

Sudden Cardiac Arrest in Pediatrics

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Abstract: Sudden cardiac arrest (SCA) in the pediatric population is a rare and potentially devastating occurrence. An understanding of the differential diagnosis for the etiology of the cardiac arrest allows for the most effective emergency care and provides the patient with the best possible outcome. Pediatric SCA can occur with or without prodromal symptoms and may occur during exercise or rest. The most common cause is arrhythmia secondary to an underlying channelopathy, cardiomyopathy, or myocarditis. After stabilization, evaluation should include electrocardiogram, chest radiograph, and echocardiogram. Management should focus on decreasing the potential for recurring arrhythmia, maintaining cardiac preload, and thoughtful medication use to prevent exacerbation of the underlying condition. The purpose of this review was to provide the emergency physician with a concise and current review of the incidence, differential diagnosis, and management of pediatric patients presenting with SCA.

Key Words: cardiac arrest, cardiovascular disease, hypertrophic cardiomyopathy, channelopathy, arrhythmia

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TARGET AUDIENCE

This CME activity is intended for emergency physicians, nurses, hospitalists, and general pediatricians.

LEARNING OBJECTIVES

After completion of this article, the reader should be able to:

1. Outline the possible cardiac causes of sudden arrest in children and adolescents.
2. Describe emergency care of a pediatric sudden cardiac arrest patient.
3. Identify disease-specific presentation of cardiac causes of sudden arrest in children and adolescents.

CASE

A previously healthy 5-year-old child experienced a sudden, unwitnessed collapse after sitting on a bench during recess. A teacher discovered him pulseless and began bystander cardiopulmonary resuscitation. After intubation by emergency medical responders on the scene, he had spontaneous return of circulation. Upon arrival to the emergency department (ED), he was noted to be persistently bradycardic and hypotensive. He received 2 doses of benzodiazepine for clinical findings concerning for seizure. Further evaluation included obtaining a focused history, resuscitation

with fluid boluses, performance of diagnostic studies, and initiation of inotropes. Chest radiograph and electrocardiogram (ECG) obtained in the ED were suspicious for underlying cardiac etiology of his arrest. Echocardiography performed in the pediatric intensive care unit (ICU) led to a diagnosis of hypertrophic cardiomyopathy (HCM).¹

Sudden cardiac arrest (SCA) occurs when cardiac output rapidly declines and will result in sudden cardiac death (SCD) if cardiac function is not restored quickly. Sudden cardiac death is further defined as a rapid and unsuspected death due to a cardiovascular cause, typically within 1 hour from the onset of symptoms and usually occurring in a patient previously considered healthy.² When resuscitation is successful, the term “aborted cardiac death” is sometimes used, although SCA alone may suffice. In discussion of SCA in pediatrics, children aged 12 months are excluded from most definitions because of more variable etiologies involved.³ Sudden cardiac arrest in children is relatively uncommon compared with adult cases of cardiac arrest.⁴ Due to the uncommon occurrence of pediatric SCA and SCD, these cases are particularly devastating for families, schools, and entire communities.⁵ This review focuses on the cardiac etiologies of pediatric SCA, with the goal of maximizing resuscitative efforts to prevent progression to SCD and result in favorable outcomes in patients who experience SCA. Specific treatment of cardiac conditions is not discussed, but rather emergency evaluation of suspected SCA.

INCIDENCE

The true incidence for SCA and SCD is difficult to discern. This is due, in part, to the occasional unclear distinctions between arrest alone and arrest with death. In the current report, the terms will be used specifically to the reference cited, with joint use of the terms (SCA and SCD) if the exact meaning is unclear or not described. Furthermore, most data for pediatric SCD are collected post mortem, such that many cases of SCA or aborted SCD are often not reported. Sudden unexpected death in childhood, which excludes patients younger than 12 months, is estimated at 1.3 per 100,000 patient years in the United States, of which cardiac causes are estimated to be present in 30% of these cases.⁶ In addition, it is estimated that approximately 2000 patients younger than 25 years will die from a sudden cardiac event each year in the United States,⁷ with an incidence estimated from 0.6 to 6.2 per 100,000 persons.⁵ The incidence may be higher in athletes, with a relative risk of sudden death of 2.1 compared with nonathletes.⁸

These numbers have also evolved over the years and seem to be increasing over time.^{9–21} Estimates differ because of unclear definitions of terms, varied methods of reporting and collecting data, and lack of mandatory reporting. Many of these reporting issues would be minimized with a standard reporting schema or a national registry.²² To further accurately define the incidence of SCD, the Centers for Disease Control and the National Institutes of Healthcare currently are enrolling patients into the “Sudden Death in the Young Registry.” This registry began in September 2014 and includes all patients in 15 states who had sudden unexpected death due to an underlying medical cause, excluding trauma. The goal for this study is to further define the relative risk

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by reviewing cases across several institutions to determine the cause of death.²³

UNDERLYING DIFFERENTIAL DIAGNOSES

The current review will summarize the literature pertaining to SCA, which for the purposes of this discussion will also encompass aborted SCD. Cardiac conditions that can result in pediatric SCA can be grouped into the following 3 general categories: structural cardiac defects, primary cardiac electrical abnormalities, and acquired conditions (due to certain drugs or chemical exposures, secondary pulmonary artery hypertension or Eisenmenger syndrome, and *Commotio cordis*). A list of etiologies is found in Table 1.

Structural etiologies include cardiomyopathies as well as congenital and postoperative cardiac lesions. The etiologies for SCA are more likely to be congenital heart disease in the young child and related to arrhythmia in the adolescent.²⁵ Congenital cardiac lesions are beyond the scope of this article and are not discussed in detail. The most common structural defect discovered at the time of a SCA is HCM (36%). Other cardiac defects include coronary artery anomalies (17%), myocarditis (6%), and arrhythmogenic ventricular dysplasia (4%).²⁶

The most common mechanism leading to SCA is arrhythmia, which can be secondary to an underlying channelopathy, cardiomyopathy, or myocarditis.²⁷ Electrophysiological abnormalities include long QT syndrome (LQTS), Wolff-Parkinson-White syndrome, Brugada syndrome, catecholaminergic polymorphic ventricular tachycardia (CPVT), short QT syndrome (SQTS), arrhythmogenic ventricular dysplasia (previously arrhythmogenic

right ventricular cardiomyopathy), and complete heart block.⁷ Arrhythmias are demonstrated secondary to ion channelopathies in 4% of cases²⁶ and may be the etiology of SCA even if no structural heart disease is found.²⁸ Ventricular fibrillation (VF) is a much less commonly reported as a cause of SCA in children (4%–10%) than in adults (50%).^{29,30} However, the lower reported incidence in pediatric patients is likely an underestimate because of lack of a documented initial rhythm at the time of collapse or because of the documentation of the rhythm, which occurred after resuscitative efforts were initiated.⁹

Electrophysiologic abnormalities can also be the result of specific exposures or ingestions. Therapeutic drugs that can result in SCA by an arrhythmogenic mechanism include prescription medications such as erythromycin, carbamazepine, and ketoconazole, as well as overdoses of stimulant or psychotropic drugs. Illicit drug use, such as cocaine, or inhalation abuse of substances such as gasoline, glue, hair or cooking sprays, cigarette lighters (also known as “huffing”) can also result in SCA.⁹

OVERALL PRESENTATION

Sudden cardiac arrest can be the first presentation of an underlying cardiac disease, but occasionally, previous signs and symptoms may help aid in the diagnosis. In cases of SCA resulting in death in children, 61% had no known cardiac disease, 45% had previous symptoms remote to the event, and 26% had symptoms immediately preceding the event.³¹ Prodromal symptoms may be nonspecific, especially in children younger than 10 years.³² When present, the most common antecedent symptoms were seizure, dyspnea, and syncope.³¹ Other preceding symptoms included chest pain, dizziness, and palpitations.⁷ Sixteen percent of children experienced an event during moderate to vigorous activity, and exercise-associated SCD was more common in children aged 10 to 19 years.³²

Risk factors for SCA and SCD have been identified in adults, but few data exist regarding the pediatric population. It has been reported that 4.5% of children with SCD had a positive family history of SCD in a first-degree relative.²⁸ Hypertrophic cardiomyopathy and many channelopathies are known to be genetic.²⁴ In these familial cases, VF or ischemia-related arrhythmias are most likely the common final pathway in adults.^{33,34}

DISEASE-SPECIFIC PRESENTATION

Hypertrophic Cardiomyopathy

Hypertrophic cardiomyopathy is the most frequently identified cause of SCD in young patients.²⁶ It is the most common form of cardiomyopathy in the general population with a prevalence of 0.2% (1 in 500)³⁵ and the second most common in pediatric and adolescent patients with an annual incidence of 0.47 in 100,000.³⁶ Potential causes of HCM are varied and more diverse in pediatric than adult populations. Inheritance in the familial form is generally autosomal dominant but often with variable penetrance of the phenotype. Left ventricular hypertrophy, which may or may not encompass HCM, can also be acquired such as that seen in longstanding hypertension. Further investigation is necessary to better understand whether an increased risk for SCA or SCD exists in this form of hypertrophy. Two recent studies have reported long-term risk for SCD in pediatric and young adult patients with HCM as 35 to 6%; however, the populations included some patients with implantable cardioverter defibrillators and are not representative of undiagnosed patients.^{37,38}

The presentation of HCM in children can range dramatically. Patients may be asymptomatic, may experience exertional symptoms such as fatigue, dyspnea, or chest pain, or may present in

TABLE 1. Predisposing Cardiac Disorders for Pediatric SCA*

Structural/functional	HCM
	Coronary artery anomalies (AOCA, ALCAPA, Kawasaki disease, etc)
	DCM
	RCM
	Myocarditis
	Congenital heart disease (tetralogy of Fallot, transposition of the great arteries, hypoplastic left heart, etc)
	<i>Other less-common causes:</i>
	Aortic rupture
	Aortic valve stenosis
	Left ventricular outflow obstruction
	Mitral valve prolapse
	Coronary arteriosclerotic disease
	Arrhythmogenic right ventricular dysplasia
	Postoperative congenital heart disease (after Fontan, Norwood angioplasty, transplantation surgeries, etc)
Electrophysiologic	LQTS
	SQTS
	CPVT
	Wolf-Parkinson-White syndrome
	Brugada syndrome
	Complete heart block
Other	Drugs/stimulants
	Commotiocordis
	Pulmonary hypertension (primary, secondary)

*Adapted from Section on Cardiology and Cardiac Surgery,⁷ Berger and Campbell,⁹ and Campbell et al.²⁴

cardiac arrest.³⁹ The most common age of presentation is older than 10 years, but it may be diagnosed at any age. Presentation at an earlier age is usually associated with a less favorable outcome because of more rapid progression.⁴⁰

Physical examination may include a systolic ejection murmur most prominent at the right upper sternal border that improves with increased preload (such as with the patient gripping the examiner's hand) as well as S3 and S4 sounds. Findings of electrocardiograms are abnormal in 75% to 90% of patients with HCM, frequently with ST changes, T-wave inversions, and prominent Q waves, especially in the inferior and midprecordial leads (II, III, aVF, V1-V3).^{41,42} Noninvasive imaging is critical for diagnosis, including echocardiogram or cardiac magnetic resonance imaging (MRI).³⁹

Coronary Artery Anomalies

The second most common cause of SCD in pediatric and adolescent patients is coronary artery anomalies, with anomalous origin of a coronary artery (AOCA) from the opposite sinus of Valsalva or anomalous left coronary artery from the pulmonary artery (ALCAPA) being the most common.⁴³ When the left coronary artery arises from the right sinus of Valsalva, as in the patients with AOCA, the artery can course between the aorta and pulmonary artery with compression of the coronary artery producing ischemia during times of increased cardiac output. In a patient with ALCAPA, coronary perfusion pressure is low because of run-off into the pulmonary artery and the myocardium experiences ischemia during times of increased demand. Coronary artery abnormalities may also be acquired, for example, coronary artery ectasia seen as a complication of Kawasaki disease.⁹

Presentation of ACOA usually occurs during adolescence or later, whereas ALCAPA is generally detected in infancy as the pulmonary artery pressures drop but, in some cases, may become evident much later in life. Complaints of early fatigue, angina, or exercise-induced syncope may lead to a directed evaluation. Unfortunately, SCD is frequently the presenting sign.⁴⁴ Patients with coronary ectasia after Kawasaki disease are at an increased risk for subsequent thrombosis or stenosis and may present with SCD.^{45,46}

Physical examination results can be normal in patients with coronary anomalies.⁴⁴ When symptomatic, ALCAPA can present in young patients with symptoms of heart failure (feeding intolerance, tachypnea) related to low cardiac output. Physical examination may reveal S3/S4 gallop, signs of pulmonary edema, and mitral valve regurgitation. Older patients are often asymptomatic at rest and may present with SCD during exertion.⁴⁴ Characteristic findings on ECG of an anterolateral infarct pattern (deep and wide Q waves in leads I, aVL, V5, and V6 with absent Q waves in leads II, III, and aVF and poor R wave progression) along with these examination findings should raise suspicion. In general, however, anomalies may be difficult to diagnose by examination or with a routine echocardiogram and may require additional screening, such as cardiac MRI, computed tomography angiography, or ultimately cardiac catheterization.⁴⁷

Arrhythmic Channelopathies

Determining the contribution of arrhythmic channelopathies in SCD in pediatric and adolescent patients is difficult. Findings on examination or autopsy are often normal, and definitive diagnosis is only made if tissue is available for genetic testing or in cases of elucidative family history. Although there are cases where SCD seems to be the presenting event, often on further review, patients may have had recurrent syncope events or presumed seizures.

Findings of physical examination are usually normal, but the surface 12-lead ECG may offer clues. More common causes of these arrhythmias include LQTS and CPVT.⁴⁸ In LQTS, ECGs

usually have corrected QT intervals that are significantly prolonged (>500 milliseconds), but 10% to 15% of patients with LQTS may have a corrected QT that is normal.⁴⁹ For CPVT, findings of baseline ECGs may be normal; however, during exercise stress testing, ventricular ectopy is enhanced.⁵⁰

Less common causes of arrhythmic channelopathies include SQTS and Brugada syndrome, both of which are associated with SCD as the presenting sign. Presentation of SQTS can be at a very early age, whereas Brugada usually presents in adulthood, with the more severe cases becoming apparent earlier.²⁴ Rare causes of arrhythmia unrelated to channelopathies include Wolff-Parkinson-White syndrome, arrhythmogenic ventricular dysplasia, and complete heart block. Wolff-Parkinson-White is caused by an anatomic anterograde accessory pathway and presents as tachyarrhythmia. Arrhythmogenic ventricular dysplasia, which most frequently presents during adulthood, is a genetically determined cardiomyopathy in which normal ventricular myocardium is replaced by fibrofatty tissue that is ultimately arrhythmogenic.⁴⁸ Complete heart block has a wide variety of causes and usually presents clinically as a bradyarrhythmia.^{24,48}

Dilated Cardiomyopathy

Dilated cardiomyopathy (DCM) is the second most common cardiomyopathy in general populations and the most common in pediatric and adolescent patients, with an incidence of 0.57 per 100,000 individuals.³⁶ Unlike HCM, SCD is not a common presentation in pediatric DCM.^{11,38} The highest risk of death occurs in the first year after diagnosis, generally as a result of heart failure.⁵¹ Two recent studies after pediatric patients with cardiomyopathy showed the long-term incidence for SCD in patients with DCM at 3% to 5%.^{38,52} Etiology of DCM includes familial or genetic, autoimmune, toxin-induced, and infectious myocarditis.⁵³

Findings on physical examination depend on disease progression. In patients with symptomatic heart failure, exaggerated precordial impulse, S3/S4 gallop, organomegaly, and signs of peripheral and pulmonary edema may be present. A mitral valve murmur can be present in cases of extreme LV dilation. Findings on chest radiographs and ECG demonstrate cardiomegaly, and echocardiogram confirms a dilated, poorly functioning left ventricle. Cardiac MRI and cardiac catheterization can be used to further define etiology and to potentially dictate therapy but are not necessary for diagnosis.

Myocarditis

Myocarditis results from infection and/or inflammation of the heart due to a variety of causes and has been stated to be the most common cause of heart failure in a previously healthy pediatric patient.⁵³ Initial presentation may be similar to any routine viral infection with nonspecific prodromal symptoms such as dyspnea, cough, irritability, diarrhea, myalgias, or fever. Respiratory symptoms are most common, with 69% of patients reporting a history of dyspnea. Additional history may include emesis (48%), poor feeding (40%), fever (36%), or lethargy (36%).⁵³

The physical examination in a patient with myocarditis is similar to that in DCM, with tachypnea (60%), hepatomegaly (50%), and tachycardia (32%).⁵³ Diagnostic testing reveals that 63% of patients had cardiomegaly on chest radiograph, 97% with abnormal echocardiogram, and 100% with abnormal ECG, with the rhythm most commonly documented to be sinus tachycardia. Other ECG findings include supraventricular tachycardia, ventricular tachycardia, atrioventricular block, low QRS voltages, T-wave abnormalities, and pseudoinfarcts.⁵³ Cardiac enzymes may be elevated, such as cardiac troponin T.^{54,55} Cardiac troponin T has a sensitivity of 100% and a specificity of 85% for myocarditis when levels are greater than 0.01 ng/mL.⁵⁵ Cardiac MRI can be used to

confirm the diagnosis in suspected myocarditis.⁵⁶ The specific etiology is difficult to define unless a myocardial biopsy is performed with polymerase chain reaction testing for the most common viruses.

Restrictive Cardiomyopathy

Restrictive cardiomyopathy (RCM) accounts for less than 5% of diagnosed cardiomyopathies and is a relatively rare cause of SCD.⁵⁷ Despite this low occurrence, transplant-free survival in RCM is typically low, in part because of arrhythmic events.⁵⁸ Given the low incidence of disease, limited follow-up data are available to track SCA and SCD in patients with RCM. Between a small, single institution series and a larger registry, SCD occurred in 10% to 12% of patients with a long-term follow-up.^{38,59}

Physical examination can include gallop, loud second heart sound (closure of the pulmonary valve or P2), or murmur.⁵⁸ Due to diastolic dysfunction, the most common presentation within a cohort of RCM patients who ultimately experienced SCD was syncope.⁵⁸ Findings of ECG are abnormal in most cases, with evidence for atrial enlargement being pathognomonic. Chest radiograph findings may also be abnormal, showing cardiomegaly, atrial enlargement, or pulmonary venous congestion.^{57,58} Echocardiograms can reveal atrial dilation in the setting of normal sized ventricles, with evidence for elevated filling pressures.⁵⁹ Cardiac catheterization, with measurement of elevated end-diastolic pressures, is diagnostic.

Other Causes

Less-common causes for SCD include congenital heart disease, *C. cordis*, and medication-related. Congenital heart disease poses the challenge of not only structural defects but also conduction abnormalities.⁴⁸ Presentation and diagnosis vary with disease process. *Commotio cordis* is a rare cause of SCD and presents when a blow to the chest occurs during repolarization (the T wave on ECG) and causes VF.⁴⁸ Finally, multiple medications, especially stimulants (such as cocaine) and QT prolonging medications (certain antibiotics, antifungals, and psychotropics, etc at high doses or multiple agents usually in patients with risk factors such as genetic predisposition, cardiac disease, and hypokalemia/hypomagnesemia), can precipitate arrhythmias and cause SCA or SCD.^{7,60}

EMERGENCY INTERVENTIONS

When treating a patient who has recently experienced or is currently in cardiac arrest, emergency care should follow Pediatric Advanced Life Support guidelines.⁶¹ While the patient is being stabilized, a brief history, focused physical examination, and initial diagnostics should be performed. As stated in Pediatric Advanced Life Support guidelines, amiodarone should be considered if a patient has arrhythmias unresponsive to defibrillation.²⁷ Intractable arrhythmias are most commonly seen in patients with HCM. Although taking an extensive history is difficult in the emergency setting, questions about history of present illness, previous history of chest pain, dyspnea and/or syncope during exertion, as well as family history of cardiac illnesses are essential in determining the etiology of the arrest.

Physical examination should focus on circulation, airway, and breathing until the patient is stable; then, a more comprehensive examination including a detailed cardiac examination may reveal pertinent findings. Cardiorespiratory monitors should be placed along with defibrillator pads to monitor for immediate recognition of an abnormal rhythm. Diagnostics should always include a 12-lead ECG and a chest radiograph. If there are additional concerns, point-of-care ultrasound has been shown to be useful

in certain situations such as in HCM; however, this should be interpreted with cardiology consultation.⁶²

If the diagnosis seems to be cardiac, care should be focused on decreasing the potential for a recurring arrhythmia and maintaining cardiac preload. Catecholamine support with agents such as dopamine, dobutamine, epinephrine, and norepinephrine for postresuscitation hypotension should be avoided or used with extreme caution because they may worsen symptoms of obstruction in a patient with HCM or potentiate arrhythmias.^{41,62,63}

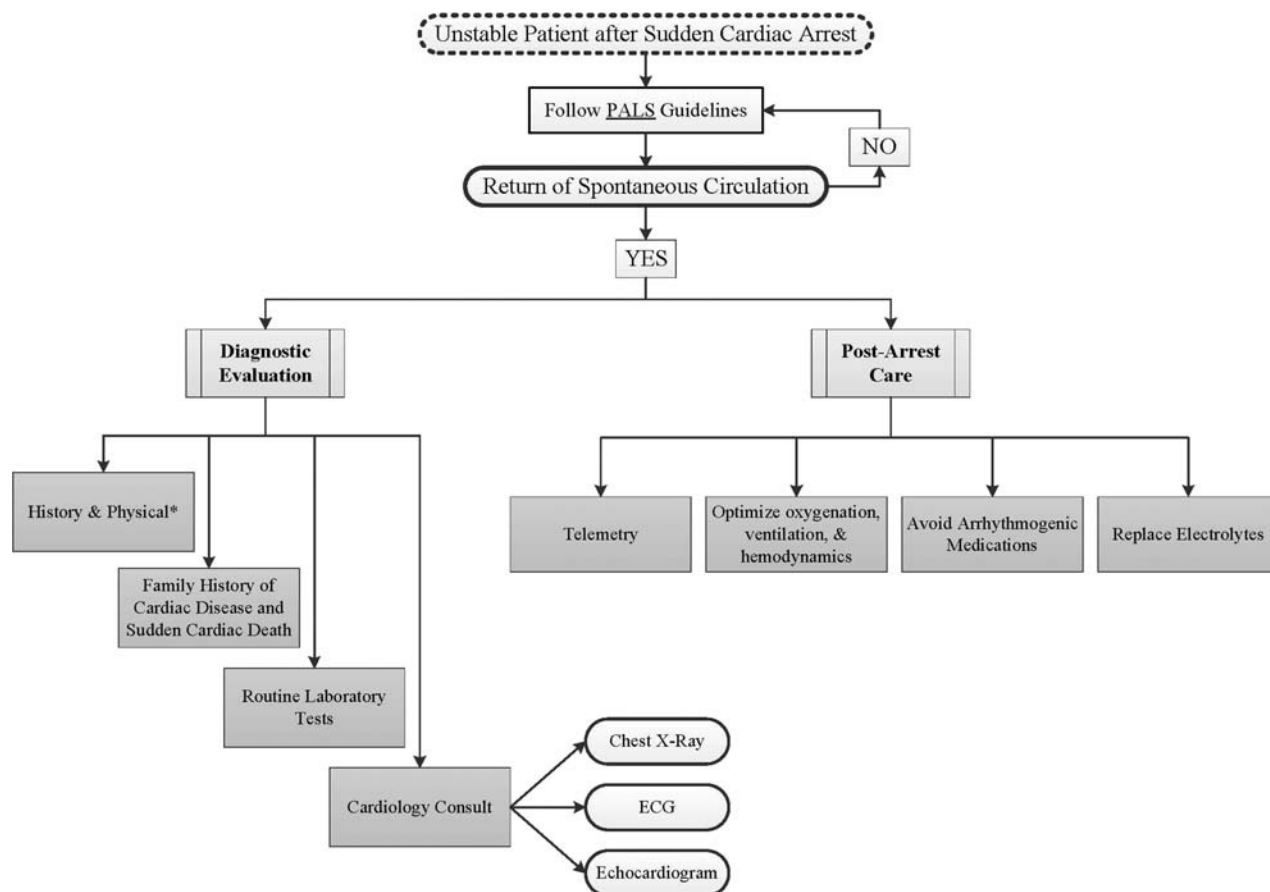
Benzodiazepines should be used with caution because they can result in hypotension, decrease afterload, and subsequently reduce coronary blood flow.⁴² Medications recommended for intubation in patients with cardiac disease include etomidate or fentanyl and midazolam for sedation with vecuronium or rocuronium for paralysis because they may produce less sympathetic response to intubation.^{42,64,65} Vecuronium has been suggested for certain structural heart disease because of its histamine release, vagolytic, and muscarinic activity, whereas some sources recommend rocuronium for arrhythmogenic heart disease because of a lower rate of bradyarrhythmias.^{42,66} A summary of these recommendations can be found in Figure 1.

Prevention of SCA is generally outside the scope of ED care, especially regarding the extensive literature and recommendations on primary prevention screening programs for athletes in an outpatient setting.⁶⁷ However, emergency physicians who treat pediatric patients may be involved in secondary preventative efforts such as evaluation of symptomatic cardiac patients before arrest and community involvement in prehospital care. Evaluation of cardiac complaints, such as exertional fatigue, chest pain, palpitations, and syncope, as well as symptoms such as vomiting, dyspnea, and abnormal movement concerning for seizure should be thoroughly and thoughtfully managed to prevent cases of SCA. It is estimated that 24% of SCA patients have had 1 or more episodes of syncope or unexplained seizure before presentation.⁶⁸ Although cardiac causes of syncope are rare, evaluation by ECG can differentiate these cases from benign causes, with 76% positive for abnormal findings in cardiac syncope versus 0% positive in noncardiac syncope.⁶⁹ In the evaluation of patients with concerning symptoms or family history, standardized screening tools such as the "12-Element American Heart Association Recommendations for Pre-participation Cardiovascular Screening for Competitive Athletes" or the American Academy of Pediatrics "Pediatric Sudden Cardiac Death Risk Assessment Form" are recommended.^{7,70} Prehospital care is vital to a positive outcome, because early cardiopulmonary resuscitation and defibrillation with an automated external defibrillator (AED) can result in survival rates of up to 74%.⁷¹ Efforts to increase AED presence on high school campuses and throughout communities are effective and continue to improve overall survival.⁷²

Even with ideal prehospital and emergency interventions, survival rates for SCA are poor, with an average of 11% survival of those with exercise-related events in school-aged children older than 6 years.⁷³ However, 50% of previously healthy children presenting to a pediatric ICU after an out-of-hospital SCA survived to discharge, with half of the identified etiologies being cardiac.⁷⁴ Of those children who survived, 50% had minimal neurocognitive disabilities with full functional capacity at 6 months; however, many continue to have memory deficits and psychological problems.⁷⁵ Despite this progress, there is a need to improve outcomes of patients presenting with SCA.

CASE RESOLUTION

The child was transferred from the ED to the pediatric ICU, where he was weaned off inotropes and extubated. A transvenous



*History: note dizziness, dyspnea, seizure, syncope, fatigue, abdominal symptoms, changes in weight
Exam: note murmur, gallop, crackles, organomegaly

FIGURE 1. Emergency provider evaluation and initial management of possible pediatric SCA.

single-coil implantable cardioverter defibrillator was placed on day 5 of hospitalization for secondary prevention due to the playground event being most likely a ventricular arrhythmia. He was discharged 6 days after presentation.

At the postresuscitation review of the ED care, there were interventions that were helpful for his condition and others that could have been detrimental. For example, fluids were important to maintain the patient's preload and therefore blood pressure. Medications for possible seizure precautions such as midazolam and fosphenytoin could have deleterious effects on maintaining his blood pressure. Use of an epinephrine drip for pressor support could have worsened afterload or potentiated an arrhythmia. As the patient was stabilized and more detailed history was provided including that the collapse occurred during exertion, a cardiac cause for his SCA became more apparent. In addition, physical examination revealed a murmur, which may have been difficult to detect in the resuscitation room. This case illustrates an episode of SCA in which successful resuscitative efforts prevented a case of SCD.¹

CONCLUSIONS

The presentation of sudden collapse can have myriad etiologies, and the underlying cause difficult to discern on initial evaluation in the ED. Maintaining a wide differential diagnosis and using diagnostics to consider cardiac etiologies are essential. If cardiac etiologies are suspected, deliberate management should

be employed to exclude treatments that could exacerbate the underlying condition. Our understanding of the etiologies of SCD and SCA is improving, and the addition of a collaborative national registry will continue to advance the field.²³

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CME EXAM

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CME EXAMINATION

September 2016

Please mark your answers on the ANSWER SHEET.

Sudden Cardiac Arrest in Pediatrics, *Scheller et al.*

1. What is the most common underlying cardiac structural abnormality that results in sudden cardiac death?
 - A. Myocarditis
 - B. Hypertrophic cardiomyopathy
 - C. Coronary artery anomaly
 - D. Idiopathic
2. The risk for sudden cardiac death in athletes compared with age matched nonathletes is:
 - A. Less than 0.5 times relative risk
 - B. Statistically similar
 - C. More than 2 times relative risk
 - D. Inadequately studied
3. ECGs findings in patients with hypertrophic cardiomyopathy are abnormal in what percentage of patients?
 - A. 1%–10%
 - B. 25%–50%
 - C. 75%–90%
 - D. 100%
4. Which medication(s) should be used with caution in a patient suspected to have arrhythmic etiology for the sudden arrest?
 - A. Dopamine
 - B. Vecuronium
 - C. Etomidate
 - D. A and B
 - E. All of the above
5. Of prevention methods for sudden cardiac death, which is in the scope of an emergency medicine provider?
 - A. School support of AEDs
 - B. Evaluating all athletes with ECGs
 - C. Screening of cardiac complaints
 - D. A and C only
 - E. All of the above

ANSWER SHEET FOR THE PEDIATRIC EMERGENCY CARE CME PROGRAM EXAM September 2016

Please answer the questions on page 637 by filling in the appropriate circles on the answer sheet below. Please mark the one best answer and fill in the circle until the letter is no longer visible. To process your exam, you must also provide the following information:

Name (please print): _____
 Street Address _____
 City/State/Zip _____
 Daytime Phone _____
 Specialty _____

1. ☐ A ☐ B ☐ C ☐ D ☐ E
2. ☐ A ☐ B ☐ C ☐ D ☐ E
3. ☐ A ☐ B ☐ C ☐ D ☐ E
4. ☐ A ☐ B ☐ C ☐ D ☐ E
5. ☐ A ☐ B ☐ C ☐ D ☐ E

Your completion of this activity includes evaluating them. Please respond to the following questions below.

Please rate this activity (1 - minimally, 5 - completely)

Was effective in meeting the educational objectives

1	2	3	4	5
<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>
<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>
<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>

Was appropriately evidence-based

Was relevant to my practice

Please rate your ability to achieve the following objectives, both before this activity and after it:

1 (minimally) to 5 (completely)

Pre					Post				
1	2	3	4	5	1	2	3	4	5
<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>
<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>
<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>

1. Outline the possible cardiac causes of sudden arrest in children and adolescents.

2. Describe emergency care of a pediatric sudden cardiac arrest patient.

3. Identify disease-specific presentation of cardiac causes of sudden arrest in children and adolescents.

How many of your patients are likely to be impacted by what you learned from these activities?

☐ <20% ☐ 20%–40% ☐ 40%–60% ☐ 60%–80% ☐ >80%

Do you expect that these activities will help you improve your skill or judgment

within the next 6 months? (1 - definitely will not change, 5 - definitely will change)

1	2	3	4	5
<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>

How will you apply what you learned from these activities (mark all that apply):

In diagnosing patients ☐

In monitoring patients ☐

In educating students and colleagues ☐

As part of a quality or performance improvement project ☐

For maintenance of board certification ☐

To consider enrolling patients in clinical trials ☐

In making treatment decisions ☐

As a foundation to learn more ☐

In educating patients and their caregivers ☐

To confirm current practice ☐

For maintenance of licensure ☐

Other _____

Please list at least one strategy you learned from this activity that you will apply in practice:

How committed are you to applying these activities to your practice in the ways

you indicated above? (1 - minimally, 5 - completely)

1	2	3	4	5
<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>

Did you perceive any bias for or against any commercial products or devices?

Yes

No

☐

☐

If yes, please explain:

How long did it take you to complete these activities? _____ hours _____ minutes

What are your biggest clinical challenges related to pediatric emergency care?

[] Yes! I am interested in receiving future CME programs from Lippincott CME Institute! (Please place a check mark in the box)

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