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REVIEW

Sudden Death in the Athlete

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ABSTRACT

Arrhythmias in the athlete can range from benign and asymptomatic to highly symptomatic and life-threatening. There is risk of not diagnosing an important cardiovascular condition which may predispose to a serious or life threatening arrhythmia. At the same time there is the risk of unnecessarily restricting the athlete with a more benign condition. Sudden cardiac death in the athlete, although relatively uncommon, remains a challenge as the screening techniques for identification of individuals at risk are insensitive. Many of the underlying cardiovascular diseases responsible for sudden death with exercise can be identified. These include hypertrophic cardiomyopathy, arrhythmogenic right ventricular dysplasia, Wolff-Parkinson-White syndrome, anomalous origin of the coronary artery, and the inherited long QT syndromes. To prevent fatalities and unnecessary restriction on athletic participation, the best available information has recently been updated with consensus recommendations and guidelines regarding eligibility. In this manuscript the cardiovascular conditions and cardiac arrhythmias that predispose to sudden cardiac death in the athlete are discussed.

KEY WORDS: *sudden cardiac death, athletes, supraventricular arrhythmias, ventricular arrhythmias, cardioverter defibrillator, hypertrophic cardiomyopathy, right ventricular cardiomyopathy, anomalous coronary artery, long QT syndrome*

ABBREVIATIONS
AED = automatic external defibrillator
CPR = cardio-pulmonary resuscitation
EKG = electrocardiogram
EMT = emergency medical team
EP = electrophysiology
ICD = implantable cardioverter defibrillator
MRI = magnetic resonance imaging
PVCs = premature ventricular contractions
SCD = sudden cardiac death
WPW = Wolff-Parkinson-White (syndrome)

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INTRODUCTION

Sudden cardiac death in the athlete has intrigued the medical community and the public since the initial report of the unexpected death of the Greek soldier Phoedippides on completing his historic run from Marathon to Athens to deliver the message of victory over the Persians in 490 B.C. To the extent that these deaths occur in the healthiest segment of society, the unexpected death of an athlete is a tragic irony. Because sudden cardiac death in the athlete is relatively uncommon and the screening techniques for identification of individuals at risk are insensitive, the problem of sudden death remains a challenge. Many of the underlying cardiovascular diseases responsible for sudden death with exercise are now identified. These include hypertrophic cardiomyopathy, arrhythmogenic right ventricular dysplasia, Wolff-Parkinson-White Syndrome, anomalous origin of the coronary artery, and the inherited long QT syndromes. To prevent fatalities and unnecessary restriction on athletic participation, the best available information has recently been updated with consensus recommendations and guidelines regarding eligibility. In this manuscript, we will discuss the cardiovascular conditions and cardiac arrhythmias that predispose to sudden cardiac death in the athlete.[1-9]

Arrhythmias in the athlete can run a range of risk from benign and asymptomatic

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to highly symptomatic and life-threatening. There is risk of not diagnosing an important cardiovascular condition which may predispose to a serious or life threatening arrhythmia. At the same time there is the risk of unnecessarily restricting the athlete with a more benign condition. Furthermore, the evaluation of the athlete is confounded by many complex, psychological, social and in some instances, economic implications. Benign arrhythmias such as many bradyarrhythmias and atrial and ventricular premature contractions are common in the young and older athlete. Supraventricular arrhythmias such as atrial fibrillation, atrioventricular nodal re-entrant tachycardia, and the Wolff-Parkinson-White syndrome are less common. The least common, but clearly the most important to diagnosis and treat appropriately, are life threatening ventricular arrhythmias which may predispose the patient to a clinical syndrome of sudden cardiac death. Although the annual risk of sudden death in the athlete appears to be low (between 5 and 10 athletes for each 1 million participants per year), selected athletes such as those with hypertrophic cardiomyopathy may be at particularly high risk.

EVALUATION AND MANAGEMENT

Evaluation and management of the athlete at the time of screening physical exam or when presenting with symptoms of a cardiac arrhythmia begins with a detailed history. The physician should always ask the individual whether they have palpitations, lightheadedness, "skipped beats", "extra beats", presyncope or syncope. When symptoms are present, the physician should determine whether these are associated with exertion, post-exertion or at rest. In general, symptoms of an arrhythmia which occur with exertion are more likely to be serious than those occurring at rest. Rarely a neurocardiogenic reflex can be the cause of syncope during exertion. The duration and frequency of the symptoms are important. In this respect, symptoms that are frequent and sustained (lasting greater than 30 seconds) are probably less likely to be serious or life-threatening compared to those that are short lived and shorter in duration. The decision to further evaluate the athlete with symptoms possibly due to an arrhythmia needs to be made in the context of the patient's family history of any significant cardiovascular condition or premature sudden death and the presence or absence of any structural heart disease in the athlete. In general, when an athlete presents with symptoms consistent with a more serious arrhythmia, such as sudden onset, syncope, exertionally related symptoms or injury associated with the arrhythmia, a full evaluation is warranted. By contrast in the athlete with symptoms which suggest a less serious condition, such as post-exertional lightheadedness or orthostatic symptoms, it may be appropriate to undertake a more limited evaluation.

Premature ventricular contractions and nonsustained VT in the absence of structural heart disease, generally have no prognostic significance and need to be treated only if the symptoms are moderate or severe. However, in the presence of structural heart disease, nonsustained ventricular tachycardia, or sustained ventricular tachycardia could be a marker of risk for sudden cardiac death.

CARDIOVASCULAR CONDITIONS PREDISPOSING TO SUDDEN DEATH

In the younger athlete (less than 35 years of age) hypertrophic cardiomyopathy is the most commonly underlying structural heart disease predisposing to sudden cardiac death. In fact, hypertrophic cardiomyopathy accounts for up to 50% of the sudden deaths in young athletes in North America. Anomalous origin of the coronary arteries, arrhythmogenic right ventricular dysplasia, as well as a number of less common conditions causing sudden death in the athlete are listed in Table 1. In the athlete older than 35 years of age, coronary artery disease is the underlying condition which predisposes to sudden death and over 75% of patients. By contrast, the experience in Italy is that arrhythmogenic right ventricular dysplasia more commonly predisposes to sudden cardiac death in the young athlete than hypertrophic cardiomyopathy.

TABLE 1. Cardiovascular Conditions Associated with Sudden Cardiac Death in the Athlete

• Hypertrophic cardiomyopathy
• Coronary artery disease
• Arrhythmogenic right ventricular dysplasia
• Left ventricular hypertrophy
• Myocarditis
• Conduction system abnormalities
• Mitral valve prolapse
• Congential heart disease
• Valvular heart disease
• Aortic dissection
• Cerebral embolus
• Pulmonary embolus
• Arteriovenous malformation
• Berry aneurysm
• Wolff-Parkinson-White syndrome
• Myocardial bridge
• Coronary aneurysm
• Subvalvular aortic stenosis
• Long QT syndrome
• Idiopathic ventricular fibrillation
• Dilated cardiomyopathy

SUPRAVENTRICULAR ARRHYTHMIAS

Supraventricular arrhythmias are not more common in the athlete with the possible exception of atrial fibrillation. Additionally supraventicular arrhythmias rarely predispose to sudden death (Table 2). However, atrial fibrillation in the presence of Wolff-Parkinson-White Syndrome rarely allows conduction to the ventricle with ventricular rates greater than 250 beats/minute. Rarely with intense physical exertion this may predispose to ventricular fibrillation in an ortherwise normal heart. Atrial fibrillation in the presence of hypertrophic cardiomyopathy can result in loss of atrial contraction, lowering of cardiac output and hemodynamic collapse.

Atrial fibrillation or flutter in the athlete is felt to be more common compared to an aged match population of non-athletes. Vagally medially atrial fibrillation may be more common in athletes. Athletes presenting with atrial fibrillation or flutter should undergo a comprehensive history and physical examination including a detailed history with regard to alcohol, caffeine or illicit drug use, an EKG, an echocardiogram and TSH to screen for thyroid abnormalities. If the individual is in sustained atrial fibrillation or flutter, the maximal ventricular response should be assessed with an exercise stress test and ambulatory monitoring while the patient is exercising during atrial fibrillation.

Wolff-Parkinson-White (WPW) syndrome, manifesting with ventricular pre-excitation in the form of a delta wave is another cause of supraventricular arrhythmias in the athlete.[10] A delta wave is found in approximately 3 out of 1,000 individuals . The evaluation of the athlete, or any individual with asymptomatic ventricular pre-excitation or WPW pattern on the surface EKG remains controversial. Some electrophysiologists recommend observation without restriction of athletic participation since the risk of sudden death appears to be extremely low. Because the WPW syndrome is associated with some congenital abnormalities such as Epstein's anomaly, it is generally agreed that a history, physical examination, 12-lead EKG, 24-hour ambulatory monitoring, stress test, and echocardiography should be performed to exclude associated cardiovascular abnormalities. .Some experts recommend electrophysiological evaluation in the asymptomatic athlete with WPW to define the properties and location of the bypass tract. If conduction to the ventricle is present through the bypass tract at rates greater than 240 bpm, consideration should be given to radiofrequency ablation to eliminate the risk of future life threatening arrhythmias.[11] Athletic restrictions are not generally recommended for the asymptomatic individual with WPW.

TABLE 2. Supraventricular Arrhythmias in the Athlete

Arrhythmia	Baseline ECG	Symptoms	Diagnosis	Treatment Options	Guidelines for Athletic Participation[1,2]
APCs	Often WNL	Palpitations	Monitor	Reassurance	No restrictions beta-blocker if highly symptomatic
Atrial Fib	Often WNL	Palpitations	Monitor	Antiarrhythmics Anticoagulation & with warfarin Rate control	Bodily contact prohibited
Atrial Flutter	Often WNL	Palpitations	Monitor	RFA Antiarrhythmics Rate control & Anticoagulation	Bodily contract prohibited prohibited with warfarin
Ventricular Preexcitation (WPW)	Short PR Delta waves	Asymptomatic	Monitor ECG EPS	No therapy RFA if high risk	Consider EPS to risk stratify
Ventricular Preexcitation (WPW)	Short PR Delta waves	Palpitations	Monitor ECG	RFA Antiarrhythmias	No restrictions after 3-6 month without symptoms
AVNRT	Normal	Palpitations	Monitor EPS	RFA Antiarrhythmics	No restrictions after 3-6 months without symptoms

APC=atrial premature contractions; AVNRT=atrioventricular nodal re-entrant tachycardia-within normal limits; ECG=Electrocardiogram; EPS=electrophysiology study, RFA=radiofrequency ablation; WNL=within normal limits; WPW=Wolf-Parkinson-White Syndrome (Modified from Ref 3).

VENTRICULAR ARRHYTHMIAS

In evaluating the athlete with ventricular arrhythmias, the nature of the ventricular arrhythmia, the presence or absence of symptoms and the presence or absence of structural heart disease are critical factors in determining the need for therapy or restriction from athletic activity. Most commonly, individuals at risk for sudden cardiac death have underlying structural heart disease, with hypertrophic cardiomyopathy being the most common. Ventricular arrhythmias in the setting of common types of congenital heart disease such as arrhytmogenic right venrticular dysplasia, [12] Epstein's anomaly, tetralogy of Fallot, or other valvular heart disease also are markers for a higher risk for sudden cardiac death. Additionally, idiopathic dilated cardiomyopathy and acute myocarditis are accepted as conditions associated with a higher risk of life threatening arrhythmic events

Premature ventricular contractions (PVCs) in the athlete are felt to be common and rarely cause sufficiently severe symptoms to warrant therapy. In the absence of any congenital or acquired structural heart disease, there is no increased risk of life threatening arrhythmias. Routine evaluation of athletes with premature ventricular contractions includes history, physical examination, EKG as well as echocardiogram. If there is no evidence of hypertrophic cardiomyopathy, arrhythmogenic right ventricular dysplasia, anomalous origin of the coronary arteries, coronary artery disease, dilated cardiomyopathy or myocarditis or long QT syndrome, the PVCs are benign and are not a marker of risk for sudden cardiac death (SCD). When there is no underlying structural heart disease, therapy with beta blockers may decrease symptoms with or without reduction in the frequency of PVCs. Drug therapy with other antiarrhythmic agents generally is not needed unless the symptoms are sufficiently severe and persistent on beta blocker therapy. In the absence of symptoms, and in the absence of structural heart disease, no therapy is needed.

Without structural heart disease, nonsustained ventricular tachycardia does not indicate any additional risk of more serious or life threatening cardiac arrhythmias or sudden cardiac death The evaluation and management of athletes with nonsustained ventricular tachycardia is similar to those with premature ventricular contractions with a few notable exceptions. In those athletes with nonsustained polymorphic ventricular tachycardia, there may be a higher risk for life threatening ventricular arrhythmias. Beta blocker therapy is considered to be the first approach to these individuals and restriction from athletic activity should be considered. When an athlete has had a prior episode of sustained ventricular tachycardia or prior episodes of ventricular fibrillation, a thorough evaluation of cardiac status with history, physical, EKG, echocardiogram and selected use of stress test, cardiac magnetic resonance imaging (MRI), cardiac catheterization, and electrophysiological (EP) evaluation is warranted. Subsets of sustained ventricular tachycardia originating from the right ventricular outflow tract or other regions of the right or left ventricle (idiopathic ventricular tachycardia) typically occur in the absence of any identifiable structural heart disease. In this setting, there is extremely low risk for sudden cardiac death. Programmed ventricular stimulation, sometimes supplemented by isoproterenol infusion generally can induce the sustained arrhythmia. When induced, mapping the site of origin and cure with radiofrequency ablation is generally possible. As with athletes who undergo ablative therapy for supraventricular arrhythmias, these athletes can return to athletic competition after a period of approximately 3 months free of arrhythmias.

In athletes presenting with sustained ventricular tachycardia or prior episodes of resuscitated ventricular fibrillation there is generally some type of underlying structural heart disease. In the athlete under age 35, hypertrophic cardiomyopathy, anomalous origin of the right coronary artery, right ventricular dysplasia, or other congenital abnormalities typically serves as a substrate for these arrhythmias. By contrast, in athletes older than 35 years of age, over 80% have underlying coronary disease with an ischemic basis for their arrhythmias. In these athletes, the implantable cardioverter defibrillator (ICD) has been shown to provide superior protection for sudden death prevention to antiarrhythmic therapy particularly in the setting of underlying coronary artery disease. Once an athlete with underlying structural heart disease has had episodes of sustained ventricular tachycardia or fibrillation, and receives an ICD, competitive sports are prohibited by current guidelines.

Exercise has been noted to exacerbate arrhythmias in many types of structural heart disease that predispose to sudden cardiac death.[13] In cases of sustained monomorphic ventricular tachycardia associated with right ventricular dysplasia and ventricular tachycardia of right ventricular outflow tract origin occurring in the absence of structural heart disease, exercise frequently exacerbates the arrhythmia. In patients with anomalous origin of the coronary arteries and congenital long QT syndrome, life threatening ventricular arrhythmias frequently occur with exertion or immediately afterwards. With the most common form of underlying structural heart disease, hypertrophic cardiomyopathy, arrhythmias are induced by exertion in approximately one-half of individuals. In patients with idiopathic ventricular fibrillation, approximately 15% of individuals have their cardiac arrest in the setting of intense physical exertion. In addition, in the setting of underlying coronary artery disease, there is a multi-fold increase in the frequency of sudden death related to exertion.

WORKUP FOR VENTRICULAR ARRHYTHMIAS

Because sudden death rarely occurs in an athlete with a structurally normal heart, an assessment for structural heart disease is of paramount importance in the evaluation of athletes with possible cardiac symptoms (Table 3). A tran-

TABLE 3. Diagnosis and management of ventricular arrhythmias in athlete

Condition	Symptoms	ECG	Diagnosis	Treatment Options	Competitive Athletics
VPCs	Palpitations	NL	Monitor	Reassurance	No restrictions
NSVT	Palpitations	Often NL	Monitor	Assess for SHD If no SHD, reassure If SHD, further Evaluation is needed	No restrictions if no SHD. If SHD present, see table
VT/VF	Palpitations	Can be NL	Monitor	RFA if no SHD ICD or AAD If SHD present	No restrictions if no SHD and cure by RFA Restricted to low intensity sports for all others

Recommendations for competitive athletics are based on the 26th Bethesda Conference. ECG=electrocardiogram; VPC=ventricular premature contractions; NL=normal; SHD=structural heart disease; BB=beta-blockers; NSVT=nonsustained ventricular tachycardia; VT=ventricular tachycardia; VF=ventricular fibrillation; RFA=radiofrequency ablation; ICD=implantable cardioverter defibrillator; AAD=antiarrhythmic drugs. (Modified from Ref. 3)

sthoracic echocardiogram and resting electrocardiogram is generally a sufficient workup for structural heart disease, although individuals with anomalous coronary arteries will not be found. Individual with risk factors for coronary artery disease (older age, male gender, high cholesterol, family history of early coronary artery disease, diabetes, hypertension, and smoking) should, in addition undergo an exercise stress test. In the absence of structural heart disease the only symptoms concerning for an increased risk of sudden death are patients with resuscitated sudden death and no structural heart disease, patients with syncope and a family history of sudden death, and patients with syncope at peak exercise. In most patients, structural heart disease can be diagnosed by an electrocardiogram, an echocardiogram, and an exercise stress test in patients over 35 years of age. However, the diagnosis of anomalous coronary arteries can be difficult and may require cardiac catheterization.

Genetic

Because many of the cardiac diseases that predispose to sudden death in athletes are genetic in origin, the family medical history is also important. These genetic disorders include hypertrophic cardiomyopathy, long QT syndrome, coronary artery disease, the Brugada syndrome and even some types of arrhythmogenic right ventricular dysplasia and idiopathic dilated cardiomyopathy. Therefore, the presence of early sudden death or hereditary cardiac abnormality in the family of an athlete should prompt a thorough cardiac workup regardless of the presenting symptoms. At the current time genetic screening is evolving rapidly and may become commercially available to assist in the diagnosis.

Invasive electrophysiologic evaluation

An invasive electrophysiologic evaluation is often performed in patients with coronary artery disease in order to ascertain the risk of future arrhythmias and whether patients should receive treatment for ventricular arrhythmias. These studies require transvenous catheters to be placed in the right ventricle and atrium and electrical stimulation of the heart. In patients with coronary artery disease, electrophysiologic evaluation is of reasonably high sensitivity (90-95%) and specificity (95%) for the induction of monomorphic ventricular tachycardia. However, the significance of induced nonsustained ventricular tachycardia and ventricular fibrillation is less well established. In patients with arrhythmogenic right ventricular dysplasia the sensitivity of electrophysiologic evaluation is 70 to 80%, and the specificity although thought to be reasonable, is not clearly defined.

Unfortunately, the sensitivity and specificity of an invasive electrophysiologic evaluation is low in patients with hypertrophic cardiomyopathy, idiopathic dilated cardiomyopathy, long QT syndrome, and congenital heart disease, which are the most common in young individuals dying suddenly, as well as in individuals with normal hearts. Thus, electrophysiologic evaluation in athletes with coronary artery disease and arrhythmogenic right ventricular dysplasia is a useful tool to evaluate symptoms, prognosticate future events, and guide treatment. However, in athletes with cardiac conditions other than coronary artery disease, an electrophysiologic evaluation cannot be relied upon to prognosticate or guide therapy.

TREATMENT

Athletes with Documented Ventricular Arrhythmias

Treatment options for ventricular arrhythmias in athletes include antiarrhythmic agents, ablation, and implantable cardioverter defibrillators (ICDs) (Table 4). In patients with life-threatening ventricular arrhythmias, ICDs have been shown to be superior to antiarrhythmic drugs. In addition

TABLE 4. Ventricular Arrhythmias in Different Forms of Structural Heart Disease

Condition	Symptoms	ECG	VT morph	Treatment Options	Competition Athletics
Idiopathic	Palpitations LH, syncope	NL	RB, left axis	RFA	No restrictions 3 months after RFA
Idiopathic RVOT, VT	Palpitations LH, syncope	NL	LB, inf axis	RFA	No restrictions 3 months after RFA
HCM	Palpitations Syncope SCD	Q's-ant		BB AAD Myomectomy ICD	Only low intensity
ARVD	Palpitations Syncope SCD	T=inv ant RBBB Epsilon wave	LB, inf axis	Sot or amio ICD RFA	Only low intensity
CAD	Palpitations Syncope SCD	Infarcts Ischemic ST	RB or LB ICD	AAD	Only low intensity
LQTS	Palpitations Syncope SCD	Long QTc	Torsades de pointes	BB PPM ICD	Only low intensity
Anomalous CAD	SCD	NL	VF	CABG	No restrictions after CABG

=electrocardiogarm; VT=ventricular tachycardia; LV=left ventricle; LH=lightheadedness; NL=normal; RB=right bundle; RFA=radiofrequency ablation; RVOT=right ventricle outlow tract; VT=ventricular tachycardia; LB=left bound; HCM=hypertrophic cardiomyopathy; SCD=sudden cardiac death; BB=beta-blockers; AAD=antiarrhythmic drugs; ICD=implantable cardioverter defibrillator; ARVD=arrhythmogenic right ventricular dysplasia; RBBB=right bundle branch block; LBBB=left bundle branch block; sot=sotalol; amio=amiodarone; PPM=permanent pacemaker; CABG=coronary artery bypass surgery. (Modified from Ref. 3)

antiarrhythmic agents are increasingly being feared as the potential for proarrhythmia is realized. In general, any athlete with a documented ventricular arrhythmia has a potentially life-threatening arrhythmia. The exception would lie in those patients with no underlying heart disease and idiopathic left ventricular tachycardia or right ventricular outflow tract ventricular tachycardia. In these patients without structural heart disease, cure rates with radiofrequency ablation approach 90%. These athletes with no structural heart disease and a successful ablation of ventricular tachycardia that is without recurrence for 6 months are allowed to participate in athletics.

ATHLETES WITHOUT DOCUMENTED VENTRICULAR ARRHYTHMIAS

Athletes with structural heart disease known to put them at risk for sudden death, but without ventricular arrhythmias or symptoms of ventricular arrhythmias (i.e. syncope or presyncope) are a more difficult group to advise and treat. If they have documented hypertrophic cardiomyopathy, idiopathic dilated cardiomyopathy, arrhythmogenic right ventricular dysplasia or congenital heart disease, competitive athletics of moderate and high intensity are generally prohibited. In the athlete with hypertrophic cardiomyopathy, risk factors for sudden cardiac death include family history of sudden death presentation with syncope or sudden death, inducible ventricular arrhythmias and septal thickness >30 mm. Spontaneous nonsustained ventricular tachycardia as a risk factor for sudden death is controversial and may not be an independent risk factor. In hypertrophic cardiomyopathy patients with one risk factor for sudden death, implantation of the ICD can be considered. With more risk factors, the ICD should be more strongly advised.

Arrhythmogenic right ventricular dysplasia patients with inducible ventricular tachycardia at ventricular stimulation, drug failure during serial testing, irregular antiarrhythmic drug intake, previous cardiac arrest, and presence of late potentials on signal averaged electrocardiograms are at increased risk for ventricular arrhythmias. Many patients with arrhythmogenic right ventricular dysplasia will meet one of these criteria. Risk factors for sudden cardiac death include presentation with syncope and markedly depressed right ventricular function. Furthermore, arrhythmogenic right ventricular dysplasia is a progressive disease and the patient's

risk of sudden cardiac death may increase with time. In the patient with arrhythmogenic right ventricular dysplasia options for treatment include antiarrhythmic agents, ablation, and ICDs. As arrhythmogenic right ventricular dysplasia is a likely progressive disease, treatments effective at one point may become ineffective later in time. Radiofrequency ablation is often acutely successful but recurrences of ventricular tachycardia are common and sudden death is described. Current opinions of the optimal treatment of these patients vary, but in those of sufficiently high risk for sudden death an ICD should be considered. Moderate and high level competitive athletics are contraindicated, in view of the frequent provocation of arrhythmias with exercise.

Although some studies have shown an increased risk of sudden death in idiopathic dilated cardiomyopathy in patients with nonsustained ventricular tachycardia and inducible arrhythmias, the most important factor is the degree of left ventricular dysfunction. The presence of syncope may also increase the risk of sudden death. In the patient with idiopathic dilated cardiomyopathy, class 1 antiarrhythmic agents are rarely effective and are possibly toxic and should generally be avoided. Beta-blockers are probably effective and should be prescribed to all patients that can tolerate them. Amiodarone may have the highest efficacy of all antiarrhythmic agents for life-threatening ventricular arrhythmias in this condition. In asymptomatic patients with idiopathic dilated cardiomyopathy, therapy other than beta-blockers and ACE-inhibitors are not generally recommended. However, in the athlete with syncope and idiopathic dilated cardiomyopathy, an ICD should be considered. In those patients with idiopathic dilated cardiomyopathy and spontaneous sustained ventricular arrhythmias competitive athletics of moderate or high intensity should be avoided.

Athletes with the long QT syndrome are at increased risk of sudden death if there is a family history of sudden death, if they personally have had syncope or sudden cardiac death or if bradycardia is present. Beta-blockers should be an initial therapy in all patients with the long QT syndrome. Permanent pacemakers should be used when bradycardia occurs with beta-blocker therapy or when recurrent symptoms occur on beta-blockers. More controversial treatments include ganglionectomy and ICD therapy.

In athletes with coronary artery disease, risk factors for sudden cardiac death include >10 premature ventricular contractions/hour, nonsustained ventricular tachycardia, inducible ventricular tachycardia, low left ventricular ejection fraction, abnormal signal averaged ECG, and decreased heart rate variability. The MADIT study has shown that patients with low left ventricular ejection fractions and inducible ventricular arrhythmias have an improved survival when treated with ICDs. However, patients with coronary artery disease and preserved ejection fractions do not generally require ventricular stimulation or ICDs in the absence of syncope or resuscitated sudden death. Indeed, athletic participation can reduce the risk of recurrent myocardial infarctions and possibly sudden death. In athletes with coronary artery disease and ventricular arrhythmias only low intensity competitive athletics are permitted.

In asymptomatic athletes with anomalous coronary arteries, a maximal exercise stress test is advised. However, since sudden death occurs without symptoms in 50% of the individuals, a normal exercise test does not automatically confer low risk. Most of the deaths occurring with anomalous coronary arteries occur with exertion. If an athlete has been diagnosed with anomalous coronary arteries, surgical therapy should be considered. If revascularization is undertaken and the patient has no inducible myocardial ischemia a return to competitive athletics can be allowed.

AUTOMATIC EXTERNAL DEFIBRILLATORS

Automatic external defibrillators, first introduced for clinical use in 1979, have revolutionized the approach to out of hospital cardiac arrest, with clear implications for their use at athletic events.[14] An AED is a lightweight portable device containing a battery, capacitors, and circuitry design to analyze cardiac rhythm and prompt the operator when a shock is indicated for a life threatening ventricular arrhythmia. Manufacturers were challenged by the American Heart Association to develop a device so reliable in its sensitivity and specificity for life threatening heart rhythms and so easy to use that fear of misuse and inappropriate shocks would be unfounded. Reductions in size, weight, cost and maintenance by manufacturers have allowed these devices to be incorporated into the American Heart Associations Public Access to Defibrillation Programs

The concept of Public Access to Defibrillation promotes expansion of the role of the defibrillation to both the minimally trained first responder, such as the trainers, coaches, police officers, fire fighters, security guards and flight attendants, and trained lay persons who are present at the time of arrest. It also promotes the placement of the AED in such areas as sports arenas, athletic fields, airports, convention centers, casinos, shopping malls, large office buildings and airlines. It is possible that the AED will become as common place as a fire extinguisher in the future. There is a growing body of evidence to suggest that the public access the defibrillation using the AED has resulted in greatly improved survival for sudden cardiac arrest

Cardiac arrest in athletics may occur in a variety of settings that should be considered in designing a response system for these emergencies. Most of the reported episodes of sudden cardiac arrest in athletes occur in those participating in football or basketball. Accordingly, a logical focus of the AED use of a response system would be on these two sports. In addition, it is well appreciated that more than half of the emergencies occurred during training sessions as opposed

to the actual competition. It is widely accepted that the team athletic trainers, rather than team physicians are most likely to be present during these sessions. Approximately 1/3 of episodes of sudden cardiac arrest occur in athletes during or immediately after a team competition when a physician is most likely to be present. Importantly, cardiac emergencies also frequently develop amongst spectators attending the athletic event. In fact, the probability of use of an AED and frequency of such emergencies is likely greater in the spectators and other attendees than it is in the athletic participant. An AED needs to be incorporated into a formal response system that adheres to the principles in the chain of survival promoted by the American Heart Association. This concept of chain of survival is straightforward with early activation of emergency medical care, early CPR, early defibrillation using the AED and early advanced life support

Multiple studies have demonstrated that EMTs and paramedics can effectively and safely use manual defibrillators. A small number of studies have shown that use of the AED was safe and effective. There has been a suggestion of a possible survival advantage with the use of AED. Multiple studies documented improved survival with addition of EMT defibrillation using the AED. These multiple studies also have documented that use by emergency personal such as police officers, fire fighters and trained first responders dramatically reduce the time to defibrillation and enhance the survival in selected.

Clinical experience with AEDs at public schools and athletic facilities are currently being evaluated. However, efficacy and cost-effectiveness data are not yet available. National programs which are designed to support AED use in schools and athletic events include Project Adam in Wisconsin. This program has already resulted in placement of AEDs in 10-15% of the regions high schools and has reportedly already saved a life. The Gregory Mayor Fund of Pennsylvania has resulted in placement of AEDs within many high schools in the county. In addition, legislation has resulted in funding for placement of up to two AEDs per high school statewide (Pennsylvania House Bill 996). In Boston, Massachusetts, Operation Heartbeat has provided AEDs for one public high school per district. In all of the programs, AEDs will be incorporated into a coordinated emergency response system that includes training for initiation of bystander CPR, and immediate activation of bystander cardiopulmonary resuscitation. While more cost effectiveness data is needed, athletic organization, educational institutions, coaches, sports medicine physicians, trainers and emergency personal should give serious consideration to incorporation of AEDs into an emergency response system.

CONCLUSION

Sudden cardiac death in the athlete is rare, albeit tragic, event.[1-23] The majority of deaths are due to underlying structural heart disease. In young athletes (<35 years old), the leading causes are hypertrophic cardiomyopathy, right ventricular cardiomyopathy, and congenital coronary artery anomalies, while less common are primary electrical diseases, such as the WPW syndrome or the long QT syndrome. In older athletes (over 35 years old) most deaths are due to coronary artery disease. Those with documented life-threatening ventricular arrhythmias or with underlying heart disease and high risk of developing sudden death can now be protected with an implantable cardioverter defibrillator. Cardiology Societies have put forth pre-participation screening strategies aiming at reducing these tragic events. Wider availability of the automatic external defibrillator may help save lives during sporting activities.

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