Cardiac Emergency in the Athlete

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KEYWORDS

• Cardiac emergency • Cardiac arrest • Collapsed athlete • Arrhythmia • CPR
• Cardiomyopathy

KEY POINTS

- Timely quality chest compressions and time to defibrillation are the greatest factors affecting survival in cardiac arrest.
- Initial management of cardiac emergencies includes activation of emergency medical services (EMS), early cardiopulmonary resuscitation (CPR), rapid defibrillation, and transfer to an appropriate medical center.
- Sudden cardiac arrest should be suspected in any collapsed and unresponsive athlete, and an automated external defibrillator (AED) should be used as early as possible, with CPR taking place until it can be applied.
- Underlying cardiac abnormalities that can predispose some athletes to sudden cardiac arrest include cardiomyopathies, coronary artery anomalies, and channelopathies, as well as genetic disorders such as sickle cell trait, and Marfan syndrome.
- Formal planning for an efficient response and effective care can help avoid catastrophic outcomes.

INTRODUCTION, BACKGROUND, AND PREVALENCE

Cardiac-related deaths are the leading nontraumatic cause of death in the young athlete and second leading cause overall behind accidents. Earlier estimates on the incidence of sudden cardiac death (SCD) varied widely, ranging from 1:3000 athlete-years (AY) to 1:917,000 AY but more recent studies have shown a rate of approximately 1:50,000 AY in collegiate athletes and 1:50,000 to 1:80,000 AY in high school athletes. The highest risk groups include men, black athletes, and basketball players, with Division I male black basketball players being found to have approximately 1:4000 AY risk.[1] Soccer and football players were also found to be among the highest risk athletes. These athletes are typically healthy at baseline but may have undiagnosed conditions that put them at an elevated risk for

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dangerous cardiac events, including cardiomyopathies, coronary artery anomalies (CAAs), or channelopathies. Due to the nature of the patient population involved, the social, health, and economic impacts of SCD are significant.[2] This has led to extensive research into preparticipation screening examinations and diagnostic tools to try to uncover these conditions before they lead to catastrophic outcomes. Despite this, cardiac events can and do still occur on a regular basis, and preparation and prompt evaluation and treatment are vital because planning for effective care to respond to cardiac emergencies can help avoid catastrophic outcomes.

Prompt evaluation and treatment of a cardiac arrest patient is crucial to their chances for meaningful long-term survival. The greatest factor affecting survival is the time between arrest and initial defibrillation, with survival rates of up to 74% if defibrillated within 3 minutes, whereas only 49% if defibrillated after 3 minutes,[3] and rates declining by 7% to 10% with every minute that defibrillation is delayed.[4] Timely bystander cardiopulmonary resuscitation (CPR) can also significantly improve outcomes, even in situations with prolonged EMS response time.[5]

In this article, we will discuss cardiac emergencies in athletes, with a primary focus on evaluation and management of on-the-field cardiac events. We will discuss the most common causes of sudden cardiac arrest, and the underlying pathophysiology of these disorders. We will also discuss the long-term management of athletes with these conditions, including postarrest care and return to play considerations.

APPROACH TO THE COLLAPSED ATHLETE

Case Vignette: An 18-year-old football player is seen to collapse while running a play. He is not responsive on arrival.

Overall approach: Regardless of inciting cause, this is a case of witnessed arrest until proven otherwise. The approach is the same: early recognition of an emergency, scene safety, EMS activation, early CPR, early defibrillation if indicated, and rapid transition to advanced life support.[6] It is important to be familiar with any established emergency action plan (EAP) for the venue to avoid any confusion and mismanagement of care.

Scene safety: Ensure that the environment is safe to render aid. Be cognizant of the surroundings, whether it be teammates, the crowd, vehicles, or nature (eg, lightning strikes).

Evaluation: On the initial approach to the collapsed athlete, it is crucial to follow the basic life support (BLS) sequence as recommended by the International Liaison Committee on Resuscitation.[6,7]

Management and stabilization: Initiation of CPR is recommended in the witnessed collapsed athlete regardless of whether cardiac arrest is the cause for collapse.[6] The steps are adapted from the original article as follows for a health-care provider after determining scene safety.

1. Assess for response.
2. If no response, activate EMS.
3. Check for apnea or agonal breathing and check for pulses simultaneously. Prepare to begin compressions.
   a. AED/emergency equipment should be retrieved immediately after the check by a second responder
4. If apneic or agonal breathing, immediately begin CPR. Second responder should apply pads from the AED/defibrillator when available.
5. As soon as AED/defibrillator is available, pause CPR to administer shock.

6. Immediately resume CPR after shock administration and perform 2-person CPR with the second responder.
7. Compressions to ventilation are 30:2.[a]

Compressions: Quality chest compressions are crucial for proper cardiac arrest management in the collapsed athlete. Chest compression depth and rate are the 2 most important factors when determining the quality of chest compressions. Chest compressions of 2 inches (5 cm) to 2.4 inches (6 cm) and a rate of 100 to 120 compressions/min are optimal. A pulse check of the carotid artery should be performed every 2 minutes with minimal interruption to compressions.[9] Chest compressions are known to be affiliated with sternal fractures, rib fractures, hemopneumothorax, and other traumatic injuries.

AED: On AED arrival and pad placement, a shock should be delivered if advised.

It is important to note that these steps are for a health-care provider but different for a trained or untrained layperson. Please refer to BLS guidelines.

SPECIAL CONSIDERATIONS

CPR-induced consciousness: This increasingly reported phenomenon occurs in 0.23% to 0.9% of CPR attempts.[10] During CPR, the athlete may become combative, begin groaning, or even open their eyes. However, with cessation of CPR and, subsequently, cerebral perfusion, the athlete will become unresponsive and lose pulses. There is no clear guidance on the benefit of sedation or analgesia but there are reports of fast-acting medications such as fentanyl, midazolam, or ketamine being used to help reduce the pain and trauma associated with CPR.[10] However, it is of the utmost importance that CPR be continued until the athlete has proper return of spontaneous circulation (ROSC).

Exercise-related collapse in the athlete with sickle cell trait: Of special importance, high flow oxygen in an athlete with exercise-related collapse in the athlete with sickle cell trait (ECAST) has a theoretic benefit in reducing sickling associated with hypoxia.[11] However, as mentioned above, high flow oxygen should already be administered in a cardiac resuscitation.

Lightning strike: In the event of a lightning strike, one or multiple athletes can be struck. Unlike other mass casualty events, lightning strike victims should be reverse triaged. Typically, those who are in cardiac arrest are “black tagged” and resources are directed to those who exhibit signs of life. However, in a lightning strike, a reverse triage system is used.[12] Victims that seem dead should be the first to receive treatment in the form of CPR because these victims will likely regain spontaneous automaticity or have a reversible arrhythmia.[12] If ROSC is achieved, respiratory support may be needed until spontaneous respirations return. Victims of a lightning strike that survive immediately after the event are unlikely to have significant mortality before transfer to a higher level of care. It should be noted that although this article only discusses cardiac system-related issues, there are other involved systems after a lightning strike.[12] In the solo athlete, proceed with approach to the collapsed athlete algorithm.

[a] Chest compression only CPR performed by bystanders in out-of-hospital cardiac arrest patients has been shown to be superior to standard CPR where rescue breaths are given.[8] About 13.3% of cardiac arrest victims survive with compression only CPR compared with 7.8% with standard CPR.[8] The reasoning is that ventilation interrupts proper organ perfusion. A nonrebreather mask at 15 L can be used to passively ventilate the patient while compressions can be performed without pause.

PATHOPHYSIOLOGY

There is a multitude of causes for cardiac arrest in the athlete. The following are a select number of important causes pertinent to sideline cardiac collapse. Signs and symptoms preceding collapse are reviewed as well.

HYPERTROPHIC CARDIOMYOPATHY

Hypertrophic cardiomyopathy (HCM) is a genetically induced left ventricular hypertrophy without other secondary causes. Typically asymmetric, it is often most severe in the intraventricular septum and histologically shows myocyte hypertrophy, disarray, and interstitial fibrosis. This can result in left ventricular outflow tract obstruction. Patients typically have nondilated left ventricles and a normal to increased ejection fraction and can also have diastolic dysfunction. Most HCM is asymptomatic but can present with chest pain, arrhythmia, syncope, or sudden cardiac arrest. Symptoms are secondary to myocardial hypoperfusion that results from reduced blood flow through thick-walled coronary arteries, as well as increased oxygen demand from hypertrophied myocardium. This can lead to nonsustained ventricular tachycardia, ventricular fibrillation, and cardiac arrest.[13]

ARRHYTHMOGENIC RIGHT VENTRICULAR CARDIOMYOPATHY

Arrhythmogenic right ventricular cardiomyopathy (ARVC) is a genetically heterogenous disease characterized pathologically by the replacement of normal myocardium with fibrofatty tissue and clinically by ventricular arrhythmias and dysfunction. The change in tissue results in wall thinning and aneurysm formation in the ventricles. Wall thinning can also result in ventricular dysfunction, often starting with the right ventricle due to its thinner wall. This can progress to biventricular dysfunction at end-stage disease. Exercise, especially endurance sports, can accelerate wall thinning. As a result, AC is a leading cause for sudden cardiac death (SCD).[14]

DILATED CARDIOMYOPATHY

Dilated cardiomyopathy is characterized by a dilated left ventricle with systolic dysfunction that is not caused by ischemic or valvular heart disease. Ventricular remodeling occurs, driven by pathologic myocyte hypertrophy, myocyte apoptosis, myofibroblast proliferation, and interstitial fibrosis. The alteration in ventricular architecture leads to an increased chamber volume, and the dilation of the left or both ventricles causes reduced systolic function with an increased preload but reduced stroke volume and contractility. Patients with dilated cardiomyopathy can develop a broad range of atrial and ventricular arrhythmias that can lead to collapse or sudden cardiac arrest.[15]

BRUGADA

Brugada syndrome is an electrical cardiac disorder that predisposes patients to SCD through the development of ventricular tachycardia and ventricular fibrillation in a structurally normal heart. The arrhythmias are secondary to mutations in cardiac ion channels that alter the transmembrane ion currents that constitute the cardiac action potential. Variants in 19 genes have been implicated but it is not known how much each of these contribute to clinical disease, and whether Brugada is caused by changes to depolarization, repolarization, or current-load match is a question still under debate.[16] The mutations lead to diagnostic electrocardiogram

(ECG) changes with right ventricular conduction delay, coved-shaped or saddle-shaped ST segment elevation, and/or T-wave inversion. This alters the excitation wavelength, which ultimately increases the risk of arrhythmia, syncope, and sudden death. Most individuals are asymptomatic, and it is not known what factors predispose to arrhythmia, although the risk of recurrence after an initial episode is approximately 50%. Ventricular fibrillation occurs more often at night during periods of increased vagal tone, although fever and certain drugs can also increase the risk.[17,18]

MYOCARDITIS

Myocarditis is an inflammatory disease of the myocardium that occurs in 3 phases: acute viral, subacute immune, and chronic phase.[19] The acute phase is commonly associated with a viral infection, although there are noninfectious causes such as autoimmune conditions (inflammatory bowel disease, rheumatoid arthritis, and so forth) or pharmacologic induced (antibiotics, amphetamines, anabolic agents, and so forth).[20] Athletes are particularly susceptible to myocarditis when traveling, competing in extreme environments, and pushing past physical limits—all potential reasons for a compromised immune system, which increases the risk for viral infection. The acute viral phase may often go undetected because it is short lived but the following subacute and chronic inflammation stages can result in the destruction of myocytes and improper cardiac remodeling. This remodeling can result in an increased risk of arrhythmia. Endurance athletes can trigger a more significant inflammatory response, which may magnify the potential for arrhythmia. After recovery, a chronic cycle of cytokine inflammation may persist. Overall, these factors make myocarditis one of the leading causes of sudden death in athletes.

Similar to other viruses, direct infiltration of SARS-CoV-2 into the myocardium produces a strong cytokine response resulting in complications such as arrhythmias and SCD.[21]

Myocarditis in the acute phase is often a mild presentation of vague symptoms that are variable from case to case. If a viral illness is the cause, it can present with symptoms such as fevers, rigors, rhinorrhea, cough, nausea, vomiting, headache, palpitations, myalgias, shortness of breath, or lightheadedness. If the athlete reports chest pain or other cardiac symptoms (eg, syncope), have an increased index of suspicion for myocarditis.

CORONARY ARTERY ANOMALIES

SCD associated with CAAs occur mostly during exercise. An anomalous coronary artery with an aortic origin is one of the most common causes of SCD in the athlete.[22,23] However, it is not the only anomaly reported—there are a vast number of other CAAs. To simplify this, the anomalies can be divided into 3 categories: anomalies of origin, course, and termination.

Anomalies of origin: Athletes with an anomalous pulmonary origin of the coronary arteries may suffer from ischemia in affected myocardial territories.[23] The level of ischemia may depend on oxygen demand and collateral circulation. About 17% of athletes with an anomalous pulmonary origin of the left coronary artery have either ventricular arrhythmia or SCD.[23]

Anomalies of course: Myocardial bridging occurs when a coronary artery courses intramurally within the heart. It is often associated with a higher risk of myocardial infarction. High-grade stenosis of the coronary ostium should be considered as a risk for heart failure and potential SCD.[23]

Anomalies of termination: Coronary fistulas typically result in right-sided heart failure or chronic dyspnea but SCD is only theoretic possibility.[23]

Dyspnea and exertional chest pain typically occur first. However, SCD may be the first presentation of CAA. This is estimated to occur in up to 50% of cases.[23]

The diagnostic gold standard for CAA IS coronary computed tomography angiography.[22] ECG, echocardiogram, and stress test are other important diagnostics to consider especially when evaluating from ischemic disease caused by CAAs.[23]

SICKLE COLLAPSE (EXERCISE-RELATED COLLAPSE IN THE ATHLETE WITH SICKLE CELL TRAIT)

The mechanism of ECAST is not completely understood. However, it is known that the risk of exercise-related death in Division 1 athletes is 37 times higher in SCT athletes compared with athletes without SCT.[24] A commonly proposed theory is that vigorous exercise can result in hyperthermia and dehydration triggering vaso-occlusion and endothelial damage from hemoglobin S polymerization. This vaso-occlusive crisis reduces blood flow and increases cellular destruction. A cascade of rhabdomyolysis, hyperkalemia, increasing acidosis, disseminated intravascular coagulation, cardiac and renal failure, and arrhythmia can occur. Poor conditioning, high ambient temperatures, dehydration, or altitude can be risk factors for a sickle collapse in an SCT athlete.[25,26]

ECAST may initially present as a “conscious collapse” where the athlete is listless but still conversant. They often report weakness more than pain. Alternatively, acute collapses have been reported.[11]

STABLE VERSUS UNSTABLE TACHYARRHYTHMIAS

Tachyarrhythmias are abnormal heart rhythms in which the heart is beating faster than usual. Causes can vary widely but can generally be broken down into atrial and ventricular tachycardias. Clinically, it is also important to differentiate between stable and unstable tachyarrhythmias because the treatment differs significantly between the two. Atrial tachycardias include atrioventricular reentrant tachycardia, atrioventricular nodal reentrant tachycardia, atrial fibrillation, atrial flutter, and Wolff-Parkinson-White syndrome. These tachyarrhythmias initiate in the atria and typically involve premature beats triggering a reentrant circuit. This circuit repeatedly generates ventricular beats, which bypass the rate control of the AV node and can generate heart rates as high as 250 to 300 bpm. The location of the circuit, as well as typical ECG findings, differentiates the atrial tachyarrhythmias. Ventricular tachyarrhythmias include ventricular tachycardia and ventricular fibrillation. These rhythms are much more likely to be unstable and are the most common causes of SCD. Ventricular tachycardia is a wide-complex tachycardia that can be sustained or nonsustained, depending on the duration, and monomorphic or polymorphic, based on the QRS morphology. The mechanism involves enhancement of normal or abnormal automaticity, early or late depolarizations, and reentry circuits, whereas causes can include ischemic heart disease, structural abnormalities, channelopathies, cardiomyopathies, electrolyte imbalances, and drug effects. Ventricular fibrillation occurs when the ventricular myocardium depolarizes erratically in an uncoordinated manner and shows fibrillation waves of varying amplitude and shape. There are no identifiable P waves, QRS complexes, or T waves, with a heart rate that can vary between 150 and 500 bpm. By definition, ventricular fibrillation is always unstable.[27] Signs and symptoms include but are not limited to palpitations (in chest or neck), chest pain, shortness of breath, generalized weakness, dizziness, diaphoresis, nausea, vomiting, syncope, or sudden death.

CORONARY ARTERY DISEASE

Atherosclerotic coronary artery disease (ASCAD) is the most common cause of death in athletes aged older than 35 years. It does still occur in a small percentage of young athletes as well, especially in those with inherited hyperlipidemia. For many, SCA is the first presenting symptom of ASCAD because acute events are caused by coronary plaque disruption and acute thrombosis rather than a gradual narrowing of coronary arteries. Vigorous exercise during competition will transiently increase the risk of acute myocardial infarction and SCA. This occurs by either an increased risk of plaque disruption or a malignant arrhythmia. Arrhythmias can develop from demand ischemia or can originate in an area of myocardial scarring. The risk of an acute exertion-related cardiac event is highest in those who have had earlier acute coronary syndrome and lower in those with "silent" ASCAD (found only through advanced/provocative diagnostic testing). The risk of SCA also increases with the extent of CAD, with lower left ventricular (LV) systolic function, with greater ischemia, and with increased electrical instability. The risk can be mitigated to some extent through aggressive lipid-lowering therapy, which can lower the lipid content of lipid-rich unstable plaques. This plaque regression can take as long as 2 years, and athletes with known ASCAD should exercise caution when deciding on their level of athletic competition.[28]

AORTIC DISSECTION OR RUPTURE (MARFAN SYNDROME)

Aortic pathologic condition in athletes is a common cause of sudden death in athletes due to the aortic stress that can occur during intense physical activity. Multiple predisposing aortic conditions besides Marfan syndrome can result in aortic dissection or rupture: familial thoracic aortic aneurysm, bicuspid aotic valve, aortic aneurysm, Loeys-Dietz syndrome. Marfan syndrome is an autosomal dominant disorder that affects connective tissue across multiple systems including the cardiac, pulmonary, musculoskeletal, skin, and ocular systems. The predominant gene mutation is in *fibrillin-1* and the incidence of disease is 1 in 5000 to 10,000.[29] Diagnosis of Marfan syndrome in adults relies on the Ghent criteria. Progressive aortic dilation with eventual aortic valve dysfunction can lead to aortic dissection, a tearing of the intima layer of the aorta, in athletes with Marfan syndrome.[30] Although it is known that mild aortic enlargement can occur from repetitive intense physical activity, aortic root dilation greater than 4 cm is abnormal and presents a risk for dissection. However, up to 12% of patients with Marfan and aortic dilation less than 4 cm still had an acute type A dissection.[31] Athletes with Marfan syndrome are recommended to participate in low and moderate static/low dynamic competitive sports if they do not meet exclusion criteria such as aortic root dilatation, moderate-to-severe mitral regurgitation, ejection fraction less than 40%, or a family history of aortic dissection at an aortic diameter of less than 5 cm.[29]

Sudden severe chest or upper back pain that may radiate to back or have a tearing or ripping sensation. Sudden severe abdominal pain, shortness of breath, sudden onset of neurologic symptoms, greater than 20 mm Hg difference in blood pressure between left and right arms or weaker pulses in one arm, decreased pulse strength, hypertension (more common), hypotension (sign of severe disease), tachycardia, syncope, or sudden death.

COMMOTIO CORDIS

Commotio cordis is ventricular fibrillation caused directly by blunt trauma to the heart, with no structural damage to the heart itself or surrounding structures. The mechanical distortion of the myocardium from the trauma creates enough mechanical energy to

cause inappropriate cardiac depolarization, resulting in an unstable arrhythmia and cardiac arrest. It most commonly results from direct impact with a hard ball, such as a baseball. The impact must occur directly over the heart in the anterior chest during ventricular repolarization, during the upstroke just before the peak of the T wave. This occurs during about 1% of the cardiac cycle, although that can be higher with increased heart rates during exercise. The force from the impact causes a stretch in myocardial cell membranes, which may activate ion channels, specifically the stretch-sensitive $K +_{ATP}$ channel, leading to depolarization and ventricular fibrillation. The estimated energy required is approximately 50 J, and the risk peaks at around 40 mph for a baseball, as higher speeds are more likely to result in structural damage to the heart or chest wall. The risk of commotio cordis is also higher with smaller objects, and highest when the impact occurs over the center of the left ventricle. The risk can be reduced by softening the impact object or with chest protection, although studies have found that standard chest protectors are not designed to prevent commotio cordis, and do not prevent VF induction in laboratory experiments.[32]

ENVIRONMENTAL

Lightning strikes cause cardiac injury and sudden death through several pathways. Lightning delivers a current during the course of 0.001 to 0.1 seconds that can vary from 30,000 to 100,000 A.[12,33] This energy may pass over the surface of the victim or may penetrate to cause internal injuries. When the heart is involved, the energy from a lightning strike can cause cardiac contusion and pericardial disease, myocardial ischemia/infarction, myocardial stunning, arrhythmias, and aortic injury, including dissection.[33] Arrhythmias can be caused by the sudden increase in voltage from the lightning strike, which can depolarize the entire myocardium. They can also originate from increased autonomic stimulation and the associated catecholamine surge, which have an additive effect on the rate and rhythm. Ventricular tachycardia and ventricular fibrillation are the most common initial rhythms, although asystole is also frequently seen and may be caused primarily or secondary by medullary dysfunction. Nonfatal changes such as atrial fibrillation and QT prolongation have also been observed, possibly related to changes in intracellular calcium metabolism.[12] Cardiac contusion and aortic dissections can occur as a direct result of current passing through the myocardium or can be caused secondarily by explosive environmental effects leading to blunt or blast trauma. Patients with cardiac contusion can develop cardiac stunning and cardiogenic shock and can suffer morbidity and mortality up to several days after the initial strike. Cardiomyopathy may also develop, leading to cardiovascular compromise and ventricular failure. Pericarditis can also be a delayed response to a lightning strike, with myocardial inflammation and necrosis leading to pericardial effusions weeks to months afterward.[33]

EMERGENCY ACTION PLAN

The EAP is essential to proper cardiac emergency preparedness on the sideline. Development and review of the EAP should include proper communication techniques, emergency equipment location and training, integration of emergency responders, hospital transportation coordination, and catastrophic event training and preparedness. The EAP should be reviewed on an annual basis, at a minimum, with all stakeholders.

In the event of an unresponsive athlete, clear communication pathways should be designated in the EAP. We suggest the practice of nonverbal communication such as predetermined hand gestures to avoid difficulty in sending and receiving verbal

communication in high volume settings such as a crowded stadium. However, the use of a primary and secondary backup verbal communication network such as walkie-talkie, cellphone, or satellite phone is recommended to allow for proper communication not only with on-site personnel but with hospital personnel as well. In large events or in remote areas, networks may be overloaded or unavailable, making equipment such as a satellite phone or landline particularly useful.

The location and availability of emergency equipment that include an AED will vary depending on the venue. However, the equipment should be placed in an easily accessible site that is not behind a locked door or gate. The location(s) should be a part of the EAP review. Emergency responder training in the use of an AED regardless of background is of the utmost importance. These responders can include school staff, students, teammates, team staff (eg, athletic trainer), EMS, or physicians. Equipment including AEDs should be checked during the annual review to ensure that they are still operational. AED battery life on standby, depending on the manufacturer, may last from 2 to 5 years in total. In the event an AED is used, the disposable pads should be replaced with a new set before being stored.

The level of expertise and involvement of EMS varies depending on the venue. The EAP should determine if EMS is on-site and off-site, what type of transportation is available, how they will be activated, and what level of technician is present. The levels of an emergency medical technician (EMT) across the different states in the United States may vary but typically are basic, intermediate, and advanced/paramedic. The EMT level will determine their capabilities and what type of equipment/medications they will have available. Coordination with EMS and the properly resourced receiving hospital is paramount in the successful resuscitation of an unresponsive athlete. The location of transportation, time and ease of access, and capabilities of the potential receiving hospitals should be listed in the EAP.

The level of preparedness of every EAP-listed emergency responder will determine the success of a cardiac resuscitation. A well-run resuscitation should be efficient, smooth, quiet, and without much wasted effort. Preassignment of duties, including a team leader, should be detailed in the EAP.

In the unfortunate circumstance of a cardiac resuscitation, a postevent protocol should be detailed in the EAP. The event should be documented, analyzed, debriefed, and used for quality improvement. Administrative, legal, and emotional support should be activated for involved parties.

GENERAL PREPARTICIPATION PHYSICAL EXAMINATION CONSIDERATIONS

A detailed history of the athlete's symptoms during and outside of exercise remains one of the most important tools in the preparticipation cardiac screening evaluation. Care should be taken to ask about any chest pain or chest pain equivalents, shortness of breath, fevers, chills, history of syncope—exertional or otherwise, family history of sudden death or cardiac disease.

The physical examination should focus on findings associated with conditions that might predispose an athlete to sudden cardiac arrest. The most common findings on examination that would prompt further workup include heart murmur, especially a systolic murmur that increases with Valsalva, Marfanoid features (long, narrow face, tall, thin body build, disproportionately long arms, legs, fingers, and toes, abnormal spine or sternum curvature, extreme nearsightedness, flat feet), or abnormal rhythm or resting tachycardia/bradycardia.

There is considerable debate about the cost-effectiveness and appropriateness of diagnostic testing as a part of routine preparticipation physical exam (PPE) screening,

which is outside the scope of this article. When further workup is indicated, however, cardiac testing may include ECG, echocardiogram, stress test, or cardiac MRI.

EVALUATION OF OUTCOME AND/OR LONG-TERM RECOMMENDATIONS

Athletes who suffer cardiac arrest events who achieve ROSC and are subsequently stabilized will need extensive postarrest care. Initial steps should include emergent transport to the nearest appropriate hospital for intensive care unit (ICU) admission and specialty Cardiology evaluation. The underlying cause of the arrest will determine what further treatment modalities are required. Patients who suffer an arrest secondary to an arrhythmia should have testing including electrocardiogram, echocardiogram, and an electrophysiological study to help determine the underlying cause of the arrest. These patients generally need an implantable defibrillator because patients who have suffered a cardiac arrest are at a much higher risk for repeat arrhythmia/arrest. Patients who also have an underlying cardiomyopathy may need symptomatic treatment as well as preventative measures to avoid future events. Those with hypertrophic cardiomyopathy that results in outflow tract obstruction may benefit from heart failure medications such as beta-blockers, calcium-channel blockers, or diuretics. HCM patients with marked outflow obstruction unresponsive to medical therapy may also be candidates for ventricular septal surgical myectomy or alcohol septal ablation.[34] ARVC patients will typically need similar treatment of heart failure symptoms related to their cardiomyopathy, with medical management, lifestyle changes, and catheter ablation.[35] Patients with cardiomyopathy not adequately responsive to these treatments may ultimately progress to a need for heart transplantation. Recommendations for patients with a definitive diagnosis of HCM or ARVC include restriction from competitive and high-intensity sports, and possible restriction from low-intensity recreational sports and athletic activities, depending on their individual risk.[34] Patients suffering a cardiac arrest following a commotio cordis event should undergo testing for underlying structural heart abnormalities but may be able to fully return to play if the heart is normal and the athlete has no other long-term effects. See **Fig. 1** for recommended management guidelines for HCM.

CORONARY ARTERY DISEASE

Athletes with ASCAD should undergo exercise testing to determine the presence and extent of inducible ischemia and electrical instability as well as to evaluate LV function. They should have aggressive statin therapy to reduce the risk of plaque disruption and must weigh the risks and benefits of exercise when determining level of future competition. Athletes with LV ejection fraction greater than 50% and no inducible ischemia or electrical instability should be allowed to participate in all levels of competition. It is reasonable to restrict athletes with a higher disease burden, and athletes who have an AMI or who undergo coronary revascularization should refrain from competitive sports for at least 3 months.[28]

MYOCARDITIS

Athletes with suspected myocarditis should have diagnostics that include an ECG, cardiac biomarkers (troponin, natriuretic peptide, creatine kinase), inflammatory markers, chest radiograph, and echocardiogram. In suspected COVID-19 infection, a COVID-19 polymerase chain reaction test (PCR) should be performed as well. The gold standard for noninvasive diagnostics is cardiac MRI (cMR), whereas an

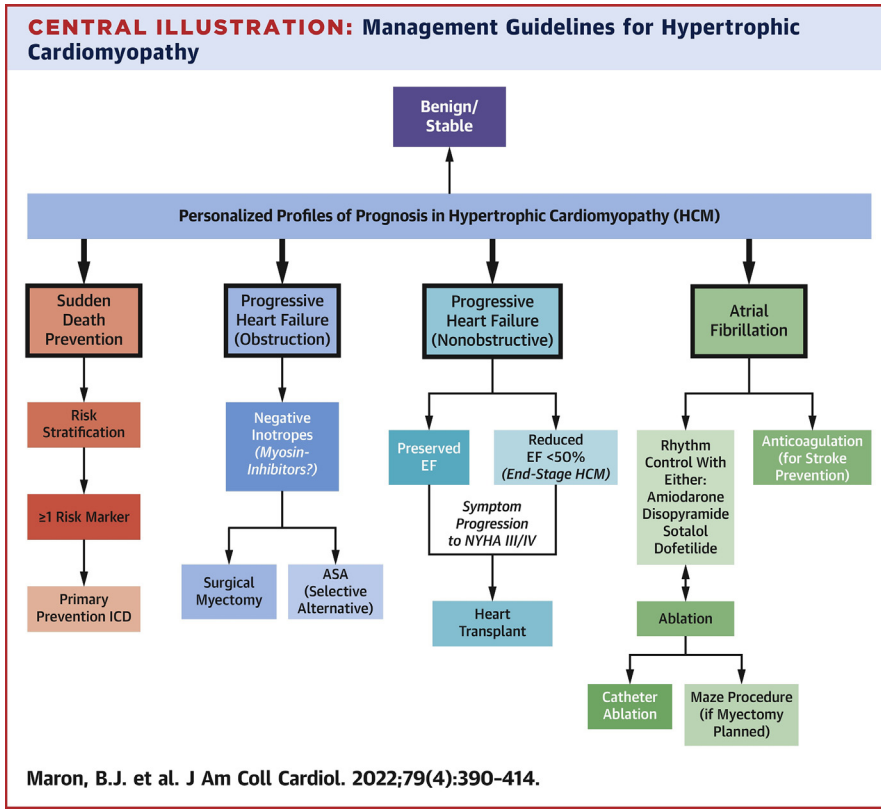

Fig. 1. Management guidelines for hypertrophic cardiomyopathy.[34]

endomyocardial biopsy is the invasive standard. In cases where the cMR is inconclusive, a fluorodeoxyglucose positron emission tomography scan can be considered.[36] Although there are multiple diagnostic modalities to evaluate for myocarditis, to date, there are no universally accepted diagnostic clinical criteria for myocarditis. However, Eichhorn and colleagues have suggested a diagnostic pathway for athletes that uses ECG, Holter monitor, stress test, cardiac biomarkers, inflammatory biomarkers, or echocardiogram as a screening tool.[19] Refer to **Fig. 2** for their proposed diagnostic and treatment algorithm for myocarditis. If one of these tests is positive, cMR is used to investigate for evidence of myocarditis. If a diagnosis of myocarditis is made, exercise and sports restriction is recommended for a minimum of 3 months. After symptoms have resolved, repeat testing with echocardiogram, Holter monitoring, and possible cMR is recommended at 3 months. If symptoms have not resolved, reevaluation every 3 months is recommended.[37]

DISSECTION

Long-term management of aortic dissection includes close regulation of blood pressure and heart rate. In athletes who survive an aortic dissection, care must be taken to minimize the risk of worsening the dissection through rupture or recurrent dissection, which can happen through increased aortic wall shear stress. This increased

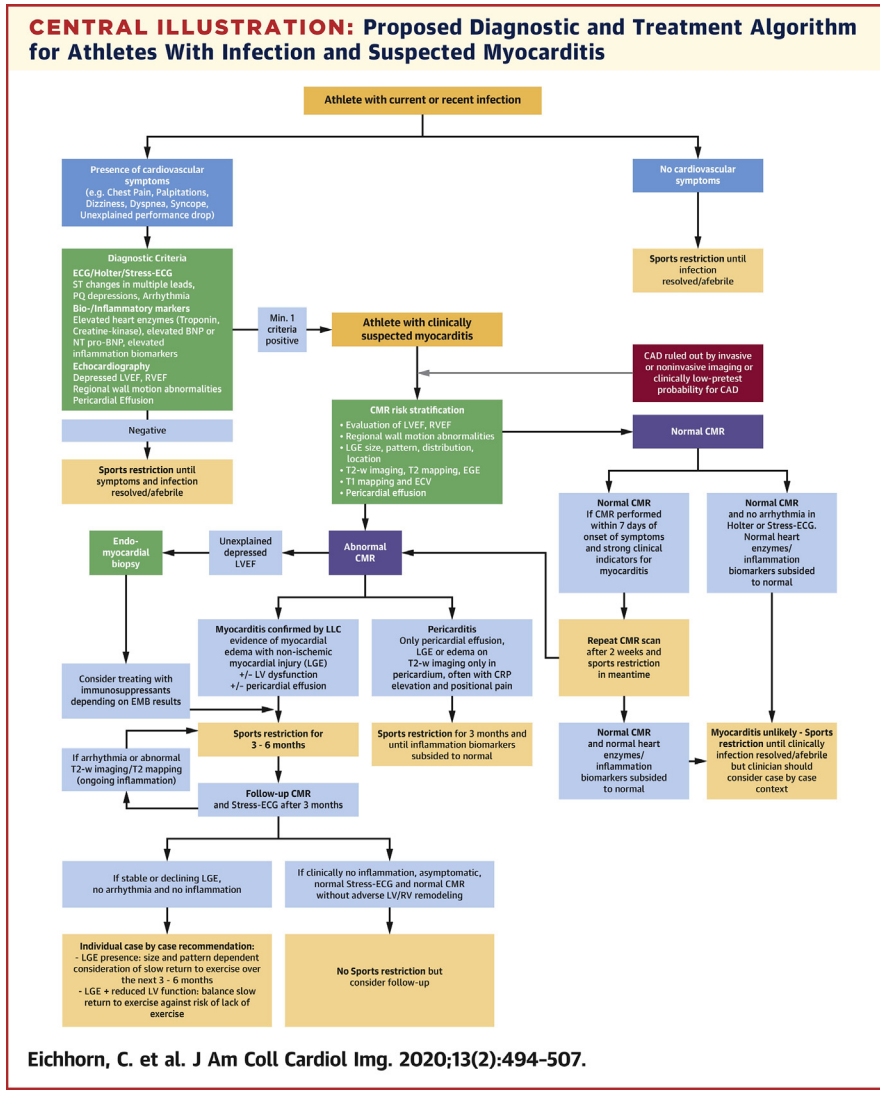

Fig. 2. Proposed diagnostic and treatment algorithm for athletes with infection and suspected myocarditis.[19]

stress can occur with sudden increases in arterial blood pressure and heart rate, such as during exercise, and is most significant in higher intensity activities such as weightlifting or running. However, maintaining regular exercise can still be a benefit to these patients in terms of reaching long-term blood pressure, heart rate, and body weight goals. Recommendations generally advise that mild-to-moderate intensity aerobic exercise (3–5 metabolic equivalents) for at least 30 minutes a day is beneficial. Weightlifting should be limited to lighter weights and athletes should stop several repetitions before failure to avoid straining. Higher intensity exercises and competition should generally be avoided.[38,39] In patients with Marfan syndrome, varying organizations have created specific activity recommendations (**Fig. 3**).[40]

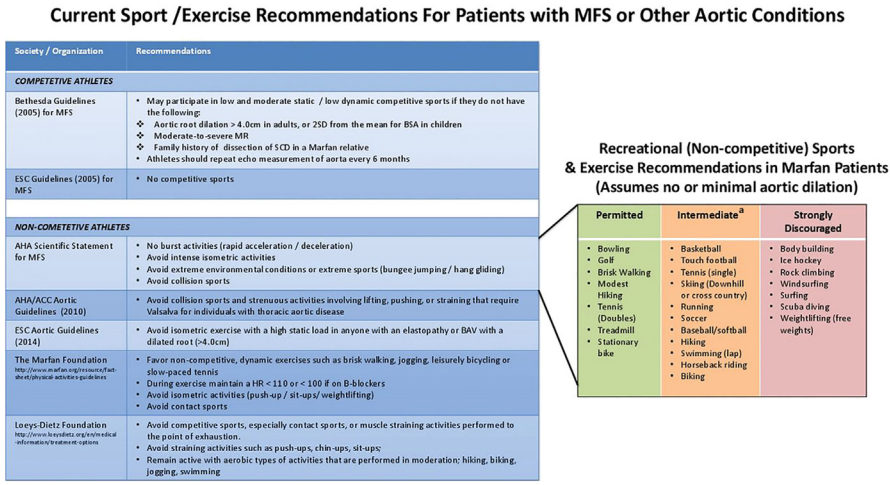

Fig. 3. Physical activity recommendations for athletes with Marfan syndrome from varying organizations.[a]Intermediate or indeterminate activities should be assessed clinically on an individual basis.

SICKLE CELL TRAIT

Although SCT is not a contraindication to participation in athletics, screening for sickle cell trait during PPEs is of the utmost importance. If SCT status is unknown, a hemoglobin solubility test and subsequent hemoglobin electrophoresis can help to identify HgbS and its subtypes, respectively.

Return to play should not be considered until the athlete is asymptomatic at rest and has normal end-organ function. Bloodwork such as a complete blood count, comprehensive metabolic panel, creatine kinase, lactate dehydrogenase, uric acid, and disseminated intravascular coagulation panel should be resolved to baseline. Assessment of identifiable event risk factors or uncovered risk factors such as hydration status, heat, altitude, pharmaceutical influence (eg, sympathomimetic, caffeine, other stimulants, and so forth.), abnormal work–rest ratio, poor electrolyte supplementation, chronic kidney disease, evidence of hyposthenuria, or other genetic anomaly will influence the decision for the athlete to return to play.[11]

A schedule to allow for gradual conditioning, exclusion from performance tests, an emphasis on hydration and electrolyte replacement, modifications for an athlete at altitude with supplemental oxygen if needed, and careful observation for concomitant acute illnesses can help prevent sickle collapse. However, despite these methods, sickle collapse can still occur.[24]

CORONARY ARTERY ANOMALIES

Long-term recommendations after diagnosis of CAA are athlete-dependent but there are generalizable recommendations. The athlete must have confirmatory imaging, a negative exercise stress test, and be asymptomatic.[23] If the athlete has surgical repair of an anomalous aortic origin of a coronary artery, the minimum time before participation is at least 3 months postoperative. The athlete must be asymptomatic and must not demonstrate ischemia on a maximal exercise stress test. Overall, as demonstrated in **Table 1**, recommendations are highly variable and depend on the specific anomaly in question.

SUMMARY/DISCUSSION/FUTURE DIRECTIONS

A moment of tragedy can be prevented and reversed with proper preparedness through a careful PPE and practiced EAP. The approach and management to SCD are the same despite multiple causes. Although the medical community has made great strides in standardizing CPR response, more evidence-based guidance should be developed for the at-risk or returning athlete.

Table 1
A comparison of current international guidelines for exercise restrictions in patients with coronary artery anomalies

Comparison of Current International Guidelines for Exercise Restrictions in Patients With CAAs

2015 ACC/AHA scientific statement for competitive athletes with cardiovascular abnormalities			2020 ESC guidelines on sports cardiology and exercise in patients with cardiovascular disease		
COR	LOE	Recommendations	COR	LOE	Recommendations
			IIa	C	When considering sports activities, evaluation with imaging tests to identify high-risk patterns and an exercise stress test to check for ischemia should be considered in individuals with AAOC.
			IIb	C	In asymptomatic individuals with an anomalous coronary artery that does not course between the large vessels or does not have a slit-like orifice with reduced lumen or intramural course, competition may be considered after adequate counseling on the risks provided that there is an absence of inducible ischemia.
			III	C	Participation in most competitive sports with a moderate or high cardiovascular demand among individuals with AAOC with an acutely angled takeoff or an anomalous course between the large vessels is not recommended.*
IIa	C	Athletes with AAORCA should be evaluated by an exercise stress test. For those without either symptoms or a positive exercise stress test, permission to compete can be considered after adequate counseling of the athlete or the athlete's parents (in the case of a minor) about the risk and benefit, taking into consideration the uncertainty of the accuracy of a negative stress test.			
III	B	Athletes with AAOLCA, especially when the artery passes between the pulmonary artery and aorta, should be restricted from participation in all competitive sports, with the possible exception of class IA sports, before surgical repair. This recommendation applies whether the anomaly is identified as a consequence of symptoms or discovered incidentally.			
III	C	Nonoperated athletes with AAORCA who exhibit symptoms, arrhythmias, or signs of ischemia on exercise stress test should be restricted from participation in all competitive sports, with the possible exception of class IA sports, before a surgical repair.			
IIb	C	After successful surgical repair of AAOC, athletes may consider participation in all sports 3 mo after surgery if the patient remains free of symptoms and an exercise stress test shows no evidence of ischemia or cardiac arrhythmias.	IIb	C	After surgical repair of an AAOC, participation in all sports may be considered, at the earliest 3 mo after surgery, if the athletes are asymptomatic and there is no evidence of inducible myocardial ischemia or complex cardiac arrhythmias during maximal exercise stress test.
I	C	Athletes with APOC artery can participate only in low-intensity class IA sports, regardless of whether they have had a prior myocardial infarction, and pending repair of the anomaly.			
IIb	C	After repair of APOC, decisions about exercise restriction may be based on presence of sequelae such as myocardial infarction or ventricular dysfunction.			
IIa	C	It is reasonable for athletes with myocardial bridging and no evidence of myocardial ischemia during adequate stress testing to participate in all competitive sports.	IIa	C	Participation in competitive and leisure-time sports should be considered in asymptomatic individuals with myocardial bridging and without inducible ischemia or ventricular arrhythmia during maximal exercise testing.
IIa	C	It is reasonable to restrict athletes with myocardial bridging of an epicardial coronary artery and objective evidence of myocardial ischemia or prior myocardial infarction to sports with low to moderate dynamic and low to moderate static demands.	III	C	Competitive sports are not recommended in individuals with myocardial bridging and persistent ischemia or complex cardiac arrhythmias during maximal exercise stress testing.
IIa	C	It is reasonable to restrict athletes who have undergone surgical resection of the myocardial bridge or stenting of the bridge to low-intensity sports for 6 mo after the procedure. If such athletes have no subsequent evidence of ischemia, they may participate in all competitive sports.			

CLINICS CARE POINTS

- Cardiac emergencies are a significant cause of morbidity and mortality in athletes and are the leading nontraumatic cause of death in the young athlete.
- Prompt evaluation and treatment of the collapsed athlete are vital to maximizing their chances of survival.
- Formal planning for an efficient and effective response care can help avoid catastrophic outcomes.
- Initial management of cardiac emergencies includes the activation of EMS, early CPR, rapid defibrillation, and transfer to an appropriate medical center.
- Sudden cardiac arrest should be suspected in any collapsed and unresponsive athlete, and an AED should be used as early as possible, with CPR taking place until it can be applied.
- Interruptions in chest compressions for rhythm analysis and defibrillation should be minimized, and compressions should resume immediately after a shock is delivered.
- Underlying cardiac abnormalities that can predispose some athletes to sudden cardiac arrest include cardiomyopathies, CAAs, and channelopathies, as well as genetic disorders such as sickle cell trait, and Marfan syndrome.
- Screening for cardiac abnormalities is an important strategy in trying to prevent sudden cardiac arrest, although the sensitivity, specificity, and cost-effectiveness of the current screening practices are ongoing topics of debate.
- Cardiac arrest secondary to commotio cordis should be suspected in any athlete who collapses shortly after being struck in the chest.
- Lightning strikes are a rare but potentially fatal cause of cardiac emergencies and can cause both short-term and long-term injuries to the patient's heart.

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