ROLE OF LOCAL BLOCKING OF ECTOPIC EXCITATION IN THE MYOCARDIUM IN THE PREVENTION OF CARDIAC ARRHYTHMIAS

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Creation of a zone with modified bioelectrical properties (surface ring perfusion with KCl solution, ring dc polarization, injection of coagulating substances) around an artificial focus of ectopic excitation in the dog myocardium blocked the spread of ectopic excitation over the myocardium and prevented disturbances of the hemodynamic activity of the heart.

The role of differences in levels of excitability of neighboring structures in the heart in preventing the spread of ectopic excitation over the myocardium has been reported in the literature [1,3,8].

The present investigation was carried out to test this hypothesis experimentally.

EXPERIMENTAL METHOD

Experiments were carried out on 20 mongrel dogs weighing 12-15 kg. The thorax was opened in layers in the 4th left intercostal space under morphine-hexobarbital anesthesia and artificial respiration, and silver bipolar electrodes were inserted into the myocardium of the left ventricle. One electrode was sutured subepicardially in the region of the apex of the left ventricle. This was equipped with special devices to modify the bioelectrical properties of the subjacent structures of the heart (Fig. 1).

In the experiments of group 1 (7 animals) the electrode used (Fig. 1A) was constructed as follows: the two central poles of the electrode (inner electrode) were surrounded by a circular bipolar electrode by means of which the subjacent myocardium was polarized with a direct current of 2-3 V. In the experiments of group 2 (8 animals) an electrode was used (Fig. 1B) in which the two inner poles were surrounded by a perfusion channel to enable surface ring perfusion of the myocardium with 30% KCl solution to be carried out. A bipolar needle electrode was inserted through the channel into the myocardium. In both designs two bipolar silver electrodes (external 1 and 2) were also provided at the circumference. In the experiments of group 3 (5 animals) changes in the bioelectrical properties of the myocardial fibers were produced by injection of a coagulating mixture (96° ethyl alcohol and 10% neutral formalin solutions, 1 :1) into a ring of heart muscle tissue surrounding the bipolar electrodes.

The pressure in the left ventricle and femoral artery was recorded by means of "Barovar" electromanometers. The bioelectrical activity of various parts of the heart and blood pressure curves were recorded on a 6-channel ink-writing "Alvar" electrocardiograph. Stimuli from a "Disa-Multistim" electronic stimulator (5 V, 3-3.5 Hz) were applied to the myocardium.

EXPERIMENTAL RESULTS AND DISCUSSION

In all the experiments, before changes were produced in the bioelectrical properties of fibers surrounding the apical bipolar electrode, stimulation of the myocardium through this electrode led to a spread of ectopic excitation to the base of the left ventricle, which was recorded by the corresponding electrode (external), and this was accompanied by disturbance of the hemodynamic activity of the heart (Fig. 2A). A similar pattern was observed after stimulation with the "external" electrode (Fig. 2B).

A different pattern was observed when the myocardium was stimulated after the creation of a zone with sharply modified bioelectrical properties of the myocardial fibers around the "internal" electrode. Stimuli applied to the myocardium by means of "internal" electrode 1 were partly (Fig. 3A) or completely

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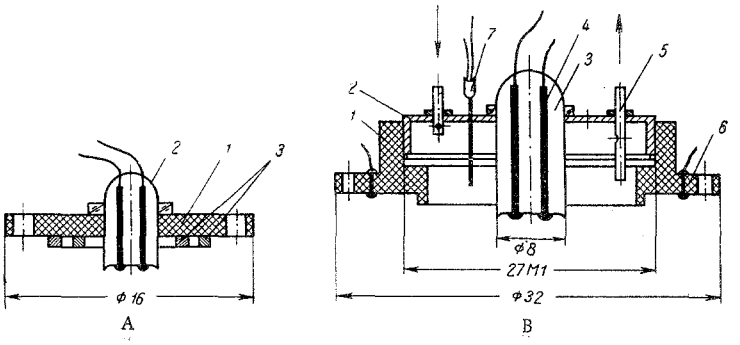

Fig. 1. Diagram of electrodes used to produce local blocking of ectopic excitation in the myocardium. A – by dc polarization; 1) base of electrode, 2) plug with central bipolar electrodes, 3) circular bipolar polarizing electrode; B – by surface ring perfusion of the myocardium with KCl solution: 1) base of electrode, 2) screw-on cover, 3) plastic plug, 4) central bipolar electrodes, 5) perfusion tube, 6) external bipolar electrode, 7) needle bipolar electrode (dimensions given in mm).

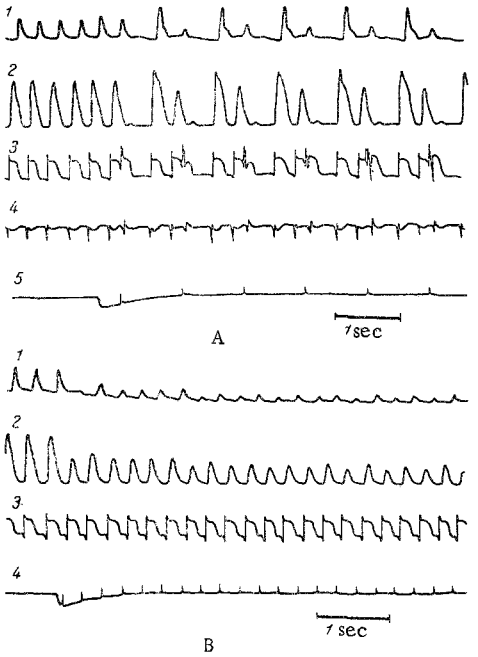

Fig. 2. Stimulation of myocardium before creation of local blocks to the conduction of ectopic excitation (by perfusion with KCl). A – stimulation through "internal" electrode: 1) pressure in femoral artery, 2) pressure in left ventricle, 3) electrogram recorded by "internal" electrode, 4) electrogram recorded by "external" electrode, 5) marker of stimulation (5 V, 1 Hz); B – stimulation through "external" electrode: 1) pressure in femoral artery, 2) pressure in left ventricle, 3) electrogram recorded by "internal" electrode, 4) marker of stimulation (5 V, 3 Hz).

(Fig. 3B and C) blocked at their point of application, no disturbances taking place as a result in the hemodynamic activity of the myocardium. When the myocardium was stimulated through the "external" electrode after the creation of a blocking zone, in every case the ectopic excitation spread to the zone of the "internal" electrode (Fig. 3D).

Stimulation of the myocardium through a needle bipolar electrode inserted superficially into the myocardium below the zone of perfusion with KCl did not lead to a spread of ectopic excitation, but if the needle electrode was inserted deeper, a spread of ectopic excitation and disturbance of the hemodynamic activity of the heart were observed.

Changes in the bioelectrical properties of fibers surrounding a focus of ectopic excitation thus prevent its spread from the blocked zone to the rest of the myocardium. During surface perfusion of an area of the myocardium with KCl solution, the resting potential is evidently lowered and the duration of the action potential shortened [4-7,10], the latter taking place on account of an increase in the gradient of the curve in phase 2, i.e., on account of a decrease in the duration of the "plateau" phase [8]. Meanwhile, before an action potential can spread over the myocardium, the duration of the "plateau" phase must not be below a particular critical value [9]. That is why in the present experiments, fibers surrounding the ectopic focus had apparently lost their ability to spread the action potential, as a result of which ectopic excitation could not pass through that part of the heart muscle.

The action of the coagulating mixture injected into the surface layers of the myocardium can be explained in a similar manner. As previous investigations showed [2,3], any alteration of the myocardium shortens the duration of the action potential in the myocardial fibers because they go into a state of energy debt.

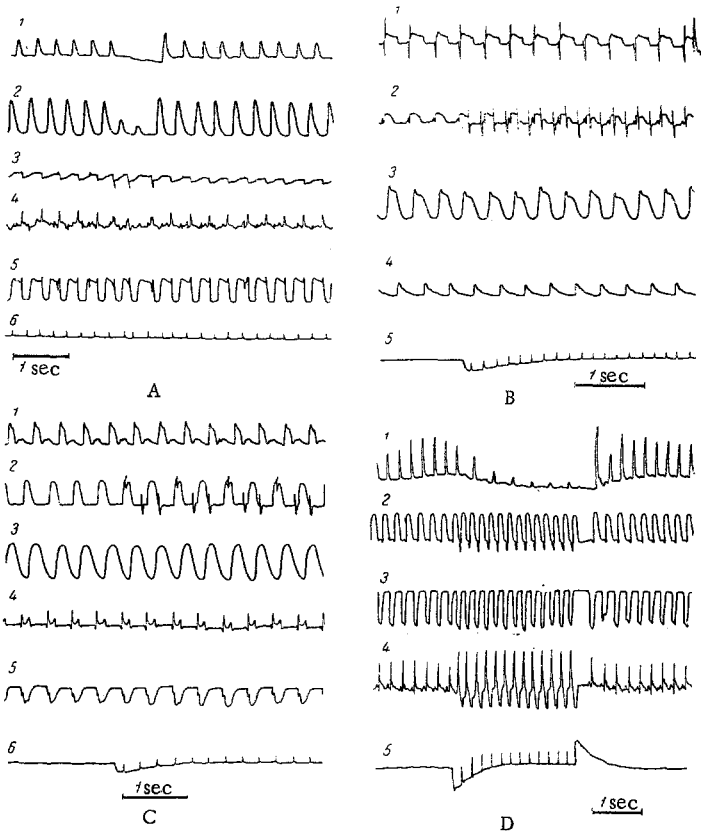

Fig. 3. Stimulation of myocardium after creation of blocks to ectopic excitation. A – incomplete block created by dc ring polarization of myocardium: 1) pressure in femoral artery, 2) pressure in left ventricle, 3) electrogram recorded by "external" electrode, 4) ECG lead III, 5) electrogram recorded by "internal" electrode 2, 6) marker of stimulation (5 V, 4 Hz); B – complete block created by injection of coagulating mixture into myocardium: 1) electrogram recorded by "external" electrode, 2) electrogram recorded by "internal" electrode 2, 3) pressure in left ventricle, 4) pressure in femoral artery, 5) marker of stimulation (5 V, 6 Hz); C – complete block created by surface perfusion of myocardium with KCl solution: 1) pressure in femoral artery, 2) electrogram recorded by "internal" electrode 2, 3) pressure in left ventricle, 4) electrogram recorded by "external" electrode, 5) electrogram recorded by needle electrode, 6) marker of stimulation (5 V, 3.5 Hz); D – absence of block (during perfusion with KCl) when myocardium stimulated through "external" electrode: 1) pressure in femoral artery, 2) electrogram recorded by "internal" electrode 1, 3) electrogram recorded by "internal" electrode 2, 4) ECG lead III, 5) marker of stimulation (5 V, 4 Hz).

The same mechanism can be considered to lie at the basis of the blocking action of the direct current applied through the bipolar ring electrode surrounding the "internal" electrode. Anodic polarization of myocardial fibers continued for a long period of time considerably shortens the duration of the action potential and reduces hyperpolarization of the membrane in phase 4 [8], thereby lowering excitability and conductivity of that particular fiber.

A block to ectopic excitation at the point of its origin of this type can thus take place under certain pathological conditions, such as in myocardial infarction, and it may play a life-saving role by preventing the development of severe cardiac arrhythmias [3].

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