RESPONSE OF GANGLION NODOSUM NEURONS TO MYOCARDIAL ISCHEMIA COMPLICATED BY VENTRICULAR FIBRILLATION

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KEY WORDS: myocardial ischemia, ventricular fibrillation, respiratory neurons of the ganglion nodosum.

In myocardial ischemia (MI) neurons of the bulbar cardiovascular center change the character of their activity in different ways depending on whether it is or is not subsequently complicated by ventricular fibrillation. In cases when MI is complicated by ventricular fibrillation, bulbar cardiovascular afferent neurons and interneurons discharge at a slower frequency; under these circumstances the degree of coordination between activity of afferent and interneurons is reduced at different stages of development of the ischemic process [1]. The reasons for the appearance of such changes in neuronal activity have not yet been explained.

Since in MI uncomplicated by ventricular fibrillation activity not only of cardiovascular, but also of cardiopulmonary and certain types of respiratory neurons of the ganglion nodosum is modified [2], it was decided to study activity of these groups of neurons in MI complicated by the subsequence development of ventricular fibrillation.

EXPERIMENTAL METHODS

Experiments were carried out on 25 cats of both sexes weighing 3-4 kg, anesthetized with pentobarbital (30-40 mg/kg, intraperitoneally) and artificially ventilated. The thorax was opened in the 4th intercostal space and the heart exposed. Ligatures were applied beneath the circumflex and anterior descending branches of the left coronary artery. Fibrillation of the heart was induced by occlusion of the branches of the left coronary artery for up to

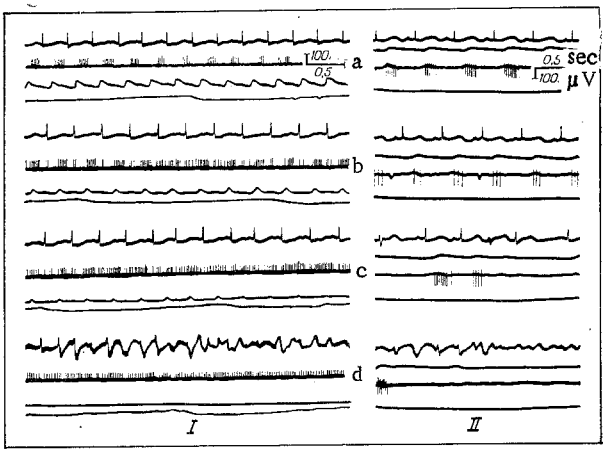

Fig. 1. Response of cardiac (I) and aortic (II) neurons to MI complicated by ventricular fibrillation. a) Spontaneous activity; b, c) established MI; d) development of ventricular fibrillation. From top to bottom: I) ECG, unit activity, BP, pneumogram; II) ECG, BP, unit activity, pneumogram.

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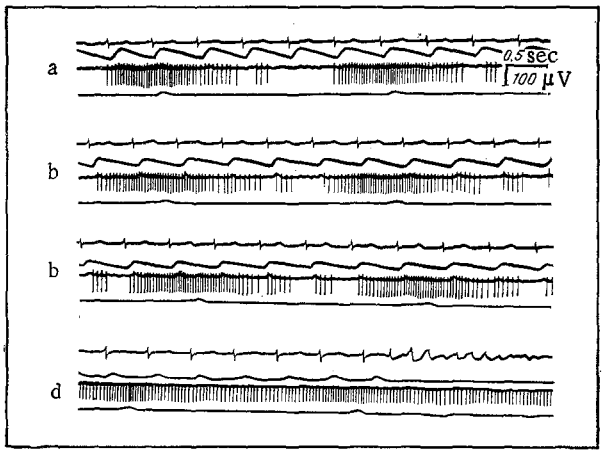

Fig. 2. Response of cardiopulmonary neuron to MI complicated by ventricular fibrillation. Legend as to Fig. 1, II.

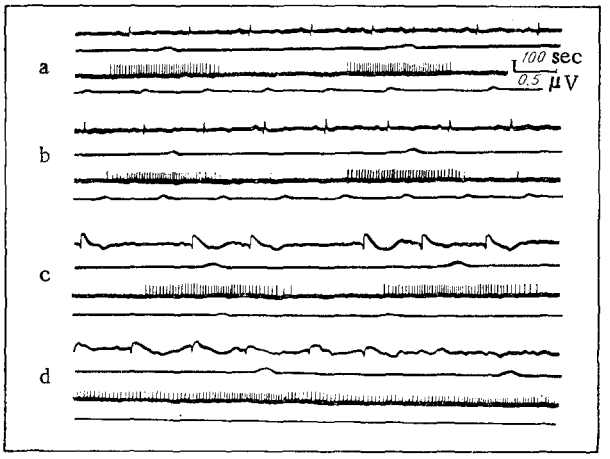

Fig. 3. Response of inspiratory-expiratory neuron to MI complicated by ventricular fibrillation. From top to bottom: ECG, pneumogram, unit activity, BP. Remainder of legend the same as to Fig. 1.

10 min. Unit activity in the ganglion nodosum was recorded by the method described previously [4-6]. Parallel with unit activity, the ECG in lead II and standard lead I, the blood pressure (BP) in the femoral artery, and the pneumogram also were recorded. The activity tested was amplified and recorded on a 4-channel M-42 myograph, and simultaneously on magnetic tape. Activity of 36 ganglion nodosum neurons was analyzed: 12 cardiovascular (five aortic and seven cardiac), nine inspiratory-expiratory, two late inspiratory and six complete inspiratory. The significance of differences was determined by the χ^2 test.

EXPERIMENTAL RESULTS

During the development of MI complicated by ventricular fibrillation, 60% of the aortic neurons had changed their firing pattern 10-15 sec after occlusion of the coronary artery. Later, with progression of the ischemic changes on the ECG and with changes in BP, virtually all the aortic neurons were involved in the response (Fig. 1, II).

Like the aortic neurons, in 67% of cases the cardiac neurons changed their firing pattern 10-15 sec after occlusion of the coronary artery. With progression of ischemic damage to the myocardium, unlike in MI uncomplicated by ventricular fibrillation, all cardiac neurons without exception changed the character of their activity, and 55% of cardiac neurons responded at all stages of MI by an increase in spike activity only (Fig. 1, I). Other workers [3] have observed marked activation of afferent impulsation in whole cardiac nerves during MI complicated by ventricular fibrillation.

In MI subsequently complicated by ventricular fibrillation cardiopulmonary neurons responded in 71% of cases to occlusion of the coronary artery. All cardiopulmonary neurons were involved in the response 10-15 sec after application of the ligature ($P < 0.05$). The cardiac (80%) and respiratory (43%) components of their activity were changed. With progression of MI, further changes took place in the firing pattern of the cardiopulmonary neurons ($P < 0.001$, Fig. 2). A response of the respiratory (58%) and cardiac (81%) components also was observed. The trend of the changes in activity of the cardiopulmonary neurons, just as in MI uncomplicated by ventricular fibrillation, differed in different experiments. In some cases (31%) the duration of the bursts of the cardiac component and the number of spikes in the bursts were reduced, whereas in other cases (51%) the changes were opposite in character. The respiratory component of some cardiopulmonary neurons shortened (20%) during the development of MI, whereas in others it lengthened (38%).

During MI subsequently leading to ventricular fibrillation, inspiratory-expiratory and late inspiratory neurons changed their firing pattern in 33% of cases in response to occlusion of the coronary artery from the first respiratory cycle after occlusion. Most inspiratory-expiratory neurons changed the character of their spike discharge 10-15 sec after application of the ligature. During development of MI, both uncomplicated and complicated by ventricular fibrillation, these changes increased progressively (Fig. 3). If the ST segment of the ECG was shifted, a change of activity was observed in 67% of neurons, and if the QRS complex was deformed, a change was observed in 73%. The trend of responses of these neurons to MI varied: the duration of the burst in some cases (36%) was reduced, whereas in others (40%) it was increased.

Complete inspiratory neurons did not respond to occlusion of the coronary artery in MI, whether complicated or not by ventricular fibrillation. As a rule no changes in their spike activity likewise were observed during the development of myocardial ischemia. In some cases, however, in the late stages of the disease, immediately preceding ventricular fibrillation, the activity of these neurons could change.

The clearest differences in the response of ganglion nodosum neurons were thus observed in activity of cardiovascular neurons. During MI complicated by ventricular fibrillation their spike activity increased considerably, unlike in MI uncomplicated by fibrillation. Meanwhile, under these conditions changes in spike activity of the cardiopulmonary and some types of respiratory neurons showed no significant differences. This fact is evidence that enhanced spike activity of cardiovascular neurons evidently plays an essential role in the modification of activity of the bulbar cardiovascular center.

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