

Clinical evaluation of the athlete

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Abstract

There is a growing body of evidence suggesting that athletic training increases the likelihood of arrhythmias in predisposed athletes, sometimes resulting in sudden cardiac death (SCD). The clinical evaluation of athletes involves a directed history and examination and diagnostic tests including a 12 lead electrocardiogram and other heart rhythm assessment, functional assessment and advanced imaging modalities including echocardiography, cardiac magnetic resonance and computed tomography. This article discusses the diagnostic challenge that sets athletes apart from sedentary individuals: namely the physiological adaptations, referred to collectively as the "athletic heart" that occur as a result of training and that can mimic cardiac pathology.

Keywords: arrhythmias; athlete's heart; clinical evaluation; physiological adaptations; sudden cardiac death.

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Introduction

Since the classical era, athletes have often been considered as some of the greatest heroes of their age. This high regard has continued into modern Western society with athletes being perceived as the supreme combination of health, training, determination and genetic good fortune. It is therefore shocking when an athlete develops cardiac disease or dies suddenly. There is evidence that athletic training can provoke arrhythmias in predisposed individuals, occasionally resulting in sudden cardiac death (SCD). These deaths are the leading cause of non-traumatic death during exercise. The majority of victims of SCD harbour asymptomatic cardiac diseases (Table 1) including inherited cardiomyopathies, channelopathies and structural heart disease, though a significant proportion die from acquired causes such as myocarditis or premature coronary artery disease. In older athletes atherosclerotic coronary artery disease is the most common cause of death.

There are discrepancies relating to the incidence of SCD in younger athletes with estimates ranging between 1 per 23,000 and 1 per 300,000 person years [1]. Regardless of the study however, this number is not insignificant and presents an important problem. Of the deaths in younger athletes, 90% are related to physical exertion, 90% occur in males and black athletes are 2.6 times more susceptible compared with Caucasians. The athlete's sport is also important; the highest incidence of SCD is observed in sports with regular bursts of explosive activity such as American football, soccer, rugby and basketball. In addition to life threatening conditions there is also evidence showing that chronic athletic training increases the risk of atrial fibrillation [2], atrial flutter [3] and sinus node dysfunction [4] in older athletes.

CONGENITAL/INHERITED CAUSES	
Myocardial disease	Hypertrophic cardiomyopathy Arrhythmogenic ventricular cardiomyopathy Dilated cardiomyopathy
Coronary artery disease / anomalies	Anomalous origin of the coronary arteries Premature atheromatous coronary artery disease (age <35 years)
Cardiac conductive tissue abnormalities	Wolff-Parkinson-White syndrome Idiopathic ventricular tachycardia Long-QT syndrome Brugada syndrome Catecholaminergic polymorphic ventricular tachycardia Short-QT syndrome
Valvular heart disease and disorders of the aorta	Mitral valve prolapse Bicuspid aortic valve Marfan's Syndrome
ACQUIRED HEART DISEASE	
Infections	Myocarditis
Drugs	Cocaine, amphetamines, overdoses
Infiltrative disease	Sarcoid, amyloid
Electrolyte disorders	
Hypothermia / hyperthermia	
Trauma (commotio cordis)	
Table 1 Summary of potentially fatal cardiac conditions seen in athletes.	

The diagnostic challenge is that physiological adaptations associated with athletic training, collectively referred to as the “athlete’s heart,” can mimic morphologically mild expressions of disorders implicated in SCD. The differentiation between physiological adaptation and pathology is critical since an erroneous diagnosis has the potential for serious and potentially life changing implications.

The “athlete’s heart”

Exercise training is associated with a number of physiological changes in the cardiovascular system [5,6]. Endurance exercise, such as running, involves a sustained elevation in cardiac output, with a normal or reduced peripheral vascular resistance, resulting in an increased blood volume flowing through all four cardiac chambers. Strength training, such as bodybuilding, involves activity with little increase in cardiac output but significant increases in peripheral vascular resistance, systolic blood pressure and left ventricular afterload. The nature of cardiac adaptation is dependent on the predominant mode of training [7], with the majority of sports combining elements of both endurance and strength training.

Endurance athletes may increase their cardiac output from a resting level of 5–6 liters/min to 40 liters/min at peak exercise. The rise in cardiac output is achieved by an increase in heart rate and augmentation of the stroke volume. Although resting systolic function in athletes is no different to sedentary controls [8], there is evidence of augmentation of early diastolic filling [9]. The majority of the increased stroke volume therefore arises primarily from increased chamber dimensions. Exercise produces an increased haemodynamic load on the aorta which results in remodeling with aortic dilatation [10].

Clinical evaluation

Athletes may present in one of three ways: as part of a pre-participation medical screening, as part of a medical consultation for another reason, for evaluation of symptoms suggestive of cardiac disease. The approach to all three is similar and requires the eliciting of a relevant clinical history and cardiovascular examination. Depending on their outcome further investigations may be indicated.

History and physical examination

Both screening and symptom-directed evaluation of the athletic patient should concentrate on identifying

the occurrence of a number of relevant cardiogenic symptoms including exertional chest pain or discomfort, breathlessness disproportionate to the amount of exercise being performed, palpitations and syncope. These symptoms occur in 35% of young patients prior to SCD [11]. In the event of syncope an eyewitness account of the episode can be helpful. Differentiating syncope from epilepsy is clinically difficult. In general all athletes with a label of epilepsy should be investigated for an arrhythmogenic cardiac disorder. A thorough family history is crucial given the inherited nature of most conditions implicated in SCD. A family history of known hereditary cardiac disease, sudden or premature cardiac death in relatives under the age of 50 years warrants comprehensive cardiac evaluation. Furthermore, a family history of epilepsy, syncope, unexplained drowning or road traffic accidents may be vital clues to a potentially fatal hereditary disorder. It is important to identify the use of medications associated with prolongation of the QT-interval and recreational or performance enhancing drugs such as cocaine, amphetamines and anabolic steroids which may be implicated in cardiomyopathy and arrhythmogenesis.

The physical examination should focus on the identification of peripheral stigmata of hypercholesterolaemia, hypertension, co-arctation of the aorta, valvular heart disease and Marfan's syndrome although most causes of SCD are associated with normal physical examination.

Pre-participation screening with an ECG

Pre-participation screening is a highly contended area with differences in opinion as to whether athletes should undergo a routine ECG screening. Using evidence of reduced mortality in athletes resulting from the program of Italian national pre-participation screening [12], the current European Society of Cardiology (ESC) guidelines [13] recommend routine acquisition of a resting 12 lead ECG. The American Heart Association take an opposing stance [14] due to the large financial and logistical requirements of implementation of such a program at National level [15]. Whether an ECG is routinely performed in the absence of relevant history or examination findings is beyond the scope of this article; however an abnormal ECG may be the only clinical manifestation of a quiescent cardiac disorder.

The 12-lead ECG

Whenever there is a clinical indication, a 12 lead ECG should be routinely performed. The athletic adaptation in autonomic activation is at least partially responsible for the common ECG findings of sinus bradycardia, first degree and Mobitz type I second degree atrioventricular nodal block, an increase in premature beats and altered repolarisation (seen as J-point elevation, ST-segment elevation and high amplitude T-waves). Up to two thirds of athletes demonstrate ECG criteria for left ventricular hypertrophy (LVH) [16,17]. Race is an important factor in repolarisation abnormalities in particular as they occur twice as often in black athletes when compared to Caucasian athletes [18]. However, there is considerable overlap between repolarization changes observed in athletes with those identified in disorders implicated in SCD. Therefore ECG changes must always be interpreted within the individual's context. The ESC have produced guidelines [19] where ECG changes are divided into Group 1 and Group 2 (Table 2). The former are considered common, training-related ECG changes that in isolation are not an indication for further investigation, while the latter are uncommon and should trigger further investigation.

The ECG is limited in that it fails to diagnose most cases of coronary artery stenoses and potentially serious coronary artery anomalies. The main concern with ECG screening is the potentially high number of false positive findings as a result of athletic heart adaptations. Using the ESC guidelines on ECG interpretation however this proportion can be significantly reduced to less than 10% [20,21]. A normal ECG however, has a very strong negative predictive value in excluding cardiomyopathies [22]. It is also useful in identifying accessory pathways and ion-channelopathies such as congenital long QT syndrome and Brugada syndrome.

Heart rhythm assessment

Detailed guidance on investigating arrhythmias has been published [23] and the following is a brief overview only. In athletes presenting with symptoms it is often useful to perform prolonged cardiac monitoring with a Holter monitor. While sinus bradycardia, first degree heart block and second degree heart block (Wenkeback type) are usually normal, second degree atrioventricular (AV) block (Mobitz Type II) or third degree AV block should be considered pathological if

Group 1: Common and training-related ECG changes	Group 2: Uncommon and training unrelated ECG changes
<ul style="list-style-type: none"> • Sinus bradycardia • First-degree AV block • Incomplete RBBB • Early repolarisation • Isolated QRS voltage criteria for left ventricular hypertrophy 	<ul style="list-style-type: none"> • T-wave inversion • ST-segment depression • Pathological Q-waves • Left atrial enlargement • Left-axis deviation / left anterior hemiblock • Right-axis deviation / left posterior hemiblock • Ventricular pre-excitation • Complete LBBB or RBBB • Brugada-like early repolarisation
<p>Table 2 Classification of abnormalities of the athlete's electrocardiogram [19]. AV atrioventricular, RBBB right bundle branch block, LBBB left bundle branch block.</p>	

the bradyarrhythmia persists after a 6-week period of detraining or in the presence of symptoms. An exercise stress test is useful for provoking arrhythmias associated with adrenergic surges such as right ventricular outflow tract ventricular tachycardia, polymorphic ventricular tachycardia associated with congenital long QT 1 and 2, and the identification of catecholaminergic polymorphic ventricular tachycardia. In individuals with infrequent palpitations without syncope, a portable Cardiomo device may be required in order to capture the cardiac rhythm during symptoms. In athletes with infrequent syncope an implantable loop recorder may be useful. In selected patients with a history suggestive of recurrent vasovagal syncope, tilt table testing may be indicated.

Functional assessment

Apart from the identification of exercise induced arrhythmias functional testing with cardiopulmonary exercise testing is used to assess fitness in athletes with structurally normal hearts who are experiencing a decline in performance. An exercise test may expose myocardial ischaemia and may be particularly useful in differentiating structural changes associated with physical training from cardiomyopathy.

Imaging assessment

An understanding of the normal limits of adaptive remodeling is critical in differentiating athletic adaptations from cardiomyopathy. In a cohort of 1309 elite male and female athletes [24] 14% had LV dilatation, with left ventricular end-diastolic diameter (LVEDD) >60mm, a dimension compatible with a dilated cardiomyopathy

diagnosis. This increase in cavity size was not observed in strength-trained athletes [6]. Factors that appear to play a role in the development of larger cavity dimensions with LVH include male gender, endurance sports, a longer duration of training, increased body mass and adult physical maturity. While mild aortic root dilatation is common, a diameter of >40mm is rare and associated with increased height, though not necessarily Marfan's syndrome [25].

LVH occurs both as eccentric hypertrophy as a result of the dilated cavity in endurance athletes, but also as a result of concentric hypertrophy in both endurance and strength-trained athletes. Within a cohort of 947 athletes however only 1.7% of male and no females participants developed a wall thickness of ≥ 13 mm—an arbitrary cut-off, but a significant one as it may overlap with hypertrophic cardiomyopathy [26]. In this study all the individuals with wall thickness ≥ 13 mm also had LV cavity dilatation which is not a feature of hypertrophic cardiomyopathy.

Transthoracic echocardiography is a cheap and effective tool to allow the exclusion of many structural abnormalities and it is still the best modality to assess valvular function. Athletes usually have excellent ultrasound windows and in a majority of cases it is even possible to visualize the coronary artery origins. In cases where better visualization of valvular or other structural abnormalities are required transoesophageal echo or other imaging modalities often offer additional benefits. The overlap between the structural findings of the athletic heart and cardiomyopathies mean echo alone is occasionally non-diagnostic and other imaging modalities are required. The role of Computed

Tomography (CT) and cardiac magnetic resonance (CMR) in athletes is reviewed in depth elsewhere [27] and while both are excellent in specific cases, they are required infrequently. CMR with gadolinium contrast is valuable for differentiating between normal myocardium, cardiomyopathies and infiltrative diseases and to localize focal fibrosis or scar. CMR is particularly useful in visualizing the left ventricular apex and right ventricle. It involves no radiation dose but is relatively expensive, time-consuming, can cause claustrophobia and is contraindicated in patients with pacemakers or other metallic foreign bodies or severe renal disease. CT has the advantage of being a quick procedure and if the exclusion of significant coronary artery plaque is required it has a high negative predictive value. The disadvantage of the previously considerable radiation dose has been relatively reduced by recent advances in radiation reduction techniques. Both CMR and CT are appropriate investigations to exclude anomalous coronary arteries.

Differentiating between athletes heart and cardiomyopathy

The overlap of athlete's heart with hypertrophic cardiomyopathy (HCM) is often referred to as the "grey zone" (Fig. 1). Repolarisation abnormalities and a maximum left ventricular wall thickness of $\geq 13\text{mm}$ are unusual but potentially normal findings in athletes. However such findings are common in HCM and so should prompt further investigation. Imaging is usually the key to diagnosis but detraining for 6 weeks may be a useful adjunct as the majority of ECG abnormalities due to athletic activity resolve. Echocardiographic features suggestive of HCM include systolic anterior motion of the anterior mitral valve leaflet, mitral leaflet elongation, LV outflow tract obstruction, impaired diastolic function and a small LV cavity. CMR has emerged as a useful imaging modality to differentiate between causes of LVH [28]. CMR in HCM demonstrates characteristic patchy late gadolinium enhancement (LGE), abnormal LV architecture, several characteristic patterns of hypertrophy, and diffuse perfusion defects. A final method to differentiate athlete's heart from pathological hypertrophy [29], is if the maximum end-diastolic wall to volume ratio $>0.15 \text{ mm/m}^2/\text{ml}$.

There is a similar overlap with the athletes heart and dilated cardiomyopathy (DCM) where again CMR is useful in demonstrating a characteristic mid-wall LGE

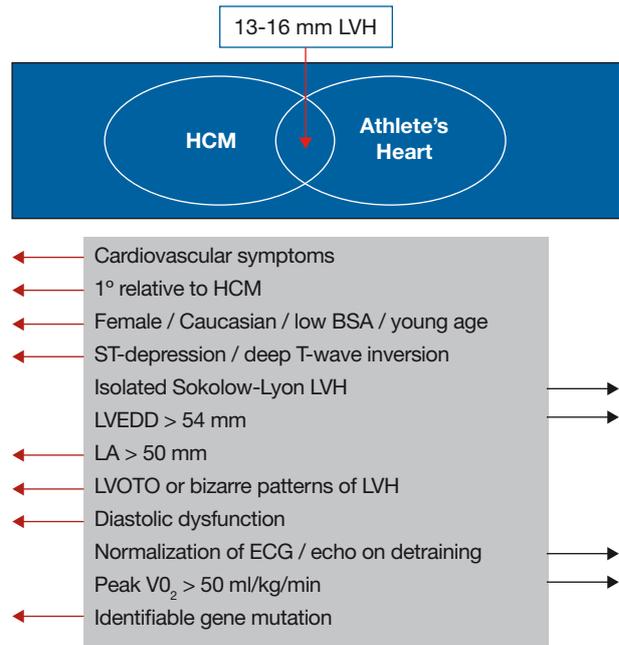


Fig. 1 Clinical criteria used to differentiate athlete's heart from HCM in cases with a borderline degree of LVH of 13–16mm [6]. LVH left ventricular hypertrophy, HCM hypertrophic cardiomyopathy, BSA body surface area, LVWT left ventricular wall thickness, LVEDD left ventricular end-diastolic diameter, LA left atrium, LVOTO left ventricular outflow tract obstruction, VO_2 oxygen consumption.

and when relevant, non-compaction. The differentiation is usually more straightforward however as impaired systolic and diastolic function will also be present in DCM. CMR is the imaging modality of choice in the diagnosis of arrhythmogenic right ventricular cardiomyopathy (ARVC) where the findings of right ventricular dilatation, focal aneurysms and regional wall motion abnormalities, and fibrofatty myocardial infiltration of the apices are part of the diagnostic criteria for the disease [30].

Summary

The initial clinical assessment of the athlete follows the same template as any other patient. However, the physiological adaptations developed as a result of chronic athletic training need to be understood to reduce the number of false positive tests and to prevent erroneous diagnoses. The differentiation between adaptive physiology and pathology has improved in the last decade with additional investigational tools becoming widely available including improved non-invasive testing and imaging modalities that make this differentiation easier to make. •

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