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Review Article

Limiting stress-induced myocardial damage by Adapting the organism to physical exertion

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Abstract

The formation of a certain systemic structural "trace" of adaptation to physical load is the basis for increasing the performance of the organism. On the other hand, it is known that adaptation to physical activity has both positive and negative cross-effects. The existence of positive cross-effects of adaptation to physical activity is that it increases the body's resistance not only to physical activity, but also to the action of other environmental factors and diseases, i.e. it is a means of preventing or correcting damage caused by these factors. This effect of adaptation has recently attracted increasing attention. The above suggests that adaptation to dosed exercise is a important factor in preventing or limiting stress-induced cardiac injury.

Keywords: myocardial damage; adapting; physical exertion

Introduction

The formation of a certain systemic structural "trace" of adaptation to physical load is the basis for increasing the performance of the organism. On the other hand, it is known that adaptation to physical activity has both positive and negative cross-effects. The existence of positive cross-effects of adaptation to physical activity is that it increases the body's resistance not only to physical activity, but also to the action of other environmental factors and diseases, i.e. it is a means of preventing or correcting damage caused by these factors. This effect of adaptation has recently attracted increasing attention. However, for successful and safe use of training for the prevention of exacerbations and treatment of diseases of the circulatory system, etc., along with an understanding of the pathogenesis of a particular disease, it is necessary to have a clear understanding of the degree of suitability of a particular type of training to influence this pathogenesis. In this regard, when considering various cross-effects of training, some scientists have revealed the connection of these effects with the peculiarities of the structural basis of adaptation. However, in the present communication, we will pay more attention to the consideration of the most essential effect of training for humans, namely, the increase in the body's resistance to factors that cause damage to the heart and circulatory system, among which stressor situations and ischemia occupy an important place. The influence of physically active lifestyle, training to physical loads on the resistance of human organism was analyzed in trained and untrained people on the expression of stress-response in response to 4 types of stressors: cold stress test (immersion of feet in ice water for l min); physical load, standard and to exhaustion; passive psychological stress (watching a film causing irritation, depression, anger); active psychological stress (solving a problem in conflict conditions). It was found out that people with better physical training had significantly lower stress reaction to 3 types of stress out of 4 (reaction to active psychological stress-test did not depend on the level of training) than untrained people. This was expressed in a smaller increase in heart rate and BP, as well as a smaller "release" of catecholamines into the blood. It is significant that in the most trained people at the action of physical test-load the reduced reaction was observed in response to the load of standard intensity; at the maximum load ("up to failure") they, on the contrary, had more powerful "release" of catecholamines and a greater rise in heart rate than in untrained people at the load "up to failure". This is due to the fact that in trained people the value of maximum physical work was much greater than that which could be overcome by untrained people, and its performance was ensured by greater mobilization of the organism. Thus, it can be noted that under the action of the same stressor factors the stress-response of the organism in people trained to physical loads is less pronounced than in sedentary, untrained people, and consequently their resistance to stressor influences is higher. The preventive effect of adaptation to physical exercise was also observed in the study of cardiac contractile dysfunctions caused by stressors. Adaptation to physical loads can play an important role as a means of prevention of cardiac contractile function disorders associated with cationic shifts in myocardium in cardiovascular diseases and as a means of prevention of potentialization of these disorders under stress. When analyzing the mechanisms of prophylactic cross-effect it should be kept in mind that under stressor influence, intensive and prolonged influence of catecholamines on the heart leads to excessive activation of free-radical oxidation, including lipid peroxidation, the products of which damage the membranes of cardiomyocytes and cells of the cardiac conducting system. This leads to disruption of mechanisms responsible for energy supply of cardiomyocytes and ion transport. Thus, in particular, the resulting damage of lipid bilayer of cardiomyocyte membranes and loss of sialic acid by glycocalyx lead to increase of membrane permeability for Ca-channels, decrease of Ca2+ content in phospholipid sites of its binding in sarcolemma, impaired ability of membranes to bind Ca2+ and, in general, to impaired transport of this cation in cardiomyocytes and destabilization of calcium homeostasis of cardiac muscle[8]. These changes together cause disturbance of excitation, contraction and relaxation of cardiomyocytes and, as a consequence, depression of amplitude and velocity of myocardial contraction and relaxation. In this regard, it can be assumed that the preventive anti-stressor

effect of adaptation to physical exercise is primarily due to the prevention of stressor activation of free-radical oxidation of lipids. Due to what can the limitation of this activation in the adapted organism be realized? Firstly, due to the fact that in such an organism, the stress reaction arising in response to the impact of environmental factors is expressed to a much lesser extent than in the untrained organism. Accordingly, in a trained organism, the "release" of catecholamines into the blood in the executive organs, including the heart, is significantly reduced during stress and, as a consequence, their activating effect on the processes of free-radical oxidation is limited. Secondly, in the adapted organism the capacity of antioxidant enzyme systems is increased, which can also limit the activation of lipid peroxidation under stress. Indeed, in the process of adaptation to endurance exercise, which includes swimming, the activity of antioxidant enzymes in skeletal muscles increases, which is accompanied by a less pronounced, than in untrained people, activation of free-radical oxidation that develops at maximum physical loads. Similar relations are observed in the myocardium of trepinated people; the increase in the activity of antioxidant enzymes in it is accompanied by the absence in these people of cardiomyocyte membrane damage and fermentemia, which naturally occur at maximum physical loads in non-adapted people. Thus, a significant role in the considered preventive effect of training under stressor influences along with the reduction of stress-response severity is played by the increase in functional capabilities of stress-limiting antioxidant system in myocardium, which develops in the process of adaptation. As shown above, stress increases the dependence of cardiomyocytes on changes in the concentration of Ca2+ in the intracellular medium and sensitivity to the action of competitors of Ca2+ for binding sites on membranes, i.e. stress reduces the ability of membrane mechanisms to bind and transport Ca2+[1,10]. Since these phenomena are directly related to the state of membrane mechanisms responsible for Ca transport and binding2+ , it can be assumed that the protective effect of adaptation is due to some specific changes occurring during exercise in the lipid bilayer or glycocalyx of sarcolemma and sarcoplasmic reticulum membranes (SRMs) of cardiomyocytes that increase the capacity of Ca2+ binding and transport mechanisms[11]. In this context, we want to emphasize that the role of adaptation to exercise is capable of preventing or limiting the stress response and, consequently, its damaging effects, it can be assumed that such adaptation can also prevent stressor-induced damage to the coronary bed and impairment of its adaptive response to ischemia. This position is particularly important for understanding the protective effects of adaptation in acute ischemia and myocardial infarction. Summarizing the above, we can conclude that certain components of the branched structural "trace" of this adaptation underlie the cross- prophylactic effect of adaptation to physical loads in cardiac contractile dysfunction caused by stressor exposure. This is, first of all, the adaptation restructuring of central and peripheral regulatory mechanisms, leading to a more economical functioning of the stress- realizing adrenergic system under extreme influences and, as a consequence, to the limitation of the stress response. This restructuring leads, in particular, to an increase in the activity of the opioid peptide system, an important stress-limiting system, which also contributes to limiting the stress response in adapted individuals. Second, is the increased capacity of the antioxidant system in the myocardium, which inhibits lipid peroxidation and therefore limits the activation of this process and the damaging effects of its products on cardiomyocytes. Thirdly, these structural changes formed in the process of adaptation at the level of cardiomyocyte membranes, which lead to an increase in the the level of cardiomyocyte membranes, which lead to an increase in the power of mechanisms responsible for binding and transport of Ca2+ , increase in the resistance of membranes to ionic loads and the damaging effect of products of activation of lipid peroxidation (POL abbreviation to enter at the first mention). The preventive effect of exercise training in cardiovascular diseases is characterized by two main features: 1) preliminary adaptation of the organism to physical loads can contribute to an easier course of the disease, for example, already "accomplished" myocardial infarction or acute transient ischemia, and faster recovery; 2) training is a factor that prevents the very occurrence of the disease, which is statistically expressed by a higher incidence of cardiovascular and other diseases among persons untrained in physical loads[9]. These features of adaptation are associated to a large extent with a decrease in the number of patients with cardiovascular and other diseases. Participation in leisure-time physical activity, even below recommended levels, has been shown to be associated with a lower risk of mortality compared with participation in no leisure-time physical activity [6]. It has also been shown that individuals who regularly engage in dynamic leisure-time physical activity have twice as few first myocardial infarctions and fatal cases of acute coronary insufficiency as those who rarely spend their leisure time actively[2]. Due to the fact that in ischemic lesions caused by myocardial infarction, an increased load falls on the non-ischemic parts of the heart. Thus, the protective effect of adaptation in ischemic heart damage can be realized by increasing the resistance of non-ischemic myocardial sections to the increased load. Thus, the preventive cross-effect of training in ischemic heart injuries is provided mainly due to the following components of the structural "trace" of adaptation. First, due to the enhancement of anti-stressor components, which limit the realization of the stressor link of the pathogenesis of these lesions. It should be emphasized that the increase in the power of antioxidant system in myocardium limits the activation of free-radical oxidation caused both by stress reaction accompanying ischemic impact and ischemia itself. Secondly, a significant place in ensuring the preventive effect of training is occupied by the components of the "trace", which increase the power of the mechanisms responsible for the blood supply to the heart muscle and its energy supply. These are, first of all, an increase in the density of coronary vessels per unit volume of myocardium and growth of coronary channel capacity, whic are realized in the process of adaptation due to the new formation o arterioles, capillaries and collaterals. As a result, the coronary reserve increases significantly in the adapted organism, and during the occlusion of small coronary vessels, the size of ischemic areas will be smaller, and during the occlusion of large vessels, the increase in blood flow in myocardial areas bordering the ischemic zone will be significantly greater than in the non-adapted organism. An important role is also played by the increased content in the "adapted" myocardium of myoglobin, a protein responsible for oxygen binding and transport, as well as by the increased capacity of the systems of aerobic and anaerobic energy conversion and energy utilization in such myocardium. Mitochondria are centers of energy metabolism, and decreased mitochondrial function, may play a key role in myocardial damage caused by impaired energy metabolism [7]. It is these changes formed in the process of adaptation in mitochondria, glycogenolysis and glycolysis apparatus, the system of energy utilization enzymes in the contractile apparatus of cardiomyocytes that increase the resistance of cardiac muscle to oxygen deficiency and, consequently, to hypoxic and ischemic effects. An important place in the preventive effect of training in ischemic lesions is occupied by the increase in the resistance of "adapted" myocardium to increased load. This feature increases the ability of non-ischemic heart parts to carry out compensatory hyperfunction. This advantage of the "adapted" myocardium is based on all components of the structural "trace" of adaptation, providing an increase in contractile capabilities of cardiomyocytes and the heart as a whole, namely, structural changes in the systems responsible for blood supply to the heart, energy conversion and utilization, and ion transport. Even more important is the fact that exercise training leads to various changes in cardiovascular function, including decreased heart rate, decreased blood pressure, increased maximal myocardial oxygen consumption, and adaptations affecting skeletal muscle, heart muscle, circulating blood volume, and various metabolic modifications [5]. The second important feature of the preventive effect of training in cardiovascular diseases is the ability to prevent the very occurrence of these diseases. Apart from its direct effects on the heart, it is definitely known that regular exercise leads to a better quality of life and longer life [4]. This effect is determined by a decrease in the probability of development of risk factors in trained people, which currently include atherosclerosis, disorders of carbohydrate metabolism and including changes in tolerance to carbohydrates, disorders of fat metabolism and obesity, hypercholesterolemia. In addition, stressful conditions are associated with higher rates of smoking, sleep disorders[3]. When evaluating the above-mentioned positive cross-effects of adaptation, it should be taken into account that they are realized only at rational dosage and adequate selection of physical loads. When adapting to excessive loads for a given organism, the general biological regularity is fully realized, which consists in the fact that all adaptive reactions of the organism have only relative expediency, i.e. even stable adaptation to physical load can

have its biological "price", which can manifest itself in two different forms: in the direct "wear and tear" of the functional system, on which the main load falls during adaptation; 2) in the phenomena of negative cross-adaptation - in the violation of the adapted organism's ability to adapt to the physical load; 3) in the phenomena of negative cross-adaptation, i.e. in the violation of the adapted organism's ability to adapt to the physical load; 4) in the phenomena of adaptation to the physical load. Direct functional insufficiency can develop in conditions of acute high load, in which direct damage to heart structures and other changes are described, which are both the result of the overload itself and of the stress-response arising in this case. This "price" of urgent adaptation is clearly manifested at the first loads of untrained people and is eliminated by the development of training. Thus, the above suggests that adaptation to dosed exercise is a important factor in preventing or limiting stress-induced cardiac injury.

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