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FUNCTIONAL CHARACTERISTICS OF THE CARDIOVASCULAR SYSTEM IN ENDURANCE ATHLETES

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Abstract

The article is devoted to a comprehensive analysis of functional changes in the cardiovascular system in endurance athletes. The mechanisms of adaptation of the heart and blood vessels to physical activity, such as eccentric myocardial hypertrophy, angiogenesis, increased mitochondrial density, and improved cardiac pumping function, are considered. The role of neural regulation, autonomic adaptation, and energy metabolism in maintaining high performance is emphasized. Potential risks associated with intense endurance exercise and their long-term health consequences are also discussed.

Keywords: Cardiovascular system, endurance, eccentric myocardial hypertrophy, angiogenesis, mitochondrial density, cardiac pump function, heart rate variability, energy metabolism, athlete's heart, training risks, adaptation, physiological changes.

Introduction

Cardiovascular adaptation in endurance athletes is one of the most striking demonstrations of the plasticity of the human body. During training, the heart and blood vessels undergo significant changes aimed at providing the body with the necessary amount of oxygen and nutrients. These changes include not only morphological restructuring, but also functional adaptations that form a highly efficient circulatory system capable of meeting the demands of intense physical activity.

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The aim of this paper is to examine the main mechanisms of cardiovascular adaptation to endurance loads, analyze their biological basis, and discuss the risks and prospects for long-term impact on the health of athletes.

Eccentric myocardial hypertrophy is a physiological process of adaptation of the heart to high aerobic loads, typical for endurance sports. It is characterized by an increase in the cavity of the left ventricle (LV) and a relative thickening of its walls, which allows the heart to pump large volumes of blood with minimal energy expenditure.

The main morphological change in eccentric hypertrophy is an increase in LV diastolic volume. This is achieved due to the constant effect of volume overload associated with high heart rate and increased venous return during physical activity. Such changes contribute to a significant increase in stroke volume and cardiac output (D'Andrea et al., 2010).

Additional increase in the size of the cardiac chamber occurs due to the elongation of cardiomyocytes, the main cellular unit of the myocardium. Unlike pathological hypertrophy, which is characterized by excessive thickening of the walls and the development of fibrosis, eccentric hypertrophy is characterized by the preservation of the normal tissue structure, which maintains high elasticity and functionality of the heart (Naylor et al., 2008).

At the molecular level, eccentric hypertrophy is regulated by the activation of signaling pathways such as IGF-1/Akt and PGC-1α, which stimulate cardiomyocyte growth, improve energy metabolism, and increase cellular resistance to hypoxia. Activation of IGF-1/Akt promotes the synthesis of proteins necessary for cell growth and also enhances the formation of mitochondria, which ensures a high level of energy metabolism under conditions of prolonged loads (McMullen et al., 2007).

PGC-1α plays a key role in the regulation of mitochondrial biogenesis, which is especially important for endurance athletes. Increasing the density of mitochondria in cardiomyocytes improves the efficiency of oxygen use and the synthesis of ATP, which is necessary for long-term cardiac work (Holloszy, 2008).

Eccentric hypertrophy provides significant improvements in cardiac pumping function. Due to the increased diastolic volume of the LV, diastolic filling is optimized, allowing the heart to pump a larger volume of blood at a lower heart

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rate. This is especially important for sports that require prolonged aerobic effort, such as marathon running, swimming, and cycling (Spirito et al., 1994).

In addition, increased LV elasticity and decreased peripheral vascular resistance contribute to improved overall hemodynamics, allowing muscles to receive sufficient oxygen even during intense exercise. The phenomenon of increased stroke volume without increasing heart rate makes the heart more economical and reduces the load on the myocardium.

Eccentric hypertrophy in athletes should be distinguished from pathological conditions such as dilated cardiomyopathy or hypertrophic cardiomyopathy. In contrast to these conditions, athletic hypertrophy is accompanied by preservation or improvement of cardiac contractile function, absence of fibrotic changes, and normal sensitivity to hormonal regulation. Echocardiographic examination is the main diagnostic method that allows distinguishing physiological hypertrophy from pathological hypertrophy (Pelliccia et al., 2000).

Long-term endurance training helps maintain cardiovascular health and prevent diseases. Enlargement of the LV cavity and improvement of its functionality increase cardiac reserves and allow the body to better cope with stress not only in sports but also in everyday life. However, some athletes may develop risks associated with excessive LV dilation, which requires regular monitoring of the heart condition.

It is important to consider that the development of eccentric hypertrophy may be partly due to genetic factors. Genetic variations such as ACE and ACTN3 polymorphisms affect the structural and functional characteristics of the heart. Individuals with certain alleles of these genes exhibit greater cardiac adaptation to physical activity (Montgomery et al., 2008).

Physiological myocardial hypertrophy is accompanied by activation of signaling pathways such as IGF-1/Akt and mTOR, which stimulate cardiomyocyte growth. These processes increase cardiac mass without causing pathological changes such as fibrosis or dysfunction (McMullen et al., 2007).

One of the key adaptation mechanisms in athletes is a decrease in resting heart rate (HR). This process is due to an increase in the activity of the parasympathetic nervous system, namely the vagus nerve, which leads to a slower but more economical heart rate (Aubert et al., 2003).

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A decrease in heart rate allows the heart to use the time to fill with blood during diastole more efficiently, which increases stroke volume. Bradycardia in athletes is an important sign of high functional activity of the autonomic nervous system. Heart rate variability (HRV) is an important marker of the adaptation of the autonomic nervous system to physical stress. Endurance athletes show an increase in HRV, indicating the body's ability to cope with increased stress and effectively regulate blood flow (Task Force of the ESC, 1996).

Adaptation of the vascular system

Angiogenesis. Endurance training stimulates the process of angiogenesis—the formation of new capillaries. This is especially important for increasing the delivery of oxygen and nutrients to working muscles. Angiogenesis is regulated by factors such as VEGF (vascular endothelial growth factor), which is activated in response to hypoxia and mechanical stress (Prior et al., 2004).

Increased elasticity of blood vessels. The vessels of endurance athletes become more elastic, which improves blood flow and reduces peripheral resistance. This is due to increased production of nitric oxide (NO), which dilates blood vessels and improves their functional state (Green et al., 2004).

Molecular mechanisms of adaptation

Mitochondrial biogenesis. One of the key processes of adaptation to endurance is an increase in mitochondrial density in the myocardium and skeletal muscle. This improves the ability of cells to use oxygen to synthesize ATP, which is critical for sustaining long-term exercise. Mitochondrial biogenesis is regulated through the activation of PGC-1α, which stimulates the expression of genes associated with oxidative phosphorylation (Holloszy, 2008).

Improving metabolism. Under endurance training conditions, aerobic metabolism enzymes such as citrate synthase and succinate dehydrogenase are activated, increasing the efficiency of energy use. These changes are associated with increased mitochondrial respiration and decreased lactate accumulation in muscle (Egan & Zierath, 2013).

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Potential Risks of Endurance Training

Arrhythmias and atrial remodeling. Long-term exercise can cause the atria to enlarge and increase the risk of developing arrhythmias such as atrial fibrillation. This is due to changes in electrical conduction and pressure in the left chambers of the heart (La Gerche et al., 2013).

Myocardial injury. Intense endurance exercise is sometimes associated with transient increases in troponin levels, a biomarker of myocardial injury. Although these changes are often reversible, their frequent recurrence may lead to fibrosis (Shave et al., 2010).

Practical significance of research. Studying the functional characteristics of the cardiovascular system in endurance athletes allows for the development of individualized approaches to training. Such programs can minimize risks and optimize adaptation changes, ensuring high athlete performance.
Modern monitoring methods, including echocardiography, heart rate variability analysis and biochemical markers, allow for timely detection of deviations and adjustment of the training process.

Conclusion

The functional features of the cardiovascular system in endurance athletes represent a complex set of adaptive changes, including cardiac remodeling, angiogenesis, improved metabolism, and regulation of heart rhythm. These processes ensure high efficiency of the heart and blood vessels, which is critical for performing long-term loads.
However, endurance training is associated with certain risks, such as arrhythmia and myocardial damage. Therefore, it is important to take into account the individual characteristics of athletes when developing training programs and to regularly monitor their condition.

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ISSN (E): 2980-4612
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