The athlete’s heart

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Athletes undergoing intensive training can exhibit cardiac changes that overlap with cardiomyopathy. Here the authors review cardiac changes that can occur in athletes and how to distinguish between normal adaptations and those that may signal pathology that can result in sudden death.

Participation in regular systematic physical activity is associated with a favourable atherosclerotic risk profile and has several other beneficial effects for the cardiovascular system. Exercise reduces obesity, improves insulin sensitivity and reduces the risk of metabolic syndrome and type 2 diabetes mellitus. Exercise also has a positive impact on blood pressure and lipid profile.1,2,3 Physically active individuals show a 50% reduction in the risk of an adverse event from coronary artery disease, in comparison with sedentary individuals.4

In addition to reducing the burden of atherosclerotic risk factors for cardiovascular disease, regular exercise has several other beneficial effects that increase cardiorespiratory fitness and have a positive impact on the cardiovascular system (see Figure 1). There is growing evidence that exercise prevents ageing of the cardiovascular system to such an extent that physically active individuals increase their life expectancy by up to seven years compared with their sedentary counterparts.5

In order to achieve some of these health benefits, the World Health Organization (WHO) instructs adults aged 18–64 years to undertake at least 150 minutes of moderate exercise, or 75 minutes of vigorous exercise, each week. Moderate exercise is defined by signs of faster breathing (but no shortness of breath) light sweating after 10 minutes of exercise, and maintenance of the ability to hold a conversation (but not sing). Prospective longitudinal studies show that maximum benefits are attained by those who exercise 4–5 times the current WHO recommendations.6

Cardiac physiological adaptations in response to athletic training
Athletes engaging in intensive exercise perform 10 to 20 times the current WHO exercise recommendations. Intensive exercise is associated with a five- to six-fold increase in cardiac output, which is achieved by a combination of increased venous return to the ventricles, rapid ventricular filling, augmentation of stroke volume, increased heart rate and a marked reduction in systemic vascular resistance. Maximal heart rate is limited by age, so regular bouts of exercise that require a prolonged and sustained increase in cardiac output necessitate a physiological increase in cardiac dimensions. Furthermore, athletic individuals demonstrate enhanced ventricular filling and can augment stroke volume even at rapid heart rates when diastole is short.

During resting conditions, there is increased vagal tone on the heart, which is associated with bradycardia. The constellation of structural, functional and electrical changes is termed the ‘athlete’s heart’. There are also peripheral adaptations in skeletal muscle; including an increase in

Figure 1. The positive impact of regular exercise on the cardiovascular system

- ↓ Body mass index
- ↑ Insulin sensitivity
- ↓ Metabolic syndrome
- ↓ Type II diabetes mellitus
- ↑ HDL
- ↓ LDL
- ↓ Triglycerides
- ↓ heart rate
- ↑ ion channel expression
- ↑ electrical stability
- ↑ capillary conductance
- ↑ endothelial function
- ↓ oxidative stress
- ↓ thrombogenicity
- ↑ cardiac size
- ↑ cardiac filling in diastole
- ↑ stroke volume
- ↓ aortic stiffness
- ↓ systemic vascular resistance
- ↑ left ventricular compliance
- ↑ plaque stability
- ↑ myokine release
- ↓ C-reactive protein
- ↑ IL-6 from muscle
- ↓ oxidative stress
- ↓ thrombogenicity
- ↓ aortic stiffness
- ↑ systemic vascular resistance
- ↑ left ventricular compliance
- ↑ plaque stability
- ↑ cardiac size
- ↑ cardiac filling in diastole
- ↑ stroke volume
- ↓ oxidative stress
- ↓ thrombogenicity
- ↓ aortic stiffness
- ↑ systemic vascular resistance
- ↑ left ventricular compliance
- ↑ plaque stability
- ↑ cardiac size
- ↑ cardiac filling in diastole
- ↑ stroke volume

Cardiovascular medicine
Abnormal ECG patterns
There are several ECG patterns that should raise suspicion of cardiac disease (see Table 1). In summary, ST segment depression, T wave inversion in the lateral leads, pathological Q waves and left bundle branch block always warrant comprehensive investigation for cardiomyopathies, which are amongst the leading causes of death in young athletes.

T wave inversion may be a normal variant in some leads. For example, T wave inversion in leads V1–V3 may represent the juvenile ECG pattern in adolescent athletes under 16 years old. T wave inversion confined to leads V1 and V2 may also constitute normal variants in adult athletes. Male athletes of African or African-Caribbean origin, may show deep T wave inversion in leads V1–4, accompanied by J point elevation and a convex ST segment.

Sudden death in sport
Exercise may trigger fatal arrhythmias in vulnerable individuals with quiescent cardiac disease. Fortunately, the incidence of such events is rare and affects 1 in 50 000 athletes. Among young athletes (<35 years), the inherited cardiomyopathies, such as hypertrophic cardiomyopathy and arrhythmogenic right ventricular cardiomyopathy and ion channel disorders, such as long QT syndrome or catecholaminergic polymorphic ventricular tachycardia, are the most common cause of death. Atherosclerotic coronary artery disease accounts for 80–90% of all deaths in older athletes.

Most deaths occur in males, with a 9:1 male/female ratio in elite sport and 20:1 ratio during recreational exercise. There are several screening initiatives to identify athletes who may be at risk, but these are confined to the elite young athletes. Current data suggest that bystander cardiopulmonary resuscitation and early application of an automated external defibrillator are the most pragmatic methods of preventing sudden cardiac death in the exercising population as a whole, and are associated with a fourfold increase in survival.

When the athlete’s heart resembles cardiomyopathy
A proportion of highly trained male athletes may show cardiac dimensions that overlap with cardiomyopathy. Endurance athletes often show an enlarged ventricle. The ventricles in these athletes may appear sluggish at rest because they do not need to contract much to eject a basal cardiac output of five litres per minute, hence there is overlap with dilated cardiomyopathy or arrhythmogenic right ventricular cardiomyopathy. Some
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Key points

- Regular exercise is associated with multiple cardiovascular benefits
- Participation in frequent intensive training leads to a constellation of structural, functional and electrical cardiac adaptations, frequently termed ‘the athlete’s heart’
- Sudden cardiac death in sport is rare and usually affects individuals with underlying cardiovascular abnormalities

Table 1. Definitive abnormal electrocardiogram patterns in athletes.

<table>
<thead>
<tr>
<th>Pattern Description</th>
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<tbody>
<tr>
<td>T wave inversion (leads I, aVL, V5 and V6 and contiguous inferior leads * in all athletes and beyond V2 in adult white athletes)</td>
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<tr>
<td>Complete LBBB</td>
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<td>ST segment depression</td>
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<tr>
<td>Pathological Q waves</td>
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<tr>
<td>Long QT interval &gt; 470 males and &gt; 480 females</td>
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<tr>
<td>Type I Brugada ECG pattern</td>
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<tr>
<td>Wolff-Parkinson-White ECG Pattern</td>
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<tr>
<td>Atrial tachyarrhythmias</td>
</tr>
<tr>
<td>Ventricular tachyarrhythmias</td>
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<tr>
<td>Two or more PVCs per 10 seconds</td>
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<tr>
<td>Mobitz Type II 2nd degree AV block</td>
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<tr>
<td>3rd degree AV block</td>
</tr>
</tbody>
</table>

LBBB = left bundle branch block; PVC = premature ventricular complexes *leads II, III and aVF

Table 1. Definitive abnormal electrocardiogram patterns in athletes.

black male athletes may show a left ventricular wall thickness between 13–15mm, which overlaps with a mild variant of hypertrophic cardiomyopathy. In such instances, the distinction between cardiac physiology and cardiomyopathy is vital, because an erroneous diagnosis may have serious consequences for the athlete – from unfair disqualification to sudden death following false reassurance.

Almost all cases can be resolved through a careful history (including family history), inspection of the ECG, an exercise test, cardiovascular MRI and prolonged ECG monitoring. The ECG is particularly important because the presence of some of the abnormal ECG patterns outlined in Table 1 – such as left bundle branch block, pathological Q waves, T wave inversion in the lateral leads, ST segment depression and ventricular arrhythmias – are indicative of cardiac pathology. The aim of the additional tests is to investigate the broader phenotypic features of cardiomyopathy. Specifically, exercise testing and prolonged ECG monitoring aim to detect ventricular arrhythmias, while the cardiovascular MRI scan is performed to check for myocardial scar or subtle wall motion abnormalities that may not be detected with echocardiography.

Exercise echocardiography is particularly useful for differentiating dilated cardiomyopathy. Athletes show a significant increase in left ventricular ejection fraction with increasing exercise, while individuals with dilated cardiomyopathy will show very limited or no increase in left ventricular ejection fraction.

Conclusion
The benefits of exercise on the cardiovascular system are well established. Individuals who regularly exercise intensively show several structural and functional adaptations, which facilitate the generation of a prolonged and sustained increase in cardiac output. The magnitude of these adaptations is greatest in large male endurance sportsmen.

The differentiation between the largest or the most hypertrophied ventricles and cardiomyopathies is crucial, since these pathological entities are leading causes of sudden death in young athletes.

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References