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Chest Pain and Syncope in Children: A Practical Approach to the Diagnosis of Cardiac Disease

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Chest pain and syncope are common in children, accounting for numerous provider visits and leading to a considerable amount of testing.¹⁻⁷ The Centers for Medicare and Medicaid Services projects that health care spending will exceed 20% of the US Gross Domestic Product by 2018.⁸ Considering the looming crisis in health care finance and impending changes in the US health care delivery system, provider-driven methods to optimize resource utilization are critical. Careful evaluation of current practices, with particular attention to use of expensive and overutilized resources, is needed.

Pediatric chest pain and syncope share a paradox in that both are common and benign in the vast majority of cases, but can be cardinal symptoms of rare and serious cardiac conditions. The prevalence of serious cardiac pathology in children presenting with these symptoms is low, but extensive and costly cardiac testing is common and may be unnecessary in many cases.^{4,5} Outpatient visits for chest pain and syncope at Boston Children's Hospital have more than doubled over the last 10 years and now account for ~15% of all outpatient visits (Figure 1; available at www.jpeds.com). In this article, we discuss causes of pediatric syncope and chest pain and propose an approach focused on careful history and physical exam that aims to optimize testing and referral patterns while maintaining a high detection rate for serious heart disease.

Chest Pain

Case

An 18-year-old male presented to the pediatrician with a 2-week history of exertional chest pain. The onset of chest pain coincided with the beginning of track season. The chest pain occurred only during high levels of exertion, and forced the patient to stop competing in track. Physical examination findings were normal.

What are the concerning symptoms for a cardiac etiology of chest pain in this patient? What further evaluation is warranted?

Chest pain is a common presenting complaint to pediatricians, pediatric cardiologists, and pediatric emergency departments (EDs),^{1-5,9-11} accounting for at least 650 000 physician visits annually in patients aged 10-21 years.^{9,10} The overwhelming majority of cases of chest pain in otherwise healthy children have a noncardiac etiology.^{1-4,9-11} Numerous previous studies in multiple practice settings have demonstrated a cardiac etiology in only 0-5% of children and adolescents presenting with chest pain (Table I; available at www.jpeds.com). In contrast to adults, in whom chest pain often signals a significant cardiac

problem, the most common etiologies of chest pain in children are benign and include musculoskeletal, gastrointestinal, pulmonary, idiopathic, and psychogenic causes (Table II).^{2-5,10-12} In the largest review of patients seen in a cardiology clinic for chest pain, Saleeb et al¹¹ reported on a cohort of ~3700 patients seen over a 10-year period. The most common causes of chest pain were idiopathic (52%), musculoskeletal (36%), respiratory (7%), and gastrointestinal (3%). Only 37 of the 3700 patients seen (1%) had a cardiac etiology for chest pain, with supraventricular arrhythmias (n = 16) and pericarditis (n = 10) the most frequent causes. Only 9 patients (0.24%) had a cardiac conditions that put them at increased risk for sudden death, including myocarditis (n = 4), anomalous coronary origin from incorrect sinus of Valsalva (n = 3), hypertrophic cardiomyopathy (HCM; n = 1), and dilated cardiomyopathy (n = 1). The potentially life-threatening and more benign causes of cardiac chest pain were all identifiable by history, physical examination, and selected use of electrocardiography (ECG). Importantly, in more than 18 000 patient-years of follow-up of this cohort, there have been no missed diagnoses leading to cardiac death.

Despite the low prevalence of serious cardiac pathology in this population, chest pain generates considerable anxiety among patients, parents, and providers and often leads to evaluations that are low-yield, extensive, and costly. Owing largely to anxiety and misconceptions about the causes of chest pain in children, patients and their families frequently seek medical attention, and providers may have difficulty providing reassurance to families. Patients and their families vastly overestimate the prevalence of cardiac causes of chest pain while underestimating the more benign causes of chest pain, with families reporting that they suspect a cardiac etiology in more than 50% of cases, compared with the actual incidence of 0-5%.^{1,2,13} In a study of 100 consecutive patients seen in a pediatric practice for noncardiac chest pain, 69% reported restricting their activity, 40% had school absences related to chest pain, 44% thought they were having a heart attack, and 12% feared they had cancer.¹⁴ High levels of anxiety disorder have been documented in children with noncardiac chest pain and their parents, with 1 study finding psychiatric disorders listed in the Diagnostic and Statistical Manual of Mental Disorders, 4th Edition (predominantly anxiety disorder) in 60% of pediatric patients with noncardiac chest pain.^{14,15} Compared with patients seen for innocent murmurs, those with noncardiac chest pain have been shown to have greater levels of functional disability, somatization, and psychosocial distress, and to use significantly more health care services.^{15,16}

Understanding the cardiac and noncardiac etiologies of pediatric chest pain helps guide evaluation and determine the need for referrals and additional testing. Causes of noncardiac chest pain are listed in a Table II. Musculoskeletal chest pain is the most common cause, accounting for up to 70% of cases; it is characteristically sharp, fleeting, and exacerbated by deep inspiration, and may be reproducible on palpation. The major cardiac causes of pediatric chest pain, and key red flag physical examination and history findings associated with each diagnosis, are listed in Table III.

Owing to the acuity and severity of symptoms, most patients with chest pain related to serious cardiac pathology present to the ED, with a smaller number, typically with less acute chest pain, presenting in outpatient clinics. In a 10-year review, Kane et al¹⁷ identified 171 patients with a serious cardiac condition who presented with chest pain. Only 41 of these patients (24%) presented through an outpatient clinic, 32 with a potentially serious cardiac diagnosis, included coronary anomalies (n = 21), HCM (n = 3), myocarditis (n = 1), pericarditis (n = 4), and pulmonary artery hypertension (HTN) (n = 1). The most common diagnoses in the ED diagnoses were pericarditis (n = 58), myocarditis (n = 45), and pulmonary embolus (n = 13). All patients who presented through outpatient clinics were identifiable by the presence of at least 1 of the following: exertional chest pain (n = 20), abnormal physical exam findings (n = 12), abnormal ECG findings (n = 5), or severe

underlying systemic illness ($n = 1$). According to the authors, their results suggest that a careful and focused history and physical examination, along with a screening ECG, will identify essentially all patients with a serious cardiac condition.

Despite the low prevalence of serious cardiac pathology, extensive and costly cardiac evaluation, including exercise stress testing (EST), echocardiography, and ambulatory ECG, is common and often low yield.^{3,4,11} EST is extremely low yield in this population, as evidenced by 3 recent studies of more than 600 pediatric patients with chest pain in whom EST detected no cardiac disorders.^{2,3,18} Ambulatory ECG also has extremely low yield in this population and has no role in evaluating chest pain in the absence of palpitations or syncope.^{3,19} Echocardiography is also low yield and is considerably more likely to uncover incidental findings than the etiology of chest pain,^{3,4} but nonetheless it remains the modality of choice for patients with concerning history, examination, or ECG findings given its ability to diagnosis most of the serious cardiac conditions that can present with chest pain, including anomalous coronary artery origin, cardiomyopathies, pulmonary HTN, pericardial effusion due to pericarditis, and left ventricular outflow tract obstruction. Indications for and utility of various cardiac testing modalities in pediatric chest pain and syncope are summarized in Table III.

As evidenced by the aforementioned findings of Saleeb et al¹¹ and Kane et al,¹⁷ an approach based on targeted history, physical exam, and selective use of ECG can identify patients requiring further evaluation by subspecialists and cardiac testing. A standardized approach to pediatric chest pain is currently being implemented in our pediatric cardiology department as part of a broader quality improvement initiative known as Standardized Clinical Assessment and Management Plans (SCAMPs). SCAMPs are designed to reduce practice variation, optimize resource utilization, and improve patient care.^{4,5,20} Although this SCAMP is intended for use by pediatric cardiologists, a similar management plan using specific red flag history and physical exam findings has been introduced for use by general pediatricians (Figure 2; available at www.jpeds.com). The algorithm uses history and physical exam red flag findings (Table IV) to trigger referral to cardiology for further evaluation.

As shown in the algorithm, patients with abnormal physical exam findings, abnormal ECG, or a high-risk medical or family history should be referred for further evaluation. Referral is also recommended for patients with anginal chest pain, exertional chest pain (ie, pain at moderate or high levels of aerobic activity) that cannot be attributed to respiratory disease or other clear etiology, chest pain with palpitations, or exertional syncope. Patients with fever, abnormal vital signs, tachypnea, and/or lung findings on physical exam should be referred to the ED or for chest radiography, depending on clinical status. This approach has been tested and validated in our outpatient cardiology clinic and has led to reductions in practice variation and in resource utilization for EST and ambulatory rhythm monitoring.^{3,4} Importantly, quality of care has been maintained as, with no known missed cardiac diagnoses in this cohort.^{3,4}

Case Resolution

Based on the exertional nature of his chest pain, the patient was referred to a pediatric cardiologist. At the cardiology visit, ECG and physical exam findings were normal. Echocardiography demonstrated a left coronary artery arising from the right sinus of Valsalva. The patient was referred for surgical correction.

Syncope

Case

A 16-year-old female presented to her pediatrician after experiencing 2 recent syncopal episodes. One episode occurred at school after she stood to leave the classroom, and the other occurred while she was taking a shower. Both episodes were preceded by nausea, lightheadedness, and “tunnel vision.”

What are concerning symptoms for a cardiac etiology of syncope? What further evaluation is warranted for this patient?

Syncope is a sudden transient loss of consciousness and postural tone as result of decreased cerebral blood flow, followed by spontaneous recovery. The mechanism of cerebral hypoperfusion is most commonly a transient reflex failure of peripheral vascular tone in combination with relative bradycardia, but also can be mechanical (eg, carotid stenosis, left ventricular outflow tract obstruction) or electrical (eg, rapid tachycardia, abrupt bradycardia). Syncope is a very common disorder with a bimodal incidence in children and adolescents, peaking in females aged 15-19 years.^{5,6} Up to one-third of the population will experience syncope at some point in life, and 15% will experience syncope before age 21.²¹⁻²⁵ Similar to chest pain, although syncope is common and usually benign, it frequently raises concern and anxiety about the possibility of serious underlying cardiac pathology and sudden cardiac death.

Although syncope is common, sudden cardiac death in young people is extremely rare, with an incidence of only 0.4-1.0 per 100 000.^{26,27} Benign etiologies, primarily neurocardiogenic syncope (NCS), are the most common cause of syncope in children, with NCS accounting for up to 75%-80% of cases.^{22,23,25} An important task of the pediatrician is to differentiate the benign forms of syncope from those associated with increased risk for sudden cardiac death requiring further evaluation. A careful history, dynamic physical exam, and ECG, along with a sound understanding the causes of pediatric syncope and red flag findings that indicate a potentially serious condition, will allow providers to accurately distinguish the vast majority of cases of serious cardiac pathology from the more common benign etiologies.^{5,23,24,28}

Various systems for categorizing syncope broadly classify syncope as cardiac, NCS (including reflex), metabolic, neurologic, or psychogenic (Table V; available at www.jpeds.com). Typical features of NCS are prominent prodromal symptoms, including nausea, dizziness, and tunnel vision, along with pallor, brief loss of consciousness, and minimal residual symptoms.^{6,24,25} NCS is often triggered by prolonged upright positioning, standing in a warm shower, or sudden orthostatic changes. Features of cardiac syncope include acute collapse, often with exertion, and with few warning symptoms preceding the event.^{6,28,29}

Potential cardiac causes of syncope can be grouped into arrhythmic and mechanical causes. In a patient with any of the red flag signs or symptoms listed in Table VI or with abnormal physical exam or ECG findings, a cardiac etiology should be suspected, and referral for further evaluation is warranted. The traditional diagnostic screening of history and physical exam along with ECG will identify the vast majority of patients with a serious underlying cardiac disease.^{6,8,21,23} History should focus on characterizing prodromal symptoms, details of events, duration and frequency of events, residual symptoms after events, and symptoms associated with events. Syncope occurring during exercise is highly concerning and should prompt urgent referral and further investigation. In contrast, postexertional syncope is often attributed to NCS and related to vasodilation and shifting autonomic states. Syncopal events

precipitated by noise or emotion are of concern for possible triggered ventricular arrhythmias, particularly long QT intervals, and also should prompt evaluation.

Physical examination should include a complete evaluation, with orthostatic vital signs, a full cardiac exam including dynamic auscultation, and neurologic exam. The presence of orthostasis supports a diagnosis of NCS or postural orthostatic tachycardia syndrome but does not exclude more serious etiologies. Pathological murmurs and/or gallop heart sounds may be detected in the setting of cardiomyopathies, myocarditis, or left ventricular outflow tract obstruction. Dynamic auscultation with the patient moving from squatting to standing may uncover a systolic ejection-type murmur related to dynamic left ventricular outflow tract obstruction in patients with HCM. A loud and/or unsplit second heart sound should raise concern for pulmonary HTN. ECG should be performed in all patients with syncope. Heart block, prolonged QT interval, Wolff-Parkinson-White syndrome, cardiomyopathies, significant right ventricular hypertrophy, and Brugada syndrome are cardiac causes of syncope that can be detected on resting ECG.

Evaluation is focused both on confirming the diagnosis of NCS and evaluating for potentially serious conditions carrying an increased risk of mortality or requiring therapy. Similar to the strategies for treating pediatric chest pain, we have instituted a structured approach to syncope through the use of a SCAMP that aims to reduce unnecessary testing and referrals while still identifying important etiologies. A diagnostic decision tree using physical exam findings, event description, family history, and ECG findings is presented in Figure 3 (available at www.jpeds.com). Red flag findings in any of these areas (Table VI) should trigger referral to a pediatric cardiologist. Routine use of echocardiography, ambulatory ECG, tilt-table testing, and EST are low-yield and expensive and may unnecessarily increase patient and family anxiety (Table III).³⁰ A recent study of 480 children with syncope demonstrated that a rapid diagnostic screen of event history, positive family history, abnormal physical exam, or abnormal ECG identified 21 of 22 patients with a cardiac etiology.⁷ In this study, echocardiography did not contribute to diagnosis in any patient. In a study of 495 patients undergoing ambulatory ECG for variety cardiac symptoms, monitoring was diagnostic only in patients with palpitations, and in patients with syncope, presyncope, or chest pain, ambulatory ECG had zero diagnostic yield.¹⁹ Tilt-table testing has been suggested as a means of provoking NCS in susceptible patients to confirm the diagnosis. However, tilt-table testing has limited specificity, with as many as 40% of adolescents with no previous history of syncope demonstrating a positive tilt-table test.^{6,31} In addition, poor sensitivity (65%-75%) and reproducibility (50%-87%) limit the usefulness of tilt-table testing.^{28,32} EST may be reasonable in patients with exertional syncope, but not in those with typical syncope.⁵

Patient and family reassurance and education on preventing or limiting the severity of further episodes are the first-line treatment for NCS syncope.^{5,6,24,28,29} Adequate oral fluid and salt intake will help improve symptoms in up to 90% of patients.²⁸ Avoiding caffeine and using postural techniques to prevent venous blood pooling, including isometric extremity contraction, toe raises, and arm folding or leg crossing, may be helpful. Patients should be instructed to assume a supine position at the onset of prodromal symptoms, both to help abort the syncopal event and to limit injury should loss of consciousness occur. Fortunately, most patients, even those who are highly symptomatic, will demonstrate improvement in symptoms with age. Should symptoms increase in severity or frequency with conservative management, referral to a pediatric cardiologist for further evaluation and/or empiric pharmacologic therapy may be warranted.

Case Resolution

This patient's physical exam and ECG findings were normal apart from a heart rate increase of 35 bpm on orthostatic vital signs. The pediatrician counseled the patient to increase her salt and water intake and to perform postural techniques to help prevent further vasovagal syncope. At follow-up 3 months later, the patient reported no additional syncopal episodes.

Discussion

Pediatric syncope and chest pain are common and generate considerable anxiety in patients, families, and providers. Owing in part to this anxiety, extensive costly and low-yield evaluation is often undertaken. In light of the impending changes in health care financing, providers need to assess areas of potential resource overutilization in their fields and to explore strategies for improving this situation. In this article, we have reviewed the causes of pediatric chest pain and syncope, as well as the red flag findings that should prompt further evaluation, and have proposed a balanced approach to evaluation of these symptoms focused on a detailed history and physical exam. This approach aims to optimize testing and referrals while recognizing the rare cases of significant heart disease presenting to the outpatient office with chest pain or syncope. Increased knowledge and use of systematic approaches and informed application of previous clinical experience should help ensure sufficient evaluation of chest pain and syncope by front-line providers.

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Glossary

ECG	Electrocardiography
ED	Emergency department
EST	Exercise stress testing
HCM	Hypertrophic cardiomyopathy
HTN	Hypertension
NCS	Neurocardiogenic syncope
SCAMP	Standardized Clinical Assessment and Management Plan

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Figure 1.
Annual cardiology clinic visits for chest pain and syncope at Boston Children's Hospital.

Figure 2.
Chest pain management algorithm.

Figure 3. Syncope management algorithm. *POTS*, postural orthostatic tachycardia syndrome; *WPW*, Wolff-Parkinson-White.

Table I

Frequency of cardiac etiology for chest pain

Study	Location	Total patients	Patients with cardiac chest pain, n (%)
Driscoll et al (1976) ¹	Ped	43	0 (0)
Pantell and Goodman (1983) ¹³	PC	100	1 (1)
Selbst et al (1998) ¹⁰	Ped ED	407	16 (4)
Gastesi et al (2003) ³³	Ped ED	161	1 (0.5)
Massin et al (2004) ³⁴	PC/Ped ED	69/168	3 (4)/8 (5)
Danduran et al (2008) ²	PC	263	0 (0)
Friedman et al (2011) ³	PC	406	5 (1.2)
Saleeb et al (2011) ¹¹	PC	3700	37 (1)

PC, pediatric cardiology office; *Ped*, pediatrician's office; *Ped ED*, pediatric ED.

Table II

Noncardiac etiologies of chest pain

Cause	Estimated incidence	Examples
Musculoskeletal	50%-68%	Costochondritis
		Precordial catch syndrome
		Muscle strain
		Trauma
		Slipping rib syndrome
Respiratory	3%-12%	Asthma
		Pneumonia
		Bronchitis
		Pleuritis
		Pulmonary embolus
Gastrointestinal	2%-8%	Pneumothorax
		Gastroesophageal reflux disorder
		Esophagitis
		Gastritis
		Pancreatitis
Psychogenic	10%-30%	Gastric ulcer
		Biliary disease
		Conversion disorder
Other	<10%	Panic/anxiety attack
		Skin infection
		Breast disease
		Acute chest syndrome in sickle cell disease

Table III

Utility and indications for cardiac testing in pediatric chest pain and syncope

Symptom/test	Indication for test	Usefulness	Conditions diagnosed
Chest pain			
ECG	Abnormal physical exam, ECG, exertional chest pain, or palpitations	Selected patients	Cardiomyopathy, myocarditis, pericarditis, with or without pulmonary HTN
Echocardiography	Abnormal physical exam, ECG, family history, or exertional chest pain	Selected patients	Anomalous coronary artery origins, cardiomyopathy, myocarditis, pericarditis, pulmonary HTN, left ventricular outflow obstruction
Troponin testing	Suspected myocarditis or pericarditis	Selected patients	Myocarditis, pericarditis, coronary ischemia
Ambulatory ECG	Chest pain and palpitations	Rarely useful for chest pain	Atrial or ventricular arrhythmia, intermittent heart block
EST	Exertional chest pain and exertional syncope or palpitations	Rarely useful	Coronary ischemia, exercise-induced asthma (if spirometry included)
Syncope			
ECG	All patients with syncope	First-line diagnostic test	Long QT interval, short QT interval, Wolff-Parkinson-White syndrome, cardiomyopathy, pulmonary HTN, Brugada, heart block, with or without ARVD
Echocardiography	Abnormal physical exam, ECG, family history, or alarming event history	Selected patients	Cardiomyopathy, pulmonary HTN, with or without ARVD, coronary anomalies
Exercise ECG/EST	Exertional syncope or exertional symptoms	Selected patients	Catecholaminergic polymorphic VT, long QT interval
Ambulatory ECG	Syncope plus palpitations, frequent symptoms, exertional syncope	Selected patients	Atrial or ventricular arrhythmia, intermittent heart block, reflex bradycardia
Tilt-table test	Very frequent, recurrent syncope or atypical syncope	Rarely useful	Confirmation of NCS (low specificity), postural orthostatic tachycardia syndrome

ARVD, arrhythmogenic right ventricular dysplasia; VT, ventricular tachycardia.

Table IV

Cardiac causes of chest pain and red flag history, exam, and ECG findings

Cause	History	Physical exam findings	ECG findings
HCM	Positive family history Exercise intolerance Exertional chest pain Syncope and/or arrhythmia	Dynamic systolic murmur	Left ventricular hypertrophy or left axis deviation ST segment or T-wave changes Q waves Arrhythmias, ventricular premature beats Ventricular preexcitation (Wolff-Parkinson-White)
Dilated cardiomyopathy	Family history Decreased exercise tolerance, syncope Heart failure symptoms	Gallop Mitral regurgitation murmur	Intraventricular conduction delay High or low QRS voltages Arrhythmia, premature beats
Anomalous coronary artery origin	Exertional chest pain Exertional syncope	Usually normal	Usually normal
Coronary ischemia	Predisposing conditions <ul style="list-style-type: none"> History of Kawasaki disease Cardiac surgery or heart transplant Systemic arteriopathy (Williams syndrome) Severe familial hypercholesterolemia Drug use: cocaine, sympathomimetics Anginal chest pain 	Tachycardia Tachypnea New murmur or gallop	ST segment depressions or elevation T wave changes Q waves
Severe left ventricular outflow tract obstruction	Exertional symptoms Exertional syncope	Loud systolic murmur	Left ventricular hypertrophy Left ventricular strain pattern
Arrhythmia	Palpitations Syncope Positive family history	Irregular rhythm	Atrial arrhythmia Ventricular arrhythmia Premature contractions Ventricular preexcitation (Wolff-Parkinson-White)
Pericarditis	Positional chest pain Predisposing factors: <ul style="list-style-type: none"> Rheumatologic conditions Malignancy Mediastinal radiation Infection (HIV, tuberculosis, viral) Renal failure Recent cardiac surgery 	Cardiac rub Tachycardia/tachypnea Distant heart sounds, JVD	Diffuse ST segment changes T wave inversions
Myocarditis	Fever Viral prodrome Short duration of symptoms	Tachycardia Tachypnea With or without gallop rhythm, ventricular ectopy	Diffuse ST segment changes T wave inversions PR depression Ventricular ectopy

Cause	History	Physical exam findings	ECG findings
Aortic dissection	New onset heart failure symptoms	Cardiovascular collapse	Low QRS voltages
	Personal or family history of bicuspid aortic valve or connective tissue disorders (Marfan, Loey-Dietz, Ehlers-Danlos type IV, others) Acute onset sharp or tearing type of pain	Marfanoid body habitus	See coronary ischemia above
Pulmonary embolus	Pain description: acute onset, pleuritic, associated dyspnea Personal or family risk factors (inherited thrombophilia, hypercoaguable states, immobilization, medications)	Right ventricular heave (elevated right ventricular pressure) Loud and/or unsplit S2 (if right ventricular pressure elevated)	Right ventricular hypertrophy Right ventricular strain pattern

JVD, jugular venous distension.

Table V

Causes of pediatric syncope

Cause	Examples
Neurally mediated	Vasovagal/NCS
	Reflex/triggered NCS <ul style="list-style-type: none"> • Stretch • Cough • Micturition • Needle phobia • Psychological stress (seeing blood, noxious trigger)
Cardiac	Orthostatic syndromes <ul style="list-style-type: none"> • Orthostatic HTN • Postural tachycardia • Breath-holding spells
	Mechanical <ul style="list-style-type: none"> • Left ventricular outflow tract obstruction • Pulmonary hypertension • Dilated cardiomyopathy (can also be arrhythmic) • Myocarditis (can also be arrhythmic) • Coronary artery anomalies • Cardiac tumors
	Arrhythmic <ul style="list-style-type: none"> • Primary electrical disease • Wolff-Parkinson-White • LQTS • Catecholaminergic PMVT • Brugada • ARVC • Congenital/acquired heart block • VT • Atrial tachycardia/SVT (rarely leads to syncope in structurally normal heart)
	Cardiomyopathy <ul style="list-style-type: none"> • HCM • DCM • Noncompaction • Arrhythmogenic right ventricular dysplasia
Neurologic	Epilepsy syndromes Concussion/postconcussion Autonomic failure (familial dysautonomia)

Cause	Examples
Metabolic/endocrine	Spinal cord
	Narcolepsy
	Addison disease
	Hypoglycemia
	Intoxication
	Hypovolemia
Environmental	Anorexia
	Heat exhaustion
Psychogenic	Anxiety triggered syncope
	Factitious
	Conversion
	Hyperventilation/panic

LQTS, long QT syndrome; *PMVT*, polymorphic ventricular tachycardia; *DCM*, dilated cardiomyopathy; *SVT*, supraventricular tachycardia.

Table VI

Syncope evaluation and red flag findings

Event history	Past medical history	Family history	Physical exam	ECG
<ul style="list-style-type: none"> • Exertional syncope • Event precipitated by noise or emotion • Prodrome/ event description not consistent with NCS 	<ul style="list-style-type: none"> • Congenital heart disease • Cardiac surgery • History of Kawasaki disease 	<ul style="list-style-type: none"> • Known familial ventricular arrhythmias * • Sudden cardiac death or unexplained death in first- or second-degree relative aged <50 y • Cardiomyopathy in first- or second-degree relative • Heart failure in first- or second-degree relative aged <50 y 	<ul style="list-style-type: none"> • Tachycardia or tachypnea • Irregular rhythm • Pathological murmur, including dynamic murmur (HCM) • Gallop • Click • Loud or single S2 	<ul style="list-style-type: none"> • Long QT interval (QTc >440 msec in males, 450 msec in adolescent females) • Short QT interval • Right ventricular hypertrophy • Brugada pattern • Pattern consistent with HCM (increased left ventricular voltages, T wave inversions) • Atrial or ventricular ectopy • Heart block • Ventricular preexcitation (Wolff-Parkinson-White)

* Includes long QT interval, Brugada syndrome, catecholaminergic polymorphic VT, arrhythmogenic right ventricular dysplasia, and others.