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ADAPTIVE CHANGES IN THE CARDIOVASCULAR SYSTEM AND OVERTRAINING SYNDROME RESULTING FROM EXCESSIVE PHYSICAL ACTIVITY

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ABSTRACT

Research objectives: Regular physical activity is widely recognized as a key component of a healthy lifestyle. While moderate effort brings numerous benefits, engaging in extreme physical activity introduces new challenges and risks. Intense exercise, despite its undeniable health benefits, can lead to unpredictable changes in the body. This paper identifies the possible adaptive changes of the cardiovascular system and the risk of developing overtraining syndrome as a result of long-term practice of intense exercise.
Methods and key findings: Two medical textbooks, thirty-eight scientific articles published between 2010 and 2024, and one article from 2005 were reviewed, with findings indicating that regular high-intensity training can lead to myocardial hypertrophy, reduced resting heart rate, and a smaller increase in heart rate in response to submaximal effort. An increased risk of arrhythmias and myocardial fibrosis has been observed in competitive athletes. Prolonged excessive exercise can result in overtraining syndrome, which may present as persistent fatigue, muscle and joint discomfort, and mood disturbances, among other symptoms.
Conclusions: The paper emphasizes the need to individualize training programs and proves the validity of regular monitoring of athletes' health. It also highlights the necessity for further research into overtraining syndrome and the adaptation of the cardiovascular system to intense exercise. Particular attention was paid to the diagnostic difficulties associated with overtraining syndrome due to the subjectivity of symptoms and the limited number of clinical studies. The significance of an interdisciplinary approach in understanding the body's adaptation to overload was emphasized, considering physiological, biochemical, and psychological factors.

KEYWORDS

Extreme Exercise, Cardiovascular System, Overtraining Syndrome

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Introduction:

In 2020, the World Health Organization (WHO) set guidelines for minimum physical activity levels. The total amount of time spent exercising for adults between the ages of 18 and 64 should be a minimum of 150 minutes per week **(**for moderate-intensity exercise) or 75 minutes (for more intense workouts). The guidelines presented above do not address the important topic of upper limits in terms of intensity, frequency and duration of exercise [1]. Studies indicate that excessive physical activity can have harmful effects on health, especially on the cardiovascular system. The Extreme Exercise Hypothesis suggests that the relationship curve between cardiovascular endurance and level of physical activity takes the shape of an inverted "J" - an increase in the amount of weekly exercise reduces the likelihood of cardiovascular disease, but these health benefits can be easily lost when the body exceeds the optimal amount of exercise for itself. The negative effects of overtraining can already be seen when physical activity exceeds 225 minutes/week [2]. Scientific studies show that professional athletes spend an average of 16 hours per week in physical training [3]. Moreover, excessive exercise is already evident among young athletes aspiring to pursue careers in competitive sports. These individuals engage in average of 11 h 41 min of structured training per week, while objective data showing approximately 4 h 31 min of total daily physical activity, of which 1 h 31 min is performed at a moderate-to-vigorous intensity [4]. Systematic, long-term performance of intense physical activity contributes to mild cardiac hypertrophy, a decrease in resting heart rate and a smaller increase in heart rate in response to submaximal effort [5]. Although these changes are primarily aimed at increasing an athlete's performance, long-term exposure to factors that promote them can result in the development of all sorts of pathologies. Myocardial fibrosis and arrhythmias, such as atrial fibrillation, can occur as a result of prolonged participation in competitive sports. Furthermore, years of intensive exercise can induce a condition where effective recovery

becomes unattainable. This overtraining syndrome is characterized by symptoms such as chronic fatigue, muscle pain, emotional lability, and a general decline in performance. The causes and possible course of the aforementioned problems are presented in the following article.

Methodology

The paper is based on information contained in two medical textbooks, thirty-eight articles written between 2010 and 2024, and one article from 2005 describing the mechanism of myocardial remodeling as a result of prolonged intense physical activity. The selection of literature was based on its alignment with Evidence-Based Medicine and the reliability of the medical data it contained. The references included in the bibliography were sourced from the PubMed database. The article addresses the following questions: how does myocardial remodeling occur as a result of intense physical activity, and what factors does it depend on? Is the cardiovascular system's adaptation to regular increased physical activity permanent? What exactly is overtraining syndrome, what are its potential health consequences and how it can be effectively treated?

Results

The concept that regular intense physical activity induces adaptive changes in the cardiovascular system is an expected conclusion. After all, the very intention to perform physical activity causes inhibition of vagus nerve tone and stimulation of the sympathetic nervous system, which leads to an increase in both heart rate and vascular resistance of extra-muscular tissues (skin, visceral organs, kidneys). Impulses from the motor cortex, hypothalamus and medulla oblongata, with the involvement of the cholinergic and vasodilator systems, lead to an increase in the diameter of the resistance vessels of the muscles involved in the activity being performed at any given time [6]. Athletes experience a certain level of stress not only during exercise but also during the immediate recovery period afterward. Physical exertion stimulates the release of catecholamines, increases the possible risk of ischemia and accelerates blood flow. An increase in vagus nerve tone and gradual bradycardia are observed during recovery after intense exercise [7]. Increased vagus nerve tone, so common in athletes, can lead to sinus bradycardia, sinus arrhythmia or first- or second-degree atrioventricular block [8]. A heart rate as low as 35 beats per minute, especially during sleep, can occur in up to 85% of professional athletes [9]. The continuous adaptation of the body to progressively greater demands results in specific modifications in the physiology of the cardiovascular system. Trained athletes show a higher ejection fraction, but a lower heart rate both at rest and during exercise (which is indirectly due to increased vagus nerve tone) [10]. Regular exercise increases the maximum oxygen consumption that can be achieved during exercise, which is associated with an increase in maximum cardiac output and maximum tissue oxygen uptake. Such efficient oxygen utilization is made possible by the frequent recruitment of new capillaries during regular exercise, which ensures better blood distribution to myocytes.

It is important to note that different forms of physical activity can lead to varying results when it comes to adaptive changes in cardiovascular function. Physical exercise can be divided into static (a.k.a. strength) or dynamic (a.k.a. endurance) exercise. During dynamic exercise (such as running), isotonic contractions occur primarily, meaning that muscles shorten, but their tension remains unchanged. In more static disciplines (such as weightlifting), the predominant muscle activity is longer-lasting isometric contractions, during which muscle length does not change, but muscle tension increases [6]. There are a number of noticeable differences between the two types of contractions, such as the fact that isotonic contractions increase blood flow through skeletal muscles, while isometric contractions decrease this flow, due to the generation of lateral compression on the arteries [10]. Sports disciplines in which endurance exercise is predominant are associated with increased blood flow in the blood vessels, while those with a predominance of strength exercise primarily result in increased pressure in these vessels [8]. However, it is worth noting that the practice of most sports requires both strength and endurance, so it actually includes both components. Prolonged and frequent exercise with a predominantly static component causes the cardiac muscle to hypertrophy inwards. In this case, its remodeling is concentric, which can also be seen in conditions such as aortic valve stenosis or hypertension. The frequent practice of sports with a predominantly endurance component results in eccentric remodeling of the heart, where there is a proportional increase in the thickness of the heart walls as well as the width of its ventricles [5, 8]. A clinically important point is that left ventricular hypertrophy in the athlete's heart is usually symmetric, whereas pathologies such as hypertrophic cardiomyopathy contribute to asymmetric ventricular growth [11]. To understand the exact physiology of the cardiovascular system’s adaptive changes in response to intense exercise, it is important to note that physiological cardiac growth is associated with an increase in cardiomyocyte volume. Unlike in certain cardiac pathologies, this growth does not involve an increase in the

number of cardiomyocytes. It is also important to highlight that an increase in the width of the left ventricle alters the overall geometry of the heart muscle, which can stretch the heart valves. This may lead to functional valve regurgitation visible on imaging, although it often remains asymptomatic in clinical evaluation [9]. The amount of oxygen consumed during exercise correlates directly with eccentric remodeling of the heart [7, 11]. Increasing the size of the heart's ventricles and wall thickness is thought to improve lusitropic and systolic function, and consequently increase stroke volume during exercise. Adaptive changes in the heart are particularly well noted in athletes practicing sports that require both strength and endurance (e.g., cycling, triathlon, rowing) [12]. Regular endurance training often leads to an increase in the transverse dimension of the left atrium (reaching ≥ 45 mm), which is directly proportional to the corresponding enlargement of the left ventricle [11].

Many of the adaptive changes in the cardiovascular system from regular exercise are crucial for improving tolerance to intense physical activity. However, these modifications do not always enhance the overall functioning of the individual. The fact that there is no clear boundary that defines the point at which physiological adaptations of the heart begin to contribute to the occurrence of arrhythmias and other pathologies is a challenging clinical problem in the context of competitive athletes [7].

Opinions are divided regarding the reversibility of cardiac remodeling caused by intense exercise. An echocardiographic study demonstrated that significant left ventricular enlargement persisted in 20% of retired professional athletes even after a 5-year period [11]. Pelliccia and colleagues conducted a prospective follow-up of 40 elite male athletes who had stopped professional activity for 1 to 13 years. They concluded that discontinuing training resulted in a significant reduction in the thickness of the initially hypertrophied left ventricle in most of the athletes. It is important to note, however, that in the study conducted on some athletes, the thickness of the left ventricle remained greater than baseline levels even after the cessation of intensive professional training. Furthermore, in 9 of the athletes, the left ventricular wall thickness exceeded 13 mm despite the cessation of training [13]. Pelliccia and colleagues also conducted a study in which they concluded that even 17 years of intensive, uninterrupted endurance training in certain athletes may still not lead to the development of abnormal cardiac dimensions or deterioration of left ventricular function [14]. The analysis of subsequent studies, in turn, leads to the conclusion that training more than 3h a day for a week can modestly alter the average resting heart rate or left ventricular wall thickness. Modification of left ventricular wall thickness can be observed after just a few years of competitive sports in professional cyclists or wrestlers [15].

It is worth noting that regular practice of various types of sports still remains extremely effective in reducing the progression of cardiovascular disease and associated mortality. Athletes who systematically exercise live, on average, 5-7 years longer compared to those with sedentary lifestyles [1]. However, this does not change the fact that excess physical activity can somehow contribute to a poorer quality of life, as well as carry unpleasant health implications. Many studies suggest that an athlete's heart, resulting from prolonged intense exercise, may be prone to arrhythmias. Moreover, several meta-analyses have shown that episodes of atrial fibrillation occur two to five times more frequently in endurance athletes than in non-athletes [16, 17, 18, 19]. Endurance athletes may also develop myocardial fibrosis induced by intense and prolonged exercise. A study of 102 marathon runners showed the onset of myocardial fibrosis in 12% of them. Similar myocardial fibrosis also occurred in 14% of 106 male endurance sports practitioners studied [16]. It is also important to note that sinus bradycardia, commonly observed in elite athletes, can cause dizziness that interferes with daily activities or lead to potentially dangerous fainting [17].

Some authors argue that adaptive changes in the cardiovascular system can resemble cardiomyopathies, which complicates diagnosis. Distinguishing between physiologic adaptations resulting from intense exercise and the benign phenotype of primary cardiomyopathies can be challenging even for the most experienced specialists [20]. Regular testing to monitor potential changes in cardiac morphology is crucial for the prevention of cardiovascular events in elite athletes. Such extensive diagnostics require prior completion of a thorough clinical history, physical examination, and a 12-lead electrocardiogram. Accurate information should be collected regarding the type and frequency of practice of the physical activity in question [21, 22]. It may be beneficial for the patient to maintain a detailed diary to track daily activities (e.g., exercise sessions and sleep hours), along with any medications taken and symptoms experienced [22]. A comprehensive assessment of the patient's nutrition is also a crucial component of the physical examination, along with a detailed account of the patient's diet, including the use of supplements and the consumption of energy drinks [23]. If any abnormalities are identified, they should be further investigated using echocardiography, which provides a detailed evaluation of the heart's structure, wall thickness, and precise ventricular measurements [21, 24]. Additionally, exercise echocardiography is a valuable tool for detecting abnormalities that may only manifest

during physical activity [21]. Cardiac magnetic resonance (CMR), although available to a lesser extent than the aforementioned tests, may be necessary to characterize the myocardium more accurately and to exclude specific diseases such as amyloidosis or Fabry disease [21, 24].

Despite the extensive coverage of cardiovascular adaptation to intense exercise in the scientific literature, this phenomenon still represents a significant research challenge that requires further analysis. In 2016, the Pro@Heart multicenter cohort study was initiated to analyze in detail the cardiovascular changes occurring over 20 years in more than 300 elite endurance athletes. Study participants undergo regular ECG, echocardiography, and CMR [25].

Many years of intense overloading of the body with physical exertion can sometimes be highly detrimental to the body. However, it does not always result in negative health outcomes. FO, or so-called Functional Overreaching, occurs when training causes a temporary decline in an athlete's form, which, after a few days of rest, regains its original physical capacity. Nonfunctional Overreaching (NFO) can occur when exercise intensity is excessive, leading to negative health consequences. If symptoms persist for more than 2 months, the condition is classified as overtraining syndrome (OTS) [26, 27]. Symptoms of overtraining syndrome include chronic muscle and joint pain, chronic fatigue, loss of motivation, insomnia, irritability, anxiety. Athletes suffering from this condition are at increased risk of depression and anxiety disorder. Physical examination of athletes has revealed the presence of either bradycardia or tachycardia, along with hypertension. These cardiovascular abnormalities, for obvious reasons, can impair the athlete's performance and negatively affect both physical and psychological well-being, which can lead to increased susceptibility to infections, emotional instability, and reduced stress resilience [28]. In addition to the aforementioned symptoms, several physiological changes may be observed in an athlete's body, including a reduced serum testosterone-to-estradiol ratio, elevated cortisol levels in both plasma and saliva, increased urinary catecholamines, elevated plasma adrenocorticotropic hormone (ACTH), and decreased prolactin levels. In individuals with overtraining syndrome, a single exercise session has been associated with a reduced neutrophil-to-lymphocyte ratio and increased levels of the interleukins IL-1β, IL-6, and tumor necrosis factor-α (TNF-α) [26, 19].

Many authors exploring the exact causes of overtraining syndrome assume that the dominance of activation of the parasympathetic part of the nervous system and reduced sympathetic activation are the primary cause of the symptoms experienced in OTS [26, 20]. However, the direct occurrence of overtraining syndrome is usually associated with a large number of high-intensity workouts without adequate rest. Excessive physical exertion can result in a progressive decline in the force or effectiveness with which a given exercise is performed. Reduced submaximal strength will require greater subjective effort to properly perform a given physical exercise. This condition without adequate recovery can exacerbate or even aggravate overtraining syndrome. The increased physical effort needed to compensate for diminished submaximal strength can also heighten perceived fatigue, as it necessitates greater recruitment of muscle fibers [29].

Malnutrition and mental stress can intensify the symptoms of OTS. Women have been found to be more susceptible to developing the overtraining syndrome. The reasons for this phenomenon are believed to be hormonal fluctuations associated with the monthly cycle. It is worth noting that overtraining syndrome is also associated with the loss of large amounts of glycogen, the resynthesis of which takes time. During intense exercise, large amounts of reactive oxygen species are also produced, which impairs post-exercise recovery [30].

Athletes undergoing training often experience gastrointestinal symptoms such as nausea, cramps, bloating, and even diarrhea or constipation. Such symptoms can be directly associated with the effects of strenuous exercise and subsequent overtraining on the gut microbiome. Studies have indicated a decrease in the abundance of Bacteroides, accompanied by an increase in Blautia and Bifidobacterium during high-intensity interval training [31, 32]. Additionally, exercise-induced stress has been shown to decrease cecal levels of Turcibacter spp. while increasing Ruminococcus gnavus, both of which play well-established roles in intestinal mucus degradation and immune modulation [33]. Alterations in the gut microbiome may lead to increased intestinal permeability, commonly referred to as leaky gut syndrome. Physical stress and intestinal hypoxia during intense exercise contribute to this increased passage of substances through the intestinal barrier by weakening tight junctions between epithelial cells and promoting inflammatory processes. Exercise-induced stress hormones, such as cortisol, further exacerbate these effects by impairing intestinal barrier function. Notably, this phenomenon can occur even during regular, long-term training at 70% of maximal capacity, and is therefore a relevant concern for individuals experiencing overtraining syndrome [31].

An interesting phenomenon is that the symptoms of overtraining syndrome tend to worsen as the condition progresses. Overtraining syndrome can suppress an athlete's appetite, often resulting in insufficient

meal intake. Caloric deficiencies during training raise cortisol levels, exacerbate fatigue and increase the likelihood of multiple injuries or hard-to-fight infections [34]. In the course of overtraining syndrome, an adequate amount of sleep is also of considerable importance. An athlete suffering from OTS due to nagging pain often suffers from insufficient night rest. Such sleep deficiency weakens the functioning of the immune system and exacerbates emotional lability [35].

Overtraining syndrome is a serious and complex issue. Its diagnosis is associated with numerous difficulties. These problems stem, among other things, from the high subjectivity of the symptoms that characterize it. Moreover, the limited number of studies on this phenomenon may, in part, be attributed to the challenges associated with collecting reliable data on overtraining syndrome. Of course, research on OTS cannot rely on intentionally inducing this condition in athletes, so it relies on data from individuals self-reporting a decline in form [36]. It is also worth noting that there is a tendency among elite athletes not to disclose problems related to their own mental or physical health due to the ever-present pressure, as well as the fear of having to involuntarily end their own careers. Thus, more research on overtraining syndrome is certainly needed to gain a broader understanding of the problem [37].

Although overtraining syndrome is a condition with multifaceted etiology and the number of publications on this topic remains limited, there are specific interventions that can be implemented to mitigate the severity of its symptoms and improve the performance of affected athletes. The treatment of OTS largely depends on the precise underlying cause of reduced physical performance, making it essential to address any chronic conditions that could exacerbate symptoms [38]. Nutritional management is also a critical component of recovery, as inadequate intake of macronutrients and micronutrients can worsen fatigue, impair immune function, and delay physiological adaptation to training. Protein intake of approximately 1.2 to 1.4 g/kg body weight per day is recommended, while carbohydrates should constitute 60-70% of total caloric intake, corresponding to 8 to 10 g/kg of body mass per day. Fat intake should not exceed 30% of total daily calories, with emphasis on unsaturated fatty acids to maintain optimal cardiovascular and metabolic health [34]. Adequate rest, sleep quality, and proper recovery strategies, including periodized training and active recovery, are equally important for restoring physiological homeostasis and preventing further deterioration. Some experts, however, suggest a strategy of gradually reducing intensity rather than complete cessation of physical activity, as complete rest may as well lead to physiological distress. Given the significant physiological component of overtraining syndrome, involvement of a sports psychologist or other mental health professional is advisable. In certain cases, pharmacological treatment with selective serotonin reuptake inhibitors (known as SSRIs) has been proposed, based on observed neuroendocrine similarities between overtraining syndrome and depression. If sleep disturbances are prominent, pharmacological interventions with trazodone and amitriptyline may also be considered [38,39]. Overall, a multidisciplinary approach that integrates nutrition strategies, individualized training modifications, psychological support, and, when necessary, pharmacotherapy, appears to be the most effective way to restore performance and well-being in athletes affected by OTS.

Discussion

There is no doubt that the average fitness of athletes who regularly practice physical exercise is significantly higher than that of people with sedentary lifestyles. The optimal amount of physical activity plays a key role in maintaining homeostasis in the human body. While appropriate levels of physical activity undoubtedly contribute to maintaining physical and mental well-being, they are certainly not the sole determinants of overall health. Even the most physically fit athletes can face health problems, often directly related to their physical exertion. Maintaining an appropriate balance between training intensity, recovery, and overall lifestyle is therefore essential for preventing long-term complications. Excessive exercise, especially when combined with inadequate rest or poor nutrition, can lead to maladaptive physiological responses, particularly within the cardiovascular system.

The main purpose of this work is to focus on the consequences of cardiovascular adaptation to excessive, prolonged exercise. Individuals who participate intensively in endurance sports may be at risk for a phenomenon known as Athlete's Heart Syndrome (AHS). This condition represents a form of physiological remodeling of the heart in response to sustained endurance training, often characterized by increased cardiac mass, enlarged ventricles, and enhances stroke volume. While these changes are considered beneficial to some extent, they may also predispose athletes to certain pathological outcomes. AHS is associated with an increased risk of a variety of arrhythmias. Arrhythmias such as atrial fibrillation and conditions like chronic sinus bradycardia, can cause deterioration of quality of life in many active athletes. In some cases, prolonged

exposure to extreme endurance activity may even increase the risk of myocardial fibrosis or electrical instability, although the clinical significance of these findings continues to be debated [40].

Excessive physical exertion can also lead to so-called Overtraining syndrome (OTS). Chronic fatigue, or muscle and joint pain, are among the many symptoms of OTS. The syndrome is often accompanied by sleep disturbances, irritability, mood swings, and noticeable decline in athletic performance, despite continuous or even increased training efforts. The mechanisms underlying OTS are multifunctional and not yet fully understood. They are believed to involve disruptions in neuroendocrine regulation, immune system function, and metabolic balance. Moreover, the absence of clear biomarkers makes early diagnosis difficult, resulting in delayed intervention and prolonged recovery times [41]. Preventing OTS therefore requires careful monitoring of both physical and psychological indicators, as well as individualization of training programs.

Despite the existing publications on the athlete's heart and overtraining syndrome, the information available in this field remains scarce. In addition to the hemodynamic load, which is directly related to the type of sport practiced and the intensity of training, there is a much broader array of factors affecting cardiovascular transformation. Age, gender, ethnicity, diet, comorbidities - these are just a few of the variables that determine an athlete's cardiac development. The duration of training, the athlete’s genetic predisposition, and environmental conditions such as altitude or temperature may also play crucial roles in shaping cardiac remodeling. Numerous publications lack consideration of these variables, which is a significant oversight. Without acknowledging such confounding factors, it is difficult to draw reliable conclusions about the true long-term impact of intense exercise on cardiac health.

It is also necessary to precisely define diagnostic methods in the context of both the athlete's heart and overtraining syndrome. Echocardiography, cardiac magnetic resonance imaging, and electrocardiographic assessment are among the most valuable diagnostic tools in differentiating between physiological and pathological changes. However, the interpretation of these tests must take into account the specific training background and individual characteristics of each athlete. The limited number of scientific publications defining in detail the symptoms of overtraining syndrome indicates an important gap in the available literature, which implies the need for further exploration of this phenomenon. Similarly, the mechanisms that distinguish adaptive cardiac hypertrophy from early pathological remodeling remain insufficiently understood.

Both Overtraining Syndrome (OTS) and Athlete's Heart Syndrome (AHS) are topics of great scientific interest. The paucity of scientific publications significantly impedes understanding of both. There is a clear need for more scientific research on OTS and AHS in order to fully recognize the health implications associated with them. Expanding research in this area could contribute not only to improved diagnosis and prevention but also to the development of safe training guidelines for both professional and amateur athletes. A deeper understanding of how the cardiovascular system responds to chronic physical stress is essential to balance the benefits of exercise with its potential risks, ensuring that physical activity continues to serve as a foundation for long-term health rather than a source of harm.

Conclusions:

Regular practice of intense physical exercise induces a wide range of adaptive changes within the cardiovascular system, reflecting the body’s physiological response to sustained and elevated workloads. One of the most notable adaptations is cardiac hypertrophy, characterized by an increase in the size and mass of the heart, particularly left ventricle. This structural modification enhances cardiac output and overall efficiency, allowing the heart to pump a greater volume of blood per beat. Consequently, individuals who engage in long-term, high-intensity training often exhibit a reduced resting heart rate, which is indicative of improved endurance and overall athletic performance.

Despite these well-documented benefits, opinions remain divided regarding the reversibility of exercise-induced cardiac remodeling. While some evidence suggests that certain structural and functional changes regress following the cessation of intense training, other studies indicate that specific alterations may persist over time, raising questions about their long-term physiological significance.

Another important consideration in the context of intense physical training is overtraining syndrome, a condition that arises from prolonged physical overload and insufficient recovery. This syndrome manifests through a variety of symptoms, including chronic fatigue, muscle and joint pain, and diminished performance capacity. However, accurate diagnosis remains challenging due to the inherently subjective nature of its symptoms. The variability in individual perception of fatigue, pain, and psychological stress complicates the establishment of objective diagnostic criteria, underscoring the need for further research into reliable assessment methods.

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