

CHAPTER 140

Syncope

ETIOLOGY

Syncope is the transient loss of consciousness and muscle tone that, by history, does not suggest other altered states of consciousness. Presyncope or near-syncope has many or all of the prodromal symptoms without loss of consciousness. Syncope is relatively common (Table 140.1) and is most commonly due to autonomic dysfunction. The frequency of episodes, amount of stress, and functional impairment caused by syncope vary. Most syncopal events are relatively benign but can represent a serious cardiac condition that may lead to death (see Table 140.1). The differential diagnosis for typical syncope includes seizure, metabolic cause (hypoglycemia), hyperventilation, atypical migraine, and breath holding.

CLINICAL MANIFESTATIONS

Typical syncopal events usually occur in the upright position or are related to changing position. Syncope may be associated with anxiety, pain, blood drawing or the sight of blood, fasting, a hot environment, or crowded places. The patient often appears pale. A prodrome, consisting of dizziness, lightheadedness, nausea, diaphoresis, visual changes (blacking out), or possibly palpitations, warns the patient and often prevents injury. Unconsciousness lasts for less than 1 minute. A return to normal consciousness occurs relatively quickly. Most of these syncopal episodes are vasovagal or neurocardiogenic in origin. The physical examination is normal.

DIAGNOSTIC STUDIES

Syncopal episodes may require no more than reassurance to the patient and family. If the episodes have a significant impact on daily activities, further evaluation may be indicated. An electrocardiogram (ECG) should be obtained with attention to the QTc and PR intervals (see Chapter 139). Although reassurance and increasing fluid and salt intake may be adequate to treat most cases of syncope, medical management is sometimes indicated.

TABLE 140.1 Syncope and Dizziness: Etiology

DIAGNOSIS	HISTORY	SIGNS/ SYMPTOMS	DESCRIPTION	HEART RATE/BLOOD PRESSURE	DURATION	POSTSYNCOPE	RECURRENCE
Neurocardiogenic (vasodepressor)	At rest	Pallor, nausea, visual changes	Brief $\pm$ convulsion	$\downarrow/\downarrow$	<1 min	Residual pallor, sweaty, hot; recurs if child stands	Common
OTHER VAGAL							
Micturition	Postvoiding	Pallor, nausea	Brief; convulsions rare	$\downarrow/\downarrow$	<1 min		(+)
Hair grooming	Standing getting hair done or ears touched	Pallor, nausea, visual changes, lightheaded	Brief		<1 min	Residual pallor, sweaty, hot, can recur if stands too soon	(+)
Cough (deglutition)	Paroxysmal cough	Cough	Abrupt onset	May not change	<5 min	Fatigue or baseline	(+)
Carotid sinus	Tight collar, turned head	Vague, visual changes	Sudden onset, pallor	Usually $\downarrow/\downarrow$	<5 min	Fatigue or baseline	(+)
CARDIAC SYNCOPE							
LVOT obstruction	Exercise	$\pm$ Chest pain, SOB	Abrupt during or after exertion, pallor	$\uparrow/\downarrow$	Any duration	Fatigue, residual pallor, and sweating	(+)
Pulmonary hypertension	Any time, especially exercise	SOB	Cyanosis and pallor	$\uparrow/\downarrow$	Any duration	Fatigue, residual cyanosis	(+)
Myocarditis	Post-viral infection exercise	SOB, chest pain, palpitations	Pallor	$\uparrow/\downarrow$	Any duration	Fatigue	(+)
Tumor or mass	Recumbent, paroxysmal	SOB $\pm$ chest pain	Pallor	$\uparrow/\downarrow$	Any duration	Baseline	(+)
Coronary artery disease	Exercise	SOB $\pm$ chest pain	Pallor	$\uparrow/\downarrow$	Any duration	Fatigue, chest pain	(+)
Dysrhythmia	Any time	Palpitations $\pm$ chest pain	Pallor	$\uparrow$ or $\uparrow/\downarrow$	Usually <10 min	Fatigue or baseline	(+)

LVOT, Left ventricular outflow tract; SOB, shortness of breath; $\pm$, with or without; (+), yes but not consistent or predictable.

From Lewis DA. Syncope and dizziness. In: Kliegman RM, ed. Practical Strategies in Pediatric Diagnosis and Therapy. Philadelphia: WB Saunders; 1996.

PEARLS FOR PRACTITIONERS

See *The Cardiovascular System: Pearls for Practitioners at the end of this section.*

CHAPTER 141

Chest Pain

ETIOLOGY

Chest pain in the pediatric patient often generates a significant amount of patient and parental concern. Although chest pain is rarely cardiac in origin in children, common knowledge about atherosclerotic heart disease raises concerns about a child experiencing chest pain. Most diagnosable chest pain in

childhood is musculoskeletal in origin. However, a significant amount remains idiopathic. Knowledge of the complete differential diagnosis is necessary to make an accurate assessment (Table 141.1).

CLINICAL MANIFESTATIONS

Assessment of a patient with chest pain includes a thorough history to determine activity at the onset; the location, radiation, quality, and duration of the pain; what makes the pain better and worse during the time that it is present; and any associated symptoms. A family history and assessment of how much anxiety the symptom is causing are important and often revealing. A careful general physical examination should focus on the chest wall, heart, lungs, and abdomen. A history of chest pain associated with exertion, syncope, or palpitations or acute onset associated with fever suggests a cardiac etiology.

TABLE 141.1 Differential Diagnosis of Pediatric Chest Pain

COMMON

Musculoskeletal
 Costochondritis
 Trauma or muscle overuse/strain
 Pulmonary
 Asthma (often exercise induced)
 Severe cough
 Pneumonia
 Gastrointestinal
 Reflux esophagitis
 Psychogenic
 Anxiety, hyperventilation
 Miscellaneous
 Precordial catch syndrome (Texidor twinge)
 Sick cell vasoocclusive crisis
 Idiopathic

UNCOMMON/RARE

Cardiac
 Ischemia (coronary artery abnormalities, severe AS or PS, HOCM, cocaine)
 Infection/inflammation (myocarditis, pericarditis, Kawasaki disease)
 Dysrhythmia
 Musculoskeletal
 Abnormalities of rib cage/thoracic spine
 Tietze syndrome
 Slipping rib
 Tumor
 Pulmonary
 Pleurisy
 Pneumothorax, pneumomediastinum
 Pleural effusion
 Pulmonary embolism
 Gastrointestinal
 Esophageal foreign body
 Esophageal spasm
 Psychogenic
 Conversion symptoms
 Somatization disorders
 Depression

AS, Aortic stenosis; HOCM, hypertrophic obstructive cardiomyopathy; PS, pulmonary stenosis.

Cardiac causes of chest pain are generally ischemic, inflammatory, or arrhythmic in origin.

DIAGNOSTIC STUDIES

Tests rarely are indicated based on the history. A chest x-ray, electrocardiogram (ECG), 24-hour Holter monitoring, echocardiogram, and exercise stress testing may be obtained based on history and examination. Referral to a pediatric cardiologist is based on the history, physical examination findings, family history, and, frequently, the level of anxiety in the patient or family members regarding the pain.

PEARLS FOR PRACTITIONERS

See *The Cardiovascular System: Pearls for Practitioners* at the end of this section.

CHAPTER 142

Dysrhythmias

ETIOLOGY AND DIFFERENTIAL DIAGNOSIS

Cardiac dysrhythmias or abnormal heart rhythms are uncommon in pediatrics but may be caused by infection and inflammation, structural lesions, metabolic abnormalities, and intrinsic conduction abnormalities (Table 142.1). Many pediatric dysrhythmias are normal variants that do not require treatment or even further evaluation.

Sinus rhythm originates in the sinus node and has a normal axis P wave (upright in leads I and aVF) preceding each QRS complex. Because normal rates vary with age, sinus bradycardia and sinus tachycardia are defined based on age. **Sinus arrhythmia** is a common finding in children and represents a normal variation in the heart rate associated with breathing. The heart rate increases with inspiration and decreases with expiration, producing a recurring pattern on the electrocardiogram (ECG) tracing. Sinus arrhythmia is normal and does not require further evaluation or treatment.

TABLE 142.1 Etiology of Dysrhythmias

DRUGS

Intoxication (cocaine, tricyclic antidepressants, and others)
 Antiarrhythmic agents (proarrhythmic agents [quinidine])
 Sympathomimetic agents (caffeine, theophylline, ephedrine, and others)
 Digoxin

INFECTION AND POSTINFECTION

Myocarditis
 Lyme disease
 Endocarditis
 Diphtheria
 Guillain-Barré syndrome
 Rheumatic fever

METABOLIC-ENDOCRINE

Electrolyte disturbances ($\downarrow K^+$, $\downarrow Ca^{2+}$, $\downarrow Mg^{2+}$)
 Cardiomyopathy
 Thyrotoxicosis
 Uremia
 Pheochromocytoma
 Porphyria
 Mitochondrial myopathies

STRUCTURAL LESIONS

Congenital heart defects
 Ventricular tumor
 Ventriculotomy
 Arrhythmogenic right ventricle (dysplasia)

OTHER CAUSES

Adrenergic-induced
 Prolonged QT interval
 Maternal systemic lupus erythematosus
 Idiopathic
 Central venous catheter

Atrial Dysrhythmias

A **wandering atrial pacemaker** is a change in the morphology of the P waves with variable PR interval and normal QRS complex. This is a benign finding, requiring no further evaluation or treatment.

Premature atrial contractions are relatively common prenatally and in infants. A premature P wave, usually with an abnormal axis consistent with its ectopic origin, is present. The premature atrial activity may be blocked (no QRS following it), conducted normally (normal QRS present), or conducted aberrantly (a widened, altered QRS complex). Premature atrial contractions are usually benign and, if present around the time of delivery, usually disappear during the first few weeks of life.

Atrial flutter and **atrial fibrillation** are uncommon dysrhythmias in pediatrics and usually present after surgical repair of complex congenital heart disease. They may also be seen in patients with myocarditis or in association with drug toxicity.

Supraventricular tachycardia (SVT) is the most common symptomatic dysrhythmia in pediatric patients. The rhythm has a rapid, regular rate with a narrow QRS complex. SVT in infants is often 280–300 beats per minute with slower rates for older children and adolescents. The tachycardia has an abrupt onset and termination. In a child with a structurally normal heart, most episodes are relatively asymptomatic other than a pounding heartbeat. If there is structural heart disease or

the episode is prolonged (>12 hours), there may be an alteration in the cardiac output and development of symptoms of heart failure. Although most patients with SVT have structurally normal hearts and normal baseline ECGs, some children have Wolff-Parkinson-White syndrome or preexcitation as the cause of the dysrhythmia.

Ventricular Dysrhythmias

Premature ventricular contractions (PVCs) are less common than premature atrial contractions in infancy but more common in older children and adolescents (Table 142.2). The premature beat is not preceded by a P wave, and the QRS complex is wide and bizarre. If the heart is structurally normal, and the PVCs are singleton, uniform in focus, and disappear with increased heart rate, they are usually benign and require no treatment. Any deviation from the presentation (history of syncope or a family history of sudden death) requires further investigation and possibly treatment with antiarrhythmic medications.

Ventricular tachycardia, defined as three or more consecutive PVCs, is also relatively rare in pediatric patients. Although there are multiple causes of ventricular tachycardia, it usually is a sign of serious cardiac dysfunction or disease. Rapid-rate ventricular tachycardia results in decreased cardiac output and cardiovascular instability. Treatment in

TABLE 142.2 Dysrhythmias in Children

TYPE	ELECTROCARDIOGRAM CHARACTERISTICS	TREATMENT
Supraventricular tachycardia	Rate usually >220 beats/min (range, 180–320 beats/min); abnormal atrial rate for age; P waves may be present and are related to QRS complex; normal, narrow QRS complexes unless aberrant conduction is present	Increase vagal tone (bag of ice water to face, Valsalva maneuver), adenosine, digoxin, sotalol, electrical cardioversion if acutely ill, catheter ablation
Atrial flutter	Atrial rate usually 300 beats/min, with varying degrees of block; sawtooth flutter waves	Digoxin, sotalol, cardioversion
Premature ventricular contraction	Premature, wide, unusually shaped QRS complex, with large inverted T wave	None if normal heart and if premature ventricular contractions disappear on exercise; lidocaine, procainamide
Ventricular tachycardia	>3 premature ventricular beats; AV dissociation; fusion beats, blocked retrograde AV conduction; sustained if >30 sec; rate 120–240 beats/min	Lidocaine, amiodarone, procainamide, propranolol, cardioversion
Ventricular fibrillation	No distinct QRS complex or T waves; irregular undulations with varied amplitude and contour, no conducted pulse	Nonsynchronized cardioversion
Complete heart block	Atria and ventricles have independent pacemakers; AV dissociation; escape-pacemaker is at atrioventricular junction if congenital	Awake rate <55 beats/min in neonate or <40 beats/min in adolescent, or hemodynamic instability requires permanent pacemaker
First-degree heart block	Prolonged PR interval for age	Observe, obtain digoxin level if on therapy
Mobitz type I (Wenckebach) second-degree heart block	Progressive lengthening of PR interval until P wave is not followed by conducted QRS complex	Observe, correct underlying electrolyte or other abnormalities
Mobitz type II second-degree heart block	Sudden nonconduction of P wave with loss of QRS complex without progressive PR interval lengthening	Consider pacemaker
Sinus tachycardia	Rate <240 beats/min	Treat cause (fever), remove sympathomimetic drugs

AV, Atrioventricular.

TABLE 142.3 Classification of Drugs for Antiarrhythmia

CLASS	ACTION	EXAMPLE(S)
I	Depression of phase of depolarization (velocity of upstroke of action potential); sodium channel blockade	
Ia	Prolongation of QRS complex and QT interval	Quinidine, procainamide, disopyramide
Ib	Significant effect on abnormal conduction	Lidocaine, mexiletine, phenytoin, tocainide
Ic	Prolongation of QRS complex and PR interval	Flecainide, propafenone, moricizine?
II	β blockade; slowing of sinus rate; prolongation of PR interval	Propranolol, atenolol, acebutolol
III	Prolongation of action potential; prolongation of PR, QT intervals, QRS complex; sodium and calcium channel blockade	Bretylium, amiodarone, sotalol
IV	Calcium channel blockade; reduction in sinus and AV node pacemaker activity and conduction; prolongation of PR interval	Verapamil and other calcium channel blocking agents

AV, Atrioventricular.

symptomatic patients is synchronized cardioversion. Medical management with lidocaine or amiodarone may be appropriate in a conscious asymptomatic patient. Complete evaluation of the etiologic picture is necessary, including electrophysiologic study.

Heart Block

First-degree heart block is the presence of a prolonged PR interval. It is asymptomatic and, when present in otherwise normal children, requires no evaluation or treatment. **Second-degree heart block** is when some, but not all, of the P waves are followed by a QRS complex. Mobitz type I (also known as Wenckebach) is characterized by a progressive prolongation of the PR interval until a QRS complex is dropped. It is often seen during sleep, usually does not progress to other forms of heart block, and does not require further evaluation or treatment in otherwise normal children. Mobitz type II is present when the PR interval does not change, but a QRS is intermittently dropped. This form may progress to complete heart block and may require pacemaker placement. **Third-degree heart block**, whether congenital or acquired, is present when there is no relationship between atrial and ventricular activity. The ventricular rate is much slower than the atrial rate. **Congenital complete heart block** is associated with maternal collagen vascular disease (such as systemic lupus erythematosus or Sjögren syndrome) or congenital heart disease. The acquired form most often occurs after cardiac surgery but may be secondary to infection, inflammation, or drugs.

TREATMENT

Most atrial dysrhythmias require no intervention. Treatment of SVT depends on presentation and symptoms. Acute treatment of SVT in infants usually consists of **vagal maneuvers**, such as the application of cold (ice bag) to the face. Intravenous (IV) **adenosine** usually converts the dysrhythmia because the atrioventricular node forms a part of the re-entry circuit in most patients with SVT. In patients with cardiovascular compromise at the time of presentation, **synchronized cardioversion** is indicated using 1–2 J/kg. In patients with palpitations, it is important to document heart rate and rhythm during their symptoms before considering therapeutic options. The frequency, length, and associated symptoms during the episodes,

as well as what is required to convert the rhythm, determine the need for ongoing treatment. Some patients require only education regarding the dysrhythmia and follow-up. Ongoing pharmacologic management with a β blocker is usually the first choice when treatment is needed. Some practitioners still use digoxin for management of SVT, but it is contraindicated in patients with Wolff-Parkinson-White syndrome. Additional antiarrhythmic medications are sometimes needed depending on the mechanism of the SVT. In patients who are symptomatic or for those not wanting to take daily medications, **radiofrequency ablation** may be performed.

A variety of antiarrhythmic agents are used to treat ventricular dysrhythmias that require intervention (Table 142.3). Management of third-degree heart block depends on the ventricular rate and presence of symptoms. Treatment, if needed, often requires placement of a pacemaker.

PEARLS FOR PRACTITIONERS

See *The Cardiovascular System: Pearls for Practitioners* at the end of this section.