MORPHOLOGICAL CHANGES IN THE MYOCARDIUM AND SKELETAL MUSCULATURE WITH EXTREME PHYSICAL LOADINGS UNDER EXPERIMENTAL CONDITIONS

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Recently, the interest in functional and morphological myocardial alterations resulting from long-lasting and exhausting physical loading increases significantly (7—9). There are also single, predominantly functional, biochemical and ultrastructural investigations of the striated musculature of the lower limbs in Marathon runners (10, 11). However, parallel morphological studies of both myocardium and skeletal striated musculature under conditions of long-lasting exhausting physical loading are still absent.

The purpose of the present work is to study the morphological alterations in the myocardium and striated musculature with extreme physical loading and the influence of some substances improving protein metabolism.

Material and methods

A total of 66 white rats of both sexes with mean body weight at the beginning of the trial of 150 — 160 g were morphologically studied. All the animals were physically loaded by means of swimming in cold water (at 10°C) after the method of Avakyan and Shiryanin (1977). Preliminary testing of untrained animals showed an average endurance of 28 min. Experimental animals of all the groups underwent training for 15 or 20 days twice daily at every 5 hours with gradually increasing training duration from 10 up to 40 min.

Animals were divided into three groups. Each group consisted of three subgroups as followed:

First group (19 animals) with 20 training days including: Ist a) subgroup (5 rats) — they were given perorally a preparation based on protein hydrolysate «Hydroprot» — «0—80—G» at a dose of 60μ g/100 g b. w.

Ist b) subgroup (6 rats) — they were given perorally Bionabol at a dose of 30 μg/100 g b. w.

Ist c) subgroup (8 rats) — they were given placebo.

Second group (23 animals) with 15 training days including:

IInd a) subgroup (8 rats) — treatment with «0—90—G» at a dose of 12μ g/100 g b. w.

IInd b) subgroup (7 rats) — treatment with Bionabol at a dose of 30μ g/.100 g b. w.

IInd c) subgroup (8 rats) — given placebo.

Third group (24 animals) with 20 training days including:

IIIrd a) subgroup — the same as IInd a) subgroup

III[rd] b) subgroup (8 rats) — the same as II[nd] b) subgroup
III[rd] c) subgroup — the same as II[nd] c) subgroup.

Animals from the first and second groups continued to swim after the end of the experiment until complete exhaustion and death (for 5 — 11 hours in various groups). Animals from the third group were put in hypobaric chamber with a pressure of 0.8 atmospheres (corresponding to 13 873 m altitude) until death (for 9 — 15 min).

Material for histologic examination from visceral organs and skeletal musculature of shoulder girdle was taken immediately after death. Paraffin sections were stained with HE, Azan after Kruchaj, toluidine blau at pH 2 and 4, PAS, PTAH and after Lee for fuxinophilic necroses. Myocardial pieces were fixed in glutar aldehyde and osmium acid and embedded in Durcopan to be electron microscopically examined.

Results and discussion

The morphological investigation of the myocardium and skeletal musculature reveals one-type morphological alterations with certain quantitative differences between single groups.

Myocyte hypertrophy (thickness up to 20 μ m) and ischemic changes can be established in the myocardium. Ischemia is manifested by myofibrillar undulations and dispersed fuxinophilic necroses mainly in subendocardial zones (fig. 1). Besides in 12 animals (in 18 per cent of them) microinfarctions of different duration are found out. Some of these infarctions undergo an organization process.

Ultrastructurally, an outlined mitochondrial hyperplasia and hypertrophia in the myocardium comes to the fore. Mitochondrial arrangement in relation to myofibrils is destroyed. The count of mitochondrial cristae increases. However, in some places cristolysis and matrix lightening can be established. Besides there is a subsarcolemmal oedema, appearance of single myelin figures and lipid inclusions as well as endoplasmic reticulum dilatation (fig. 2). Glycogen granulae in myocyte cytoplasm are strongly reduced and even absent. Nuclei possess deep invaginations of the nuclear membrane and marginally located chromatin. There are severe destructive shanges including also a complete necrosis in single myocytes.

Capillary endothelial cells show an increased pinocytotic activity, vacuolization and formation of luminal protrusions sometimes completely remoted from endothelial cells. Sludge and thrombocyte aggregations are observed as manifestation of rheological disturbances.

Severe morphological alterations are sound out in the striated musculature of the shoulder girdle. It is the musculature with the most intensive loading in the experiment carried out. Myofibrils are hypertrophic and their thickness reaches here and there up to 45 μ m. Manifested ischemic changes are also observed — contracture changes, disappearing of striatedness, fuxinophilic necroses and even almost complete myocyte destruction in single regions (fig. 3).

Comparison between single groups demonstrates that myocardial infarctions (i. e. microinfarctions) develop more often in animals given placebo (e. g., there are 4 myocardial infarctions in the subgroup I-c (including a total of 8 rats) than in animals given «0—80—G» (in the subgroup I-a (one rat out of

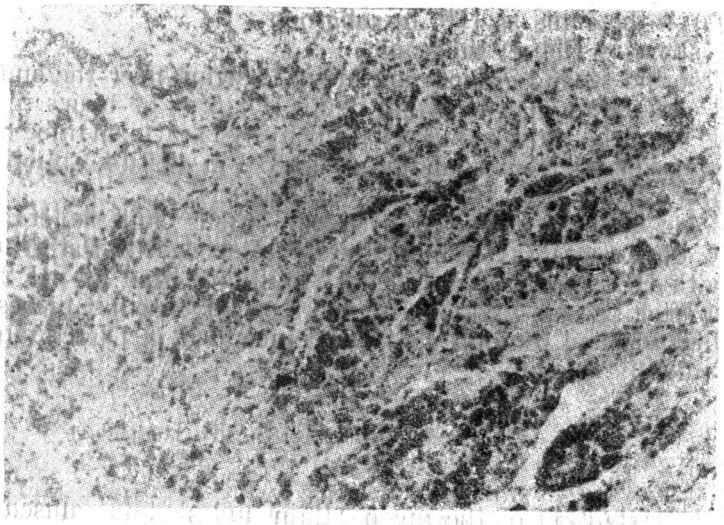

Fig. 1. Scattered fuxinophilic necroses mainly in the subendocardial zones of the myocardium. Stain after Lee. Magn. 10 x 6.3.

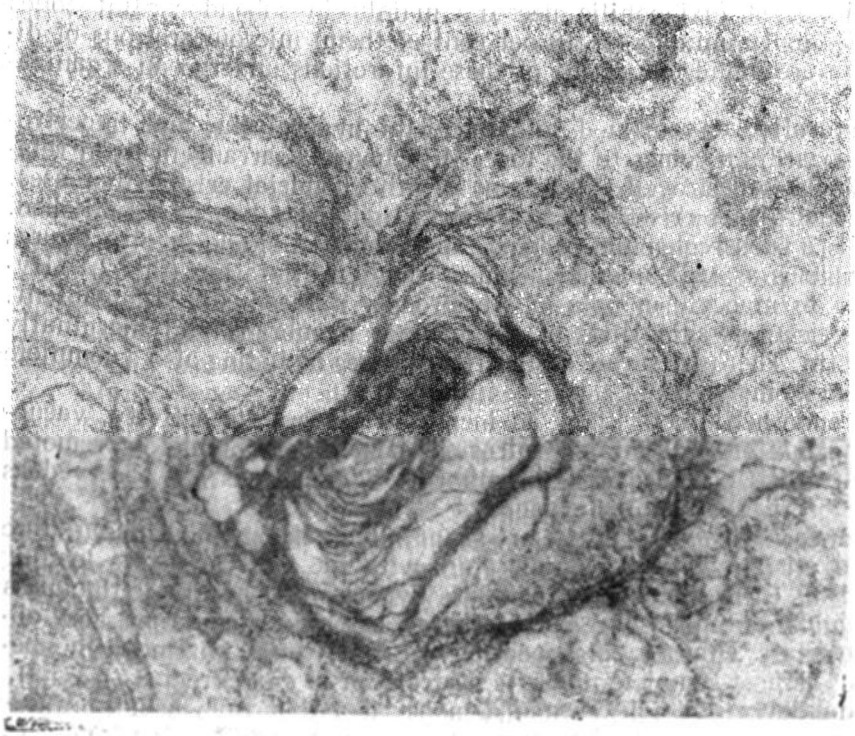

Fig. 2. Myelin-like body formation in the cytoplasm of myocardial myocytes. Electronogram. Magn. x 40000.

5 has a microinfarction) and in rats given Bionabol (in the subgroup I-b) (no infarctions are found).

There are certain differences between animals from various groups when maximal endurance is concerned. Endurance is the strongest one in animals from the first group and given Bionabol (for 11. 16 hours) or placebo (for 10

Fig. 3. Contracture alterations, disappearance of striatedness and fuxinophilic necroses in the skeletal musculature. Stain after Lee. Magn. 10 x 16.

hours) during a 20-day training. Differences are smaller in the second group (with a 15-day training). Animals given Bionabol show the strongest endurance, too (7.26 hours long).

These results obtained show that extreme physical loading for many hours induces severe irreversible structural damages not only in the myocardium but also in the skeletal musculature of trained rats.

It is known that sudden death can set in in Marathon runners. Echocardiographically, data about depression of left ventricular contractile function combined with a sharp serum creatinine-kinase level increase in such athletes has been established. It is accepted that creatinine-kinase increase is due to the damage of skeletal musculature mainly (8). On the other hand, some experimental investigations on untrained rats (7, 8) immediately after exhausting swimming (about 3 hours long at water temperature of 32°C) indicate that in myocardium interstitial oedema, swelling and oedema of mitochondria and contracture changes in sarcomeres without necroses set in.

In the present study we found out that despite preliminary training exhausting and long-lasting (for 5—11 hours) swimming in cold water (at .0°C) caused considerably more severe alterations including even dispersed fuxionophilic necrosses in the myocardium and striated musculature.

Undoubtedly, some of the changes observed such as mitochondrial hyperplasia and well-outlined microinfarctions some of which are already at the stage of organization have set in during the intensive training process.

Morphological myocardial alterations do not significantly differ from lesions occurred after severe hypoxia, shock, and under hypobaric conditions (2—4,6). Severe hypoxia presents probably a common pathogenetic factor in all these states. Long-lasting exhausting physical loading combined with an increase of the requirements of oxygen in myocardium and striated musculature on the background of severe metabolic changes, acidosis and hypercatecholaminaemia (6) makes these organs particularly vulnerable. Cold water acting as a stress factor and contributing to an increased liberation of endogenous catecholamines plays a certain role, too. Severe microcirculatory distyrbances in visceral organs resulting in formation of microthrombi with different localization in 38 per cent of the animals (unpublished data) thus arguing for development of intravascular coagulation are undoubtedly important, too (5). All that enables us to accept that severe ischemic changes in myocardium and striated musculature have a complex genesis. Both microcirculatory and metabolic disorders combined with hypercatecholaminaemia intensify hypoxia which appears during long-lasting physical loading and present the most important factors in this genesis.

Our results demonstrate also that during extreme long-lasting phy sical loading means improving protein metabolism do not influence essentially upon the degree of lesions, i. e. their protective effect is reduced to a minimal level. These morphological alteration in myocardium and skeletal musculature are irrevocable and, therefore, it cannot be expected that they can be influenced by means which improve protein metabolism.

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МОРФОЛОГИЧЕСКИЕ ИЗМЕНЕНИЯ МИОКАРДА И ПОПЕРЕЧНО-ПОЛОСАТОЙ МУСКУЛАТУРЫ ПРИ ЭКСТРЕМАЛЬНЫХ ФИЗИЧЕСКИХ НАГРУЗКАХ В ЭКСПЕРИМЕНТАЛЬНЫХ УСЛОВИЯХ

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РЕЗЮМЕ

Морфологически исследовано 66 белых крыс, тренированных предварительно плавать в течение 15—20 дней. Животные были распределны в три основные группы: получавшие препарат на базе а) белкового гидролизата («0—80—Г»), б) бионабола, в) плацебо. После окончания тренировочного процесса они оставлялись плавать до полного истощения и смерти, или же помещались в гипобарическую камеру до наступления смерти вследствие асфиксии.

В сердечной мышце животных всех групп обнаруживаются значительные ультраструктурные изменения, включающие в себя гиперплазию митохондриев, дилатацию саркоплазматического ретикулума, появление липидных вакуол и миелоноподобных фигур. Методом светового микроскопирования устанавливаются фуксинофильные некрозы, а у некоторых животных и микроинфаркты различной давности. В поперечно-полосатой скелетной мускулатуре также наблюдается фуксинофильные некрозы и тяжелые контрактурные изменения.

Полученные результаты показывают, что при продолжительной экстремальной физической нагрузке в миокарде и скелетной мускулатуре наступают необратимые морфологические увреждения, которые невозможно предупредить средствами, способствующими улучшению белкового обмена организма.