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Evaluation of Cardiac Arrhythmia among Athletes

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Abstract

Due to the growing awareness of exercise related arrhythmias and improved sensitivity of diagnostic modalities, physicians are increasingly faced with choices that may have life changing impact for the athlete. This article surveys recent research and expert opinion addressing benign and pathogenic cardiac changes underlying arrhythmias in athletes.

Keywords

Arrhythmia; Athletics; Exercise; Syncope; Review

Introduction

The growing awareness of exercise related arrhythmias and improved sensitivity of diagnostic modalities, has led to increasing arrhythmia evaluations in athletes. As a result, physicians are frequently faced with choices regarding referral for electrophysiology studies, implantable cardioverter defibrillators, and/or limiting activities. Such potentially life changing decisions must depend upon a careful review and understanding of the available literature.

Intense physical activity can result in cardiac electrical and structural changes that mimic diseased hearts. When observed, pathologic cardiac changes in an athlete are rarely caused by exercise. However, increased after load and cardiac output, electrolyte imbalances, release of catecholamines, and autonomic changes in response to intense activity can trigger arrhythmia, syncope and sudden cardiac death in athletes with an undetected underlying genetic or acquired disease.[1] This article reviews recent research and expert opinion regarding arrhythmias among athletes. For brevity, non-arrhythmic conditions associated with sudden death (e.g. Marfan syndrome, valvular heart disease, etc) have been excluded. Information on such conditions can be obtained from comprehensive resources such as the European Society of Cardiology (ESC) and the 36th Bethesda Conference (BC#36) consensus documents.[2,3]

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Benign changes associated with athleticism

Normal structural changes

Physiologic adaptations associated with intense athleticism are known as “Athlete's Heart,” and generally serve to meet the increased oxygen demand of skeletal muscle during rigorous activity. In a meta-analysis of 59 studies (1451 athletes), Pluim et al found that the mean left ventricular septal wall thickness of strength trained athletes was greater than endurance athletes, and both were greater than controls (11.8 vs. 10.5 vs. 8.8 mm, respectively). Similarly, left ventricular mass among strength trained athletes was greater than endurance athletes, and both were greater than controls (267 vs. 249 vs. 174 gm, respectively). Left ventricular internal diameter was largest in endurance athletes, followed by strength trained athletes and control subjects (53.7 vs. 52.1 vs. 49.6 mm, respectively). Importantly, no significant differences were found between athletes and control subjects in left ventricular ejection fraction.[4] Thus, intense activity may result in modest increases in wall thickness and chamber volume compared to the “normal” range, but cardiac function should remain preserved. The typical range of echocardiographic measurements in athletes has been summarized in Table 2.[4]

Normal electrical changes

Athletes often present with electrocardiographic (ECG) features that differ from control subjects. In a cohort of 1005 athletes, Pelliccia et al found abnormal ECG patterns in 40% of participants. Such changes included increased voltage suggestive of left ventricular hypertrophy, inverted T-waves, left or right axis deviation, first degree atrioventricular block, and QRS prolongation. The greatest ECG changes were noted in athletes with more pronounced structural modifications on echocardiography.[5] Sinus bradycardia and type I second degree atrioventricular block can also be noted in trained athletes. The predictive value of such ECG changes for identification of true pathology in the asymptomatic athlete is poor. Inverted T waves are particularly difficult to interpret in the young athlete. The T wave vector is typically directed posteriorly in children. Therefore, inverted T waves are a common finding in the right precordial lead electrograms (V1-V3) of the young athlete. The T wave vector usually rotates anteriorly, resulting in upright T waves in the right precordial leads as the athlete ages. Pelliccia et al reviewed ECGs of 12550 trained athletes and identified 81 with diffusely distributed and deeply inverted T-waves (≥2 mm in at least three leads).[6] These athletes had undergone echocardiography and had no apparent structural heart disease. During follow-up 6% ultimately developed a cardiomyopathy, one of whom died of sudden death and another had aborted sudden death. Among 229 matched control athletes in the study, no cases of cardiomyopathy or sudden death were identified during 9±3 years of follow-up. Therefore, diffusely distributed and deeply inverted T-waves in athletes may be associated with the initial expression of an underlying cardiomyopathy and continued clinical surveillance appears warranted in this setting.

Pathologic conditions among athletes

The following section addresses primary arrhythmias and pathologic conditions underlying arrhythmias among athletes. The disorders are arranged roughly by prevalence, though rates vary by region and demographics. A brief description of each condition and summary recommendations have also been provided in Table 1.

Hypertrophic cardiomyopathy

Hypertrophic cardiomyopathy is an autosomal dominant disease of the cardiac sarcomere with manifestations ranging from no symptoms to atrial fibrillation, heart failure, syncope, or ventricular tachycardia / fibrillation and sudden cardiac death. Hypertrophic

cardiomyopathy is the most common cause of sudden cardiac death among athletes in the United States.[7] Anatomically, the disease is characterized by generalized or localized hypertrophy of the left ventricle. Although usually more extreme, these changes may mimic Athlete's Heart, and diagnosis in athletes can be difficult. Hypertrophy unaccompanied by dilation, focal hypertrophy, bizarre ECG patterns, abnormal left ventricular filling, and family history of hypertrophic cardiomyopathy, should trigger workup in the athlete.[3] Additionally, decreased left ventricular wall thickness (by 2–5 mm) after a break from athletic activity is suggestive of Athlete's Heart rather than hypertrophic cardiomyopathy.[8] Genetic testing for hypertrophic cardiomyopathy is available, but its utility is reduced by a relatively high false negative chance. The difficulty of distinguishing between hypertrophic cardiomyopathy and Athlete's Heart has been reduced by pre-participation screening in Italy; but may be limited by progression of disease after initial screening.

If hypertrophic cardiomyopathy is diagnosed, both the ESC and BC#36 consensus documents recommend that athletes be excluded from competitive sports.[2,3] However, if an athlete is genotype positive for hypertrophic cardiomyopathy but does not express the phenotype the BC#36 document allows participation, whereas the ESC restricts competitive sports.[1,3] The decision to implant an implantable cardioverter defibrillator rests upon family history of sudden cardiac death, syncope, non-sustained ventricular tachycardia, and severe left ventricular hypertrophy. It should be noted here that regardless of the underlying disease, implantation of an implantable cardioverter defibrillator does not allow, but rather excludes athletes from competitive sport participation.[1,7]

Commotio cordis

Blunt force injury to the chest can cause ventricular fibrillation and sudden cardiac death in individuals with or without preexisting heart disease. This type of impact is called commotio cordis and accounts for 3% of sudden cardiac death among athletes in the United States.[7] Commotio cordis occurs in sports with potential for high impact such as baseball, hockey, and boxing. The impact transiently increases left ventricular pressure, which stretches the myocardial cell membrane and activates stretch-sensitive ion channels, ultimately inducing ventricular fibrillation by increasing the dispersion of repolarization.[9] To induce ventricular fibrillation, the impact must occur within a narrow window before the T-wave peak.

Baseballs or hockey pucks manufactured with softer materials, may decrease the likelihood of commotio cordis.[10] The BC#36 consensus document recommends workup to rule out underlying heart disease following resuscitation from commotio cordis. Data regarding the safety of returning to full activity if structural defects are ruled out is lacking.[3]

Anomalous origin of coronary arteries

Anomalous origin of coronary arteries, the most common of which is left coronary origin from the right sinus of Valsalva, can cause sudden cardiac death among athletes. Hypo-perfusion due to deformation of the coronary artery by variant angle of origin or constriction of the artery by surrounding muscle tissue or pulmonary/aortic trunks can be exacerbated by exercise, leading to ventricular fibrillation and sudden cardiac death. Among 27 cases of sudden cardiac death among athletes with anomalous origin of coronary arteries, 55% had no coronary related symptoms prior to the event. Exertional or recurrent syncope and chest pain predominated in those with symptoms, but tests including baseline and stress ECG, and echocardiography were normal.[11] Therefore, methodologies that directly visualize the aortic root and coronary artery ostia (such as cardiac computed tomography) are required when anomalous origin of coronary arteries is suspected.

Surgery can be curative for anomalous origin of coronary arteries by reimplantation in the correct sinus or un-roofing the myocardium that covers the intramyocardial segment. The BC#36 document recommends exclusion of athletes with anomalous origin of coronary arteries from competition if the coronary artery passes between the great arteries. Activities may resume if a normal stress study is obtained 3 months after successful corrective surgery. [3] Although rare, other diseases of coronary arteries such as myocardial bridging and atherosclerotic coronary artery disease can also affect young athletes.

Arrhythmogenic right ventricular dysplasia / cardiomyopathy

Arrhythmogenic right ventricular dysplasia is characterized by enlargement, dysfunction, and fibro-fatty infiltration of the right ventricle. Ventricular tachycardia with left bundle branch block morphology is often triggered by exercise. The predominant symptoms of athletes with arrhythmogenic right ventricular dysplasia are exertional pre-syncope, syncope, or sudden cardiac death. T-wave inversion in leads V1–3 are observed in >85% of arrhythmogenic right ventricular dysplasia patients. Epsilon waves which are distinct deflections that occur after the QRS complex and prior to the T-wave, are observed in <1/3 of arrhythmogenic right ventricular dysplasia patients. Although Athlete's Heart can also cause T-wave inversion in leads V1–3, the T-waves typically normalize during exercise in athletes whereas they remain inverted in patients with arrhythmogenic right ventricular dysplasia.[12] If right ventricular dilation is observed in an athlete, depressed right ventricular ejection fraction and right ventricular outflow tract shortening fraction can help identify the patient with arrhythmogenic right ventricular dysplasia.[13] Cardiac magnetic resonance can serve as an adjunct for the diagnosis by comprehensive right ventricular imaging and visualization of fibro-fatty infiltration on T1-weighted images. However, the normal presence of fat in the atrioventricular groove and anteroapical right ventricular epicardium, and artifacts due to motion, arrhythmia, and surface coil proximity can reduce specificity. Altered distribution of desmosomal proteins on immunohistochemical analysis of endomyocardial biopsy samples also holds promise as a test with high sensitivity and specificity. [14]

Treatment with an implantable cardioverter defibrillator is advised to prevent sudden cardiac death, especially if the family history is concerning. Treatment with beta-blockers can help reduce arrhythmias. Catheter ablation has 60–90% initial success in ventricular tachycardia suppression, but the arrhythmia generally recurs with new arrhythmogenic foci. [15] Both the ESC and BC#36 consensus documents recommend exclusion of athletes with probable or definite arrhythmogenic right ventricular dysplasia from competitive sports.[2,3]

Idiopathic ventricular tachycardia and premature ventricular contractions

Ventricular tachycardia unassociated with obvious structural disease occurs sporadically among athletes and young individuals with little increased risk of sudden cardiac death. Right ventricular outflow tract ventricular tachycardia is the most common idiopathic ventricular tachycardia and can occur in athletes. Exercise increases cyclic adenosine monophosphate levels thus leading to intracellular calcium overload, and triggering right ventricular outflow tract ventricular tachycardia.[16] Absence of right ventricular dilation and hypokinesis helps in distinguishing affected individuals from those with arrhythmogenic right ventricular dysplasia.[17] Treatment with beta-blockers and calcium channel blockers has low efficacy (25–50%), but catheter ablation can be curative.[18] Implantable cardioverter defibrillator implantation or limitation of physical activity is generally not indicated.

Premature ventricular contractions and non-sustained ventricular tachycardia deserve attention in athletes. Both the ESC and BC#36 consensus documents recommend that

athletes with premature ventricular contractions and non-sustained ventricular tachycardia undergo workup for underlying structural diseases such as hypertrophic cardiomyopathy and arrhythmogenic right ventricular dysplasia.[2,3] However, if the athlete is asymptomatic and has no family history of sudden cardiac death, structural disease is ruled out, ectopy with short RR intervals is absent, and the arrhythmia does not worsen with exercise, sports participation is not limited by the BC#36 consensus document.[2,3]

Atrial fibrillation / flutter

Atrial fibrillation is the most common arrhythmia in athletes and may be associated with long-term endurance training.[19] Mechanistically, atrial fibrillation in athletes may be related to elevated vagal tone, left atrial enlargement, or other underlying substrates such as Wolff-Parkinson-White, myocarditis, or arrhythmogenic right ventricular dysplasia. Pulmonary vein isolation can be curative in the athlete with symptomatic lone atrial fibrillation.[20] Atrial flutter can co-exist in the athlete with atrial fibrillation or present in isolation, and is likely due to right atrial dilation in the setting of Athlete's Heart. When cavotricuspid isthmus dependent, catheter ablation effectively treats atrial flutter and may suppress coexistent atrial fibrillation.

Both the ESC and BC#36 consensus documents allow participation in competitive sports if structural heart disease and rapid ventricular rate are absent and anticoagulation is not deemed necessary. Individuals with rapid ventricular rate or need for long term anticoagulation should be excluded from competitive sports. However, successful catheter ablation of lone atrial fibrillation or flutter without evidence of recurrence during a 4–6 week observation period can lift restrictions.[2,3]

The long QT syndrome

The long QT syndrome, a condition resulting in prolonged and abnormal cardiac repolarization, may be acquired or a result of one of a multitude of genetic aberrations. The condition is rare, with an estimated prevalence of 0.4% among athletes.[21] The three most common forms of long QT syndrome are: (1) long QT syndrome −1, a slow outward delayed rectifier potassium current abnormality encoded by mutation of gene *KCNQ1* on chromosome 11 with sudden cardiac death associated with exertion; (2) long QT syndrome −2, a rapid component outward delayed rectifier potassium current abnormality encoded by mutation of gene *KCNH2* on chromosome 7 with sudden cardiac death associated with autonomic nervous system arousal and stress; and (3) long QT syndrome −3, a gain of function sodium channel abnormality encoded by mutation of gene *SCN5A* on chromosome 3, with sudden cardiac death associated with rest and bradycardia.

The BC#36 consensus document recommends QTc diagnostic cutoffs of 0.47 and 0.48 for male and female athletes, respectively.[2,3] Genetic tests for the long QT syndrome are available and may be useful among asymptomatic athletes with prolonged QTc or those with family history of sudden cardiac death. Importantly, many genetically proven long QT syndrome patients have borderline prolonged or normal QTc intervals at baseline. Epinephrine, isoproterenol, or adenosine challenge can be used to diagnose the long QT syndrome, by paradoxic prolongation of the QT interval with tachycardia. Medical treatment for long QT syndrome −1 and 2 includes beta-blockers, whereas long QT syndrome −3 is typically treated with sodium channel blockers. History of syncope, QT intervals >480 ms, and family history of sudden cardiac death are associated with greater risk and treatment with an implantable cardioverter defibrillator should be considered. If the long QT syndrome is diagnosed, both the ESC and BC#36 consensus documents recommend that athletes be excluded from competitive sports.[2,3] The BC#36 also recommended swimming restriction in athletes with a genotype diagnosis of long QT syndrome −1 without phenotypic effects,

however, the document allows competitive sport participation for other phenotype negative genotype positive long QT syndrome patients.[3] In contrast, The ESC recommendations exclude all genotype positive athletes from competitive sport regardless of phenotype.[2]

Wolff-Parkinson-White syndrome

Wolff-Parkinson-White syndrome, characterized by the presence of ECG manifest accessory pathway conduction, can lead to palpitations due to atrio-ventricular reentry tachycardia and rarely (<0.6% incidence) sudden cardiac death due to antegrade conduction of atrial fibrillation. Indicators of low risk include concealment of the accessory pathway with increasing heart rate, while high risk indicators include symptoms, multiple pathways, persistence of the delta wave during exercise treadmill test or accessory pathway effective refractory period < 240 ms. Both the ESC and BC#36 consensus documents recommend electrophysiology study and catheter ablation in symptomatic athletes with Wolff-Parkinson-White syndrome. The ESC (but not BC#36) recommends electrophysiology study to assess the pathway effective refractory period in all athletes with Wolff-Parkinson-White syndrome regardless of symptoms.[2,3]

Brugada syndrome

Brugada syndrome is the result of an autosomal dominant mutation in the *SCN5A* gene on chromosome 3, resulting in a loss of function sodium channel abnormality. An ECG can be diagnostic if a characteristic type-1 ST segment elevation in the right precordial leads followed by a negative T-wave is present. However, ECG abnormalities can be concealed, and a challenge with sodium channel blockers is often required for diagnosis. Heat, hypokalemia, and many compounds such as glucose and insulin can precipitate not only diagnostic changes, but also ventricular fibrillation. Benign ST-segment elevation encountered in athletes, is distinguished from Brugada syndrome by lower ST segments, shorter QRS duration,[22] and by lack of change with sodium channel blocker challenge.

Higher risk is associated with history of syncope / sudden cardiac death, family history of sudden cardiac death, and a type-1 ECG; while lower risk is assumed if the ECG abnormalities are not spontaneously present. Implantable cardioverter defibrillator implantation can prevent sudden cardiac death in Brugada syndrome. Quinidine in relatively high doses may be beneficial for infants or if implantable cardioverter defibrillator implantation is not possible.[23] Both the ESC and BC#36 consensus documents recommend that athletes with a diagnosis of Brugada syndrome be excluded from competitive sports. The ESC (but not BC#36) also excludes genotype positive / phenotype negative athletes from competitive sport. [2,3]

Catecholaminergic polymorphic ventricular tachycardia

Catecholaminergic polymorphic ventricular tachycardia is an inherited autosomal dominant disorder of Ryanodine receptors which leads to calcium loading and propensity for ventricular tachycardia or fibrillation triggered by either emotional or physical stress.[24] Structural changes are absent, and arrhythmias and baseline ECG changes are not seen in the absence of stress, therefore catecholaminergic polymorphic ventricular tachycardia can elude detection under normal conditions. The diagnosis of catecholaminergic polymorphic ventricular tachycardia can be made by exercise treadmill test or Isoproterenol infusion, and confirmed via genetic tests. If confirmed, catecholaminergic polymorphic ventricular tachycardia is treated by implantable cardioverter defibrillator implantation and beta-blockers. Both the ESC and BC#36 consensus documents recommend that athletes diagnosed with catecholaminergic polymorphic ventricular tachycardia be excluded from competitive sports. Additionally, the ESC excludes genotype positive / phenotype negative

athletes from competitive sport, whereas the BC#36 allows competition so long as the phenotype remains negative.[2,3]

The Short QT syndrome

The short QT syndrome appears to be a highly lethal but rare condition typically caused by potassium channel gene mutations. The ECG shows a QT interval of ≤ 300–320 ms. [25] Patients with short QT syndrome often suffer from atrial fibrillation, and can also present with sudden cardiac death either at rest or with exertion. The diagnosis of short QT syndrome is primarily made based upon the surface ECG. If an athlete is suspected to have short QT syndrome, especially in the setting of other affected relatives, genetic testing may be considered. Implantable cardioverter defibrillator implantation may be effective against sudden cardiac death, although data is limited. The arrhythmias associated with short QT syndrome may also be mitigated with quinidine.[26] Given the poor outcomes observed within the growing but small experience with this condition, the BC#36 consensus document recommends excluding patients with short QT syndrome from competitive sports.[3]

Athleticism and arrhythmia: association versus causality

Generally, it is accepted that cardiac disease in athletes is due to undetected pre-existing defects, and that, while athleticism can trigger symptoms and events, it is not the etiology of cardiac disease or arrhythmia. In an interesting recent study however, La Gerche et al demonstrated elevation of serum Troponin-I and B-type natriuretic peptide levels in 58% of athletes who completed a triathlon. New left ventricular regional wall motion abnormalities were also seen in 27% of athletes, and right ventricular function was reduced in all athletes. [27] Almost all abnormalities resolved within a week after the triathlon; however the study challenges previous thinking regarding the benign nature of intense exercise. These findings may share a mechanism with reversible left ventricular dysfunction in the setting of emotional stress and high catecholamine levels.[28] Further studies to assess the association and potential for causality between exercise and myocardial dysfunction and arrhythmia are necessary.

Conclusion

The majority of arrhythmias on the athletic field are due to undetected cardiac structural defects or channelopathies. The symptoms of cardiac disease including ECG changes, dilation, and hypertrophy can be masked in athletes by the morphological adaptations that take place in response to athletic training, thus making the diagnosis difficult. The contents of this review are meant as a succinct review and the reader is encouraged to read comprehensive resources such as the ESC and BC#36 consensus documents.[2,3]

Clinical Significance

- The growing awareness of exercise related arrhythmias has led to increasing arrhythmia evaluations in athletes.
- Intense physical activity can result in cardiac electrical and structural changes that mimic diseased hearts. However, even in elite athletes, these adoptive changes are generally modest.
- Athletes with cardiovascular symptoms, family history of sudden death, or structural or ECG abnormalities outside the observed range for their level of training, merit further investigation.

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Table 1

Summary of Conditions Associated with Cardiac Arrhythmia in Athletes

Condition	Description	Recommendation [2,3]
Hypertrophic Cardiomyopathy	Autosomal dominant inheritance, generalized or localized hypertrophy of the left ventricle, heart failure, atrial and ventricular arrhythmia, syncope, sudden death	No competitive sports, consider defibrillator implantation
Commotio Cordis	Blunt force injury to the chest resulting in ventricular fibrillation	Workup to rule out underlying heart disease
Anomalous origin of coronary arteries	Variant site of origin of right or left coronary artery, myocardial hypoperfusion, ventricular arrhythmia	No competitive sports, consider referral for surgery if myocardial hypo-perfusion is demonstrated
Arrhythmogenic right ventricular dysplasia / cardiomyopathy	Enlargement, dysfunction, and fibro-fatty replacement of the right ventricle, T-wave inversions in leads V1–3 or Epsilon waves on baseline ECG of some but not all patients, ventricular tachycardia (with left bundle branch like morphology), exertional pre-syncope, syncope, or sudden death	No competitive sports, defibrillator implantation, beta blockers or catheter ablation for ventricular tachycardia suppression and shock reduction
Idiopathic ventricular tachycardia	Monomorphic ventricular tachycardia, normal cardiac structure and function	Consider catheter ablation
Atrial fibrillation / flutter	Most common arrhythmia in athletes, associated with endurance training	Competitive sports ok if structural heart disease and rapid ventricular rate are absent, consider catheter ablation
The long QT syndrome	Prolonged and abnormal cardiac repolarization, acquired or inherited	No competitive sports, consider defibrillator implantation
Wolff-Parkinson-White syndrome	Delta wave, atrio-ventricular reentry tachycardia, sudden death risk due to antegrade conduction of atrial fibrillation	Electrophysiology study and catheter ablation in symptomatic athletes, investigate pathway effective refractory period if asymptomatic
Brugada syndrome	Autosomal dominant inheritance, RSR' and ST segment elevation in right precordial leads, syncope, ventricular fibrillation	No competitive sports, defibrillator implantation
Catecholaminergic polymorphic ventricular tachycardia	Autosomal dominant inheritance, polymorphic ventricular tachycardia or fibrillation triggered by exercise	No competitive sports, defibrillator implantation, beta blocker to reduce events
The short QT syndrome	Shortened and abnormal cardiac repolarization, atrial fibrillation, sudden death	No competitive sports, consider defibrillator implantation

Table 2

Summary of Normal Echocardiographic Measurements in Athletes

	Endurance-Trained Athletes	Strength-Trained Athletes
Left Ventricular End Diastolic Dimension (cm)	5.28–5.46	5.06–5.36
Diastolic Posterior Wall Thickness (cm)	1.00–1.06	1.02–1.17
Diastolic interventricular Septum Thickness (cm)	1.01–1.09	1.09–1.27
Left Ventricular Mass (gm)	233–264	234–300
Left Ventricular Ejection Fraction (%)	65.1–72.6	60.7–71.9

Values represent 95% confidence intervals for echocardiographic data extracted from the meta-analysis by Pluim et al.[4]