



REVIEW

Athletes and Arrhythmias

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Abstract

Sudden cardiac death related to athletic competition is a rare but tragic event. The victims are typically young with no previous cardiovascular symptoms or limitations. The majority of sudden cardiac death events in athletes are due to ventricular arrhythmias as a result of underlying molecular and/or structural level pathologic substrate. In this article, we will review the physiologic cardiac adaptations to exercise along with arrhythmias seen in athletes with a focus on those commonly associated with sudden cardiac death.

Keywords: athletes; cardiac remodeling; arrhythmias; sudden cardiac death

Introduction

The occurrence of sudden cardiac death (SCD) in an athlete is a tragic event that deeply affects family members, teammates, and at times, an entire community. Questions usually arise:

- How could this have happened to a young, fit, healthy athlete?
- Were there warning signs that were not recognized?
- Was this a preventable event in this athlete?
- Can we prevent this from happening to other athletes?

Unfortunately, the precise cause of death is frequently not determined definitively, but it is well accepted that the root cause frequently stems from ventricular arrhythmias as a result of an underlying molecular and/or structural level pathologic substrate.

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In 1899, Henschen [1] reported on his clinical findings after performing percussion and auscultation on the thorax of elite Nordic cross-country skiers. He concluded that enlargement of the heart was based on both dilation and hypertrophy, and he found that skiing champions had enlargement of both sides of the heart. Since that time there has been intense scientific interest in the study of cardiac adaptation to repetitive bouts of vigorous activity, athletic performance, and the pathophysiology of structural, functional, and electrical cardiac diseases in competitive athletes. This study has been led by science at the molecular level as well as by advances in imaging that allow us to “look inside the heart” using electrocardiography, echocardiography, cardiac computed tomography, and cardiac magnetic resonance imaging techniques.

Cardiac Adaptation to Exercise

Physiologic adaptation to exercise is dependent on the type of exercise being performed and the degree with which it is performed. In general, two types of exercise are appreciated: static (strength or isometric) exercise and dynamic (endurance or isotonic) exercise. Endurance-based sports activities (e.g., rowing,

swimming, cycling, and long-distance running) result in sustained elevations in cardiac output and normal or decreased peripheral vascular resistance, and importantly, impose significant volume challenges to all cardiac chambers and vessels leading away from the heart. Strength-based sporting activities (e.g., track and field throwing events, weightlifting, karate/judo, American football) result in a normal or slightly increased cardiac output, an increase in peripheral vascular resistance, and transient hypertension, imposing a significant pressure load on both the left ventricle and the right ventricle. There are, of course, many sports that have overlap in the types of hemodynamic load on the heart, such as soccer, lacrosse, basketball, and hockey [2, 3]. This sport-specific cardiac remodeling was described in 1975 and is referred to as the Morganroth hypothesis [4].

Beyond chamber size and thickness, advanced techniques have shown that coexistent with structural cardiac remodeling, functional remodeling occurs and results in both an enhanced stroke volume and enhanced diastolic filling necessary to maintain cardiac output at elevated heart rates [5, 6]. The fundamental reason for cardiac chamber enlargement appears to be due to recurrent episodes of significant volume challenge imposed during athletic training. The changes can be seen in as little as several months with training and can resolve with detraining [7–10]. Multiple factors have been shown to influence cardiac adaptation to exercise, including age, sex, ethnicity, body surface area, type of sport being played, training intensity, and level of sport competitiveness [11].

Chamber-Specific Cardiac Adaptation

Each chamber of the heart responds differently depending on the type and intensity of exercise. Physiologic increase in left ventricular size is commonly seen in athletes, especially in endurance athletes. The left ventricular enlargement is both in chamber dimension and in chamber thickness (eccentric hypertrophy) as opposed to that in isometric athletics, where left ventricular chamber size is usually preserved but increased chamber thickness is seen (concentric hypertrophy) [2]. Differentiating pathologic hypertrophy from physiologic ventricular

hypertrophy is a significant clinical challenge for cardiologists. Fortunately, evolving imaging strategies exist to help differentiate the overlap between physiologic conditioning and pathologic disease processes.

Cardiac remodeling is also known to occur in the right ventricle and may be more instrumental as an arrhythmogenic substrate than left ventricular remodeling. In endurance athletics, right ventricular enlargement is common and coincides with left ventricular enlargement and hypertrophy. Right ventricular enlargement creates a diagnostic challenge when one is attempting to differentiate a physiologic from a pathologic process as a result of its position and shape. The impact of endurance athletics on right ventricular structure and function has received less overall attention than that on the left ventricle. Physiologic adaptation of the right ventricle differs from that of the left ventricle because the work requirement of the right ventricle, at rest, is relatively less than that of the left ventricle, as a result of low right ventricular afterload. With increasing demand, as occurs with increasing exercise intensity, right ventricular systolic pressure progressively rises as a result of increasing pulmonary artery pressure from increased right-sided volume loading. This increasing workload is thought to relatively exceed that of the left ventricle and results in a disproportionate increase in right ventricular wall stress and right ventricular oxygen demand. Relative to the left ventricle, intense endurance exercise has been shown to cause a large reduction in right ventricular function that increases with race duration and correlates with increases in the levels of biomarkers of myocardial injury [12].

Prolonged high-endurance exercise has been shown in other studies to induce cardiac fatigue and even myocardial injury, resulting in a decrease in right ventricular systolic function. This physiologic burden on the right ventricle in some endurance athletes causes chronic and inappropriate right ventricular remodeling, right ventricular dilatation, and right ventricular systolic and diastolic dysfunction and can progress to a syndrome coined “exercise-induced arrhythmogenic right ventricular cardiomyopathy” [13].

Left atrial dimension, volume, and volume index are known to be increased in both endurance athletes and strength athletes [14, 15]. There is grow-

ing evidence that suggests there is an association between endurance athletics and the development of significant atrial arrhythmias. Although precise mechanisms are not well established, contributions of left ventricular volume and/or pressure overload, increased vagal tone with bradycardia, and inflammation are felt to play roles [14, 16, 17].

“Exercise Paradox”

For years the term “exercise paradox” has been used to describe a higher risk of coronary artery events and significant arrhythmias during vigorous exercise [18, 19]. The occurrence of myocardial fibrosis in endurance athletes versus controls has led some to surmise that fibrosis leads to an arrhythmogenic substrate [20]. Excessive endurance exercise has been postulated to be responsible for adverse cardiovascular events, and a beneficial upper limit beyond which adverse effects occur is argued [21].

Bradycardias

Many well-conditioned athletes will have resting bradycardia, with sinus bradycardia being the most common bradycardia. For many years it was felt endurance conditioning resulted in an increase in parasympathetic activity (i.e., vagal tone), leading to slower heart rates at rest. More recently, there is evidence that suggests sinus automaticity and atrioventricular (AV) nodal conduction of athletes are actually determined by alterations of the intrinsic physiologic properties of these specialized conduction tissues [22]. It is likely interplay between the long-term autonomic adaptations and the mechanical anatomic changes results in these intrinsic physiologic changes.

Resting sinus bradycardia with rates in the 30–40 bpm range is fairly common in well-trained athletes. Sinus pauses up to 3 s in duration have been seen in nearly one third of athletes and are typically of no clinical significance unless they are associated with symptoms [23]. Longer pauses or symptoms related to bradycardia should be further evaluated with a 12-lead electrocardiogram (ECG), ambulatory monitoring, echocardiogram, and exercise testing. Electrophysiologic testing may be necessary in some instances.

AV nodal conduction disturbances, such as first-degree heart block and Mobitz I AV block, are also quite common and typically asymptomatic. These changes tend to resolve with hyperventilation, exercise, or detraining and do not require further investigation unless they are symptomatic or a higher-degree AV block develops [24]. For athletes with Mobitz II AV block or complete heart block, permanent pacing is generally recommended. Athletes with permanent pacemakers should avoid sports in which significant bodily contact is anticipated.

Supraventricular Arrhythmias

Atrial Arrhythmias

Premature atrial contractions are frequently seen both in the general population and in athletes. They are typically not associated with structural heart disease or symptoms and do not usually require any further investigation beyond a 12-lead ECG. Atrial fibrillation (AF) is the most common sustained arrhythmia in the athletic community and is more frequently observed in middle-aged athletes [25]. Endurance sports participation increases the risk of developing AF by more than five times the risk for the general population [26].

Several mechanisms are felt to be acting together in some capacity in the induction and maintenance of exercise-related AF. It is well accepted that arrhythmias depend on triggers, substrates, and modulators, and these factors may be present in relation to physical activity [27]. Atrial ectopy, particularly pulmonary vein ectopy, has been shown to be the trigger in most episodes of paroxysmal AF [28]. Enhanced vagal tone, seen with athletes, shortens and increases the dispersion of the atrial refractory period, creating the conditions necessary for reentry [29]. As mentioned previously, long-term endurance training may induce structural changes in the atrium (increased left atrial size, fibrosis) secondary to chronic volume and pressure overload and provide the substrate necessary for facilitating AF maintenance [27].

The evaluation and treatment of AF in athletes is similar to that in the general age-matched population. An echocardiogram to evaluate the heart for structural changes, a 24-h ambulatory monitor to

evaluate heart rate and arrhythmia burden, and an exercise stress test to evaluate ventricular response should all be included as standard evaluation. Treatment for most athletes involves reestablishing and maintaining normal sinus rhythm, especially for the younger population. A rate control strategy with beta blockers or calcium channel blockers is typically reserved for asymptomatic, older athletes. AV nodal blocking agents are generally poorly tolerated in younger athletes and may be prohibited in some competitive sports. Antiarrhythmic drugs are usually effective as most athletes are young and free of structural heart disease. Pulmonary vein isolation (AF ablation) remains a viable first-line alternative because of its higher efficacy in the younger patient population [30]. Anticoagulation therapy to prevent thromboembolic complications is no different from that in nonathletes and should be based on current guidelines (CHADS₂ or CHA₂DS₂-VASc score). Those athletes who ultimately require anticoagulation should not participate in sports with danger of bodily collision. Returning to athletic competitive sports depends on multiple factors, including the frequency of episodes, the presence of structural heart disease, heart response with activity, and clinical response to antiarrhythmic or ablation therapy [31].

Atrial flutter, as well as other forms of atrial tachycardia, in athletes is not typically seen without the presence of structural heart disease. When it does occur, a thorough examination including an echocardiogram and stress test is required. Catheter-based ablation is preferable to antiarrhythmic agents for these particular arrhythmias because of the higher success rates and low risk of the procedure. If ablation is pursued, athletes can participate in all athletic activities after 2–4 weeks without recurrence, or in several days if a further electrophysiology study shows noninducibility [31].

AV Nodal Reentrant Tachycardia/AV Reciprocating Tachycardia

When an athlete presents with symptomatic supraventricular tachycardia, definitive evaluation and treatment with an electrophysiology study and ablation is generally preferred because of the young age and high success rate of ablation and lifetime risk of these arrhythmias despite pharmacologic

therapy. This approach is particularly justified in patients who present with near syncope or syncope because of the unpredictable nature of the tachycardia and the difficulty in defining appropriate end points with noninvasive medical therapy [32]. Symptomatic athletes who opt for medical therapy should be restricted from participating in athletic activities until they have been adequately treated for at least 1 month. At that time, participation in low-intensity activities is permitted [31].

Wolff-Parkinson-White Syndrome

A Wolf-Parkinson-White (WPW) pattern is a manifestation of ventricular preexcitation on baseline ECG. When this ECG pattern is associated with documented clinical tachycardia, the athlete is reported to have WPW syndrome. WPW syndrome is not more common in the athlete, but athletes with WPW syndrome may be at higher risk of SCD because of the high sympathetic drive seen with competition and athletic training [33]. SCD appears to be confined to those WPW syndrome patients with short refractory periods of the accessory pathway along with clinical AV reciprocating tachycardia that degenerates to AF and subsequently to ventricular fibrillation. Athletes with the shortest pre-excited RR interval during AF of less than 250 ms at rest and less than 220 ms when taking isoproterenol are felt to be at higher risk and should be offered catheter ablation. Recently, however, it has been suggested that the risk of an arrhythmic event in asymptomatic WPW syndrome patients goes beyond the conduction properties of the accessory pathway and includes male sex, presence of multiple accessory pathways, and inducibility (AV reciprocating tachycardia/AF) during invasive evaluation [34]. Athletes with symptoms of palpitations and/or syncope are required to undergo invasive evaluation to assess the functional capabilities and electrophysiologic properties of the accessory pathway [31]. Optimal treatment of the asymptomatic athlete, however, remains controversial and continues to be debated by experts in the field [34, 35].

Younger athletes may not have had enough time to develop symptoms, and so the distinction between symptomatic and asymptomatic WPW syndrome may be less meaningful in the pediatric

population [31]. It has been well established that the sudden disappearance of preexcitation during exercise is indicative of a low-risk accessory pathway. It is therefore recommended that all young athletes with asymptomatic preexcitation undergo noninvasive exercise testing for further risk stratification. In those athletes not showing sudden block in their accessory pathway during exercise, further invasive evaluation should be considered after a thoughtful discussion with the athlete, the athlete's family, training staff, etc. [31].

Ventricular Arrhythmias and SCD

Premature Ventricular Contractions

Premature ventricular contractions are common in athletes and can occur in those with and without underlying structural heart disease. They are often asymptomatic and confer no increased risk of sustained ventricular arrhythmias or sudden cardiac death, regardless of the frequency, in the absence of structural heart disease [36]. Deconditioning typically results in a reduction of ventricular ectopy, providing further evidence of their benign clinical nature [37]. Those athletes with frequent or multi-form premature ventricular contractions, however, should undergo further investigation to exclude asymptomatic cardiac disease that could increase their risk of an arrhythmic event. An increase in the frequency or complexity of ventricular ectopy during exercise should also prompt further investigation, especially if it produces symptoms [31]. In the absence of structural heart disease, pharmacologic treatment is only warranted for those athletes who are highly symptomatic.

Idiopathic Ventricular Tachycardia

Athletes with a structurally normal heart and monomorphic ventricular tachycardia (VT) are felt to have idiopathic VT. The two most common cardiac sites for idiopathic VT are the right/left ventricular outflow tract and the left posterior fascicle [33]. Most of the idiopathic VTs can be exacerbated by exercise and can lessen during periods of deconditioning [31]. Catheter ablation has the potential to cure these arrhythmias and is recommended for most athletes, especially if symptoms or ven-

tricular ectopy worsen during periods of exercise. Following a successful ablation, the athlete can resume full competitive activity within 2–4 weeks if further electrophysiologic testing is negative. A more conservative approach is recommended for the athlete who chooses pharmacological therapy because catecholamines released during athletic activity can counter the suppressive effects of the drug, and the VT can reemerge [31].

Sustained Ventricular Arrhythmias and SCD

SCD in an athlete is uncommon but generates significant media attention and debate among the medical community. The true incidence of SCD remains unclear and the incidence can be influenced by geographic location, sex, ethnicity, and method of reporting/collecting events. For many years only publications from the Veneto region of Italy consistently showed an increased risk of SCD for young athletes [38]. Recently, however, data from the National Collegiate Athletic Association and the National Registry for AED Use in Sports in the United States have provided further evidence and support for an increased risk of SCD in young athletes [39, 40].

The cause of SCD is typically sustained ventricular arrhythmias, probably due to exercise-induced catecholamine surges acting on underlying arrhythmogenic substrate. Other contributing influences include dehydration, hyperpyrexia, electrolyte imbalances, and increased platelet aggregation associated with exercise [41].

It is clear that SCD rarely happens in young athletes without the presence of underlying cardiac disease (i.e., structural, channelopathy, etc.), and its prevalence is influenced by the underlying mechanism causing the arrest, sex, sport played, and ethnicity [33].

In athletes older than 35 years, SCD is frequently associated with the presence of atherosclerotic coronary artery disease. Ventricular arrhythmias can occur either from acute ischemia during exercise or from myocardial scar from previous myocardial infarctions [42].

Athletes with underlying cardiac conditions that result in cardiac arrest are typically treated with an implantable cardiac defibrillator as there is no guarantee that medical therapy (either pharmacologic or

invasive) will prevent recurrent events. Those athletes, therefore, cannot participate in any moderate- or high-intensity athletic sports [31].

Structural Cardiac Abnormalities Associated with SCD

Structural myopathies, including hypertrophic cardiomyopathy, arrhythmogenic right ventricular cardiomyopathy, and myocarditis, along with congenital coronary anomalies are the most common causes of SCD in young athletes worldwide [41].

Hypertrophic cardiomyopathy is a genetic disorder caused by mutations in one of the sarcomere genes resulting in various morphologies and severity of left ventricular hypertrophy [43]. SCD due to ventricular arrhythmias is often the first clinical manifestation of hypertrophic cardiomyopathy [44]. Risk factors for SCD include septal thickness greater than 30 mm, family history of SCD, nonsustained VT hypotensive response to exercise, unexplained syncope, and the presence of late-gadolinium enhancement on cardiac MRI [45, 46]. Athletes with a probable or unequivocal clinical diagnosis of hypertrophic cardiomyopathy should not participate in most competitive athletic sports regardless of phenotypic expression, symptoms, medical therapy, etc. [47].

Arrhythmogenic right ventricular cardiomyopathy is an inherited myocardial disease primarily caused by mutations in genes encoding cardiac desmosomal proteins. This alteration results in fibrofatty infiltration and thinning of the myocardium, predominantly affecting the right ventricle [48]. Exercise appears to exacerbate the pathologic changes of myocardial stretching and myocyte detachment which serve as the nidus for the development of ventricular arrhythmias and SCD [41]. Because of the marked increased risk of SCD associated with exercise, athletes with arrhythmogenic right ventricular cardiomyopathy should be restricted from participating in most athletic sports [47].

Anomalous coronary arteries are a common cause of SCD in young athletes. The two most common anomalies associated with SCD are a left coronary artery originating from the right sinus of Valsalva and a right coronary artery originating from the left sinus of Valsalva [49]. Coronary blood flow is impaired by the abnormal ostium of the anomalous

vessel or by compression of the vessel between the aorta and the pulmonary artery [50]. Exercise appears to worsen myocardial ischemia, resulting in ventricular arrhythmias and SCD. Unfortunately, many athletes have few to no symptoms before cardiac arrest. Regardless of symptoms, an athlete with an uncorrected anomalous coronary artery traversing between great arteries should be excluded from participating in athletic activities [51].

Electrical Cardiac Abnormalities Associated with SCD

Long QT syndrome (LQTS) is a heterogeneous group of inherited ion channelopathies resulting in abnormalities of myocardial repolarization and increased risk of SCD. There are at least 13 types of congenital LQTS that have been identified as a result of hundreds of mutations within ten separate genes. Nearly 80% of LQTS cases, however, are due to either loss-of-function mutations involving *KCNQ1* (I_{Ks} -LQT1) and *KCNH2* (I_{Kr} -LQT2) or gain-of-function mutations involving *SCN5A* (I_{Na} -LQT3) [52]. Exercise appears to increase the risk of ventricular arrhythmias and SCD in some of the LQTS subtypes, particularly LQT1 [53]. Athletic restrictions will vary depending on the genotype, symptoms, and baseline QT interval [31].

Catecholaminergic polymorphic VT is a genetic disorder characterized by adrenergically driven polymorphic ventricular arrhythmias resulting in syncope or SCD during periods of exertion or emotional stress [41]. It has been linked to mutations in either the gene encoding the cardiac ryanodine receptor or the gene encoding cardiac calsequestrin [54]. In light of the strong association of arrhythmic events during exercise, most catecholaminergic polymorphic VT patients are restricted from participating in athletic activities [31].

Brugada syndrome is an autosomal dominant sodium channelopathy that is characterized by an accentuated J-wave pattern with ST elevation in leads V_1 - V_3 [41]. There has been no clear association between exercise and SCD in Brugada syndrome patients. Hyperthermia resulting from periods of extreme exertion and enhanced vagal tone resulting from long-term training may enhance the propensity for ventricular arrhythmias [55]. Intensive athletic competition, therefore, is not typically recom-

mended [31]. Moderate- and low-intensity athletic participation is considered relatively safe, except for participation in those activities that would incur significant risk with impaired consciousness, such as swimming alone and rock climbing [42].

Conclusion and Take-Home Message

1. The medical management of athletes presents many unique challenges.
2. The normal cardiac adaptations that occur with intense physical training can be misinterpreted and lead to unnecessary testing and withdrawal from competition.
3. Athletes, however, can have underlying asymptomatic cardiac disease which can be exacerbated by exercise and, rarely, results in SCD.
4. Preventative strategies such as preparticipation cardiac screening to better identify those athletes

at higher risk of sudden death are currently under investigation and remain controversial.

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Conflict of Interest

The authors declare no conflict of interest.

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