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Playing it safe: exercise and cardiovascular health

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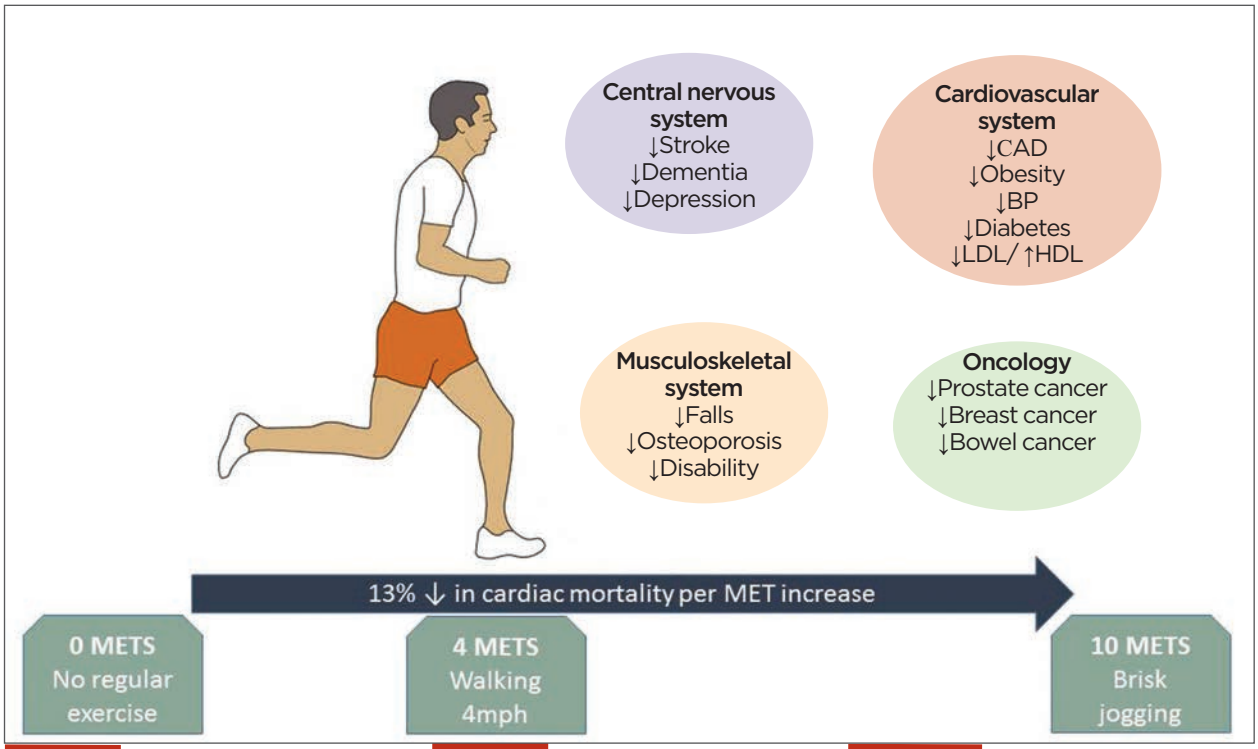


FIGURE 1
The benefits of regular exercise (MET = metabolic equivalent of task)

What are the benefits of regular exercise?

THE PROMOTION OF EXERCISE AS A POSITIVE AND POWERFUL HEALTH INTERVENTION HAS NEVER been more important given the global epidemic of disease states related to a sedentary lifestyle such as obesity and diabetes.

However, intensive exercise may be a trigger for sudden death in individuals harbouring quiescent cardiovascular disease. Indeed, hereditary and congenital abnormalities of the heart are the most common cause of nontraumatic death during sport in young athletes.

The issue of screening athletes for cardiac disease is an important focus of the scientific community but the actual protocol is still debated.

This article will discuss the relationship between exercise and the cardiovascular system.

What are the potential risks?

BENEFITS OF EXERCISE

Participation in regular physical activity is established as a principal lifestyle choice to optimise cardiovascular health, see figure 1, above. Regular physical activity controls acquired cardiovascular risk factors such as obesity, diabetes mellitus, hypertension and hyperlipidaemia.[1] Exercise is generally associated with a 50% reduction in adverse events from coronary artery disease (CAD).[2,3]

'Exercise is generally associated with a 50% reduction in adverse events from coronary artery disease'

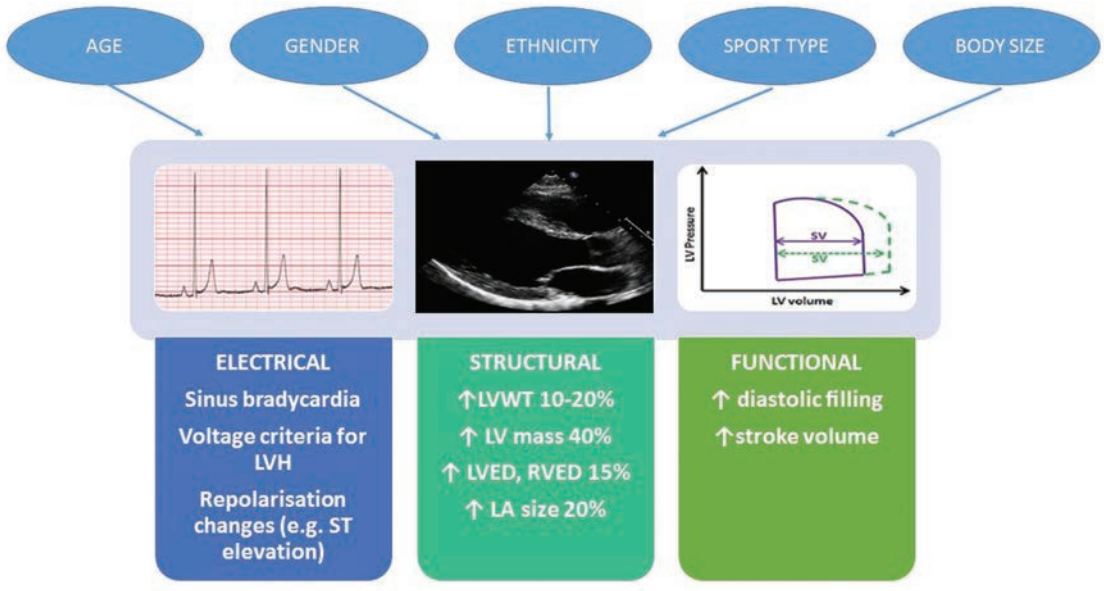
Which individuals should be screened?

The benefits of exercise extend well beyond the cardiovascular system. Recent evidence suggests that exercise prevents cell senescence, and active individuals are at lower risk of developing certain malignancies including cancer of the prostate and the colon, osteoporosis, depression and dementia.[4,5] Indeed, individuals who exercise regularly extend life expectancy by three to seven years.[6,7]

Exercise should be championed as one of the most effective therapeutic interventions a doctor can prescribe. Unlike most interventions, it is free, can be performed at any time and is generally devoid of side-effects.

The British Association of Sport and Exercise Sciences recommends that healthy individuals should engage in 150 minutes of moderate-intensity aerobic exercise per week.[8] Recent »

FIGURE 2
The cardiovascular adaptations to exercise – the athlete's heart



LA = left atrium, LV = left ventricle, LVED = left ventricle end diastolic diameter, LVH = left ventricular hypertrophy, LVWT = left ventricular wall thickness, RVED = right ventricle end diastolic diameter

studies have demonstrated that even lower volumes of exercise below these recommendations confer health benefits, which is highly relevant to individuals with established cardiac disease including heart failure.[9,10]

THE ATHLETE'S HEART

Individuals who engage in at least four hours of intensive exercise per week develop a constellation of physiological alterations in autonomic tone, cardiac structure and cardiac function,[11] see figure 2, above. Sinus bradycardia, sinus arrhythmia and repolarisation anomalies including ST segment elevation on the ECG are common in athletes and occur as a result of increased vagal tone, see figure 3A, below.

Young males also frequently exhibit voltage criteria for left ventricular hypertrophy (LVH) but without other features suggestive of pathological LVH such as ST segment depression and T-wave inversion in the lateral leads.

Black athletes of African/Afro-Caribbean origin reveal profound repolarisation changes on the ECG. Nearly a quarter of healthy male black athletes exhibit T-wave inversions, but these are considered as normal variants when they are confined to leads V1-V4, whereas T-wave inversion in the infero-lateral leads should raise suspicion of cardiomyopathy in all athletes,[12] see figure 3B, below.

Structural adaptations include a 10-20% increase in ventricular wall thickness and a 10-15% increase in ventricular and atrial dimensions.[13,14,15,16,17]

'Active individuals are at lower risk of developing cancer of the prostate and colon, osteoporosis, depression and dementia'

The spectrum of changes is governed by age, gender and ethnicity and sporting discipline. In general, male endurance athletes display the largest ventricular dimensions and male

FIGURE 3A
ECG of a 30-year-old Caucasian rower demonstrating sinus bradycardia, Sokolow-Lyon voltage criteria for left ventricular hypertrophy (‡), U-waves (arrow) and early repolarisation (*). These changes are common in highly trained athletes and do not require further investigation

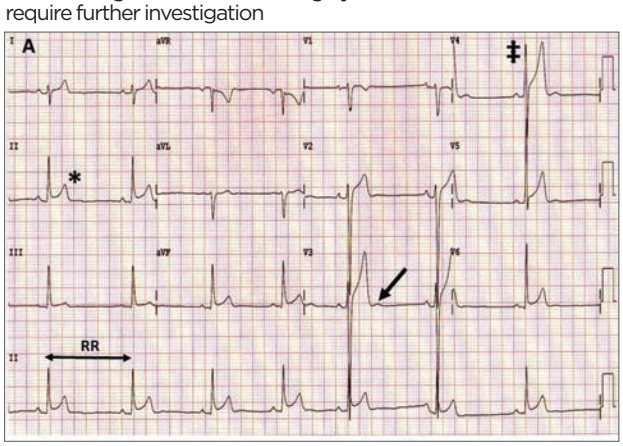


FIGURE 3B
ECG of an 18-year-old black athlete demonstrating deep T-wave inversion in leads V1-4 preceded by convex ST segment elevation (arrows) and Sokolow-Lyon voltage criteria for left ventricular hypertrophy. This ECG pattern is common in black athletes and does not warrant further investigation

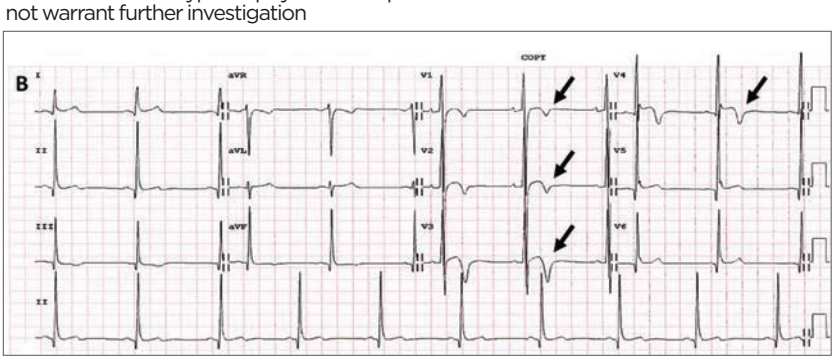
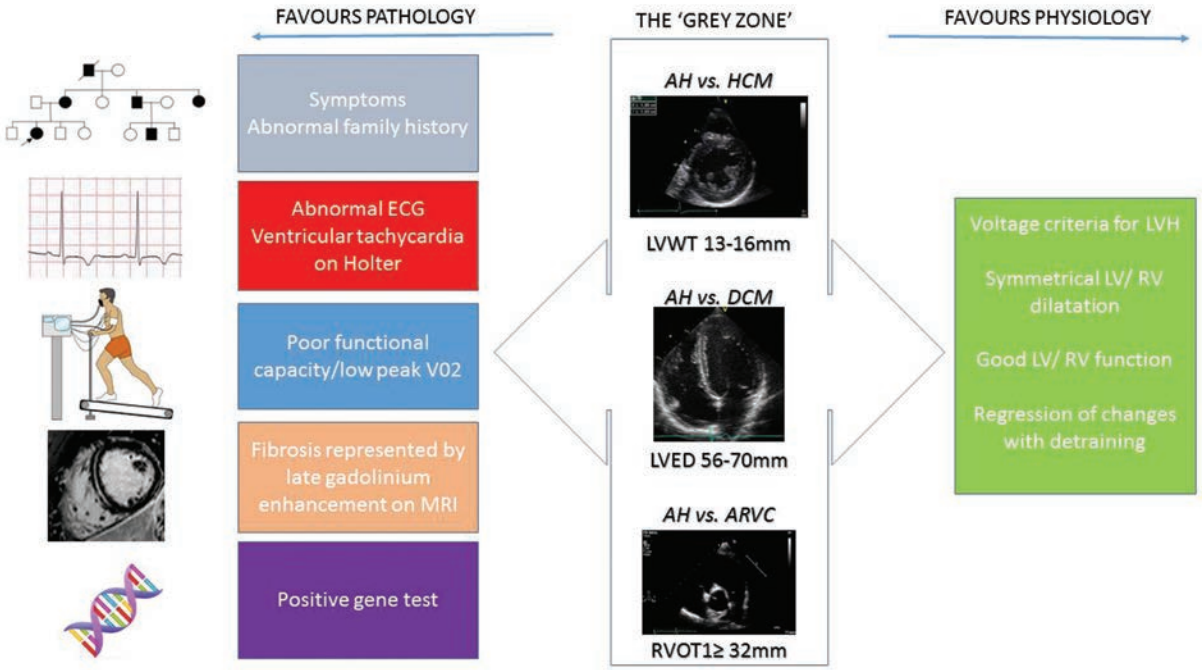


FIGURE 4
Algorithm to differentiate physiological structural changes of exercise from those that represent pathology



AH = athlete's heart, ARVC = arrhythmogenic right ventricular cardiomyopathy, DCM = dilated cardiomyopathy, HCM = hypertrophic cardiomyopathy, LV = left ventricle, LVED = left ventricle end diastolic diameter, LVH = left ventricular hypertrophy, LVWT= left ventricular wall thickness, RV = right ventricle, RVOT1 = right ventricular outflow tract, V02 = maximal oxygen consumption

'Exercise should be championed as one of the most effective therapeutic interventions a doctor can prescribe'

athletes of Afro-Caribbean origin exhibit a greater degree of LVH compared with other athletes.[13,16] Such adaptations are benign and regress on cessation of training.

On occasion, the magnitude of physiological increases in cardiac dimensions overlaps with those characteristic of the cardiomyopathies which are the leading cause of exercise-related sudden cardiac death (SCD) in young athletes.

In such cases, multimodal algorithms including advanced cardiac imaging, exercise tests, reassessment after a period of detraining or even family evaluation and genetic testing are useful in facilitating the differentiation between cardiac pathology and cardiac physiology.

It is prudent that these assessments are conducted in an expert centre given the potential consequences of an erroneous diagnosis, see figure 4, above.

SUDDEN CARDIAC DEATH IN ATHLETES

SCD in athletes under the age of 35 is rare with estimates ranging from 1 in 50,000 to 1 in 200,000.[18,19] The most common cause of death in young athletes worldwide is hypertrophic cardiomyopathy (HCM) which is implicated in over one-third of all SCDs in young American athletes.[20] Arrhythmogenic right ventricular cardiomyopathy (ARVC) is the most common cause of death in Italy.[21] In a quarter of cases, the heart is structurally normal and when the toxicology screen is negative, such cases are often attributed to primary cardiac ion channel disorders such as the long QT syndrome, Brugada syndrome, catecholaminergic polymorphic ventricular tachycardia, or the congenital accessory pathways.[22]

Almost 90% of deaths occur in males and 40% of athletes are aged under 18.[20] Data from the USA suggest that black athletes may be more prone to SCD, with a reported incidence of 5.6 per 100,000.[23,20] Most deaths occur in explosive sports of a start-stop nature such as football and basketball.[21,24]

'The most common cause of death in young athletes is hypertrophic cardiomyopathy'

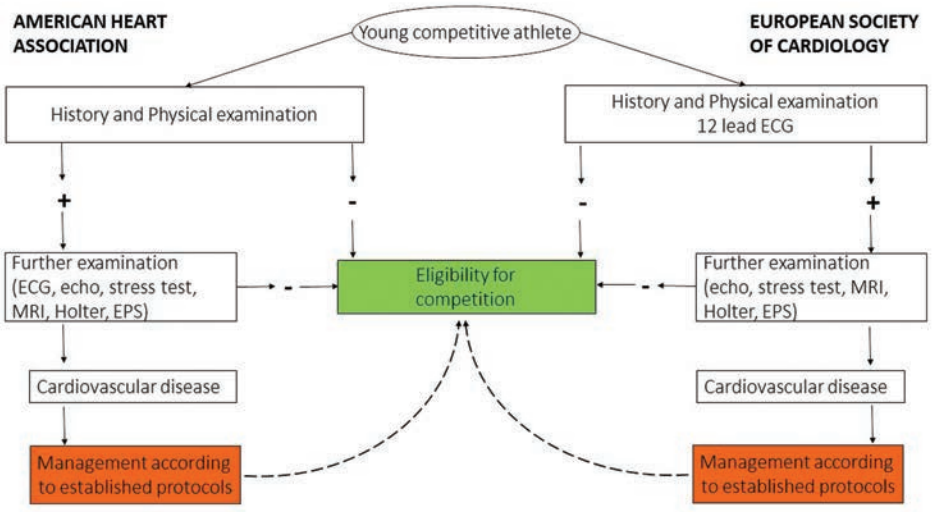
Research related to SCD has largely focused on young professional athletes. However, the overwhelming majority of exercise-related SCDs occur in middle-aged recreational athletes.[25,26] More than 90% of such deaths occur in males and more than 90% are caused by atherosclerotic CAD. [25,26,27]

Prevention

Although the incidence of SCD during sport is relatively low, such events are magnified through the media and impact on society. Identification of athletes at risk of SCD is an important focus of the medical community on the premise that the majority of responsible diseases can be detected during life, and acceptable interventions such as lifestyle, pharmacological therapy, radiofrequency ablation of accessory pathways or implantation of a cardioverter defibrillator are available to reduce the risk of SCD.

Pre-participation screening (PPS) is carried out in the highest echelons of sport in the UK including the Football Association, the English Institute of Sport, and Rugby Football Union, but grassroots sport and secondary »

FIGURE 5
Recommendations for pre-participation screening of young athletes



echo = echocardiogram, EPS = electrophysiology study

schools are neglected. The most persuasive evidence that early detection of disease saves lives is derived from a large prospective Italian study, which reported an 89% reduction in SCD in athletes screened by ECG over a 25-year period.[18]

The American Heart Association (AHA) and the European Society of Cardiology (ESC) advocate PPS of young athletes, but differ in their protocols,[28,29] see figure 5, above. The AHA advocates a health questionnaire pertaining to cardiac symptoms and relevant family history, and physical examination. The ESC model advocates the inclusion of the 12-lead ECG. The AHA model is cheap and pragmatic but exhibits poor sensitivity because more than 80% of athletes are asymptomatic prior to death.[22]

The ECG has the ability to detect congenital accessory pathways and ion channelopathies, and is frequently abnormal in individuals with cardiomyopathy, see figures 6A and B, left. Indeed, the ECG is abnormal in more than 90% and 75% of cases of HCM and ARVC respectively,[30] see figure 6B, left. The ECG cannot detect all disorders predisposing to SCD in athletes and is unable to identify athletes with premature CAD or congenital coronary anomalies. Furthermore, ECG interpretation by inexperienced physicians is associated with a high false-positive rate.[18,31,32,33,34] More modern evidence-based guidelines for the interpretation of the athlete's ECG have reduced the false-positive rate to 6% without compromising sensitivity to detect serious cardiac disease.[31,34,35]

Screening with a 12-lead ECG in older athletes is of limited value given the overwhelming contribution of atherosclerotic CAD to SCD. In this group of athletes, risk assessment should include a questionnaire pertaining to symptoms of myocardial ischaemia, conventional risk factors for atherosclerosis and a prior cardiac history.[36]

Prior symptoms suggestive of myocardial ischaemia, and acquired risk factors for CAD are present in 36% and 50% of older victims of exercise-related sudden cardiac arrest respectively.[27] In this regard, routine ECG and exercise stress testing should be reserved for individuals with symptoms and

FIGURE 6A
ECG of an 18-year-old Caucasian tennis player demonstrating a prolonged QT interval (> 500 msec) with T-wave notching (arrow); these features are diagnostic of long QT syndrome

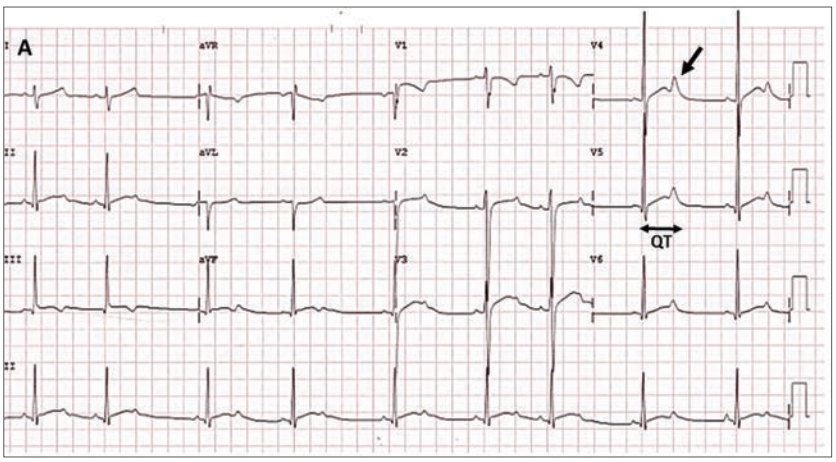
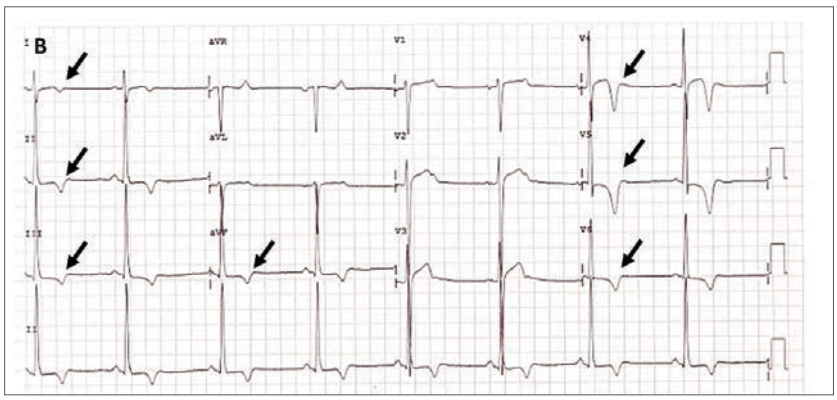


FIGURE 6B
ECG of a 16-year-old Caucasian footballer demonstrating deep T-wave inversion in the inferior and lateral leads (arrows). Further investigations led to a diagnosis of hypertrophic cardiomyopathy



key points

SELECTED BY

Dr Peter Saul
GP, Wrexham and Associate GP Dean for North Wales

Regular physical activity controls acquired cardiovascular risk factors such as obesity, diabetes mellitus, hypertension and hyperlipidaemia. Exercise is generally associated with a 50% reduction in adverse events from coronary artery disease (CAD). The benefits of exercise extend well beyond the cardiovascular system. Recent evidence suggests that exercise prevents cell senescence, and active individuals are at lower risk of developing certain malignancies including cancer of the prostate and the colon, osteoporosis, depression and dementia. Individuals who exercise regularly extend their life expectancy by three to seven years.

The British Association of Sport and Exercise Sciences recommends that healthy individuals should engage in 150 minutes of moderate-intensity aerobic exercise per week. Recent studies have demonstrated that even lower volumes of exercise below these recommendations confer health benefits, which is highly relevant to individuals with established cardiac disease including heart failure.

Sinus bradycardia, sinus arrhythmia and repolarisation anomalies including ST segment elevation on the ECG are common in athletes and occur as a result of increased vagal tone.

Sudden cardiac death in athletes under 35 is rare with estimates ranging from 1 in 50,000 to 1 in 200,000. Hereditary and congenital abnormalities of the heart are the most common cause of nontraumatic death during sport in young athletes. Data from the USA suggest that black athletes may be more prone to sudden cardiac death. In middle-aged recreational athletes more than 90% of sudden cardiac deaths occur in males and more than 90% are caused by atherosclerotic CAD.

The American Heart Association and the European Society of Cardiology advocate pre-participation screening of young athletes. The ECG has the ability to detect congenital accessory pathways and ion channelopathies, and is frequently abnormal in individuals with cardiomyopathy. Screening with a 12-lead ECG in older athletes is of limited value given the overwhelming contribution of atherosclerotic CAD to sudden cardiac death.

There is mounting evidence that there may be a threshold of physical activity beyond which there are no further health benefits, and some studies have shown that lifelong intensive exercise may even prove detrimental for some individuals with previously normal hearts. However, given that adverse cardiac event rates among lifelong marathon runners is low, the prognostic implications of these observations is speculative and sequential follow-up studies of large numbers of veteran athletes are required to confirm whether too much exercise is cardiotoxic in some athletes.

established risk factors for CAD but even this strategy has limitations.[36] The resting ECG will fail to detect quiescent CAD in the absence of prior myocardial infarction and an exercise stress test will only reveal individuals with marked luminal stenosis, but not those with minor soft plaques that are prone to fissuring.[36] Although computed tomography (CT) calcium scoring or CT coronary angiography will detect silent atherosclerotic plaques, the availability, cost and radiation exposure are currently limiting factors for this approach.

Given the exponential rise of middle-aged individuals participating in recreational sport, efficient emergency response planning including public awareness of bystander cardiopulmonary resuscitation and early deployment of automated external defibrillators may be more pragmatic. Two large studies in French and Dutch recreational athletes have shown that early bystander cardiopulmonary resuscitation was associated with a three-fold increase in survival from exercise-related sudden cardiac arrest.[25,26]

'The incidence of sudden cardiac death during sport is relatively low'

CARDIOVASCULAR RISK

Based on the numerous benefits of exercise, highly trained athletes are rightly perceived as the healthiest segment of society. These individuals exercise five to ten times above the usual exercise recommendations for cardiovascular health.

However, there is mounting evidence that there may be a threshold of physical activity beyond which there are no further health benefits, and some studies have shown that lifelong intensive exercise may even prove detrimental for some individuals with previously normal hearts.[37,38]

The most compelling evidence to support this hypothesis is the relationship between intensity of exercise and atrial fibrillation (AF), the most common arrhythmia in elderly patients. Although light-moderate physical activity is associated with a lower incidence of AF in older adults, males participating in long-term endurance exercise exhibit a five-fold increase in the prevalence of AF which suggests a U-shaped relationship between the intensity of exercise and AF.[39,40,41] Smaller studies have also demonstrated myocardial fibrosis, right ventricular dysfunction with ventricular arrhythmia and increased coronary calcium scores in middle-aged athletes performing lifelong exercise.[42,43,44,45,46]

'There was an 89% reduction in sudden cardiac death in athletes screened by ECG'

However, given that adverse cardiac event rates among lifelong marathon runners is low, the prognostic implications of these observations is speculative and sequential follow-up studies of large numbers of veteran athletes are required to confirm whether too much exercise is cardiotoxic in some athletes.

CONCLUSION

The health benefits and increased life expectancy associated with regular exercise are unsurpassable. Very occasionally, an athlete may die during exercise from an underlying cardiac disorder.

The early detection of potentially serious disease is an important issue but is hampered by the overlap between the manifestations of physiological cardiac adaptation and cardiac pathology.

State sponsored screening of athletes with cardiac symptoms, abnormal cardiovascular examination or a family history of cardiovascular disease is available.

However, the lack of financial resources, expertise and infrastructure does not currently support mass screening of all exercising individuals in the UK. The responsibility for the professional athlete should fall to their employers, whereas charitable organisations such as Cardiac Risk in the Young (www.c-r-y.org.uk) may support grassroots sports and secondary school athletes through subsidised screening.

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Useful information

Cardiac Risk in the Young
www.c-r-y.org.uk

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