The Exercise Paradox

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In 2009, a woman pleaded guilty to reckless homicide and faces up to five years in prison for exercising her husband to death in a swimming pool. He suffered a 'heart attack.' [1] We cannot know, however, whether this was an ischaemic event or an arrhythmia. Exercise is promoted and encouraged in society; it is considered a healthy pursuit with benefits to the heart and mind. We know that certain heart diseases make exercising dangerous, but what risk is exercise to a healthy person without known heart disease?

Ancient history records the death of the Greek messenger Phidippides who ran 26 miles from Marathon to Athens to deliver the news of the victory over Persian invaders, only to collapse and die soon after his arrival.

In the past few years we have heard of professional athletes collapsing during soccer and basketball games and on the athletics track. These are graphically represented and frequently viewed on YouTube. In September last year, Evander Sno, a midfielder for Dutch soccer giants, Ajax, suffered a cardiac arrest during a match. He was successfully resuscitated after four shocks from an external defibrillator – an outcome unfortunately not shared by several athletes in recent years.

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Can these deaths be prevented?

Not so long ago, there was evidence to suggest that marathon runners were immune to coronary artery disease, [2] and this idea has pervaded public perception. If someone can compete in countless marathons and triathlons, how could they possibly be at risk of dying from a heart attack? This has been debunked however, with the finding that coronary disease is the major cause of exercise related deaths in the over 35 age group; a phenomenon also seen in younger individuals. [3] To confuse matters more, there is evidence that strenuous activity kills patients with known heart disease but the risk is reduced if they exercise on a regular basis compared with those who are sedentary. [4] To top it off, recent Australian research shows evidence of damage to the right ventricle detected by MRI following a triathlon in normal hearts. [5]

One of the problems in identifying athletes at risk is the similar appearances of the athletic heart to abnormal pathological hearts. Physiologic changes can occur which mimic the appearance of these conditions (so-called 'athlete's heart'). They can manifest as morphologic changes (such as wall thickening mimicking hypertrophic cardiomyopathy), ECG changes (usually voltage changes, non-specific ST segment and T wave changes) and an increase in ventricular arrhythmias. Ventricular arrhythmias can be a harbinger of serious heart disease and portend a bad prognosis, but they can also be benign and occur in individuals with no structural heart disease and normal life expectancy. Deconditioning has been suggested as a means of differentiating normal from pathological, [6] with regression of hypertrophy, resolution of ECG changes and reduction in ventricular arrhythmia frequency when the athlete stops exercise for a few months. However, reversal of these conditions does not guarantee absence of disease manifestation at a later stage, with a significant proportion of those with resolution of arrhythmia diagnosed with a cardiomyopathy in later years.

In a recent meta-analysis of sudden death in sport, [7] cardiac abnormalities were found in more than 90% of cases with the commonest causes being coronary artery disease (either due to atherosclerosis or congenital anomalies) and hypertrophic cardiomyopathy (HCM). Dilated cardiomyopathy, arrhythmogenic right ventricular dysplasia (ARVD) and mitral valve prolapse made up a significant proportion. These are conditions that are apparent at autopsy, but a group of arrhythmic causes of sudden death, including long QT syndrome, Wolff Parkinson White syndrome (WPW) and conduction disease can only be identified during life or after successful resuscitation from a cardiac arrest. Drug doping, often implied as a feature in high profile athletes by the media, is thought to be an unlikely cause of exercise related death on the basis of histopathologic studies. [7]

How do we identify those at risk?

Pre-participation screening for heart disease would seem logical in an athletic population. A rigorous program has been implemented in Italy for the past 30 years. It includes history taking, a physical examination and ECG; an abnormality then prompts an echocardiogram and further assessment. A history of syncope during exercise and a family history of premature sudden death are examples of red flags, which should prompt cardiac referral. Exclusion from participation in competitive sport in Italy, amongst individuals with anomalies identified during screening, has been credited with an 89% reduction in the incidence of sudden cardiac death. [8] This type of screening program has not been universally adopted because of the perceived financial cost, the large number of people needing screening, the low yield in identifying at-risk athletes and the recognition that it is impossible to eliminate this risk entirely. ECG abnormalities may be present in up to 40% of athletes, leading to unnecessary further testing; but specific syndromes such as HCM, long QT syndrome and WPW may be identified in this way.

What should we do when we identify an at risk athlete?

The diagnosis of a cardiac condition in a prospective athlete can be devastating. Their dreams and aspirations, years of hard work and high achievement can suddenly dissolve in an instant with forced exclusion from sport participation. There are strong financial disincentives to making an adverse diagnosis. This was illustrated in the case of Reggie Lewis, a professional basketball player for the Boston Celtics in the 1990's on a multimillion dollar contract. He was diagnosed with HCM after an episode of syncope whilst playing, and was advised to quit the

game. He sought several expert opinions in the Boston area, all but one of which concurred with the diagnosis. He followed the advice of the physician who 'passed him fit' with a diagnosis of vasovagal syncope on the basis of a positive tilt test. He continued to play after rejecting the other opinions offered and died suddenly during a practice game. In the Italian experience, athletes excluded from competitive sport participation in Italy because of heart disease had rare cardiac events, whereas a number who defied the ban and went on to play competitively in another country had a significant incidence of sudden death.

Is it feasible to allow these people to compete with protection?

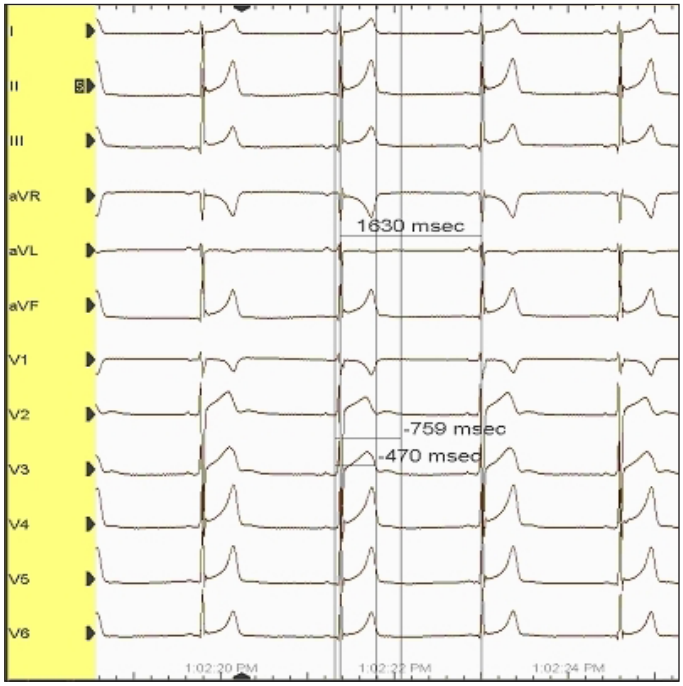
One option that has been debated among cardiologists is to allow athletes with potentially life threatening arrhythmias to continue to play, but with an implanted defibrillator. [8] Evander Sno underwent defibrillator implantation and returned to training two weeks later. This, however, is not a failsafe solution. It is an issue that has not been resolved by medical and sporting bodies and there are medical concerns with this practice. These include damage to the implanted apparatus from direct trauma, the possibility of inappropriate shock delivery because of sinus tachycardia and the possible failure of the device to revert a ventricular arrhythmia under the 'adrenaline-charged' conditions during vigorous activity. Beta-blockers, which have a protective role, are banned in competitive sports.

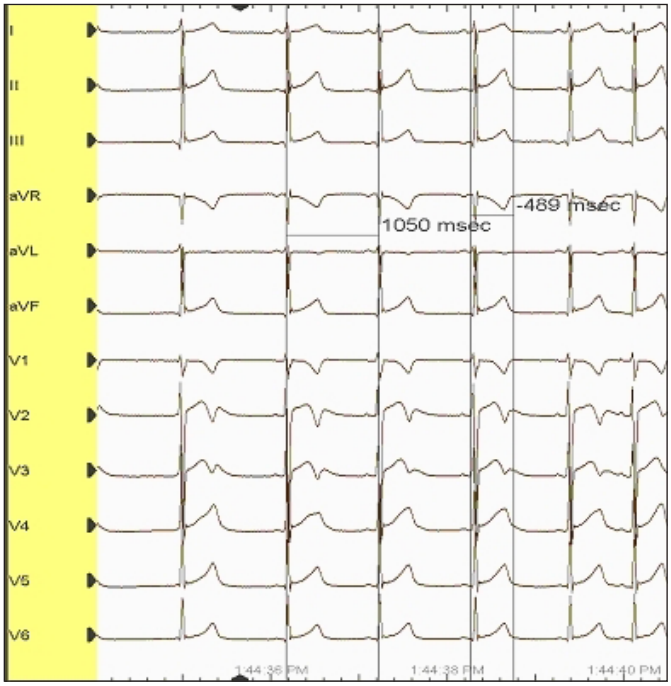
I recently saw an eighteen year old European-based soccer player who had a syncopal episode during a half-marathon. He underwent rigorous testing including exercise testing, an echocardiogram, Holter monitoring, a cardiac MRI and cardiac electrophysiologic testing. A provisional diagnosis of long QT syndrome was made on the basis of his post syncope ECG in the Emergency Department and the response to an adrenaline infusion during electrophysiologic testing. Abnormal prolongation of the QT interval together with characteristic bizarre T wave changes were noted during the infusion (Figure 1). This provocative test is felt by many to enhance the diagnostic accuracy for this condition. [10]

The diagnosis of long QT syndrome would mean exclusion from competitive sport participation. How confidently can the diagnosis be made? Unfortunately, not all cardiologists agree with the implications of this test, arguing that it produces non-specific responses that may mimic long QT in some cases. Many argue that it is better to over-diagnose than under-diagnose this potentially lethal condition, [10] as the stakes are high if the diagnosis is missed and the individual continues to exercise at a high level. Genetic testing may offer an element of added confidence to the diagnosis, but it can take six months to obtain a result and around 20% of patients with the condition will not have a known genetic mutation identified.

So where does that leave the athlete? Does he go on and continue to play at a high level? Does he really care that he may have a condition that might cause him to faint or even die, when he aspires to hero status in a game with millions of zealous followers? Do we monitor him closely with an implantable heart rhythm recording device? Do we follow him around with an external defibrillator? Do we pass him fit to compete in an unrestricted manner? Evander Sno's life was saved by the fortunate availability and judicious use of an external defibrillator. These devices should be seriously considered in public sporting facilities, particularly if at-risk athletes are known to be playing at these venues.

Time will tell if the right decision has been made.





Figures 1 and 2: *Pre (left) and post adrenaline infusion ECG traces: In the right hand trace, after infusion of adrenaline, with a heart rate of ~60/min (RR interval 1050ms), the QT interval is >480ms in duration (normal <440). Note also the marked notching in the T waves in V2 and V3.*

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