The athlete's heart

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In this article, the authors highlight the spectrum, magnitude and determinants of the athlete's heart and provide a practical guide for differentiating physiological left ventricular hypertrophy from hypertrophic cardiomyopathy.

Regular physical exercise is associated with a five-fold increase in cardiac output to facilitate oxygen delivery to exercising muscles. Cardiac output is a product of both stroke volume and heart rate. The ability of the heart to generate a large stroke volume is determined by a benign reversible cardiac remodelling process comprising ventricular hypertrophy, increase in cardiac chamber size and enhanced diastolic ventricular filling. Athletes also exhibit a slower resting heart rate from the functional effects of a large stroke volume with an appropriate increase in the electrical activity to generate a large and sustained increase in cardiac output during exercise.

An increased cardiac chamber size and a slow heart rate are commonly acknowledged features of the 'athlete's heart' (Figure 1). Occasionally the manifestations of 'athlete's heart' overlap with phenotypic expressions of morphologically mild cardiomyopathies. The distinction between athlete's heart and cardiomyopathy is crucial when one considers that the cardiomyopathies are the main causes of sudden cardiac death (SCD) in young athletes.

THE ATHLETE'S ELECTROCARDIOGRAM

The electrocardiogram in athletes exhibits features reflecting a high resting vagal tone and increased cardiac size (Figure 2). Sinus bradycardia, sinus arrhythmia, first- and second-degree heart block and voltage criteria for left ventricular hypertrophy (LVH) are common. Repolarisation anomalies...
comprising of j-point, ST-segment elevation and high-amplitude T-waves are also common, particularly in athletes of African/Afro-Caribbean (black) origin (Box 1). In contrast, ST-segment depression is rare and suggestive of cardiac pathology. T-wave inversion in leads V1–V4 is acceptable in all athletes aged <15 years and all black athletes (Box 2). The persistence of T-wave inversion beyond V1 in Caucasians after 16 years of age is rare and should raise suspicion of underlying arrhythmogenic right ventricular cardiomyopathy, a recognised cause of SCD in young athletes. T-wave inversion in leads III and aVf in isolation is of little significance. T-wave inversion in the lateral leads always warrants further investigation to exclude hypertrophic cardiomyopathy (HCM), which is the commonest cause of SCD in young athletes worldwide.

Holter monitoring demonstrates sinus pauses and Mobitz type I second-degree block in up to 25 per cent of highly trained endurance athletes. Ventricular extrasystoles are common but rarely exceed 2000 over a 24-hour period.

THE ATHLETE’S ECHOCARDIOGRAM
Echocardiographic meta-analyses in male athletes reveal a 15–20 per cent increase in left ventricular wall thickness and a 10 per cent increase in left ventricular cavity size compared with sedentary controls of similar age. In absolute terms most Caucasian (98 per cent) athletes exhibit a left ventricular wall thickness within normal limits (<12mm). In contrast, up to 50 per cent of male athletes, particularly those competing in endurance sports, reveal a dilated left ventricular cavity (>55mm).

Cardiac dimensions are influenced by several demographic factors (Figure 3); in general adult males participating in endurance/high dynamic intensity sporting disciplines exhibit the greatest structural changes. Black athletes more commonly demonstrate increases in left ventricular wall thickness compared with other ethnicities; up to 13 per cent of black athletes reveal a left ventricular wall thickness >12mm compared with just 2 per cent of Caucasian athletes.

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

Figure 2. (a) Example of a black athlete’s ECG showing sinus bradycardia, voltage criteria for left ventricular hypertrophy (LVH), ST-segment elevation in leads V1–V5 and deep T-wave inversion in V1–V4. (b) Example of a Caucasian athlete’s ECG showing sinus bradycardia, LVH and high-amplitude T-waves
Sinus bradycardia
Brugada-like early repolarisation
Inverted T-wave V1–V4
Left ventricular hypertrophy
Ventricular pre-excitation
Complete left or right bundle branch block
Left atrial enlargement
Juvenile ECG pattern
J-point ST-segment elevation
Large QRS complexes
Isolated QRS voltage criteria for left ventricular hypertrophy
Left axis deviation
Large left/right ventricle cavities
First-degree atrioventricular block
T-wave inversion
Large QRS complexes
Notched T-waves
Left ventricular hypertrophy
ST-segment depression
Marked repolarisation changes
Pathological Q-waves

UPPER LIMITS OF CARDIAC DIMENSIONS IN ATHLETES
Studies in large groups of Olympian athletes in Italy and the UK have revealed an upper limit of left ventricular wall thickness in male athletes of 16mm. Caucasian females do not reveal left ventricular wall thickness >12mm. In contrast, up to 3 per cent of black female athletes show a wall thickness >12mm but never exceeding 13mm.

Athletes exhibit a broad distribution of left ventricular cavity dimensions depending on age, size and type of sport. Almost 25 per cent of all males and 1 per cent of females reveal a left ventricular cavity size >60mm. The upper limit of left ventricular cavity size in males and females is 70mm and 66mm respectively.

There are fewer data relating to right ventricular adaptation in athletes given the inherent limitations of imaging the chamber with echocardiography. Unlike the left ventricle, which has an ellipsoid shape, the right ventricle has a crescent-like shape; therefore dimensions vary according to the portion of the right ventricle that is measured. Emerging data from cardiac magnetic resonance imaging (MRI) have demonstrated balanced chamber enlargement. Upper limits for cardiac MRI-derived right ventricular end-diastolic cavity size are 58mm in male and 52mm in female elite athletes. Our own experience of echocardiographic assessment of the right ventricle in male football players reveals that over 40 per cent exhibit right ventricular dimensions exceeding predicted upper limits.

ECG PATTERNS SUGGESTIVE OF CARDIAC DISEASE IN ATHLETES
- T-wave inversion
- ST-segment depression
- Pathological Q-waves
- Left atrial enlargement
- Left axis deviation
- Ventricular pre-excitation
- Complete left or right bundle branch block
- Brugada-like early repolarisation

COMMON ECG PATTERNS IN ATHLETES
- Sinus bradycardia
- First-degree atrioventricular block
- J-point ST-segment elevation
- Isolated QRS voltage criteria for left ventricular hypertrophy

CARDIAC FUNCTION
There are usually no differences in resting left ventricular systolic function between athletes and non-athletic individuals. Endurance athletes occasionally demonstrate lower fractional shortening at rest, which normalises with brief exercise. Invasive studies in athletes during exercise have demonstrated enhanced diastolic filling and augmentation of stroke volume even at periods of rapid heart rate. Most athletes demonstrate a high peak oxygen consumption ranging from 55ml/kg per minute to over 70ml/kg per minute.

SUDDEN DEATH IN YOUNG ATHLETES
The benefits of exercise on the cardiovascular system are well established and exercising individuals live for an average of six years longer than sedentary persons. Rarely, a young athlete may die during or shortly after exercise. The incidence of SCD in young athletes aged 12–35 years old is estimated between 1 in 50 000 to 1 in 200 000. Hypertrophic cardiomyopathy is the commonest cause of SCD worldwide, followed by coronary artery anomalies. Among Italian athletes, the greatest number of fatalities was caused by arrhythmogenic right ventricular cardiomyopathy. Acquired conditions implicated in the death of young athletes include acute myocarditis, commotio cordis and illicit drug use. Atherosclerotic coronary artery disease is the commonest cause of SCD in older athletes.

BOX 1. Classification of the athlete's electrocardiogram

COMMON ECG PATTERNS IN ATHLETES
- Sinus bradycardia
- First-degree atrioventricular block
- J-point ST-segment elevation
- Isolated QRS voltage criteria for left ventricular hypertrophy

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BOX 2. Impact of age, ethnicity and sport on the athlete's ECG

ADOLESCENT ATHLETES*
- Juvenile ECG pattern
- Notched T-waves
- Large QRS complexes

ENDURANCE ATHLETES*
- Marked sinus bradycardia
- Large QRS complexes
- Left ventricular hypertrophy
- Large left/right ventricle cavities

BLACK ATHLETES*
- Marked repolarisation changes
- Inverted T-wave V1–V4
- Left ventricular hypertrophy

*Females have a lower prevalence of repolarisation changes and large cardiac dimensions.

PRE-PARTICIPATION SCREENING OF ATHLETES
A mandatory state-sponsored pre-participation screening (PPS) programme in Italy has proved effective in identifying athletes with cardiomyopathies and electrical disorders of the heart. Such individuals are disqualified from sport to minimise the risk of SCD. The screening model is unable to detect anomalous coronary arteries or premature coronary artery disease. A 25-year follow-up study in Italy has revealed a significant (90 per cent) reduction in SCD since the implementation of screening from 36/100 000 person-years to 0.4/100 000 person-years. However, PPS remains a controversial issue in most Western countries because of issues relating to cost-effectiveness, false-positive and negative results, the financial and logistical implications of developing an adequate infrastructure and manpower to implement a similar system.

In the UK, several sporting organisations, including the Lawn Tennis Association,
Football Association, Premier Rugby League and the English Institute of Sport, advocate PPS. The charitable organisation Cardiac Risk in the Young has facilitated the screening of more than 20,000 athletes since 1997 and report a 0.3 per cent identification rate for potentially sinister disorder at the expense of a false-positive rate of 3 per cent.

THE DIAGNOSTIC CHALLENGE:
ATHLETE’S HEART OR HYPERTROPHIC CARDIOMYOPATHY?
Physical training is occasionally associated with electrical and structural cardiac changes that overlap with cardiac disease. One of the most important diagnostic dilemmas encountered is the differentiation of physiological LVH from morphological mild HCM when confronted with an athlete with a left ventricular wall thickness of 13–16mm. In most instances the differentiation between the two entities is possible using clinical history, athlete demographics, ECG, echocardiography and cardiopulmonary exercise testing (Figure 4). The presence of cardiac symptoms and/or a family history of HCM in an athlete with LVH suggests HCM. The athlete’s demographic profile provides a clue to the differentiation between the two entities; for example, LVH is most unusual in any adolescent or female Caucasian athlete and would be highly suggestive of HCM. A small cavity, dynamic left ventricular outflow obstruction and abnormal indices of diastolic function on echocardiography favour HCM. Cardiac MRI has the advantage of identifying myocardial fibrosis, which would be consistent with pathological LVH.

The ECG can prove extremely helpful in making the differentiation between athlete’s heart and HCM. Whereas both conditions are associated with voltage criterion for LVH, individuals with HCM also exhibit additional abnormalities including ST-segment depression, T-wave inversion, pathological Q-waves and left bundle branch block.

A high peak oxygen consumption >50ml/kg per minute or >120 per cent predicted for age during cardiopulmonary exercise testing is a feature of athlete’s heart. Individuals with HCM have impaired myocardial relaxation and consequently fail to augment a stroke volume, which is associated with a low peak oxygen consumption. Detraining an athlete is not an easy task as it risks fitness and team selection, but the resolution of LVH after a six-week period of cessation of exercise is suggestive of athlete’s heart.

Recent advances in methods for analysis of genetic mutations may permit the use of genotyping for resolving the diagnostic dilemma in cases where doubt persists in spite of comprehensive clinical

Figure 3. Key determinants of left ventricular hypertrophy in athletes

Figure 4. Clinical criteria for differentiating athlete’s heart from hypertrophic cardiomyopathy in athletes with left ventricular hypertrophy (LVH) 13–16mm

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evaluation. Unfortunately such tests can prove timely and a negative gene test does not necessarily exclude HCM with certainty.

CONCLUSION
The athlete’s heart is characterised by increased cardiac dimensions that occasionally overlap with cardiomyopathies. A systematic clinical approach enables differentiation of athlete’s heart from HCM in most cases.

Declaration of interests: none declared.

REFERENCES