GENERAL CARDIOLOGY

The athlete's heart

David Oakley
Sheffield, UK

Over the last decade the culture of physical exercise has changed. While elite athletes are training to ever more rigorous schedules, our middle aged sedentary population, no doubt seeing the writing on the wall, are hurrying to sign up for the fashionable, local fitness centres. When talking about athletes' hearts, we must distinguish between elite athletes, recreational sports men and women, non-athletes who wish to maintain cardiovascular fitness, and athletic patients with known cardiovascular disease. This article is mainly about athletes with serious sporting ambitions at club or higher level, who train most days for more than one hour. I will make it clear when I am referring to other types of athletes.

A regular training programme causes favourable changes in skeletal muscle performance (the realm of the sports physiologist) and two clear cut cardiovascular effects—namely, enlargement of the heart and a slow pulse rate at rest. These are the components of a characteristic clinical picture known as the "athlete's heart".[1]

Athletes, as a group, tend to be somewhat introspective about their health and will frequently consult doctors complaining of palpitations, dizziness, fatigue, chest pain, and undue dyspnoea. Physical examination may reveal some unusual signs, which will do little to reassure the doctor, already aware of widely publicised high profile cases of sudden death in association with sporting activity. The finding of an apparently abnormal ECG will cause further anxiety. A knowledge of the characteristic features of the athlete's heart is therefore important if the patient is to be advised wisely.[2]

Cardiac enlargement

Regular training causes the heart to enlarge. This is the result of a combination of left ventricular cavity enlargement (dilatation) and increased wall thickness (hypertrophy). The stimuli and processes involved are complex but appear to be akin to the normal growth of the heart in childhood.[3] It is widely held that resistance or isometric training (weight lifting, etc) stimulates hypertrophy with normal cavity dimensions (concentric), whereas aerobic, isotonic training (running, etc) stimulates hypertrophy and cavity dilatation (eccentric).[4] Athletes include both types of training in their schedules, so the distinction is blurred but it is probably a genuine phenomenon, as demonstrated by a recent meta-analysis of the available data.[w1] When the cardiac dimensions are corrected for surface area or lean body mass, it becomes more difficult to demonstrate concentric hypertrophy in resistance athletes. There is a large body of literature concerning the extent of these changes and what might be considered within the normal range for an athlete.[w2] The evidence is inconsistent, but a useful meta-analysis of the available data by Fagard suggests that a wall thickness more than 1.6 cm is very unusual in a healthy athlete, most of whom will be less than 1.3 cm.[5] Oarsmen, cyclists, and Nordic skiers appear to have the largest hearts, but the degrees of hypertrophy observed do not correlate well with the intensity of training or the performance of the athletes. Many Olympic champions have cardiac dimensions within the normal range, whereas a college athlete may exhibit pronounced hypertrophy. This observation implies that the cardiac response to training is not simply induced by the haemodynamic stresses during exercise, which is hardly surprising bearing in mind the much longer periods of rest during which, by the same argument, hypertrophy should regress.

Other influences including hormonal stimuli and genetic susceptibility are thought to play a part. Indeed, there is increasing evidence that the propensity to hypertrophy is partly genetically determined, with physical training acting as the trigger. Studies of identical twins support this thesis.[w3] Also, there is some indication that ACE gene (and probably other) polymorphism affect the hypertrophic response to training. For instance the DD genotype seems to predispose to greater hypertrophy in response to training and the I allele occurs more frequently in elite athletes and high altitude mountaineers.[6]

Whatever the mechanism, it is evident that hypertrophy is a genuine response to training. Athletes who train seasonally exhibit a seasonal variation in left ventricular dimensions[7]. Regression of hypertrophy, even after many years of training, can frequently be observed on de-training.

Cardiac dilatation and hypertrophy may be sufficiently pronounced to resemble a pathological state, but markers of left ventricular function, both systolic and diastolic, are consistently normal.[8] [w4] There is no convincing evidence that healthy subjects can train themselves into a pathological state manifest by fibrosis or fibre disarray.[w5]

Most published reports of cardiac enlargement refer to young male athletes but similar, though less notable, changes are reported in children, elderly subjects, and women.[w6–8]

Bradycardia

A resting bradycardia is characteristic of the trained athlete. In exceptional cases this may be less than 40 beats/min (bpm). While denervated hearts beat slower in response to training the bradycardia in athletes is mainly mediated by increased parasympathetic and reduced sympathetic input during the resting state.[9] [w9]

Correspondence to: Dr David Oakley, 54 Broomgrove Road, Sheffield S10 2NA, UK
oakley@ shef1.demon.co.uk

This may result in sinus pauses with junctional escape rhythms, first degree heart block and Wenckebach type second degree block especially at night.[10] Resting bradycardia may predispose to increased atrial or ventricular ectopic activity and, in some cases, atrial fibrillation.[1]

As with hypertrophy, there is little evidence to support the view that bradycardia is harmful. The separation of athlete's bradycardia from sick sinus syndrome is somewhat academic if the latter is asymptomatic. One large series of athletes monitored continuously revealed no pauses in excess of 4 seconds.[10] Some subjects do develop symptoms of dizziness or syncope in response to training and require de-training or even permanent pacing. Careful characterisation of this subgroup, however, reveals a previously subclinical sick sinus syndrome rendered symptomatic by the additional stimulus of training.[11]

The effects of increasing age may modify some of the adaptations discussed above. Many athletes maintain their training into middle age and compete in "master" and "veteran" competitions. There is evidence that continued training results in improved diastolic function in the elderly but in one study some left ventricular dysfunction was seen in veteran Japanese cyclists.[12] It was not possible to say whether this was related to training or the development of some other problem such as coronary artery disease. It has also been shown that bradycardias are more pronounced in the older athlete whose maximum heart rate is also less. This may account in part for the drop off in performance with age.[w10]

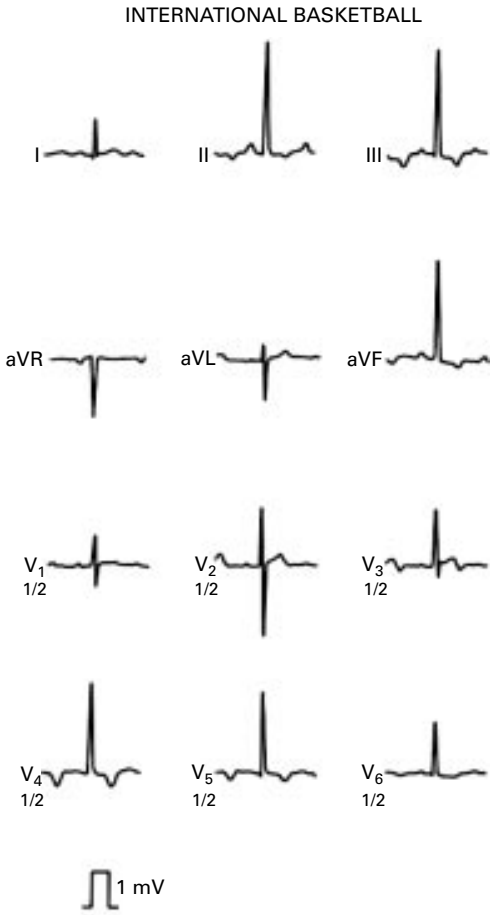

Figure 1. Twelve lead ECG of an international basketball player. Note the left ventricular hypertrophy and inferior T wave changes.

The patient

Athletes can present with symptoms, or because an "abnormality" has been noted incidentally during screening, etc. Symptoms frequently have a relatively benign explanation—dizziness caused by dehydration or post-exercise hypotension, chest pain due to tracheitis or musculoskeletal pain (particularly in contact sports and heavy weight trainers), palpitations caused by benign premature contractions, and shortness of breath due to chest infection or exercise induced bronchospasm. Nevertheless, thorough investigation is often needed to provide patient and physician with the reassurance they need.

Investigational procedures

Physical examination

A physical examination reveals a normal or slow pulse. Loss of the resting bradycardia may be seen in overtrained subjects. The venous pressure and arterial blood pressure are normal. The left ventricle may be prominent to feel and displaced laterally. Third and fourth sounds are permissible, as is a soft midsystolic flow murmur.[2]

ECG

The ECG is usually normal, but left ventricular hypertrophy on voltage criteria is common. Many abnormal ECG patterns have been described. Two of the most common are illustrated here:

- Pronounced left ventricular hypertrophy on voltage with inferolateral T wave changes (fig 1)
- Early depolarisation changes with biphasic T waves in the anterior leads (fig 2).

The second pattern presents a particularly characteristic biphasic T wave morphology with early repolarisation, convex proximally. These changes reflect non-homogenous repolarisation caused by reduced resting sympathetic drive and resolve rapidly on exercise.[13]

Incomplete RBBB, deep anterolateral T wave inversion, and "left ventricular hypertrophy and strain" pattern are described elsewhere.[14 15] The latter is rare.

Various slow rhythms as previously mentioned may be present (fig 3).

The ECG is a constant source of confusion in athletes with numerous variations on the basic patterns. The changes are related to the extent of training and vary in athletes whose training is seasonal. Even quite gross changes may not indicate cardiovascular disease, though thorough further evaluation will often be needed to prove this point. It seems that

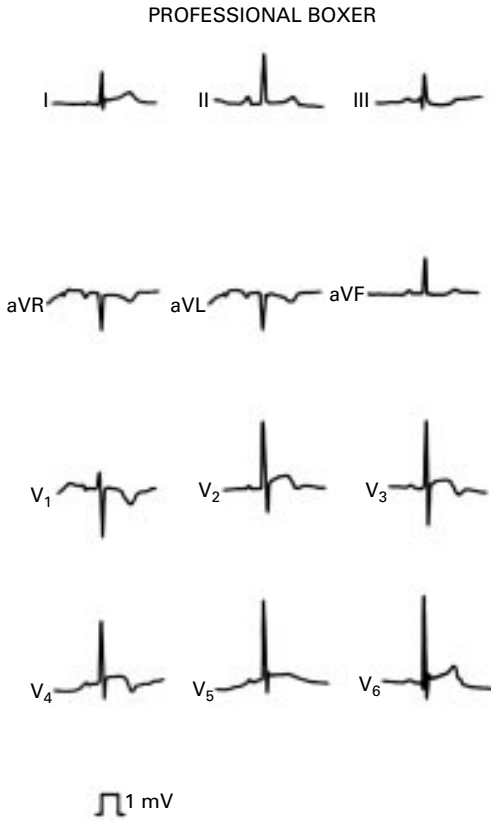

Figure 2. Twelve lead ECG of a European boxing champion. Note the large voltage complexes and bizarre early repolarisation changes in V2–V4.

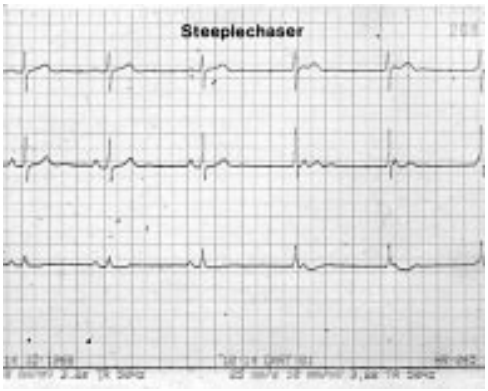

Figure 3. Short rhythm strip of a national steeplechaser. Note the bradycardia and junctional escape rhythm.

such changes are more common in athletes of African Caribbean origin.[w11] The unpredictability and variability of the ECG in athletes seriously limits its value in screening for cardiac disease in these subjects.

Radiography

The chest radiograph is usually normal or may show mild cardiac enlargement.

Echocardiography

Cardiac ultrasound will frequently reveal a modest uniform increase in wall thickness seldom to more than 1.6 cm and usually less than 1.3 cm.[w12] Mild left ventricular cavity dilatation is also observed. Trivial regurgitation of mitral and tricuspid valves is reported more frequently than in the sedentary population. Indices of systolic and diastolic function are normal. In some extreme cases, however, a pattern indistinguishable from hypertrophic cardiomyopathy is observed, even though exhaustive further investigations of the subject and immediate family yield no confirmatory evidence. Echocardiographic features of other confounding conditions may be present. Transoesophageal echocardiography has a role in excluding a patent foramen ovale in scuba divers.

24 hour ECG

Dynamic ECG monitoring may show some of the bradycardic features mentioned above.[w13] Complete heart block and ventricular tachycardia (sustained or unsustained) are not features of the athlete's heart and should be investigated thoroughly. Premature atrial and ventricular contractions are common,[w14] and more complex forms are seen, especially in the elderly.[w15]

Exercise ECG

Stress testing reveals an outstanding exercise capacity with rapid recovery of heart rate in the resting phase. The heart rate response is slower than in untrained people, but the eventual maximum rate is the same. Previously abnormal early repolarisation changes (thought to be related to reduced resting sympathetic tone) and T wave inversion will usually "normalise". It is widely known that patients with coronary disease may develop pseudonormalisation of T wave changes on exercise, but in the context of a fit young athlete this response is reassuring (fig 4). The blood pressure response is normal which may be a helpful distinguishing feature from hypertrophic cardiomyopathy.

Other investigations

Radionuclide studies, cardiac catheterisation, and magnetic resonance imaging provide useful insight into the athlete's heart but, in clinical practice, are largely reserved for specific cases where some suspected cardiac pathology requires elucidation. Multiple gated imaging shows that the increment in stroke volume on exercise in athletes is the result of a normal ejection fraction and increased end diastolic volume, rather than any demonstrable enhancement in contractility.

Vigorous exercise can cause elevation in cardiac enzymes including creatine phosphokinase (CPK). Small rises in MB CPK have also been reported.[w16] This can be confusing if an athlete is admitted having collapsed. The rises are modest, however, and the time scale of enzyme release is not typical of myocardial infarction. Troponin T and I are more specific and should be used in cases of doubt.[w17]

Reversibility

Reversibility of these changes in the detrained athlete can occur quite rapidly in athletes whose training is seasonal (fig 5) or over longer

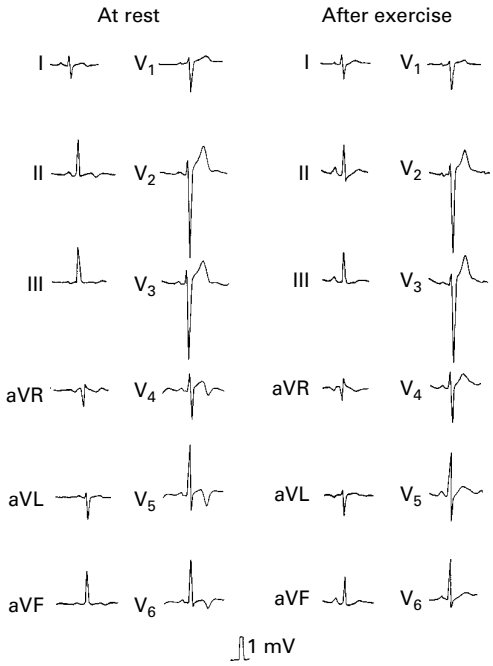

Figure 4. Twelve lead ECG before and after exercise of a county squash player. The striking T wave changes in V4–V6 become normal immediately after exercise.

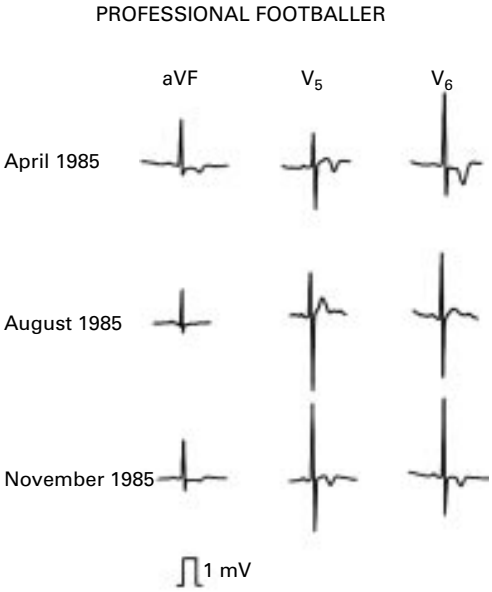

Figure 5. Selected leads of a premier league football player while training (April) after a period of detraining (August) and after resumption of training.

periods of time. It can be used as a distinguishing feature between athlete's heart and hypertrophic cardiomyopathy. A very long term follow up of competitors in the Tokyo Olympics showed eventual regression of hypertrophy in virtually all subjects.[16] Athletes frequently carry on training into middle and old age, competing in veteran and master events. These subjects are particularly confusing as the features of the athlete's heart are superimposed on other changes associated with aging.

Normal or abnormal?

The obvious concern facing a physician consulted by an athlete is "am I overlooking a potentially fatal cardiac condition?". In the young athlete exhibiting extreme hypertrophy or bradycardia the differential diagnosis will lie between hypertrophic cardiomyopathy and sick sinus syndrome, respectively. No single test can be guaranteed to separate athlete's heart from hypertrophic cardiomyopathy; not surprisingly, bearing in mind the great clinical variability of the latter. Helpful features include the extent and symmetry of hypertrophy on echocardiography, the response to three months detraining, the finding of unexplained hypertrophy in close relatives, the blood pressure and metabolic responses to exercise,[17] and other echocardiographic features such as the left ventricular filling profiles.

Asymptomatic bradycardia is less of a problem, but symptoms may merit a period of detraining monitored with serial 24 hour tapes.

In the older subject, coronary disease is more important and such patients with symptoms will require investigation for this with the usual battery of investigations whose fallibility is only too well known.

Sudden death

The death of a fit young athlete is extremely rare. The frequency is estimated as around 1 in 200 000, with considerable variability depending on different reports.[w18–20] Death rates will, of course be higher in recreational athletes and middle aged joggers, but even in these groups the risk is very small and less than in the sedentary population. When death occurs, however, it is more likely to be in association with exercise than during the resting period.[18 w20]

Death can be non-cardiac—for example, traumatic, hypo- or hyperthermia, dehydration or associated with drug abuse. Sudden death is usually cardiovascular, however, and careful necropsy frequently reveals an underlying cause. In younger subjects hypertrophic cardiomyopathy is the most frequent cause of death, though some authors claim that arrhythmogenic right ventricular dysplasia is even more common.[w21] Other causes include anomalous origins of the coronary arteries,[w22] aortic stenosis, myocarditis (possibly related to exercise during the febrile phase of a viral illness), Marfan syndrome, Wolff-Parkinson-White syndrome, long QT syndromes, mitral valve prolapse, and coronary disease. The estimated number of people who might be at risk from these conditions is summarised in a useful paper by Sharma.[17] In older subjects coronary disease assumes greater importance.

No sport has a monopoly of sudden death, though squash has been singled out as posing special risks, perhaps related to its very vigorous and competitive nature.[w23] There is also now a growing interest in extreme sports (for example, triathlons, 24 hour races, and fell

running). The state of complete physical exhaustion encountered by some participants can lead to metabolic changes which adversely affect cardiac performance.[w24]

Screening athletes for cardiovascular disease

The frequent finding of pathology in necropsies of athletes who die suddenly implies that a prior diagnosis of their cardiac condition might have been made on routine screening.[19] Appropriate treatment and advice to avoid extreme exertion could have prevented death. In some countries, such as Italy, screening is mandatory and evidence from this country shows that the programme can be effective.[w25] There are, however, critics of this approach who point out that we are screening for rare abnormalities, for which there is often no curative treatment, with imperfect tests.[20] [w26] Even if a screening programme is embraced, there is no general agreement as to how it should be organised and which tests employed, though some attempts have been made in this area.[w27] The tests we have at our disposal are insensitive and non-specific, and the pre-test probability of cardiac disease is extremely low. This will inevitably lead to missed diagnoses and, more importantly, numerous false positive results. The misdiagnosis of a cardiac problem in a normal athlete is disastrous in someone for whom the state of physical fitness is, by definition, all important. Once the suggestion of cardiac disease has been raised, it is extremely difficult to eradicate it.

While the arguments about the merits of screening continue, it is clear that symptoms or suspicious signs in athletes demand thorough investigation.

Conclusion

Athletes are a challenging group for the cardiologist both in terms of diagnosis and management. The stakes are high. The cardiovascular system is going to be pushed to the limit. The consequences of an erroneous diagnosis are potentially devastating, be it the death of the athlete or a career and way of life in ruins. These patients always merit the most careful evaluation. When still in doubt, the physician is well advised to seek help from others. The author of this paper certainly does!

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website *extra*

Additional references appear on the Heart website

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