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Effect of Transcardial Galvanization on the Microcirculatory Bed of the Periinfarcted Region in Experimental Myocardial Infarction

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Since the state of the microcirculatory bed (MCB) of cardiac muscle determines in many respects the nature of the adaptive processes and metabolic activity of cardiomyocytes, the mass of necrosis forming in acute myocardial infarction (MI) depends largely on the blood supply of the ischemic region [4], a factor which has to be taken into account in treatment aimed at minimizing ischemic damage to cardiac muscle during disturbed coronary circulation. As previously noted, the use of transcardial galvanization (TCG) in patients with stenocardia of functional class 1-2 improves the microcirculation in the underlying organs, increases fibrinolytic activity, and decreases blood coagulation (enhancing heparin tolerance) [2,3]. All this argues in favor of expanding the uses of TCG, including it in cardiac ischemia therapy, particularly in combined therapy of acute MI.

The aim of this investigation was to study the effects of TCG on MCB state in periinfarcted regions in experimental MI.

MATERIALS AND METHODS

Experiments were carried out on 30 male Wistar rats weighing 280-320 g. A model of experimental MI (EMI) was used. With the animals under nembutal anesthesia (20 mg/kg) and breathing naturally, the chest was opened at intercostal space 4-5, and the anterior descending coronary artery was ligated in its upper third. The operative wound was then tightly closed with sutures. For control an ECG was recorded in 12 standard leads after the initial narcosis and 1 h after MI simulation. There were 3 experimental groups: the first group (n=10) comprised sham-operated rats, the second

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TABLE 1. State of the Microcirculatory Bed of the Periinfarcted Region in Experimental Myocardial Infarction

Index	Normal myocardium	Control, 1 day EMI	Experiment, 1 day EMI+DC
S_c, c.u.	0.20±0.01	0.15±0.01*	0.18±0.005*,**
%	100	75	90
S_a, mm²	0.004±0.0002	0.0028±0.0002*	0.0034±0.0001*,**
%	100	70	85
N_c, c.u.	5010.0±181.02	3760.0±29.6*	4600.0±104.8*,**
%	100	75	90
D_c, m	5.70±0.38	7.13±0.49*	8.29±0.45**
%	100	124	145
D_u, m	28.45±1.55	50.92±4.76*	48.76±3.81*
%	100	178	171

Note. Asterisks refer to significance of differences ($p<0.05$): *) in relation to normal myocardium, **) in relation to control; c.u. – conventional units.

group ($n=10$) animals with a natural course of MI, and the third group ($n=10$) rats treated with TCG for 60 min with a current strength of 4 mA 1 and 12 h later MI simulation. The direct current source (DC) was Potok-1. All the animals were decapitated 1 day after EMI. The heart was fixed in 10 % neutral formalin and embedded in paraffin. For study of the capillary bed the PAS-reaction with amylase as a control was performed in paraffin sections. A morphometric analysis of the MCB of the periinfarcted myocardium was carried out using a grid with equally spaced points [1]. The specific capillary area (S_c, units), the absolute capillary area (S_a, mm²) in a visual field, the number of capillaries per unit area (N_c, units), the diameter of the capillaries (D_c, m), and the diameter of pericapillary ultracirculation of metabolites (D_u, m) were estimated. Results were processed statistically using Student t test.

RESULTS

As is shown in Table 1, there was a decrease compared to the normal myocardium of S_c by 25 % ($p<0.05$), S_a by 30 % ($p< 0.05$), and N_c by 25 % ($p< 0.05$) in the periinfarcted region by the end of the first day of the natural course of MI. The D_c, on the other hand, increased by 24 % ($p< 0.05$), and D_u by 78% ($p< 0.05$).

These changes of the quantitative indexes of MCB by the end of the first day of EMI indicate pronounced disturbances in the capillary circulation of the periinfarcted regions, as evidenced by the reduction of S_c and S_a, and the decrease of N_c. There is a direct correlation between the D_c increase and the distance across which oxygen and different metabolites diffuse from the capillaries to the sarcoplasm and mitochondria of the cardiomyocytes, owing to increased permeability of the capillary wall and pronounced tissue edema.

The MBC changes detected against the background of developing hyperfunction of the periinfarcted myocardium membrane stabilizing effect, and intensification of its metabolic processes promote a deficiency of the metabolic and energy-producing processes in preserved myocardium and aggravate the ischemia. This in turn leads to a further expansion of the area of damaged heart muscle.

TCG resulted by the end of the 1st day of EMI in comparison with nongalvanized animals in marked changes of the MCB quantitative indexes in the periinfarcted region. There was an increase of S_c, S_a, and N_c by 15%, and of D_c by 21% ($p<0.05$). D_u dropped 7% ($p>0.05$).

The findings testify to a positive effect of galvanic DC on the coronary circulation, leading to activation of the capillary bed function and improvement of metabolite exchange between the capillaries and cardiomyocytes in ischemized, periinfarcted regions of cardiac muscle.

Such changes of the periinfarcted MBC induced by TCG treatment are probably due to the membrane stabilizing effect of DC and to the maintenance of the water-electrolyte balance and the control mechanisms of metabolism, which lead to a decrease of capillary wall permeability, a reduction of intra- and intercellular hyperhydration, and, as a result, an enlargement of the MCB area, an increase of the number of reserve capillaries opening, and a lessened distance of oxygen and metabolite diffusion from the capillaries to the cardiomyocytes. These shifts provide favorable conditions for the still-viable myocardium and allow for stabilization of the infarcted region. Thus, there is good justification of using TCG in combined therapy aimed at limiting the damaged zone in MI patients in the acute stage of the disease.

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Effects of Weightlessness on Amphibians
I. The Ultimobranchial Body

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This is the first in a series of articles describing our studies, extending over many years, into the effects of short-term space flights on various amphibian organs and systems, as exemplified by the newt *Pleurodele waltlii*.

During a space flight, anemia develops and signs of skeletal demineralization appear both in astronauts and in experimental animals [4,5]. These abnormalities may be caused by disorders of calcium and phosphorus metabolism at the cellular, tissue, organic, and/or systemic levels. The most important role in the regulation of mineral metabolism is played by the synthesis of calcitonin, a hormone that controls the mechanism of calcium binding in vertebrates. In mammals, including man, calcitonin-secreting cells (calcitoninocytes, or C cells) become incorporated into the thyroid gland during early stages of development [3] to be diffusely arranged among the thyroid follicles, which complicates their quantitative evaluation and makes impossible the analysis of nondifferentiated cells. Attempts at postflight analysis of C cells in mammals have failed to produce unequivocal results, although their secretory activity has been shown to deviate from normal [6]. To assess the impact of weightlessness on calcitonin secretion, amphibians were used as test animals [2]. The ultimobranchial body or gland (ULT) in amphibians is a distinct organ that is anatomically separated from the thyroid gland and is largely composed of C cells aggregated into follicles and surrounded by a dense network of capillaries arising from the arterial arch adjacent to the ULT. This gland contains both secretory and nonsecretory cells. Being an anatomically separate and asymmetrical organ that comprises cells in various phases of differentiation, the amphibian ULT appears to be an optimal model for studying changes in calcitonin secretion brought about by weightlessness.

The purpose of the present study was to examine morphological changes in the ULT of the newt *Pleurodeles waltlii* after a space flight and in particular during the readaptation period.

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MATERIALS AND METHODS

In this study, 32 young adult *Pleurodeles waltlii* newts that had been on board a biosatellite (Kosmos 1887, Kosmos 2044, Bion, or Foton - a to-

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