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MORE THAN JUST A RACING HEART: PALPITATIONS UNCOVER HYPERTROPHIC CARDIOMYOPATHY IN ATHLETES

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ABSTRACT

Heart palpitations are a common symptom reported by athletes presenting to sports medicine clinics and cardiology departments. While often benign, palpitations in this population can indicate underlying structural heart disease, notably hypertrophic cardiomyopathy, which is a leading cause of sudden cardiac death in young athletes.
Methods: This narrative review was conducted using PubMed and Google Scholar, focusing on studies published between 2016 and 2024. Standard textbooks in cardiology and internal medicine were also consulted to ensure comprehensive coverage of current knowledge.
Results: The analysis showed that palpitations can result from both mild and severe arrhythmias and structural heart disease. The importance of differential diagnosis and the role of imaging studies and ECGs in identifying the cause was emphasized. Special attention was paid to hypertrophic cardiomyopathy as a common genetic cause of symptoms. The need for an individualized therapeutic approach and patient education was also pointed out.
Conclusions: Palpitations in athletes should never be overlooked, as they may be the first sign of hypertrophic cardiomyopathy, a potentially life-threatening condition.. Special attention should be paid to hypertrophic cardiomyopathy, which is a common genetic cause of cardiac arrhythmias. Diagnosis should be based on careful differentiation of causes and the use of imaging studies and ECGs. Successful management requires an individualized therapeutic approach and patient education in recognizing and responding to symptoms.

KEYWORDS

Palpitations, Arrhythmia, Hypertrophic Cardiomyopathy, ECG, Cardiology

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Introduction.

Heart palpitations are one of the most commonly reported symptoms among athletes presenting to both outpatient clinics and emergency medical settings. Often described by patients as a sensation of a rapid, pounding, fluttering, or irregular heartbeat, palpitations may be perceived as alarming and are frequently associated with anxiety or physical exertion. While in many cases palpitations are benign and self-limiting, they can also serve as an early warning sign of serious cardiovascular conditions, including arrhythmias, structural heart abnormalities, and metabolic disorders. [1]

The clinical challenge lies in distinguishing between palpitations of functional origin and those that may indicate potentially life-threatening disease. A comprehensive understanding of the underlying mechanisms, classification, and diagnostic approach is therefore essential for appropriate evaluation and management.[2]

This article aims to provide a descriptive overview of the phenomenon of heart palpitations, emphasizing both benign and pathological etiologies, with particular focus on hypertrophic cardiomyopathy (HCM) as a key example of a structural cardiac disorder frequently associated with this symptom.[3]

Pathomechanism

The normal number of beats of the human heart is between 60 and 80 beats per minute. The heart rhythm is regulated by electrical impulses that originate in the sinus node. It is through these that the heart chambers contract and pump blood to all the organs of the body. If the stimulus formation in the sinus node, which triggers the contraction of the heart muscle, is disturbed, palpitations occur. Factors that can cause palpitations include: heart rhythm disturbances, heart arrhythmias, taking certain medications, use of psychoactive substances, stress, emotions or physical exertion. [4]

The most commonly observed arrhythmias include sinus bradycardia, defined as a sinus rhythm rate of less than 60 beats per minute, which can result in cerebral hypoperfusion and symptoms such as dizziness or weakness. Tachycardias, including sinus tachycardia, supraventricular tachycardia and ventricular tachycardia, are characterized by accelerated heart rate and often manifest as sudden, intense palpitations. Another group is characterized by premature contractions, both ventricular (PVCs) and atrial (PACs), which can be felt as a "falling out" of the beat or an intensified perception of it. A special form of arrhythmia is the sick sinus syndrome (SSS), especially its form with alternating tachy- and bradyarrhythmias (tachy-brady syndrome), which is associated with dysfunction of sinus node automaticity and hemodynamic instability. The aforementioned disorders require etiological differentiation and appropriate diagnostic and therapeutic management depending on the frequency, severity of symptoms and presence of comorbidities.[5,6]

Non-Arrhythmic Causes of Palpitations

Palpitations may also originate from non-arrhythmic cardiovascular causes, which should be considered during differential diagnosis. Structural abnormalities such as valvular heart disease, particularly aortic regurgitation or aortic stenosis, can lead to increased myocardial workload and compensatory changes in cardiac output, resulting in an irregular or forceful heartbeat sensation. [7] Septal defects, including atrial septal defect (ASD) and ventricular septal defect (VSD), contribute to volume overload and altered intracardiac hemodynamics, which may manifest as palpitations. Cardiomyopathies, especially hypertrophic or dilated forms, disturb normal myocardial function and conduction, promoting mechanical and electrical instability. Congenital heart defects of various types, depending on severity and anatomical presentation, may also induce palpitations through altered chamber compliance or turbulent blood flow. [8] Additionally, pericarditis, characterized by inflammation of the pericardial layers, can cause irritation of the myocardium and provoke symptoms such as chest discomfort and palpitations, particularly in the acute phase. These non-arrhythmic etiologies warrant thorough clinical evaluation and targeted imaging to confirm diagnosis and guide management.

Some substances can directly or indirectly cause palpitations by affecting the electrical conduction, rhythm or contractility of the heart muscle. Among the most common are agents with stimulant or arrhythmogenic effects. Cocaine is a potent sympathicomimetic that can cause tachyarrhythmias, accessory contractions and myocardial ischemia. Alcohol, especially when consumed in excess or in the so-called "holiday heart" syndrome, can lead to arrhythmias, mainly atrial fibrillation. Caffeine, present in coffee, energy drinks and tea, has a stimulating effect on the sympathetic nervous system and can cause palpitations in susceptible individuals. Among arrhythmogenic drugs, digoxin is particularly noteworthy, as it can cause atrioventricular arrhythmias and arrhythmias if overdosed or interacted with. Amitriptyline, a drug in the tricyclic antidepressant group, can prolong the QT interval and predispose to dangerous arrhythmias. Erythromycin and other drugs that prolong the QT interval have a similar effect, increasing the risk of torsade

de pointes. Class I antiarrhythmic drugs, which act by blocking sodium channels, can paradoxically induce ventricular arrhythmias. Calcium channel blockers, although used to treat hypertension and some arrhythmias, can lead to bradycardia and conduction disturbances, especially when combined with other cardiotropic drugs. All of the substances mentioned should be considered in the differential diagnosis of palpitation, especially in patients without previously diagnosed heart disease.[9]

Temporal Pattern of Palpitations

Other causes of palpitations, not directly related to the cardiovascular system, are often systemic and metabolic in nature. Fever can lead to accelerated heart rate as a physiological response to increased body temperature. Dehydration results in decreased circulating blood volume, which activates the sympathetic nervous system and can lead to tachycardia. Hypoglycemia triggers an adrenaline rush, manifested by palpitations, tremors and anxiety, among other symptoms. Anemia, especially in the severe or chronic form, results in a decrease in the blood's ability to transport oxygen, leading to a compensatory acceleration of heart rate to maintain adequate tissue oxygenation. Thyrotoxicosis (hyperthyroidism) affects the cardiovascular system by increasing the sensitivity of β-adrenergic receptors, which can result in tachycardia, atrial fibrillation and increased palpitations. Adrenal pheochromocytoma is a rare tumor that secretes catecholamines, leading to paroxysmal hypertension, tachycardia and severe palpitations. The above conditions require appropriate diagnosis and causal treatment, and should be considered in the context of palpitation diagnosis, especially when no palpable cardiac pathology is found.[10]

Based on the timing of palpitations, two main types can be distinguished:

Paroxysmal (paroxysmal) palpitations - are characterized by sudden onset and abrupt cessation of the symptom. They are often associated with supraventricular arrhythmias, such as paroxysmal atrial tachycardia, and can occur in people without significant structural -changes in the heart.

Non-paroxysmal (non-paroxysmal, non-stress) palpitations - develop gradually, with increasing heart rate, and then gradually subside. This type is often observed in physiological situations such as physical exertion, emotional stress or anxiety disorders, and less often indicates a serious arrhythmia.

Analysis of the nature of the onset and termination of palpitations is an important part of the initial differential diagnosis and can guide further clinical management.[11]

Clinical Differentiation

In evaluating a patient with complaints of palpitations, it is crucial to determine the potential cause of the symptom. Consider whether the palpitations are related to:

Arrhythmia, which is one of the most common causes and can range from mild accessory contractions to severe arrhythmias such as atrial fibrillation or ventricular tachycardias. Cardiovascular disease, such as cardiomyopathy, in which structural changes in the heart muscle lead to impaired mechanics and conduction, generating symptoms of palpitation. Cardiac manifestations of systemic disease, such as hyperthyroidism (thyrotoxicosis), which, by increasing sensitivity to catecholamines and accelerating metabolism, can cause tachycardia and palpitations. Mild somatic symptoms aggravated by psychological factors, such as sinus tachycardia in the course of anxiety disorders, where the autonomic system responds to emotional stress, leading to a sensation of rapid heartbeat despite the absence of significant organic changes. Careful history taking and targeted diagnostics are essential for correct diagnosis and implementation of appropriate treatment. [12,13]

The Significance of Hypertrophic Cardiomyopathy in Sports

Among the various causes of palpitations outlined in the clinical differentiation, hypertrophic cardiomyopathy (HCM) deserves special attention. This is particularly true for athletes and physically active individuals, in whom HCM is a leading cause of sudden cardiac death during exertion. Unlike many other causes of palpitations, HCM is a genetic structural heart disease that may remain clinically silent or present with subtle symptoms such as palpitations, exertional dyspnea, or syncope. Early identification of HCM in this population is crucial because it enables the implementation of preventive strategies and tailored management to reduce potentially fatal outcomes. Therefore, the following section focuses in detail on the pathophysiology, clinical presentation, diagnosis, and management of hypertrophic cardiomyopathy, emphasizing its relevance in the athletic population.

Due to the increased risk of sudden cardiac death during intense physical activity, approximately 50-70% of individuals diagnosed with HCM are advised to discontinue competitive or high-intensity sports. International cardiology guidelines recommend that athletes with symptomatic HCM, significant left ventricular outflow tract obstruction, or high-risk arrhythmias avoid strenuous sports. However, in asymptomatic patients with low-risk profiles, decisions regarding sports participation are individualized but still require careful evaluation and ongoing monitoring. [14]

Hypertrophic cardiomyopathy (HCM)

a. Pathomechanism

Hypertrophic cardiomyopathy is a heart muscle disease that is genetically determined. It is inherited autosomal dominantly in approximately 50% of all cases. In the population, it occurs with a frequency of 1:500. Between 5 and 10% of all cases are due to other genetic disorders, including inherited metabolic and neuromuscular diseases (e.g. amyloidosis, Friedreich's ataxia and mitochondrial diseases).

Mutations in genes encoding sarcomeric proteins such as: beta-myosin heavy chain (chromosome 14), myosin-binding protein C (chromosome 11), troponins (chromosome 1) and alpha-tropomyosin (chromosome 15), lead to their compensatory hypertrophy with the typical irregular arrangement of myocytes, diffuse local fibrosis, as well as abnormalities of the coronary vessels(thickened walls, intramural course, reduced cross-sectional area). [15,16]

Hypertrophic cardiomyopathy is characterised by myocardial hypertrophy that is not due to any of the causes characteristic of the condition, e.g. aortic stenosis or hypertension, which can cause left ventricular overload. Depending on the presence or absence of left ventricular outflow tract stenosis, cardiomyopathies with or without left ventricular outflow tract stenosis are distinguished. [17,18]

b. Subjective and physical symptoms

Table 1. Subjective symptoms and physical findings characteristic of hypertrophic obstructive cardiomyopathy [7]

Subjective symptoms	Physical symptoms
● Exertional dyspnea (the most common symptom) ● Angina pain ● Heart palpitations ● Dizziness ● Syncope or pre-fainting states (especially in forms with left ventricular outflow tract narrowing)	● Systolic murmur, along the left edge of the sternum (it may radiate to the upper edge of the right edge of the sternum and to the apex of the heart) ● Increase in the loudness of the systolic murmur with attempts to reduce the preload or afterload on the left ventricle, e.g., after getting up from a sitting or squatting position and after nitroglycerin administration ● Silence of the murmur after passive elevation of the patient's lower extremities, assuming a squatting or sitting position ● Possible presence of a rapid, two-beat peripheral pulse

c. The most significant findings confirming the diagnosis and severity of the changes

Echocardiography- the primary test used to diagnose and monitor the course of hypertrophic cardiomyopathy. In most patients, significant myocardial hypertrophy is observed, especially of the basal segments of the interventricular septum, but the lateral wall, the posterior part of the septum and the left ventricular apex are also frequently affected. Hypertrophy of the basal part of the septum causes narrowing of the left ventricular outflow tract, which in 30% of cases coexists with mitral valve leaflet regurgitation and forward motion during systole. In 25% of cases, a gradient between the left ventricular outflow tract and the aorta is evident - a gradient of more than 30mmHg can be used to diagnose left ventricular outflow tract narrowing.[19]

Standard 12-lead ECG: patients may have a normal ECG at the time of the study, but complex abnormalities are generally evident, which include left ventricular hypertrophy, abnormal T-waves and ST-segments and pathological Q-waves present.

An important role is played by laboratory tests that can detect non-cardiac conditions that exacerbate symptoms of ventricular dysfunction - e.g. renal failure, diabetes or thyroid disease - and secondary organ failure in the course of severe heart failure. One example is a low haemoglobin level (anaemia) resulting in increased chest pain and breathlessness.[20]

d. Criteria for diagnosis

The diagnosis is made on the basis of any imaging study that shows myocardial wall thickening of more than 15mm in more than one segment of the left ventricular myocardium. In addition to increased wall thickness, myocardial fibrosis, morphologic abnormalities of the mitral valve apparatus, abnormal coronary microcirculatory function and electrocardiographic abnormalities are also noted. [21,22]

e. Management depending on the severity of symptoms

Chest pain

Patients usually complain of pain after exertion or a period of rest. Pain may also occur after eating a heavy meal or alcohol. The management should include basic vital signs (respiration, pulse, blood pressure), subject and physical examination to determine the type and severity of pain. An ECG should be performed in all cases.[23]

Shortness of breath

The management of dyspnea involves communicating with the patient and gaining his trust, teaching effective breathing and expectoration of secretions, developing coping strategies for episodes of dyspnea. It is necessary to give the patient written information about the management: adopting an appropriate body position, such as sitting or lying on the side, breathing exercises, adequate supply of fluids(to dilute the retained secretions). [24,25,26]

Conclusions

Heart palpitations are a frequent and non-specific symptom encountered in various medical settings. While often benign, they may also be the initial manifestation of serious underlying cardiac or systemic disorders. Careful clinical evaluation, including detailed history-taking, physical examination, and appropriate use of diagnostic tools such as ECG and echocardiography, is essential in differentiating between functional and pathological causes. Among structural heart diseases, hypertrophic cardiomyopathy (HCM) stands out as a significant and potentially life-threatening condition frequently presenting with palpitations. Given its genetic basis, variable clinical presentation, and risk of sudden cardiac death, early recognition and management of HCM are crucial. Personalized therapeutic strategies, patient education, and long-term monitoring play a key role in optimizing outcomes for patients experiencing palpitations. Ultimately, palpitations should not be underestimated, as they may serve as an important clinical clue to hidden cardiovascular pathology.[16,17,21]

Disclosures

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