The athlete's heart

The deaths of a number of high-profile athletes within the sporting community have focused attention on the phenomenon of 'the athlete's heart'. This review highlights the electrical and morphological changes observed in the hearts of athletes, and provides practical methods for differentiating physiology from disease.

It has been recognized for over a century that the hearts of athletes differ from those of non-athletic individuals. Henschen (1899) described dilatation and hypertrophy of both sides of the heart in cross-country skiers, with manual chest percussion as his only diagnostic tool. In the past three decades, a considerable amount of literature has focused on the physiological aspects of electrical and structural remodelling of the athlete's heart. This topic has been brought into the spotlight by the tragic and untimely deaths of a number of young professional sportsmen as a result of undiagnosed inherited or congenital cardiac disease. Diagnostic difficulty may arise during cardiovascular evaluation of highly trained athletes, since physiological changes resulting from training may closely resemble cardiac diseases associated with potential fatality, particularly hypertrophic cardiomyopathy. This article discusses the physiology of the athlete's heart, and guides the reader through the differentiation between healthy cardiac remodelling and the pathological conditions that can lead to sudden death.

Definition

The term 'athlete's heart' describes the constellation of cardiac morphological, functional and electrical adaptations resulting from systematic training (*Figure 1*). Such changes are generally regarded as physiological and benign.

Physiology

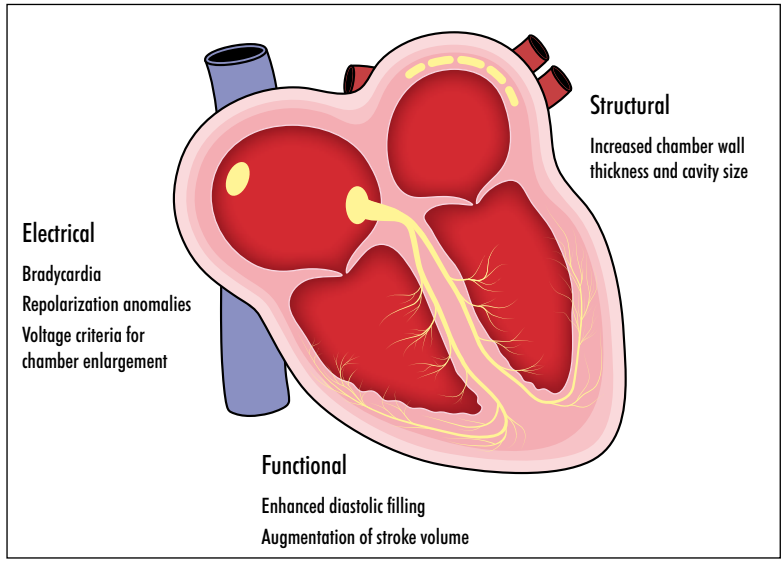
Cardiovascular responses to exercise differ according to the nature of the activity. Dynamic exercise, for example long-distance running, results in increased heart rate and stroke volume, reduced peripheral vascular resistance and a modest rise in blood pressure, creating a volume load on the left ventricle. Static exercise, such as weight lifting, results in a slight rise in heart rate and a significant rise in blood pressure, causing a pressure load on the left ventricle. Most sporting disciplines involve a combination of static and dynamic exercise in varying proportions.

Sudden death in young athletes and pre-participation screening

The incidence of sudden cardiac death in young athletes is estimated to range between 1 in 50 000 (Corrado et al, 2003) and 1 in 200 000 per year (Maron et al, 1998). Strenuous exercise may act as a trigger for lethal arrhythmias in athletes harbouring potentially sinister conditions. Indeed, the risk of sudden death in young athletes with cardiac disease is increased 2.8-fold during competitive sports (Corrado et al, 2003), and sudden cardiac death is often the first presentation.

The vast majority of deaths in athletes are the result of hereditary or congenital disorders affecting the structural, functional and electrical properties of the myocardium. Hypertrophic cardiomyopathy is the commonest cause of sudden cardiac death in young athletes worldwide (Maron et al, 2007), accounting for over a third of these deaths (36%). Coronary artery anomalies are the second most common underlying pathology (17%). A variety of acquired conditions are also implicated, including acute myocarditis, commotio cordis and illicit drug abuse. Arrhythmogenic right ventricular cardiomyopathy accounts for the greatest number of fatalities in Italian athletes (Corrado et al, 2003). This may in part represent a unique genetic substrate, but may also be a consequence of the Italian programme of mandatory pre-participation cardiovascular screening of all young (≤35 years) competitive athletes. Since 1982, this scheme has led to a 90% mortality reduction in young Italian athletes (Corrado et al, 2006), with the near-eradication of deaths caused by hypertrophic cardiomyopathy (Corrado et al, 1998).

Figure 1. Structural, functional and electrical changes in the athlete's heart.



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The European Society of Cardiology advocates pre-participation cardiovascular screening of all young competitive athletes via 12-lead electrocardiogram in addition to history and clinical examination (Corrado et al, 2005). These guidelines have been endorsed by the International Olympic Committee, as well as international football governing bodies. The evidence for this strategy is derived largely from the Italian experience. Pre-participation cardiovascular screening with history, examination and electrocardiogram is cost effective (Wheeler et al, 2010), although the addition of routine echocardiography is prohibitively expensive. Despite the European Society of Cardiology guidelines, most countries have not instituted systematic state-sponsored pre-participation cardiovascular screening of young athletes, because of a lack of expertise, resources and infrastructure. In the UK, pre-participation cardiovascular screening of athletes is dependant on the work of charitable organizations.

Morphology of the athlete's heart

It has been well established through echocardiographic and cardiac magnetic resonance imaging studies that around 50% of trained athletes exhibit morphological cardiac changes consisting of chamber enlargement and hypertrophy. An early study by Maron (1986) compared more than 1000 male athletes with non-athletic controls. Mean values for maximal left ventricular wall thickness and end-diastolic diameter were 19% and 10% greater respectively in the athletic group.

Limits of normality

Athletes demonstrate on average a 10–20% increase in left ventricular wall thickness and left ventricular end-diastolic diameter. However, this is highly variable and there is considerable overlap with controls matched for age and gender. European Society of Cardiology reference limits for non-athletes are left ventricular wall thickness ≤10 mm and left ventricular end-diastolic diameter ≤59 mm in males, and left ventricular wall thickness ≤9 mm and left ventricular end-diastolic diameter ≤53 mm in females (Lang et al, 2005). Upper reference limits for left ventricular end-diastolic diameter, left ventricular wall thickness and right ventricular end-diastolic diameter in athletes are summarized in *Table 1*, according to gender, race and age, with reference limits for non-athletes given for comparison.

Pelliccia et al (1991) demonstrated a left ventricular wall thickness greater than the reference limit for non-athletes in over a quarter of 947 elite male and female athletes. However, left ventricular wall thickness in the range compatible with hypertrophic cardiomyopathy (≥13 mm) was extremely uncommon (2%). Moreover, this degree of wall thickening was always accompanied by a dilated left ventricle, and exclusively found in rowers, canoeists and cyclists. None of the athletes had left ventricular wall thickness >16 mm (*Figure 2a*).

Pelliccia et al (1999) also studied left ventricle cavity dimensions in 1309 elite male and female athletes. A wide range of values was seen, with greater left ventricular end-diastolic diameter values seen in male athletes (*Figure 2b*). Marked cavity dilatation (left ventricular end-diastolic diameter ≥60 mm) in the range compatible with possible dilated cardiomyopathy was seen in 14% of athletes. No athletes had a left ventricular end-diastolic diameter >70 mm.

Determinants of cardiac dimensions in athletes

Physiological adaptation to exercise is influenced by a number of demographic factors including age, sex, size and ethnicity, as well as the sporting discipline. In general, large males participating in endurance sports have the greatest cardiac dimensions and most profound electrocardiographic changes. Athletes of African or Afro-Caribbean origin (black athletes) exhibit a greater magnitude of left ventricular hypertrophy and more marked repolarization changes than their caucasian counterparts. The key determinants of cardiac dimensions in the athlete's heart are summarized in *Figure 3*.

Table 1. Upper reference limits* for left ventricular end-diastolic diameter, left ventricular wall thickness and right ventricular end-diastolic diameter in athletes and non-athletes

		Gender	Left ventricular end-diastolic diameter (mm)	Left ventricular wall thickness (mm)	Right ventricular end-diastolic diameter (mm)
Non-athletes		M	≤59 (Lang et al, 2005)	≤10 (Lang et al, 2005)	≤55 (Prakken et al, 2010)†
		F	≤53 (Lang et al, 2005)	≤9 (Lang et al, 2005)	≤48 (Prakken et al, 2010) †
Athletes	Caucasian adult	M	≤63 (Pelliccia et al, 1999)	≤12 (Pelliccia et al, 1991)	≤58 (Prakken et al, 2010) †
		F	≤56 (Pelliccia et al, 1999)	≤11 (Pelliccia et al, 1996)	≤52 (Prakken et al, 2010) †
	Caucasian adolescent	M	≤58 (Makan et al, 2005)	≤12 (Sharma et al, 2002)	Not known
		F	≤54 (Makan et al, 2005)	≤11 (Sharma et al, 2002)	Not known
	Black adult	M	≤62 (Basavarajaiah et al, 2008)	≤15 (Basavarajaiah et al, 2008)	Not known
		F	≤56 (Rawlins et al, 2010)	≤12 (Rawlins et al, 2010)	Not known

* Upper reference limits are 95th percentile values (mean + 2 standard deviations), derived from the study referenced in each case. † Value derived from magnetic resonance imaging study.

Sex

Male athletes have greater increases in cardiac dimensions than female athletes. Pelliccia et al (1996) compared 600 female athletes with female sedentary controls and male athletes of similar age and sporting discipline. Although left ventricular wall thickness and left ventricular end-diastolic diameter were greater in female athletes than female controls, both dimensions were greatest in male athletes. Left ventricular wall thickness >12 mm was seen in 2% of male athletes compared with none of the female athletes, while left ventricular end-diastolic diameter ≥60 mm was exhibited by 24% of male athletes but less than 1% of female athletes.

Age

Adult athletes demonstrate a greater degree of cardiac remodelling than adolescent athletes of similar sex and sporting discipline, who are physically less mature and have been exposed to shorter periods of intense training. Makan et al (2005) reported significantly greater left ventricle cavity dimensions in 900 elite adolescent athletes compared with sedentary controls. However, unlike adult athletes in previous studies, no adolescent athletes had left ventricular end-diastolic diameter >60 mm. Sharma et al (2002) studied left ventricular wall thickness in 720 elite junior athletes. The extent of left ventricular hypertrophy demonstrated was less profound than previously documented in adult athletes, with only 0.4% of adolescent athletes having left ventricular wall thickness >11 mm, and none greater than 12 mm.

Ethnicity

Basavarajaiah et al (2008) compared 300 nationally ranked, normotensive, black male athletes with white male athletes and controls. Left ventricular wall thickness ≥13 mm was seen in 18% of black athletes, compared with 4% of white athletes matched for age, size and sporting discipline. Left ventricular wall thickness ≥15 mm was seen in 3% of black athletes but none of the white athletes. Their data suggest upper limits of left ventricular wall thickness ≤15 mm and left ventricular end-diastolic diameter ≤62 mm to be considered the normal physiological responses to exercise in male black athletes.

Rawlins et al (2010) compared 240 black female athletes with matched white female athletes. Left ventricular wall thickness ≥12 mm was seen in 3% of black female athletes but none of the white female athletes.

Sporting discipline

According to Morganroth's hypothesis (Morganroth et al, 1975), volume load in dynamic (i.e. endurance) exercise should lead to predominant dilatation of the left ventricle and a proportional increase in wall thickness ('eccentric left ventricular hypertrophy'). Conversely, pressure load in static (i.e. strength) exercise should result in thickening of the left ventricle wall with preserved cavity dimensions ('concentric left ventricular hypertrophy'). The reality is usually less clear-cut, as most endurance sports involve elements of strength training and vice versa.

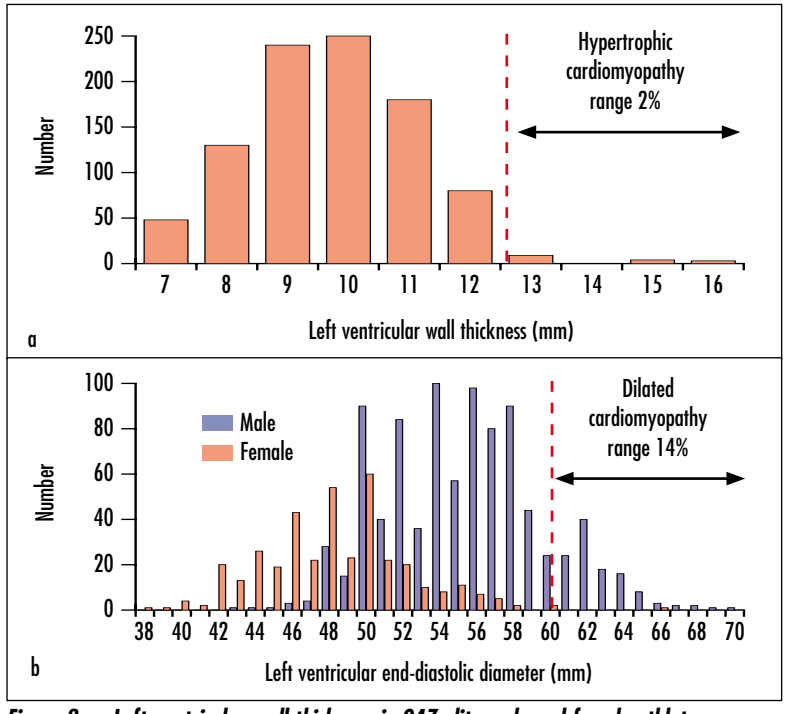


Figure 2. a. Left ventricular wall thickness in 947 elite male and female athletes (Pelliccia et al, 1991). b. Left ventricular end-diastolic diameter in 1309 elite male and female athletes (Pelliccia et al, 1999).

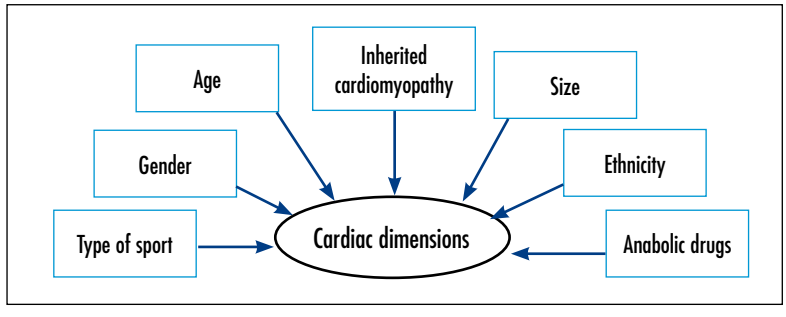


Figure 3. Key determinants of left ventricular dimensions in athletes.

However, as demonstrated by Spirito et al (1994), the extent of left ventricular remodelling is influenced by the type of sporting discipline, with the most marked increases in cavity dimensions and wall thickness seen in elite rowers, cross-country skiers, cyclists and swimmers, followed by long-distance runners, soccer and tennis players.

Morphology of the right ventricle

The response of the right ventricle to systematic exercise has been less extensively studied than that of the left ventricle. Pulmonary artery pressure increases significantly on exercise. However, in contrast to the left-sided circulation, there is minimal concomitant reduction in pulmonary vascular resistance. Cardiac output and heart rate must be equivalent in both ventricles, and conse-

quently the right ventricle is subject to a greater work load than the left ventricle during exercise.

Exercise results in similar changes in left ventricle and right ventricle mass, volume and function (Scharhag et al, 2002), lending support to the concept of a 'balanced' enlargement of the heart (*Figure 4*). Echocardiographic normal values for right ventricle dimensions in athletes have not been established, since currently used reference limits are derived from small studies of non-athletes. A cardiac magnetic resonance imaging study by Prakken et al (2010) suggested upper reference limits for right ventricle end-diastolic diameter of 58 mm in elite male athletes (*vs* 55 mm in controls), and 52 mm in elite female athletes (*vs* 48 mm in controls). Most studies have shown that right ventricle function does not differ between athletes and non-athletic controls (Scharhag et al, 2002).

Cardiac function

Numerous studies, including a meta-analysis by Fagard (1996), have shown no difference between left ventricular systolic function in athletes and non-athletic controls. This is supported by data demonstrating unchanged systolic function in athletes in differing training states (Fagard et al, 1996) and after long periods of de-training (Pelliccia et al, 2002). The majority of studies have also demonstrated normal left ventricular diastolic function in athletes. In fact, 'supra-normal' diastolic function is frequently seen, which may improve left ventricular filling during periods of rapid heart rate (Fagard, 1997).

Figure 4. Echocardiographic apical four-chamber views of the heart of (a) a non-athlete and (b) an athlete. Physiological dilatation of both ventricles and both atria is seen in the athlete, giving rise to a 'balanced' cardiac enlargement.

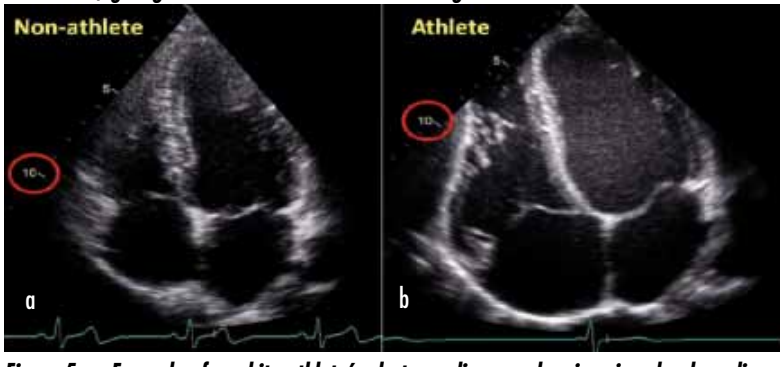
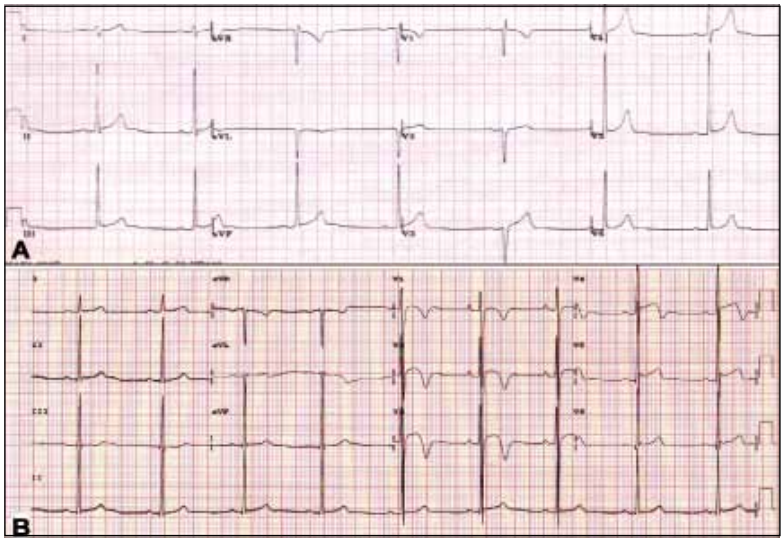


Figure 5. a. Example of a white athlete's electrocardiogram showing sinus bradycardia, Sokolow–Lyon voltage criteria for left ventricular hypertrophy, and tall T waves. b. Electrocardiogram of a healthy African marathon runner showing voltage criteria for left ventricular hypertrophy as well as marked repolarization changes including ST segment elevation in leads V1–V5 and deep T wave inversion in leads V1–V4.



The electrocardiogram

Electrocardiogram changes are common in athletes and usually reflect physiological electrical and structural cardiac remodelling. Frequently observed changes include rhythm and conduction alterations, increased voltages and repolarization anomalies. Pelliccia et al (2000) correlated electrocardiogram findings with echocardiography in 1005 athletes competing in 38 sporting disciplines. A distinctly abnormal electrocardiogram was seen in 40% of cases, yet only 5% exhibited structural cardiac abnormalities on echocardiography. The majority of electrocardiogram changes in athletes therefore reflect physiological structural remodelling as well as the effects of increased vagal tone. Occasionally, specific repolarization anomalies overlap with incomplete expression of, or morphologically mild, cardiomyopathy. The differentiation between physiological changes and cardiac pathology is crucial in order to prevent mortality, and conversely to avoid unnecessary disqualification from sport.

Sinus bradycardia and Sokolow–Lyon voltage criteria for left ventricular hypertrophy are common findings in the athlete's electrocardiogram (*Figure 5a*). A small number of athletes (5%) also exhibit nodal rhythm or Mobitz type 1 (Wenkebach) second-degree atrioventricular block at rest, but revert to sinus rhythm with mild exertion, which should be taken as evidence of normal physiological conditioning. Common repolarization changes include J point and ST segment elevation, and high amplitude T waves. The ST segment usually exhibits a concave shape in the inferior and lateral leads and occasionally a convex shape in the anterior praecordial leads.

The European Society of Cardiology has produced consensus guidelines for interpretation of the 12-lead electrocardiogram in athletes (Corrado et al, 2010), which categorize changes seen in the athlete's electrocardiogram into two groups (*Table 2*). Group 1 includes common and training-related electrocardiogram changes that should not cause alarm in the absence of a positive family history, symptoms or abnormal physical findings. Group 2 consists of uncommon and training-unrelated changes, which may represent underlying heart disease with an increased risk of sudden cardiac death. Group 2 electrocardiogram changes therefore usually require additional diagnostic evaluation.

However, these guidelines are derived from adult caucasian athletes and cannot be fully extrapolated to juvenile athletes (less than 16 years of age) or adult athletes of

African or Afro-Caribbean descent. The authors' personal experience of over 10 000 cardiac evaluations in athletes in the UK suggests that right axis deviation is normal in athletes of all age groups, as is isolated voltage criterion for left atrial enlargement in adolescent athletes. Right axis deviation and incomplete right bundle–branch block are thought to represent increased right ventricle size in athletes. Specific repolarization anomalies, notably T wave inversion, should warrant further investigation in all caucasian athletes over 16 years of age. T wave inversion confined to leads V1–V4 in juvenile athletes and black adult athletes may be normal (see below).

Electrocardiogram repolarization patterns in athletes of African or Afro-Caribbean origin

Ethnic variation in physiological remodelling of the athlete's heart is also reflected in the electrocardiogram. Magalski et al (2008) reported that electrocardiogram abnormalities were twice as common in black compared to white athletes. Basavarajaiah et al (2008) reported electrocardiogram and echocardiographic findings in 300 black athletes and 300 matched white athletes, demonstrating Sokolow–Lyon voltage criteria for left ventricular hypertrophy in 68% of black athletes compared with 40% of white athletes. More striking were the differences in repolarization patterns between the two ethnic groups. ST segment elevation was present in 85% of black and 62% of white athletes, while deep T wave inversions (>0.2 mV) were seen in 12% of black athletes but none of the white athletes.

Papadakis et al (2010) reported similar findings in a 911 black and 858 white athletes. Deep T wave inversions were seen in 16% of black athletes compared with 2% of white athletes. However, these changes were largely confined to the anterior praecordial leads (*Figure 5b*). Full clinical evaluation of those athletes with anterior T wave inversions did not reveal any underlying cardiac pathology. Furthermore, a period of de-training in a subset of athletes led to resolution of these changes. One black athlete with deep T wave inversions in the inferior and lateral leads, but a structurally normal heart on imaging, experienced aborted sudden cardiac death. A second black athlete with similar inferior and lateral T wave inversions was subsequently diagnosed with hypertrophic cardiomyopathy. In black athletes, therefore, deep T wave inversions in leads V1–V4 are likely to represent a benign finding, while T wave inversion extending to the inferior and lateral leads should raise the suspicion of cardiac pathology.

The diagnostic dilemma: athlete's heart or hypertrophic cardiomyopathy?

The key challenge in the athlete's heart lies in the differentiation of physiological adaptation to exercise from cardiac pathology that may predispose to sudden death. The commonest scenario is the athlete with left ventricular hypertrophy ranging between 13 and 16 mm. In such cases, differentiation between physiological left ventricular hypertrophy and hypertrophic cardiomyopathy is made on the basis of the following:

- Personal and family history
- Demographics
- Echocardiography
- Electrocardiogram
- De-training
- Cardiopulmonary exercise testing
- Cardiac magnetic resonance imaging
- Genetic testing.

Figure 6 summarizes the clinical criteria used to differentiate athlete's heart from hypertrophic cardiomyopathy.

Table 2. Classification of abnormalities of the athlete's electrocardiogram

Group 1: Common and training-related electrocardiogram changes	Sinus bradycardia
	First-degree atrioventricular block
	Incomplete right bundle–branch block
	Early repolarization
	Isolated QRS voltage criteria for left ventricular hypertrophy
Group 2: Uncommon and training unrelated electrocardiogram changes	T wave inversion
	ST segment depression
	Pathological Q waves
	Left atrial enlargement
	Left-axis deviation or left anterior hemiblock
	Right-axis deviation or left posterior hemiblock
	Ventricular pre-excitation
	Complete left or right bundle–branch block
	Brugada-like early repolarization

From Corrado et al (2010)

Personal and family history

The presence of cardiovascular symptoms such as exertional chest pain, dyspnoea, palpitations or syncope, or a family history of hypertrophic cardiomyopathy or sudden death should raise the index of suspicion of an underlying pathological process.

Demographics

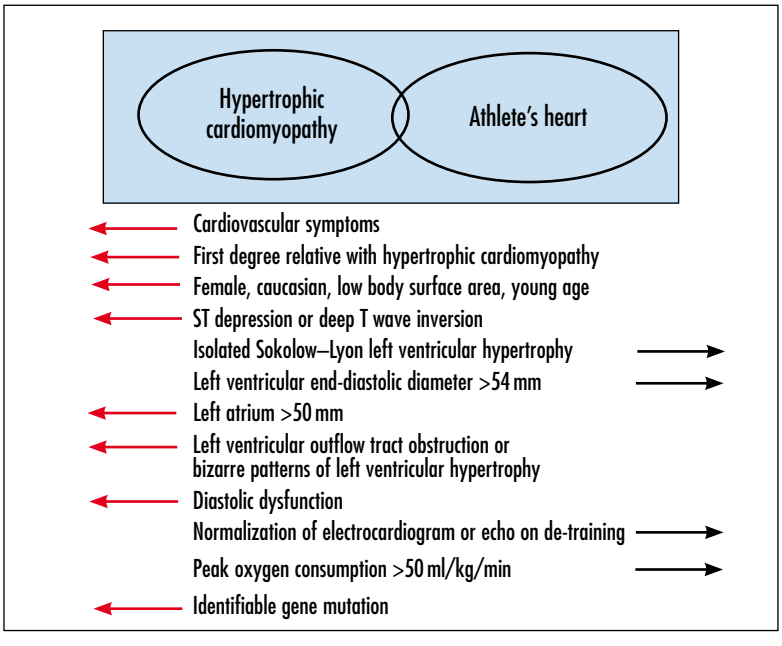
Left ventricular hypertrophy is confined to large male athletes participating in endurance sports, and athletes of African or Afro-Caribbean origin.

Echocardiography

Left ventricular wall thickness values exceeding reference values for athletes of similar age, gender, size and sporting discipline (*Table 1*) warrant further investigation to exclude hypertrophic cardiomyopathy. When left ventricular wall thickness falls in the 'grey zone' between 13 and 16 mm, other echocardiographic markers of pathology may be of use. The first of these markers is an abnormal distribution of left ventricular hypertrophy. In the athlete's heart, the pattern of hypertrophy is almost always symmetrical, while hypertrophic cardiomyopathy results in asymmetrical septal hypertrophy in 60% of cases and apical hypertrophy in 10% of cases. Second, the ratio of left ventricular wall thickness to left ventricular end-diastolic diameter is preserved in athletes, while left ventricular hypertrophy with reduced cavity size is characteristically seen in hypertrophic cardiomyopathy (*Figure 4*). Finally, abnormal morphology and architecture of the papillary muscles and mitral valve apparatus are frequently seen in hypertrophic cardiomyopathy, leading to systolic anterior motion of the mitral valve and outflow tract obstruction in around a third of cases. Systolic anterior motion and mitral valve and outflow tract obstruction should not be seen in the athlete's heart.

Echocardiographic assessment of cardiac function may also help to differentiate athlete's heart from hypertrophic cardiomyopathy. Impaired diastolic function is an abnormal finding in the athlete's heart but is a common finding in hypertrophic cardiomyopathy, often preceding the appearance of overt structural or systolic functional abnormalities.

Figure 6. Clinical criteria used to differentiate athlete's heart from hypertrophic cardiomyopathy in cases with a borderline degree of left ventricular hypertrophy (13–16 mm).



Electrocardiogram

The electrocardiogram is extremely useful in aiding differential diagnosis. The presence of pathological Q waves, inferior or lateral T wave inversion, left bundle–branch block, left axis deviation with voltage criteria for left atrial enlargement, or ST segment depression is highly suggestive of hypertrophic cardiomyopathy in an athlete with left ventricular hypertrophy on echocardiography.

De-training

A period of cessation of sporting activity (de-training) can lead to the resolution of left ventricular hypertrophy in the athlete's heart (Pelliccia et al, 2002; Basavarajaiah et al, 2006). Conversely, de-training will not result in a reversal of the abnormalities associated with underlying hypertrophic cardiomyopathy, and is thus a powerful discriminator of disease. However, de-training is undesirable for competitive athletes striving for honours, and its role in the diagnostic process is therefore limited.

Cardiopulmonary exercise testing

The combination of left ventricular hypertrophy, small left ventricle cavity size and impaired myocardial relaxation in hypertrophic cardiomyopathy is associated with a failure to augment stroke volume on exercise, and low peak oxygen consumption (peak VO_2). In contrast, athletes with a large left ventricle cavity and supra-normal diastolic function achieve a high cardiac output and peak VO_2. Sharma et al (2000) showed that peak VO_2 >50 ml/kg/min, or greater than 120% predicted for age, gender and size is consistent with athlete's heart rather than hypertrophic cardiomyopathy.

Magnetic resonance imaging

Cardiac magnetic resonance imaging can help to differentiate athlete's heart from hypertrophic cardiomyopathy. It permits excellent visualization of the cardiac apex and lateral left ventricle wall, which are often poorly seen on echocardiography. Areas of myocardial fibrosis, which are consistent with hypertrophic cardiomyopathy, may also be detected by cardiac magnetic resonance imaging.

Genetic testing

In families in which a definitive gene mutation has already been identified, genetic testing of a new suspected

case can often confirm the diagnosis. However, the heterogeneity of the molecular genetics of hypertrophic cardiomyopathy makes gene testing time consuming, labour intensive and expensive. Results are usually not available for several months, which is impractical for competitive athletes. There is therefore a limited role for genetic testing in most cases.

Conclusions

Systematic training results in cardiac morphological, functional and electrical changes that collectively comprise the athlete's heart. These changes can closely resemble pathological conditions that may lead to sudden death. Differentiation between healthy remodelling and cardiac disease can save lives. This process requires careful consideration of evidence gathered from history, clinical examination and cardiac investigations. Such information must be interpreted in the context of each individual athlete's unique demographic profile. Future research is likely to focus on the complete characterization and quantification of the right ventricular response to chronic exercise, which will complement our existing knowledge of the athlete's heart. BJHM

Conflict of interest: none.

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KEY POINTS

- Systematic training results in morphological, functional and electrical cardiac adaptations.
- Changes seen on the electrocardiogram and imaging of the athlete's heart may closely resemble hypertrophic cardiomyopathy.
- The extent of cardiac remodelling may vary considerably according to each individual athlete's demographic profile.
- Differentiation between health and pathology requires careful interpretation of history, clinical examination and cardiac investigations.
- Pre-participation cardiovascular screening with 12-lead electrocardiogram, history and clinical examination is recommended in all young (≤35-year-old) competitive athletes.