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REVIEW

# Sudden Death and Ventricular Arrhythmias in Athletes: Screening, De-Training and the Role of Catheter Ablation

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Athletes enjoy excellent health outcomes including greater longevity relative to non-athletic counterparts. Paradoxically, however, endurance athletic conditioning is associated with an increase in some arrhythmias. This review discusses the potential mechanisms for this paradox and strategies enabling early identification of potentially serious pathologies. Screening remains contentious due to the challenges of identifying relatively rare entities amongst a healthy cohort. The imperfect diagnostic accuracy of all current tests means that screening strategies have potential for harm through incorrect diagnoses as well as the potential for identification of important sub-clinical pathologies. Management of athletes at risk of ventricular arrhythmias and sudden cardiac death is similarly complex. There is much yet to learn about the specific patterns of ventricular arrhythmias in athletes, and the separation of benign from potentially life-threatening remains imperfect. There are some promising advances, however, such as specialised imaging modalities combined with improved electrophysiological diagnostics and therapeutics. Some unique clinical patterns are emerging to advance our understanding and management of athletes with ventricular arrhythmias, requiring specialised skill-sets for evaluation and management.

**Keywords** Arrhythmias • Athlete • Sudden death • Screening

## Introduction

The sudden death of an athlete is a catastrophic event affecting everyone in the community and often resulting in widespread attention. These tragedies engender strong emotions both in the medical community and the wider community in general. Athletes are the healthiest among us and, if this can happen to them, it can happen to anyone.

One of the reasons that sudden death of an athlete is so jarring is the paradox of it occurring in the context of a pursuit generally associated with cardiovascular protection and excellent health outcomes. Athletes live longer and have less disease than non-athletes [1] and there is even evidence that the most elite athletes live longest of all [2–4]. Therefore, the sudden death of an athlete raises many questions, some of which we will attempt to address in this review.

## Why Do Athletes Experience Sudden Cardiac Death?

When sudden cardiac death (SCD) occurs in an athlete, as in the population in general, the mechanism is almost certainly a ventricular arrhythmia. While bradyarrhythmias are common in athletes and a known cause of SCD in some populations [5], there is no evidence that athletes are at increased

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risk of SCD due to bradycardia. As illustrated in Figure 1, in younger athletes, SCD is most frequently due to cardiomyopathies such as hypertrophic cardiomyopathy (HCM), arrhythmogenic right ventricular cardiomyopathy (ARVC) or dilated cardiomyopathy (DCM). Channelopathies such as long QT Syndrome, Brugada syndrome and catecholaminergic polymorphic ventricular tachycardia (VT) are less common causes. With increasing age, coronary artery disease becomes an increasingly common cause and is one of the most common causes of death in middle-aged adults. In more advanced ages, SCD becomes less common due to competing causes of death.

## Are Athletes at Increased Risk of SCD?

When compared to the general, non-athlete population, athletes probably have a lower *lifetime* incidence of SCD than non-athletes, although this is difficult to measure. It is an extrapolation of the fact that athletes appear to experience a lower incidence of coronary disease [1,6] and, given that SCD associated with an acute coronary event is the dominant cause of SCD across the lifespan, it stands to reason that athletes would be somewhat protected. Despite recent studies suggesting an association between endurance exercise and an increasing coronary calcification [7,8], this does not seem to translate to clinical events [9].

In younger age groups, SCD is very uncommon. Young athletes may be at a slightly greater risk of SCD than non-athletes [10,11]; although, this excess risk has not been observed in all studies [12]. It is thought that an excess in SCD in young athletes likely reflects the fact that intense exercise can serve as a trigger for events in those with an underlying inherited or acquired cardiovascular disorder. It is this theory that underpins efforts at cardiovascular screening to prevent SCD in young athletes.

## What is the Incidence of SCD in Athletes?

The reported incidence of SCD amongst athletic groups varies massively. This is a result of considerable variation in the definition of what constitutes an athlete and difficulties in quantifying the cases (numerator) and, more especially, estimating the athletic population from which cases are derived (denominator). The issues of sports-related SCD and SCD occurring in athletes are frequently confused but need to be distinguished. Marijon et al. reported 4.6 cases of sports-related SCD per million population aged 10–75 years [13]. Even though exercise comprises a high risk activity [14], sports-related SCD may be expected to represent only a minority of SCD cases because time spent exercising represents only a very small proportion of total time. For example, 10 hours of athletic training per week represents only 6% of total time and less than a fifth of the time spent sleeping. Thus, it is important to note that, when only SCD cases occurring during exercise are considered, this will grossly underestimate the total number of SCD cases in all groups,

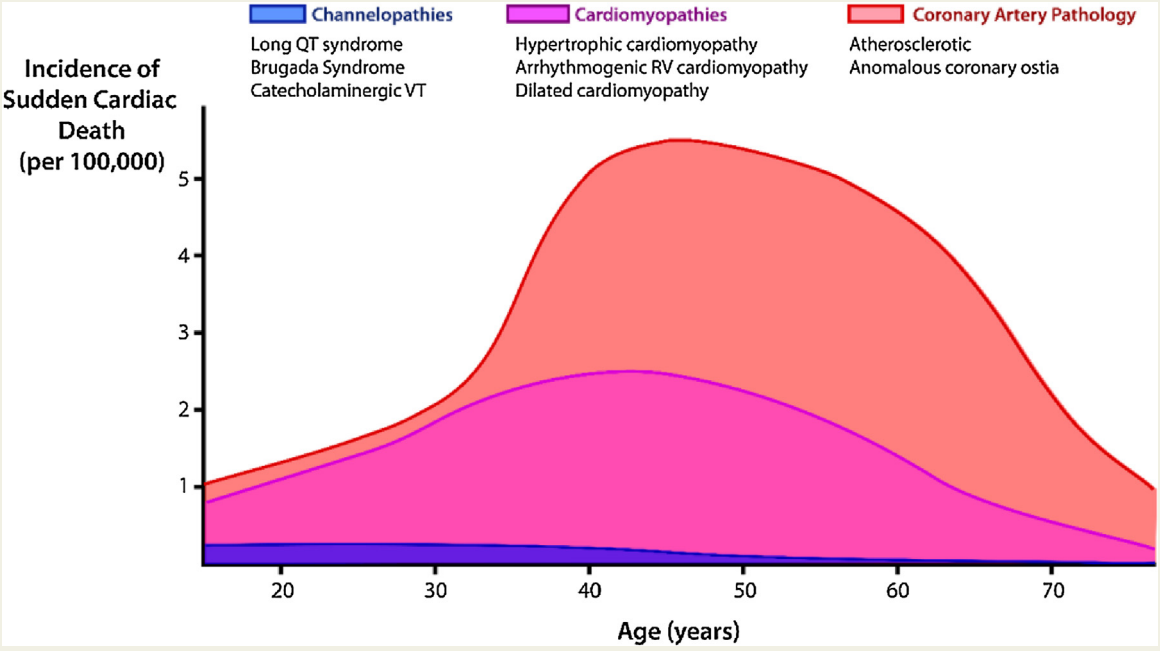


**Figure 1** Age-dependent changes in incidence and aetiology of sudden cardiac death.
This figure represents an interpretation of the combined experience from studies which have assessed the causes of sudden cardiac death in athletes. Whilst the majority of deaths may be attributed to inherited cardiomyopathies and channelopathies in those aged less than 30 years, there is no absolute cut-off. Thus athletes aged in their 30s and 40s (the median age in many competitive sports) are at greatest risk of sudden cardiac death caused by inherited and acquired causes (reproduced with permission from La Gerche et al. *JACC Cardiovasc Imaging 2013* [26]).

including athletes. Furthermore, athletes represent a minority of the population and only a small proportion of those who die during exercise will meet definitions for being an athlete. It may not be surprising, therefore, that only 6% of the sports-related SCD cases described by Marijon et al. were young, competitive athletes [13].

However, the definition of an athlete varies greatly from study to study and estimating how many people in the general population can be considered athletes is difficult. Smaller groups are easier to measure. For example, determining the risk of sudden cardiac death in the highest levels of a professional sport is far easier than determining the risk of SCD in those who regularly participate in sporting activity. A recent screening study of professional English Football Association athletes identified a risk of 1 SCD per 14,794 person-years [15]. This approximates the incidence identified within the National Collegiate Athletic Association in the United States in which the highest ranked basketball players had a concerningly high incidence (1 in 5,451 athletes per year) [16]. On the other hand, larger population-based studies of athletes have consistently reported much lower rates of SCD, typically around 1 in 50,000 [17]. One thing that is clear in all studies is that males are far more commonly affected than females. The reason for this is not entirely clear but is consistent with data in non-athletes in which women are under-represented in SCD in most age groups. Understanding why women are relatively protected from fatal arrhythmias may provide some insight into arrhythmogenic mechanisms and preventative strategies.

## Can We Prevent SCD in Athletes?

Prevention of SCD has frequently been simplified to a focus on screening and particularly screening with electrocardiogram (ECG). However, an effective preventative strategy should involve better education of the public about risks, concerning symptoms, better by-stander cardiopulmonary resuscitation and better access to public defibrillators. Small improvements in each of these steps could result in significant gains although they are all reactive and the best prevention is going to require better understanding of individual risk identification and personalised interventions.

Screening athletes for cardiac abnormalities as part of preparticipation screening is a controversial topic. Both the ACC/AHA (American College of Cardiologists/American Heart Association) and ESC (European Society of Cardiology ) guidelines advocate some form of preparticipation cardiac screening, however, there are differences in the approaches recommended [18,19]. The goal of screening is to identify those athletes who may have a cardiovascular predisposition to SCD as a result of a sub-clinical cardiomyopathy or channelopathy and that early identification may lead to management strategies that mitigate the increased SCD risk. However, once a cardiac abnormality is identified there is limited evidence for how to best manage the patient, particularly regarding ongoing sport participation.

The best evidence for screening comes from observational studies following the initiation of screening programs [20]. This evidence is limited by all of the problems usually associated with observational studies. In the Veneto region in Italy, the observed incidence in SCD in athletes decreased following the initiation of a mandatory screening program [15,20]. This same decrease was not seen in the unscreened non-athlete population. The main criticism of this study is that it does not prove that the observed decreased in SCD was caused by screening. The decrease may simply represent regression to the mean. It is noteworthy that the baseline incidence of SCD was significantly higher than that described in most population-based studies and then decreased to approximate a more expected SCD incidence, thus supporting the concept of regression to the mean.

A recently published study reported outcomes on professional footballers who underwent comprehensive mandatory screening [15]. In brief, the English Football Association mandates thorough cardiovascular screening for high-level academy players aged 15–17 years of age prior to progression to a senior football career. This involves history, physical examination, ECG and an echocardiogram. Out of over 11,000 screened in the study, roughly 800 (7%) were found to have an abnormality. Following further testing, most of these were cleared to continue participation, with ongoing surveillance in approximately 500. Forty-two (42) of the screened athletes were found to have a disorder associated with SCD. Most of these (26 athletes) had a WPW ECG pattern. Five had coronary artery or valve pathology which were treated, allowing return to sporting activity. In total, of over 11,000, only 11 (<0.1%) athletes had disorders for which sport cessation was recommended following investigation and treatment. Unfortunately, despite the comprehensive screening program, eight athletes died of cardiac causes during the follow-up period of, on average, 10 years. Two of these deaths were potentially preventable as both were identified as having HCM and advised to cease sporting activity. The other six athletes who died had normal screening investigations. This study raises many questions. It certainly does suggest that screening can save lives. Clearly two of the deaths probably could have been prevented. Unfortunately, this study also demonstrates that even comprehensive screening will not prevent all cardiac deaths. What is not clear from this study is how many lives were saved. Of the 42 at risk identified, it is unknown how many, if any, would have died without screening.

One of the main criticisms of screening is that it may exclude athletes from sport who would never have experienced an adverse event. Indeed, as stated previously in the English FA study nearly one in ten of the athletes screened had some form of abnormality detected [15]. Following expert evaluation and treatment, however, almost all were cleared to return to sport. Unfortunately, not all screening programs result in such low exclusion rates. In the previously described screening program in Italy, a similar number (9%) had abnormalities detected, with 2% ultimately excluded from sport participation [20].

Athletes who are advised to cease sporting participation may become sedentary out of the very real fear of SCD. While this may be beneficial in reducing SCD in young athletes, this group is then denied the important benefits of exercise. They may then be exposed to the very real risks of a sedentary lifestyle such as increased risk of coronary artery disease (CAD). Furthermore, there is mounting evidence that not all exercise is bad for all cardiomyopathies and channelopathies. A more permissive approach to sports participation may have psychosocial benefits without an increase in the risk of arrhythmias, although this is a topic in which evidence remains scarce [21]. Thus, whilst screening has the potential to identify athletes with sub-clinical disease at risk of SCD, there is also some potential to cause harm. It is important, when cardiac abnormalities are identified, that the athlete is advised on the evidence and, in conjunction with their cardiologist, a management plan is instituted which mitigates risk and also, importantly, minimises harm.

## How Should We Screen Athletes to Prevent SCD?

Though there is some consensus that screening should be performed, there is less agreement on how best to perform preparticipation screening. The AHA/ACC guidelines recommend a comprehensive history and examination, with particular attention paid to family history as part of preparticipation screening [18]. Routine ECG screening is not recommended due to the significant risk of false positives and negatives. Conversely, the European guidelines recommend routine ECG in addition to clinical history and physical examination due to the added sensitivity that ECG provides [19]. Unfortunately, with increased sensitivity comes less specificity [22]. Specificity is further reduced if ECG interpretation is performed without consideration of the athlete's training history. Normal physiological adaptations to exercise cause a number of ECG changes which would be considered abnormal in a non-athlete and thus ECG interpretation should be performed by someone with experience in athletic evaluations. Excellent summaries of the unique considerations in ECG interpretation of athletes have been published [23] and international guidelines have been developed to aid in the interpretation of athletes' ECGs [24]. However, even with the best ECG interpretation, athletes will still be identified with abnormal ECGs who do not, in fact, have significant cardiac disease and, conversely, athletes with a normal screening ECG can suffer SCD, as has been recently demonstrated [15].

As noted previously, some sporting bodies such as the International Football Federation (FIFA) and the International Cycling Union (UCI) mandate more comprehensive screening including echocardiography. There is little evidence supporting the use of echocardiography for routine screening. Zeltser et al. provide some insight into the potential limitations of a pre-participation screening program inclusive of an echocardiographic examination [25]. They screened 2,051 high-school athletes with a 'limited' echocardiographic examination comprising two-dimensional measures of cardiac structure, volumes and ejection fraction;14 athletes (0.7%) were considered to have changes on echocardiogram suspicious of disease (10 HCM, one non-compaction and one DCM) — all but three of which had ECG changes that would have prompted further evaluation in any case. Of the 14 athletes with suspicious changes on screening echocardiography, six refused follow-up, five were considered normal after a more comprehensive echocardiogram and three athletes were diagnosed with a cardiomyopathy (two HCM, one non-compaction). However, none of these three diagnoses were subsequently confirmed by a blinded expert at an independent institution. Thus, this initial real-world experience would suggest that routine echocardiography has the potential to inappropriately exclude athletes whilst having limited or no efficacy in detecting pathological disease with potential for SCD.

A final point on screening is that there is real uncertainty with regards to who should be screened. A rational application of screening would be directed to those groups who are at highest risk. Thus, questions may be raised about the benefits vs. risks in screening female athletes with a SCD risk 5–10-times lower than males. Furthermore, what is the most appropriate age for screening and is it acceptable to screen at only one time point?

## If Screening is Positive, Can Athletes Continue to Participate in Sport?

When screening is performed, it is imperative that it is performed consistently and by clinicians expert in the interpretation of athlete-specific cardiovascular investigations. As highlighted previously, approximately 10% of athletes will have abnormal investigations. If investigations are interpreted correctly then relatively few athletes will be excluded from sports participation given that only approximately 1 in 300 athletes are likely to have a cardiovascular pathology rendering them at increased risk of SCD [26]. The two most common conditions for which sport cessation is recommended are hypertrophic cardiomyopathy (HCM) and arrhythmogenic right ventricular cardiomyopathy (ARVC). Competitive sport has clearly been shown to promote ARVC disease progression and increase burden of arrhythmias [27,28]. An athlete diagnosed with ARVC should be counselled on the available evidence and advised to cease competitive sport.

For the athlete diagnosed with HCM, the evidence is less clear. Hypertrophic cardiomyopathy is the leading cause of SCD in athletes and in young people in general [29]. However, most people with HCM have an excellent prognosis and never develop symptoms or experience a significant arrhythmic event. A recent small randomised control trial demonstrated that moderate intensity exercise was beneficial in HCM with improved quality of life and exercise capacity with less arrhythmias [30]. Although this study was not powered to detect differences in serious arrhythmias, it represents a turning point in investigations into this topic that was previously considered contraindicated. Larger trials including higher risk subjects and more intense exercise

training have been commenced and will provide important evidence as to whether current advice regarding exercise abstinence is justified. Observational data from an implantable cardioverter defbrillator (ICD) registry would suggest that high intensity exercise is safe with similar rates of events in both those who exercise and those who do not [31]. In the absence of compelling data to guide decision making, it would seem prudent to recommend that most athletes with HCM should be advised against competitive sport given the fact that sport is a proven trigger for arrhythmic events. However, it will be exciting to see whether regular exercise training can attenuate the risk of acute exercise bouts as is true for the general population [14].

## Aside from SCD, Are Ventricular Arrhythmias More Prevalent in Athletes?

While athletes seem to be at lower lifetime risk of SCD than the general population, this is not true of all rhythm disturbances. Endurance athletes are at greater risk of developing atrial fibrillation than non-athletes [32]; and, also appear to have a higher burden of ventricular ectopy than non-athletes [33–35]. These studies have not been powered to assess whether this is limited to an excess of ventricular ectopy or includes more complex and sustained ventricular arrhythmias.

### Why Do Athletes Experience Ventricular Arrhythmias?

Like the general population most athletes who have ventricular arrhythmias have underlying cardiac disease, most commonly ischaemic heart disease.

Though the incidence of symptomatic ischaemic heart disease is reduced in comparison to non-athletes, athletes are certainly not immune from coronary disease and myocardial infarction, particularly as they age. Consequently, athletes can and do develop ischaemic heart disease and some develop the associated ventricular arrhythmias. It is important to recognise these risk factors and manage them as aggressively in the athlete as the non-athlete.

As stated previously, endurance athletes have increased ventricular ectopy even in the absence of known cardiac disease. There are a number of potential mechanisms for this excess. The simplest explanation is that there is a diminishing threshold for spontaneous depolarisation the longer the interval between successive beats. Thus, the relative bradycardia in athletes explains some of the excess in ectopic activity. An alternative and potentially less benign proposed mechanism is re-entry due to areas of slow conduction. Athletes have been shown to have more myocardial fibrosis as evidenced by late gadolinium enhancement (LGE) on cardiac magnetic resonance (cMRI) [8,36–38]. Areas of fibrosis have also been documented in athletes who undergo biopsies for ventricular arrhythmias and in autopsies of athletes who experience sudden cardiac death [39–41]. These areas of fibrosis may allow slow conduction promoting re-entry and facilitating ventricular arrhythmias.

Myocardial fibrosis may occur in athletes for several reasons. Athletes who have ischaemic heart disease, manifest or occult, may well have areas of myocardial fibrosis detected by cMRI. Not surprisingly this pattern of myocardial fibrosis predicted future cardiac events in German marathon runners [42]. Athletes may also have fibrosis due to other cardiac disease. As highlighted previously, athletes are not immune to cardiac diseases such as HCM and ARVC. An otherwise asymptomatic athlete may also have myocardial fibrosis due to a past episode of myocarditis [43]. Even in the absence of underling cardiac disease, many athletes have myocardial fibrosis. A pattern of myocardial fibrosis involving the interventricular septum and RV insertion points has been described in endurance athletes [37,44]. This pattern of myocardial fibrosis, however, has not been shown to increase risk of arrhythmias in athletes. On the other hand, recent reports of epicardial scar affecting the LV and/or RV have been associated with serious ventricular arrhythmias [45,46]. Figure 2 illustrates a case of epicardial scar and complex arrhythmias in an elite endurance athlete.

Endurance sport training has also been associated with right ventricular dysfunction and right ventricular arrhythmias. Often considered a spectrum of ARVC, this syndrome is associated with relatively preserved RV dysfunction compared to ARVC, sparing of the left ventricle and rapid ventricular arrhythmias arising from the right ventricular outflow tract (RVOT) [36,39]. Recently this phenotype has been found to be associated with epicardial scar detected by electro-anatomical mapping in the RVOT [47]. The mechanism by which this scar forms may be related to repetitive injury to the RV as result of long-term endurance training. Most athletes, however, do not develop RV dysfunction and arrhythmias. The reason some athletes do develop this syndrome may relate to a combination of genetic predisposition and exercise exposure. Most athletes who have this syndrome do not have a mutation in the desmosomal genes currently associated with familial ARVC [48]; but, it is possible that the predisposition to RV remodelling and arrhythmias is explained by another gene or a complex genetic trait. Amidst this discussion, it is critical to remember that the vast majority of endurance athletes never develop ventricular arrhythmias and that it is likely that an unfortunate combination of genetic predisposition, intercurrent illness and training factors may need to combine (see Figure 3).

## What is the Management of Ventricular Arrhythmias in Athletes?

The management of ventricular arrhythmias in athletes largely depends on the underlying cardiac condition. However, there are some important factors unique to athletes to consider. One of the important considerations in athletes is whether the athlete should be advised to reduce physical activity. The

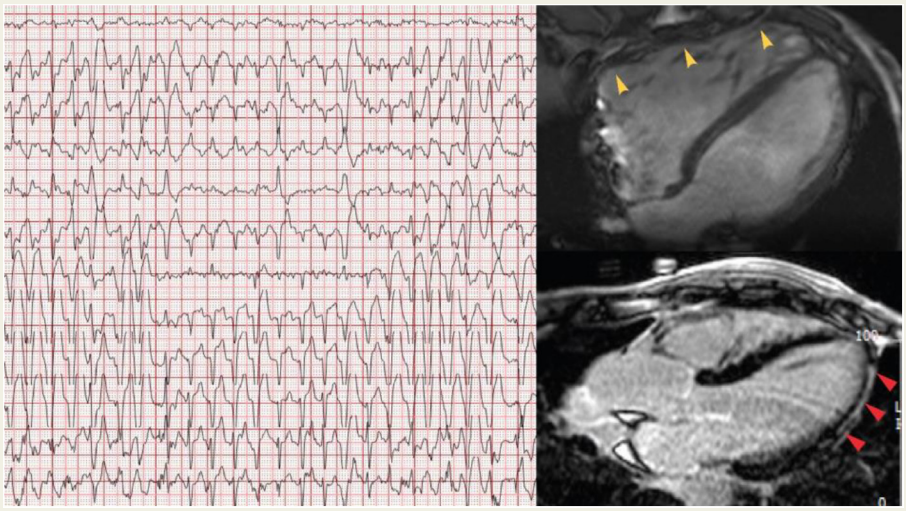

**Figure 2** An elite endurance athlete with complex ventricular arrhythmias.
An exercise electrocardiograph (ECG) demonstrating repeated episodes of non-sustained ventricular arrhythmias of varied morphologies. These complex arrhythmias raise suspicion of underlying cardiac injury. The cardiac MRI (magnetic resonance imaging) images on the right demonstrate right ventricular dilation often resulting in an irregular appearance of the free wall (yellow arrowheads). The bottom right image demonstrates T1 imaging after gadolinium administration. An epicardial-based scar can be seen in the apical lateral wall of the left ventricle (red arrowheads).

ultimate decision should be part of a shared decision making process, however, some sporting bodies may not allow continued participation in a particular sport or may mandate certain treatments prior to resuming activity.

Medical management of ventricular arrhythmias in athletes can also pose some unique challenges. Aside from the usual issues of medication intolerance, side effects and compliance, athletes may also raise concerns regarding the

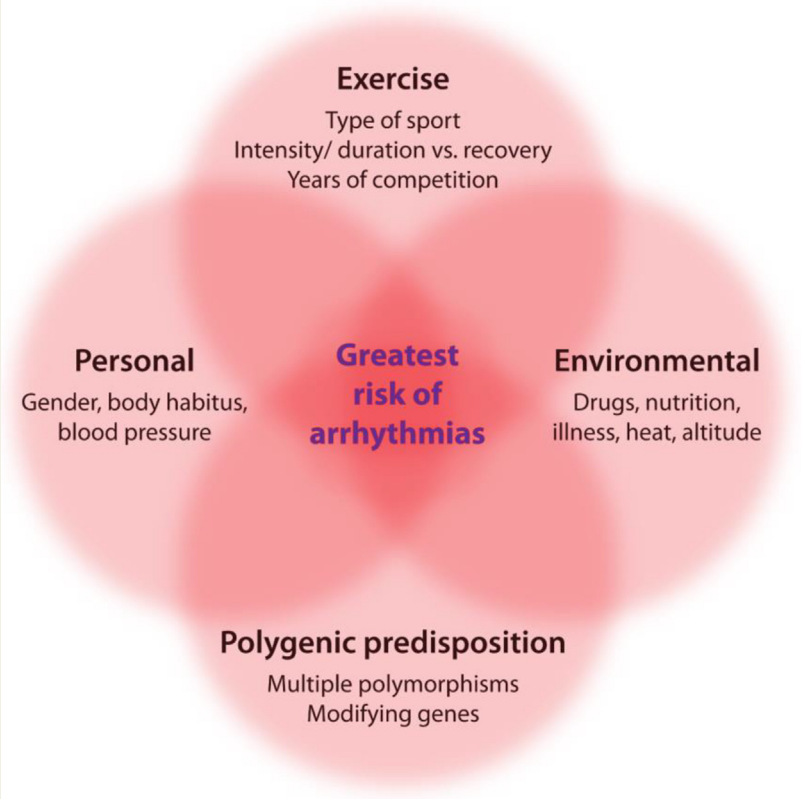


**Figure 3** A complex interaction of multiple factors is likely necessary for an athlete to develop arrhythmias.

potential performance-altering effects of medications. Almost all medications used to treat ventricular arrhythmias will reduce the heart rate response to exercise and some will reduce cardiac contractility. This effect is quite modest [49]; but, can be acutely felt by athletes striving for their best performance.

In athletes with more significant arrhythmias and/or underlying structural heart disease, an ICD may need to be considered for prognostic reasons and some unique factors then come into play. For example, whether to place a transvenous or subcutaneous device, how to program the device to avoid inappropriate shocks, and how to program a device to prevent syncope when arrhythmias occur.

Compared to a subcutaneous ICD (S-ICD), transvenous ICD lead placement allows for the provision of anti-tachycardia pacing (ATP), a smaller device, longer battery longevity and a smaller risk of inappropriate shocks [50]. There are, however, several disadvantages to a transvenous lead. The biggest disadvantage is the long-term risk of an endovascular infection. This risk increases if an ICD is placed in a young athlete who faces a lifetime of device changes [51]. Another disadvantage in athletes is the long-term risk of lead fracture [52]. Certain sports, such as rowing, place increased mechanical stress on the lead increasing the risk of fracture over millions of rowing cycles. Lead fracture carries significant risk of inappropriate shocks which can be a traumatic experience [53]. Tricuspid valve dysfunction and lead-related thromboembolism may exacerbate right ventricular dysfunction, particularly in athletes with ARVC [54]. Additionally, lead extraction is a potentially hazardous procedure. Of course, the significant ECG changes present in some athletes (especially those with HCM) may mean they have increased risk of T wave oversensing-related inappropriate shocks with the S-ICD. For this reason, both resting and stress-test screening of their ECGs are required prior to considering an S-ICD.

Athletes routinely exercise with heart rates near their maximal rate. Consequently, it is important to program ICDs to not deliver therapy at these rates. A dual chamber ICD may help in discriminating sinus tachycardia from ventricular tachycardia though this carries the risk of an extra lead [55]. With subcutaneous or single chamber ICDs, slower VT can be problematic but tends not to cause syncope.

A final consideration for device programming in athletes is how device therapy or lack of device therapy may affect an athlete during sport participation. A swimmer who develops a ventricular arrhythmia is at significant risk of drowning, and a rock climber may be saved from an arrhythmic death but could suffer significant injury, in the event of syncope or an ICD discharge.

For athletes who continue to experience symptomatic ventricular arrhythmias despite medical therapy, catheter ablation can offer significant benefits. There are no randomised trials of catheter ablation in athletes. In fact there have been very few randomised trials of catheter ablation in ventricular tachycardia in any population except ischaemic cardiomyopathy [56]. The evidence for ablation in athletes must be extrapolated from trials in non-athletes and observational data. In patients with structurally normal hearts, catheter ablation is safe and efficacious [57]. Catheter ablation may be the preferred option in athletes who do not wish to take medications. It should be noted, however, that the chance of a successful procedure is dependent on the origin of the arrhythmia [57]. The VT ECG may provide clues as to the origin. This allows for better pre-procedure planning and better estimation of the chances of a successful procedure.

In endurance athletes with RV arrhythmias, Venlet et al. identified epicardial scar in the RVOT as an almost universal substrate in endurance athletes, and demonstrated that radiofrequency ablation was a very effective treatment [47]. Ventricular tachycardia was non-inducible in 10 of the 11 athletes after epicardial ablation of the RVOT, and, in the remaining athlete, there was a marked improvement in arrhythmia burden. At last follow-up (median 27 months), none of the athletes had recurrent VT. Although these results are promising, there are some important caveats. Obviously, this is a small cohort with relatively limited follow-up and, also, athletes were advised to cease intense sports activity. It is not yet known whether resumption of exercise may re-create the same substrate and whether VT may recur. It is also important to note that the localisation of the low-voltage signals, indicating scar, was largely confined to the epicardium suggesting that the more common endocardial approach to ablation would be expected to have limited efficacy in this setting. In Australia, relatively few electrophysiologists routinely perform epicardial ablation. Based on this early evidence, referral of athletes with this syndrome should be directed to expert centres experienced in epicardial ablation.

## Conclusions

On average, athletes enjoy excellent health outcomes including greater longevity relative to non-athletic counterparts. Paradoxically, however, endurance athletic conditioning is associated with an increase in some arrhythmias. This includes benign ventricular ectopics; but, there is also evolving evidence that athletic training is associated with RV epicardial scar and arrhythmias in some predisposed individuals.

Very rarely, athletes will develop ventricular arrhythmias which tragically, can be fatal on first presentation. In most of these cases, inherited heart disease is the underlying cause and screening programs have been developed with the hope of identifying at-risk athletes and preventing SCD. There is, however, considerable debate about who to screen, how to screen, and whether these programs are effective.

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