tensive process compensatory mechanisms are established and the cardiovascular system is functioning at a new level with a new range of adaptational possibilities for the heart. Actually, however, the adaptation proves to be limited because the LV functions under conditions of a stably increasing load. In other words, the system becomes more rigid and inevitably involves the RV too, which thereby loses degrees of its freedom, i.e., its inherent capacity for a very plastic adaptation to changing conditions; as a result, it undergoes decompensation more rapidly than the LV.

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Effect of Direct (Galvanic) Current on the Ultrastructure of the Normal Isolated Perfused Heart and during Postischemic Reperfusion

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Galvanization by itself and as a means of therapeutic electrophoresis is used to treat cardiac patients with stenocardia of the 1st, 2nd, and 3rd functional classes and myocardial infarction also in the convalescence and postconvalescence stages [1]. There have been a few reports of heparin electrophoresis being used to prevent thromboembolic complications, and magnesium electrophoresis has been used to arrest stenocardia in acute myocardial infarction [3]. The clinical effect of direct current (DC) therapy in the treatment of different cardiac ischemic disorders is well known, but the mechanisms of the cardiac component involved in the action of this physical factor have not been clarified. There is hardly any information about the effect of DC on the subcellular organization of the normal and ischemic myocardium.

The aim of the present investigation was to study the effect of DC on the ultrastructure of the normal isolated perfused heart and in the course of its postischemic reperfusion.

MATERIALS AND METHODS

Experiments were carried out on 34 male Wistar rats weighing 280-320 g using a model of an iso-

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TABLE 1. Changes in Indexes of Quantitative Ultrastructure Morphometry of Isolated Rat Heart ($M \pm m$)

Experimental series	Number of Mc per cm²	Number of PTA Mc per cm²	Number of MEF Mc per cm²	Mean Mc S per EG, mm²	Mean S of 1 Mc, mm²	CESM
Normal perfusion, $n=7$, %	2425.53±189.16 100	70.21±6.54 100	2037.44±164.52 100	181.49±16.55 100	15.86±1.75 100	0.84±0.03 100
Normal perfusion + DC, $n=8$,%	2595.74±201.37 107	55.31±4.32* 78	2310.20±189.11 113	259.55±22.21* 143	21.27±2.18* 140	0.89±0.02 105

Note. Here and in Table 2 : an asterisk means that the difference is significant relative to the corresponding control value ($p<0.05$).

lated heart, beating under isotonic conditions (after Langendorff). After heparin premedication (i.p. 400 units) the animals were decapitated, and the heart was removed and perfused with Krebs-Henseleit physiological solution while spontaneously beating at 37°C. In the first variant the perfusion lasted 30 min and was performed after 15 min adaptation. In the second variant, after an adaptation period perfusion was terminated for 30 min, after which reperfusion was performed for 30 min. At the end of the experiment a biopsy of the left ventricle was performed for electron microscopic examination. Myocardial samples were embedded in araldite and ultrathin sections were prepared with an LKB-III ultramicrotome (Sweden) and studied under a UEMB-100A electron microscope (×20,000). An examination of cardiomyocyte (CMC) ultrastructure was performed in 3 regions : perinuclear, myofibril, and subsarcolemal. Together with the qualitative changes obtained, a quantitative analysis of some components of fine myocardial structure was performed after B.S.Paukov (1971), including the total number of mitochondria (Mc) per unit volume (1 cm²), the number of pathologically altered Mc (PTA Mc) and metabolically effective Mc, determined by the succinate dehydrogenase reaction (MEF Mc), measurement of the Mc mean area per electronogram (S Mc per EG mm²), the mean area of one mitochondrion (S 1 Mc, mm²), and the mean length of a sarcomere (Sm). The coefficient of the energy supply of the myocardium (CESM) was determined as described previously [2] in order to get more objective and accurate data on the effect of DC on the energetic processes in the myocardium. In the control series the heart was not exposed to DC. In the experimental series the heart was treated with DC with a strength of 0.4 mA, comparable to the previously defined current strength that acts upon the human myocardium during transcardiac galvanization. The DC treatment lasted the 30 min of the normal perfusion and the whole period of the total ischemia and reperfusion (60 min). Silver electrodes for DC were situated as follows: the negative electrode was fixed at the base of the aorta, while the positive electrode was placed on the apex cordis. A Potok-1 galvanization apparatus was the DC source. The data were processed with variational statistics using the Student's t test.

RESULTS

The qualitative and quantitative analysis of the ultrastructure of the normal and perfused myocardium revealed the following.

The DC-affected CMC contained hyperplastic Mc with condensed, compactly arranged cristae more often than in the control. Enlargement of the sarcoplasmic reticulum (SPR) cisternae was less pronounced. The number of glycogen (Gl) granules in the sarcoplasm was less than in the control, and they were situated near the SPR. In both

TABLE 2. Changes in Indexes of Quantitative Ultrastructure Morphometry of Isolated Rat Heart for Reperfusion ($M \pm m$)

Experimental series	Number of Mc per cm²	Number of PTA Mc per cm²	Number of MEF Mc per cm²	Mean Mc S per EG, mm²	Mean S of 1 Mc, mm²	Sarcomere length, cm	CESM
Total ischemia + reperfusion, $n=10$, %	2294.1±214.85 100	1110.0±99.81 100	1445.3±114.82 100	284.6±25.14 100	26.4±2.37 100	1.83±0.003 100	0.63±0.05 100
Total ischemia + reperfusion + DC, $n=9$, %	3856.3±289.21* 168	531.91±52.12* 48	3123.66±294.45 216	372.1±29.63* 130	20.53±1.97* 76	2.14±0.006* 117	0.81±0.03* 128

experimental series the myofibril (Mf) and nucleus structure were not significantly changed; there were no features of intra- and intercellular, pericapillary hydration.

The total number of DC-affected Mc and the number of MEF Mc per unit area remained unchanged. The number of PTA Mc diminished by 22%, and the average Mc area per EG and average S of a Mc rose by 43% and 40%, respectively. CESM did not change appreciably (Table 1).

The findings suggest that the DC produced a normalizing, membrane-stabilizing effect on the subcellular CMC organization in the isolated heart perfused at the normal temperature.

The mechanism of the DC membrane-stabilizing effect under these conditions remains in many respects unclear. However, it may be suggested that the current enhances the polarization both between the extra- and intracellular medium at the sarcolemma and on both sides of the membranes of the different intracellular compartments, such as Mc and SPR, and it has a pronounced effect on the electrogenic processes in the membranes themselves, causing their conformational and structural changes.

It is to be noted that the obtained hyperplasia of the intact Mc, the increase of the sarcosome area, and the spatial redistribution of the Gl granules, together with the absence of a significant dynamics of the CESM, attest to an adaptive trend of the structural changes in cardiac muscle treated with DC under conditions of preserved coronary perfusion. This creates the conditions for increasing the resistance of the myocardium to different adverse agents, in particular, to hypoxia and ischemia.

The DC-induced qualitative changes in the myocardium ultrastructure toward the end of reperfusion were less pronounced. There were no CMC with an altered sarcolemma, in contrast to the control series. Mc with features of destruction, vacuolization, and disorientation of the cristae appeared more seldom. The number of small Mc with condensed cristae increased. Phenomena of supercontraction, separation, and lysis of Mf were not well expressed. The structure of the SPR elements was more distinct and better preserved. Cell hyperhydration was not so evident in DC-treated case.

The quantitative analysis of the CMC ultrastructure at the 30th min of reperfusion showed that the total number of DC-treated Mc rose by 68%, and the number of PAT Mc fell by 52%. In spite of an increase of Mc S per EG by 30%, the S of one Mc decreased by 24% as a result of an increase of their total number owing to the fraction of small Mc, which correlates with the qualitative trend of Mc changes. The energetic supply of the cardiac muscle was markedly improved, as is evidenced by the increase of the MEF Mc by 116% and of the CESM by 28% . The increased effectiveness of the energy production induced by the DC normalized such an energy-dependent process as diastolic relaxation of the Mf, lowering the magnitude of their contracture changes, as is proved by the smaller number of Mf supercontracted bands and the increase in the mean sarcomere length by 17% (Table 2).

The findings demonstrate that the DC had a pronounced membrane-stabilizing effect on the CMC in the postischemic reperfusion of the isolated heart. Such an effect of the DC on the membranous CMC structures probably relates to the influence of this physical factor on the physicochemical properties and electrostatic relations between the integral membranous components, promoting a decrease of the conformational, spatial, and functional disturbances of the lipoprotein complexes. Improvement of the CMC membrane architectonics undoubtedly promotes the normalization of their functions in different compartments, leading to enhanced ATP synthesis and to normalization of the water-electrolyte exchange, the mechanism of cell volume control, and the function of the contractile apparatus.

The noted increase in the number of Mc due to the fraction of small sarcosomes testifies to stimulation of the intracellular repair processes in the myocardium damaged during ischemia-reperfusion, and, in particular, Mc division under the influence of this physical factor.

Thus, the DC, by stabilizing the CMC membranes, had a pronounced correcting effect on the leading component of pathogenesis of the ischemic and reperfused myocardium, providing for more successful and complete recovery of the metabolism and contractility of the cardiac muscle, and reducing the number of dead CMC.

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