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Balancing Cardiovascular Health: Assessing the Impact of Physical Activity on Athletic Cardiac Adaptations and Cardiac Pathology

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Abstract

Introduction:

Physical activity is an effective therapy for preventing and treating cardiovascular diseases. However, studies show a curvilinear relationship between exercise and survival, with diminishing returns beyond optimal fitness levels. Extreme athletic training can harm some individuals, as long-term training in professional athletes can lead to Athlete's Heart Syndrome (AHS), characterized by structural heart changes and altered cardiac conduction due to high-intensity exercise. Intense exertion also increases the risk of sudden cardiac death by exacerbating underlying cardiac conditions. A step-by-step multimodality approach,

starting with personal and family history, clinical evaluation, and 12-lead ECG, followed by further investigations like exercise testing, echocardiography, 24-hour ECG Holter monitoring, cardiac MRI, CT, nuclear scintigraphy, or genetic testing, is essential for differentiating between extreme physiological adaptations and cardiac pathology. In this process, cardiovascular imaging is crucial for identifying structural abnormalities in athletes within the grey zone between normal adaptations and early cardiovascular disease.

Purpose: The aim of this scientific paper is to review the current understanding of the pathophysiological changes in the cardiovascular system of athletes, the incidence and causes of sudden cardiac death in athletes, and the multimodality approach to distinguishing athletic cardiac remodelling from cardiomyopathy.

Review Methods: We conducted our study as a literature review based on information gathered from PubMed, Embase, GoogleScholar using combinations of the following

Conclusion: Regular exercise reduces mortality, but intense exertion can alter the heart, increasing arrhythmia risk. Sudden deaths in young athletes often stem from inherited heart conditions, while older athletes primarily succumb to CAD. Differentiating athlete's heart from cardiomyopathy requires thorough evaluation using available techniques.

Keywords: athlete's heart; cardiomyopathy; physical activity; ventricular hypertrophy

Introduction

Physical activity is recognized as an effective therapy for preventing and treating cardiovascular diseases. Exercise offers added advantages such as weight loss, lowered blood pressure, enhanced insulin sensitivity, improved lipid profile, reduced risk of certain cancers,

and better physical and cognitive function during aging, along with enhanced mental well-being [1]. Studies suggest a curvilinear relationship between exercise and survival, demonstrating that beyond an optimal fitness level, diminishing returns may occur. Extreme athletic training has been implicated in potential harm for some individuals. Professional athletes prioritize peak performance in competition, with long-term training linked to increased left ventricle size, thickness, and mass, leading to Athlete's heart syndrome (AHS). AHS is characterized by structural heart changes and altered cardiac conduction due to high-intensity exercise [2]. The physiological factors driving this remodeling are not fully understood but depend on non-modifiable athlete characteristics, exercise type and duration, as well as environmental and genetic factors. There are several theories describing structural adaptations of the heart to the physical activity. In athletes, sinus bradycardia often coincides with first-degree atrioventricular (AV) block, while more severe AV conduction issues may stem from parasympathetic hypertonia [3]. Athletes also face an increased risk of sudden cardiac death (SCD), primarily due to intense exertion exacerbating underlying cardiac conditions. In young athletes, SCD is typically caused by genetic or congenital structural cardiac disorders, while in athletes over 35, the most common cause is atherosclerotic coronary artery disease (CAD) [1]. Recognizing these changes is crucial for distinguishing between normal physiological responses and pathological conditions in cardiovascular health. Various tests and examinations aid in this process, starting with a 12-lead ECG and followed by further investigations such as exercise testing, echocardiography, 24-hour ECG Holter monitoring, cardiac MRI, CT, nuclear scintigraphy, and genetic testing [1].

Review methods

We conducted our study as a literature review based on information gathered from PubMed, Embase, GoogleScholar and explored the methods of diagnosis and management to overcome intracranial hypotension.

Physiology of the athlete’s heart

Regular training enhances cardiorespiratory fitness and sports performance by inducing significant cardiovascular adaptations to changing hemodynamic conditions [4]. Optimal cardiovascular efficiency is crucial for physical performance, providing muscles with more oxygen through increased blood flow and enhanced oxygen extraction. Palermi *et al*. state that a minimum of 3 hours of training per week for at least 3 months may suffice to observe initial morpho-functional heart adaptations, but significantly more training is needed to identify an

athlete's heart [4, 5]. These adaptations depend on the duration, type, and intensity of the individual's sports activity [6]. The Morganroth hypothesis, firstly presented in 1975, proposes that endurance and strength training each cause unique changes in cardiac structure [7]. Due to the hypothesis endurance athletes typically develop eccentric left ventricle (LV) hypertrophy and right ventricle (RV) dilation due to increased LV volume and diastolic wall stress while in contrast, strength athletes exhibit concentric LV hypertrophy, with normal LV dimensions but increased wall thickness and mass due to pressure overload and elevated systolic wall stress [7, 8]. This hypothesis is limited because many sports combine endurance and strength training, leading to an intermediate hypertrophy phenotype. Recent studies challenge it by suggesting that LV mass increases proportionally with LV volume (balanced remodelling) regardless of the sport, and normal LV geometry is common among elite athletes [9, 10]. There is occasionally overlap between physiological adaptations in an athlete's heart and certain pathological conditions like hypertrophic cardiomyopathy (HCM) or arrhythmogenic cardiomyopathy (ACM), increasing the risk of sudden death [11, 12]. Thus, distinguishing between physiological and pathological cardiac anomalies in athletes is challenging yet crucial, as misdiagnosis can lead to significant consequences such as exclusion from competitive sports, false reassurance, and missed opportunities for effective treatment.

ECG adaptations in athletes are common, resulting from electrical and structural remodelling due to repeated intense training sessions [13, 14, 15]. These alterations are common and may coincide with patterns indicative of latent cardiovascular disease. Athletes are often presented with a range of electrocardiographic changes considered within the typical spectrum for this population.

Regular, long-term participation in intensive exercise leads to unique ECG changes reflecting enlarged cardiac chambers and increased vagal tone. These ECG findings in athletes are normal physiological adaptations and do not require further evaluation [16].

Table 1. Normal EKG findings in athletes table 1 [16]

Normal ECG findings	**Definition**
Increased QRS voltage	Isolated QRS voltage criteria for left (SV1 6 RV5 or RV6 > 3,5 mV) or right ventricular hypertrophy (RV1 + SV5 or SV6 > 1,1 mV)
Incomplete RBBB	rSR` pattern in lead V1 and a QRS pattern in lead V6 with QRS duration <120 ms
Early repolarization	J-point elevation, ST-segment elevation, J-waves, or terminal QRS slurring in the inferior and/or lateral leads
Black athlete repolarization variant	J-point elevation and convex ("domed") ST-segment elevation followed by T-wave inversions in leads V1-V4 in black athletes
Juvenile T-wave pattern	T-wave inversion V1-V3 in athletes aged <16 y
Sinus bradycardia	≥30 bpm
Sinus arrhythmia	Heart rate variation with respiration: rate increases during inspiration and decreases during expiration
Ectopic atrial rhythm	P-waves are a different morphology compared with the sinus P-wave
Junctional escape rhythm	QRS rate is faster than the resting P-wave or sinus rate and typically <100 bpm with narrow QRS complex unless the baseline QRS is conducted with aberrancy
1st degree AV block	PR interval: 200-400 ms
2nd degree AV block (Mobitz 1 or Wenckebach)	PR interval progressively lengthens until there is a non-conducted wave with no QRS complex

ECG changes include sinus bradycardia or arrhythmia, first-degree or Mobitz type 1 atrioventricular block, voltage criteria for ventricular hypertrophy, incomplete right bundle branch block, T-wave inversion and J-point elevation with ascending ST segments [17].

Proarrhythmic tendencies in athletes may result from several mechanisms, including atrial and ventricular dilation, hypertrophy, bradycardia, increased vagal tone at rest, ionic changes, early repolarization, elevated sympathetic tone during exercise and high wall stress [18].

Cardiovascular adaptations in an athlete's heart involve balanced enlargement of all chambers. While past research mainly focused on the LV, recent attention has shifted to the RV, atria, and aorta. Physical activity results in proportional increases in both left and right cardiac cavity sizes, greater LV wall thickness and mass, and enhanced systolic and diastolic function. These changes, which depend on the duration, type, and intensity of training, are usually benign and physiological but can occasionally predispose to pathological conditions [19, 20, 21]. Athletes typically exhibit a 10–20% increase in left ventricular (LV) wall thickness and a 10–15% increase in both left and right ventricular cavity size compared to non-athletes of similar age and size [22]. Additionally, athletes show enhanced diastolic filling, increased stroke volume even at high heart rates, and greater oxidative capacity and capillary conductance in skeletal muscle, resulting in high peak oxygen consumption during exercise [17].

Sudden cardiac death in athletes

Occasionally, an athlete may experience sudden death during or immediately after a competition. The prevalence of sudden cardiac death (SCD) varies depending on data collection methods, but the most reliable figures show a prevalence of about 1 in 50,000 among young competitive athletes and middle-aged marathoners [23]. Ninety percent of the victims are male. While deaths in competitive athletes attract significant media attention, over 90% of all exercise-related SCDs occur in recreational athletes [24]. In young athletes, sudden cardiac death (SCD) is typically caused by genetic or congenital structural cardiac disorders, such as hypertrophic cardiomyopathy (HCM), arrhythmogenic cardiomyopathy (ACM), or anomalous coronary artery origin [11, 25]. In athletes over 35, most SCDs are due to atherosclerotic coronary artery disease (CAD) [25]. Many of these conditions may first present with sudden death and not be clinically apparent. Although estimating the proportion of athletes with prior symptoms is challenging and subject to bias, around 30% of athletes with SCD reported symptoms such as chest pain, shortness of breath, performance decline, palpitations, pre-syncope, or syncope beforehand. Evaluating these exertional symptoms by sports medicine and cardiology specialists is crucial for the medical care of athletes and preventing SCD [26].

As the population ages and the popularity of endurance sports like running and cycling increases, the burden of SCD risk in this group is likely to grow. In older athletes, the most common cause of SCD is atherosclerotic CAD. While regular physical activity reduces the risk of developing CAD and related cardiovascular events, those with existing CAD face an elevated risk of cardiovascular events during vigorous exercise [27]. SCD during exercise is more common in males, older sports participants who are not regularly active, and individuals with known cardiac disease or risk factors for CAD. These characteristics allow clinicians to focus screening and risk reduction strategies on those at highest risk [26].

In most U.S. studies, the leading cause of SCD in young athletes is HCM, a genetic condition marked by left ventricular hypertrophy and cardiac myocyte disarray, which predispose to ventricular arrhythmias. HCM affects up to 1 in 200 people, with SCD often being the first symptom. The second most common cause, accounting for around 17% of cases, is congenital coronary artery anomalies. Other causes include various inherited or acquired myocardial diseases, other structural cardiovascular diseases, and primary arrhythmogenic disorders [28].

ARVC is a another significant cause of SCD in young athletes, with a prevalence of about 1 in 5,000. This genetic cardiomyopathy is characterized by fibro-fatty infiltration and dilation of the right ventricle. Exercise increases the risk of ventricular arrhythmias and may accelerate the phenotypic expression of ARVC [28, 29].

Discriminating between AHS and cardiovascular pathology

Athlete's heart exhibits cardiac remodelling features that can mimic pathological conditions, posing a common challenge for physicians assessing athletes [19]. The overlap between physiological adaptation and pathology necessitates a thorough evaluation of cardio-circulatory changes specific to the sport, such as left ventricular wall thickening, dilation, right ventricular dilation, and left ventricular hypertrabeculation. Clear definitions and stringent criteria are essential for accurately managing athletes, enabling differentiation from conditions like hypertrophic cardiomyopathy (HCM), dilated cardiomyopathy (DCM), left ventricular noncompaction (LVNC), and arrhythmogenic cardiomyopathy (ACM) [30].

To distinguish between physiological adaptations in athletes and structural cardiovascular disease, the diagnostic process should commence with a comprehensive medical history, including family background, thorough physical examination, and a 12-lead ECG. Additional tests may be warranted only if there is clinical suspicion or abnormalities on the ECG.

In this context, the most common, accessible, and cost-effective second-line examinations include echocardiography, exercise stress testing (EST) and 24-hour Holter ECG monitoring.

When athlete follow-up is necessary, using any combination of the three examinations is highly effective in detecting subtle changes over time [31]. However, cardiopulmonary exercise testing (CPET) can play a crucial role in diagnosing athlete's heart. Despite its importance, its use is limited to select cases due to the need for experienced personnel, high costs, and time-intensive nature [32]. If these evaluations yield highly suspicious results or fall into a grey zone, third-line evaluations using more advanced and costly techniques, such as exercise stress echocardiography (ESE), cardiovascular magnetic resonance (CMR), coronary computed tomography (CCT), genetic testing, single photon emission computed tomography (SPECT), and positron emission tomography (PET), are necessary [5, 16].

Conclusions

Regular physical exercise offers numerous health benefits, including reducing mortality, with increasing exercise intensity potentially leading to greater benefits, though possibly levelling off in a curvilinear pattern. However, intense and frequent exertion can induce cardiac changes in susceptible individuals, including morphological, functional, and electrical alterations, which may increase the risk of ventricular arrhythmias and sudden death. Most sudden deaths among young athletes stem from underlying inherited or congenital cardiac disorders, notably cardiomyopathies, whereas athletes over 35 primarily succumb to atherosclerotic CAD. Distinguishing an athlete’s heart from early-phenotype cardiomyopathy or hidden cardiovascular pathology requires a thorough diagnostic work-up incorporating morphological, electrical, structural, and functional evaluations. Given the wide availability of various multimodality techniques, a practical step-by-step approach is essential. This systematic evaluation proceeds only if second- and third-line diagnostic modalities are indicated after initial screening. Many athletes fall into a grey zone where pathology and physiological remodelling overlap, making a multimodality cardiovascular diagnostic approach crucial for an accurate final diagnosis.

Disclosures

Author's contribution

Conceptualization: Izabela Hądzlik and Jan Piotrowski; Methodology: Julia Biały-Karbowniczek; Software: not applicable; Check: Katarzyna Słychan, Blanka Łuczak and Aleksandra Jędrasek; Formal analysis: Tomasz Jędrasek; Investigation: Klaudia Bulska; Resources: not applicable; Data curation: Julia Pogoda; Writing - rough preparation: Izabela

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