**The Athlete’s Heart**

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The benefits of exercise on the cardiovascular system

Participation in regular systematic physical activity is associated with a favourable atherosclerotic risk profile and has several other beneficial effects on 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 profile1,2,3. Physically active individuals show a 50% reduction in the risk of an adverse event from coronary artery disease, in comparison to sedentary individuals4. 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 (Figure 1). There is growing evidence that exercise prevents ageing of the cardiovascular system, such that physically active individuals increase their life expectancy by up to 7 years compared with their sedentary counterparts5.

In order achieve some of these health benefits, the World Health Organisation (WHO) instructs adults of 18-64 years of age to carry out 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.



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

Cardiac physiological adaptations in response to athletic training

Athletes engaging in intensive exercise perform between 10 to 20 times the current WHO exercise recommendations. Intensive exercise is associated with a 5-6 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, therefore 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” (Figure 2). There are also peripheral adaptations in skeletal muscle; including an increase in capillary density around the muscle fibres and the concentration of mitochondria in the muscle cells, which facilitate rapid extraction of oxygen from the blood and cause a widening of the arterio-venous oxygen difference2. The net effect is an increase in peak oxygen consumption.



Figure 2: Physiology of cardiovascular response to exercise.

*Structural cardiac changes*

Increases in cardiac preload and afterload from repetitive exercise training is associated with bilateral and symmetrical enlargement of all cardiac chambers. The size of both ventricles increases by 10-15%, left ventricular wall thickness increases by 10-20% and left ventricular mass increases by 45%. Such adaptations are governed by several demographic factors and type of sport. In general, black male athletes who engage in explosive sports such as sprinting, football and basketball, have the greatest increases in left ventricular wall thickness1,2,3 and large male athletes (all races) who engage in endurance sports have the largest ventricular cavity dimensions. In rare instances, these physiological adaptations overlap with those observed in patients with cardiomyopathy and can result in major clinical dilemma (see below). Female athletes and adolescent athletes below the age of 16 years old, rarely show cardiac dimensions that stimulate cardiac pathology.

The athlete’s ECG

The athlete’s ECG generally reflects increased vagal tone and/or increased cardiac chamber size. Common electrical changes include sinus bradycardia (70%), first degree atrioventricular block (25%), early repolarisation (J-point and ST-segment elevation) (25-40%) and high voltage T waves (20%). Voltage criterion for left ventricular hypertrophy (50%) and incomplete right bundle branch block (30%) are also common2,3 (Figure 3).

  
Figure 3: The athlete’s ECG showing sinus bradycardia, voltage criteria for left ventricular hypertrophy (LVH), incomplete right bundle branch block and early repolarisation (ER) changes.

*Abnormal ECG Patterns*

There are several ECG patterns which should raise suspicion of cardiac disease (Table 1)6. 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 aged below 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 Afro-Caribbean origin (black athletes), may show deep T-wave inversion in leads V1-4 that is also accompanied by J-point elevation and a convex ST segment7.

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| **T wave inversion**  (leads I, aVL, V5 and V6 and contiguous inferior leads\* in all athletes and beyond V2 in adult white athletes)  **Complete LBBB**  **ST segment depression**  **Pathological Q waves**  **Long QT interval** > 470 males and > 480 females  **Type I Brugada ECG pattern**  **Wolff-Parkinson-White ECG Pattern**  **Atrial tachyarrhythmias**  **Ventricular tachyarrhythmias**  **2 or more PVCs per 10 secs**  **Mobitz Type II 2nd degree AV block**  **3rd degree AV block** |

Table 1: Definitive abnormal ECG patterns in athletes.

Abbreviations: LBBB left bundle branch block; PVC premature ventricular complexes.

\*leads II, III and aVF

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 athletes8. Among young (< 35 years old) athletes, the inherited cardiomyopathies, such as hypertrophic cardiomyopathy and arrhythmogenic right ventricular cardiomyopathy and ion channel disorders, such as long QT syndrome or catecholeaminergic polymorphic ventricular tachycardia, are the commonest 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 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 most elite young athletes. Current data suggests that by-stander cardiopulmonary resuscitation and early application of an automated external defibrillator are the most pragmatic methods of preventing SCD in the exercising population as a whole and are associated with a 4-fold increase in survival9.

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 5 litres per minute and hence, there is overlap with dilated cardiomyopathy or arrhythmogenic right ventricular cardiomyopathy. Some black male athletes may show a left ventricular wall thickness between 13-15 mm, which overlaps with a mild variant of hypertrophic cardiomyopathy10. In such instances, the distinction between cardiac physiology and cardiomyopathy is vital, because an erroneous diagnosis may have serious consequences for the athletes; which range 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 magnetic resonance imaging 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 magnetic resonance imaging 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, whereas 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 exercise intensively on a regular basis 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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| **Key Points** |
| * Regular exercise is associated with multiple benefits on the cardiovascular system. * Participation in frequent intensive training leads to a constellation of structural, functional and electrical cardiac adaptations, frequently termed “the athlete’s heart”. * Large male endurance athletes show the largest cardiac dimensions whereas black male athletes show the most profound electrical changes. * On occasion, the electrical and structural changes observed in may overlap with cardiomyopathy. * Sudden cardiac death in sport is rare and usually affects individuals with underlying cardiovascular abnormalities. |