Syncope In Pediatric Patients: A Practical Approach To Differential Diagnosis And Management In The Emergency Department

Abstract

Syncope is a condition that is often seen in the emergency department. Most syncope is benign, but it can be a symptom of a life-threatening condition. While syncope often requires an extensive workup in adults, in the pediatric population, critical questioning and simple, noninvasive testing is usually sufficient to exclude significant or life-threatening causes. For low-risk patients, resource-intensive workups are rarely diagnostic, and add significant cost to medical care. This issue will highlight critical diseases that cause syncope, identify high-risk “red flags,” and enable the emergency clinician to develop a cost-effective, minimally invasive algorithm for the diagnosis and treatment of pediatric syncope.
Case Presentations

A 10-year-old previously healthy boy presents after “passing out” and experiencing chest pain while playing basketball with friends earlier that evening. The patient reports occasional chest pain with exertion. Today, he also had chest pain while running, collapsed, and had a loss of consciousness for 4 to 5 seconds. He then returned to baseline. He has no prior history of syncope and no recent infections. He denies drug use. On physical examination, there is no evidence of acute distress, and he has normal pulmonary and cardiac examinations. You immediately order an ECG. Do you also need to obtain troponins, D-dimer, or coagulation studies? Does he also need an echocardiogram? You want the patient to see a cardiologist, but does this need to happen in the middle of the night?

A 16-year-old previously healthy adolescent girl presents with multiple episodes of syncope over the last 24 hours. Her preceding symptoms include the sensation that her heart was racing, seeing spots in her visual fields, and feeling short of breath. She had been feeling unwell for 4 days with a dry cough, but no other cold-like symptoms. The first episode of syncope occurred the previous night after getting up from seated position and walking. Her second episode of syncope was this morning, again, after getting up and walking. She had her third episode of syncope today while seated on a couch. This episode was witnessed by friends who state she was unconscious for a few seconds. She denies any pain with these episodes. She currently has no chest pain, but feels short of breath. She has no risk factors for pulmonary embolism and no family history of early cardiac death or clotting disorders. Her last menstrual period was 2 weeks ago. As you order an ECG and a pregnancy test, you think about what else you need to do for this patient.

An 18-year-old previously healthy adolescent girl presents after fainting. She was standing and waiting for the subway when she “felt the room closing in” and the “world going dark.” The next thing she remembers is lying on the ground with people looking down at her. The patient reports recent cold symptoms and decreased appetite. She denies the use of drugs or alcohol. She denies pregnancy. This has happened one other time, several years ago. Now she feels she has returned to her baseline. What diagnostic testing is helpful in the diagnosis of this patient? What further evaluation does she need, if any? How should she be managed? Does she need admission?

Introduction

Syncope is defined as a transient loss of consciousness and postural tone due to an alteration in cerebral perfusion, usually associated with spontaneous recovery. In pediatric patients, syncope is most often a brief episode with complete recovery, without sequelae. These typical episodes, however, must be differentiated from those with rare, life-threatening etiologies. Syncope is most common in teenagers, with the incidence peaking in patients aged between 15 and 19 years. Fifty percent of people report an episode of syncope during adolescence. Syncope accounts for 1% to 3% of emergency department (ED) visits, with an overall incidence in the pediatric population of 0.1% to 0.5%. Syncope must be distinguished from all other causes of loss of consciousness, such as seizures, head trauma, and psychiatric causes.

There are many ways to classify syncope, but the simplest is to divide the causes of syncope into 2 groups: cardiac and noncardiac etiologies. While cardiac causes represent the minority of syncope cases, they should not be missed, as they can result in sudden death. As many as 1% to 5% of syncopal events may be related to underlying cardiac disease. This issue of Pediatric Emergency Medicine Practice will help emergency clinicians develop a broad differential diagnosis, use a classification scheme to identify the causes of syncope, and determine appropriate evaluation of the patient. Most importantly, this issue will help emergency clinicians identify the red flags for etiologies that must not be missed in the evaluation of pediatric syncope in the ED.

Critical Appraisal Of The Literature

A literature search was performed in PubMed using the terms syncope, fainting, blackout, and vasovagal. The search was limited to articles published since 1960 that involved patients aged 0 to 18 years. The term emergency department was also included in a subsequent search. This search was limited by age, English language, and human subjects. These searches identified several thousand articles that were screened by title, which resulted in approximately 150 articles that were considered for inclusion. The Cochrane Database of Systematic Reviews, Evidence-Based Medicine Reviews: Best Evidence (American College of Physicians), Database of Abstracts of Reviews of Effectiveness (DARE), and the National Guideline Clearinghouse were all queried for articles related to syncope in adults or children. The results of these queries produced more than 130 articles that were reviewed in full.

Etiology And Pathophysiology

Syncope, while technically an ICD (International Statistical Classification of Diseases) diagnosis, is the presenting symptom of relative cerebral hypoperfusion that is the result of neurocardiogenic, cardiac, neurologic, metabolic, toxin-mediated, or psychiatric etiologies. With respect to the pediatric population, syncope is more common in the adolescent population (ages 13 to 18 years) and is more common in girls than boys. In the adolescent population, syncope most commonly results from a benign etiology and is most often neurocardiogenic in nature. Young
children rarely present with syncope, except in the setting of breath-holding spells, seizures, and cardiac dysrhythmias. In young children, unless the history is classic for a breath-holding spell, additional evaluation is warranted.

**Pathophysiology Of Neurocardiogenic (Vasovagal) Syncope**

Neurocardiogenic syncope (NCS), or vasovagal syncope, is the most common cause of pediatric syncope, accounting for 60% to 80% of syncope cases. The loss of consciousness caused by NCS is due to cerebral hypoperfusion. In NCS, cerebral perfusion decreases 30% to 50% from baseline. The body has multiple compensatory measures to prevent this drop. When standing from a sitting or lying position, the decreased venous return results in a decreased stroke volume. In order to compensate for this subsequent drop in mean arterial pressure, high-pressure receptors in the aortic arch and carotid sinus, as well as low-pressure receptors in the lungs and heart, are activated. This activation of the sympathetic nervous system results in a compensatory increase in heart rate and blood pressure. Additionally, venous return is increased through muscle contraction in the distal extremities, which increases peripheral vascular resistance. If these compensatory measures are engaged, cerebral perfusion is maintained and syncope is prevented. When these measures are altered or are insufficient, syncope occurs.

Cerebral hypoperfusion occurs in the setting of autonomic dysregulation with inappropriate withdrawal of sympathetic stimulation and increased vagal stimulation. This manifests in 3 ways: (1) inadequate compensatory tachycardia, (2) insufficient vascular tone, and (3) decreased cardiac output.

Cerebral vascular perfusion is particularly sensitive to changes because, in the standing position, the mean cerebral pressure is only slightly above the lower limit of cerebral autoregulation. The most important sympathetic receptors in preventing syncope are those in the carotid sinus that affect peripheral vascular resistance, where 60% of the body’s blood volume resides.

**Differential Diagnosis**

Differentiating cardiogenic syncope from the other causes can be especially challenging for the emergency clinician. Several highly publicized cases have drawn attention to life-threatening diagnoses such as hypertrophic cardiomyopathy (HCM) and cardiac dysrhythmias. Although these make up a small proportion of syncope cases that present to the ED, they account for the vast majority of morbidity and mortality in the pediatric population. Extensive evaluation of children presenting with syncope is low-yield and often costly. Fortunately, several simple historical and physical examination features can aid the emergency clinician in distinguishing between cardiac and noncardiac syncope. Table 1 notes various etiologies of syncope.

**Cardiogenic Etiologies Of Syncope**

**Hypertrophic Cardiomyopathy**

Hypertrophic cardiomyopathy (HCM) is a rare, genetic, structural heart condition that has received substantial public attention. One of the most famous cases was the sudden unexpected death of basketball player Reggie Lewis in 1993; additional cases in other famous athletes have occurred since then. HCM is the most commonly identified cause of sudden death in athletes. It is frightening, as it can afflict athletes or young adults in their prime. Despite the public perception of its high mortality rate, the actual prevalence of this disease is relatively low. The true prevalence of HCM in the pediatric population is not certain, but some studies have identified the prevalence in young adults to be 1 in 500.

HCM is defined as an asymmetric, hypertrophied, and nondilated left ventricle without other underlying cardiac disease. It is caused by disorganized, irregular, and hypertrophied cardiac myocytes. When these myocytes undergo necrosis, they are replaced by fibrotic tissue, which creates hypertrophy of the left ventricular wall that is typically asymmetric and preferentially affects the septum or apex. Once this hypertrophy becomes larger, it can cause left ventricular outflow tract (LVOT) obstruction. Heart function becomes preload-dependent to overcome the obstruction. Persistent obstruction can lead to additional ventricular hypertrophy due

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**Table 1. Etiologies Of Syncope**

<table>
<thead>
<tr>
<th>Cardiac Causes</th>
<th>Noncardiac Causes</th>
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<tbody>
<tr>
<td><strong>Structural</strong></td>
<td>Neurocardiogenic/vasovagal (most common)</td>
</tr>
<tr>
<td>• Hypertrophic cardiomyopathy</td>
<td>Postural orthostatic tachycardia syndrome</td>
</tr>
<tr>
<td>• Anomalous coronary artery*</td>
<td>Psychogenic pseudosyncope</td>
</tr>
<tr>
<td>• Myocarditis*</td>
<td>Drugs and toxins*</td>
</tr>
<tr>
<td>• Pericarditis*</td>
<td>Metabolic derangements, hypoglycemia</td>
</tr>
<tr>
<td>• Valvular dysfunction*</td>
<td>Breath-holding spells</td>
</tr>
<tr>
<td><strong>Dysrhythmias</strong></td>
<td>Seizure</td>
</tr>
<tr>
<td>• Commotio cordis*</td>
<td>Trauma</td>
</tr>
<tr>
<td>• Long QT syndrome</td>
<td>Pregnancy</td>
</tr>
<tr>
<td>• Wolff-Parkinson-White syndrome</td>
<td>Pulmonary hypertension*</td>
</tr>
<tr>
<td>• Supraventricular tachycardia</td>
<td>Pulmonary embolism*</td>
</tr>
<tr>
<td>• Brugada syndrome</td>
<td></td>
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<tr>
<td>• Short QT syndrome*</td>
<td></td>
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<tr>
<td>• Heart block</td>
<td></td>
</tr>
<tr>
<td>• Sick sinus syndrome*</td>
<td></td>
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<tr>
<td>• Arrhythmogenic right ventricular dysplasia*</td>
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</table>

*Not discussed in detail in this article.
to the increased force needed by the left ventricle to overcome the LVOT obstruction, and these cellular changes can generate dysrhythmias, heart failure, and sudden death. The dysrhythmias associated with HCM and sudden death are ventricular tachycardia and ventricular fibrillation. There are over 450 genetic mutations that have been found to be associated with HCM. Of patients with an identified genotype, 70% have a mutation in either the beta-myosin heavy chain or the myosin-binding protein in cardiac myocytes. Pathogenic mutations are inherited in an autosomal-dominant pattern.13,14

There are varying types of HCM that can present with different degrees of obstruction and symptomatology. Many patients with HCM can live for years without symptoms. For adults, the mortality rates range from 1% to 6% per year. Higher mortality is associated with HCM in patients aged < 20 years. Sudden death in the setting of HCM is more prevalent in the younger population (< 30 years) and is equal between genders. In the pediatric population, HCM tends to be associated more with a syndromic phenotype, such as underlying genetic or neuromuscular syndrome, whereas in the adult population, HCM tends to be a primary disease.

HCM causes sudden death in approximately 5% of cases, but in cases of sudden cardiac death among athletes and young people, it is the most common identified cause. Sudden death is often the first clinical manifestation of HCM. In a study by Maron et al, HCM was the identifiable cause in 36% of cases of sudden pediatric death.15

Patients with HCM who do not present with sudden death typically present for evaluation of an incidental murmur or positive family history; this includes most patients. Patients presenting in distress, however, are likely to have symptoms such as heart failure, syncope, arrhythmias, or cardiac arrest. Patients who present with these symptoms have a poorer prognosis.13 Of those patients who present with syncope to the pediatric ED, the number of patients identified as having HCM is exceptionally low, but could be determined by taking a thorough family history of cardiac disease and sudden death, and by abnormal ECG findings. For a patient with HCM, the physical examination may be notable for a systolic murmur associated with a diastolic murmur or crescendo-decrescendo murmur. Patients presenting with these symptoms have a poorer prognosis.13

Because many QT-prolonging medications are regularly administered in the ED, providers must be aware of the potential for the cumulative effects of these medications in prolonging the QT interval, in addition to any home medications. It may be prudent to use these medications with caution in patients with a history of syncope or to consider obtaining an ECG prior to administration.

**Long QT Syndrome**

Long QT syndrome is a potentially fatal cardiac dysrhythmia that can be either acquired or congenital. For children with long QT syndrome, there is an alteration in cardiac cell membrane ion channel permeability that results in prolonged depolarization that is seen on ECG as a prolonged QT interval. (See Figure 2, page 6.) This abnormality has varying degrees of clinical manifestation, but usually presents between the ages of 9 and 15 years, with seizures, syncope, or sudden death. The most commonly described dysrhythmia associated with long QT syndrome is a polymorphic ventricular dysrhythmia, or torsades de pointes, which describes the oscillating QRS axis. The diagnosis of long QT syndrome is based on an abnormal 12-lead ECG obtained at rest.16

The QTc (corrected for heart rate) interval can be calculated by dividing the QT interval by the square root of the preceding R-R interval. If this value is > 460 milliseconds, it is considered abnormal or prolonged.17,18 Dysrhythmias are most often associated with a QTc interval > 500 milliseconds.19

Congenital long QT syndrome is associated with 2 syndromes: Jervell and Lange-Nielsen syndrome (which is also associated with congenital deafness and higher rates of sudden death) and Romano-Ward syndrome.20 The genetic risk associated with these syndromes makes obtaining a family history a crucial aspect of the syncope evaluation. Women are known to have an increased likelihood of recurrent cardiac events and are at an increased risk of developing syncope or sudden death, especially during menses.20-23

The frequency of a medication-induced long QT interval is uncertain but tends to be associated with cumulative QT-prolonging medications or an underlying risk of long QT syndrome. (See Table 2, page 7.) Because many QT-prolonging medications are regularly administered in the ED, providers must be aware of the potential for the cumulative effect of these medications in prolonging the QT interval, in addition to any home medications. It may be prudent to use these medications with caution in patients with a history of syncope or to consider obtaining an ECG prior to administration.

**Wolff-Parkinson-White Syndrome And Narrow QRS Complex Tachycardia**

Wolff-Parkinson-White (WPW) syndrome is an accessory pathway-induced re-entrant tachycardia, either anterograde (90%) or retrograde (10%). The typical WPW pattern on ECG (a short PR interval and associated ventricular pre-excitation as seen by the delta wave) (see Figure 3, page 8), is found in approximately 0.2% of the population, but only a
small proportion of these patients will be symptomatic. Not all patients with WPW syndrome will have the classic ECG findings, unless the accessory pathway is activated. The accessory pathway is most likely to be activated during periods of tachycardia. In a prospective study published in 2013, 446 children from a cardiology database were identified as having WPW. Of this identified cohort, 4% were diagnosed after presenting with syncope. Although WPW is a rare cause of syncope, it is often identifiable by screening ECG. In fact, 25% of the patients within the previously mentioned cohort were diagnosed with WPW based on an incidental ECG finding.

The most common tachydysrhythmia in WPW is anterograde atroventricular re-entrant tachycardia, which produces a narrow QRS complex tachycardia that can evolve into a ventricular tachycardia. There is also the less-common retrograde conduction from the atrioventricular node to the atria, generating a wide QRS complex tachycardia that is difficult to distinguish from polymorphic ventricular tachycardia. Santinelli et al assessed 184 asymptomatic children with WPW who were diagnosed by screening ECG in Italy, and 10% had a potentially treatable dysrhythmia. These identified dysrhythmias had a variety of presenting symptoms that ranged from cardiac arrest or syncope to mild or atypical symptoms such as gastrointestinal distress. This study helped to emphasize the significant morbidity and mortality of WPW. The current interventional cardiology practice includes more aggressive attempts to induce and ablate these accessory pathways.

Supraventricular Tachycardia
Supraventricular tachycardia (SVT) is the most common symptomatic pediatric dysrhythmia. SVT is defined as a narrow QRS complex tachycardia, with a rate > 220 beats/min in infants or > 180 beats/min in children and adolescents. In addition, P waves are absent and there is a lack of beat-to-beat variability. The compromised cardiac output that can result from SVT may lead to syncope. SVT can be induced by caffeine intake, which has become increasingly common in adolescents, given that energy drinks

Figure 1. Hypertrophic Cardiomyopathy On Electrocardiogram

Normal sinus rhythm, left anterior hemiblock, left ventricular hypertrophy, right ventricular hypertrophy, nonspecific T-wave abnormalities in anterior (V3, V4), and lateral leads (V5, V6).
Image courtesy of Manuella Lahoud-Rahme, MD and Joseph Spinner, MD.
can have > 200 mg of caffeine in 1 to 2 ounces of volume. Questions about caffeine use as well as illicit drug use should be part of the history taken for all children presenting with syncope.

**Heart Block**

Heart block occurs in several forms and may also cause syncope. (See Figure 5, page 9.) First-degree heart block, or PR prolongation without dropped ventricular beats, is often asymptomatic. Second-degree heart block is either Mobitz I or II, with the latter being more likely to progress to third-degree or complete heart block. Mobitz I is characterized by gradual lengthening of the PR interval until a ventricular beat is dropped, whereas Mobitz II exhibits stable PR intervals and ventricular beats are dropped in a specified ratio to P waves that are conducted (eg, 3:1, 4:1, etc). Third-degree or complete heart block occurs when there is atrioventricular node dysfunction and the electrical activity of the atria is no longer communicating with the ventricles and the ventricular beats are generated from the ventricle itself.

Heart block can be either congenital or acquired.

Complete heart block is the most common cause of symptomatic bradycardia in children and infants. In children, acquired heart block can be due to an infectious cause. Heart block can also occur as a result of iatrogenic causes such as medications or as a result of prior heart surgery. Additionally, it can be associated with a syndrome such as lupus erythematosus.

Delayed conduction from any form of heart block can result in symptomatic bradycardia that can decrease cardiac output enough to cause syncope. High-degree heart block (ie, second-degree type II or third-degree) is more dangerous than low-degree heart block (first-degree or second-degree type I), with high-degree heart block more likely to evolve into ventricular dysrhythmias and sudden death.

**Brugada Syndrome**

Brugada syndrome was first described in 1992 as a cause of sudden cardiac death. It is a rare, autosomal-dominant disease, with an estimated pediatric prevalence of 0.0098% in one study population and 0.0005% in the general population in another study. Overall, Brugada syndrome has a poorly defined...
prevalence in children. Since the classic ECG findings are only intermittently present, the true prevalence is unknown. The syncope and sudden death that results from symptomatic disease may occur at rest, during sleep, or with hyperpyrexia, large meals, cocaine use, or excessive alcohol consumption. In the pediatric population, fever is a well-documented precipitant of cardiac events, and it is recommended that children with Brugada syndrome be treated early with antipyretics and brought to the hospital for cardiac evaluation, ECG, and cardiac monitoring during febrile episodes. Although age of onset is typically in the third to fourth decade of life, neonatal and pediatric cases of Brugada syndrome are well documented.

Brugada syndrome is defined by ECG findings of ST-segment elevations of at least 2 mm in at least 2 of 3 right precordial leads with a “coved morphology,” followed by a negative T wave with little or no isoelectric separation (type 1) or a saddle-back appearance with a high take-off ST segment followed by a descending ST segment (type 2). Type 3 may have either a coved or saddleback appearance with < 1 mm of ST-segment elevation.

In the largest pediatric study to date, with 30 children aged < 16 years, syncope was the most common presenting symptom in one-third of the cases. Thirteen percent of the patients had associated atrial fibrillation or flutter, which are very uncommon pediatric dysrhythmias. Half of the cases in this study were detected by family screening.

Patients with spontaneous (not medication-induced) type 1 ECG findings and patients with syncope are at high risk for malignant dysrhythmias.

Other Cardiogenic Etiologies

Additional cardiogenic causes of syncope include anomalous left coronary artery, commotio cordis, myocarditis, and pericarditis. These topics have been covered in previous issues of Pediatric Emergency Medicine Practice. For more information regarding the management of these conditions, please see the following Pediatric Emergency Medicine Practice issues:

- “Congenital Heart Disease In Pediatric Patients: Recognizing The Undiagnosed And Managing Complications In The Emergency Department,” May 2016, available at: www.ebmedicine.net/CHD

Noncardiac Etiologies Of Syncope That Require Further Evaluation

Pregnancy And Ectopic Pregnancy

Pregnant adolescents make up a small proportion of pediatric ED visits (< 1%), but adolescent pregnancy accounts for significant morbidity in the pediatric population, with an incidence of teen pregnancy of 57.4 per 1000 adolescents in the United States. Syncope and near-syncope are common in pregnancy, particularly during the third trimester. Of all pregnant women, 5% experience syncope and almost 30% of pregnant women experience presyncope.

Menstrual history should be obtained and documented in all adolescent girls; however, it should not be used to determine whether testing will be performed, as adolescent sexual history can be unreliable. Given the unpredictable nature of adolescent menstrual cycles, the high risk of missing a pregnancy, and low-cost, noninvasive testing methods (point-of-care urine analysis), a urine human chorionic gonadotropin (HCG) pregnancy test is indicated in all girls of reproductive age. Because the age of menarche is variable, and a patient can become pregnant during her first cycle, assessment of Tanner staging is useful when determining a patient’s potential for pregnancy. Menarche does not occur until late in Tanner stage III. All patients with appropriate Tanner staging could, theoretically, be pregnant.

Ectopic pregnancy occurs at a rate of 19.7 per 1000 pregnancies; a ruptured ectopic pregnancy can be life-threatening. Pregnancy-associated syncope can occur with ectopic pregnancy and ruptured ectopic pregnancy, resulting in hemorrhage and

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Table 2. Medications That Prolong The QT Interval

<table>
<thead>
<tr>
<th>Medication Type</th>
<th>Drugs Commonly Used In The Pediatric Population</th>
</tr>
</thead>
<tbody>
<tr>
<td>Macrolides (account for 77% of antimicrobial-induced torsades de pointes)</td>
<td>Azithromycin, Erythromycin, Clarithromycin</td>
</tr>
<tr>
<td>Fluoroquinolones</td>
<td>Levofloxacin, Moxifloxacin</td>
</tr>
<tr>
<td>Antifungals</td>
<td>Voriconazole, Fluconazole, Ketoconazole</td>
</tr>
<tr>
<td>Gastrointestinal agents</td>
<td>Ondansetron, Metoclopramide</td>
</tr>
<tr>
<td>Psychiatric medications</td>
<td>Haloperidol, Lithium, Quetiapine, Risperidone, Citiplompr, Escitalopram</td>
</tr>
<tr>
<td>Other</td>
<td>Trimeprprim-sulfamethoxazole, Tacrolimus, Methadone, Chloroquine</td>
</tr>
</tbody>
</table>
Hypoglycemia

Syncope in the setting of hypoglycemia is rare in the adult population, comprising approximately 1%, in one study population. The incidence of hypoglycemia as a cause of syncope in the pediatric population is uncertain, but, in one study of adults by Massin et al, the combined incidence was < 3 per 226. (However, hypoglycemia was not specifically separated out from other causes.) Because syncope is, by definition, a transient phenomenon, it is unlikely to be due to hypoglycemia, which would cause persistent symptoms until the hypoglycemia was reversed. In the 2009 European Society of Cardiology guidelines on syncope, hypoglycemia was not even included as a cause. Hypoglycemia is most common in diabetic patients and, within that subgroup, it is more common among the elderly – not pediatric patients. Standard testing of glucose is therefore not recommended in routine syncope evaluation.
Figure 5. Types Of Heart Block On Electrocardiogram

A. First-degree heart block

Arrow indicates the buried P wave.

B. Second-degree Mobitz I heart block

Oval indicates the P wave without a subsequent QRS complex in a random fashion.

C. Second-degree Mobitz II heart block

Ovals indicate the lack of a QRS complex after the P wave in specific intervals.

D. Third-degree heart block

Independent P waves and QRS complexes indicative of ventricular beats generated from the ventricles.

All images used with permission from www.lifeinthefastlane.com.
Clinical Pathway For The Management Of Pediatric Patients With Syncope

Patient presents to ED for episode of syncope

- Obtain history\(^b\) (Class II)
- Perform physical examination\(^c\) (Class II)
  
Patient returned to baseline?

- Follow PALS guidelines
- Perform primary assessment
- Obtain focused history\(^a\) (Class II)

Spontaneous return to consciousness?

- Obtain additional history\(^b\)
- Perform physical examination\(^c\) (Class II)

Obtain cardiology or neurology specialist examination to determine disposition

- Initiate care as warranted

Abnormal cardiac or neurologic examination?
- Known cardiac disease?

Obtain ECG (Class II-III)

Abnormal findings?\(^d\) (Class II)

Pregnancy possible?

- Administer supportive care
- IV fluids if symptomatic hypovolemia
- Consider review of ECG by pediatric cardiologist

- Discharge if no red flags\(^e\) and patient is asymptomatic
- Recommendations to patient and caregiver:
  - Ensure adequate oral intake
  - Avoid provoking events
  - Recognize vasovagal symptoms

- Perform obstetric assessment, rule out ectopic pregnancy, involve OB as indicated, discharge if stable
- Refer to obstetrician/gynecologist
- Consider social services involvement

POSITIVE

Obtain urine HCG testing

NEGATIVE

\(^a\)Focused history: onset of symptoms, details of events, underlying medical problems, allergies, medications, last meal.

\(^b\)History to determine etiology: personal or family history of seizures, metabolic disease, or diabetes; history of medication or toxic ingestion, history of recurrent syncope, menstrual history in females, history of trauma. See Table 3, page 12, for additional history details.

\(^c\)Physical examination: general appearance, full set of vital signs, injuries, neurologic examination (intracranial injury, seizure), cardiac examination (heart rate, regularity, murmurs, and other sounds such as rubs or gallops).

\(^d\)Positive findings of any one of the following is indicative of a cardiac etiology: (1) any syncope that surrounded activity, (2) a family history of cardiac disease or sudden death, (3) physical examination findings suggestive of cardiac disease, and (4) an abnormal ECG.

\(^e\)Red flags: Multiple episodes of syncope in a short time period, syncope associated with chest pain, syncope occurring during exercise or while sitting, syncope in patients with a family history of cardiac disease, deafness, or sudden death.

Abbreviations: ECG, electrocardiogram; ED, emergency department; HCG, human chorionic gonadotropin; IV, intravenous; OB, obstetrician; PALS, pediatric advanced life support.
for children. However, if on careful history there is report of prolonged fasting, drug ingestion, diabetes, or metabolic disease, point-of-care glucose testing may be useful to guide treatment. 

Outside of the hospital, hypoglycemia is often diagnosed in the field with a fingerstick glucose measurement by emergency medical services (EMS) providers and is treated prior to arrival to the ED. Unless the patient is still symptomatic (ie, with lethargy or confusion), routine glucose testing in the ED is unlikely to be diagnostic, as glucose levels will have likely normalized. A study by Salins et al showed that while patients may experience transient hypoglycemia during a syncopal episode, their glucose levels returned to normal after the event once their symptoms subsided. 

Other Noncardiac Etiologies
Syncope may also be caused by ingestion of drugs or toxins, but would present with signs and symptoms related to the associated toxidromes. It is possible for syncope to occur in the setting of metabolic derangements, but since metabolic derangements are not typically transient, they are more likely to cause altered mental status, lethargy, or coma than the transient loss of consciousness that defines syncope. The routine testing of electrolytes in the workup of syncope has not been shown to contribute to diagnosis or management.

Noncardiac, Non–Life-Threatening Etiologies Of Syncope
Neurocardiogenic Syncope
NCS is the most common cause of pediatric syncope, and it is benign. NCS tends to present in 3 phases: prodrome, loss of consciousness, and recovery. Often, a good history of prodromal symptoms can elucidate the diagnosis. Typical triggers for NCS can include postural changes, prolonged sitting or standing, or noxious stimuli. NCS can occur in otherwise-healthy patients in the setting of exercise (due to venous pooling), hyperthermia, hypovolemia or dehydration, and hyperventilation. These key historical findings must be distinguished from syncope that occurs during exertion (“mid-stride” syncope) that is more worrisome for a cardiac etiology and would not have the same prodromal symptoms. Associated prodromal symptoms for NCS include lightheadedness, headache, vision or hearing changes, pallor, nausea, and diaphoresis, and they are more likely to occur with NCS than with syncope from a cardiac etiology. The neurologic symptoms of headache, visual changes, and lightheadedness are thought to be directly related to the transient cerebral hypoperfusion that occurs during syncope.

The environment and preceding events are also key to taking a prodromal history. In a study by Tretter and Kavey, a history of prolonged standing prior to a syncopal episode was present in 82% of NCS patients, but was not present in any patients who experienced cardiac syncope. In addition, fewer patients with NCS had loss of consciousness during activity or during peak activity as opposed to patients with cardiac syncope. Acute stressors such as anxiety, pain, and strong emotions can also trigger syncope. This stress response is postulated to trigger a parasympathetic firing of the autonomic nervous system, resulting in syncope. A detailed history to document volume status to assess for hypovolemia as a contributing factor can also support the diagnosis of NCS.

The second phase of NCS is the transient loss of consciousness. Typically, once the patient is supine, cerebral hypoperfusion is reversed and symptoms resolve in seconds to minutes, with complete resolution in 1 to 2 minutes. Most of the morbidity of NCS occurs during the loss-of-consciousness phase, particularly if the patient sustains a head injury or injures another body part. Cases of traumatic injuries have been reported. In a study by Johnson et al, 12% of patients had injury with syncope. A detailed history, particularly from bystanders, is helpful to determine whether additional injury was sustained during the syncopal event. During the loss-of-consciousness phase, the patient may appear

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Class Of Evidence Definitions

Each action in the clinical pathways section of Pediatric Emergency Medicine Practice receives a score based on the following definitions.

**Class I**
- Always acceptable, safe
- Definitely useful
- Proven in both efficacy and effectiveness

**Level of Evidence:**
- One or more large prospective studies are present (with rare exceptions)
- High-quality meta-analyses
- Study results consistently positive and compelling

**Class II**
- Safe, acceptable
- Probably useful

**Level of Evidence:**
- Generally higher levels of evidence
- Nonrandomized or retrospective studies: historic, cohort, or case control studies
- Less robust randomized controlled trials
- Results consistently positive

**Class III**
- May be acceptable
- Possibly useful
- Considered optional or alternative treatments

**Level of Evidence:**
- Generally lower or intermediate levels of evidence
- Case series, animal studies, consensus panels
- Occasionally positive results

**Indeterminate**
- Continuing area of research
- No recommendations until further research

**Level of Evidence:**
- Evidence not available
- Higher studies in progress
- Results inconsistent, contradictory
- Results not compelling
pale, diaphoretic, or clammy, and may even have abnormal movements. The patient typically does not remember this phase of the event.

In the third, recovery phase of NCS, the patient returns to baseline over the subsequent 5 to 30 minutes, and during this time the patient may experience symptoms of headache, nausea, weakness, or fatigue.

Often, NCS can be recurrent. Evaluation and diagnosis in the ED should include a thorough history of the event with emphasis on the typical prodromal symptoms described previously. A history negative for red flags (see Table 3) (including a family history of cardiac disease, syncope occurring during exertion or while sitting, and associated chest pain) is also key to the diagnosis of NCS. Physical examination should be thorough and should focus on pertinent negatives, as patients with NCS have a normal physical examination, barring any trauma sustained during the episode. Normal vital signs, neurologic examination, and cardiac examination are reassuring. While used in the past, the head-up tilt-table test is not necessary for the diagnosis of NCS.

Particularly in the ED setting, this test is time and labor-intensive and does not add value over a thorough history and physical examination.

Seizure

Seizure is often high on the differential diagnosis of syncopal events. Syncope from various causes can be convulsive, with transient stiffening, clonus, or myoclonus due to the transient cerebral hypoxia that suppresses the limbic and cortical structures. These movements occur after the loss of consciousness. In contrast, during a seizure, the abnormal movements occur before or at the onset of loss of consciousness. The abnormal movements during syncope tend to be few or brief rather than sustained tonic-clonic seizure activity. Furthermore, the prodrome prior to typical NCS is different from the symptoms associated with seizure. The associated symptoms more consistent with seizure include supine posture; warm, flushed, or cyanotic skin; loss of bowel function or bladder function; and tongue biting. Additionally, a true seizure will have a postictal phase, characterized by lethargy or confusion, while syncope may be followed by headache, nausea, or weakness—a subtle, yet important, distinction.

Breath-Holding Spells

Breath-holding spells are a common cause of syncope in toddlers. Breath-holding typically occurs in young children who begin to cry either after injury or while frightened or frustrated. In the setting of this crying episode, the child then becomes quiet and cyanotic or pallid and goes limp, often with loss of consciousness. The pathophysiology is unclear. Initial episodes most often occur before 2 years of age and are most often not associated with other developmental abnormalities. The child usually stops crying in full expiration with mouth open. Loss of consciousness lasts for < 1 minute. The patient should then return to baseline. These episodes can be associated with posturing, body “jerks,” or incontinence. In general, workup is not required if the history is consistent. Reassurance is the best treatment, although since anemia has been associated with these episodes, complete blood cell count testing may be considered either in the ED or as an outpatient.

Postural Orthostatic Tachycardia Syndrome

Postural orthostatic tachycardia syndrome (POTS) is similar to NCS in that they can both result in syncope and presyncope through the presumed mechanisms of cerebral hypoperfusion. Distinct from NCS, however, POTS does not produce observed changes in blood pressure. In POTS, symptoms are characterized by sympathetic activation with palpitations, chest pain, vasomotor symptoms, or tremulousness elicited by orthostatic changes. When standing, patients can also experience symptoms of orthostatic intolerance, such as syncope and near-syncope, dizziness, light-headedness, visual changes, weakness, headache, and nausea. Patients may also have chronic fatigue. In patients with POTS, orthostatic changes elicit a change in heart rate by > 30 beats/min or an absolute systolic blood pressure > 120 mm Hg within 10 minutes of active standing. These changes may cause presyncope and syncope in patients with POTS, but unlike patients with NCS, there is no associated orthostatic hypotension, or only modest hypotension. The diagnosis is based on the chronicity of sympathetic activation symptoms such as palpitations, chest pain, or tremulousness, as well as the subjective symptoms of orthostatic intolerance.

The estimated prevalence of POTS in adults is 170 per 100,000, but the incidence is unknown in the pediatric population. Patients with POTS are most commonly aged between 12 and 40 years.

<table>
<thead>
<tr>
<th>Table 3. “Red Flags” And “Green Lights” In Patients With Syncope</th>
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<tbody>
<tr>
<td><strong>Red Flags</strong></td>
</tr>
<tr>
<td>• Multiple episodes in a short time period</td>
</tr>
<tr>
<td>• Associated chest pain</td>
</tr>
<tr>
<td>• Episode occurred during exercise or while sitting</td>
</tr>
<tr>
<td>• Family history of cardiac disease, deafness, or sudden death</td>
</tr>
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</table>

Red flags are worrisome aspects of the history. Green lights are reassuring aspects of the history that suggest neurocardiogenic syncope.
and there is a 4:1 female-to-male prevalence. In a study by Jajour of adolescents with POTS, the most common symptoms were chronic fatigue, nausea, and disordered sleeping. Patients with POTS may present to the ED with syncope and presyncope. A thorough history of the chronicity of the symptoms as well as vital sign abnormalities are sufficient to diagnose this syndrome. Typically, the patient history is notable for a stressor around the time of onset of these symptoms. Occasionally, these patients go on to present with subsequent alternative pseudoneurologic disorders that are not attributed to an organic etiology. In patients with psychogenic pseudosyncope, a thorough history is key to the diagnosis. As compared with patients who experience true loss of consciousness with syncope, these patients tend to have longer periods

**Psychogenic Pseudosyncope**

Psychogenic pseudosyncope is defined as an apparent transient loss of consciousness without having true loss of consciousness. It is associated with chronicity and progressive worsening of symptoms, causing disability and impaired quality of life. While poorly characterized, particularly in the pediatric population, it has been estimated to account for 0% to 8% of patients presenting with syncope. Typically, the patient history is notable for a stressor around the time of onset of these symptoms. Occasionally, these patients go on to present with subsequent alternative pseudoneurologic disorders that are not attributed to an organic etiology. In patients with psychogenic pseudosyncope, a thorough history is key to the diagnosis. As compared with patients who experience true loss of consciousness with syncope, these patients tend to have longer periods

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**Figure 6. Brugada Syndrome Patterns On Electrocardiogram**

Precordial leads of a resuscitated patient with Brugada syndrome. Arrows indicate coved ST segment changes. Note the dynamic electrocardiogram (ECG) changes over time. All 3 patterns are shown. Arrows denote the J wave. The left panel shows a clear type 1 ECG. Between 7–2-99 and 13–2-99, types 2 and 3 are shown. Calibrations are given.

Arthur A.M. Wilde, Charles Antzelevitch, Martin Borggrefe et al. Proposed Diagnostic Criteria for the Brugada Syndrome. *Circulation*. 106(19):2514-2519. DOI: [https://doi.org/10.1161/01.CIR.0000034169.45752.4A](https://doi.org/10.1161/01.CIR.0000034169.45752.4A) Reprinted with permission from Wolters Kluwer Health, Inc.
Prehospital Care

Prehospital care may differ widely depending on the specific type of syncope. If a patient had a brief syncopeal event in a public area, bystanders may have been present to help minimize injury (eg, fall prevention or securing the location of the patient). Depending on the length of the loss of consciousness, the patient may or may not have returned to baseline by the time of arrival of medical providers, specifically EMS.

If a patient is unconscious or unresponsive on EMS arrival, then the PALS or APLS guidelines should be followed with evaluation and support of airway, breathing, circulation, and disability (ABCD). Depending on the level of consciousness, airway support may not be required or may range from noninvasive with placement of oxygen to advanced airway management. For the layperson caring for a patient who is unconscious and unresponsive, the 2011 PALS guidelines recommend cardiopulmonary resuscitation (CPR). The guidelines have been updated for both bystanders (laypersons) and medical providers, because it has been found that only 30% of victims of sudden death receive bystander CPR. Current recommendations support the hands-only (compression-only) option, especially for layperson CPR, as this is easier to perform, requires no equipment, and is simpler to instruct over the phone. In adult studies of mostly cardiac syncopeal events, survival rates have been found to be similar or better without rescue breathing, but no definitive pediatric trial of compression-only CPR has been conducted.

Modification of the classic A-B-C training for resuscitation to the current model of C-A-B emphasizes the circulation aspect of CPR even for children. For trained providers, ventilations and compressions are the preferred mode of CPR to increase survival, with ventilations being most important for hypoxic/asphyxial arrest versus sudden cardiac arrest. As 70% of pediatric arrests are respiratory in etiology, current recommendations still strongly support ventilations and compressions, due to increased survival rates. If ventilations are not possible or cannot be performed safely, then compressions alone are preferable to no CPR. The use of automated external defibrillators has become the standard of care, with improved outcomes for pediatric and adult out-of-hospital arrests that occur in schools and public spaces.

If a patient has experienced syncope but has regained consciousness by the time of arrival of EMS, then care is largely supportive and dependent on history and physical examination. If the patient has ongoing mental status changes, a fingerstick glucose measurement should be obtained and acted upon, if abnormal. In the asymptomatic patient, monitoring is often the only required intervention prior to ED arrival.

Emergency Department Evaluation

Initial Management

The evaluation for syncope in the ED depends on whether or not the patient has returned to baseline or remains unconscious or has continued altered mental status. All patients who have altered mental status or are unconscious should be treated using standard PALS guidelines, with an evaluate-identify-intervene continuous cycle of assessment with primary and secondary assessments taking place in a rapid fashion. Of note, if the patient does not have a spontaneous return to consciousness within seconds to minutes and remains unconscious, then this should no longer be treated as syncope; the treatment of the obtunded patient is beyond the scope of this review.

History

Initial history in a patient with altered mental status should be brief and focused toward pertinent information such as onset of symptoms, details of events, and underlying medical problems. Additional pieces of history that are useful to the emergency clinician in the acute setting may be obtained using the “SAMPLE” mnemonic (Signs and Symptoms, Allergies, Medications, Past medical history, Last meal, and surrounding Events).

Additional history that is useful in patients with syncope who present either unconscious then recover or for patients who have already returned to baseline focuses on delineating the underlying cause of the syncope. History should be focused on worrisome and reassuring aspects. See Table 3, page 12 for “red flags” ( worrisome aspects of the history) and “green lights” (reassuring aspects of the history that suggest NCS). Other aspects of the history that may elucidate the etiology of syncope...
can include a personal or family history of seizures, metabolic diseases, and diabetes. Additionally, a history of medication or toxic ingestion would be relevant, as well as history of recurrent syncope, menstrual history and risk of pregnancy, and possible trauma history.

**Physical Examination**

Physical examination in the patient who has recovered from syncope should focus on general appearance, evaluation for injuries, and an overall cursory examination of the patient. Special attention should be directed toward the neurologic and cardiac examinations. A full set of vital signs should also be obtained in patients who have experienced syncope. Abnormal neurologic findings may indicate intracranial injury or seizure and would warrant additional directed evaluation that may include EEG or head imaging. A cardiac examination is also warranted in each patient to evaluate for rate, regularity, murmurs, and other adventitious sounds such as rubs or gallops. Any abnormality would require additional cardiac evaluation unless the emergency clinician is confident that the patient has a benign flow murmur. In NCS, the neurologic and cardiac examinations will be normal.

**Diagnostic Studies**

Vasovagal syncope/NCS is a largely clinical diagnosis. Because the biggest challenge for the emergency clinician is to evaluate whether or not a patient’s syncope is from a benign or vasovagal etiology or from a more concerning cause such as cardiac syncope, the most useful test is an ECG (though they are only diagnostic a small portion of the time). While there are no definitive data to suggest that a normal ECG ensures a patient’s syncope is vasovagal, the majority of current algorithms in the literature include an ECG as part of the evaluation of syncope. More specifically, in several current algorithms to evaluate for syncope, a normal ECG is a common branch point when distinguishing between vasovagal syncope and other more concerning etiologies. In studies that attempt to distinguish cardiac versus noncardiac causes of syncope, an ECG is used in both decision rules and algorithms. Additionally, there are no studies that have specifically demonstrated the negative predictive value of a normal ECG in the evaluation of routine syncope, but there are multiple studies that have evaluated the negative predictive value of a normal ECG in cardiac syncope. Because of the low cost and noninvasive nature of an ECG, it is recommended to include a screening ECG in the evaluation of routine syncope.

In a retrospective review that compared pediatric cardiology clinic patients with vasovagal syncope to a 10-year chart review of cardiogenic causes of syncope, Tretter and Kavey devised a simple screening rule to identify patients who require cardiology referral. The study showed that a 4-part screen had a sensitivity of 100% and a specificity of 60% for identifying a cardiac cause of syncope in that patient population. The screening consisted of: (1) a history of exertional syncope, (2) a family history of known cardiac disease or unexplained sudden death, (3) an abnormal physical examination supporting a cardiac diagnosis, or (4) an abnormal finding on ECG. Using a similar screen, a study of 480 children with syncope identified 21 of 22 patients with a cardiac etiology.

In addition to an ECG, urine testing should be considered if pregnancy is possible. These noninvasive, low-cost tests, as well as a thorough physical examination, make up most ED evaluations.

**Evidence Against Routine Testing**

When pediatric patients present to the ED after a syncopal event, they often undergo unnecessary and expensive evaluations without additional diagnostic yield. In a study by Goble et al, 90% of patients had electrolytes checked, 58% had head computed tomography (CT) scans, and 37% had chest x-rays. Diagnostic tests are costly and have not been shown to be helpful in the workup of routine syncope. In contrast, the majority of guidelines reviewed for this paper have recommended ECG as part of routine workup, yet only 58% to 85% of patients with routine syncope had ECG studies performed.

In the ED, following standardized clinical pathways has been shown to be effective and produce significant cost savings by preventing unnecessary low-yield testing and admissions. In spite of this, workup for routine syncope in the pediatric population remains an area that needs improvement in more effective resource utilization. Routine use of additional testing, including but not limited to, head-up tilt-table testing, orthostatic vital signs, head imaging, EEG, laboratory testing (such as electrolytes), echocardiograms, and x-rays, is not indicated and has been shown by multiple analyses to be low-yield in determining cause and management of pediatric syncope. Special cases, based on history and initial evaluation (including ECG and urine HCG), may require additional diagnostic tests as outlined above, but these should be obtained on an individual basis rather than a routine basis.

**Head-Up Tilt-Table Test**

The head-up tilt-table test is neither efficient nor necessary for the diagnosis of NCS. In one protocol, the test is performed in more than 30 minutes. A test is considered positive for NCS if it results in age-specific bradycardia, hypotension, or dysrhythmia, such as sinus or atrioventricular node block or transient asystole. In one study, as many as 13% of
patients with syncope received head-up tilt-table testing, but the data have shown consistently that this type of testing does not provide additional information beyond that obtained from the history, physical examination, and ECG, nor is it helpful to predict outcomes. The specificity of head-up tilt-table testing has been estimated to be between 48% and 100%, and in one study, 87% of patients with NCS were diagnosed without results of head-up tilt-table testing. Orthostatic Vital Sign Measurements Orthostatic vital sign abnormalities were defined in 1995 by the American Autonomic Society, but they are not correlated with symptomatic orthostatic. Orthostatic vital signs are often obtained in the ED setting, but their clinical significance is unclear in pediatric patients with syncope. In large studies of healthy school-age children, significant variations in heart rate and blood pressure were detected. In a study by Koziol-McLain et al of adults in an ED, 43% of presumed euvolemic patients met criteria for being orthostatic. In healthy adolescent volunteers, 44% had significant orthostasis. Since so many nonsyncopal children can have “positive” or abnormal results, the diagnostic value is questionable. The sensitivity and specificity of pulse change in adolescents has been shown to be 61% and 56%, respectively. Even with known volume depletion from blood loss, sensitivity was shown to be < 30% for heart rate and blood pressure change. Any patient with persistent symptoms of orthostasis (i.e., lightheadedness with sitting or standing), regardless of change in heart rate or blood pressure, should be evaluated and treated.

Electroencephalogram And Computed Tomography Head imaging is frequently performed as part of the syncope evaluation. In the setting of neurologic deficit or trauma, imaging is warranted, but in a study by Massin et al of routine syncope evaluation, EEG and head imaging did not contribute to diagnosis of any patients. While EEG may be a useful evaluation tool in diagnosing seizures, it is very low-yield for routine evaluation as part of a syncope evaluation. In the Massin et al study, there was a positive yield for EEG in < 1 in 300 instances. An EEG would only be useful to rule out seizure in the setting of concerning history (as noted in the “Seizure” section, page 12) or postictal state on examination. Additionally, even if there is history to suggest that a seizure has occurred but has since stopped, the utility of an EEG in the ED versus inpatient or outpatient settings is variable. If seizure is suspected, then consultation with neurology is indicated to determine the appropriate timing of the EEG (e.g., the need for admission for EEG or deferral to outpatient evaluation).

CT scanning was found to be performed as part of routine syncope evaluation in 58% of pediatric pa-
tients, but this study was costly ($1100 to $1500 per scan), and all CT scans were normal in the absence of abnormal neurologic findings. Based on these results, the use of CT in the setting of routine syncope is costly, low-yield, and also subjects patients to unnecessary radiation. Radiologic imaging should only be obtained if there is significant concern for intracranial bleeding.

Cardiac Enzymes Cardiac enzymes for routine pediatric syncope should not be obtained unless there is a suspicion for aberrant coronary artery or myocarditis. As these are very rare causes in the pediatric population, this type of testing should only be performed in the setting of chest pain or history of chest pain with suggestive ECG findings. Despite this recommendation, the National Hospital Ambulatory Medical Care Survey from 2005 to 2007 showed that 15% of patients had such testing performed.

Treatment Supportive care is the primary treatment for the vast majority of pediatric patients presenting to the ED with syncope, as most syncope is neurocardiogenic. For those patients who have symptomatic hypovolemia, fluids are indicated. Up to 90% of patients with syncopal symptoms will improve with adequate fluid and salt intake. Intravenous fluid is a common treatment for patients who present with persistent symptoms or who have evidence of dehydration and cannot readily hydrate orally. In addition, anticipatory guidance should be given to patients regarding adequate oral intake at home, avoiding provoking events, and early identification of vasovagal symptoms to adjust behaviors and prevent syncope.

All cases with a suspected cardiac etiology should have a pediatric cardiac consultation and close pediatric cardiology follow-up. If a patient is to be discharged home after discussion with cardiology, exercise limitation is warranted until follow-up is obtained. Cardiac events or sudden cardiac death can occur at any time and often occur at rest, but physical activity increases the risk. As symptomatology cannot always be predicted in patients with presumed cardiac syncope, patients discharged to home must be limited in their exercise and competitive sport participation until evaluated by cardiology. ECG interpretation by a pediatric cardiologist can be helpful if the results are being used to determine disposition. In some studies, it has been documented that pediatric emergency clinicians may underappreciate significant ECG findings, or conversely, be unnecessarily concerned about QT intervals. ECG review by pediatric cardiology may be useful in determining patient disposition.
1. “The teenage patient assured me that she could not be pregnant, so I did not order an HCG test.”
Ensuring that adolescent patients have a chance to speak with providers without their parents present is an expected part of adolescent medicine and often allows capture of sensitive information. The emergency clinician must verify that teenage girls who report no sexual intercourse are really not pregnant. Results should be provided in a confidential manner based upon individual state law.

2. “It was such a classic story for neurocardiogenic syncope, except for the family history of sudden death, that I did not perform an ECG.”
Cardiac abnormalities can easily be overlooked. Most of the rules regarding limiting extensive testing presume a normal ECG. A family history of sudden death could suggest a genetically inherited cause of cardiac syncope.

3. “I asked if there were any medical problems that run in the family, but the patient didn’t tell me that her sister is deaf.”
Many families do not recognize deafness as a reportable medical problem, so this must be asked specifically. This is also true for sudden unexplained deaths in the family. Patients may not offer this information unless it is explicitly asked.

4. “The coach, parents, and patient all told me he was just overexerted while running. They think he can play in the state championship game tomorrow.”
Don’t be swayed by elite athletes, coaches, or family members minimizing symptoms. The primary goal is to ensure the safety of the patient. If syncope occurred during activity, then the patient should refrain from strenuous activity until cleared by cardiology.

5. “The patient had a history and physical examination consistent with neurocardiogenic syncope without any evidence of injury. The mother was very concerned about a brain tumor, so I obtained a CT scan to reassure her.”
Sometimes the path of least resistance can do more harm than good. Do not unnecessarily irradiate pediatric patients, as this exposes them to radiation that increases their long-term risk of cancer.

6. “I looked at the ECG quickly to check for ischemic changes as I do for my adult patients and was reassured by the ECG.”
Remember ECG analysis in pediatric patients is not primarily to assess for myocardial infarctions, and emergency clinicians must change their point of reference and concentrate on cardiac abnormalities that can cause syncope in children (eg, prolonged QT, Wolff-Parkinson-White syndrome, Brugada syndrome, or myocarditis/pericarditis).

7. “The nurse checked orthostatic vitals. I knew the patient could not be volume depleted, so we discharged her without fluid resuscitation.”
Orthostatic vitals have been shown to be neither sensitive nor specific for volume depletion. Patients who are orthostatic by symptom description should be hydrated and reassessed prior to discharge.

8. “The patient had known congenital cardiac disease, but the episode sounded neurocardiogenic so I sent him home.”
Children with underlying cardiac disease warrant consultation with pediatric cardiology prior to discharge to ensure that the syncope is not related to their underlying condition.

9. “A pediatrician referred this patient with classic syncope to the ED. His ECG was normal, but the primary care physician wanted him admitted for overnight observation.”
Routine admission for a patient who has returned to baseline, has no cardiac risk factors, and has a normal ECG is unnecessary and not cost-effective. To date, there are no data that show that routine admission after a syncopal event alters morbidity or mortality, and admission increases healthcare costs and may expose patients to additional risks.

10. “I know the patient had no cardiac risk factors, but I wanted to be thorough, so I ordered electrolytes, an EEG, an echo, and head-up tilt-table testing.”
Extensive testing in low-risk groups rarely improves diagnostic yield and results in unnecessary medical expenses.

Risk Management Pitfalls In Pediatric Patients With Syncope

- Don’t be swayed by elite athletes, coaches, or family members minimizing symptoms.
- The primary goal is to ensure the safety of the patient.
- If syncope occurred during activity, the patient should refrain from strenuous activity until cleared by cardiology.
- Irradiating pediatric patients unnecessarily exposes them to radiation that may increase their long-term risk of cancer.
- Extensive testing in low-risk groups rarely improves diagnostic yield and results in unnecessary medical expenses.
Table 4 summarizes management approaches for various etiologies of syncope.

**Special Populations**

**Patients With Known Cardiac Disease**

In the European Society of Cardiology syncope guidelines (for adult patients), it is recommended that all patients with syncope who have known structural heart disease should be admitted for further workup. The Canadian Cardiovascular Society made similar recommendations. The incidence of heart disease in children is estimated to be 0.7% to 1%. At this time, there are no clear guidelines about routine admission for pediatric patients.

Factors that would dictate additional evaluation or admission in these patients include their repair status, current functionality or disease, and any existing known residual defects. At this time, any patient with known cardiac history presenting to the ED would warrant cardiology evaluation. In addition to

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**Table 4. Management Of Various Etiologies Of Syncope**

<table>
<thead>
<tr>
<th>Cardiac Etiology</th>
<th>Management</th>
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| Hypertrophic cardiomyopathy       | • Management is supportive.  
• Follow the PALS or APLS algorithm for persistent dysrhythmias and to treat any underlying heart failure.  
• Advise restriction from all competitive athletics to reduce the risk of sudden death.  
• In an otherwise asymptomatic patient, activity restriction, adequate hydration, and urgent cardiac evaluation are indicated.  
• For high-risk patients, treatment may include medical management, surgical myectomy, or the use of implantable defibrillators. |
| Long QT syndrome                  | • Therapy is aimed at reducing sympathetic activity to the heart.  
• Treat patients with torsades de pointes with IV magnesium sulfate (25-50 mg/kg, maximum of 2000 mg), beta-blockers, and/or temporary cardiac transcutaneous pacing.  
• Provide specific instructions both verbally and in writing to patients and parents when discharging patients from the ED with cardiology follow-up. Patients should be advised to:  
  ▪ Avoid competitive sports, although possible participation in specific recreational activities may be allowed.  
  ▪ Drink electrolyte-rich fluids and eat potassium-rich foods during exercise in hot environments.  
  ▪ Be aware of QT-prolonging medications and avoid them, if possible. |
| Wolff-Parkinson-White syndrome     | • Treatment of these patients in the ED depends on their presenting symptoms and their ECG findings.  
• All patients with WPW ECG morphology will need cardiology evaluation. Consider electrophysiologic consultation to induce and ablate the accessory pathways.  
• Treat any persistent dysrhythmia per established PALS guidelines with immediate electrical cardioversion for unstable patients.  
• For patients presenting with irregular wide QRS complex tachycardias, the emergency clinician must differentiate WPW-induced atrial fibrillation from polymorphic VT, which is mostly a concern for the adult population. |
| Supraventricular tachycardia       | • Follow the PALS or APLS algorithm, with emphasis on early cardioversion in an unstable patient.  
• Vagal maneuvers are appropriate for the stable patient who is normotensive and without signs or symptoms of cardiorespiratory compromise.  
• For infants, a slurry of ice water to the face, and for older children, valsalva maneuvers (bearing down as if to defecate or blowing through an obstructed straw) can be effective in aborting the dysrhythmia.  
• If vagal maneuvers are not effective, the appropriate next treatment for a stable patient is a rapid push of adenosine (0.1 mg/kg with a maximum of 6 mg given as a rapid IV bolus) using the IV line closest to the heart. A second dose of 0.2 mg/kg of adenosine (with a maximum of 12 mg) may be given, if needed.  
• If the patient becomes unstable or shows signs of cardiorespiratory compromise, the treatment of choice is synchronized cardioversion with 0.5-1 J/kg. |
| Heart block                       | • For symptomatic bradycardia, treat with supportive measures (eg, oxygen, IV fluids).  
• For cardiorespiratory compromise, administer epinephrine (0.01 mg/kg IV, every 3-5 minutes) to increase the patient’s heart rate. Atropine (0.02 mg/kg IV, can be repeated once) can also be given if there are concerns about primary AV node dysfunction.  
• In the unstable patient with cardiorespiratory compromise, external pacing is warranted after addressing airway and breathing with airway adjuncts and oxygen. This should be done while addressing the underlying cause, if one is identified.  
• A cardiology consultation is warranted in patients with syncope who have an identified heart block on ECG (see Figure 5, page 9), because the presence of syncope implies that the patient is having symptomatic heart block. |
| Brugada syndrome                  | • Recommend exercise restriction and that the patient avoid medications including antidysrhythmics, antiarrhythmics, tricyclic antidepressants, local anesthetics (eg, bupivacaine), and opioids.  
• Cardiology consultation is warranted if a Brugada pattern is identified on ECG in the ED. |
the baseline ECG, these patients may be more likely to require additional testing such as echocardiogram, chest x-ray, or laboratory studies. While in the ED, these patients should remain on a cardiac monitor.

**Controversies And Cutting Edge**

The use of pharmacologic agents in the treatment of NCS is controversial. Fludrocortisone has been used in patients with recurrent syncope and presyncope associated with hypotension. Fludrocortisone is a mineralocorticoid agent that raises blood pressure by stimulating renal sodium and fluid retention. Side effects and treatment–limited effects include hypertension, hypokalemia, and hypomagnesemia. Fludrocortisone tends to be the first-line pharmacologic treatment for recurrent NCS, but recent data suggest that fludrocortisone is not effective at preventing NCS and is not better than placebo. The data, however, are generally limited, and it is difficult to draw conclusive recommendations.

### Table 4. Management Of Various Etiologies Of Syncope

<table>
<thead>
<tr>
<th>Noncardiac Etiology</th>
<th>Management</th>
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| Pregnancy                                       | • Early referral to obstetric services, as well as to social work for additional support, is recommended.  
  • Ultrasound may be indicated to confirm intrauterine pregnancy, if there are additional symptoms of vaginal bleeding or abdominal pain when urine HCG is positive. |
| Hypoglycemia                                    | • IV glucose is recommended with glucose-containing fluids to provide 0.5-1 g/kg of glucose. The fluids can also be taken orally (if the patient is able). |
| Neurocardiogenic syncope                         | • Treatment is largely supportive. In general, patients who are diagnosed with NCS do not require any specific treatment. However, there are several pharmacologic agents used for recurrent NCS with limited supporting data; these are discussed in detail in the “Controversies And Cutting Edge” section, page 19.  
  • Correcting fluid deficits in a dehydrated patient may be useful.  
  • To prevent recurrent NCS, exercise has also been recommended to increase orthostatic tolerance.  
  • Patient education should be provided on ways to minimize the risk of future episodes through behavioral modifications (eg, staying hydrated, avoiding prolonged standing or time spent in the supine position, and increasing salt intake).  
  • Additionally, advising patients to recognize the syncope prodrome and encouraging them to lower themselves to the ground prior to loss of consciousness may minimize potential injuries associated with syncope. |
| Seizure                                          | • History alone is sufficient to distinguish most cases of syncope from seizure; but rarely, for cases when true seizure is unclear, neurology referral or EEG may be useful. |
| Breath-holding spells                            | • Treatment with iron has been shown to decrease subsequent episodes, but it is unclear whether treatment is effective if the child is not anemic.  
  • As breath-holding episodes can recur until the child reaches school age, expectant guidance and reassurance that there are no long-term sequelae are recommended. |
| Postural orthostatic tachycardia syndrome (POTS) | • If a patient is suspected of having POTS, then referral to a specialist should be considered to establish the diagnosis.  
  • Treatment for POTS is controversial, but the mainstays of therapy include increased fluid intake (to 2 to 2.5 L per day) and increased salt intake.  
  • Reconditioning, exercise, and avoiding circumstances that may worsen the symptoms are also important.  
  • Pharmacologic treatments include fludrocortisone, low-dose beta blockers, and midodrine, but there are currently no United States Food and Drug Administration-approved treatments for pediatric patients with POTS. |
| Psychogenic pseudosyncope                        | • No additional management is necessary other than reassurance.  
  • Avoid excessive testing, but psychiatry referral may be of the most benefit to these patients. |

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4 For maintenance management of long QT syndrome, beta blockers are generally considered the first line of therapy, and have been shown to decrease mortality from 71% to 6% and to eliminate dysrythmia in 60% of patients.  
5 A complete list with the degree of risk can be found in the American Heart Association consensus statement published in 2004.  
6 With atrial fibrillation, the ECG will more likely reveal an irregular, wide QRS complex tachycardia that has beat-to-beat variation. This finding, in conjunction with a faster ventricular rate (can exceed 300 beats/min), can be more suggestive of atrial fibrillation due to WPW.  
7 Drinking water profoundly affects sympathetic tone and increases heart rate in patients with autonomic failure, and it is possible that this may be extrapolated to patients with NCS.  
8 EEG has not, however, been shown to be an efficacious component of a routine syncope workup. In patients who have been referred to pediatric neurology for evaluation of seizure, up to 20% do not have epileptic seizures, and NCS is a common finding in this population.  
9 The diagnostic evaluation for POTS is often extensive and may involve testing for metabolic and hematologic abnormalities as well as potentially performing autonomic function tests. This evaluation is not appropriate in an ED setting, and while POTS does cause significant personal distress for patients, there is no additional management that is necessary in the ED. A referral to a provider who specializes in POTS may be useful.

Abbreviations: APLS, Advanced Pediatric Life Support; AV, atrioventricular; ECG, electrocardiogram; ED, emergency department; EEG, electroencephalogram; HCG, human chorionic gonadotropin; IV, intravenous; PALS, Pediatric Advanced Life Support; VT, ventricular tachycardia.
Alpha-1 agonists, such as midodrine, have also been used to increase blood pressure in patients with primary autonomic failure, and the effects of this may be extrapolated to patients with recurrent NCS. The data, however, are still inconclusive. A study of 26 children with recurrent syncope demonstrated that patients with NCS who were taking midodrine hydrochloride had a lower recurrence of events. Low-dose beta blockers have also been used to treat recurrent NCS. Despite some promising early data, in general, there is no definitive treatment for recurrent NCS beyond management of symptoms.

**Disposition**

Admission is rarely indicated for patients presenting with syncope. After evaluation and stabilization in the ED, the vast majority of patients with syncope who have no red flags and are asymptomatic can be discharged home safely with supportive management and explanation of risk factors for future episodes. For patients with concerning cardiac findings, consulting cardiology from the ED is indicated to help establish safe disposition and further workup. The decision of inpatient admission, outpatient specialist referral, or discharge home with reassurance is dependent on the individual etiologies. Patients with NCS can be discharged home safely with instructions to watch for warning signs of a more worrisome syncopal etiology (ie, red flags for cardiac etiology), and to follow up with their primary care provider. Additionally, patients should receive instructions about adequate hydration and early identification of vasovagal symptoms. Routine inpatient evaluation of syncope is not warranted unless a specific etiology is being considered and is either dependent on further workup that cannot be performed in the ED (eg, echocardiograms at some institutions), or if their specific etiology (eg, myocarditis, dysrhythmias) poses significant risk of anticipated morbidity or mortality. When considering admission based on ECG findings, a review by a pediatric cardiologist may be useful; such practice may decrease unnecessary admissions.

Patients with likely seizure should have a neurology consultation and likely early follow-up as an outpatient, unless there is a high enough index of suspicion for an infectious etiology or if the patient does not return to baseline in an acceptable period of time. Patients with positive pregnancy tests do not require routine admission, but should be evaluated for possible ectopic pregnancy and assessed by obstetrics and social work, if indicated.

In cases where the patient is clinically unstable, unable to maintain adequate hydration, or has a concerning history or physical examination and requires continued monitoring or evaluation that cannot be performed as an outpatient, admission is warranted.

**Time-And Cost-Effective Strategies**

Ensuring that the appropriate questions are asked to rule out red flags and capture green lights is a cost-efficient and effective way to avoid missing a critical cardiac etiology of syncope. Extensive workups do not generally provide more information for the routine case of pediatric syncope. Screening with low-cost, noninvasive tests such as ECG and pregnancy testing (when indicated) is the most reasonable approach to most pediatric patients with syncope.

Admission rates tend to be high and are often based on ECG findings. When considering admission based on ECG findings, a review by a pediatric cardiologist is warranted. Given the underappreciation of significant ECG findings by emergency clinicians and unnecessary concern about prolonged QT syndrome that is diagnosed by emergency clinicians but not deemed concerning by cardiology, this process should happen early in the course of treatment. Such a practice pathway could decrease unnecessary admissions and ensure that critical findings are not missed.

Head CT scans and chest x-rays are often obtained in pediatric patients presenting with syncope. In one study in a community ED, of the 113 pediatric patients seen, 58% had head CT scans and 37% had chest x-rays. None of the CT scans had positive findings, but this resulted in a large percentage of patients being deemed high-resource utilizers. Other studies have assessed noncardiac testing and found low utility and high average patient costs. Having standardized algorithms and clinical pathways has been shown to increase diagnostic yield, reduce admissions, and decrease overall costs.

**Summary**

Syncope in the pediatric population is a common presenting complaint to EDs. Etiologies can range from life-threatening cardiac causes to more benign etiologies. There are certain red flags that require careful attention, but most cases can be safely and efficiently managed with screening ECGs and pregnancy screening. Extensive and expensive workups have not been shown to be useful or diagnostic, unless there are specific signs and symptoms to suggest the need for in-depth testing. Establishing a care algorithm for these patients has been shown to improve efficiency and decrease cost and length of stay. The causes of pediatric syncope and syncope in adults are often different. The evaluation of pediatric patients who have had a syncopal event must be tailored to the individual patient.
Case Conclusions

Multiple red flags existed for the 10-year-old who collapsed on the basketball court. You immediately suspected cardiac causes due to the event occurring during vigorous exercise. In addition to his screening ECG, cardiology was consulted from the ED and the patient had an urgent echocardiogram. The patient’s echocardiogram showed a significantly thickened interventricular septum with outflow obstruction. The patient was started on beta blockers, but later had a recurrent episode and had an implantable cardiac defibrillator placed. The patient was put on exercise limitation and was restricted from competitive sports.

The case of the 16-year-old girl demonstrated many red flags for a cardiac etiology of her syncope. She had multiple episodes during activity with persistent symptoms of shortness of breath. The patient’s ECG showed sinus tachycardia at 164 beats/min with low voltages, right-axis deviation, and diffuse 1-mm ST elevations. You ordered a chest x-ray that did not show cardiomegaly, infiltrate, or edema. You called for an emergent cardiology consultation, and the patient had an initial echocardiogram that showed a small pericardial effusion and normal ejection fraction. While in bed, the patient had a presyncope sensation and then had a generalized seizure with hypoxia and relative bradycardia at 100 beats/min. After receiving lorazepam, the patient’s symptoms resolved. Cardiology was called again, and repeat echocardiogram showed poor ejection fraction. During the echocardiogram, the patient went into a 2:1 AV block and became pulseless. Immediate CPR resulted in return of spontaneous circulation and the ECMO team was mobilized. The patient was cannulated in the ED and transported to the pediatric ICU. She remained on ECMO for 9 days and was discharged from the hospital after 1 month with the diagnosis of influenza myocarditis. On follow-up echocardiogram 9 months after her admission, she continued to have normal cardiac function and was given approval to return to normal activity levels.

The 18-year-old girl had a normal ECG, but had a positive HCG test. The patient denied abdominal pain or bleeding and had an ultrasound that showed a 7-week estimated gestational age intrauterine pregnancy. Social work was consulted, and reassurance and support information was provided. No further testing was performed. After tolerating oral intake and demonstrating ability to ambulate, she was discharged home with obstetrician follow-up information.

References

Evidence-based medicine requires a critical appraisal of the literature based upon study methodology and number of subjects. Not all references are equally robust. The findings of a large, prospective, randomized, and blinded trial should carry more weight than a case report.

To help the reader judge the strength of each reference, pertinent information about the study, such as the type of study and the number of patients in the study is included in bold type following the references, where available. The most informative references cited in this paper, as determined by the authors, are noted by an asterisk (*) next to the number of the reference.

30. Caughrey RW, Humphrey JM, Thomas PE. High-degree atrioventricular block in a child with acute myocarditis. Ochsner J. 2014;14(2):244-247. (Case report; 1 patient)


1. A teenager feels the “world going dark” and then collapses while standing. What is the most likely cause of her syncope?
   a. Neurocardiogenic
   b. Hypertrophic cardiomyopathy
   c. Psychogenic pseudosyncope
   d. Breath-holding spell

2. A 17-year-old deaf patient with a history of syncope presents to the ED for cough, nausea, and vomiting and is found to have pneumonia on chest x-ray. Which of the following medications could be prescribed without an ECG?
   a. Cefuroxime
   b. Levofloxacin
   c. Azithromycin
   d. Ondansetron

3. Which of these findings is reassuring that a patient most likely has vasovagal syncope?
   a. QTc of > 500 milliseconds
   b. Family history of sudden cardiac death
   c. Prodrome of nausea, diaphoresis, and lightheadedness
   d. Event occurred while running

4. A teenager collapsed while playing basketball and had some “shaking” movements. He has a history of syncope with lightheadedness when having his blood drawn and has a family history of syncope. Which of the following would heighten your concern for an underlying cardiac etiology of his syncope?
   a. Syncope is associated with lightheadedness
   b. The patient’s mother experienced syncope as a teenager
   c. Syncope occurred while running at basketball practice
   d. Rhythmic shaking during the loss-of-consciousness phase

5. While seated playing a video game, a 10-year-old has an episode of unresponsiveness with loss of tone followed by several minutes of shaking movements of his upper extremities. There was no loss of continence. What is the most likely cause of this altered mental status?
   a. Neurocardiogenic
   b. Psychogenic pseudosyncope
   c. Breath-holding spell
   d. Seizures

6. Which of the following is more likely associated with psychogenic syncope?
   a. Episodes lasting for several seconds
   b. Closed eyes
   c. The event does not recur during evaluation
   d. A patient who is a boy

7. In the setting of syncope due to traumatic cardiac arrest on the ball field, what is the most effective measure for reducing morbidity and mortality?
   a. Being located near a hospital
   b. Having an automated external defibrillator available
   c. Having oxygen available
   d. Having a doctor at all games

8. What is the most useful screening test for children who present with syncope?
   a. Electrolytes
   b. ECG
   c. Head CT
   d. Fingerstick glucose

9. A teenager collapses while sprinting during football tryouts. He wants to go back to practice tomorrow. When should he be allowed to return?
   a. As soon as he is asymptomatic
   b. Once he is cleared by cardiology
   c. The next day, if his ECG is normal
   d. After 1 week without recurrent symptoms

10. What follow-up should be arranged for a patient who presents with syncope and has a QTc on ECG of 500 milliseconds who is currently asymptomatic?
    a. Emergent pediatric cardiology referral in the ED
    b. Admission to the hospital
    c. Primary care physician follow-up within the week
    d. Competitive sports limitations until outpatient pediatric cardiology appointment
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- **Points**
  - Patients diagnosed with a cervical artery dissection targets are up to individual providers based when cervical artery dissection is suspected. After acute strokes can receive thrombolytics safely even when cervical artery dissection is suspected.
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