Pediatric Syncope: Cases from the Emergency Department

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Pediatric syncope is a common presentation in the emergency department (ED). The emergency physician (EP) must handle the complaint with finesse, as an abrupt loss of consciousness can be dramatic and alarming to patients, family, friends, and caregivers. The EP should perform a thorough evaluation of cause, care for any resulting injury and emotional reassurance in a focused and resource-effective manner.

Syncope is defined as the sudden loss of consciousness and postural tone with spontaneous and complete recovery after a brief duration. Pre-syncope is the feeling that one is about to pass out but remains conscious with a transient loss of postural tone. Pre-syncope may or may not reflect the same pathology as syncope but the approach should be the same.

Syncope follows a common pathophysiologic pathway with many potential inciting stimuli. Cerebral perfusion is compromised by a transient decrease in cardiac output caused by vasomotor changes decreasing venous return, a primary dysrhythmia, or impairment of vascular tone. Frank syncope occurs when cerebral blood flow decreases to less than 30% to 50% of baseline.

The incidence of pediatric syncope is common with 15% to 25% of children and adolescents experiencing at least one episode of syncope before adulthood. Incidence peaks between the ages of 15 and 19 years for both sexes. There appears to be a female predominance. Before age 6, syncope is unusual except in the setting of

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seizure disorders, breath holding, and cardiac arrhythmias. Pediatric syncope may account for as high as 3% of ED visits.\(^8\)

Most causes of pediatric syncope are benign, but an evaluation must exclude rare life-threatening disorders. In contrast to adults, vasodepressor syncope is the most frequent cause of pediatric syncope (61%–80%).\(^9\) Other causes include neuropsychiatric, cardiac, pregnancy, and metabolic disorders (Table 1).\(^9–11\) Cardiac disorders represent 2% to 6% of pediatric cases.\(^7\) Although the annual incidence of nontraumatic sudden death in the pediatric population is very low (less than 4/100,000), underlying cardiac disease is very common among these cases.\(^12\)

**WHY IS THE HISTORY SO IMPORTANT IN PEDIATRIC SYNCOPE?**

The lack of objective findings in pediatric syncope can pose a challenge for the EP. An accurate and detailed history becomes essential for clinical decision making (Fig. 1). However, pediatric patients may not be able to provide a complete or specific history.

<table>
<thead>
<tr>
<th>Causes of pediatric syncope</th>
<th>Cardiac</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Noncardiac</strong></td>
<td><strong>Cardiac</strong></td>
</tr>
<tr>
<td>Vasodepressor syncope</td>
<td>Arrhythmias</td>
</tr>
<tr>
<td>Orthostatic hypotension</td>
<td>Ventricular tachycardia</td>
</tr>
<tr>
<td>Postural orthostatic</td>
<td>No congenital heart disease</td>
</tr>
<tr>
<td>tachycardia syndrome</td>
<td>Postoperative congenital heart disease</td>
</tr>
<tr>
<td>Exercise-related syncope</td>
<td>Supraventricular tachycardia (ie, Wolff-Parkinson-White syndrome)</td>
</tr>
<tr>
<td>Breath-holding syncope</td>
<td>Long-QT syndrome</td>
</tr>
<tr>
<td>Tussive syncope</td>
<td>Congenital</td>
</tr>
<tr>
<td>Situational syncope</td>
<td>Drug induced</td>
</tr>
<tr>
<td>Dysautonomia</td>
<td>Congenital</td>
</tr>
<tr>
<td>Carotid sinus hypersensitivity</td>
<td>Hypertrophic cardiomypathy</td>
</tr>
<tr>
<td>Neuropsychiatric Seizure</td>
<td>Atrioventricular block</td>
</tr>
<tr>
<td>Migraine</td>
<td>Congenital</td>
</tr>
<tr>
<td>Hyperventilation Conversion syncope</td>
<td>Acquired (ie, Lyme disease)</td>
</tr>
<tr>
<td>Intracranial tumors</td>
<td>Arrhythmogenic right ventricular disease</td>
</tr>
<tr>
<td>Metabolic</td>
<td>Pacemaker malfunction</td>
</tr>
<tr>
<td>Hypoglycemia</td>
<td>Sick sinus syndrome</td>
</tr>
<tr>
<td>Electrolyte disorder</td>
<td>Commotio cordis</td>
</tr>
<tr>
<td>Malnutrition</td>
<td>Obstructive Lesions</td>
</tr>
<tr>
<td>Endocrine disease Drugs and toxins</td>
<td>Valvular aortic stenosis</td>
</tr>
<tr>
<td>Carbon monoxide poisoning</td>
<td>Primary pulmonary hypertension</td>
</tr>
<tr>
<td>Pregnancy</td>
<td>Eisenmenger syndrome</td>
</tr>
<tr>
<td>Normal pregnancy</td>
<td>Myocardial dysfunction</td>
</tr>
<tr>
<td>Ectopic pregnancy Pulmonary embolism</td>
<td>Secondary ventricular dysfunction</td>
</tr>
<tr>
<td>Pregnancy-related cardiac disease</td>
<td>Inflammatory disease</td>
</tr>
<tr>
<td>Data from Refs.(^8–11)</td>
<td>Acute myocarditis</td>
</tr>
<tr>
<td></td>
<td>Kawasaki disease</td>
</tr>
<tr>
<td></td>
<td>Ischemia</td>
</tr>
<tr>
<td></td>
<td>Anomalous coronary artery</td>
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<tr>
<td></td>
<td>Kawasaki disease</td>
</tr>
<tr>
<td></td>
<td>Cardiac tumors</td>
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Witnesses, parents, relatives, friends, teachers, and coaches are often needed but may not always be present in the ED. Moreover, the nuances of the history (eg, did the tonic-clonic movement begin before or after loss of consciousness) may not be apparent to the lay-person and further confound the presentation. Despite these obstacles, the history and physical examination prove sufficient to define the cause of syncope in up to 77% of pediatric cases.  

**WHAT ARE THE CRITICAL ELEMENTS OF THE PEDIATRIC SYNCOPE HISTORY?**

The key elements or “red flags” the EP must elicit are a history of exercise-induced syncope, pre-syncope or chest pain, and/or a family history of sudden death, including dysrhythmia, drowning, sudden infant death syndrome (SIDS), or pacemaker placement. These historical elements and/or a history of congenital heart disease should prompt immediate cardiovascular monitoring, a cardiology consultation, and further testing.  

Healthy patients may experience vasodepressor syncope after vigorous exertion due to venous pooling, high ambient temperature, dehydration, and hyperventilation. This condition must be differentiated from “mid-stride” syncope, which may indicate cardiac pathology.  

Patients with known cardiac pathology should be evaluated for interval change. Patients who have previously undergone surgical repair or palliation of congenital heart disease may have acquired or residual structural lesions, myocardial dysfunction, and supraventricular or ventricular dysrhythmias.  

A detailed history should include the time of day, time of last meal, activities leading up to the event, and associated symptoms such as palpitations or racing heart beat, chest pain, headache, shortness of breath, nausea, diaphoresis, amnesia, visual changes, and auditory changes. The duration of symptoms, the patient’s position
when symptoms began, and the patient’s appearance during and immediately following the episode are also important.¹

In addition to the pertinent medical history, a family history should expose familial syncope, congenital heart disease, seizures, metabolic disease, and deafness.¹,¹⁶ A medication history including herbal, over-the-counter or illicit drug use, and access to other medications or toxins should be gathered.¹ Risk of pregnancy should be assessed by gathering a menstrual history.¹³ Practitioners should also note the patient’s syncope burden and the timing and frequency of episodes in patients with repeat syncope.¹⁷

WHAT ARE THE CRITICAL ELEMENTS OF THE PHYSICAL EXAMINATION?

Each patient should undergo a complete physical examination in the ED. Most examinations are normal, but physical findings can help identify cardiac and neurologic causes of syncope.⁸,¹⁸

Prehospital, triage, and orthostatic vital signs should be reviewed.

Cardiac auscultation should be performed in the supine and standing position to determine the presence of dynamic obstruction. Abnormalities of cardiac rhythm should also be sought.¹⁷ The neurologic examination should focus on fundoscopy, cranial nerves, Romberg sign, gait, deep tendon reflexes, and cerebellar function.⁸

Physical features associated with cardiac disease (eg, abnormal facies, Marfan habitus, deafness, ataxia) or neurologic disease (eg, ash-leaf spots, cafe-au-lait spots, cleft palate) should be sought.¹³

WHAT DIAGNOSTIC STUDIES ARE NEEDED?

An electrocardiogram (ECG) should be obtained for all pediatric syncope patients. The ECG is low yield but is inexpensive, noninvasive, and highly sensitive. In combination with a detailed history and physical examination, the ECG has demonstrated a sensitivity of 96% for cardiac syncope.¹⁹ Some forms of structural heart disease and primary electrical disorders may not otherwise be apparent.¹⁷ Attention should be paid to the rhythm and the potential presence of a delta wave or a prolonged QT interval. Furthermore, the ECG may identify conduction disorders and cases of Brugada syndrome (Table 2).

<table>
<thead>
<tr>
<th>Table 2</th>
<th>Selected electrocardiographic findings in pediatric syncope</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cause</td>
<td>ECG Findings</td>
</tr>
<tr>
<td>Wolff-Parkinson-White</td>
<td>Short PR interval, wide QRS complex, and a delta wave or positive slurring in the upstroke of the QRS complex</td>
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<tr>
<td>Long-QT syndrome</td>
<td>QTc &gt;0.45</td>
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<tr>
<td>Brugada syndrome</td>
<td>ST elevation in anterior precordial leads, V₁ and V₂. Type 1 coved</td>
</tr>
<tr>
<td>Hypertrophic cardiomyopathy</td>
<td>LAE and LVH, ST-segment abnormalities, T-wave inversions, Q waves and diminished or absent R waves in the lateral leads.</td>
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</tbody>
</table>

Abbreviations: LAE, left atrial enlargement; LVH, left ventricular hypertrophy.

Any ECG abnormalities identified by the EP should be reviewed by a cardiologist before admission or ancillary testing. Despite low rates of discordance between EP and cardiologist ECG interpretation, suspected diagnoses can often be excluded by the cardiologist. Use of web-based or faxed ECG sharing can avoid unnecessary admission and testing.20,21

Other ED testing should be limited and patient focused. A urine pregnancy test should be sent for all menstruating females. A history suggesting electrolyte disturbance, malnutrition, an eating disorder, or endocrine abnormality should prompt serum electrolytes, including magnesium, calcium, and phosphate levels. Physical examination findings may guide the physician to check a hemoglobin level, a urine or serum toxicologic screen, or thyroid function tests.

Bedside ultrasound evaluation of fluid status and gross cardiac function may aid the EP in identifying dehydration, dilated cardiomyopathy, and hypertrophic cardiomyopathy.22–26 Further studies are needed to determine the role of this evolving technology.

Ancillary tests are of limited use except for patients with a specific indication prompted by the history, physical or ECG.19–21,27,28 Echocardiogram, Holter monitor, and tilt-table testing should only be ordered in consultation with a cardiologist. Electroencephalography (EEG), neuroimaging studies (including computer tomography or magnetic resonance imaging) and neurologic consultation may be indicated with specific neurologic findings.20,29

CASE 1: VASODEPRESSOR SYNCOPE

Sally is an 11-year-old girl brought in by ambulance to the ED after “fainting” this morning during math class. She describes feeling “dizzy” and “wobbly” when she stood up from her desk to answer a question. Witnesses report Sally became pale then collapsed into her chair, unconscious for 15 seconds. There was no abnormal movement or trauma noted. She woke up spontaneously feeling “weak.” Sally was late for the school bus this morning and missed breakfast. Her mother denies any prior episodes of syncope or a family history of sudden cardiac death. Emergency medical services (EMS) report normal vital signs during transport and a point-of-care glucose of 92 mg/dL. Vital signs and physical examination are normal. An ECG is performed and interpreted as normal. Sally is diagnosed with vasodepressor syncope. Precautions and reassurance are given and she is discharged home.

How Does Vasodepressor Syncope Present?

Vasodepressor syncope is also known as vasovagal syncope, neurocardiogenic syncope, neurally mediated syncope, reflex syncope, and common fainting. Patients classically experience 3 distinct phases during an event: prodrome, loss of consciousness, and recovery. A good prodrome history can determine the diagnosis. This phase can last seconds to minutes and is usually recalled by the patient.1,17

Premonitory symptoms include a warm, hot, or cold sensation, lightheadedness, dizziness, nausea or abdominal pain, yawning, shortness of breath, pallor, diaphoresis, hearing or visual change (decreased acuity, tunnel vision, or double vision), headache, and anticipated loss of consciousness.1,17,30 Any patient with abrupt syncope in which there is little or no premonitory symptoms must undergo a more extensive evaluation because of increased risk of serious cardiac disease and the risk of future injury.17

Actual loss of consciousness usually lasts somewhere between 5 and 20 seconds, but rarely extends to minutes. During this time patients appear pale or ashen, with cold skin and/or profuse sweating, and occasionally have dilated pupils and, rarely,
seizure-like activity. In general, this phase is not recalled by the patient; although some patients may describe feeling “disconnected,” or able to hear bystander voices but unable to respond. The recovery phase lasts 5 to 30 minutes. Fatigue, dizziness, weakness, headache, and nausea are common. Episodes tend to be short but may recur if patients are helped up too quickly from the recumbent position.1

What Provokes Vasodepressor Syncope?

Several factors can provoke a common pathway causing vasodepressor syncope. These triggers include prolonged upright posture during sitting, standing, and walking; cough; menses; and carotid sinus pressure sensitivity.1 Episodes may also be initiated by emotional stress, such as fear, anxiety, pain, phlebotomy, or the sight of blood. Precipitants that may contribute to an event include: anemia, relative dehydration, hunger, recent or concurrent illness, physical exhaustion, cessation of prolonged exercise, noxious stimuli, and/or crowded, warm, and poorly ventilated confines.31 Patients with situational syncope generally have stereotypical triggers for recurrence such as swallowing, micturition, defecation, instrumentation (eg, colonoscopy), post-exercise, sneezing, diving, and pain.17

How Well Do We Understand the Mechanism for Vasodepressor Syncope?

Vasodepressor syncope is thought to be a normal reflex triggered at unintended situations.32 There are 3 observed forms of vasodepressor syncope: primary bradycardia, which often includes transient asystole with subsequent hypotension; primary vasodepressor response with hypotension and relative preservation of heart rate; and a mixed response with simultaneous hypotension and bradycardia.1 The common mechanism for all 3 forms has long been believed to be the Bezold-Jarisch phenomenon. Forceful contractions on an underfilled left ventricle causes excessive stimulation of the ventricular mechanoreceptors or C fibers, which leads to paradoxical signals to central nervous system pathways. A sudden conversion from vasoconstriction to vasodilation and bradycardia then follows.32

The magnitude of hypotension recorded may be substantial, with systolic blood pressure decreasing by 40 to 82 mm Hg. The average decrease in heart rate is variable, with an average decrease of 40 beats/min, and cardiac asystole lasting 3 to 40 seconds in 4% to 6% of patients.33 Mounting evidence suggests this neurocardiogenic pathway is inadequate to fully explain vasodepressor syncope. Aberrant autonomic regulation, endogenous vasodilators, disordered baroreflex function, paradoxical cerebral autoregulation, and low serum ferritin may all play a role.32,34

What is the Treatment for Vasodepressor Syncope?

Therapy in the ED is supportive, focusing on education and reassurance. Patients and caregivers should be encouraged to maintain adequate hydration and enhance dietary salt intake. Patients should learn to recognize prodrome symptoms and promptly sit or lie supine for up to 10 minutes or until symptoms resolve. Potential triggers should be identified and avoided. Recurrence in adolescence is common.

Referral to cardiology and subsequent medical therapy may be required for those with recurrent syncope unresponsive to behavioral modification, abrupt syncope associated with significant injury, or chronic dizziness.

Are There Similar Forms of Syncope?

Orthostatic hypotension results from a failure of compensatory mechanisms to maintain cardiac output with any postural change. These mechanisms (vasoconstriction,
muscular contractions of the lower extremities, and venous valve competency) are mediated by increased sympathometric activity and parasympathometric inhibition. Symptoms include: dizziness, lightheadedness, blurred or tunnel vision, weakness, and syncope. Episodes occur frequently in the morning, after meals, or during exercise. Dehydration, blood loss, pregnancy, prolonged bed rest, and medication use (calcium channel blockers, vasodilators, phenothiazines, and diuretics) may contribute. Orthostatic testing is diagnostic. Standing blood pressures should be delayed to observe for possible, subsequent hypotension and syncope.\(^\text{16}\)

Postural orthostatic tachycardia syndrome (POTS) is diagnosed by observing posturally induced symptoms with robust tachycardia and without significant blood pressure decline.\(^\text{32}\) In POTS, the heart rate increases by more than 30 beats/min with the development of vasopressor symptoms.\(^\text{6}\) The incidence is unknown. Patients may or may not develop frank syncope, and often complain of orthostatic palpitations, profound fatigue and exercise intolerance, lightheadedness, and cognitive impairment.\(^\text{35}\) Symptoms improve with sitting. These patients have generalized malaise and are often misdiagnosed with chronic fatigue syndrome or psychiatric illness.\(^\text{36}\)

**CASE 2: NEUROPSYCHIATRIC DISEASE**

Bill is a 3-year-old boy brought in by his mother to the ED after an episode of “fainting” this afternoon. His mother states Bill went into the bathroom to “pee pee.” Two minutes later she heard the toilet seat bang and found him on the floor unresponsive. She witnessed no abnormal movement but his eyes were deviated to the left. He woke up 2 minutes later but remained confused for another 15 minutes. She noted he had wet his pull-ups but showed no visible signs of trauma. Bill had a febrile seizure at age 2 but is otherwise healthy. Review of systems is negative. Vital signs and physical examination are normal. An ECG is performed and interpreted as normal. Bill is diagnosed with a seizure. His primary care provider is contacted. Outpatient EEG and a follow-up with a neurologist is scheduled. Seizure precautions are given and he is discharged home.

**How Does One Differentiate Seizure from Pediatric Syncope?**

A detailed history and physical examination are typically sufficient to distinguish seizure from syncope. Typical seizures have a premonitory aura, generalized tonic-clonic activity, and a postictal period of lethargy and confusion. Patients with seizures do not experience the prodrome symptoms of syncope. Seizure findings typically include supine rather than upright posture at onset, convulsions before rather than after loss of consciousness, frothing at the mouth, tongue biting, incontinence, and warm, flushed, or cyanotic skin rather than pallor and diaphoresis.\(^\text{16}\)

**Can Pediatric Syncope Cause Seizure-Like Movements?**

Syncope can cause stiffening, myoclonic, and limited clonic movements, known as *convulsive syncope*, and is a common reason for the misdiagnosis of pediatric epilepsy.\(^\text{37}\) Transient global hypoxia leads to cortical suppression and disinhibition of limbic and subcortical structures.\(^\text{38}\) Movements are usually during the loss of consciousness phase, not before or at the onset of that phase; and are brief with multifocal or 1 to 2 clonic jerks rather than prolonged clonic-tonic movements.\(^\text{32}\) There is no postictal period.

**Can Migraine Cause Pediatric Syncope?**

Migraine can cause syncope; specifically, vertebrobasilar vascular spasm. Symptoms are atypical and may include severe a occipital headache before and/or after loss of
consciousness, unilateral vision change, ataxia, emesis, and vertigo. These episodes are longer in duration with stable hemodynamics.\(^1\) Scintillating scotomatas may precede onset.\(^{13}\)

**Are There Psychiatric Mimics of Pediatric Syncope?**

**Hyperventilation** and **conversion syncope** commonly occur in adolescents, usually in a highly emotional setting, and can result in a loss of consciousness. Both conditions are rare in children younger than 10 years.

**Hyperventilation** begins with an apprehensive feeling and deep sighing respirations. These subtle changes may not be obvious to the patient.\(^8\) Dyspnea, air hunger, and chest tightness may progress to loss of consciousness. Other symptoms include light-headedness, abdominal discomfort, palpitations, dizziness, paresthesias, and visual disturbances.\(^{16}\)

**Conversion syncope** or hysteria typically occurs in the presence of an audience and rarely produces injury. Episodes are not posture dependent and may last up to an hour.\(^8,^{13}\) In addition, there are no neurologic, autonomic, or cardiovascular changes. Patients often describe feeling calm and may remember the surrounding environment during the episode.\(^{16}\)

**CASE 3: CARDIAC DYSRHYTHMIA**

Frank is a 10-year-old boy brought in by ambulance to the ED after “fainting” this afternoon during recess. He says he was putting on his jacket and then woke up on the ground with a bleeding chin. Witnesses report Frank collapsed to the ground and failed to protect his chin from striking the floor. He was unconscious for 30 seconds before awaking spontaneously. There was no abnormal movement noted. His father states he has “fainted” twice this year but has never injured himself or visited a physician. There is no family history of sudden cardiac death. EMS report a point-of-care glucose of 90 mg/dL and normal vital signs during transport. Vital signs are normal. Physical examination reveals a 3-cm simple laceration to the chin. An ECG is performed with a delta wave, short PR interval with wide QRS complex identified. Frank is diagnosed with Wolff-Parkinson-White (WPW) syndrome and placed on a cardiac monitor. The laceration is repaired and he is admitted to the pediatric cardiology service.

**Is Cardiac Syncope Common in Pediatric Patients?**

Cardiac syncope is a serious concern but an uncommon diagnosis. A focused history and physical examination, screening ECG, and cardiology consultation when indicated can detect the majority of cardiac syncope and ultimately be life-saving. Cardiac causes account for 85% of sudden death in children and adolescent athletes, and 17% of young athletes with sudden death have a history of syncope.\(^{39}\) The ECG should be reviewed for any abnormality, with specific attention to the corrected QT interval (QTc), evidence of an abnormal or irregular rhythm, ischemia, axis deviation, and hypertrophy.

**What is the Correct Way to Calculate the QTc?**

Bazet’s formula, QTc = (QT)/square root (R-R) is the correct way to calculate the QTc. The QT interval should be measured from the beginning of the QRS complex to the end of the T wave in leads II, V5, or V6. The measured R-R interval should immediately precede the measured QT. Automated calculation from ECG machines cannot be relied on. The measurement should be averaged from several successive beats.
QTc calculation can be difficult to analyze in patients with very slow or fast heart rates, and those with sinus irregularities.

**What if the ECG Reveals QTc >0.450 Milliseconds?**

Long-QT syndrome (LQTS) is the prolongation of ventricular repolarization secondary to impaired ion channels in the myocardium. There are multiple genetic mutations that lead to dysfunction of potassium, sodium, and/or calcium ion channels which, in turn, leads to an overabundance of cations within the myocardium. LQTS puts the heart at risk for ventricular dysrhythmias such as torsades de pointes and ventricular fibrillation (Fig. 2). These ventricular dysrhythmias can cause syncope. If self-correction does not occur, then sudden death may follow.

**What Congenital Syndromes are Associated with LQTS?**

Romano Ward and Jervell-Nielsen-Lange are 2 commonly described syndromes associated with LQTS. Romano Ward is inherited in an autosomal dominant fashion, whereas Jervell-Nielsen-Lange is autosomal recessive and associated with congenital deafness; a family history of deafness is an important feature to ask about in screening for this syndrome. Cardiology referral and/or screening with an ECG is recommended for all identified family members of LQTS patients.40

**Can Medications Induce or Exacerbate LQTS?**

Several medications have been known to prolong the QT interval, including tricyclic antidepressants, antipsychotics, antibiotics (eg, macrolides), organophosphates, anti-histamines, and antifungals.41 Not all patients on these medications will develop LQTS. However, those with multiple risk factors are at greater risk for drug-induced LQTS. These risk factors include genetic predisposition, electrolyte abnormalities, female gender, multiple medications causing drug interactions, and structural heart disease.42

**What is Brugada Syndrome?**

Brugada syndrome is a heritable disorder of the cardiac sodium channels that creates a susceptibility to polymorphic ventricular tachycardia. This recurrent dysrhythmia can degenerate to ventricular fibrillation and cardiac arrest. Self-termination usually results in syncope or pre-syncope. The ECG is often dynamic with an ST elevation in the anterior precordial leads, that is, V1 and V2 being pathognomonic (Fig. 3).8,43,44 Patients who present with syncope have a 2-year risk of sudden cardiac death of approximately 30% if untreated. Placement of an internal defibrillator leads to an excellent prognosis with zero mortality at 10 years.44 Systematic familial study can identify asymptomatic affected family members who can benefit from early treatment to prevent complications.45

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**Fig. 2.** Rhythm strip demonstrating long-QT syndrome and the degeneration to torsades de pointes. (Courtesy of Amandeep Singh, MD, Oakland, CA.)
What Should One Consider if Heart Block is Found on the ECG?

First-degree heart block is usually an incidental finding in the setting of syncope, although it can be found in children with myocarditis, congenital heart disease, or cardiomyopathy. Second- and third-degree heart block detected on ECG is of concern in the setting of syncope. Neonatal lupus syndrome can cause heart block secondary to the passage of anti-Ro or anti-La antibodies in infants and young children. Maternal presence of these antibodies can occur with or without a history of maternal lupus. Congenital heart block may also be present in the setting of structural anomalies.

Acquired heart block can be caused by infectious myocarditis (most commonly Lyme disease), rheumatic heart disease, digoxin toxicity, and cardiac surgery. These cases require cardiology consultation for further testing and treatment. Symptomatic heart block may require pacing in the ED.

What is the Most Common Pediatric Dysrhythmia?

Supraventricular tachycardia (SVT) is the most common symptomatic pediatric dysrhythmia. SVT is a narrow complex tachycardia with a heart rate of more than 220 beats/min for infants and young children and greater than 180 beats/min for older children and adolescents. SVT can be due to a junctional tachycardia, ectopic atrial tachycardia, or an accessory atrioventricular pathway that causes reentrant tachycardia, as described in WPW syndrome.

SVT creates a relative anoxia from compromised cardiac output that can lead to syncope. Narrow complex tachycardia, absent or polymorphic p waves, and lack of beat-to-beat variability are common ECG findings. When SVT has resolved (either spontaneously or with treatment in the ED), ECG findings may also suggest the presence of an accessory pathway. In WPW syndrome, a delta wave (due to accessory conduction through the Bundle of Kent), and other associated ECG findings can be seen (Fig. 4).

What Rare Dysrhythmias Cause or Mimic Pediatric Syncope?

Sick sinus or sinus node dysfunction can result in a variety of symptomatic arrhythmias and syncope. These arrhythmias can be caused by cardiac surgery (eg, Fontan procedure), infectious or postinfectious changes (eg, myocarditis, pericarditis), congenital heart disease (sinus venous atrial septal defect, Ebstein anomaly), antiarrhythmic drugs (eg, digitalis, propranolol), hypothyroidism, or can be idiopathic with an otherwise normal heart. Observed bradytachyarrhythmia is very concerning. Profound bradycardia following a period of tachycardia (overdrive suppression) can lead to syncope and even death. Postoperative cardiac patients with a history of surgery involving the ventricles (eg, tetralogy of Fallot or a ventricular septal defect) are at risk for complete atrioventricular block or ventricular tachycardia.
Commotio cordis is a life-threatening dysrhythmia caused by a direct, nonpenetrating, often low-impact blow to the chest that may be confused with syncope. Two-thirds of cases occur during sporting events but reports include chest blows from physical abuse, fighting, snowballs, and hollow plastic toys. Ventricular fibrillation is typical but heart block, ventricular tachycardia, bundle branch block, ST-T wave abnormalities, and asystole can also occur. Patients are often amnestic to the event and have a contusion on the chest at the point of impact. Short-term observation and a cardiology release to return to athletics are required.

CASE 4: STRUCTURAL CARDIAC DISEASE

Steve is a 14-year-old boy brought in by ambulance to the ED after “fainting” this evening while helping his dad with yard work. He says he was running to the garage and then woke up on the ground. His father reports Steve collapsed and was unconscious for 1 minute before awaking spontaneously. He had 3 to 4 clonic jerks during the event. Steve denies prior events but does endorse periodic chest discomfort when participating in recent neighborhood soccer games. There is no family history of sudden cardiac death. Review of systems is otherwise negative. EMS report a point-of-care glucose of 80 mg/dL and normal vital signs en route. Vital signs and physical examination are normal. ECG reveals left ventricular hypertrophy. An ECHO is performed that shows hypertrophic cardiomyopathy. He is placed on a cardiac monitor and admitted to the pediatric cardiology service.

What Congenital Cardiac Structural Anomalies are Important to Consider in Pediatric Syncope?

Important cardiac structural anomalies that can present as syncope include hypertrophic cardiomyopathy (HCM), valvular disease such as aortic stenosis, and coronary anomalies causing ischemic heart disease. Chest pain, dizziness, and shortness of breath (particularly in the setting of exercise) are important historical elements to elicit. On physical examination, a systolic murmur may be apparent in HCM and aortic stenosis. Cardiac auscultation should also be performed with various maneuvers to increase and decrease blood flow to the heart. In HCM, the systolic murmur will increase when venous return is decreased to the heart (eg, during Valsalva or in standing position). The murmur will decrease with increased flow to the heart (eg, sitting, or bringing legs to chest). The opposite is true for the murmur of aortic stenosis.
HCM results from a diverse group of mutations and phenotypes that ultimately leads to abnormal hypertrophy of cardiac tissue.\textsuperscript{51} The estimated incidence is 1 in 500 individuals. Although obstruction can be the cause of syncope or sudden death in HCM, dysrhythmias from thickened muscle are more common. Left ventricular hypertrophy, axis deviation, and ST- or T-wave abnormalities may be identified on ECG (see Table 2).

**Can Pediatric Patients Have Ischemic Heart Disease?**

Myocardial ischemia or infarction is rare in children but must be considered. Syncope, chest pain (usually with exertion), shortness of breath, fatigue, and in infants, difficulty or sweating with feeds are all important historical features.

Anomalous left coronary artery from the pulmonary artery is a congenital cardiac anomaly in which the left coronary artery is attached to the pulmonary artery instead of the aorta. This anomaly results in deoxygenated blood being delivered to the myocardium, and potential ischemia and infarction. ECG findings of ST elevation, T or Q wave anomalies are consistent with infarction. Syncope and sudden death commonly present in infancy but may occur later in life.\textsuperscript{52}

Moreover, Kawasaki disease and the development of coronary artery aneurysms can also lead to ischemia. Patients with a known history of aneurysms or those with previously undiagnosed Kawasaki disease are also at risk for ischemic heart disease. The EP should always be mindful of ECG interpretation, as it can clearly delineate ischemic disease.

**CASE 5: ADDITIONAL CAUSES OF PEDIATRIC SYNCOPE**

Jill is a 14-month-old girl brought in by her grandmother to the ED after “fainting” this evening. Her grandmother states Jill became “very upset” when it was time to go to bed and then collapsed into her crib. She was unconscious for 10 seconds before awaking spontaneously. She turned blue briefly but had no abnormal movements. Her mother arrives and confirms this has happened one other time while being dropped off at daycare. There is no family history of sudden cardiac death. Review of systems is negative. The vital signs and physical examination are normal. An ECG is performed and interpreted as normal. Jill is diagnosed with a breath-holding spell. Precautions are given and she is discharged home.

**What is a Breath-Holding Spell?**

Breath-holding spells occur when children typically aged 6 to 18 months are startled by an intense emotional trigger: pain, fright, or anger. The child holds their breath and becomes limp or falls to the ground within seconds. The entire event usually lasts less than a minute and ends with gasping breaths. Cyanosis is common but pallor may be observed due to the vasodepressor mechanism.\textsuperscript{53} Convulsive syncope can occur.\textsuperscript{54}

Breath-holding spells are reported in 2% to 5% of children. Therapy is rarely required.\textsuperscript{55} Approximately 10% to 20% of children with a diagnosis of breath-holding spells develop more typical vasodepressor syncope in later life.\textsuperscript{56}

**Does Coughing Cause Pediatric Syncope?**

Tussive syncope is associated with bronchospasm from an acute infection, asthma, pertussis, or cystic fibrosis.\textsuperscript{13} Episodes are triggered by reduced cardiac output due to high intrathoracic pressures from the severe paroxysms of coughing. The transmission of high intrathoracic pressures to the subarachnoid space reducing cerebral blood flow may also contribute.\textsuperscript{16}
Does Hypoglycemia Cause Pediatric Syncope?

Hypoglycemia does not cause isolated syncope, as symptoms have a gradual onset without spontaneous resolution. Patients describe feeling weak, hungry, sweaty, agitated, and confused, and eventually develop altered mental status. Hypoglycemia is rare in children and adolescents except in the setting of fasting, insulin-dependent diabetes or suspected toxic ingestion. Measuring a serum glucose in all pediatric syncope patients is low yield and unnecessary.

Does Pregnancy Matter in Pediatric Syncope?

All menstruating females should have a urine pregnancy test performed. Syncope and near-syncope are common with normal pregnancy, especially in the third trimester, but can be associated with ectopic pregnancy, pulmonary embolism, and pregnancy-related cardiac disease. These high-risk diagnoses must be ruled out.

Can Carbon Monoxide Poisoning Present as Pediatric Syncope?

In the acute phase following carbon monoxide (CO) exposure, headache, nausea, and dizziness are common. As exposure increases, patients develop more pronounced and severe symptoms, including syncope. The brain and heart are the most oxygen-dependent organs and are also the most sensitive to the toxic effects of CO. A significant number of CO poisoning cases may go undetected as the signs and symptoms are diverse and easily confused with other illnesses.

SUMMARY

Pediatric syncope is a common presentation in the ED, with a large differential diagnosis. Most causes are benign but an evaluation must exclude rare life-threatening disorders. The keys to identifying high-risk patients include a detailed history, a focused but thorough physical examination, and a screening ECG on all patients. Key features on history and physical examination for identifying high-risk patients include exercise-related symptoms, a family history of sudden death, a history of cardiac disease, an abnormal cardiac examination, or an abnormal ECG. Ancillary testing should be limited to corroborate concerning findings from the history and physical examination; routine blood work and diagnostic imaging are not indicated. Education and reassurance are important because recurrence is common.

REFERENCES