Nontraumatic chest pain in children and adolescents: Approach and initial management

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INTRODUCTION

Nontraumatic chest pain is a common symptom in children and adolescents and is a frequent complaint in patients seeking primary, emergency, or subspecialty care [1-4]. Although the etiology is benign in most cases, this symptom may lead to school absences, restriction of activities and causes considerable anxiety in patients and their families or caregivers. A thorough history and physical examination usually can determine the cause and identify patients who require acute intervention and those who can be managed with reassurance and continued follow-up. Laboratory testing is necessary only in a small number of patients [5]. In the absence of associated symptoms of illness, positive findings on physical examination related to the cardiac or respiratory systems, or symptoms during exertion, a serious organic cause is unlikely.

This topic will discuss the approach to nontraumatic chest pain in children and adolescents. The causes of nontraumatic chest pain in children and adolescents, pediatric thoracic trauma, and the emergent evaluation of respiratory distress in children and adolescents, with or without chest pain, is discussed separately:

- (See "Causes of nontraumatic chest pain in children and adolescents".)
- (See "Overview of intrathoracic injuries in children".)
- (See "Thoracic trauma in children: Initial stabilization and evaluation".)
- (See "Chest wall injuries after blunt trauma in children".)
- (See "Pulmonary contusion in children".)
- (See "Acute respiratory distress in children: Emergency evaluation and initial stabilization".)

EVALUATION

The clinician should perform a careful history and physical examination in all children with nontraumatic chest pain with the goal of identifying those patients who warrant further diagnostic studies or referral.

History — Most children with nontraumatic chest pain appear well during acute evaluation. Thus, the history is of utmost importance for identifying serious underlying causes of pediatric chest pain.

Findings of life-threatening causes — The following symptoms are associated with potentially life-threatening cardiac or pulmonary causes of pediatric chest pain (table 1) [3] (see "Causes of nontraumatic chest pain in children and adolescents", section on 'Life-threatening conditions'):

- Pain description as follows:
  - Classic anginal pain (ie, substernal, crushing, with radiation down the left arm or up into the jaw and associated with vomiting, diaphoresis, altered mental status, or dyspnea)
  - Severe, tearing type pain often radiating to the back (aortic dissection)
  - Pleuritic pain with difficulty breathing (pneumonia, pneumothorax, pulmonary embolus, or in patients with sickle cell disease, acute chest syndrome)
  - Exertional pain or exercise intolerance due to tachypnea or fatigue caused by limitations in myocardial oxygen delivery (myocardial ischemia, coronary anomalies, cardiomyopathy)
  - Sharp, retrosternal pain exacerbated by lying down, sometimes radiating to the left shoulder, and often associated with fever (pericarditis)
  - Pain precipitated by the use of cocaine, amphetamines, bath salts, synthetic cannabinoids, marijuana or other vasoactive drugs (eg, cough and cold preparations) suggesting variant angina
- Symptoms occurring with pain as follows:
  - Exertional syncope or dizziness (coronary anomalies, left ventricular outflow obstruction, cardiomyopathy)
  - Palpitations

- Difficulty breathing

- Fever and heart failure symptoms (eg, dyspnea on exertion, orthopnea, or in infants, tachypnea with feedings) suggestive of myocarditis

- Concerning past medical history such as:

  - Congenital or acquired heart disease, Kawasaki disease, or sickle cell anemia

  - Receipt of an mRNA coronavirus disease 2019 (COVID-19) vaccine within the preceding 30 days (see "COVID-19: Vaccines", section on 'Myocarditis')

  - Conditions that predispose to pericarditis, for example, rheumatologic disease, malignancy, recent cardiac surgery, mediastinal radiation, renal failure or infections such as tuberculosis, human immunodeficiency virus

  - Risk factors for pulmonary embolism including immobility, oral contraceptive use, pregnancy termination, ventriculoatrial shunts for hydrocephalus, in-dwelling central venous catheters, solid tumors, heart disease, hypercoagulable states, and obesity

  - Previous aortic dissection, particularly in a patient known to have Marfan, Loeys-Dietz, type IV Ehlers-Danlos, or Turner syndrome

- Family history of:

  - Hypertrophic cardiomyopathy or of sudden death in first-degree relatives (eg, parents or siblings) younger than 50 years of age

  - Marfan, Loeys-Dietz, type IV Ehlers-Danlos, or Turner syndromes because of predisposition for aortic root dissection

  - Inherited hypercoagulable state (factor V Leyden, protein C or protein S deficiency, and other conditions)

Other important features — When history does **not** suggest a life-threatening cause, an accurate description of the chest pain can help establish a specific diagnosis as follows (table 2) [3,6-8]:

- **Temporal elements** – The duration and time course of the onset of chest pain may be a useful distinguishing feature. Chronic pain is unlikely to have a serious underlying cause and often is musculoskeletal, psychogenic, or idiopathic.

  Acute pain is more likely to be caused by a medical condition. As examples, pain caused by pulmonary conditions (eg, asthma or pneumothorax) or vascular events (eg, aortic

dissection or acute pulmonary embolism) typically has an abrupt onset. However, ischemic myocardial pain, which is unusual in children and is described in greater detail above, may have a gradual onset with increasing intensity over time.

- **Quality** – A description of the quality of the pain may be helpful. The pain associated with costochondritis typically is described as midsternal in location and sharp in quality with minimal radiation. It occurs mainly at rest and lasts for seconds to a few minutes. The pain may increase in intensity with deep inspiration because of stretching of the costochondral junctions or muscle fibers.

  Patients with slipping rib syndrome may describe "something slipping or giving away," "a popping sensation," or "hearing a clicking sound" [9]. In that condition, pain frequently occurs with bending over or deep breathing [10].

- **Location** – Pain that localizes to a small area on the chest more likely is of chest wall or pleural origin rather than visceral. Ischemic pain is a diffuse discomfort that may be difficult to localize.

  Radiation usually is associated with causes of pain that are uncommon in children. As an example, the pain of myocardial ischemia may radiate to the neck, throat, lower jaw, teeth, upper extremity, or shoulder. Other unusual causes include acute cholecystitis, which can present with right shoulder pain (although concomitant right upper quadrant or epigastric pain is more typical), aortic dissection, which may be associated with chest pain that radiates between the scapulae, or pericarditis that can radiate to the left shoulder.

- **Precipitating factors** – The patient should be asked about factors that induce or make the pain worse:

  - Deep breathing may exacerbate chest pain of musculoskeletal origin.

  - Pain made worse by swallowing likely is of esophageal origin. Discomfort that occurs with eating also may suggest upper gastrointestinal disease. History of a choking spell or witnessed foreign body ingestion, especially in a preschool child warrants evaluation for an esophageal foreign body.

  - Chest discomfort provoked by exertion often has a cardiac or respiratory cause.

  - Pleuritic chest pain is worsened by inspiration.

  - Chest pain that increases when lying supine and decreases when sitting suggests pericarditis.

  - Pain associated with coronary artery anomalies typically occurs with exertion.

- **Associated symptoms** – Associated symptoms may help determine etiology.
  - Fever, especially when associated with tachypnea or cough, may suggest a respiratory infection. Fever is also present in patients with pericarditis, myocarditis, Kawasaki disease, or multisystem inflammatory syndrome in children (MIS-C). (See "Clinical manifestations and diagnosis of myocarditis in children" and "Kawasaki disease: Clinical features and diagnosis" and "COVID-19: Multisystem inflammatory syndrome in children (MIS-C) clinical features, evaluation, and diagnosis".)
  - Dyspnea or cough may indicate pulmonary disorders, including pathology of the airways, lung parenchyma, or pulmonary vasculature, or be a sign of cardiac disease. In addition to chest pain, for example, patients with pulmonary embolism can develop dyspnea, hypoxemia, apprehension, cough, and diaphoresis [11]. Patients with myocarditis may have dyspnea and fatigue. (See "Clinical manifestations and diagnosis of myocarditis in children".)
  - Vomiting or regurgitation, painful swallowing, or heartburn associated with eating suggest gastrointestinal disease, such as gastroesophageal reflux and esophagitis.
  - Recurrent somatic complaints, including headache or abdominal or extremity pain, occur in most children with psychogenic chest pain [12,13]. Approximately one-third have significant sleep disturbances [12].
  - Lightheadedness or paresthesias frequently accompany chest pain resulting from hyperventilation.
  - Exertional syncope or palpitations suggest an underlying cardiac disorder. (See "Emergency evaluation of syncope in children and adolescents".)

Physical examination — Children and adolescents with chest pain are typically in no acute distress or have minimal discomfort during examination.

Patients with acute onset of chest pain and who exhibit respiratory distress or hemodynamic instability require immediate management according to the principles of Pediatric Advanced Life Support. This should be initiated while the evaluation proceeds. (See 'Unstable' below.)

A thorough physical examination should be performed in all patients. Important vital sign changes include [3,6]:

- Fever (associated with cardiac or pulmonary disease such as pericarditis, myocarditis, pneumonia, or a rheumatologic condition)
- Tachycardia for age (table 3) (present in several common and life-threatening causes of pediatric chest pain and warrants evaluation of the patient's rhythm and cardiac

function)

- Tachypnea for age (table 3) (seen in patients with a variety of conditions but raises concerns for asthma, pneumonia, spontaneous pneumothorax, pulmonary embolism, pulmonary congestion from cardiac causes, and hyperventilation syndrome)
- Hypertension (frequently present in patients with aortic dissection)
- Hypotension (associated with serious cardiac, pulmonary, and infectious causes of chest pain)
- Narrow pulse pressure or pulsus paradoxus >10 mmHg (present in patients with a large pericardial effusion associated with cardiac tamponade)
- Hypoxia (eg, abnormal pulse oximetry; the threshold varies based on several factors, but, for normal individuals at sea level, many experts consider a resting oxygen saturation ≤95 percent as abnormal. Trends in oxygen saturation and the underlying disease process are important for interpretation.) (See "Pulse oximetry", section on 'Optimal oxygen saturation'.)

General appearance may indicate the following [3,6,8]:

- Presence of genetic conditions that are associated with congenital heart disease (eg, Turner syndrome) or aortic dissection (eg, Marfan (table 4) or Ehlers-Danlos syndrome). (See "Ehlers-Danlos syndromes: Clinical manifestations and diagnosis", section on 'Clinical manifestations' and "Clinical manifestations and diagnosis of Turner syndrome", section on 'Clinical manifestations'.)
- Signs of chronic disease such as weight loss, fatigue, or pallor suggesting an underlying condition such as systemic lupus erythematosus, lymphoma, or a solid tumor.
- Tetany or carpopedal spasm indicating hyperventilation syndrome. (See "Hyperventilation syndrome in adults", section on 'Clinical presentation'.)
- Drooling associated with an esophageal foreign body

Next, the clinician should focus on the chest, lungs, and heart as follows [3,6,8]:

- **Chest wall** – Chest wall deformity, such as pectus excavatum or carinatum (although uncommonly causes of chest pain), asymmetry of breathing (pneumothorax), rapid, shallow breathing with normal oxygenation (hyperventilation) or asymmetry of the breasts (gynecomastia) are important findings on inspection.

  Chest wall tenderness indicates a musculoskeletal cause of pain, typically costochondritis. Bruises associated with trauma sometimes are seen. However, serious

underlying thoracic injury can occur without obvious chest wall bruising. (See "Overview of intrathoracic injuries in children" and "Pulmonary contusion in children".)

Examination should also include palpation of the costochondral junctions, the insertion site of the pectoralis major muscle group (by grasping the head of the muscle between the examiner's fingers and thumb), the inframammary area, and other regions of the chest where pain is reported. In patients with costochondritis, involvement of the costochondral junctions often is asymmetric, with the left side more frequently affected. The diagnosis is confirmed when palpation reproduces the discomfort. Provocative maneuvers for costochondritis include horizontal arm traction (figure 1) and the "crowing rooster" maneuver (figure 2). (See "Causes of nontraumatic chest pain in children and adolescents", section on 'Musculoskeletal'.)

Diagnosis of slipping rib syndrome can be confirmed by the "hooking" maneuver in which the examiner's fingers are curled around the lower costal margin. Lifting anteriorly will elicit a click and reproduce pain (figure 3). (See "Causes of nontraumatic chest pain in children and adolescents", section on 'Musculoskeletal'.)

Tenderness on palpation of breast tissue often can be elicited in patients who complain of breast pain. Tender subareolar masses may also be palpable. (See "Breast disorders in children and adolescents", section on 'Breast pain'.)

- **Lungs** – Signs of a respiratory etiology may include tachypnea and respiratory distress. Patients with respiratory distress warrant urgent assessment of airway and breathing and administration of supplemental oxygen. Airway management in patients with signs of impending respiratory failure should be initiated prior to full evaluation. (See "Acute respiratory distress in children: Emergency evaluation and initial stabilization", section on 'Rapid assessment'.)

  In addition to lung pathology, tachypnea with or without wheezing can also be an important sign of heart failure in children with myocarditis, dilated cardiomyopathy, or other cardiac causes of chest pain. (See "Heart failure in children: Etiology, clinical manifestations, and diagnosis", section on 'Clinical manifestations'.)

  Diminished breath sounds over affected areas of the lung raises suspicion for pneumothorax. Rales or tubular breath sounds suggest pneumonia whereas wheezing may be audible in patients with asthma. Pneumomediastinum or esophageal rupture may produce subcutaneous emphysema that is detected as crepitus on palpation of the supraclavicular or neck region. (See "Causes of nontraumatic chest pain in children and adolescents", section on 'Spontaneous pneumothorax' and "Causes of nontraumatic chest pain in children and adolescents", section on 'Respiratory'.)

Hyperventilation, associated with chest pain of psychogenic origin, may be present at the time of the examination. Patients will display light, shallow breathing with normal lung sounds and oxygenation. If the patient is not actively hyperventilating, it may be possible to reproduce the chest pain by asking the patient to hyperventilate. However, hyperventilation for as long as 20 minutes may be required to reproduce symptoms [14].

In patients with atypical angina due to coronary vasospasm, hyperventilation for six minutes can frequently reproduce symptoms [15].

- **Heart** – A cardiac cause of chest pain is identified by abnormal heart sounds on auscultation (eg, cardiac murmur, gallop, muffled heart sounds, or a pericardial friction rub) or by an abnormal pulse or blood pressure (table 1). Any new heart murmur or abnormal heart sound in association with chest pain should prompt additional evaluation and pediatric cardiology consultation. (See 'Ancillary studies' below and 'Indications for pediatric subspecialty consultation or referral' below.)

  Findings associated with specific cardiac causes of chest pain include:

  - **Hypertrophic cardiomyopathy (HCM)** – Patients with HCM may develop several types of systolic murmurs, but the two most common are related to left ventricular outflow tract (LVOT) obstruction and mitral regurgitation as follows (see "Hypertrophic cardiomyopathy: Clinical manifestations, diagnosis, and evaluation", section on 'Systolic murmurs'):

    - Significant LVOT obstruction results in a harsh crescendo-decrescendo systolic murmur that begins slightly after S1 and is heard best at the apex and lower left sternal border. The murmur may radiate to the axilla and base but usually not into the neck. It increases with a Valsalva maneuver.

    - Centrally directed mitral regurgitation, usually associated with primary mitral valve pathology, classically results in a holosystolic murmur heard loudest at the apex which radiates to the axilla.

  - **Dilated cardiomyopathy** – Pediatric patients with dilated cardiomyopathy may display findings of heart failure that vary by age but include tachycardia, poor perfusion, an S3 gallop, tachypnea, wheezing, hepatomegaly, jugular venous distension, dyspnea on exertion, and peripheral edema [16]. (See "Heart failure in children: Etiology, clinical manifestations, and diagnosis", section on 'Physical examination'.)

  - **Coronary ischemia** – Myocardial ischemia may result in tachycardia, increased blood pressure, new murmur consistent with mitral valve regurgitation, paradoxical

splitting of the second heart sound if left bundle branch block is present, or a third or fourth heart sound. Some patients have none of these findings. (See "Approach to the patient with suspected angina pectoris", section on 'Physical examination'.)

- **Left ventricular outflow obstruction** – Signs of left ventricular outflow obstruction include a systolic ejection murmur at the right upper sternal border and occasionally along the left sternal border. Coarctation of the aorta is associated with elevated blood pressure in the arms and a lower blood pressure in the legs, and frequently with a systolic murmur on the back between the scapulae. If the coarctation is long-standing (present for more than five to seven years), collateral vessels may form that connect the upper and lower portions of the aorta; these vessels create a continuous murmur over the lateral aspect of the ribs. (See "Clinical manifestations and diagnosis of coarctation of the aorta".)

- **Arrhythmia** – An irregular heart rate associated with chest pain increases the likelihood of an underlying arrhythmia. Very rapid tachycardia for age (180 to 240 beats per minute in older children and adolescents) suggests supraventricular tachycardia, including Wolff-Parkinson-White (WPW) syndrome. Exposure to a variety of sympathomimetic agents, including cocaine, amphetamines, bath salts, and synthetic cannabinoids may cause sinus tachycardia, hypertension, and anxiety. (See "Irregular heart rhythm (arrhythmias) in children", section on 'Symptomatic child' and "Clinical features and diagnosis of supraventricular tachycardia (SVT) in children", section on 'Clinical features'.)

- **Pericarditis** – In patients with pericarditis, pain increases when manual pressure is applied to the sternal region. The pain typically improves with sitting up and leaning forward. Signs of pericarditis depend upon the size of the pericardial effusion. Patients with a small effusion typically have an audible pericardial friction rub, caused by rubbing together of the inflamed parietal and visceral pericardial surfaces. The rub often is continuous in systole and diastole. It is easier to hear with the diaphragm of the stethoscope when the patient is sitting and leaning forward.

  A rub will not be heard if the effusion is large because the two pericardial surfaces of the pericardium are not in contact with each other. A large effusion may result in cardiac tamponade, manifested by a narrow pulse pressure, elevated pulsus paradoxus (>10 mmHg), elevated jugular venous pressure, distant heart sounds, hepatomegaly, ascites, and peripheral edema.

- **Myocarditis** – Myocarditis may on occasion present with chest pain. Signs of myocarditis include tachycardia, diminished heart sounds, a gallop rhythm, and a murmur of mitral regurgitation, usually accompanied by fever. Patients often complain of dyspnea and fatigue and may display tachypnea on examination.

Tachycardia out of proportion to age and poor perfusion can be findings of myocarditis in children with an otherwise normal examination. (See "Clinical manifestations and diagnosis of myocarditis in children".)

- **Pulmonary hypertension (pulmonary embolism)** – Patients with pulmonary hypertension caused by a pulmonary embolus typically have a right ventricular heave and a single loud S2. They may have a murmur consistent with tricuspid or pulmonary regurgitation.

Additional findings that suggest specific diagnoses include [6,8]:

- Epigastric abdominal tenderness associated with gastritis or, less commonly, pancreatitis
- Rashes (eg, evanescent salmon pink rash (picture 1) or malar rash (picture 2)) characteristic of specific collagen vascular diseases
- Arthritis arising from underlying collagen vascular disease (eg, juvenile rheumatoid arthritis or systemic lupus erythematosus)
- Posterior oral ulcers and rash on the palms and soles in associated with pleurodynia in patients with enterovirus infection (coxsackievirus B)

Ancillary studies — Most children with chest pain have no history suggestive of life-threatening conditions (table 1) and either a normal physical examination or findings consistent with a musculoskeletal etiology. Further investigations are not needed in patients for whom a clear etiology, other than cardiac disease, can be established [3,6,8]. Furthermore, in children with findings that indicate gastroesophageal reflux disease or medication-induced esophagitis, presumptive treatment prior to testing is appropriate [8]. (See "Gastroesophageal reflux disease in children and adolescents: Management", section on 'Lifestyle changes' and "Gastroesophageal reflux disease in children and adolescents: Management", section on 'Pharmacotherapy' and "Pill esophagitis", section on 'Management'.)

Diagnostic studies may help establish a diagnosis in patients with associated symptoms that suggest pulmonary or cardiac conditions or an esophageal foreign body [3,6]. Although cardiac causes of chest pain are uncommon in children, patients with clinical manifestations that are concerning for life-threatening cardiac conditions warrant prompt consultation with a pediatric cardiologist (table 1). (See 'Indications for pediatric subspecialty consultation or referral' below and "Causes of nontraumatic chest pain in children and adolescents", section on 'Cardiac disease'.)

Bedside ultrasonography — When performed by properly trained and experienced clinicians, bedside ultrasound can rapidly confirm the presence of pneumothoraces and pericardial effusions and guide emergent intervention (eg, chest tube thoracostomy or pericardiocentesis (figure 4)) in unstable patients. (See "Emergency pericardiocentesis", section on 'Ultrasound-guided pericardiocentesis technique' and "Bedside pleural ultrasonography: Equipment, technique, and the identification of pleural effusion and pneumothorax".)

Electrocardiogram — An electrocardiogram (ECG) should be obtained if cardiac disease is suspected based upon history or physical examination (table 1) and when a noncardiac etiology (eg, costochondritis, pneumonia, gastroesophageal reflux, pill esophagitis, or esophageal foreign body) **cannot** be established.

In patients with a rapid pulse rate or palpitations, an ECG can identify the type of arrhythmia (algorithm 1). However, if the arrhythmia is intermittent and the initial ECG is normal, referral to a pediatric cardiologist for a Holter monitor or event monitor recording may be needed.

Other ECG abnormalities can help with diagnosis and indicate the need for further testing as follows [3]:

- Patients with hypertrophic cardiomyopathy may have evidence of left ventricular hypertrophy or strain. Formal exercise tolerance testing may be needed to assess the development of arrhythmia, left ventricular outflow tract obstruction, or ischemia during exertion.

- In pericarditis with effusion, the ECG changes during the clinical course. Generalized ST segment elevation involving limb and precordial leads is seen in the initial 10 to 14 days (waveform 1). This is followed by T wave flattening or inversion that can persist for an additional two weeks. Voltages will be reduced in amplitude if the effusion is large. Abnormal Q waves typically are not seen in pericarditis. ST-T wave abnormalities occur in myocarditis. (See "Clinical manifestations and diagnosis of myocarditis in children".)

- In children with anomalous origin of the left coronary artery from the pulmonary artery, the typical ECG pattern is of an anterolateral infarction with deep and wide Q waves and T wave inversions in leads I, aVL, V5, and V6 (waveform 2).

- Patients with pulmonary hypertension typically have signs of right ventricular hypertrophy and right axis deviation. The ECG may show right ventricular strain (abnormal T waves in the anterior leads).

- ECG findings in pulmonary embolism usually are nonspecific ST-T segment changes or sinus tachycardia. If acute right ventricular hypertension occurs, the classic pattern of S

wave in lead 1, Q wave in lead III, and T wave inversion in lead III can be seen [11].

Chest radiograph — A chest radiograph should be obtained in children in whom a cardiac or pulmonary disorder or esophageal foreign body is suspected including those with the following findings [3,6] (see 'Physical examination' above and "Causes of nontraumatic chest pain in children and adolescents", section on 'Gastrointestinal'):

- Respiratory distress
- Pleuritic chest pain
- Pathologic cardiac murmur or heart sounds
- Pulmonary rales
- Hypoxia
- Persistent tachypnea (table 3)
- History of choking or witnessed ingestion of a foreign body

Signs of cardiac enlargement may be apparent in conditions causing left ventricular outflow obstruction, heart failure, myocarditis, pericarditis, or pericardial effusion.

Patients with pulmonary hypertension may have prominent main and central pulmonary arteries. The peripheral lung fields may be dark if pulmonary vascular resistance is chronically elevated.

In patients with suspected pulmonary disorders, a chest radiograph may show infiltrates caused by pneumonia or areas of atelectasis and air trapping caused by aspiration of a foreign body. Hyperinflation typically is seen in patients with asthma. In addition, pneumothorax, pneumomediastinum, or pleural effusions can be detected.

An anteroposterior and lateral chest radiograph can localize and help identify the type of radio-opaque esophageal foreign body (eg, button battery, coin, or magnet). (See "Foreign bodies of the esophagus and gastrointestinal tract in children", section on 'Imaging'.)

Echocardiogram — Based upon observational studies of pediatric patients referred for cardiology evaluation, an echocardiogram is indicated in the patients with the following findings [17,18]:

- History of exertional chest pain or exertional syncope.
- Chest pain associated with fever (>38.5°C).
- Chest pain that radiates to the back, jaw, left arm or left shoulder, or that increases with supine position.
- Past medical history of congenital heart disease, heart transplant, Kawasaki syndrome, or diseases that raise cardiac risk (eg, malignancy, collagen vascular disease,

hypercoagulable state, immobilization).

- Family history of cardiomyopathy, sudden death, or hypercoagulable state in first-degree relatives (eg, parents or siblings) younger than 50 years of age.
- A new murmur, gallop, distant heart sounds, pericardial friction rub, increased intensity of the pulmonary component of S2, or peripheral edema on cardiac examination. (See 'Physical examination' above.)
- An abnormal ECG (eg, right or left ventricular hypertrophy, ST segment change >2 mm, low QRS voltage, PR segment depression, S wave in lead 1, Q wave in lead III, or an inverted T wave in lead III).

The utility of echocardiography for such patients includes the following:

- Establish the diagnosis of pulmonary hypertension and evaluate ventricular function and associated structural abnormalities.
- Determine the severity and site of left ventricular outflow obstruction.
- Determine left ventricular function and assess for the presence of dilated or hypertrophic cardiomyopathy.
- Assess the size of a pericardial effusion and signs of tamponade, which include variation in Doppler peak velocity across the valves during the cardiac cycle, atrial free wall collapse, or ventricular septal paradoxical motion into the left ventricle during inspiration.
- Identify coronary artery abnormalities, including abnormal origin or course, fistula, or aneurysm or stenosis caused by Kawasaki disease.
- Diagnose aortic root dissection (image 1). Alternatively, this diagnosis can be made with magnetic resonance imaging (image 2), computed tomography, or aortography (image 3 and image 4A-B). The most efficient imaging technique available at the institution should be used to establish the diagnosis. (See "Clinical features and diagnosis of acute aortic dissection", section on 'Cardiovascular imaging'.)
- Diagnose ruptured sinus of Valsalva aneurysm.

Other tests — Less commonly, additional testing may be indicated based upon associated signs and symptoms and the results of the initial evaluation as follows:

- **Cardiac troponin** – Cardiac troponin testing may assist with diagnosis when myocardial infarction or ischemia is suspected. Troponin is a highly sensitive biomarker that aids in the detection of myocardial cell damage, which is often but not always, due to

thrombotic obstruction of a coronary artery. Thus, while troponin may be useful to "rule out" a non-ST elevation myocardial infarction, the clinician must recognize the limitations of using troponin to "rule in" MI in patients with low clinical likelihood of an acute coronary syndrome (ACS) which includes most children and adolescents. While troponin is highly specific for myocardial injury, it is not specific for ACS as the cause. As a result, if troponin testing is applied indiscriminately in broad populations with a low pretest probability of thrombotic disease, the positive predictive value for ACS is greatly diminished. Causes of troponin elevations other than acute coronary syndrome are noted in the table (table 5) and discussed in detail separately. In children, myocarditis is frequently the cause of an elevated troponin test. (See "Elevated cardiac troponin concentration in the absence of an acute coronary syndrome".)

- **Ambulatory cardiac rhythm monitoring** – Cardiac rhythm monitoring may be appropriate in some patients with chest pain associated with palpitations, dizziness, or syncope. The type of device for monitoring depends upon several factors and is discussed separately. (See "Ambulatory ECG monitoring".)

  However, these tests should be obtained and interpreted in conjunction with a pediatric cardiologist.

APPROACH AND INITIAL MANAGEMENT

The approach and initial management of children and adolescents with chest pain is determined by their clinical status on presentation and findings on history and physical examination (algorithm 2).

Unstable — Although rare in children and adolescents with a primary complaint of chest pain, patients with marked respiratory distress, hemodynamic instability, or sudden collapse require rapid supportive care of the airway, breathing, and circulation according to the principles of Pediatric Advanced Life Support (PALS) (see "Pediatric advanced life support (PALS)") including timely management of the following conditions when present:

- **Airway foreign body with obstruction** – Emergent securing of the airway and treatment of the underlying cause (algorithm 3 and algorithm 4). (See "Emergency evaluation of acute upper airway obstruction in children", section on 'Determining the cause of upper airway obstruction'.)

- **Tension pneumothorax** – Needle or chest tube thoracostomy followed by management of the underlying cause (eg, pulmonary blebs). (See "Spontaneous pneumothorax in children", section on 'Evacuation of the pleural space'.)

- **Severe status asthmaticus** – Rapid administration of oxygen, inhaled bronchodilators (eg, albuterol), and systemic corticosteroid therapy (algorithm 5). (See "Acute severe asthma exacerbations in children younger than 12 years: Intensive care unit management", section on 'Preintubation therapies' and "Acute severe asthma exacerbations in children and adolescents: Endotracheal intubation and mechanical ventilation", section on 'Endotracheal intubation and mechanical ventilation'.)

- **Pulmonary embolism** - Anticoagulant therapy or, in children with a large embolus or hemodynamic compromise, thrombolytics (eg, tissue plasminogen activator or urokinase), or in patients who fail thrombolytic therapy, embolectomy. (See "Treatment, prognosis, and follow-up of acute pulmonary embolism in adults", section on 'Embolectomy' and "Venous thrombosis and thromboembolism (VTE) in children: Treatment, prevention, and outcome", section on 'Pulmonary embolism'.)

- **Ischemia or infarction** – Patients with persistent chest pain consistent with myocardial infarction should receive rapid treatment including anticoagulation, pain management, nitroglycerin, beta blocker if not contraindicated, and, depending upon degree of risk, catheterization (algorithm 6). (See "Initial evaluation and management of suspected acute coronary syndrome (myocardial infarction, unstable angina) in the emergency department", section on 'Management'.)

- **Tachyarrhythmia** – Tachyarrhythmias should be managed according to Pediatric Advanced Life Support principles (algorithm 1). (See "Pediatric advanced life support (PALS)", section on 'Tachycardia algorithm'.)

- **Heart failure with cardiogenic shock** – Patients with heart failure (eg, dilated cardiomyopathy or myocarditis) warrant therapy as determined by the severity of shock (algorithm 7) and degree of failure (table 6). (See "Heart failure in children: Management", section on 'Approach to HF management'.)

Acute chest pain with concerning findings — Most children with a primary complaint of nontraumatic chest pain will be stable at initial evaluation. Those with concerning features of a potential cardiac etiology on history or physical examination (table 1) warrant an ECG (see 'Electrocardiogram' above and "Causes of nontraumatic chest pain in children and adolescents", section on 'Cardiac disease'). Patients with findings of a serious cardiac etiology on physical examination or ECG should have consultation with a pediatric cardiologist and treatment of the underlying abnormality. Patients with a normal physical examination and ECG but a concerning history deserve follow-up with a pediatric cardiologist within one to two weeks.

A chest radiograph is helpful for patients with respiratory distress, pleuritic chest pain, abnormal murmur or heart sounds, or a suspected foreign body. (See 'Chest radiograph'

above.)

Pleuritic or positional chest pain suggests pneumonia, pulmonary embolism, spontaneous pneumothorax, pericarditis, and, in patients with sickle cell disease, acute chest syndrome (algorithm 2). These patients warrant timely intervention based upon the underlying cause as follows:

- **Pneumonia** – Supplemental oxygen, endotracheal intubation, and/or mechanical ventilation as needed; hemodynamic support as needed; and antibiotics tailored to the type of pneumonia (eg, community-acquired, nosocomial, or aspiration pneumonia). (See "Pneumonia in children: Inpatient treatment", section on 'Empiric therapy'.)

- **Pulmonary embolism** – Diagnostic testing and imaging to establish the diagnosis and extent of embolism followed by anticoagulant therapy or, in children with a large embolus or hemodynamic compromise, thrombolytics (eg, tissue plasminogen activator or urokinase) or, in patients who fail thrombolytic therapy, embolectomy. (See "Treatment, prognosis, and follow-up of acute pulmonary embolism in adults", section on 'Embolectomy' and "Venous thrombosis and thromboembolism (VTE) in children: Treatment, prevention, and outcome", section on 'Pulmonary embolism' and "Venous thrombosis and thromboembolism (VTE) in children: Risk factors, clinical manifestations, and diagnosis", section on 'Diagnosis'.)

- **Spontaneous pneumothorax** – For patients with large pneumothoraces, significant pain, dyspnea, or hypoxia, needle or chest tube thoracostomy followed by management of the underlying cause (eg, pulmonary blebs). (See "Spontaneous pneumothorax in children", section on 'Evacuation of the pleural space'.)

- **Pericarditis** – Urgent echocardiogram to determine the presence and amount of pericardial fluid, pericardiocentesis in patients with tamponade, and treatment of the underlying cause. (See "Acute pericarditis: Treatment and prognosis", section on 'Adjunctive therapies' and "Acute pericarditis: Treatment and prognosis", section on 'Treatment'.)

- **Acute chest syndrome** – Respiratory support to maintain oxygenation, pain control, bronchodilators for wheezing, broad spectrum antibiotics, and blood transfusion, either simple or exchange transfusion depending upon the degree of severity. (See "Acute chest syndrome (ACS) in sickle cell disease (adults and children)", section on 'Management'.)

Other key findings include history or physical appearance suggestive of Marfan, Ehlers Danlos, Loeys-Dietz, or Turner syndrome indicating a predisposition to aortic root dissection; history of a foreign body or drooling on examination suggesting an esophageal foreign body; pain with swallowing, severe retrosternal pain, and hematemesis seen in patients with

esophageal rupture; and fatigue, pallor, weight loss, or abdominal mass in association with a chest wall or intrathoracic neoplasm.

Initial management for these patients consists of the following:

- **Aortic root dissection** – Symptomatic patients warrant emergent evaluation by a pediatric cardiologist and a pediatric cardiothoracic surgeon and typically require emergent surgical intervention. Acute management of these patients is discussed in greater detail separately. (See "Overview of acute aortic dissection and other acute aortic syndromes", section on 'Acute medical management' and "Management of acute type B aortic dissection", section on 'Anti-impulse therapy' and "Management of acute type A aortic dissection", section on 'Acute management'.)

- **Esophageal foreign body** – The management of an esophageal foreign body depends upon the type of object. Symptomatic patients and those with sharp or long foreign bodies, objects in the esophagus for a prolonged period of time, impacted disc batteries, or ingested magnets warrant urgent removal (algorithm 8). (See "Foreign bodies of the esophagus and gastrointestinal tract in children", section on 'Indications for urgent removal'.)

- **Esophageal rupture** – Management of esophageal rupture requires urgent involvement of a surgeon with pediatric expertise. Specific treatment depends upon whether the perforation is free or contained (algorithm 9). (See "Boerhaave syndrome: Effort rupture of the esophagus", section on 'Management'.)

- **Tumor** – Patients with constitutional symptoms such as intermittent fevers, pallor, and weight loss or chest mass on radiography require a work-up designed to assess for the presence of an underlying neoplasm (eg, lymphoma, leukemia, neuroblastoma, or sarcoma). This evaluation should be performed in consultation with a pediatric oncologist. (See "Approach to the adult patient with a mediastinal mass", section on 'Presumptive clinical diagnosis' and "Overview of common presenting signs and symptoms of childhood cancer".)

Common findings — In most pediatric patients, nontraumatic chest pain is not life-threatening. Reassurance is an important component of initial management in these patients. Although the etiology usually is benign, this symptom causes considerable anxiety in patients and their families or caregivers because of the more serious implications of chest pain in adults. Cases with a psychiatric etiology typically need additional counseling and in selected patients, referral to a child psychiatrist.

Important findings of common causes and initial management by etiology include (table 2):

- **Chest wall pain** – Pain on chest wall palpation strongly suggests a musculoskeletal cause such as costochondritis, slipping rib syndrome, or muscle strain from recent exertion or chronic cough. Mild musculoskeletal pain not associated with cough typically responds to analgesics (eg, acetaminophen or ibuprofen) and rest within a few days, although frequently no medication is necessary [6,8].

  Patients with chronic cough may need further diagnostic evaluation (algorithm 10). Chest pain usually resolves with treatment of the underlying cause (eg, asthma, cystic fibrosis, postnasal drip, or infection). (See "Approach to chronic cough in children".)

- **History of stressful event with or without hyperventilation** – A history of a recent stressful event (eg, parental divorce or death in the family), sleep disturbance, other somatic complaints (eg, headache or abdominal pain) or physical findings of hyperventilation suggest an underlying psychiatric cause of chest pain. Appropriate initial interventions consist of acknowledgement of the stressful event, reassurance, and follow-up counseling. Patients with known mental illness or significant emotional distress despite reassurance should be referred to a psychiatrist [6]. (See "Causes of nontraumatic chest pain in children and adolescents", section on 'Psychiatric'.)

- **Fever and cough** – Fever, cough, tachypnea, and abnormal lung examination describe findings consistent with viral or community-acquired bacterial pneumonia in stable patients. These patients warrant treatment based upon age and type of pneumonia. Patients with probable bacterial pneumonia warrant empiric oral antibiotic therapy (table 7). (See "Community-acquired pneumonia in children: Outpatient treatment" and "Community-acquired pneumonia in children: Outpatient treatment", section on 'Indications for hospitalization'.)

- **Cough or dyspnea at night or with exercise** – Night-time cough or cough and difficulty breathing with exercise suggests exercise-induced or subclinical bronchospasm. Patients with exercise symptoms that overlap with possible cardiac etiologies warrant an ECG and a chest radiograph. In patients with normal findings, appropriate management consists of a trial of bronchodilators (eg, albuterol) with assured follow-up in patients with known asthma or referral for formal exercise testing in patients without an asthma history. (See "Exercise-induced bronchoconstriction", section on 'Diagnosis' and "Exercise-induced bronchoconstriction", section on 'Management'.)

- **Heart burn** – Substernal burning pain after meals supports the diagnosis of gastroesophageal reflux disease. Patients with frequent and severe pain should be referred to a pediatric gastroenterologist for evaluation of possible esophagitis. Patients with mild symptoms can begin “step up” therapy as described in detail

separately. (See "Gastroesophageal reflux disease in children and adolescents: Management", section on 'Heartburn'.)

- **Pain after taking medications** – Pain temporally associated with taking medications known to cause esophagitis (eg, tetracycline, doxycycline, aspirin, or nonsteroidal antiinflammatory medications), often without water and at night, strongly suggests pill esophagitis. Whenever possible, the medication should be discontinued. Acid suppression with antacids or histamine-2 receptor blockers (eg, famotidine) should be performed to address any associated gastroesophageal reflux disease. Some patients can have severe pain with swallowing and may not be able to eat or drink. Such patients warrant intravenous hydration and urgent consultation with a pediatric gastroenterologist. (See "Pill esophagitis", section on 'Clinical manifestations' and "Pill esophagitis", section on 'Management'.)

- **Breast pain** – Breast tenderness on palpation in association with gynecomastia in males and thelarche, pregnancy, mastitis, or fibrocystic disease in females. Reassurance is appropriate for patients with gynecomastia and thelarche. (See "Gynecomastia in children and adolescents", section on 'Management'.)

- **Normal history and physical examination** – Patients with idiopathic chest pain have no obvious cause on history and physical examination. They should receive reassurance, anticipatory guidance that the pain may recur, and follow-up with their primary care provider in four to six weeks. (See "Causes of nontraumatic chest pain in children and adolescents", section on 'Idiopathic' and 'Disposition' below.)

INDICATIONS FOR PEDIATRIC SUBSPECIALTY CONSULTATION OR REFERRAL

Children with chest pain and known heart disease or any of the findings that suggest a cardiac cause on history, physical examination, or ECG (table 1) warrant prompt consultation with a pediatric cardiologist.

Urgent consultation with other pediatric specialists is indicated according to the identified condition as follows:

- Children with recurrent spontaneous pneumothorax or persistent large air leaks warrant pediatric surgical consultation. (See "Spontaneous pneumothorax in children", section on 'Subsequent management'.)

- In addition to the involvement of a pediatric cardiologist, children with aortic root dissection require emergent involvement of a pediatric cardiothoracic surgeon.

- Children with pulmonary embolism should undergo evaluation by pediatric specialists with expertise in providing antithrombotic therapy to children and treating the underlying cause of venous thrombosis and thromboembolism (eg, systemic lupus erythematosus, inherited thrombophilia, heart valve disease, or structural venous anomalies).

- Patients with sickle cell disease complicated by acute chest syndrome warrant consultation and management by a pediatric hematologist.

- Patients with pulmonary hypertension require multidisciplinary consultation with a pediatric cardiologist and pulmonologist.

Other consultations or referrals may be appropriate depending upon the degree of illness and the specific diagnosis. As an example, children with moderate to severe gastrointestinal symptoms, especially those with dysphagia, weight loss, or hematemesis and children with persistent or recurrent pain with no apparent etiology, warrant referral to a gastroenterologist. Evaluation of the upper gastrointestinal tract may reveal esophagitis, gastritis, and/or motility disorders such as diffuse esophageal spasm or achalasia. These abnormalities may be detected in some children with significant chest pain but no gastrointestinal symptoms [19]. (See "Causes of nontraumatic chest pain in children and adolescents", section on 'Gastrointestinal' and "Gastroesophageal reflux disease in children and adolescents: Management".)

Children and adolescents with serious emotional disturbance should receive psychiatric consultation.

DISPOSITION

Disposition and follow-up are determined based upon the etiology of the chest pain as follows:

- **Unstable patients** – Initially unstable patients warrant hospitalization in facilities with pediatric intensive care, pediatric cardiology, and other pediatric subspecialty capability. (See 'Unstable' above.)

- **Stable patients with concerning findings** – Patients with abnormal findings suggesting a life-threatening condition should have the disposition determined in consultation with the appropriate specialist. In many of these patients, hospitalization for further testing is appropriate. (See 'Acute chest pain with concerning findings' above.)

- **Stable patients with common findings** – Up to 99 percent of children and adolescents with nontraumatic chest pain have a benign cause, most frequently musculoskeletal chest pain, and can be discharged with additional primary care or specialty follow-up as determined by the underlying etiology. (See "Causes of nontraumatic chest pain in children and adolescents", section on 'Etiology' and 'Common findings' above.)

  Idiopathic chest pain may persist or recur. However, serious underlying illness is rare [17,20,21]. As an example, in an observational study that reported on the outcomes of 1938 patients with idiopathic chest pain followed by pediatric cardiologists for a median of four years, none died or had a serious underlying cause identified [17]. Thus, the clinician should avoid extensive ancillary testing in patients with a normal history and physical examination. However, these patients should have periodic follow-up until the pain resolves.

SUMMARY AND RECOMMENDATIONS

- **Epidemiology** – Chest pain is a common symptom in children and adolescents. Its underlying cause is typically benign in this patient population (table 2). (See "Causes of nontraumatic chest pain in children and adolescents", section on 'Epidemiology'.)

- **Evaluation** – A thorough history and physical examination usually can determine the cause and differentiate patients who require further diagnostic studies and acute intervention from those who can be managed with reassurance and continued follow-up. (See 'History' above and 'Physical examination' above.)

  Most children with chest pain have no history suggestive of life-threatening conditions (table 1) and either a normal physical examination or findings consistent with a musculoskeletal etiology. Further investigations are not needed in patients for whom a clear etiology, other than cardiac disease, can be established. Diagnostic studies (eg, ECG, chest radiograph, or echocardiogram) are warranted in patients with abnormal physical findings or with associated symptoms (eg, exertional syncope, palpitations, dyspnea, fever, or chest pain associated with exertion) that suggest underlying pulmonary or cardiac disease (table 1). (See 'Ancillary studies' above.)

  An ECG should also be obtained when a noncardiac etiology (eg, costochondritis, pneumonia, gastroesophageal reflux, pill esophagitis, or esophageal foreign body) **cannot** be established. (See 'Electrocardiogram' above.)

- **Approach and initial management** – The approach and initial management of children and adolescents with chest pain is determined by their clinical status on

presentation and findings on history and physical examination (algorithm 2). (See 'Approach and initial management' above.)

Children with chest pain and known heart disease or any of the findings that suggest a cardiac cause on history, physical examination, or ECG (table 1) warrant prompt consultation with a pediatric cardiologist. Urgent consultation with other pediatric specialists is indicated according to the identified condition. (See 'Indications for pediatric subspecialty consultation or referral' above.)

- **Disposition** – Up to 99 percent of children and adolescents with nontraumatic chest pain have a benign cause, most frequently musculoskeletal chest pain, and can be discharged with additional primary care or specialty follow-up determined by the underlying etiology. (See 'Disposition' above.)

REFERENCES

1. Selbst SM, Ruddy RM, Clark BJ, et al. Pediatric chest pain: a prospective study. Pediatrics 1988; 82:319.
2. Driscoll DJ, Glicklich LB, Gallen WJ. Chest pain in children: a prospective study. Pediatrics 1976; 57:648.
3. Friedman KG, Alexander ME. Chest pain and syncope in children: a practical approach to the diagnosis of cardiac disease. J Pediatr 2013; 163:896.
4. Danduran MJ, Earing MG, Sheridan DC, et al. Chest pain: characteristics of children/adolescents. Pediatr Cardiol 2008; 29:775.
5. Selbst SM. Chest pain in children. Pediatrics 1985; 75:1068.
6. Selbst SM. Approach to the child with chest pain. Pediatr Clin North Am 2010; 57:1221.
7. Massin MM, Bourguignont A, Coremans C, et al. Chest pain in pediatric patients presenting to an emergency department or to a cardiac clinic. Clin Pediatr (Phila) 2004; 43:231.
8. Collins SA, Griksaitis MJ, Legg JP. 15-minute consultation: a structured approach to the assessment of chest pain in a child. Arch Dis Child Educ Pract Ed 2014; 99:122.
9. Porter GE. Slipping rib syndrome: an infrequently recognized entity in children: a report of three cases and review of the literature. Pediatrics 1985; 76:810.
10. Coleman WL. Recurrent chest pain in children. Pediatr Clin North Am 1984; 31:1007.
11. Evans DA, Wilmott RW. Pulmonary embolism in children. Pediatr Clin North Am 1994; 41:569.

12. Asnes RS, Santulli R, Bemporad JR. Psychogenic chest pain in children. Clin Pediatr (Phila) 1981; 20:788.
13. Kashani JH, Lababidi Z, Jones RS. Depression in children and adolescents with cardiovascular symptomatology: the significance of chest pain. J Am Acad Child Psychiatry 1982; 21:187.
14. Pantell RH, Goodman BW Jr. Adolescent chest pain: a prospective study. Pediatrics 1983; 71:881.
15. Nakao K, Ohgushi M, Yoshimura M, et al. Hyperventilation as a specific test for diagnosis of coronary artery spasm. Am J Cardiol 1997; 80:545.
16. Kane DA, Fulton DR, Saleeb S, et al. Needles in hay: chest pain as the presenting symptom in children with serious underlying cardiac pathology. Congenit Heart Dis 2010; 5:366.
17. Saleeb SF, Li WY, Warren SZ, Lock JE. Effectiveness of screening for life-threatening chest pain in children. Pediatrics 2011; 128:e1062.
18. Friedman KG, Kane DA, Rathod RH, et al. Management of pediatric chest pain using a standardized assessment and management plan. Pediatrics 2011; 128:239.
19. Glassman MS, Medow MS, Berezin S, Newman LJ. Spectrum of esophageal disorders in children with chest pain. Dig Dis Sci 1992; 37:663.
20. Selbst SM, Ruddy R, Clark BJ. Chest pain in children. Follow-up of patients previously reported. Clin Pediatr (Phila) 1990; 29:374.
21. Rowland TW, Richards MM. The natural history of idiopathic chest pain in children. A follow-up study. Clin Pediatr (Phila) 1986; 25:612.

Topic 6469 Version 32.0

GRAPHICS

Serious cardiac causes of pediatric chest pain

Cause	History	Physical exam findings	Additional findings
Hypertrophic cardiomyopathy	Positive family history Exercise intolerance Exertional chest pain Atypical chest pain Angina Palpitations Syncope and/or arrhythmia	Dynamic systolic murmur	Laboratory: Not helpful ECG: Left ventricular hypertrophy or left axis deviation ST segment or T wave changes P-wave abnormalities Q waves Arrhythmias, premature ventricula beats
Dilated cardiomyopathy	Family history Decreased exercise tolerance, syncope Heart failure symptoms	Gallop Mitral regurgitation murmur	Laboratory (insufficient for diagnosis): Elevated D-dimer, BNP, and/or cardiac troponin I and T if ischemia and/or heart failure ECG: Intraventricular conduction delay High or low QRS voltages T-wave inversion Arrhythmia, premature ventricula beats
Anomalous coronary artery origin	Exertional chest pain Exertional syncope	Usually normal	Laboratory and ECG: Usually normal, unless coronary ischemia (refer to coronary ischemia below)

Coronary ischemia	Predisposing conditions: ▪ 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	Laboratory: Elevated cardiac troponin I and T ECG: ST segment depressions or elevation T wave changes Q waves
Severe left ventricular outflow tract obstruction	Exertional symptoms Exertional syncope	Loud systolic murmur	Laboratory: Not helpful ECG: Left ventricular hypertrophy Left ventricular strain pattern
Arrhythmia	Palpitations Syncope Positive family history	Irregular rhythm	Laboratory studies: Check serum potassium and calcium levels ECG: Atrial arrhythmia Ventricular arrhythmia Premature contractions Ventricular pre-excitation (Wolff-Parkinson-White)
Pericarditis	Positional chest pain Predisposing factors: ▪ Rheumatologic conditions ▪ Malignancy ▪ Mediastinal radiation ▪ Infection (HIV and other viral, TB) ▪ Renal failure ▪ Recent cardiac surgery	Pericardial friction rub Tachycardia/tachypnea Distant heart sounds, JVD	Laboratory: elevated CRP, ESR, WBC (nonspecific) Elevated cardiac troponin I and T (minority of patients) ECG: PR-segment depression ST-segment: ▪ Early phase ST-segment

			elevation ▪ Late phase T wave inversion
Myocarditis	Fever Viral prodrome Short duration of symptoms New onset heart failure symptoms	Tachycardia Tachypnea With or without gallop rhythm, ventricular ectopy Cardiovascular collapse	Laboratory studies: Elevated cardiac troponin I and T, elevated BNP ECG: Diffuse ST segment changes T wave inversions Ventricular ectopy Low QRS voltages
Aortic dissection	Personal or family history of bicuspid aortic valve or connective tissue disorders (Marfan, Loeys-Dietz, Ehlers-Danlos type IV, others) Acute onset sharp or tearing type of pain	Marfanoid body habitus Bifid uvula	Refer to coronary ischemia above
Pulmonary embolus	Pain description: Acute onset, pleuritic, associated dyspnea Calf or thigh pain/swelling Personal or family risk factors (inherited thrombophilia, hypercoagulable states, immobilization, medications)	Tachypnea Right ventricular heave (elevated pulmonary artery pressure) Loud and/or unsplit S_2 (if right ventricular pressure elevated)	Laboratory studies (insufficient for diagnosis): Decreased pulse oximetry, decreased PaO_2 Elevated D-dimer, BNP, and/or cardiac troponin I and T ECG: Right ventricular hypertrophy Non-specific ST-segment and T wave changes; minority with S1Q3T3 pattern

BNP: B-type natriuretic peptide; CRP: C-reactive peptide; ECG: electrocardiogram; ESR: erythrocyte sedimentation rate; HIV: human immunodeficiency virus; JVD: jugular venous distension; PaO_2: partial pressure oxygen (arterial); S_2: second heart sound; WBC: white blood cell count.

Original table modified for this publication. From: Friedman KG, Alexander ME. Chest pain and syncope in children: A practical approach to the diagnosis of cardiac disease. J Pediatr 2013; 163: 896. Table used with the permission of Elsevier Inc. All rights reserved.

Graphic 96306 Version 6.0

Causes of nontraumatic chest pain in children and adolescents

Life-threatening conditions (1 to 6% of patients)

Cardiac conditions

- Hypertrophic cardiomyopathy
- Aortic stenosis
- Coarctation of the aorta
- Coronary artery abnormalities (eg, Kawasaki disease, anomalous coronary arteries)
- Variant angina after recreational drug use (eg, cocaine, amphetamines, bath salts [methcanthinones], marijuana, and synthetic cannabinoids)
- Classic angina (early atherosclerotic disease from hyperlipidemias or diabetes mellitus)
- Pericarditis
- Myocarditis
- Dilated cardiomyopathy
- Tachyarrhythmias
- Aortic aneurysm or dissection
- Ruptured sinus of Valsalva aneurysm

Airway foreign body

Spontaneous pneumothorax

Pulmonary embolism

Pulmonary hypertension

Sickle cell disease with acute chest syndrome

Tumor (chest wall, pulmonary, or mediastinum)

Nontraumatic esophageal rupture (Boerhaave syndrome)

Spinal cord compression (tumor, vertebral collapse, or epidural abscess)

Common conditions (94 to 99% of patients)

Musculoskeletal conditions

- Muscle strain
- Costochondritis
- Slipping rib syndrome
- Precordial catch (Texidor twinge)
- Fibromyalgia
- Pectus excavatum or carinatum

Psychiatric

- Anxiety
- Panic disorder with or without hyperventilation syndrome
- Depression
- Hypochondriasis
- Somatic symptom disorder

Respiratory

- Pneumonia (can be life threatening)
- Asthma (can be life threatening)
- Chronic cough with muscle strain or, if severe, fractured rib
- Spontaneous pneumomediastinum

Gastrointestinal

- Gastroesophageal reflux disease
- Medication-induced ("pill") esophagitis
- Esophageal foreign body
- Esophageal spasm and achalasia
- Gastritis
- Peptic ulcer
- Irritable bowel disease
- Cholecystitis
- Pancreatitis

Breast

- Male adolescents: Gynecomastia
- Female adolescents: Pregnancy, thelarche, mastitis, or fibrocystic disease

Idiopathic

Other conditions

Tietze syndrome

Pleurodynia

Herpes zoster

Graphic 96739 Version 5.0

Pediatric respiratory rate and heart rate lower limit, normal range, and upper limit by age*

Age	Respiratory rate (breaths/minute)			Heart rate (beats/minute)		
	Lower limit (1st percentile)	Normal range (10th to 90th percentile)	Upper limit (99th percentile)	Lower limit (1st percentile)	Normal range (10th to 90th percentile)	Upper limit (99th percentile
0 to 3 months	25	34 to 57	66	107	123 to 164	181
3 to <6 months	24	33 to 55	64	104	120 to 159	175
6 to <9 months	23	31 to 52	61	98	114 to 152	168
9 to <12 months	22	30 to 50	58	93	109 to 145	161
12 to <18 months	21	28 to 46	53	88	103 to 140	156
18 to <24 months	19	25 to 40	46	82	98 to 135	149
2 to <3 years	18	22 to 34	38	76	92 to 128	142
3 to <4 years	17	21 to 29	33	70	86 to 123	136
4 to <6 years	17	20 to 27	29	65	81 to 117	131
6 to <8 years	16	18 to 24	27	59	74 to 111	123
8 to <12 years	14	16 to 22	25	52	67 to 103	115
12 to <15 years	12	15 to 21	23	47	62 to 96	108
15 to 18 years	11	13 to 19	22	43	58 to 92	104

* The respiratory and heart rates provided are based upon measurements in awake, healthy infants and children at rest. Many clinical findings besides the actual vital sign measurement must be taken into account when determining whether a specific vital sign is normal in an individual patient. Values

for heart rate or respiratory rate that fall within normal limits for age may still represent abnormal findings that are caused by underlying disease in a particular infant or child.

Data from: Fleming S, Thompson M, Stevens R, et al. Normal ranges of heart rate and respiratory rate in children from birth to 18 years of age: A systematic review of observational studies. Lancet 2011; 377:1011.

Graphic 78097 Version 11.0

Characteristics of the Marfanoid habitus

Name of measurement	Value consistent with Marfanoid habitus*	Technique for measurement and for calculation of ratio¶
Span/height ratio	>1.05	The span/height ratio (S-HR) is measured by asking the patient to stand with their back to a wall and in contact with the wall, with the arms out to 90 degrees at the shoulder, and hands and fingers fully extended. The arm span is the measure from the tip of the middle finger of one hand to that of the other. This is then divided by the height to obtain the S-HR.
Hand/height ratio	>0.11	The hand/height ratio (H-HR). Hand length is taken to be the distance between the distal palmar crease to the tip of the middle finger. This is then divided by the height to obtain the H-HR.
Foot/height ratio	>0.15	The foot/height ratio (F-HR). Foot length is taken to be the distance between the base of the posterior edge of the heel and the tip of the hallux. This is then divided by the height to obtain the F-HR.
Upper segment/lower segment ratio	<0.89	The upper segment/lower segment ratio (US/LSR) LS is taken to be the distance from the mid-point of the pubic symphysis and the floor with the patient standing erect. The US is calculated by subtracting the LS from the height. Thus US/LSR = (Height – LS)/LS.
Other features of the Marfanoid habitus include:		
▪ Dolichocephaly (skull width/length x 100 <76%) ▪ Scoliosis (Bunnell Scoliometer >5 degrees) ▪ Pectus deformities (excavatum or carinatum) ▪ Jaw deformities with overcrowding of teeth ▪ High-arched palate ▪ Long, flat feet, often with hammer toes, which flatten and pronate on weightbearing		

* The cut-off levels for the Marfanoid habitus are based upon the original data collected by McKusick[1].

¶ Equipment needed to perform the necessary measurements comprises an accurate height-measuring scale, a tape measure for measuring the arm span, a Bunnell scoliometer for establishing whether or not a significant scoliosis is present[2], and a calculator.

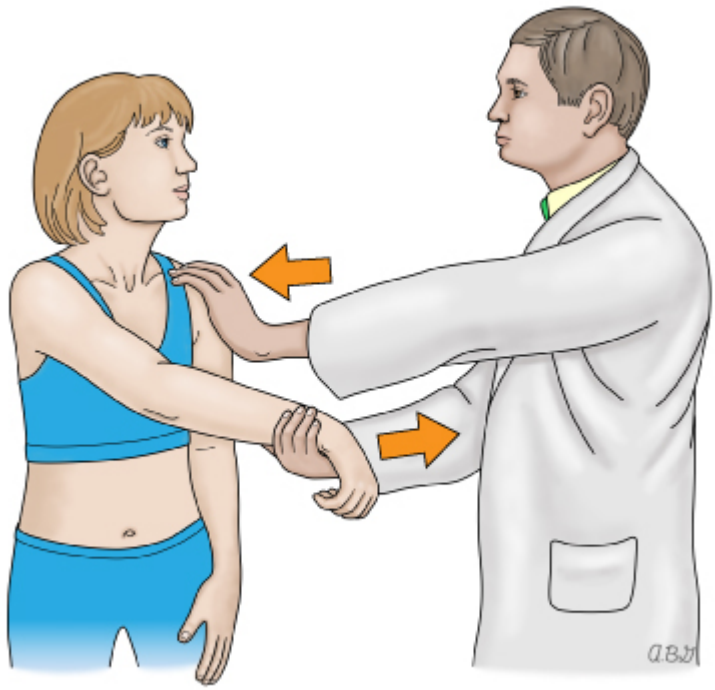
References:

1. McKusick VA. The marfan syndrome. In: Heritable Disorders of Connective Tissue. 4th ed, C.V. Mosby, St. Louis 1972.

2. Bunnell WP. Outcome of spinal screening. Spine 1993; 18:1572.

Graphic 95551 Version 2.0

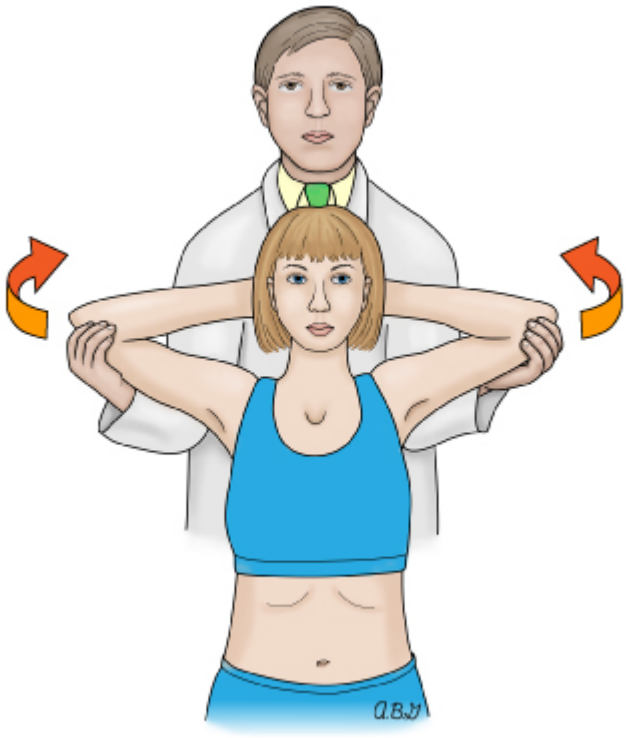
Horizontal arm traction maneuver in a pediatric patient



The physician pulls the flexed arm across the anterior chest with steady, prolonged traction while providing pushing against the opposite shoulder.

Graphic 96607 Version 1.0

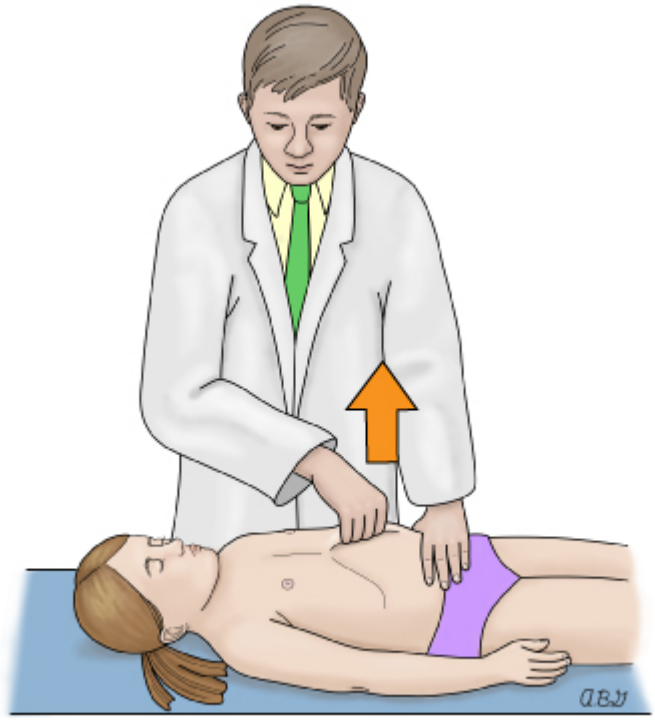
Crowing rooster maneuver in a pediatric patient



The physician asks the patient to clasp his or her hands behind the head. While standing behind the patient, the physician grasps the elbows and exerts backward and superiorly directed traction as pictured.

Graphic 96606 Version 1.0

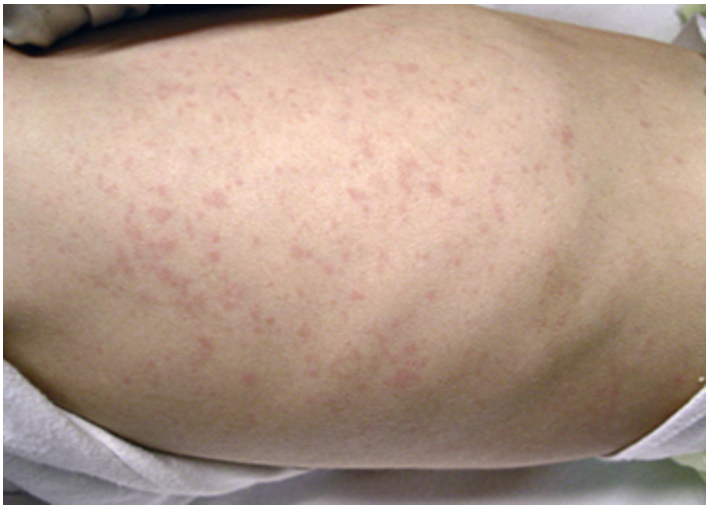
Hooking maneuver in a child



The clinician performs the hooking maneuver by hooking the curled fingers under the anterior costal margins and gently pulling the rib cage anteriorly.

Graphic 96608 Version 1.0

Systemic juvenile idiopathic arthritis rash

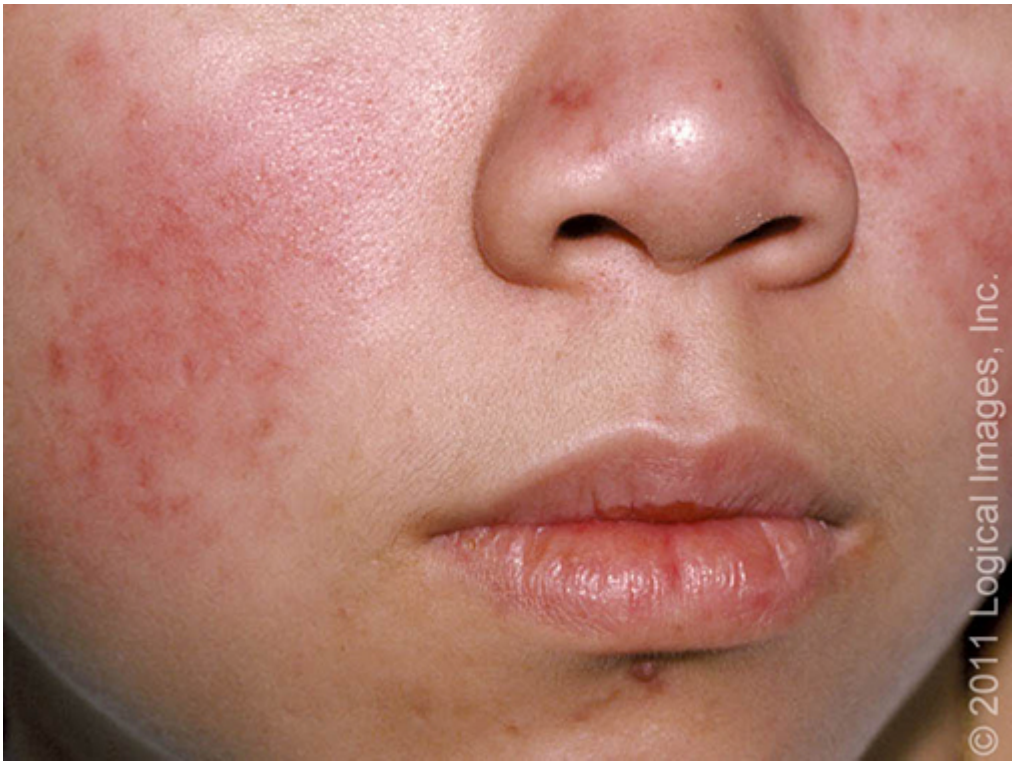


A salmon-pink rash is characteristic of this juvenile idiopathic arthritis (JIA) subtype. The rash is brought out by heat and often can be found in the axillae and around the waist but may be present anywhere on the body.

Courtesy of Robert Sundel, MD.

Graphic 62959 Version 9.0

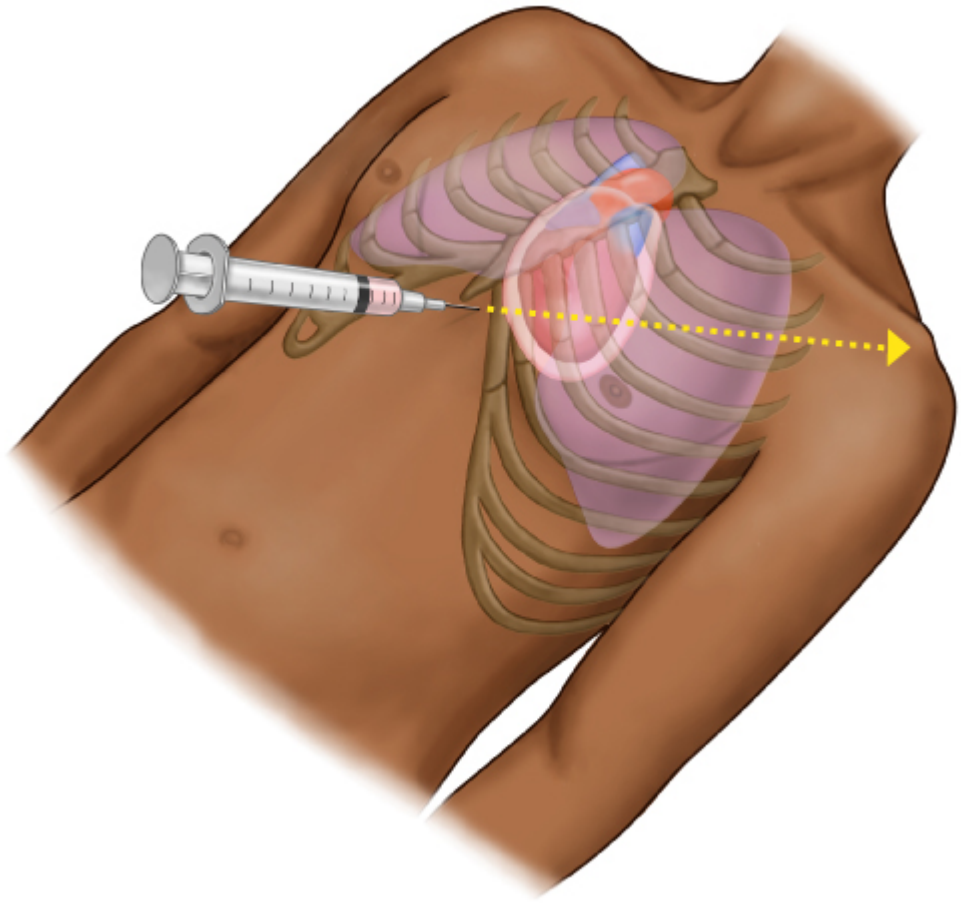
Acute cutaneous lupus erythematosus



Malar erythema and subtle edema are present in this patient with systemic lupus erythematosus.

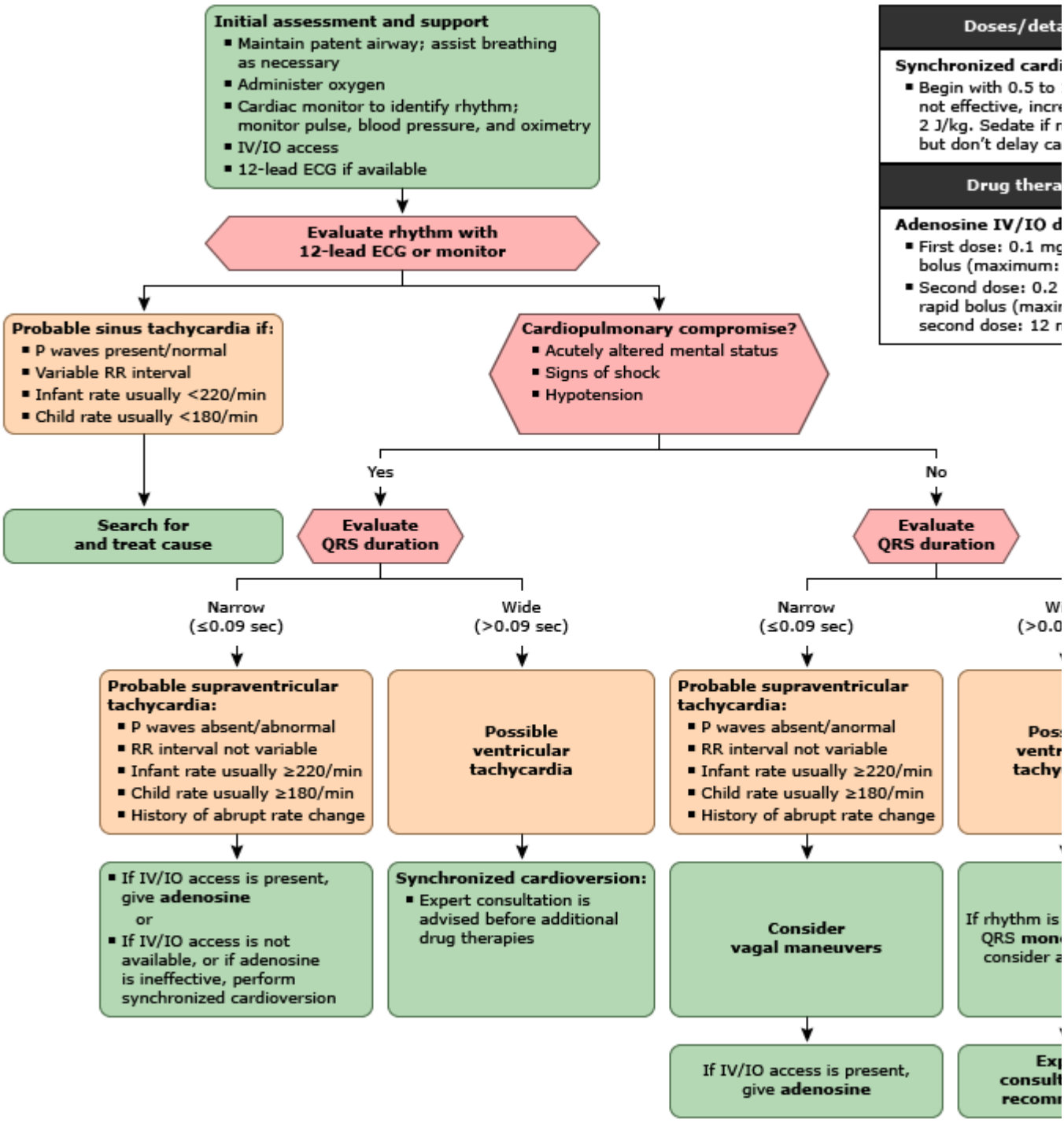
Graphic 75781 Version 6.0

Emergency pericardiocentesis subxiphoid approach



Graphic 81442 Version 3.0

Pediatric tachycardia with a pulse 2020 update



IV: intravenous; IO: intraosseous; ECG: electrocardiogram; J/kg: joules per kilogram.

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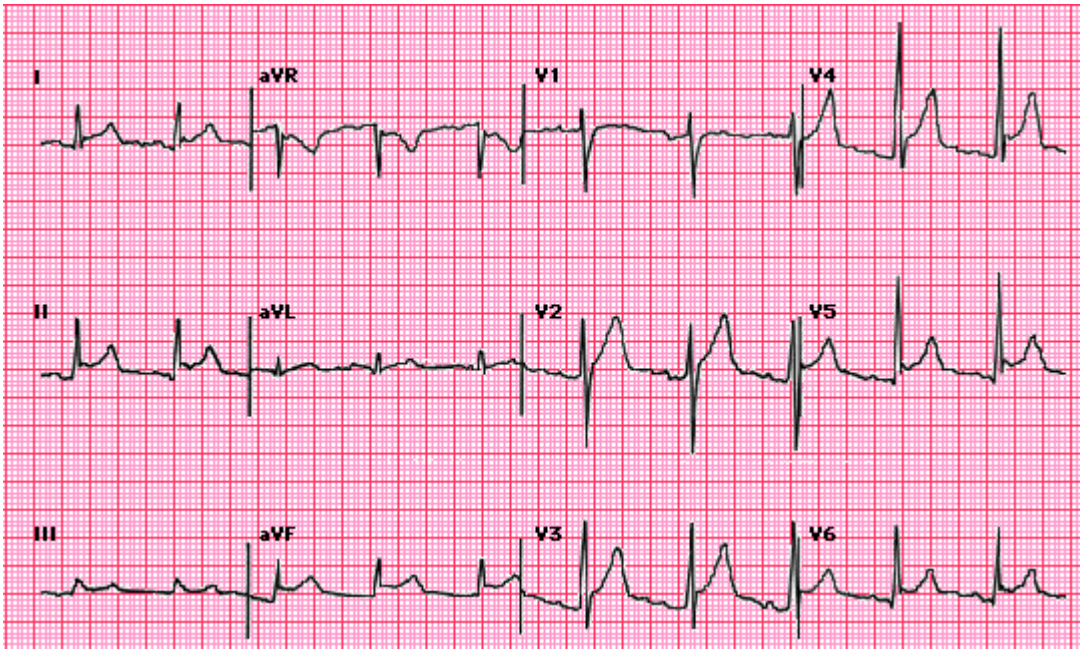
Graphic 129940 Version 10.0

Electrocardiogram (ECG) in pericarditis

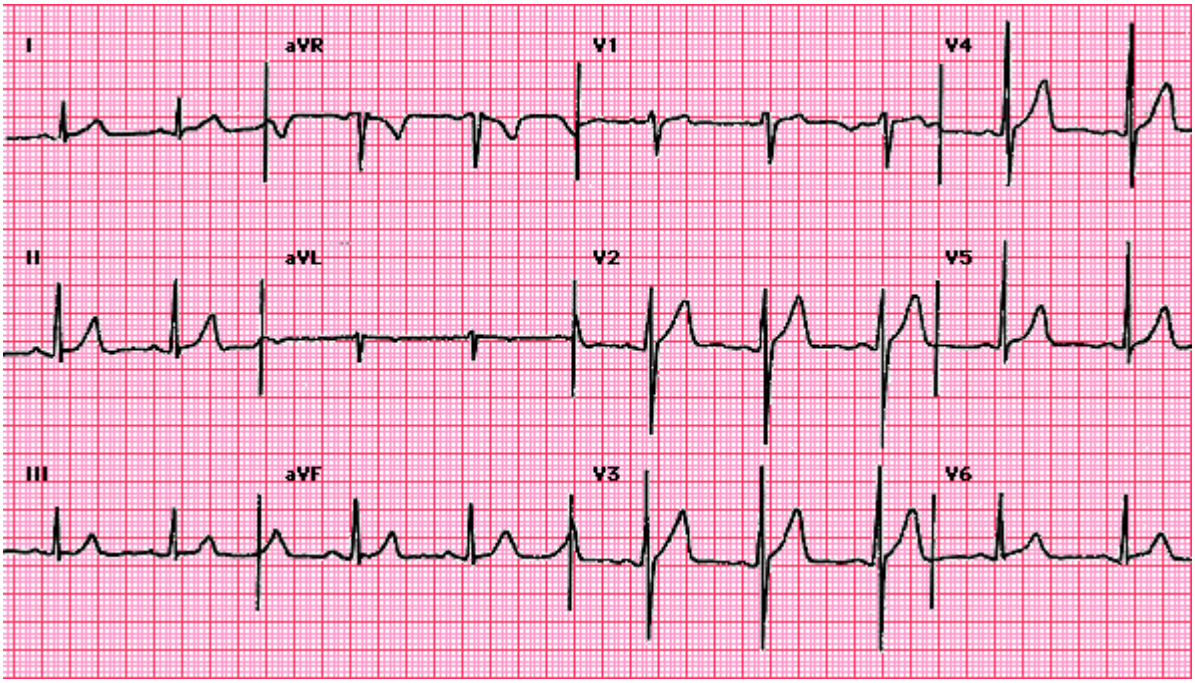


Electrocardiogram in acute pericarditis showing diffuse upsloping (concave up) ST-segment elevations seen best here in leads II, III, aVF, and V2 to V6. There is also subtle PR-segment deviation (positive in aVR, negative in most other leads). ST-segment elevation is due to a ventricular current of injury associated with epicardial inflammation; similarly, the PR-segment changes are due to an atrial current of injury, which, in pericarditis, typically displaces the PR segment upward in lead aVR and downward in most other leads.

Courtesy of Ary Goldberger, MD.

Graphic 77572 Version 5.0

Normal ECG

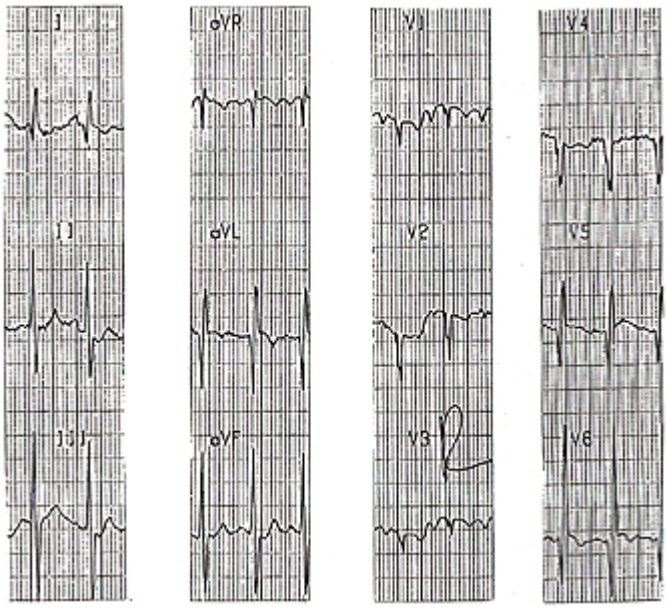


Normal electrocardiogram showing normal sinus rhythm at a rate of 75 beats/minute, a PR interval of 0.14 seconds, a QRS interval of 0.10 seconds, and a QRS axis of approximately 75°.

Courtesy of Ary Goldberger, MD.

Graphic 76183 Version 4.0

An electrocardiogram obtained from a patient with anomalous left main coronary artery arising from the pulmonary artery

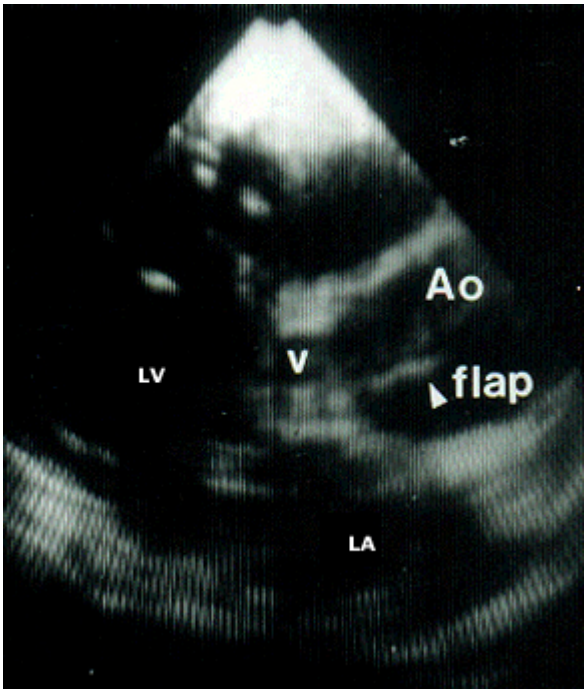


There is a pattern of anterolateral infarction with deep wide Q waves in the anterior (I, aVL) and lateral (V5, V6) leads. There are T wave inversions in these leads as well.

Courtesy of Robert L Geggel, MD.

Graphic 72881 Version 3.0

Ascending aortic dissection on echocardiogram

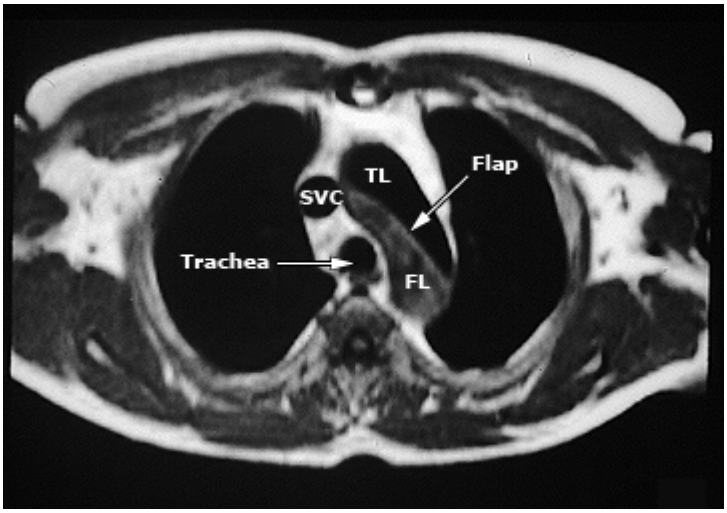


Modified long axis view shows a proximal dissection of the aortic root (Ao), with a flap extending to the aortic valve (V). This aortic pathology is seen by imaging the ascending aorta one interspace above the usual long axis precordial window.

LA: left atrium; LV: left ventricle.

Graphic 75705 Version 3.0

Aortic dissection on magnetic resonance imaging



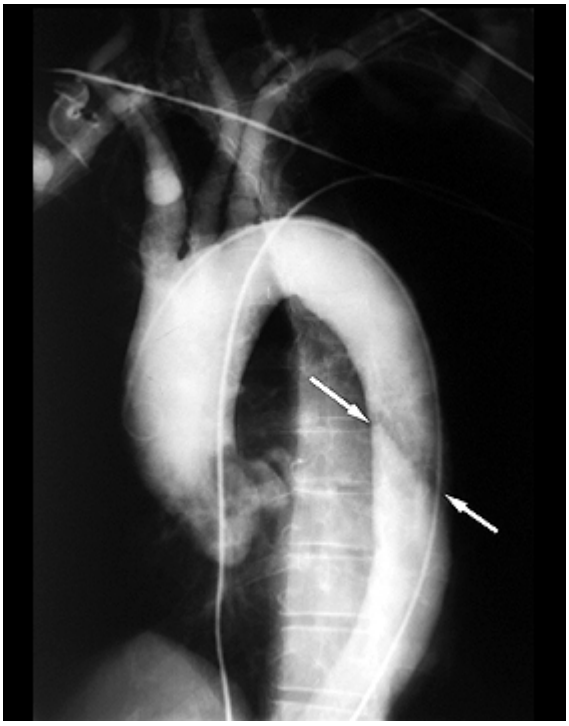
Transverse (axial) spin-echo CMR in a patient with an aortic dissection at the level of the aortic arch. The true lumen (TL), false lumen (FL), and intimal flap can be easily identified. The trachea and superior vena cava (SVC) are also seen.

CMR: cardiovascular magnetic resonance.

Courtesy of Warren Manning, MD.

Graphic 69616 Version 6.0

Aortogram showing descending thoracic aortic dissection

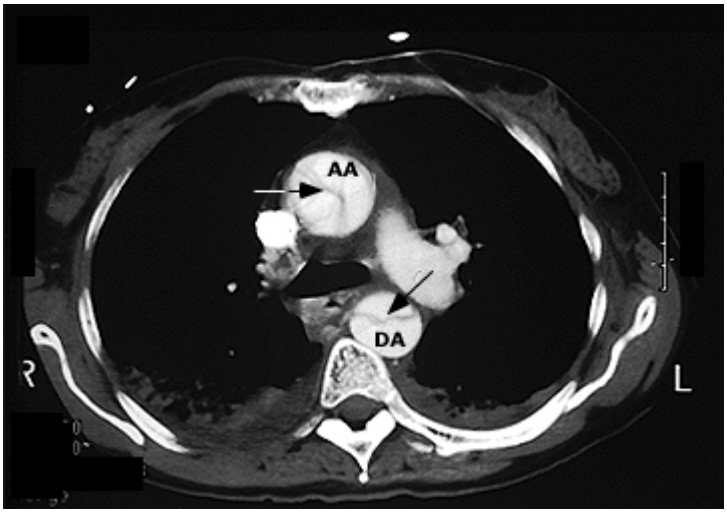


This aortogram demonstrates dissection of the descending thoracic aorta, arising immediately distal to the origin of the left subclavian artery. An oblique lucency is noted within the lumen of the aorta, which is a diagnostic feature (arrows).

Courtesy of Jonathan Kruskal, MD.

Graphic 55207 Version 3.0

Thoracic aortic dissection on computed tomography

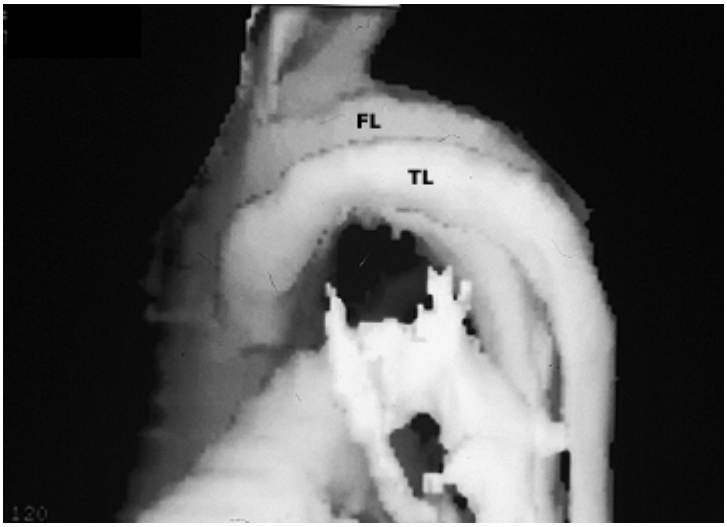


Transverse plane through ascending (AA) and descending (DA) thoracic aorta showing the intimal flap (arrows) and both lumens of a type A aortic dissection. One cannot distinguish between the true and false lumens based on this view alone.

Courtesy of Vassilios Raptopoulos, MD.

Graphic 81919 Version 3.0

Thoracic aortic dissection on reconstructed computed tomography



Sagittal plane reconstruction of transverse and descending thoracic aorta showing the true (TL) and false (FL) lumens of the same patient as the previous CT.

Courtesy of Vassilios Raptopoulos, MD.

Graphic 58395 Version 3.0

Causes of elevated troponin

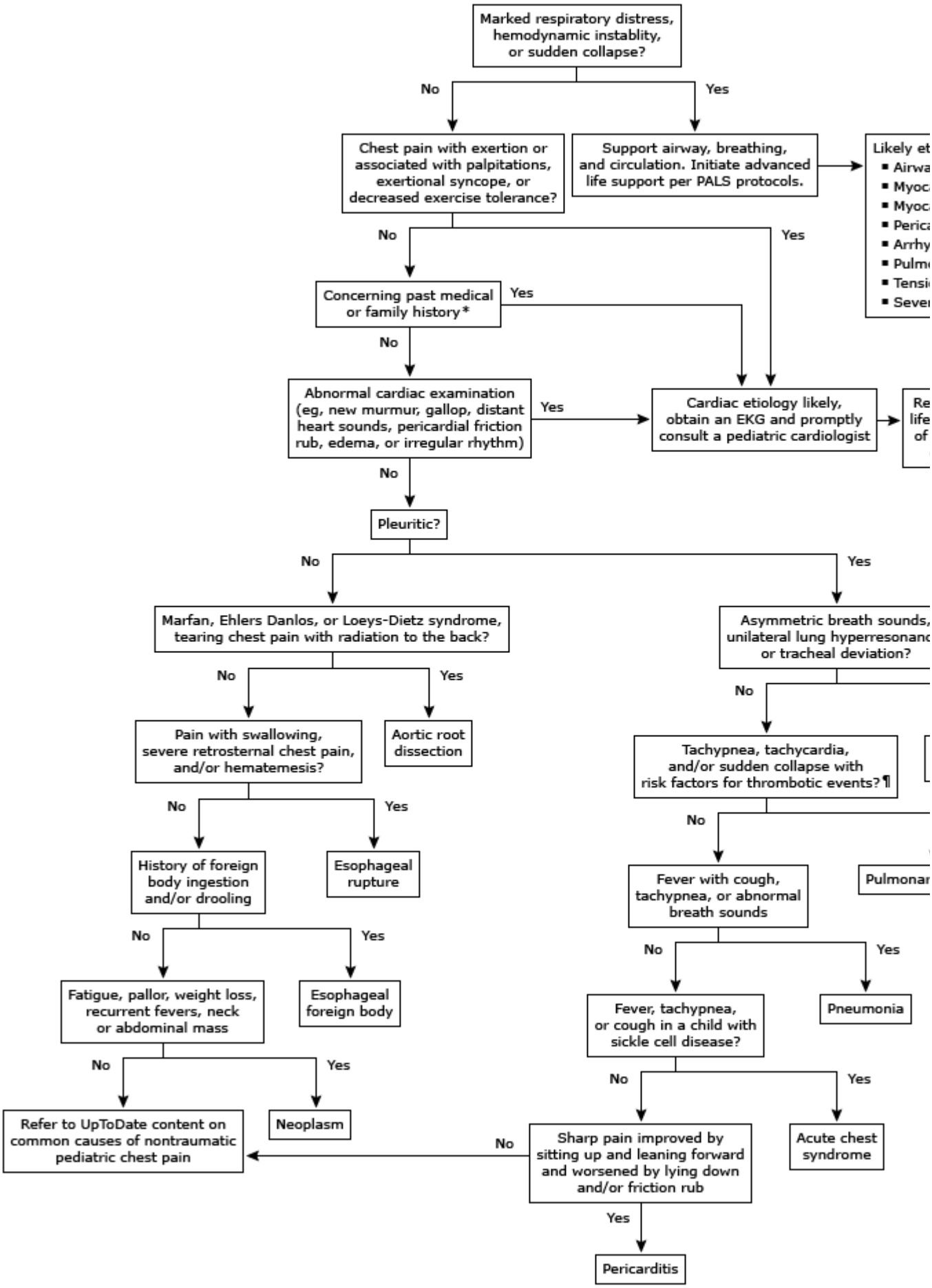
Myocardial ischemia
Acute coronary syndrome
STEMI
NSTEMI
Other coronary ischemia
Arrhythmia: tachy- or brady-
Cocaine/methamphetamine use
Coronary intervention (PCI or cardiothoracic surgery)
Coronary artery spasm (variant angina)
Stable coronary atherosclerotic disease in setting of increased O_2 demand (eg, tachycardia)
Severe hypertension
Coronary embolus
Aortic dissection
Coronary artery vasculitis (SLE, Kawasaki)
Noncoronary ischemia
Shock (hypotension)
Hypoxia
Hypoperfusion
Pulmonary embolism
Global ischemia
Cardiothoracic surgery
Myocardial injury with no ischemia
Comorbidities
Renal failure
Sepsis
Infiltrative diseases
Acute respiratory failure
Stroke
Subarachnoid hemorrhage
Specific identifiable precipitants
Extreme exertion

Cardiac contusion
Burns >30% BSA
Cardiotoxic meds: anthracyclines, herceptin
Electrical shock
Carbon monoxide exposure
Other
Stress (takotsubo) cardiomyopathy
Myocarditis
Myopericarditis
Rhabdomyolysis involving cardiac muscle
Hypertrophic cardiomyopathy
Peripartum cardiomyopathy
Heart failure, malignancy, stress cardiomyopathy

STEMI: ST elevation myocardial infarction; NSTEMI: non-ST elevation myocardial infarction; PCI: percutaneous coronary intervention; SLE: systemic lupus erythematosus; BSA: body surface area.

Graphic 54910 Version 15.0

Approach to pediatric nontraumatic chest pain



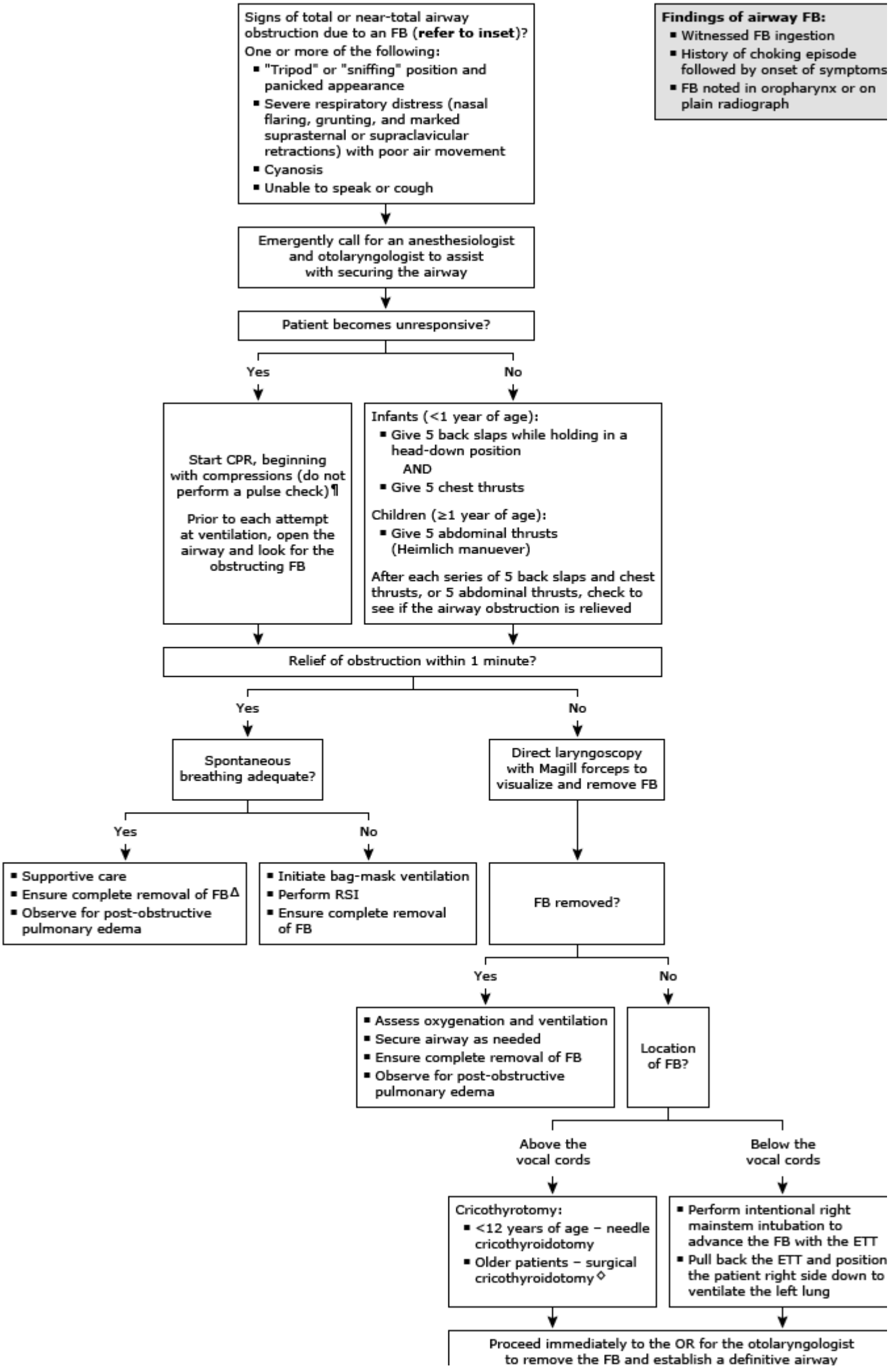
PALS: pediatric advanced life support; EKG: electrocardiogram.

* For example, history of congenital heart disease, heart transplant, Kawasaki disease in the patient or family history of cardiomyopathy or relatives who died suddenly before the age of 50 years.

¶ Risk factors for pulmonary embolism in children include immobility, oral contraceptive use, pregnancy termination, ventriculoatrial shunts for hydrocephalus, central venous catheters, solid tumors, heart disease, infection, dehydration, hypercoagulable states, low cardiac output, and obesity.

Graphic 97535 Version 3.0

Management of upper airway obstruction due to a foreign body in children*


Signs of total or near-total airway obstruction due to an FB (refer to inset)?
One or more of the following:
"Tripod" or "sniffing" position and panicked appearance
Severe respiratory distress (nasal flaring, grunting, and marked suprasternal or supraclavicular retractions) with poor air movement
Cyanosis
Unable to speak or cough
Findings of airway FB:
Witnessed FB ingestion
History of choking episode followed by onset of symptoms
FB noted in oropharynx or on plain radiograph
Emergently call for an anesthesiologist and otolaryngologist to assist with securing the airway
Patient becomes unresponsive?
Yes
No
Start CPR, beginning with compressions (do not perform a pulse check)¶
Prior to each attempt at ventilation, open the airway and look for the obstructing FB
Infants (<1 year of age):
Give 5 back slaps while holding in a head-down position
AND
Give 5 chest thrusts
Children (≥1 year of age):
Give 5 abdominal thrusts (Heimlich manuever)
After each series of 5 back slaps and chest thrusts, or 5 abdominal thrusts, check to see if the airway obstruction is relieved
Relief of obstruction within 1 minute?
Yes
No
Spontaneous breathing adequate?
Direct laryngoscopy with Magill forceps to visualize and remove FB
Yes
No
Supportive care
Ensure complete removal of FBΔ
Observe for post-obstructive pulmonary edema
Initiate bag-mask ventilation
Perform RSI
Ensure complete removal of FB
FB removed?
Yes
No
Assess oxygenation and ventilation
Secure airway as needed
Ensure complete removal of FB
Observe for post-obstructive pulmonary edema
Location of FB?
Above the vocal cords
Below the vocal cords
Cricothyrotomy:
<12 years of age – needle cricothyroidotomy
Older patients – surgical cricothyroidotomy◇
Perform intentional right mainstem intubation to advance the FB with the ETT
Pull back the ETT and position the patient right side down to ventilate the left lung
Proceed immediately to the OR for the otolaryngologist to remove the FB and establish a definitive airway

FB: foreign body; CPR: cardiopulmonary resuscitation; RSI: rapid sequence intubation; ETT: endotracheal tube; OR: operating room.

* The following findings suggest upper airway obstruction:
- Inspiratory stridor, wheezing, or stertor
- Suprasternal or supraclavicular retractions
- Prolonged inspiratory phase
- Oral mucosa or tongue swelling
- Drooling
- Dysphagia

Positions of comfort to help maintain airway patency in patients with severe obstruction:
- "Sniffing" position (neck is mildly flexed and head is mildly extended)
- Tripod position (leaning forward while bracing on the arms, with neck hyperextended and chin thrust forward)

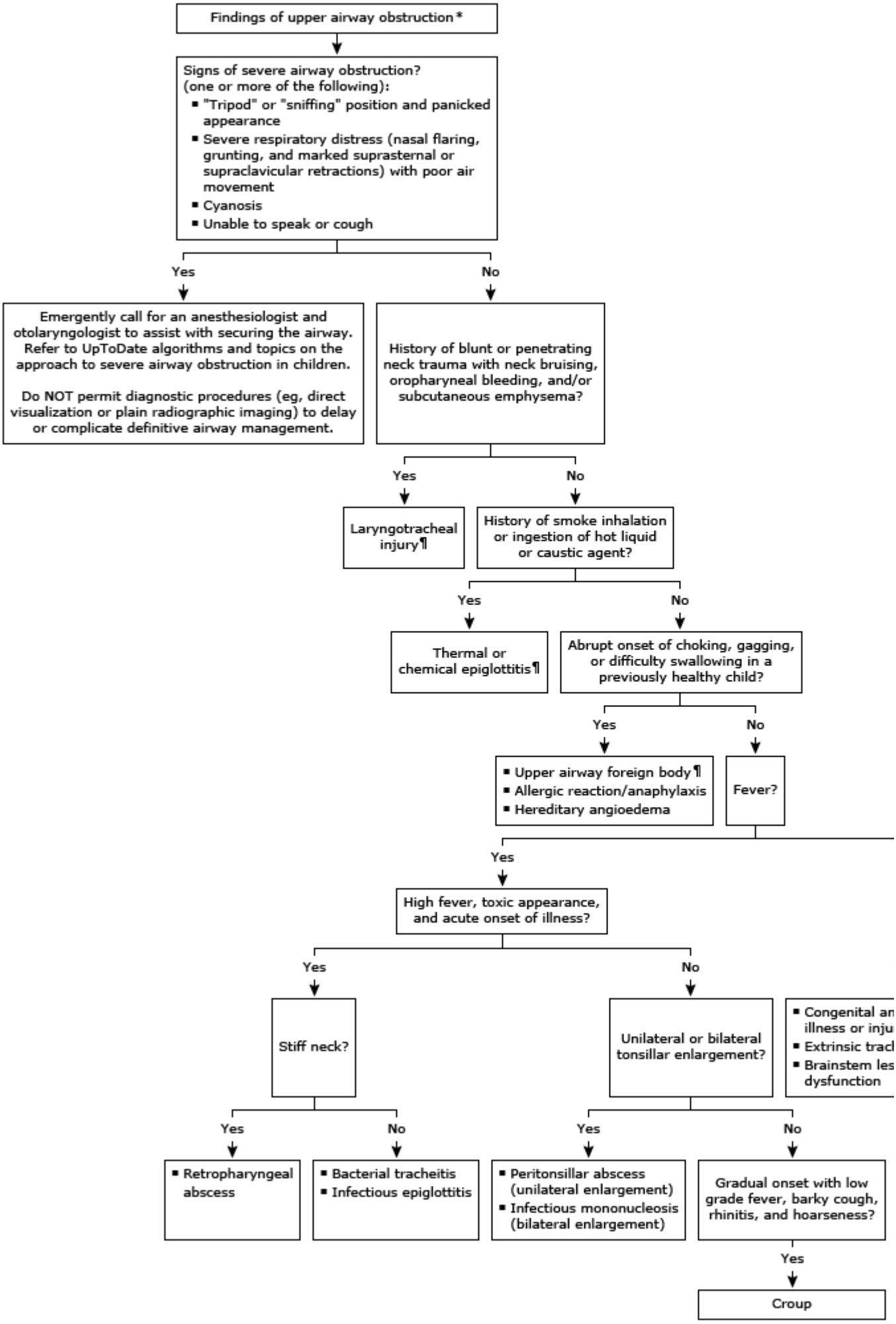
¶ Refer to UpToDate algorithms and topics on pediatric basic life support for health care providers and FB obstruction.

Δ Refer to UpToDate topics on evaluation of upper airway obstruction in children.

◇ Surgical cricothyrotomy may be appropriate in selected patients younger than 12 years of age, as determined by cricothyroid membrane size. Refer to UpToDate topics on needle and surgical cricothyroidotomy.

Graphic 55990 Version 5.0

Diagnostic approach to upper airway obstruction in children


Findings of upper airway obstruction*
Signs of severe airway obstruction?
(one or more of the following):
"Tripod" or "sniffing" position and panicked appearance
Severe respiratory distress (nasal flaring, grunting, and marked suprasternal or supraclavicular retractions) with poor air movement
Cyanosis
Unable to speak or cough
Yes
No
Emergently call for an anesthesiologist and otolaryngologist to assist with securing the airway. Refer to UpToDate algorithms and topics on the approach to severe airway obstruction in children.
Do NOT permit diagnostic procedures (eg, direct visualization or plain radiographic imaging) to delay or complicate definitive airway management.
History of blunt or penetrating neck trauma with neck bruising, oropharyneal bleeding, and/or subcutaneous emphysema?
Yes
No
Laryngotracheal injury¶
History of smoke inhalation or ingestion of hot liquid or caustic agent?
Yes
No
Thermal or chemical epiglottitis¶
Abrupt onset of choking, gagging, or difficulty swallowing in a previously healthy child?
Yes
No
Upper airway foreign body¶
Allergic reaction/anaphylaxis
Hereditary angioedema
Fever?
Yes
High fever, toxic appearance, and acute onset of illness?
Yes
No
Stiff neck?
Unilateral or bilateral tonsillar enlargement?
Congenital an
illness or inju
Extrinsic tracl
Brainstem les
dysfunction
Yes
No
Yes
No
Retropharyngeal abscess
Bacterial tracheitis
Infectious epiglottitis
Peritonsillar abscess (unilateral enlargement)
Infectious mononucleosis (bilateral enlargement)
Gradual onset with low grade fever, barky cough, rhinitis, and hoarseness?
Yes
Croup

* The following findings suggest upper airway obstruction:
    - Inspiratory stridor, wheezing, or stertor
    - Suprasternal or supraclavicular retractions
    - Prolonged inspiratory phase
    - Oral mucosa or tongue swelling
    - Drooling
    - Dysphagia

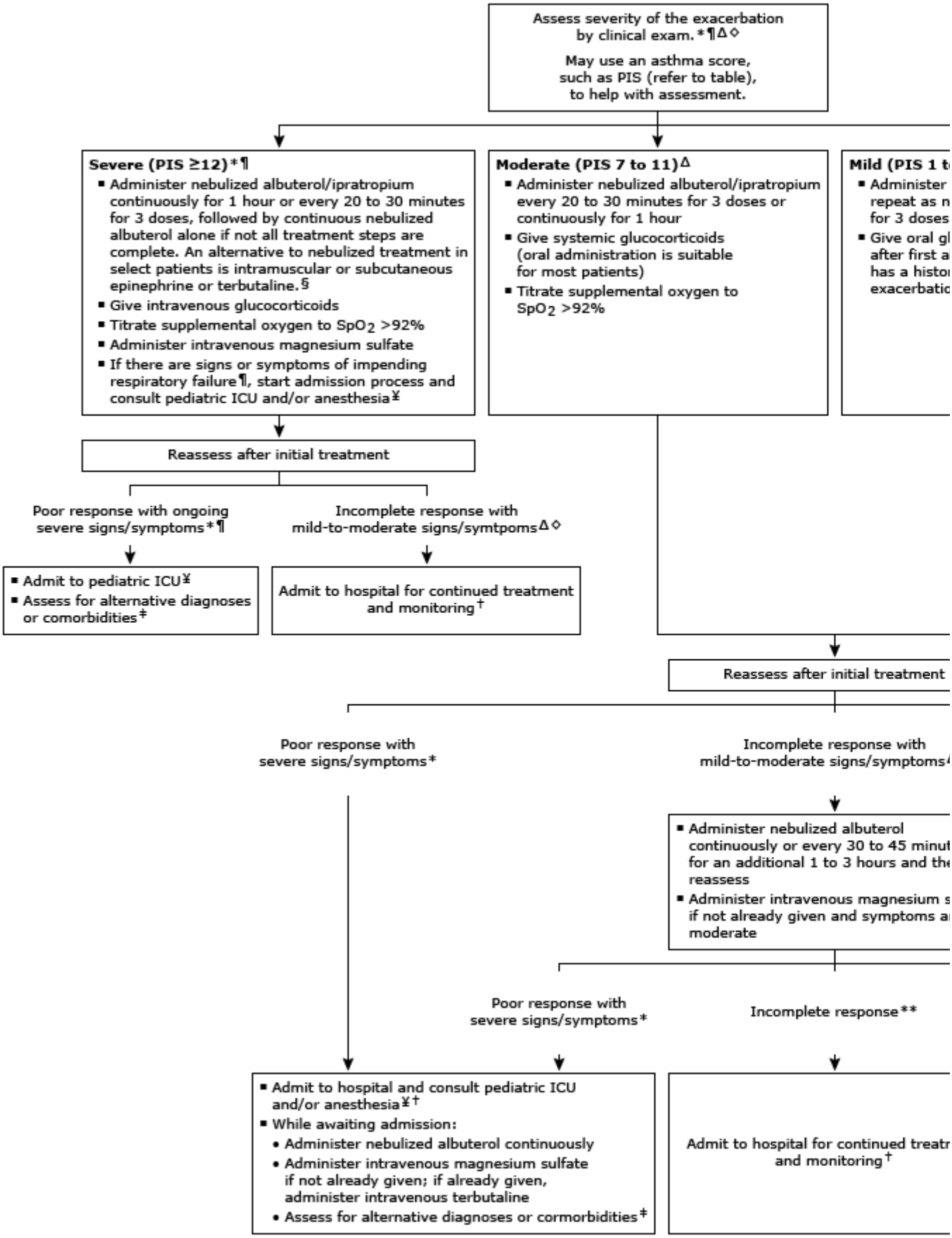
Positions of comfort to help maintain airway patency in patients with severe obstruction:

- "Sniffing" position (neck is mildly flexed and head is mildly extended)
- Tripod position (leaning forward while bracing on the arms with neck hyperextended and chin thrust forward)

¶ Given the risk of sudden decompensation, patients with significant laryngotracheal injury, thermal or chemical epiglottitis, or symptomatic upper airway obstruction warrant emergency consultation with an anesthesiologist or pediatric intensivist and an otolaryngologist to help secure the airway.

Graphic 54996 Version 4.0

Management of asthma exacerbations in children <12 years old in the emergency department



PIS: pulmonary index score; SpO_2: pulse oxygen saturation; ICU: intensive care unit; SVN: small-volume nebulizer; MDI: metered-dose inhaler; pCO_2: partial pressure of carbon dioxide.

* A severe exacerbation is characterized by inability to repeat a short phrase, extreme tachypnea, inspiratory and expiratory wheezing, an inspiratory-expiratory ratio exceeding 1:2, very poor aeration, significant use of accessory muscles, and an oxygen saturation that is typically <92%.

¶ Signs of impending respiratory failure include cyanosis, inability to maintain respiratory effort (respiratory rate may be inappropriately normal to low), depressed mental status (lethargy or agitation), SpO_2 <90%, and respiratory acidosis (elevated pCO_2 noted on venous, arterial, or capillary blood gas sample).

Δ A moderate exacerbation is characterized by normal alertness, tachypnea, wheezing throughout expiration with or without inspiratory wheezing, an inspiratory-expiratory ratio of approximately 1:2, significant use of accessory muscles, and an oxygen saturation that is typically 92 to 95%.

◇ A mild exacerbation in characterized by normal alertness, slight tachypnea, expiratory wheezing only, a mildly prolonged expiratory phase, minimal accessory muscle use, and an oxygen saturation of >95%.

§ An alternative in children with poor inspiratory flow or who cannot cooperate with nebulized therapy is intramuscular or subcutaneous epinephrine or terbutaline.

¥ Patient may need to be transported to a hospital with the appropriate pediatric resources.

‡ Alternative diagnoses or comorbidities include, but are not limited to, upper airway obstruction, pneumothorax, cardiac tamponade, sepsis, pneumonia, heart failure, and mediastinal mass. Refer to UpToDate topics on these disorders and on acute respiratory distress in children.

† Refer to UpToDate topics on inpatient and ICU management of severe asthma exacerbations in children.

** Good response includes resolution of symptoms or marked improvement in a patient/family with good adherence and understanding of asthma management. Marked improvement is manifested by diminished or absent wheezing and retractions and increased aeration that is sustained for at least 60 minutes after the most recent albuterol dose. Children who were moderately to severely ill on arrival should demonstrate a more sustained period of improvement before discharge to home. Patients with incomplete response have continued mild-to-moderate symptoms and do not meet criteria for discharge.

¶¶ Discharge medications include an inhaled albuterol (every 4 to 6 hours for 3 days and then weaned as tolerated), oral glucocorticoids if given with initial treatment (3- to 5-day course for most children), and resumption or initiation of inhaled glucocorticoids if controller therapy is indicated. Patients should also receive discharge education of medications (purpose, side effects, proper administration technique), a written asthma action plan, prevention of exacerbations, and follow-up instructions.

Table inset: Courtesy of Richard Scarfone, MD, FAAP.

Graphic 123081 Version 1.0

Overview of approach to patients with suspected acute myocardial infarctio in the emergency department

ACS: acute coronary syndrome; aPTT: activated partial thromboplastin time; BUN: blood urea nitrogen; CBC: complete blood count; ECG: electrocardiogram; ED: emergency department; INR: international normalized ratio; IV: intravenous; MI: myocardial infarction; PCI: percutaneous coronary intervention; PT: prothrombin time; SL: sublingual.

* Initial laboratory work may vary by institution, but often includes: serum cardiac biomarkers, CBC with platelet count, PT and INR, aPTT, basic electrolytes, magnesium, BUN, creatinine, blood glucose, and serum lipid profile. Contraindications to nitrates include: severe aortic stenosis, hypertrophic cardiomyopathy, suspected right ventricular infarct, hypotension, marked bradycardia or tachycardia, and recent use of phosphodiesterase 5 inhibitor (eg, Viagra).

¶ The timing of repeat ECG and cardiac biomarkers depends on the troponin assay and institutional protocols, but is typically obtained after 2 to 6 hours.

Graphic 56813 Version 11.0

Initial shock management in children in settings with access to ICU care


Clinical signs of shock present (2 or more):
Diminished peripheral pulses
Cool, pale, or mottled skin
Altered mental status
Tachycardia or bradycardia
Provide supplemental oxygen, as needed, to avoid hypoxemia
Identify and treat life-threatening conditions:
Arrhythmia
Obstructive shock caused by tension pneumothorax, hemothorax, or cardiac tamponade
Anaphylaxis
Initiate continuous monitoring of HR, BP, and pulse oximetry
Treat respiratory failure with HFNC, NIV, or mechanical ventilation as needed*
Within 5 minutes, obtain venous access:
Preferred: two peripheral IVs of largest caliber that can be reliably inserted
If peripheral access not achieved: place IO cannula or central venous line
Obtain bedside glucose and laboratory studies (refer to inset)¶
If no signs of fluid overload, give IV or IO fluid resuscitation with balanced crystalloid solution (eg, Ringer's lactate) or normal saline as follows:Δ
Hypotensive shock: 20 mL/kg over 5 to 10 minutes
Compensated shock: 10 to 20 mL/kg over 5 to 20 minutes
Identify need for and give time-sensitive treatment based upon underlying condition:
Blood transfusion for hemorrhage
Empiric antimicrobial therapy for septic shock
Dextrose infusion for hypoglycemia
Prostaglandin E1 for ductal-dependent congenital heart disease
Systemic corticosteroids for adrenal insufficiency
Treatment of electrolyte abnormalities (eg, hyperkalemia, hypokalemia, or hypocalcemia)
Evaluate clinical targets; are all of the following present?
BP >5th percentile
Strong central and distal pulses
Warm skin with capillary refill <2 seconds
Alert mental status
Urine output 1 mL/kg or greater after circulating volume is restored
Yes
No
Continue monitoring
Treat underlying condition
Admit to appropriate inpatient setting
Signs of fluid overload (one or more of the following):
Pulmonary rales
Worsening respiratory distress with new or worsening oxygen requirement
Gallop rhythm
Jugular venous distension
Hepatomegaly
Cardiomegaly or pulmonary edema on bedside US or chest radiograph
Yes
No
Probable cardiogenic shock:
Evaluate cardiac function (bedside US, ECHO◇, and pediatric cardiology consultation)
If indicated, give smaller fluid boluses over a longer period of time (eg, 5 to 10 mL/kg over 15 to 30 minutes)§
Start vasoactive therapy¥
For persistent signs of shock, repeat 10 to 20 mL/kg f
up to 60 mL/kg within the first hour
Evaluate clinical targets after each bolus
Signs of shock persist
Re-evaluate presumed cause of shock
For suspected hemorrhagic shock, evaluate for source of bleeding and give blood products
For suspected hypovolemic shock, re-estimate fluid losses and continue fluid replacement
For patients with suspected distributive shock, start vasoactive agents¥
Obtain consultation with a pediatric critical care specialist and arrange transfer to a pediatric ICU
Inset: Initial laboratory stud
All patients:
Rapid blood glucose
Complete blood count with d
Serum electrolytes
Venous or arterial blood gas
Blood lactate
Blood urea nitrogen and seru
Urine dipstick
Septic shock suspected:
Serum total bilirubin
Alanine aminotransferase
Ionized blood calcium
PT and INR
PTT
Fibrinogen and D-dimer
Blood culture
Other cultures as indicated b
Diagnostic serologic testing a
Inflammatory biomarkers (eg
Urinalysis
Urine culture
Hemorrhagic shock suspected:
Type and cross-match
PT and INR
PTT
Additional studies based upo
(refer to UpToDate content o
Other studies:
ECG (arrhythmia or signs of
Bedside US (suspected obstr
Pediatric cardiology specialis
Chest radiograph for patients
60 mL/kg of isotonic fluid ad
Two-view abdominal radiogra
suggest possible perforation

ICU: intensive care unit; HR: heart rate; BP: blood pressure; HFNC: high-flow oxygen by nasal cannula; NIV: noninvasive ventilation; IV: intravenous; IO: intraosseous; US: ultrasound; ECHO: echocardiography; PT: prothrombin time; INR: international normalized ratio; PTT partial thromboplastin time; ECG: electrocardiography; e-FAST: extended focused assessment with sonography for trauma.

* A trial of HFNC or NIV, such as continuous positive airway pressure ventilation or bi-level positive airway pressure ventilation, may avoid the need for endotracheal intubation in selected patients. Patients with hemodynamic instability should receive appropriate interventions to treat shock prior to or during intubation. Refer to UpToDate content on HFNC, NIV, and rapid sequence intubation in children.

¶ Ancillary studies are determined by patient presentation and suspected type or types of shock present. Other laboratory and ancillary studies may also be indicated based upon the suspected underlying condition that is causing shock.

Δ Fluid volume should be calculated based upon ideal body weight (eg, 50^{th} percentile for age).

◇ When performed by trained and experienced physicians, bedside ECHO can provide rapid evidence of myocardial dysfunction, including dysfunction due to obstructive shock.

§ Patients with signs of fluid overload who continue to receive fluid boluses warrant close monitoring for respiratory and cardiac failure. The clinician should have a low threshold for endotracheal intubation and mechanical ventilation to treat pulmonary edema in these patients.

¥ Suggested vasoactive therapy depends upon type of shock and clinical findings; refer to UpToDate topics and graphics on management of shock in children.

Graphic 129655 Version 1.0

Stages of heart failure in infants and children and recommended therapy

Stage	Definition	Examples	Therapy
A	Patients with increased risk of developing HF, but with normal cardiac function and chamber size	▪ Exposure to cardiotoxic agents ▪ Family history of heritable cardiomyopathy ▪ Univentricular heart ▪ Congenitally corrected transposition of the great arteries	None
B	Patients with abnormal cardiac morphology or function, with no symptoms of HF, past or present	▪ Aortic insufficiency with LV enlargement ▪ History of anthracycline exposure with decreased LV systolic function	▪ ACE inhibitor for patients with systemic ventricular dysfunction
C	Patients with structural or functional heart disease, and past or current symptoms of HF	▪ Symptomatic cardiomyopathy ▪ Congenital heart defect with ventricular pump dysfunction	▪ ACE inhibitor and an aldosterone antagonist; oral diuretic therapy as needed for fluid overload; low-dose digoxin if needed for additional symptom relie ▪ After a few weeks of stability, a beta blocker is added in patients with persistent LV dilation and dysfunction
D	Patients with end-stage HF requiring specialized interventions	▪ Marked symptoms at rest despite maximal medical therapy	▪ Pharmacologic therapy consists of intravenous diuretics and/or inotropes ▪ Other interventions may include positive pressure ventilation, cardiac resynchronization therapy, mechanical circulatory support, and heart transplantation

HF: heart failure; LV: left ventricular; ACE: angiotensin-converting enzyme.

Original table modified for this publication. Rosenthal D, Chrisant MR, Edens E, et al. International Society for Heart and Lung Transplantation: Practice guidelines for management of heart failure in children. J Heart Lung Transplant 2004; 23:1313. Table

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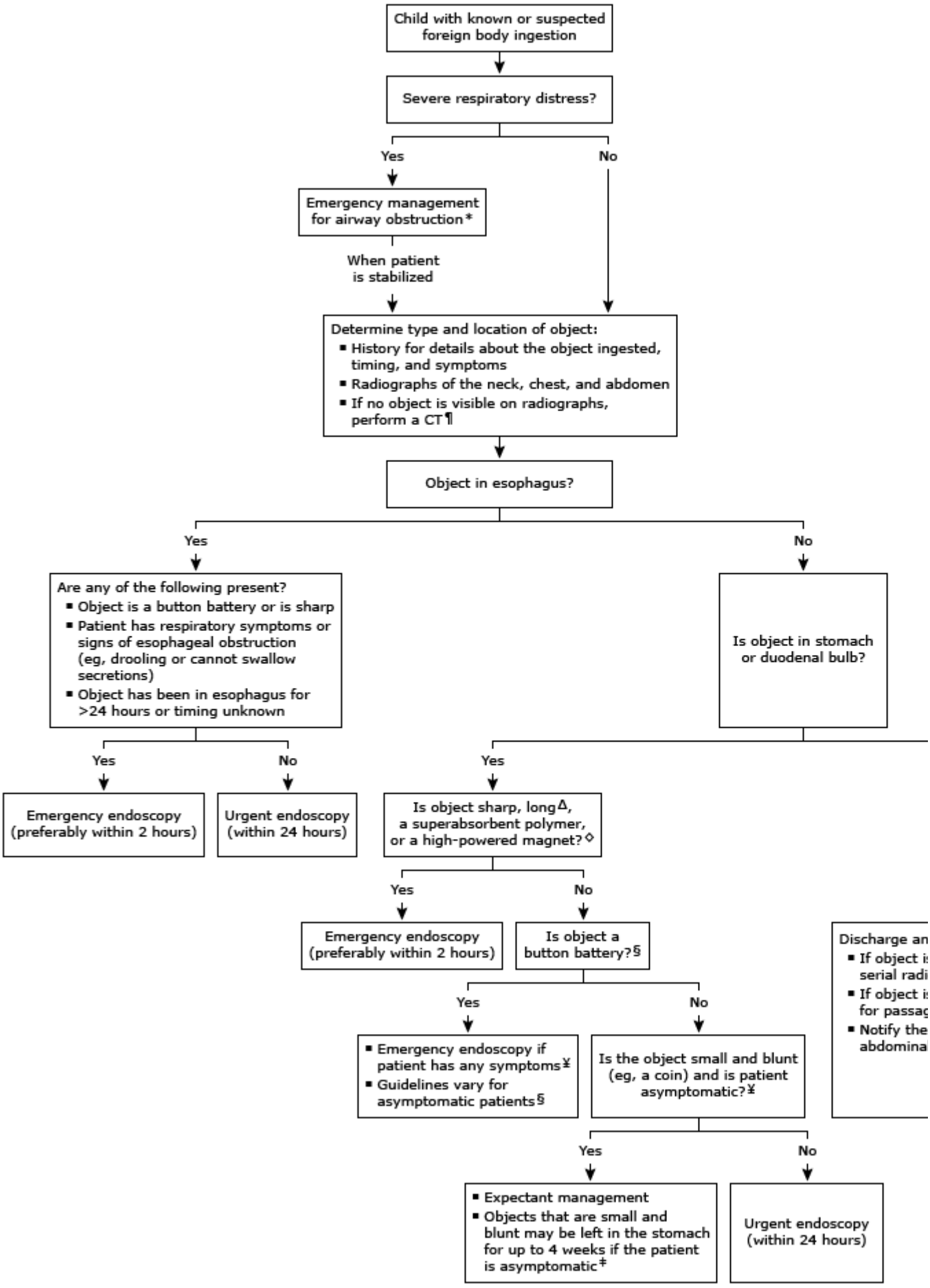
Graphic 60553 Version 7.0

Overview of the management of suspected foreign body ingestion in childre



CT: computed tomography.

* Refer to UpToDate content on the management of upper airway obstruction.

¶ In a child with negative conventional radiographs (no foreign body identified), a CT is not necessary if the child is asymptomatic and if suspected object is small (<2 cm) and blunt.

Δ In adults or older children, a long object is defined as one that is ≥5 cm; such objects should be removed from the stomach because they may not pass the duodenal sweep. There is no guidance for defining long objects in younger children. We generally remove an object from the stomach of a child <5 years if the object is longer than 2.5 cm.

◇ High-powered magnets are also known as "rare earth" or neodymium magnets. Management depends on the number of magnets present. Refer to UpToDate content and algorithms about magnet ingestion.

§ Guidelines vary for management of battery ingestion. Refer also to UpToDate topic on disc ("button") or cylindrical battery ingestion.

¥ Symptoms suggesting complications include vomiting, abdominal pain, fever, hematemesis, or melena.

‡ If an object remains in the stomach for more than 4 weeks, it should be removed because this means that the object is unlikely to pass beyond the pylorus. If the object is radiolucent and in the stomach, it is reasonable to remove the object initially if endoscopic expertise is readily available, especially if the object is larger than 2 cm, in which case it may not easily pass the pylorus. If observation is chosen, the parents should monitor stools until they determine that the object has passed and should notify the clinician if any symptoms develop.

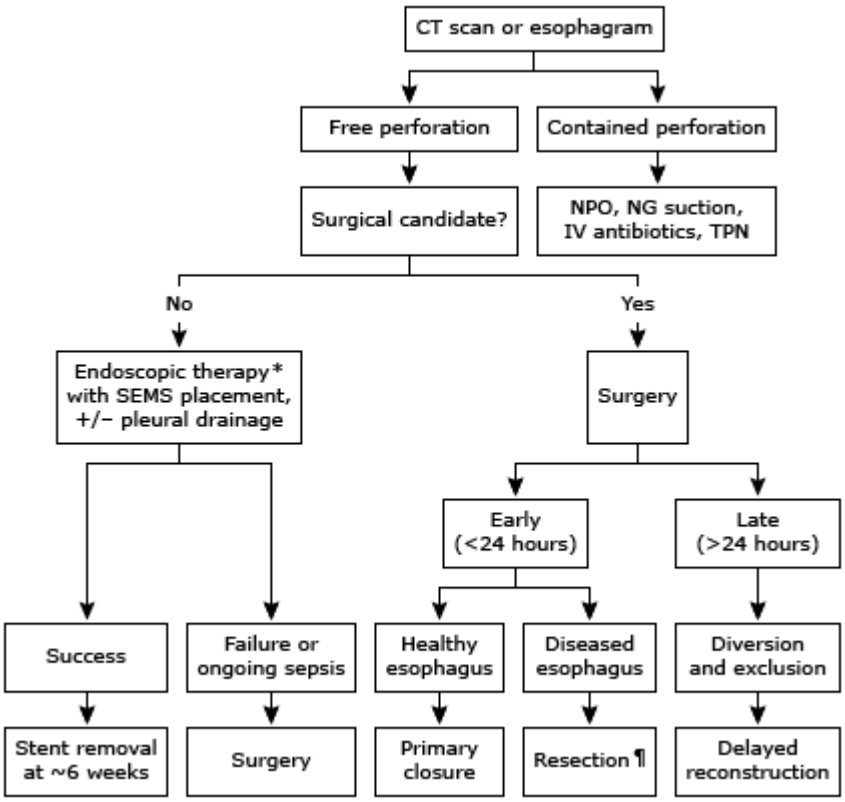
† For objects beyond the duodenal bulb that are radiopaque, monitor with serial radiographs (weekly for blunt objects, daily for sharp or long objects). Surgery is reserved for patients who develop complications (eg, obstruction, perforation) and for nonprogression of a foreign body (a blunt object that remains in the same location distal to the duodenum for more than 1 week or a sharp foreign body that does not advance radiographically for 3 consecutive days).

**If no object is identified, further evaluation depends on the patient's symptoms, type of object thought to be ingested, and likelihood that the ingestion occurred. If there is concern for aspiration of the object or for ingestion of an object that could lead to complications (large, long, or sharp), the evaluation might include diagnostic endoscopy or bronchoscopy, further imaging (eg, CT if not already done), and/or close observation.

Courtesy of Mark Gilger, MD.

Graphic 57363 Version 10.0

Management of esophageal perforation



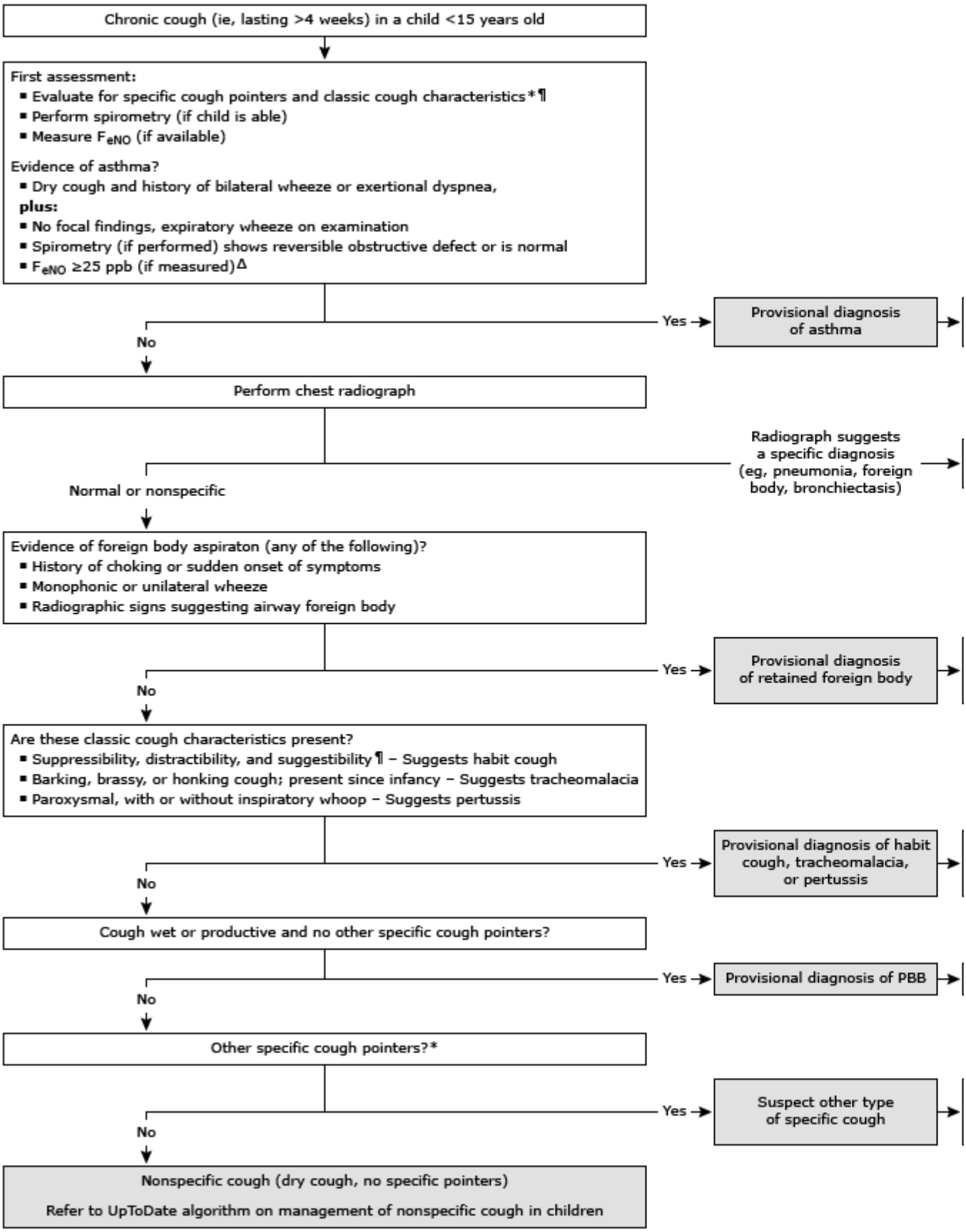
CT: computed tomography; NPO: nothing by mouth; NG: nasogastric; IV: intravenous; TPN: total parenteral nutrition; SEMS: self-expandable metallic stents.

* Endoscopy should only be performed in centers of expertise.

¶ Depending upon the type, location, severity of the esophageal disease, and size of the perforation.

Graphic 73379 Version 5.0

Algorithm for the evaluation of chronic cough in children



F_{eNO}: exhaled nitric oxide fraction; PBB: protracted bacterial bronchitis; TB: tuberculosis; CF: cystic fibrosis; PCD: primary ciliary dyskinesia.

* Specific cough pointers include[1]:

- Symptoms – Chronic wet/productive cough, chest pain, history suggestive of inhaled foreign body, dyspnea, exertional dyspnea, hemoptysis, failure to thrive, feeding difficulties (including choking/vomiting), cardiac or neurodevelopmental abnormalities, recurrent sinopulmonary infections, immunodeficiency, or epidemiologic risk factors for exposure to TB
- Signs – Respiratory distress, digital clubbing, chest wall deformity, or auscultatory crackles
- Tests – Chest radiographic changes (other than perihilar changes) or lung function abnormalities

Refer to UpToDate content on chronic cough in children.

¶ Habit cough (also known as tic cough) is typically absent at night or when distracted and may be honking or short/dry[2].

Δ F_{eNO} value ≥25 ppb with asthma symptoms supports a diagnosis of asthma[3].

◇ For diagnostic evaluation, refer to UpToDate content on pertussis and tracheomalacia. Tic (habit) cough is diagnosed based on characteristic symptoms.

References:

1. *Kantar A, Chang AB, Shields MD, et al. ERS statement on protracted bacterial bronchitis in children. Eur Respir J 2017; 50: 1602139.*
2. *Weinberger M, Hoegger M. The cough without a cause: Habit cough syndrome. J Allergy Clin Immunol 2016; 137:930.*
3. *Gaillard EA, Kuehni CE, Turner S, et al. European Respiratory Society clinical practice guidelines for the diagnosis of asthma in children aged 5-16 years. Eur Respir J 2021; 58:2004173.*

Graphic 113691 Version 4.0

Initial oral empiric antibiotics for outpatient treatment of pediatric community-acquired pneumonia

Age group	Empiric regimen
1 to 6 months	
Bacterial (not *Chlamydia trachomatis*)	**Infants <3 to 6 months of age with suspected bacterial pneumonia should be hospitalized**
C. trachomatis	Refer to UpToDate topic on *C. trachomatis* infections in the newborn
6 months to 5 years	
Typical bacterial*	Amoxicillin¶ 90 mg/kg per day in 2 or 3 divided doses (MAX 4 g/day), **or**
	Amoxicillin-clavulanate 90 mg/kg per day of the amoxicillin component in 2 or 3 divided doses (MAX 4 g/day amoxicillin component)
	For children with mild reactions to a penicillin and no features of an IgE mediated reaction△:
	▪ Amoxicillin 90 mg/kg per day in 2 or 3 divided doses (MAX 4 g/day), **or**
	▪ Amoxicillin-clavulanate 90 mg/kg per day of the amoxicillin componen in 2 or 3 divided doses (MAX 4 g/day amoxicillin component), **or**
	▪ An extended-spectrum cephalosporin, such as cefuroxime axetil 20 to 30 mg/kg per day in 2 divided doses (MAX 1 g/day), cefpodoxime 10 mg/kg per day in 2 divided doses (MAX 400 mg/day) cefdinir 14 mg/kg per day in 2 divided doses (MAX 600 mg/day) **in communities with a low rate of pneumococcal resistance to penicillin**
	For children with IgE-mediated or serious delayed reaction to a penicillin:
	▪ Levofloxacin◇ 16 to 20 mg/kg per day in 2 divided doses (MAX 750 mg/day), **or**
	▪ Clindamycin 30 to 40 mg/kg per day in 3 or 4 divided doses (MAX 1.8 g/day), **or**
	▪ Linezolid 30 mg/kg per day in 3 divided doses (MAX 1.8 g/day)
	In communities with a high rate of pneumococcal resistance to penicillin:
	▪ Levofloxacin◇ 16 to 20 mg/kg per day in 2 divided doses (MAX 750 mg/day), **or**
	▪ Linezolid 30 mg/kg per day in 3 divided doses (MAX 1.8 g/day)
≥5 years	

Mycoplasma pneumoniae or *Chlamydia pneumoniae*	Azithromycin[¶] 10 mg/kg on day 1 followed by 5 mg/kg daily for 4 more days (MAX 500 mg on day 1 and 250 mg thereafter), **or**
	Clarithromycin 15 mg/kg per day in 2 divided doses (MAX 1 g/day), **or**
	Erythromycin 40 to 50 mg/kg per day in 4 divided doses (MAX 2 g/day as base, 3.2 g/day as ethylsuccinate), **or**
	Doxycycline 4 mg/kg per day in 2 divided doses (MAX 200 mg/day), **or**
	Levofloxacin[◇] 8 to 10 mg/kg once daily for children 5 to 16 years (MAX 500 mg/day); 500 mg once daily for children ≥16 years, **or**
	Moxifloxacin[◇§] 400 mg once daily (≥18 years)
Typical bacterial[*]	Amoxicillin[¶] 90 mg/kg per day in 2 or 3 divided doses (MAX 4 g/day)
	For children with mild reactions to a penicillin and no features of an IgE mediated reaction[Δ]:
	▪ Amoxicillin 90 mg/kg per day in 2 or 3 divided doses (MAX 4 g/day), **or**
	▪ An extended-spectrum cephalosporin, such as cefuroxime axetil 20 to 30 mg/kg per day in 2 divided doses (MAX 1 g/day), cefpodoxime 10 mg/kg per day in 2 divided doses (MAX 400 mg/day), or cefdinir 14 mg/kg per day in 2 divided doses (MAX 600 mg/day) **in communities with a low rate of pneumococcal resistance to penicillin**
	For children with IgE-mediated or serious delayed reaction to a penicillin:
	▪ Levofloxacin[◇] 8 to 10 mg/kg once daily for children 5 to 16 years (MAX 750 mg/day); 750 mg once daily for children ≥16 years, **or**
	▪ Clindamycin 30 to 40 mg/kg per day in 3 or 4 divided doses (MAX 1.8 g/day), **or**
	▪ Linezolid 30 mg/kg per day in 3 divided doses (MAX 1.8 g/day) for children <12 years; 20 mg/kg per day divided in 2 doses (MAX 1.2 g/day) for children ≥12 years
	In communities with a high rate of pneumococcal resistance to penicillin:
	▪ Levofloxacin[◇] 8 to 10 mg/kg once daily for children 5 to 16 years (MAX 750 mg/day); 750 mg once daily for children ≥16 years, **or**
	▪ Linezolid 30 mg/kg per day divided in 3 doses (MAX 1.8 g/day) for children <12 years; 20 mg/kg per day divided in 2 doses (MAX 1.2 g/day) for children ≥12 years
Aspiration pneumonia	
Community-acquired	Amoxicillin-clavulanate 40 to 50 mg/kg per day in 2 or 3 divided doses (MAX 1.75 g/day amoxicillin component)

	For children with mild reactions to a penicillin and no features of an IgE mediated reaction[Δ]:
	▪ Amoxicillin-clavulanate 40 to 50 mg/kg per day in 2 or 3 divided doses (MAX 1.75 g/day amoxicillin component)
	For children with IgE-mediated or serious delayed reaction to amoxicillin:
	▪ Clindamycin 30 to 40 mg/kg per day divided in 3 or 4 doses (MAX 1.8 g/day)
	▪ Moxifloxacin[◇] 400 mg once daily (for ≥18 years)

IgE: immunoglobulin E; MAX: maximum.

* For the infant or child who is suspected to have bacterial community-acquired pneumonia and is unable to tolerate liquids at the time of presentation, a single initial dose of ceftriaxone (50 to 75 mg/kg) may be administered intramuscularly or intravenously before starting oral antibiotics.

¶ Preferred agent.

Δ The choice is individualized according to the drug allergy history and the ability to safely conduct an oral challenge if necessary. Refer to UpToDate content on penicillin allergy.

◇ In the United States, fluoroquinolones (eg, levofloxacin and moxifloxacin) are approved by the US Food and Drug Administration for community-acquired pneumonia for patients ≥18 years of age. However, they may be used in younger children if other antibiotics are inappropriate (eg, due to hypersensitivity or local antimicrobial resistance patterns).

§ Also covers typical bacterial pathogens.

Data from:

1. *McIntosh K. Community-acquired pneumonia in children. N Engl J Med 2002; 346:429.*
2. *Bradley JS, Byington CL, Shah SS, et al. The management of community-acquired pneumonia in infants and children older than 3 months of age: Clinical practice guidelines by the Pediatric Infectious Diseases Society and the Infectious Diseases Society of America. Clin Infect Dis 2011; 53:e25.*
3. *American Academy of Pediatrics. Tables of antibacterial drug dosages. In: Red Book: 2021-2024 Report of the Committee on Infectious Diseases, 32nd ed, Kimberlin DW, Barnett ED, Lynfield R, Sawyer MH (Eds), American Academy of Pediatrics, Itasca, IL 2021. p.876.*

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Contributor Disclosures

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