Sports Cardiology Essentials
Christine E. Lawless, MD, MBA
Editor

Sports Cardiology Essentials
Evaluation, Management and Case Studies

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Part I

Prevention of Sudden Cardiac Arrest in Athletes: Application of appropriate cardiac evaluation and treatments
Incidence

Introduction

Exceeding 300,000 cases annually, sudden cardiac death (SCD) is the leading cause of death in the USA [1, 2]. SCD is also the leading cause of death in athletes [3]. Defined as occurring within 1 h of participation in sports [4], exercise-related SCD occurs in one to five cases per one million athletes per year. Of the approximately 25 million competitive athletes in the USA, there are 25–125 documented cases of SCD per year, likely a significant underestimation [5]. The National Federation of High School Coaches estimates 10–25 cases of SCD per year in individuals younger than 30 years [6]. A study of Minnesota high schools revealed three deaths due to SCD in a period of 12 years, translating to a risk of one death per 200,000 athletes/year [6]. The overall occurrence rate of SCD in high school athletes is estimated to be 1:100,000–1:300,000 athletes/year [7]. Despite significant efforts nationally to identify the ideal mechanism for detecting and preventing these events, cardiovascular collapse as the cause of athletic fatalities in high school and collegiate athletes outnumbers death by trauma by 2:1 [8].

SCD in older athletes (>35 years of age) is most often related to atherosclerotic coronary arterial disease with myocardial infarction. However, in young athletes (<35 years of age), the majority of these cases are caused by defined and hereditary cardiovascular disorders. Cardiac electrical instability, due to these underlying pathologies, deteriorating into fatal ventricular tachycardia or ventricular fibrillation, appears to be the most common immediate cause of death [1, 5, 9, 10].
A retrospective review, by Maron, of sudden deaths of competitive athletes from 1985 to 1995 found that 134 deaths (85%) were due to cardiovascular disorders [11]. The majority of these athletes were involved in high school sports (62%), with collegiate athletes a distant second (22%), and professional athletes ranking third (7%). Of the athletes who experienced sudden death, 90% were male. From a sport-specific standpoint, 68% of these deaths occurred during football and basketball participation, likely a population-participation-based statistic, but also potentially an intensity-related phenomenon. Ninety percent of these athletes collapsed during or immediately after a training session or competition.

Unfortunately, although symptoms or family history of SCD may precede the event, most episodes of arrest are the first manifestation of disease in an “apparently healthy” individual. Many athletes are capable of exceptionally high levels of performance for long periods of time, even while harboring occult and potentially lethal cardiovascular malformations [11]. Traumatic to the family and the surrounding community, such events are typically excruciating, prompting public outcry and ever-increasing efforts by sports medicine professionals to detect, prevent, and respond aggressively to these rare but catastrophic events.

To further complicate the issue, one response to this difficult problem has been to focus significant energy and resources on the preparticipation examination. This, however, has yielded modest benefits at best [12]. The ability of the preparticipation examination alone to optimally identify all athletes with these underlying cardiovascular predispositions to sudden cardiac arrest has been limited, and noninvasive cardiovascular screening techniques have not yet been adopted on a broad scale in the US [7, 13–16]. Particularly, the relative rarity of actual events, approximately 1:200,000 US athletes/year, demands an extremely sensitive and specific screening test, and clear indications for definitive action, to be deemed a “cost-effective” intervention [17].

Another approach has been to aggressively implement emergency response protocols to optimally respond to a sudden cardiac arrest event [18]. While there has been significant success in the development of nationally standardized recommendations in emergency action planning [19], particularly from the National Athletic Trainer’s Association [20], the success of defibrillation in the young athlete may be lower than that of defibrillation in the general population [21]. We discuss these studies later in the chapter.

Ultimately, successful efforts to save every young athlete will require further understanding of the optimal preparticipation evaluation, considerable expanded research in the detection and management of the various underlying cardiac pathologies commonly associated with arrest in active youth, and continued pursuit of optimal response to arrest episodes.

**Causes**

Greater than 20 cardiac pathologies have been identified as causes of SCD in athletes. In a retrospective review from 1985 to 1995 of SCD in US athletes [11], Maron
reported pathological findings at autopsy revealing a predominance of hypertrophic cardiomyopathy (HCM) (26%) and aberrant coronary arteries (13%) as the underlying cause of death. Only 18% of these patients had symptoms attributed to the cardiovascular system in the preceding 36 months prior to death (i.e., chest pain, exertional dyspnea, syncope, or dizziness). Further, 115 of these athletes had standard preparticipation examinations performed. Of these, only 3% were suspected of having cardiovascular disease and only one athlete (0.9%) had the correct cardiovascular abnormality identified (see Fig. 1.1) [11].

The lack of standardization in the quality and type of preparticipation physical examination performed on these athletes as well as the high rate of unrecognized cardiovascular disease illustrates the need for health care professionals, particularly in the sports medicine community, to explore adjunctive means for preventing and/or treating SCD [23].

SCD in older athletes (>35 years old) is most often related to coronary artery disease [4], with the incidence of SCD increasing with age [24]. Including this older population of vigorous exercisers, estimates of SCD incidence approach 1:15,000–1:18,000 [25]. Most of these deaths, of young and old athletes, are thought to be related to electrical instability leading to ventricular tachycardia and, eventually, ventricular fibrillation [4, 26].

### Specific Causes

#### Hypertrophic Cardiomyopathy

The single most common cardiac abnormality leading to SCD in the USA in athletes is hypertrophic cardiomyopathy or HCM [11]. Responsible for approximately 26% of all SCD events in US athletes, it is the most significant cardiac pathology with which we as a sports medicine community struggle. Affecting approximately one in every 500 adults in the US, it seems to have a fairly equal male:female genetic distribution ratio.
Less prevalent in the athletic population, likely due to selection and some screening practices, the mortality rate is 6% per year in children and adolescents with HCM, and 1–3% per year in adults [12].

Genetically, 11 mutant genes with over 200 specific mutations in these genes have been identified and implicated in clinical HCM [27]. Mutations involving the beta-myosin heavy chain, the myosin-binding protein C, and the cardiac troponin T are responsible for approximately 50% of the population HCM. Of these mutations, the beta-myosin heavy chain mutations and the troponin T mutations tend to be of the highest risk, while the cardiac myosin-binding protein C and alpha-tropomyosin mutations tend to be of lower risk. HCM is generally inherited via an autosomal dominant pattern. Pathologic disease of the myocardium typically develops in adolescence in conjunction with rapid growth of the body [27]. Only 60% of all individuals with HCM have an affected first degree relative, due in large part to the occurrence of spontaneous mutations [28].

Pathologic evaluation of the heart with HCM reveals a larger than normal heart with a particularly enlarged left ventricle [27]. Total left ventricular mass is increased without compensatory dilation of the chamber; thus, ventricular filling is decreased in diastole. Thickness of the ventricular septum is typically markedly increased from 15 to 50 mm, with less than 13 mm considered normal [5, 27]. The marked septal hypertrophy is particularly asymmetric when compared to the left ventricular free wall, causing the characteristic left ventricular outflow tract obstruction [22]. Also commonly seen in the setting of HCM is significant anterior motion of the mitral valve during systole [29]. Microscopic pathology reveals abnormal small arteries, with “myocardial disarray” and a complex, disorganized arrangement of the myocytes with interstitial fibrosis [30].

On clinical evaluation, HCM can be asymptomatic [31], or patients may present, with exertional chest pain, shortness of breath, dizziness, or syncope with exertion. On examination, one may find a laterally displaced, enlarged point of maximal impulse. A systolic murmur may be heard, typically at the left lateral sternal border, and is characteristically increased by maneuvers that decrease preload. The Valsalva maneuver or a squatting-to-standing position change should make the murmur audibly louder. This is in contrast to more flow-dependent benign murmurs, which will often become quiet or disappear with these maneuvers.

ECG testing is fairly sensitive for HCM, likely greater than 90% [31, 32]. Characteristic findings include left ventricular hypertrophy, abnormal axis, q waves, and ST-T wave changes [22, 31, 32]. The ECG, however, can be normal, particularly in cases of nonobstructive or less obstructive disease. In cases of nonobstructive HCM, the sensitivity of ECG drops to approximately 75%. Asymmetric septal hypertrophy, systolic anterior motion of the mitral valve, abnormal diastolic filling, and often, diminished left ventricular cavity size can all be seen on echocardiogram [16, 33, 34]. Cardiac MRI, particularly with contrast, can often identify the abnormal myocardium if the diagnosis is in doubt. Genetic testing is available but can cost up to $4,500 [31, 35]. (See Chapter 5 for further information on role of genetic testing.)
Commotio Cordis

Responsible for 20% of all SCD in US athletes, commotio cordis is particularly concerning in high-velocity ball sports [36, 37]. A sudden forceful impact to the chest wall during ventricular repolarization can elicit electrical instability leading to asystole or ventricular fibrillation [37]. The mean age of victims is 13 years of age. No symptoms are present prior to the event [37]. No underlying cardiac abnormalities are found at autopsy. Unfortunately, although rapid defibrillation is the treatment of choice, defibrillation attempts are often unsuccessful, due to the fact that some athletes deteriorate immediately into asystole [21, 22]. The resuscitation rate is 15%, even with rapid response. A commotio cordis registry is tracking injuries and response, and hopefully it will provide important information in furthering our understanding of this deadly condition (see Chapter 14).

Coronary Arterial Abnormalities

The second leading cause of nontraumatic SCD is actually a group of disorders of the coronary arteries [1, 4, 18, 22, 34, 38, 39]. Anomalous origin of the left anterior descending from the right sinus of Valsalva is considered the most lethal of a variety of congenital anomalies of the coronary vessels [11]. Tunneled coronaries, myocardial bridging, single coronary arteries, and others have been found to contribute to transient myocardial ischemia during intense activity, that then induces malignant arrhythmia, often ventricular tachycardia/ventricular fibrillation [10, 40].

Most often asymptomatic, some one-third of athletes will experience angina, syncope, or exertional dyspnea prior to an SCD event [41]. In one study in the USA and Italy, 10 of 12 athletes with underlying coronary abnormalities had symptoms prior to death [42]. Physical examination is typically unremarkable [26].

The ECG is typically normal [7, 12, 31, 40], the resting echocardiogram is typically normal [23, 42], and even exercise treadmill stress testing is often normal [4, 42]. All nine of the athletes in the above study who had ECGs had normal findings, including six individuals who had completed exercise stress testing in addition to routine ECG [42]. One may see segmental wall motion abnormalities on stress echocardiography, but definitive diagnosis is often made by coronary angiography, computed tomography of the coronary arteries, or magnetic resonance imaging [31, 34, 42] (see Chapter 3 and 15).

Myocarditis

Inflammation or infection in the myocardial tissue can lead to electrical instability and SCD in athletes. Involvement of the conduction system can also lead to fatal complete heart block [10, 26, 34]. Typically related to Coxsackie B virus infection (approximately 50%), cardiac inflammation can be due to a great number of organisms [45, 46]. Affected individuals may present with chest pain, dyspnea, syncope,
and/or dizziness, but they may also present with more systemic symptoms related to the infectious process [45]. Fever, chills, nausea, vomiting, upper respiratory symptoms, diarrhea, and/or sore throat may be present. Often, however, an athlete’s presenting symptom is the SCD event [5, 18, 21, 46].

Clinically, athletes at risk may be identified by symptoms of infection as mentioned above or of cardiac involvement with chest pain, dyspnea, syncope, or dizziness [46]. On exam, they may have evidence of congestive heart failure such as distended neck veins, a laterally displaced enlarged PMI, audible S3 gallop, rales, peripheral edema, and/or hepatojugular reflux [34, 45, 46].

Chest X-ray is typically normal, but it could reveal cardiomegaly or pulmonary edema [45]. ECG may indicate myocarditis with ST segment elevation, but sensitivity is relatively low and variable (10–50%) [34, 45, 46]. Echocardiogram may reveal hypokinesis, depressed ejection fraction, or may be normal. Cardiac MRI with contrast may be helpful in demonstrating myocardial inflammation [46].

Definitive diagnosis depends on myocardial biopsy [46]. Pathologic analysis typically demonstrates myocardial inflammation but may reveal only idiopathic myocardial scarring [46, 47]. It may be this scarring that generates the arrhythmogenic focus in some asymptomatic athletes with SCD events [5, 10, 45]. Treatment for myocarditis is geared toward the clinical cardiac disease, treating any underlying heart failure, and protecting from excessive stress during the active phase of the disease. Echocardiography, electrophysiology, and stress testing may be utilized to document resolution of the condition and lack of arrhythmogenic potential [1, 5, 26, 45].

Marfan’s Syndrome

Accounting for only 2% of SCD in US athletes, Marfan’s syndrome is an important underlying problem for the sports physician and cardiologist to be familiar with, as it is one of the more recognizable pathologies with a largely clinical diagnosis. It is an autosomal dominant inherited connective tissue disorder occurring in approximately one in 10,000 individuals [46]. An athlete with this condition will often have a family history significant for Marfan’s syndrome. The physician’s ability to diagnose and subsequently monitor Marfanoid athletes to reduce their risk of SCD is unique among the pathologies here and warrants some attention. Marfan’s is one of the family of connective tissue disorders that leads to increased risk of progressive dilatation of the aortic root, potentially leading to dissection and/or rupture of the aorta with massive hemorrhage, pericardial tamponade, coronary artery dissection, acute aortic valve insufficiency, and congestive heart failure [46, 48].

Marfan’s syndrome is typically asymptomatic but does have characteristic morphologic/clinical findings related to the connective tissue pathology [48]. Athletes are characteristically very tall and thin. They often have disproportionately long limbs, with an arm span greater than their height and an upper body to lower body ratio more than one standard deviation below the mean, as well as long thin facies [46]. They often have an arm span greater than their height.
Hyperextension and significant overlap of the thumb and fifth digit when circumferentially wrapped around the thin wrist is also common [46, 48, 49]. Kyphoscoliosis and anterior thoracic deformities such as pectus excavatum and carinatum are often present [49].

Once identified clinically, the typical athlete with Marfan’s syndrome is evaluated via echocardiogram for dilation of the aortic root, evaluated by ophthalmology for ectopia lentis, and limited appropriately from activity, depending on the sport [49]. Athletes are then typically studied via echocardiogram every 6 months to 1 year for aortic dilatation [46, 48]. See Chapter 16 for a complete discussion of this important syndrome.

**Long QT Syndrome and Wolff–Parkinson–White Syndrome**

**Long QT Syndrome**

A reported rare (1% of all SCD episodes in US) but dramatic group of congenital ion channel defects may lead to SCD in an athlete [3]. Approximately one in 10,000 Americans have prolonged QT syndrome by current estimations [3, 50]. In this syndrome, the period of the cardiac cycle between ventricular firing and repolarization known as the QT interval is longer than normal. In men, this period is considered prolonged if the interval, after correction for rate, is greater than or equal to 440 ms. In women, the corrected QT interval is considered prolonged if it is greater than or equal to 460 ms.

Of those individuals with long QT syndrome, 60% have a positive family history of long QT or SCD [3, 50]. One-third of all patients present with symptoms referable to the disorder, with palpitations, seizures, and syncope being most common [23, 51]. Statistically, these syndromes are likely very underreported due to the inconclusive autopsy findings that are common in these disorders [50]. Because the heart is structurally normal in most patients, with the primary abnormality being a tendency toward life-threatening ventricular arrhythmias as a result of an ion channel defect, these syndromes are often asymptomatic leading up to a catecholamine surge and SCD under extreme stress [3, 9, 48].

If long QT syndrome is suspected, the ECG is almost always abnormal, displaying the prolonged QT interval [1, 3, 31, 32]. Occasionally, only an atypical U wave is seen. In the related Brugada syndrome, ECG shows incomplete right bundle branch block and ST segment elevations in the right precordial leads, with a structurally normal heart. Genetic testing may allow identification of the disease and the underlying subtype. The testing typically will detect up to 75% of the most common genetic mutations leading to prolonged QT syndrome (QT1, QT2, QT3, QT5, QT6). The three major types of testing for this condition are focused on slightly different genetic tendencies. The Comprehensive Cardiac Ion Channel Analysis provides analysis for variants in all five genes and is the most comprehensive test. Sodium Channel Analysis tests only for the SCN5A gene and is appropriate in cases of suspected Brugada syndrome. There is also a family-specific analysis looking to
the specific subtype of prolonged QT syndrome that is carried in the family genome [3, 31, 48].

This testing, however, can be very expensive and often requires repeated negotiation with the patient’s insurance company to get the test reimbursed by their insurance. Testing can be further investigated through http://www.familion.com.

Interestingly, of the four major subtypes of long QT syndrome that account for the majority of mutations, each has a different predictable trigger that precedes a SCD episode [3, 27, 31, 50]. In QT1, the common trigger is swimming. Although these athletes may tolerate an extreme level of exertion of most types, the unique cardiovascular demands of swimming typically produce a sudden death event due to malignant tachyarrhythmia, often ventricular tachycardia decompensating into ventricular fibrillation. In QT2, the most common trigger is extreme emotion. Often a frightening surprise sends those athletes with prolonged QT2 into the aberrant tachyarrhythmia and SCD. Interestingly, in QT3 the most common trigger is not activity at all, but inactivity. The malignant tachyarrhythmia seems to erupt almost spontaneously during low levels of activity. In QT4, the most common trigger of a SCD episode is a sudden malignant tachyarrhythmia precipitated by a loud noise, such as an alarm clock.

Treatment often involves therapy with β-blockers, a pacemaker, and/or implantable cardiac defibrillator, typically then refraining from activity. There are, however, a growing number of athletes, after extensive consultation and assessment, who are returning to competitive sport even with an ICD in place [31, 52]. Please see Chapter 18 and 19.

Unlike the channelopathies, Wolff–Parkinson–White Syndrome (WPW) is a result of an accessory pathway connecting the atrium to the ventricle that can promote the development of “reentrant tachycardias.” Typically manifesting as a relatively benign intermittent supraventricular tachycardia, or SVT, patients will often present with symptoms of palpitations, dizziness, or lightheadedness [26, 34, 53]. The ECG during an episode may reveal a narrow complex tachycardia. Once the rhythm is back to normal sinus rhythm, a characteristic “delta wave,” a slurred, early upstroke in the QRS complex, can typically be seen [26, 31] (see Fig. 1.2). The acute episode of SVT can often be terminated with stimulation of the vagal system. Maneuvers such as unilateral carotid sinus massage, the Valsalva maneuver,
or even facial immersion in ice water will often break the tachycardia and return the patient to normal sinus rhythm [53]. If not, often a calcium channel blocker or even adenosine can break the abnormal rhythm medically.

SCD is rare in WPW syndrome, but it can occur, particularly with rapid ventricular rates during atrial fibrillation that may degenerate into ventricular fibrillation. In cases of aborted SCD in WPW, or for patients with recurrent or severe symptoms, an electrophysiology study with radiofrequency catheter ablation of the abnormal accessory pathway will be performed [53].

**Arrhythmogenic Right Ventricular Dysplasia**

Arrhythmogenic right ventricular dysplasia (ARVD), also known as arrhythmogenic right ventricular cardiomyopathy, is a genetic disorder characterized by right ventricular enlargement and dysfunction as well as potentially life-threatening ventricular arrhythmias. The incidence of SCD in athletes with ARVD is estimated to be 0.5/100,000 persons/year [54]. Typically, the disease becomes manifest during adolescence and early adulthood, with a male predominance. ARVD is the most common pathological finding in athletes who have died suddenly in the Veneto region of Italy, accounting for approximately one-fourth of fatal events. The identification of ARVD as the cause of SCD in the USA is much less frequent, with an estimated incidence of 3% [46].

ARVD is a genetic disorder with autosomal dominant transmission in about one-third of cases. In many cases it is recognized to be a desmosomal disease caused by defective cell adhesion proteins such as desmoplakin, plakoglobin, and others [55]. There is also a mutation in the cardiac ryanodine receptor that has been identified as the cause of ARVD in other patients. Progressive myocyte death occurs with fibro-fatty infiltration. Thinning of the right ensues occurs along with localized aneurysmal dilatation. The disease affects the right ventricle primarily, with a much lower incidence of left ventricular dysfunction.

Athletes with ARVD have a 5.4-fold higher risk of dying during competitive sports than when they are sedentary [54]. The reasons for this observation are not known but may relate to increases in right ventricular afterload during exertion along with denervation hypersensitivity to catecholamines that may lead to an increase in ventricular arrhythmias during exercise.

Athletes with ARVD may present with palpitations or syncope, but others may be asymptomatic when first evaluated. Physical examination is usually normal unless extensive disease and right ventricular failure is present. The typical ECG abnormality in an individual with ARVD is T-wave inversions in the right precordial leads beyond V1. Ventricular arrhythmias, including premature ventricular beats and ventricular tachycardia, are frequent and usually have a left bundle branch block morphology. Signal-averaged electrocardiography is typically abnormal as well. The diagnosis may be confirmed by noninvasive imaging studies such as echocardiography, angiography, or magnetic resonance imaging. In some cases, endomyocardial biopsy is used to demonstrate fibro-fatty infiltration of the right
ventricle. The diagnosis of ARVD in an athlete would lead to disqualification from sports participation, given the high risk of SCD during competition. ICDs are often implanted in symptomatic individuals.

**Immediate Treatment**

Following a thorough preparticipation exam, the next step in prevention of SCD is the implementation of a well thought out emergency action plan (EAP). Careful planning and consideration must be taken into account when developing the EAP. Several factors must be considered including, but not limited to, the physical facilities, emergency equipment requirements, designated personnel, communication strategies, and the local hospital settings. Because of its high participation levels and variety of venues, athletics has become a key EAP target, not only for athletes, but for coaches, staff, and spectators as well [21, 56]. The National Athletic Trainers’ Association (NATA) and the National Collegiate Athletic Association (NCAA) have developed statements and positions regarding the EAP and its utilization for sporting events [20, 56].

The following components have been identified as fundamental to the organization and development of a comprehensive EAP [20]:

(a) **Personnel** – mandating personnel trained in cardiopulmonary resuscitation (CPR), AED, and first aid. It is imperative to have all key personnel who are directly involved with the athletes trained in emergency care. Key personnel may include coaches, athletes, administrators, equipment managers, and officials. The implementation of the EAP is not limited to only those who are certified in emergency care. Personnel not trained in emergency care can be instrumental in coordinating communication, directing emergency medical response teams to the correct location, and in helping to maintain the area around the emergency situation. These individuals need to be accounted for in your EAP.

(b) **Equipment** – Having the appropriate equipment on-site, or nearby, assists in the smooth implementation of the EAP [18]. One of the most essential pieces of emergency equipment is the Automated External Defibrillator (AED) [57]. Access to an AED is critical in many SCD cases and can make the difference between saving or losing a life [18, 20, 21]. Other equipment needed for CPR would include masks and oxygen supplies. Personnel participating in the EAP must be familiar with and trained to use the necessary equipment [18].

(c) **Communication** – A communication device could easily be listed in the equipment section as the most essential piece of emergency rescue equipment. Access to communications in the event of an emergency including SCDS should be the first step in initiating your EAP. The EAP should clearly identify where the closest land line is located, who should have cell phones on site, and/or who is in charge of radio communication. In the event of utilizing radio communication,
the plan must be clear on the duties of the person receiving the emergency message and how they should proceed as well. It is important that the person being identified to make the emergency call know the correct information to give on a 911 call and to make sure they are calm and listening to the dispatcher so that the information is clear and action can be taken quickly. Key elements of a 911 call include the nature of emergency, the patient’s symptoms, care being given currently, approximate age of patient, and any medical history you can ascertain from the patient, or those involved in his/her care. Specifically, concern regarding a cardiac event must be relayed to the dispatcher as, often, it is possible to expedite AED arrival to the scene, even prior to full EMS response.

(d) Venue – The EAP should be specific for each venue in which sporting activities take place. For example, in an intercollegiate athletics program, you will need to have an EAP for each sport and for each site in which athletes may compete or practice. Even when practicing at a site for a short period of time, it is imperative to have a plan in place for an emergency. Items that should be considered for each venue include communication (are there any land lines for telephone), access to the field/court, the location and number of gates around the venue that may require keys in order to unlock them to allow emergency access, and the location of the nearest AED. Hospital access from the site is another consideration and should be included in the plan.

(e) Emergency Care Facilities – Access to the emergency room facilities and personnel are additional key components to a comprehensive EAP. It is important to communicate beforehand with the emergency facility closest to your venue and to establish how information can best be relayed to these emergency providers during an emergency event. Making contact with the hospital and local EMTs/paramedics can help establish improved implementation of the EAP and its efficacy when applied in an emotionally charged setting.

(f) Practice and Review – The EAP should not only be reviewed but should also be practiced at each site. The importance of practicing the EAP prior to an event cannot be overstated. A verbal review of the EAP procedures with personnel involved in the emergency plan is important, but implementing a practice run with selected scenarios for the EAP at specific venues will enhance the ultimate performance and plan. The results of these reviews should be well documented and indicate any changes in the plan.

The NATA consensus statement on Emergency Planning in Athletics is a thorough, detailed document that forms a comprehensive blueprint on customizing an EAP for any venue for athletics. The NCAA guidelines for Emergency Care and Coverage (NCAA Sports Medicine Handbook) is another source for universities and colleges to help design, construct, and implement an EAP. While the statistics show that the survival rate of a SCD is low, especially in cases of young, otherwise healthy athletes, the numbers also show that there have been successful outcomes with revival attempts. Well-planned EAP procedures play an integral part in those successful cases of survival.
AED Utilization: NCAA Collegiate Experience

In 2002, Coris et al. examined current AED access, ownership, and utilization among NCAA Division 1 athletic departments [18]. Of the 186 (61%) of 303 institutions responding to the survey, 72% (133) reported having access to AED units for their athletic programs, either through departmental ownership of the units or through the university system or local emergency medical services, while slightly more than half, 54% (101), reported departmental ownership of AED units for their programs. The mean number of total students attending the responding departments’ institutions was 16,360 (SD = 11,026.00) with a reported 498 (SD = 222) mean number of varsity athletes per institution [18].

At the time of the survey, 8.6% (16) of the 133 programs with either access to, or ownership, of AED units reported having had to use the AED in an SCD event since purchase (see Fig. 1.3).

Reported recipients of the AED interventions included 62.5% (ten) officials, fans, and athletic department staff; 18.7% (three) athletes; and 6.25% (one) events involving a department’s coach. Twelve and one-half percent (two) reported having had to use their AED units multiple times since purchase [21] (see Fig. 1.4).

Cases of AED utilization reported by athletic departments are outlined in the table above. Twenty percent of AED uses were attributed to student athletes, with 33% of utilizations for athletic department staff, and 47% for fans. Defibrillation was actually administered in 53% of AED unit applications. Time to shock was an average of 3.4 min, with average EMS response time of 8.2 min for those events without EMS on site (see Fig. 1.5).

Reported survival in this university athletic department setting for SCD was 0% for students, 75% for staff, 57% for fans, and 61% overall [21]. Eighty-eight percent of the reported AED interventions occurred during athletic events or training venues, with the exception of two events which occurred on campus locations unrelated to the athletic department. Of departments directly involved with the AED unit intervention (67%), 56% reported the AED units as easy to use, with all 16 respondents denying problems specifically with the units during the SCD event (see Fig. 1.6).

![Fig. 1.3 Sudden cardiac death in collegiate sports medicine programs – victims](image-url)
<table>
<thead>
<tr>
<th>Case #</th>
<th>Patient</th>
<th>Outcome</th>
<th>Time to Shock</th>
<th>Location</th>
<th>EMS response time</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Student-athlete</td>
<td>Head athletic trainer notified 5-10 minutes after event, defibrillator applied within 9 minutes, non-shockable, deceased</td>
<td>No shock</td>
<td>Student weight room</td>
<td>11 min</td>
</tr>
<tr>
<td>2</td>
<td>Student-athlete</td>
<td>Pt with cardiac history, reported chest pain, monitored within 30 seconds, survived, diagnosis: non-cardiac chest pain</td>
<td>No shock</td>
<td>Practice field</td>
<td>8-10 min</td>
</tr>
<tr>
<td>3</td>
<td>Student-athlete</td>
<td>Defibrillator applied within 90 seconds, non-shockable rhythm (PEA), deceased, diagnosis: hypertrophic cardiomyopathy</td>
<td>No shock</td>
<td>Campus recr</td>
<td>8 min</td>
</tr>
<tr>
<td>4</td>
<td>Coach</td>
<td>Defibrillator applied within 2 minutes, non-shockable rhythm, deceased, diagnosis: myocardial infarction</td>
<td>No shock</td>
<td>Bleachers</td>
<td>Unknown</td>
</tr>
<tr>
<td>5</td>
<td>Graduation attendee</td>
<td>Suspected MI, Defibrillator applied in 5-7 min, no shock advised, survived to hospital discharge</td>
<td>No shock</td>
<td>Gymnasium</td>
<td>10 minutes</td>
</tr>
<tr>
<td>6</td>
<td>Lecture attendee</td>
<td>Defibrillator x 3, once more by EMS, survived to discharge, diagnosis: MI</td>
<td>2 min</td>
<td>Lecture hall</td>
<td>5-10 min</td>
</tr>
<tr>
<td>7</td>
<td>Staff</td>
<td>Monitored, survived to discharge</td>
<td>No shock</td>
<td>Training room</td>
<td>7 min</td>
</tr>
<tr>
<td>8</td>
<td>Football official</td>
<td>Defibrillator applied within one minute, ventricular fibrillation noted, defibrillated x1, survived to hospital discharge, diagnosis: myocardial infarction</td>
<td>55 sec</td>
<td>Football stadium</td>
<td>Immediate, on site</td>
</tr>
<tr>
<td>9</td>
<td>Fan</td>
<td>Defibrillated x1, survived to discharge</td>
<td>≤ 5 min</td>
<td>Arena</td>
<td>Immediate, on site</td>
</tr>
<tr>
<td>10</td>
<td>Fan</td>
<td>Defibrillated x2, resumed normal sinus rhythm, survived to hospital discharge</td>
<td>≤ 2.5 min</td>
<td>Basketball arena</td>
<td>Within 5 min</td>
</tr>
<tr>
<td>11</td>
<td>Fan</td>
<td>CPR provided until EMS arrival, no defibrillation prior, patient did not survive, diagnosis: MI</td>
<td>5-10 min</td>
<td>Basketball arena</td>
<td>5-10 min</td>
</tr>
<tr>
<td>12</td>
<td>Fan</td>
<td>VF/VT, Defibrillated x3, resumed normal sinus rhythm, survived to hospital discharge</td>
<td>4 min</td>
<td>Parking area</td>
<td>Within 5 min</td>
</tr>
<tr>
<td>13</td>
<td>Fan</td>
<td>VF/VT, Defibrillated x1, resumed normal sinus rhythm, survived to hospital discharge</td>
<td>2 min</td>
<td>Unknown</td>
<td>15 min</td>
</tr>
<tr>
<td>14</td>
<td>Fan</td>
<td>Notified within minutes, Defibrillated x1, presumed VF/VT, precipitated non-shockable rhythm, did not survive</td>
<td>3 min</td>
<td>Stadium</td>
<td>5 min</td>
</tr>
<tr>
<td>15</td>
<td>Fan</td>
<td>Hockey trainer notified, EMS on site, CPR immediately, Defibrillator applied in 7-8 min due to crowd related delay, non shockable rhythm, did not survive, diagnosis: massive MI</td>
<td>No shock</td>
<td>Hockey arena</td>
<td>On site</td>
</tr>
</tbody>
</table>

Fig. 1.4  Sudden cardiac death experience in NCAA Division I Sports Medicine Programs

Fig. 1.5  NCAA Division I – EMS response time to collegiate SCD episode
Figures 1.6 and 1.7 illustrate the AED unit program utilization and maintenance policies for the subsample, which were not significantly different from the total sample of NCAA respondents. When compared with the total sample of Division I Athletic departments reporting access to or ownership of AED units (n = 133), departments reporting having experienced an SCD event were significantly more likely to report a greater number of units per department (4.5 SD = 2.7 vs. 2.84 SD = 2.4; t = −2.32, p = 0.03) at a greater per unit cost ($2,568.00 SD = $1,115.00 vs. $1,872.00 SD = $1,597.00, t = −2.21, p < 0.03) than departments without an SCD event. Five (31%) of departments reported purchasing additional AED units to expand their program since their initial implementation of their AED programs, three (18.5%) reported that they were actively attempting to fund additional units, while 50% reported no changes in their current AED programs.

Case 1

As a 20-year-old softball pitcher is making her way back to the mound during routine pitching drills, she reaches up to her head as she states she “doesn’t feel good” and then suddenly collapses face first to the turf. Her athletic trainer, witnessing her fall, rushes to her aid. There did not appear to be any trauma involved in the incident. After a log rolling maneuver, assessment of her airway revealed that she had some dirt in her mouth, but that her airway was otherwise unobstructed. There was no evidence of respiration and she was beginning to appear cyanotic peripherally, and then centrally. The EAP was activated. Following the look, listen, and feel protocol, no respiration or pulse could be found and CPR was immediately
initiated. The AED was called for and chest compressions and rescue breaths were begun by one person CPR while awaiting the AED. Upon arrival of the AED, approximately 1½ min later, chest pads were applied and a shock was advised. All were cleared and one shock was delivered. Return of color was witnessed in the athlete and gasping respirations were observed. A pulse was palpated and the patient was placed on her side in the recovery position. Two minutes later, the AED advised another shock, but the patient was alert and responsive at the time, and the AED pads were removed. After the initial shock event, she converted to a sinus tachycardia with a right bundle branch block.

EMS arrived approximately 5 min after the initial shock event. At this time, she was still responsive and found to be in a normal sinus tachycardia rhythm with a right bundle branch block. The patient was placed on oxygen, an IV was started, and she was placed on a transport gurney and subsequently taken to the nearest ER. In the emergency room, her ECG showed a mild U wave and a right bundle branch block, but it was otherwise normal. The chest X-ray was normal and her lab reports revealed mild hypokalemia. The rest of her lab reports was normal, and the patient was admitted to a cardiac care unit for observation.

No further ectopy or ventricular arrhythmia was noted. Echocardiogram was normal, cardiac MRI was normal, with normal muscle signal and normal takeoff of the coronary arteries. Given the patient’s SCD event, extensive discussion with the patient and her parents was completed, and an implantable cardiac defibrillator was placed. The patient tolerated the procedure well and was discharged to home after hospital day 3 (see Fig. 1.7).

Summary

There are significant concerns with the rare, but catastrophic case of SCD in athletes. There are a variety of conditions, many asymptomatic, which can lead to sudden death. Preparticipation examinations can identify some athletes at risk and should be done in accordance with the AHA guidelines [7] for preparticipation examination and by the recommendations for the Preparticipation Physical Evaluation endorsed by the American Academy of Family Physicians, the American Academy of Pediatrics, The American College of Sports Medicine, the American Medical Society for Sports Medicine, the American Orthopaedic Society for Sports Medicine, and the American Osteopathic Academy of Sports Medicine [17].

Many cardiovascular abnormalities will not be identified by even the most thorough preparticipation evaluation. As more data becomes available in the US population, the role of noninvasive cardiovascular screening in the large scale preparticipation examination, particularly the screening ECG, may become more clear. Further research into the underlying causes of SCD in athletes is also ongoing and should provide invaluable insights into the detection and treatment of underlying cardiac disease in athletes.
Fig. 1.7  (a) AED tracing of case 1: Ventricular fibrillation resulting in SCD. (b) Post defibrillation tracing
Finally, a robust EAP is imperative to optimally respond to the inevitable emergencies that will infrequently, but emergently, present themselves to the sports medicine team. Establishing, teaching, and practicing this plan may save lives that otherwise would be lost to this difficult collection of hidden disorders.

References


34. Koster MC. A Review of Sudden Cardiac Death in Young Athletes and Strategies for Preparticipation Cardiovascular Screening. *Journal of the American College of Cardiology* 2003;42(9):1687–1713.


