Child with Syncope

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1 Introduction

Syncope is defined as a sudden, self-limited loss of consciousness, and loss of postural tone resulting from transient global cerebral hypoperfusion. The recovery is complete, spontaneous and without any neurologic sequelae [1, 2]. In its most common form, it is typically preceded by a prodromal phase consisting of nonspecific symptoms such as nausea, blurry vision, dizziness, sweating, and pallor or cold skin lasting from few seconds to couple minutes [3]. Presyncope is another term commonly used in the context of syncope. This is usually defined as the feeling that one is about to pass out but the child remains conscious albeit a transient loss of postural tone [4]. Although pediatric syncope is usually due to non-life-threatening causes and requires minimal evaluation in the emergency department, most children with syncope, especially recurrent syncope will experience stress and anxiety and will be referred for cardiac and or neurologic evaluation. The primary objective of evaluation of a child with syncope is to determine the etiology and rule out life-threatening cardiac and neurological disorders. This can be accomplished in most situations by obtaining a detailed history and performing a thorough physical examination.

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2 Epidemiology

Syncope occurs in approximately 15–25% of children with predominance in girls [3, 5]. Despite being so commonly encountered in the first two decades of life, only a small percentage of children seek medical attention. Syncope accounts for in 1 in 2000 visits to the emergency department, i.e., approximately 1% of all pediatric ED visits. It occurs at any age, with different frequencies and different etiologies and accounts for 126/100,000 children seeking medical attention [6–8]. Fifteen percent of all children will experience at least one episode of syncope before the age of 18 years [9].

3 Etiology

Syncope is caused by transient global hypoperfusion of the brain. In adults, the most common cause of syncope is cardiac disease while in children the most frequent cause of syncope is neurocardiogenic, also known as vasovagal syncope. Vasovagal syncope accounts for 75% of all causes while cardiac disease accounts for 10% of causes of syncope in children. See Table 1 for the different forms of syncope that can be encountered in children. Seizure disorders and neurologic disorders are another category that can present as syncope in 5% of children. This section will focus on how to distinguish benign from life-threatening causes of syncope with

Table 1 Causes of pediatric syncope

1.	Neurally mediated syncope
	Neurocardiogenic or vasovagal syncope
	Orthostatic hypotensive syncope
	Postural orthostatic tachycardia syndrome (POTS)
	Breath holding spells
	Situational syncope (coughing, micturition, defecation, hair grooming)
2.	Cardiac syncope
	Hypertrophic cardiomyopathy
	Aortic stenosis
	Coronary anomalies
	Myocarditis or cardiomyopathy
	Arrhythmias
3.	Other causes of syncope
	Seizures
	Convulsive syncope
	Psychogenic syncope
	Metabolic (hypoglycemia, hypoxia, or electrolyte abnormalities)
	Drug-induced syncope

some pointers to distinguish syncope from seizures. Please see Table 1 in chapter "Child with Attention Deficit Disorder/Child with Attention Deficit Hyperactivity Disorder (ADHD)" where differential diagnosis of seizures is discussed.

4 Neurally Mediated Syncope

4.1 Orthostatic Intolerance Syndromes

Orthostasis refers to the upright position. The conditions listed below all share a common thread, i.e., the symptoms are brought on by change in posture and there is inability to tolerate the upright posture.

4.2 Orthostatic Intolerance and Orthostatic Hypotension

Orthostatic intolerance is a common disorder in children, and it is frequently seen in the outpatient setting. It is characterized by the presence of symptoms that start with a change to upright position and are relieved by lying down. If symptoms start in the supine position, then orthostatic intolerance is ruled out. The most frequently reported symptoms (also called orthostatic symptoms) include palpitations, lightheadedness, headache, fatigue, weakness, nausea, abdominal discomfort, pallor, and diaphoresis [10].

Orthostatic hypotension or postural hypotension is defined as a sustained decrease in systolic blood pressure of at least 20 mmHg or diastolic blood pressure of at least 10 mmHg within 3 min of standing. Orthostatic hypotension is a sign and it is not always associated with symptoms. If the fall in blood pressure is prominent, cerebral blood perfusion is decreased and it can lead to syncope [11].

4.3 Neurocardiogenic Syncope (Vasovagal Syncope)

In vasovagal syncope, also referred to as "common faint," there is a series of events leading to loss of cerebral function. See Fig. 1 for pictorial depiction. Following certain triggers such as sudden or prolonged standing, hot shower, the sight of blood, any orthostatic stress, or venous pooling, there is decrease in ventricular preload. This results in initial hypotension followed by increased heart rate. Reflexive tachycardia is not able to compensate for the decrease in cardiac output leading to cardiovascular collapse with progressive hypotension, vasodilation, and bradycardia. This phase corresponds with symptoms of autonomic activation such as nausea and warmth with symptoms of cerebral hypoperfusion: blurry vision and

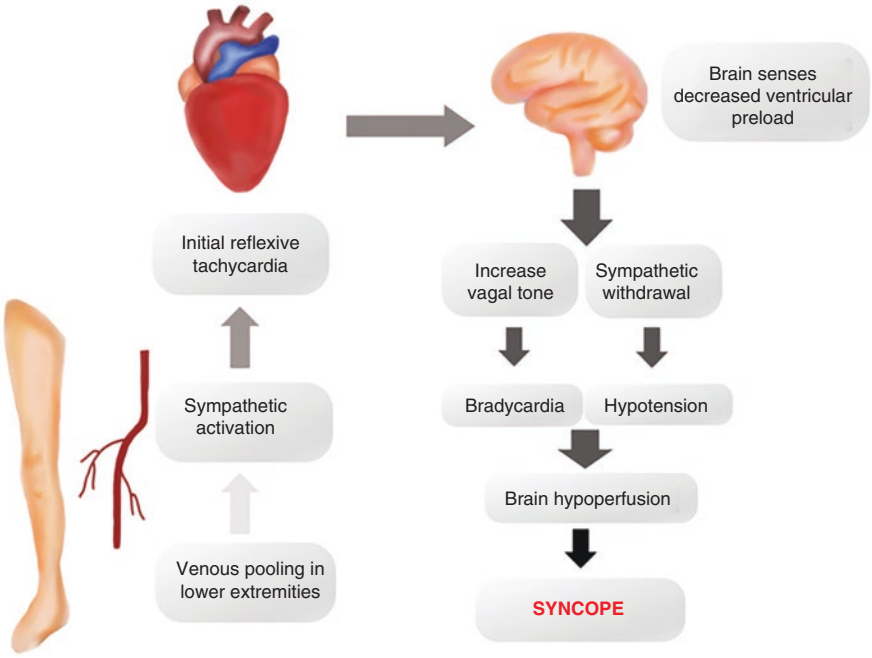

Fig. 1 Mechanism of vasovagal syncope

dizziness. Progressive decrease in blood pressure, systolic below 60 mmHg or mean arterial blood pressure of 30–40 mmHg results in loss of consciousness. Once the child is in the supine position, there is rapid increase in venous blood return to the heart, resulting in improved mean arterial pressure and improvement of symptoms which usually occurs within 30 seconds of being placed back in the horizontal position [12, 13].

4.4 Postural Orthostatic Tachycardia Syndrome (POTS)

POTS is a clinical syndrome characterized by symptoms of orthostatic intolerance with a change from supine position to an upright position. It is defined by an increase in heart rate of ≥30 beats/min, or a rate that exceeds 120 beats/min within 10 min of standing or head-up tilt, without orthostatic hypotension (a decrease in systolic blood pressure of 20 mmHg or more and/or decrease in diastolic blood pressure of 10 mmHg or more) [14]. This condition has a prevalence of 0.2% in the United States, is seen more frequently between ages of 15 and 25 years, and also has a female predominance. Symptoms experienced by patients with POTS include: light-headedness, palpitations, tremor, generalized weakness, blurred vision,

exercise intolerance, and fatigue. Many of these patients will faint although presyncope is seen more frequently. Patients with POTS will have chronic symptoms that can typically last for 6 months or longer and will often experience chronic fatigue with limitations to complete their daily activities [8, 14].

5 Cardiac Syncope

Cardiac syncope is the result of a sudden decrease in cardiac output, leading to decreased brain perfusion with subsequent loss of consciousness [5]. Although this group accounts for only 10% of all causes of syncope in children, it has the highest risk for sudden death compared to other causes of syncope [5]. History and physical examination serve as the main pillars in distinguishing cardiac causes of syncope from the more common vasovagal syncope. Several features that will be discussed in subsequent sections may serve as red flags for potentially life-threatening cardiac causes of syncope. In this review, we have divided the cardiac causes of syncope into two main categories: structural or anatomical and cardiac arrhythmias. The main cardiac diseases in the first category include: aortic stenosis, hypertrophic cardiomyopathy, and coronary abnormalities. Although myocarditis and other forms of cardiomyopathy are primarily disorders of myocardial function, they will be included in this category of cardiac syncope for practical purposes. Among arrhythmias presenting as syncope in children, the most common are: complete heart block and tachyarrhythmias including Long QT syndrome, Wolff–Parkinson–White syndrome, and Brugada syndrome [6, 7, 15]. Table 2 below presents an overview of common causes of cardiac syncope in children.

Table 2 Causes of cardiac syncope

1. Structural or anatomic	
Hypertrophic cardiomyopathy (HCM)	Syncope during exercise Family history of HCM or sudden death Systolic murmur that increases with Valsalva maneuvers Left ventricular hypertrophy on EKG
Aortic stenosis	Syncope during exercise Systolic murmur and ejection click Left ventricular hypertrophy on EKG
Myocarditis and/or cardiomyopathies	Symptoms of heart failure Abnormal vital signs: tachycardia and hypotension S3 or S4 gallop ST- T wave abnormalities on EKG
Coronary abnormalities	History of chest pain with exercise Abnormal EKG
Pulmonary hypertension	Loud P2 component or abnormal S2 splitting Right ventricular hypertrophy on EKG

(continued)

Table 2 (continued)

2. Arrhythmias	
Wolff–Parkinson–White syndrome	History of palpitations Ventricular pre-excitation or delta wave on EKG
Supraventricular tachycardia	History of palpitations of sudden onset and offset
Bradycardia	Abnormal heart rate for age Second-degree or third-degree heart block on EKG
Long QT syndrome	Syncope during exercise or startle Family history of sudden death or congenital deafness Prolonged QTc on EKG

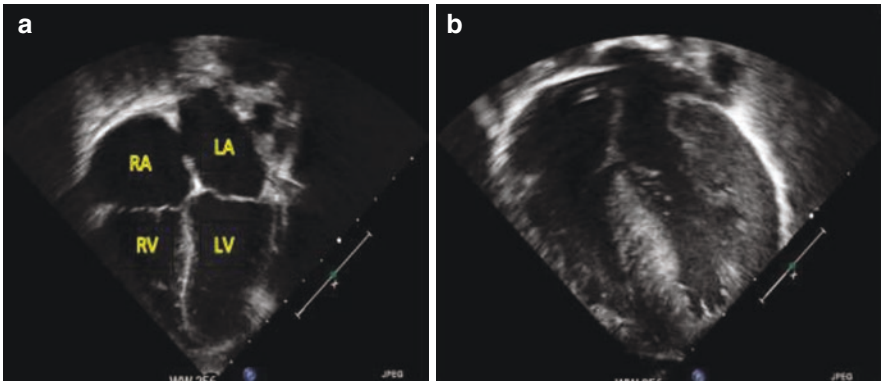

Fig. 2 Echocardiogram of a patient with normal heart (**a**) and a patient with hypertrophic cardiomyopathy (**b**). *RA* right atrium, *RV* right ventricle, *LA* left Atrium, *LV* left ventricle

5.1 Hypertrophic Obstructive Cardiomyopathy (HOCM)

Hypertrophic obstructive cardiomyopathy (HOCM) is a genetic disorder that is characterized by asymmetric septal hypertrophy, which produces a dynamic left ventricular outflow tract obstruction (LVOTO). The majority of patients have a benign course and are mostly asymptomatic. Palpitations, presyncope, and syncope in these patients might be caused by arrhythmia, usually ventricular tachycardia, or LVOTO. When arrhythmias and/or syncope are present they are considered a major risk factor for sudden cardiac death. A systolic ejection murmur at the mid and lower left sternal borders or at the apex, a left ventricular lift and a systolic thrill at the apex or along the lower left sternal border may be present. Electrocardiographic abnormalities include left ventricular hypertrophy, ST segment, T wave abnormalities, and abnormal deep Q waves. The treatment is aimed at providing symptomatic relief and decreasing the risk of sudden cardiac death. Since HOCM is the most common cause of sudden cardiac death in young competitive athletes, patients with HOCM need to be evaluated and cleared by a cardiologist before returning to sports [1, 6]. Figure 2 is an echocardiogram of a child with HOCM.

5.2 Aortic Stenosis

Aortic stenosis is the most common type of congenital left ventricular outflow tract obstruction, which causes decreased blood flow to the aorta and poor coronary perfusion. Abnormalities of the aortic valve can range from asymptomatic malformations (bicuspid aortic valve, which is the most common form of aortic valve disease) to severe ductal dependent lesions (critical aortic stenosis). On physical examination, an ejection click may be identified and if aortic stenosis is severe, there might be paradoxical splitting of S2. A harsh, mid-systolic ejection murmur might also be heard at the second right or left intercostal space. Most children with mild-to-moderate aortic stenosis are asymptomatic and they can present with occasional exercise intolerance, exertional chest pain, and easy fatigability. Children with severe obstruction can present with syncope; and even sudden cardiac death from ischemia in 1–2% of patients with severe aortic stenosis. Treatment options include balloon valvuloplasty or surgical relief of the obstruction [2, 7].

5.3 Congenital Coronary Artery Abnormalities

Congenital coronary anomalies are those in which there is an abnormal origin of the coronary arteries that lead to an increased risk for myocardial ischemia. Congenital coronary abnormalities are the second leading cause of sudden cardiac death in young athletes in the United States. Most affected children are asymptomatic and have a normal physical examination, and sudden cardiac death may be their first symptom. Therefore, when patients present with palpitations, exertional chest pain, or syncope, anomalous coronary artery must be considered under the differential diagnosis [1].

5.4 Pulmonary Hypertension

Pulmonary hypertension is a group of conditions with multiple etiologies and it is defined as mean pulmonary arterial (PA) pressure of 25 mm Hg or above in a resting individual at sea level. Regardless of its cause, pulmonary hypertension involves pulmonary arteriolar constriction which results in increased pulmonary vascular resistance. This increases the afterload of the right ventricle resulting in right ventricular hypertrophy and in severe/chronic cases, right ventricular dysfunction. Children with pulmonary hypertension can present with decreased cardiac output when there is volume and pressure overload of the right ventricle that leads to poor cardiac function and decreased coronary perfusion with left ventricular dysfunction. Similar to conditions described previously, patients with pulmonary hypertension will present with fatigue, dyspnea, exertional chest pain, or syncope. On physical

examination, a single S2, loud P2, and diastolic decrescendo murmur (due to pulmonary regurgitation) can be noted. Electrocardiogram, echocardiogram, and chest radiography will confirm the diagnosis if there is suspicion for pulmonary hypertension. Treatment will depend on the underlying cause of pulmonary hypertension [2, 7].

5.5 *Long QT Syndrome*

Long QT syndrome is a genetic disorder caused by mutations in genes that affect development of cardiac ion channels. EKG of a child with long QT syndrome is shown in Fig. 3. This syndrome is characterized by abnormal myocardial repolarization and prolonged QT interval on the electrocardiogram. Prolonged QT interval is defined as a corrected QT interval (QTc) longer than 440 ms in males and 460 ms in females [2, 7]. Symptoms can occur during exercise or be associated with emotional upheaval. It is important to identify activities that the child may have participated in prior to onset of syncope since long QT syndrome is associated with adrenergic arousal, intense emotion, and exercise (specifically swimming). Other associated triggers are auditory, such as alarm clocks, cellphones, or doorbells. Once prolonged QTc is identified, it is recommended that the child be referred to a pediatric cardiologist for further evaluation and management due to the high risk for arrhythmias and sudden cardiac death [6].

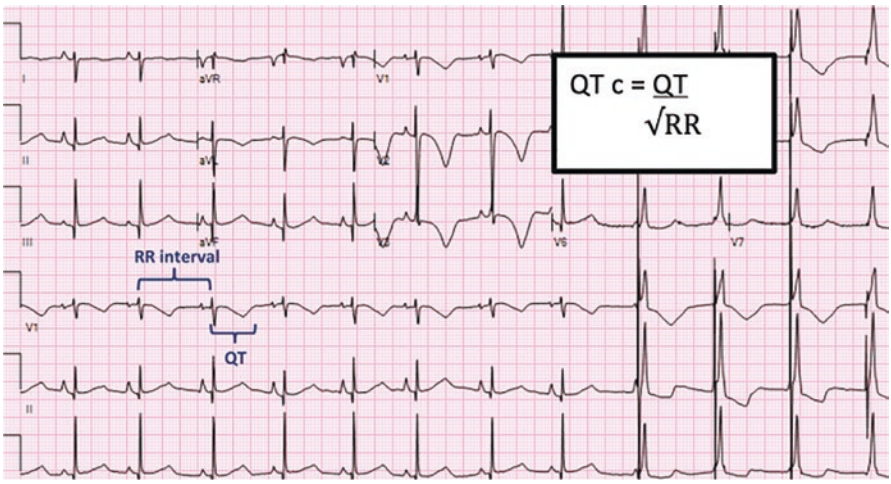

Fig. 3 Electrocardiogram in a patient with prolonged QT

5.6 *Other Dysrhythmias*

Wolff–Parkinson–White (WPW) syndrome is a condition characterized by a short PR interval associated with ventricular pre-excitation and a characteristic delta wave and wide QRS on electrocardiogram. Clinical presentation ranges from asymptomatic to paroxysmal episodes of supraventricular tachycardia with symptoms of palpitations, sudden cardiac arrest and rarely sudden cardiac death [2, 7].

Congenital complete atrioventricular block is a condition that has been strongly associated with exposure to maternal SSA/Ro and/or SSB/La autoantibodies and it is usually seen with structurally normal hearts. Clinical presentation and prognosis vary depending on time of diagnosis. It ranges from asymptomatic bradycardia to syncope, heart failure, or sudden death [6, 7].

EKGs of children with WPW syndrome and third degree atrioventricular blocks are depicted in Figs. 4 and 5, respectively.

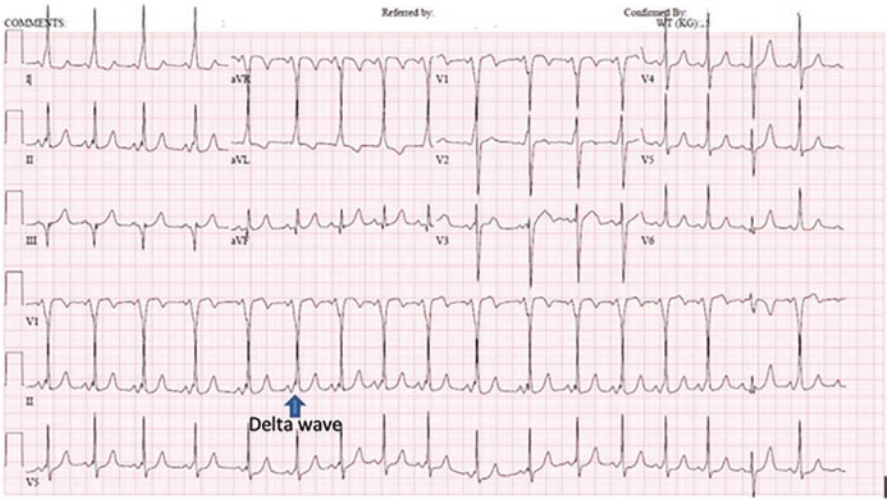

Fig. 4 Wolff–Parkinson–White syndrome or pre-excitation

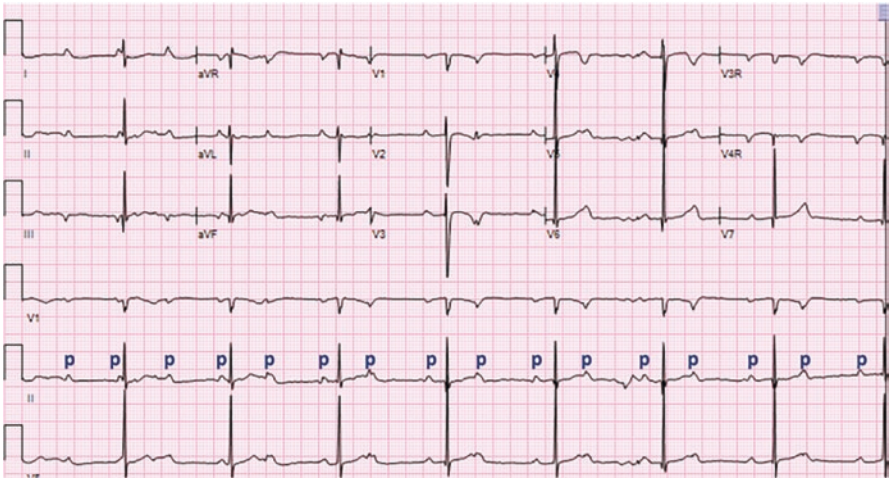

Fig. 5 Third-degree atrioventricular block. P waves marching out independently of QRS complexes

6 Differential Diagnosis

Syncope should be considered as a symptom of an underlying disorder. Although vasovagal syncope is the most common cause of syncope, this must be differentiated from life-threatening cardiac and neurological causes of syncope. Precipitating factors might help to distinguish these conditions. A family history of cardiomyopathy, pacemaker placement or internal cardiac defibrillator dependent conditions at a young age, history of sudden cardiac death, or congenital sensorineural hearing loss should raise suspicion for possible cardiac syncope. Similarly, presence of a pathological heart murmur and/or abnormal vital signs such as tachycardia or bradycardia, hypotension, or hypoxia will indicate a possible cardiac cause of syncope.

Other, not uncommon causes of syncope seen in infants are breath-holding spells which are a type of reflex or situational syncope. The key to the diagnosis of breath holding spells is the presence of a trigger: pain, fear, or any other emotional insult. The children can appear cyanotic or pale before losing consciousness. Breath-holding spells are generally benign and represent a variant of neurocardiogenic syncope. No treatment is required other than behavioral management [3].

Psychogenic or psychiatric causes of syncope should also be considered in older children. For patients presenting with unusual loss of consciousness, with multiple episodes per day, not associated or triggered by change in position, and usually lasting longer than 3–4 min, a diagnosis of conversion disorder should be considered. Likewise, a thorough clinical history including psychosocial triggers such as stressors or anxiety will aid in syncope evaluation [6].

Seizures may occasionally be confused with neurally mediated syncope. In atonic seizures, there may be sudden loss of tone when the child is standing causing the episode to resemble a syncopal spell. However, there are no prodromal symptoms or orthostatic symptoms such as pallor, diaphoresis or light-headedness before the onset of the event. Furthermore, atonic seizures may occur even when a child is sitting down unlike orthostatic symptoms that are typically brought on by change in posture. A child who is experiencing a convulsive seizure (also called a generalized tonic–clonic seizure) may fall before the actual seizure starts. Witnesses may note the typical sequence of events such as stiffening of limbs, clenching of teeth, lateral/upward deviation of eyes, followed by rhythmic jerking of limbs, and finally a period of altered consciousness when a child has a convulsive seizure.

7 Diagnostic Approach

7.1 History

A detailed history is the most important aspect to identify the cause of syncope and distinguish it from other conditions. The prodrome or premonitory factors such as warm or clammy sensation, nausea, light-headedness, or visual changes will suggest vasovagal syncope. Absence of these symptoms should raise suspicion for cardiac causes of syncope [16]. Circumstances surrounding the event such as prolonged standing, change in position, hot weather, poor hydration or hunger is usually seen in vasovagal syncope. If the child presents with syncope mid-exercise, that should raise concern for cardiac syncope [17]. In contrast, postexertional syncope, as is commonly seen in adolescents walking off the field after the game, often indicates vasovagal syncope. This phenomenon is secondary to peripheral vasodilation and hypotension [16]. Abrupt onset of syncope triggered by a loud noise or emotions like frightening or other startle events without any other prodromal symptoms should raise concern for ventricular arrhythmias secondary to long QT syndrome and requires urgent evaluation [18].

It is essential to ask questions about precipitating factors. Factors such as the sight of blood, hair grooming, micturition/defecation, injuries causing pain as well as hyperventilation secondary to emotional stress are usually seen with vasovagal syncope. Chest pain and palpitations during exercise or immediately before syncope should raise concern for cardiac syncope [19].

Similarly, a detailed family history is absolutely essential in differentiating cardiac from non-cardiac causes of syncope. Family history of sudden unexplained death at a young age, congenital deafness, cardiomyopathy, arrhythmia, placement of a pacemaker or defibrillator, or death from an unknown cause should raise suspicion for a cardiac cause for syncope. Family history of seizures, migraine, sleep or vestibular disorders as well as mental disorders in the family will help in identifying other possible causes of syncope.

Other diagnostic hallmarks of vasovagal syncope are the duration and recovery after the event. In general, the loss of consciousness is brief, lasting for few seconds to couple minutes with rapid and spontaneous recovery and no neurologic sequelae. Patients might have brief tonic–clonic seizure like activity during the episode and on rare occasions syncope is accompanied by urinary incontinence. Prolonged episodes, lasting over 5 min, may indicate neurologic or psychiatric causes of syncope (seizures, stroke, and somatization disorder). Another way to differentiate a seizure from vasovagal syncope is the postictal state. In seizures, the state of confusion can last from minutes to hours whereas after vasovagal syncope, the patient can be tired but there is no confusion [20].

Questions pertinent to seizures include inquiry regarding an aura. The aura of a seizure may take the form of an unpleasant smell/taste or a déjà vu phenomenon. An older child may be able to relay these symptoms but sometimes a directed history is important. Most children who experience a seizure, no matter their age, will have their eyes open unlike in syncope [21].

Uprolling of eyes can be present in both seizures and syncope and does not constitute a salient differentiating feature. Enuresis can occur both during syncope and seizure as well, as mentioned above. However, most children are confused or sleepy after a seizure for a variable length of time, whereas there is rapid return to baseline following syncope.

7.2 Physical Examination

In general, physical examination in patients with neurally mediated syncope will be normal. However, the presence of abnormal findings on physical examination will help tailor further evaluation.

7.3 General Examination

A careful evaluation for heart murmurs, distinguishing innocent murmurs from pathologic murmurs can raise or quell concerns for anatomical cardiac conditions like aortic stenosis and hypertrophic obstructive cardiomyopathy [22]. Assessment of vital signs with orthostatic changes are helpful in assessing hydration status and demonstrating dysautonomic responses to orthostatic stress. However, the absence of positive orthostatic vital signs does not rule out vasovagal syncope. Similarly, about 40% of euvolemic teenagers have positive orthostatic signs [23]. Therefore, vital signs should not be used in isolation to direct the evaluation of syncope.

7.4 Neurological Examination

Focal neurologic symptoms or persistent neurologic deficits: motor weakness, ataxia, altered mental status, or slurred speech should raise concern for neurologic conditions such as seizures, complex migraine or stroke [24].

Most children will present to the clinician's office several hours or days after a seizure and therefore will have a normal neurological examination. In the immediate aftermath of a seizure a child may be noted to be drowsy, confused, combative or irritable. In rare instances, Todd's paralysis (motor weakness of a limb or one-half of the body) may be present.

7.5 Evaluation (Laboratory Studies/Imaging)

Diagnostic evaluation is usually guided by history and physical examination. However, all patients with syncope should have an electrocardiogram (EKG) especially if syncope occurred during exercise. When EKG is integrated with a detailed medical history, family history, and physical examination, it has a sensitivity of up to 96% for ruling out cardiac syncope and is Class I recommendation per the 2017 American College of Cardiology/American Heart Association guidelines for evaluation of patients with syncope [23, 25]. If a patient presents with additional risk factors for cardiac cause of syncope, referral to a pediatric cardiologist should be prompt for comprehensive cardiac evaluation that includes but is not limited to echocardiogram, and 24-h or 30-day ambulatory rhythm monitor (Holter monitor or event monitor).

Routine use of blood tests is not usually recommended and has low diagnostic value in the evaluation of a patient with syncope. If necessary, laboratory evaluation should be directed by the history, physical examination and underlying medical condition [16].

Tilt-table test or head-up tilt table test is a non-invasive test frequently performed in adults but also used in children for diagnosis of dysautonomia. It has been safely used in children greater than 6 years of age but it can be used in younger children. The test is performed by positioning the patient upright at an angle of 60°–80° for 15–60 min on a tilt table. The test is concluded with reproduction of clinical symptoms, accompanied by hypotension and or bradycardia. Tilt table test is not helpful if the diagnosis of vasovagal syncope is clear by history and physical examination as it is fraught with a high rate of false positive test results. Neuroimaging studies and electroencephalography may be indicated if patients have specific neurologic findings, but otherwise they are not routinely recommended [26].

8 Treatment/Management and Prevention

The approach to management of patients with syncope should be guided by the underlying mechanism of syncope. See Fig. 6 for our diagnostic approach and referral patterns. For children with vasovagal syncope, the objective is to prevent the recurrence or reduce the frequency of syncopal episodes, to identify triggers and provide reassurance to the family. Most patients with vasovagal syncope do not require hospitalization and have few recurrences. In general, an increase in fluid intake, of at least 60–80 oz, is recommended and additional salt intake in the diet. Equally important is educating the family and patient about physical maneuvers to counteract the effects of gravity that decrease the pooling of venous blood into the lower limbs and abdomen. These maneuvers include elevation of the legs and lowering the head, leg crossing, and tensing of the abdominal muscles [27]. For prevention of vasovagal syncope, patients should be taught other physical techniques like crossing legs or buttocks-clenching while upright or squatting that have been proven to effective in adults and are safe to practice in children [28]. Parents should also be reassured that vasovagal syncope is not life threatening, provided patients learn how to protect themselves from trauma or injuries caused by sudden falls during syncope.

Pharmacologic therapy is not the first choice of treatment due to potential side effects and controversy regarding benefits of long-term prophylaxis. It should be considered if vasovagal syncope is refractory to at least 6 months of conservative management [14]. Among the pharmacologic agents: beta blockers,

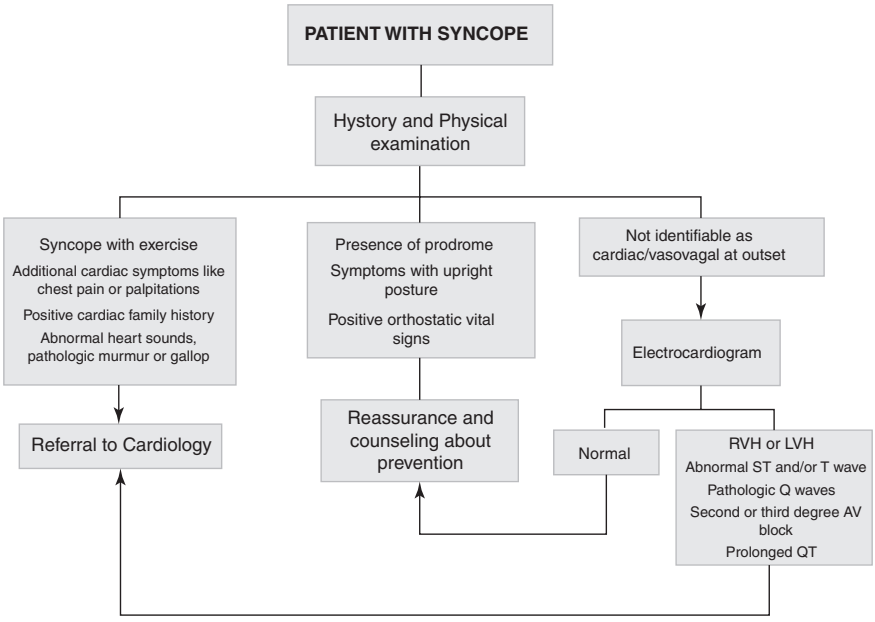

Fig. 6 Diagnostic approach to syncope

mineralocorticoids like fludrocortisone, an alpha-adrenergic receptor agonist like midodrine are the most commonly prescribed medications in children [29].

Beta blockers reduce cardiac inotropy and mechanoreceptor activation, and increase the peripheral arterial resistance. It might not be beneficial in patients that require sympathetic compensatory mechanisms such in postural tachycardia syndrome, highly competitive athletes and patients with asthma. Physicians should inform patients and their families about the side effects of beta-blockers that include headache, hypotension, irritability, depression, and suicide attempts. Only atenolol has been effective in the treatment of vasovagal syncope in children [30]. Fludrocortisone is a corticosteroid with mineralocorticoid activity and acts by enhancing central blood volume by increasing sodium and fluid retention. The dose is usually 0.1 mg once daily or twice daily, with maximum dose of 1 mg/day. At lower doses it is well tolerated with adverse effects usually seen with higher doses which include hypertension, hypokalemia, edema, depression, migraine, or acne. The benefit of fludrocortisone is weak as shown in a small randomized clinical trial, where symptoms and recurrence of syncope were better with placebo than with fludrocortisone [31]. Midodrine is a selective α-1 adrenergic agonist with peripheral effects only that increases peripheral vascular resistance and diminishes the venous pooling. It can be used alone or in combination with fludrocortisone. The dose of midodrine ranges from 2.5–10 mg three times per day with maximum dose of 40 mg/day. It needs to be administered every 6–8 h due to its short duration of action. Among the side effects, hypertension, headache, and edema are common. Low-dose midodrine is considered as first line therapy for vasovagal syncope and small randomized study has shown that midodrine prevents recurrence of vasovagal syncope when compared with placebo [14, 32].

9 Prognosis/Outcomes

Children with vasovagal syncope have an excellent prognosis. The majority of children will have resolution of symptoms within the first year of onset of syncope and 5–10% would continue to present with symptoms up to 5 years from diagnosis [33]. For patients with cardiac syncope, prognosis will depend on prompt recognition and treatment of the underlying cause. Although these patients are at increased risk for sudden death, the majority of children with cardiac syncope can be treated effectively following timely referral to a cardiology specialist.

10 When to Refer/Admit

Whereas most syncopal episodes of childhood are of benign etiology, there is small group of patients with syncope of cardiac origin who are at risk for life-threatening events. It is important to recognize the unusual characteristics that these patients

demonstrate in order to initiate a timely referral to a pediatric cardiologist. These include exertional syncope, absence of a prodrome/premonitory symptoms, syncope preceded by chest pain or palpitations, an event requiring cardiopulmonary resuscitation, neurologic sequelae, prior cardiac history, positive family history of cardiac disease or sudden cardiac death, an abnormal cardiac physical examination, and an abnormal EKG [33].

11 Clinical Pearls/Key Points

- Syncope is common in childhood, especially in adolescent girls.
- Vasovagal syncope is the most common cause of syncope in adolescents.
- Cardiac syncope is rare and can be differentiated from vasovagal syncope based on key elements on history and physical examination.
- Syncope with physical exercise is cardiac syncope until proven otherwise and should initiate a prompt referral to the pediatric cardiologist.

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