 39 cats of both sexes weighing 3-4 kg under pentobarbital anesthesia (30-40 mg/kg, intraperitoneally) with artificially ventilation by the Vita-I apparatus. The thorax and pericardium were opened and ligatures applied to the circumflex and anterior descending branches of the left coronary artery. Ischemia was produced by compressing the branches of the left coronary artery for 5-10 min. Unit activity in the gang-

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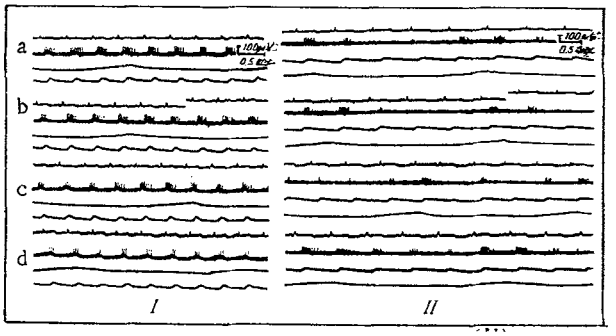

Fig. 1. Response of aortic (I) and cardiac (II) neurons to development of MI: a) spontaneous activity, b) compression of coronary artery, c, d) development of MI. From top to bottom; I) ECG (shift of isoelectric line corresponds to beginning of compression), unit activity, pneumogram, blood pressure; II) ECG (shift of isoelectric line corresponds to beginning of compression), unit activity, blood pressure, pneumogram.

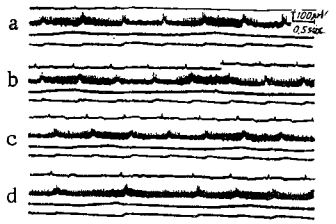

Fig. 2

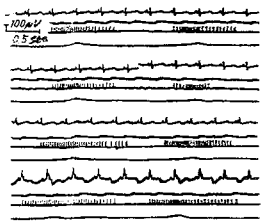

Fig. 3

Fig. 2. Response of cardiopulmonary neuron to development of MI. Legend as to Fig. 1, I.

Fig. 3. Response of inspiratory-expiratory neuron to development of MI. From top to bottom: ECG (shift of isoelectric line corresponds to beginning of compression), blood pressure, unit activity, penumogram. Remainder of legend as to Fig. 1.

lion nodosum was recorded by the method described previously [2]. Unit activity, the ECG in the standard leads I or II, the blood pressure in the femoral artery, and pneumogram were recorded on a four-channel M-42 myograph ("Medicor," Hungary). The activity of 92 neurons was analyzed: 27 cardiovascular (10 aortic and 17 cardiac), 25 cardiopulmonary, 14 inspiratory-expiratory, 12 late inspiratory, and 14 full inspiratory neurons. The significance of differences was determined by the chi-square test.

EXPERIMENTAL RESULTS

In 70% of cases aortic neurons changed their activity 10-15 sec after compression of the coronary sinus. With aggravation of ischemic injury to the myocardium these changes became more marked and were observed in all aortic neurons ($P < 0.05$; Fig. 1, I). Incidentally, the trend of responses of the aortic neurons coincided with the trend of changes in arterial pressure.

Cardiac neurons, like aortic, do not as a rule respond to compression of the coronary artery, but altered their firing pattern during the development of MI. Activity of 43% of cardiac neurons recorded increased progressively with increased severity of ischemic damage to the myocardium (Fig. 1, II). Discharges of 29% of neurons showed phasic changes; in some neurons, moreover, a period of activation preceded the period of inhibition, whereas in others these time relations were reversed. In 12% of cases cardiac neurons gradually inhibited their firing under these same conditions, and 12% of neurons did not change their firing pattern. These results are in agreement with the character of changes in spike activity in single fibers of the vagus nerve carrying information from cardiac receptors discovered under the same conditions [3-6].

Unlike the cardiovascular neurons, in 64% of cases cardiopulmonary neurons responded to compression of the coronary artery during the first second. Between 10 and 15 sec after application of the ligature all cardiopulmonary neurons changed their firing pattern ($P < 0.01$). Changes in activity of this group of neurons increased progressively with aggravation of MI ($P < 0.001$; Fig. 2). The trend of the changes in the firing pattern of the neurons during ischemia varied. In 36% of neurons the response of the cardiac component to MI consisted of an increase in the duration of the bursts and in the number of spikes in each burst, whereas in 31% of neurons changes in the cardiac component were of the opposute kind. Similar changes also were observed in the respiratory component.

Late inspiratory neurons responded in 42% of cases to compression of the coronary artery with effect from the first respiratory cycle after compression. With the development of MI the firing pattern of these neurons changed. From 10 to 15 sec after ligation of the coronary vessel a larger number of late inspiratory neurons became involved in the response, and in later stages of ischemia, when marked ischemic damage to the myocardium was present, this number continued to increase (80%). The firing patterns of the neurons changed differently under these circumstances: in some neurons there was an increase in duration of the bursts and in the number of spikes in the bursts; in others, on the contrary, the bursts became shorter and each burst contained fewer spikes.

Inspiratory-expiratory neurons changed their firing pattern in 50% of cases with effect from the first respiratory cycle after compression of the coronary artery. With development of ischemia the number of responding neurons increased. In some neurons the duration of the bursts and the number of spikes in the bursts increased (Fig. 3), whereas in others they decreased. Inspiratory-expiratory, like late inspiratory neurons, changed their firing pattern significantly during the development of myocardial ischemia ($P < 0.05$).

Full inspiratory neurons of the ganglion nodosum did not respond to compression of the coronary artery and did not change their firing pattern during the development of MI ($P < 0.001$).

Unlike cardiovascular neurons, the cardiopulmonary, late inspiratory, and inspiratory-expiratory neurons, which receive information from two systems of the body, thus changed their firing pattern with effect from the first second after compression of the coronary artery, evidence of the rapid activation of compensatory mechansims of respiration in response to an acute disturbance of the coronary blood flow. Later, as MI developed, the firing pattern of all groups of neurons of the ganglion nodosum, except full inspiratory neurons, changed significantly. It can accordingly be tentatively suggested that reorganization of the activity of the various types of neurons of the bulbar cardiovascular center during MI may be due to information arriving not only from cardiovascular neurons, but also from cardiopulmonary, late inspiratory, and inspiratory-expiratory neurons of the ganglion nodosum.

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