EFFECT OF AN ELECTROMAGNETIC FIELD AT LOW TEMPERATURES ON THE CONDUCTING SYSTEM OF THE HEART

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The isolated hearts of rabbits and albino rats were cooled with solid carbon dioxide in an electromagnetic field to temperatures of −4 and −10°C. In most experiments, when the organs were reheated to normal temperature, the functional connection was restored (completely or partly) between the specific muscle of the heart and the myocardium. The synaptic connection between the specific muscle of the right atrium and the myocardium of the same atrium was the most resistant, while the connection between the left branch of the bundle of His and the myocardium of the left ventricle was functionally the most vulnerable.

The connection between the specific muscle and myocardium of the ventricles of warm-blooded animals has been studied for many years in the authors' laboratory [1, 2, 4-6]. Investigations have not only confirmed observations of Western and Japanese workers [7-11] regarding a possible functional block and recovery of the link between the specific muscle and myocardium, but have also yielded facts on the basis of which the existence of a functional synapse between these two muscular structures of the heart has been postulated [5, 6]. Work in this laboratory has shown that the specific muscle of the ventricles is not a static conducting system, but an actively contracting muscle tissue, capable of functioning even under unfavorable physical conditions [2, 3], as is seen particularly clearly when a complete block exists in the region of the functional synapse.

The object of the present investigation was to study the resistance of the synaptic connection between the specific muscle and myocardium after cooling the isolated heart to −4 and −10°C and during exposure to an electromagnetic field.

EXPERIMENTAL METHOD AND RESULTS

Experiments were carried out on 32 rabbits weighing 2-3 kg and 20 albino rats weighing 150-200 g. The rabbits were anesthetized with urethane (25%, intravenously), and the rats by inhalation of ether. The heart was removed within 1.5-3 min, and 10-20 min after isolation it was placed in the chamber of the apparatus and cooled by carbon dioxide from a cylinder to −4 or −10°C. In the experimental series (22 rabbits and 10 albino rats) an electromagnetic field (50 Hz, 360-600 Oe) was created in the chamber of the apparatus and maintained throughout the period of cooling.

In the control series (10 rabbits and 10 albino rats) cooling took place without the application of an electromagnetic field. The temperature of the heart was measured by a mercury thermometer introduced into the left ventricle through an incision in the auricle. The temperature of the heart was maintained at the assigned level (−4 or −10°C) for 15-20 min. Reheating to 12-20° took place in the same apparatus, after which the heart was taken from the chamber and placed in Ringer−Locke solution at 12-20°C. A cannula was introduced into the aorta and coronary perfusion begun with Ringer−Locke solution, saturated

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TABLE 1. Types of Recovery of Synaptic Communication between Specific Muscle and Myocardium of Isolated Heart after Cooling in an Electromagnetic Field to −4 and −10°C and Subsequent Rewarming

Species of animals	Number of animals	Type of synaptic communication				Contraction of specific muscle		Absence of functional manifestations
		1	2	3	4	Right or left branch	Region of sinoatrial node	
Rabbits	22	4	4	3	3	4	3	1
Rats	10	2	—	2	—	2	—	4

Legend: 1) Complete synaptic connection between specific muscle and myocardium of both ventricles (contractions of whole heart); 2) disturbance of synaptic connection in left ventricle (contractions of atria and right ventricle); 3) disturbance of synaptic connection between specific muscle and myocardium in both ventricles (contractions of atria); 4) focal manifestations of synaptic connections between individual area of the specific muscle and ventricular myocardium along course of descending branches of left or right coronary arteries.

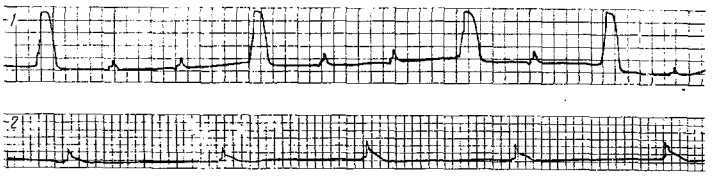

Fig. 1. Recovery of contractions of all parts of isolated (rabbit) heart rewarmed to 37° after cooling to −7°C. ECG recorded by needle electrodes connected to base and apex of heart (ÉKT-02 apparatus, frequency 50 Hz, sensitivity 10mm/mV, paper winding speed 50 mm/sec). Signs of myocardial hypoxia visible on ECG. 1) Experiment on March 25, 1968; 2) experiment on April 17, 1968.

with oxygen, at 38-39°C. Observations were kept on the heart for 1 h, after which the walls of the left and right ventricles were divided and the edges retracted above the interventricular septum (by Smirnov's method [4]) to allow observation of the branches of the bundle of His by means of a binocular loupe with a magnification of 12-15 times.

The state of function of the myocardium and of the specific muscle was determined by recording its electrical and mechanical activity. To enable the cardiac potentials to be differentiated, the ECG was recorded beforehand. When a straight line was obtained on the ECG, the electrodes for recording the activity of the specific muscle were connected and visual observations made through the binocular loupe. Fuller details of the method of recording the electrical and mechanical activity were described previously [3].

Neither the specific muscle nor the myocardium regained its electrical or mechanical function after rewarming of the heart in any experiment of the control series. Examination under the microscope revealed absence of contractions and of fibrillation of the Purkinje fibers [2, 6]. Visual observation during perfusion revealed transudation of the perfusion solution through the endocardium and epicardium, gross swelling of the heart, and pallor and flabbiness of the myocardium. These changes were considered to be due to death of cellular structures following crystallization of extra- and intracellular water under the influence of the subzero temperatures.

In only 5 of the 32 experiments in which cooling took place in an electromagnetic field was it impossible to restore the contractions of either the specific muscle or the myocardium after rewarming of the

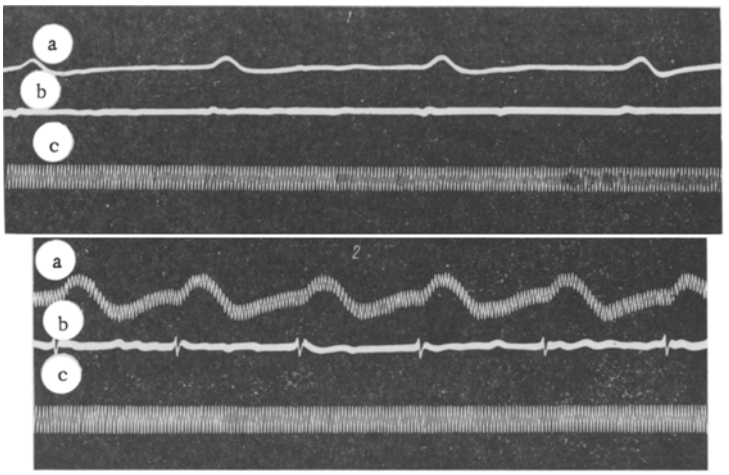

Fig. 2. Electrical activity and mechanical contractions of specific muscle of ventricles (myocardium of atria and ventricles not contracting) of isolated rabbit heart rewarmed to 37°C after cooling to −10°C. Experiment on January 30, 1968: 1) recording from right branch of bundle of His; 2) from left branch of bundle of His; A) mechanical activity; B) electrical activity; C) time marker.

heart by coronary perfusion for 1 h. In 9 cases rhythmic contractions of the specific muscle only were observed, in 18 experiments contractions of the myocardium were obtained in different parts of the heart (atrium and ventricle), while in 13 experiments synaptic communication was restored (completely or partly) between the specific muscle and myocardium of the ventricles (Table 1). These observations showed that the negative results could not be attributed entirely to the cooling temperature, because lowering the temperature of the heart to −7° did not cause death of the cardiac structures, and complete recovery of synaptic communication took place after rewarming of the heart (Fig. 1). Meanwhile, cooling the heart to −4°C in some cases produced changes in the tissues of such severity that no manifestations of function could be obtained even in the specific muscle, a more resistant structure of the heart than the myocardium.

In 18 experiments synaptic communication began to recover in the first 15 min of coronary perfusion, and in those cases in which contractions of all parts of the heart were observed (6 experiments), the atrial rhythm (100-120 contractions/min) was restored in the first 3-5 min. Later contractions of the right ventricle appeared (60-80 contractions/min), and these were followed (10-15th min of perfusion) by contractions of the left ventricle.

The isolated heart could still change the frequency of its contractions with a change in temperature of the perfusion solution (the frequency was reduced at a temperature of 32-33°) and in response to adrenalin and caffeine (injection of these drugs into the perfusion fluid increased the heart rate). In experiments in which it was impossible to restore completely the contractile function of all parts of the heart after coronary perfusion for 1 h (Table 1), disturbances of synaptic communication between the specific muscle and myocardium were found, to begin with in the left ventricle and later in the right ventricle also (Fig. 2).

In these experiments the place most vulnerable to the effects of subzero temperatures was apparently the connection between the left branch of the bundle of His and the myocardium of the left ventricle, followed by the junction between the right branch and the myocardium of the right ventricle. In some cases, a "focal" manifestation of functional communication was found between isolated areas of the specific muscle of the bundle branches and the ventricular myocardium, recorded visually as rhythmic contractions of isolated parts of the myocardium, as a rule along the course of the descending branches of the left and right coronary arteries.

Hence, on rewarming of the isolated heart of warm-blooded animals after cooling in an electromagnetic field to −4 or −10°C, recovery of the contractions of all its parts can take place. The functional disconnection between two structures of the heart observed under certain conditions, and the development of a block in isolated parts of the specific muscle are evidence of the heterogeneity of the homoiothermic heart.

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