Approach to the adult with dyspnea in the emergency department

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INTRODUCTION

Dyspnea is the perception of an inability to breathe comfortably [1]. Acute dyspnea in the adult patient presents challenges in diagnosis and management. The emergency clinician must provide appropriate initial treatment for a potentially life-threatening illness while working through a wide differential diagnosis. Airway, breathing, and circulation are the emergency clinician's primary focus when beginning management of the acutely dyspneic patient. Once these are stabilized, further clinical investigation and treatment can proceed.

In this topic, we use the term "dyspnea" to encompass all disordered or inadequate breathing and provide a stepwise approach to the initial evaluation and management of the life-threatening and common causes of dyspnea in the adult, describe important historical and clinical findings that can help to narrow the differential diagnosis, and discuss common diagnostic studies. Detailed discussions of pathophysiology and focused evaluations of dyspnea are reviewed separately:

- (See "Physiology of dyspnea".)
- (See "Approach to the patient with dyspnea".)
- (See "Measures of oxygenation and mechanisms of hypoxemia".)
- (See "Maternal adaptations to pregnancy: Dyspnea and other physiologic respiratory changes".)

- (See "Cardiopulmonary exercise testing in the evaluation of unexplained dyspnea".)
- (See "Overview of pulmonary function testing in adults".)
- (See "Assessment and management of dyspnea in palliative care".)

PATHOPHYSIOLOGY

The respiratory system maintains homeostasis with respect to gas exchange and acid-base status. Derangements in oxygenation and/or acidemia can lead to dyspnea via a complex pathway generally involving stimulation of a variety of mechanoreceptors throughout the upper airway, lungs, and chest wall; and chemoreceptors at the carotid sinus and the medulla. The pathophysiology of dyspnea is discussed in detail separately. (See "Physiology of dyspnea" and "Measures of oxygenation and mechanisms of hypoxemia".)

EPIDEMIOLOGY

Dyspnea is a common chief complaint among emergency department (ED) patients, accounting for approximately four million visits (3 percent) annually in the United States [2]. Other dyspnea-related chief complaints (eg, cough, chest discomfort) comprise an additional 9 percent of ED visits.

In males and females over the age of 65, dyspnea and related problems were a major reason for ED visits [3].The most common diagnoses among older adult patients presenting to an ED with a complaint of acute shortness of breath and manifesting signs of respiratory distress (eg, respiratory rate >25, oxygen saturation [SpO_2] <93 percent) are decompensated heart failure, pneumonia, chronic obstructive pulmonary disease (COPD), pulmonary embolism (PE), and asthma [4].

OVERVIEW OF APPROACH

The primary goals for the emergency clinician faced with an acutely dyspneic patient are to:

- Optimize arterial oxygenation
- Determine the need for emergency airway management and ventilatory support
- Identify whether a life-threatening condition exists, such as:
    - Acute coronary syndrome

- Acute heart failure
- Arrhythmia
- Pericardial tamponade
- Pulmonary embolism (PE)
- Pneumonia or other infection
- Chronic obstructive pulmonary disease (COPD) exacerbation
- Asthma
- Angioedema and anaphylaxis
- Poisoning (eg, carbon monoxide)
- Trauma (eg, pneumothorax, hemothorax)

- Determine the most likely cause of dyspnea (table 1)

- Initiate treatment and stabilize if critically ill

Potential pitfalls in the approach of an acutely dyspneic emergency department patient include the following:

- Failure to secure the airway in a timely manner
- Failure to recognize and act on abnormal vital signs and signs of impending respiratory failure
- Over-reliance upon a single finding (physical examination or test result) to establish a diagnosis
- Failure to generate a proper differential diagnosis
- Failure to monitor the patient's clinical course
- Failure to consider carbon monoxide poisoning, methemoglobinemia, or PE
- Misinterpreting tachypnea, which may not represent a respiratory abnormality and may reflect nonpulmonary disease (eg, metabolic acidosis or impending herniation of the brainstem)
- Allowing patients with a tenuous respiratory status to leave the ED and deteriorate in the radiology suite or inpatient floor
- Discharging patients with inadequate follow-up or unclear instructions

INITIAL RAPID ASSESSMENT AND STABILIZATION

Clinical danger signs — All patients complaining of acute dyspnea should be screened for signs of imminent respiratory arrest or significant respiratory distress. A brief physical examination (including pulse oximetry) is often sufficient for this purpose.

- **Signs that portend imminent respiratory arrest include**:
  - Depressed mental status, which can occur with severe hypoxia or hypercarbia
  - Inability to maintain respiratory effort (bradypnea, poor inspiratory effort, or agonal respirations)
  - Cyanosis, which is uncommon and indicates severe hypoxia or methemoglobinemia
- **Signs suggestive of severe respiratory distress include:**
  - Retractions and the use of accessory muscles – Retractions occur with airway obstruction (eg, asthma, chronic obstructive pulmonary disease [COPD], foreign body) and can be seen in the suprasternal, intercostal, and subcostal areas [5]. They are an ominous sign suggesting extreme respiratory distress, fatigue of the respiratory muscles, and the potential for respiratory failure. Patients with neuromuscular disease may not manifest retractions due to muscle weakness, even in the face of severe respiratory compromise.
  - Brief, fragmented speech (patient is unable to answer questions with anything more than a few words), which can be quickly assessed by asking the patient to count to 10 in one breath.
  - Significant tachypnea (ie, greater than 25 breaths per minute). This cutoff is not absolute, and a respiratory rate of over 20 breaths per minute should prompt a timely evaluation.
  - Inability to lie supine – Many patients in respiratory distress sit bolt upright or in a tripod position. An exception is hepatopulmonary syndrome, where patients may breathe more comfortably when recumbent. (See "Hepatopulmonary syndrome in adults: Prevalence, causes, clinical manifestations, and diagnosis", section on 'Dyspnea'.)
  - Profound diaphoresis, which reflects extreme sympathetic stimulation associated with severe disease (eg, myocardial infarction, severe asthma flare, diastolic cardiac dysfunction).
  - Audible stridor or wheezing, which can represent upper airway obstruction or severe bronchospasm.
  - Dusky skin, which indicates poor perfusion or cyanosis.

- Agitation, somnolence, or other altered mental status in the dyspneic patient suggests severe hypoxia or hypercarbia. Patients with a depressed mental status from carbon dioxide (CO_2) retention may look comfortable and lackadaisical.

Emergency stabilization of patients with danger signs

General measures — The following measures are required in a patient with danger signs (see 'Clinical danger signs' above), who appears clinically ill, or is hypoxic (pulse oxygen saturation [SpO_2] <90 percent):

- **Provide supplemental oxygen** – For hypoxic patients with respiratory difficulty, provide 50 to 60 liters per minute (LPM) of oxygen via a nonrebreather mask. To deliver this much oxygen, open the flow meter valve until the indicator lies well beyond the 15 LPM mark. An SpO_2 of 94 percent is an appropriate target for most patients.

  Patients breathing 100% oxygen deliver five times as much oxygen to the alveoli per unit of ventilation as those breathing room air and, in the absence of parenchymal disease, can maintain a normal SpO_2 with only two or three breaths per minute. However, the best nonrebreather oxygen-delivery systems provide only 85% oxygen.

  For patients likely to require tracheal intubation, higher levels of oxygenation are necessary to increase their oxygen reserves for the apneic phase of rapid sequence intubation (RSI). Patients that are apneic or nearly so may require bag-mask ventilation. In certain situations, noninvasive ventilation may be used as a preoxygenation delivery system. Preoxygenation for RSI is reviewed in detail separately. (See "Rapid sequence intubation in adults for emergency medicine and critical care", section on 'Preoxygenation'.)

- **Establish intravenous (IV) access and obtain blood for laboratory measurements**. (See 'Complete blood count, serum chemistries' below.)

- **Begin continuous cardiac and pulse oximetry monitoring.**

- **Obtain airway management equipment and request a respiratory therapist to the bedside.**

- **Continue focused history and examination** – Assess for a potentially difficult airway and search for rapidly reversible causes (anaphylaxis, tension pneumothorax, pericardial tamponade, upper airway obstruction).

- **Determine the need for airway management and ventilatory support** – The initial decision to provide noninvasive or invasive ventilatory support is made based upon clinical grounds, **not** laboratory values. (See "The decision to intubate" and 'Options for oxygenation or ventilatory support' below.)

- **Obtain an electrocardiogram (ECG)** – (See 'Electrocardiogram' below and "Diagnosis of acute myocardial infarction" and "Clinical presentation, evaluation, and diagnosis of the nonpregnant adult with suspected acute pulmonary embolism".)

- **Obtain a portable chest radiograph (CXR)** – However, some life-threatening causes of dyspnea may not manifest any abnormality on CXR. (See 'Plain chest radiograph' below.)

- **Perform bedside ultrasound (if available)** – Emergency physician-performed ultrasound can rapidly identify pericardial effusions, pulmonary edema, and clinically significant pneumothorax. (See 'Role of bedside ultrasound' below.)

- **Optimize hemodynamic physiology** – Control the rate or rhythm of a tachydysrhythmia and improve right ventricular filing pressures if needed (eg, IV fluids, vasopressors). When volume overload is suspected, can administer diuretics and/or preload and afterload reduction (eg, nitrates) depending on underlying physiology. (See "Overview of the acute management of tachyarrhythmias" and "Evaluation and management of suspected sepsis and septic shock in adults", section on 'Volume' and "Right heart failure: Causes and management", section on 'Optimization of volume status' and "Use of vasopressors and inotropes".)

- **Maintain appropriate infection control measures** – If tuberculosis, coronavirus disease 2019 (COVID-19), or other highly transmittable infections are a concern, appropriate infection control measures are crucial (table 2). (See "COVID-19: General approach to infection prevention in the health care setting" and "COVID-19: Infection prevention for persons with SARS-CoV-2 infection", section on 'Infection prevention in the health care setting'.)

Options for oxygenation or ventilatory support — In a patient that requires ventilatory assistance to overcome an infraglottic challenge (eg, bronchospasm or parenchymal disease) or nonpulmonary disease (eg, neuromuscular disease), there are noninvasive and invasive strategies:

- **High-flow nasal cannula (HFNC)** – The main use of HFNC is to provide a relatively high fraction of inspired oxygen (FiO_2) to patients with severe nonhypercapnic hypoxemic respiratory failure. HFNC is often used in patients with hypoxia due to COVID-19. Oxygen

flows from the source, is heated and humidified, and is then delivered to the patient through a wide-bore nasal cannula (picture 1). (See "Heated and humidified high-flow nasal oxygen in adults: Practical considerations and potential applications" and "COVID-19: Respiratory care of the nonintubated hypoxemic adult (supplemental oxygen, noninvasive ventilation, and intubation)", section on 'Patients with requirements for advanced respiratory support'.)

- **Noninvasive ventilation (NIV)** – NIV with a mask delivering continuous positive airway pressure (CPAP) or bilevel positive airway pressure (BiPAP) can increase minute ventilation, reduce the work of breathing, recruit alveoli, and improve hemodynamics. Contraindications to NIV include inability to protect the airway, significant airway obstruction, severely depressed mental status, active vomiting, hemodynamic instability unlikely to stabilize with NIV or other appropriate intervention (eg, cardioversion), facial trauma, and recent facial, esophageal, or gastric bypass surgery. These are discussed in detail elsewhere. (See "Noninvasive ventilation in adults with acute respiratory failure: Benefits and contraindications" and "Treatment of acute decompensated heart failure: General considerations".)

- **Mechanical ventilation** – Endotracheal intubation and controlled mechanical ventilation should be pursued aggressively when ventilatory support is needed and NIV is **not** expected to improve outcomes, such as for acute exacerbations of asthma and diseases that do not respond rapidly to medical therapy (eg, pneumonia and acute respiratory distress syndrome [ARDS]). (See "Acute exacerbations of asthma in adults: Home and office management" and "Treatment of community-acquired pneumonia in adults who require hospitalization" and "Acute respiratory distress syndrome: Fluid management, pharmacotherapy, and supportive care in adults" and "Mechanical ventilation of adults in the emergency department".)

- **Extracorporeal membrane oxygenation (ECMO)** – In patients with hypoxemic respiratory failure who are mechanically ventilated and not responding to conventional measures, ECMO is an option (if available), but appropriate services need to be consulted early to mobilize resources. (See "Extracorporeal life support in adults: Management of venovenous extracorporeal membrane oxygenation (V-V ECMO)".)

Patients with inadequate respiratory effort — Inadequate respiratory effort can occur from respiratory fatigue (eg, asthma, COPD, pneumonia) or the underlying disease process (eg, neuromuscular disease). (See "Mechanisms, causes, and effects of hypercapnia".)

- **Identifying and monitoring hypercapnic respiratory failure** – A venous blood gas may be useful in the assessment of the patient presumed to be somnolent from CO_2 retention. The partial pressure of carbon dioxide ($PaCO_2$) should be low in the acutely dyspneic patient, who is usually hyperventilating. A normal or elevated CO_2 in the setting of dyspnea and tachypnea portends respiratory failure.

  Capnography (ie, end-tidal CO_2 [$EtCO_2$]) can identify CO_2 retention and provide dynamic monitoring of ventilatory status in patients with acute respiratory distress. By measuring $EtCO_2$ and respiratory rate with each breath, capnography provides instantaneous feedback, while trends enable the clinician to determine whether the patient's ventilation is worsening despite treatment (increasing $EtCO_2$), stabilizing (stable $EtCO_2$), or improving (decreasing $EtCO_2$). The $EtCO_2$ may be falsely low due to dilution in high-flow open systems (eg, nonrebreather masks, CPAP) [6]. (See "Carbon dioxide monitoring (capnography)".)

- **Initial management** – Initial therapies are targeted towards reversing the etiology, if possible, and ventilatory support. (See "The evaluation, diagnosis, and treatment of the adult patient with acute hypercapnic respiratory failure".)

  The following disease processes can lead to inadequate respiratory effort and often require ventilatory support:

  - **Primary pulmonary disease** – COPD, asthma, pneumonia, interstitial fibrosis, and others can lead to hypercapnic respiratory failure from hypoventilation, increased dead space, decreased respiratory drive, and respiratory muscle fatigue.

  - **Sepsis** – Severe sepsis often causes respiratory compromise secondary to tachypnea and respiratory muscle fatigue, which may stem from underlying pneumonia, compensation for metabolic acidosis, or some other process.

    Intubation and mechanical ventilation can support the increased work of breathing and are often performed as part of the resuscitation for the anticipated clinical course. (See "Evaluation and management of suspected sepsis and septic shock in adults", section on 'Immediate evaluation and management'.)

  - **Stroke** – Although dyspnea is not the chief complaint of patients with an acute stroke, a number of respiratory abnormalities may result from a sufficiently severe injury or one affecting regions involved in respiration. Such abnormalities may include aspiration pneumonia, neurogenic pulmonary edema, and various abnormal respiratory patterns, including apnea. (See "Complications of stroke: An overview", section on 'Pulmonary complications'.)

- **Neuromuscular disease** – Several neuromuscular diseases, including multiple sclerosis, Guillain-Barré syndrome, myasthenia gravis, amyotrophic lateral sclerosis, West Nile virus, and enterovirus D68 (seen primarily in children), can cause weakness of the respiratory muscles, leading to acute respiratory failure. Botulism may cause generalized descending muscle weakness involving the respiratory muscles. (See "Manifestations of multiple sclerosis in adults" and "Guillain-Barré syndrome in adults: Pathogenesis, clinical features, and diagnosis" and "Clinical features of amyotrophic lateral sclerosis and other forms of motor neuron disease" and "Myasthenic crisis" and "Botulism".)

Targeted measures based on signs and symptoms — The definitive stabilization of respiratory failure is endotracheal intubation and mechanical ventilation. However, there are many processes where targeted intervention may improve respiratory distress and avoid mechanical ventilation, which are presented below based on signs and symptoms:

Stridor — Stridor occurs when there is airway obstruction. Inspiratory stridor suggests obstruction above the vocal cords (eg, foreign body, epiglottitis, angioedema). Expiratory stridor or mixed inspiratory and expiratory stridor suggests obstruction below the vocal cords (eg, croup, bacterial tracheitis, foreign body).

Patients with signs of upper airway obstruction may need emergency airway management, although angioedema from anaphylaxis often improves with medical therapy (eg, epinephrine, antihistamines, and glucocorticoids). The decision to secure the airway and optimal approach (ie, endotracheal intubation, fiberoptic, or emergency cricothyrotomy) are discussed in detail elsewhere. (See "Rapid sequence intubation in adults for emergency medicine and critical care" and "Approach to the difficult airway in adults for emergency medicine and critical care" and "Emergency cricothyrotomy (cricothyroidotomy) in adults".)

Potential causes of airway obstruction with targeted interventions include:

- **Tracheal foreign objects** – Food, coins, bones, dentures, and medication tablets are common causes of upper and lower airway obstruction, but a multitude of other objects can be placed in the mouth and become lodged in the airways. Foreign body obstruction is an uncommon cause of acute dyspnea in adults but relatively common in children.

  In a conscious adult, chest blows and abdominal thrusts are first-aid maneuvers that can be attempted prehospital or the ED. The procedure is described elsewhere. (See "Airway foreign bodies in adults", section on 'Foreign body removal'.)

Once the patient is sedated, foreign body retrieval with Magill forceps or endotracheal intubation with an attempt to push the foreign body into a major bronchus can be attempted. Management is discussed in detail elsewhere. (See "Airway foreign bodies in adults".)

- **Angioedema** – Angioedema can cause significant swelling of the lips, tongue, posterior pharynx, and most dangerously the larynx; this can occur over minutes to hours and may cause severe dyspnea. Etiologies include idiopathic, allergic (ie, anaphylaxis), medication-related (eg, nonsteroidal antiinflammatory drug [NSAID], angiotensin-converting enzyme [ACE] inhibitor, angiotensin receptor blocker), and complement-related (eg, C1-esterase inhibitor deficiency or a nonfunctional allele). Patients who receive IV tissue plasminogen activator (tPA) for acute ischemic stroke are also at risk for developing angioedema, which tends to be hemilingual and contralateral to the ischemic hemisphere [7].

  C1 inhibitors and bradykinin B2-receptor antagonists are targeted therapies for hereditary angioedema and reviewed separately. (See "Hereditary angioedema: Acute treatment of angioedema attacks" and "An overview of angioedema: Pathogenesis and causes".)

- **Anaphylaxis** – Anaphylaxis may cause severe swelling of the upper airway and tongue, and possibly airway occlusion. Bronchospasm may also be prominent. Symptoms and signs, which are often triggered by foods, insect bites, and various medications, develop over minutes to hours and may include skin and mucosal findings (eg, hives, flushing, oropharyngeal swelling), respiratory compromise (eg, wheezing, stridor, hypoxia), cardiovascular compromise (eg, hypotension, tachycardia, syncope), and gastrointestinal complaints (eg, abdominal pain, vomiting, and diarrhea).

  Intramuscular or IV epinephrine often improves symptoms if given promptly after the onset of symptoms. Since anaphylaxis has a variable course, preparations for endotracheal intubation or emergency cricothyrotomy should be ongoing while assessing clinical response. Airway management and medications for anaphylaxis are discussed in detail elsewhere. (See "Anaphylaxis: Emergency treatment".)

Unilateral diminished breath sounds — Diminished breath sounds can be caused by anything that prevents air from entering the lungs.

- **Pneumothorax** – Any simple pneumothorax can develop into a life-threatening tension pneumothorax. Patients in extremis with a suggestive history and examination findings consistent with a tension pneumothorax (eg, hypotension, distended neck veins, unilateral diminished or absent breath sounds) should be treated with immediate needle

decompression followed by a tube or catheter thoracostomy. (See "Thoracostomy tubes and catheters: Placement techniques and complications".)

- **Pleural effusion, hemothorax** – Respiratory distress from a large pleural effusion or hemothorax can be improved with a large-volume thoracentesis or tube/catheter thoracostomy, respectively. (See "Large volume (therapeutic) thoracentesis: Procedure and complications" and "Thoracostomy tubes and catheters: Indications and tube selection in adults and children", section on 'Hemothorax'.)

Rales — Rales (crackles) suggest the presence of intra-alveolar fluid, which can be caused by many processes. NIV can be attempted to improve oxygenation and preoxygenate for rapid sequence intubation, but preparations for endotracheal intubation should be ongoing while assessing clinical response. With the exception of pulmonary edema, patients with hypoxemic respiratory failure from alveolar processes are less likely to benefit from NIV and will often require mechanical ventilation. (See 'Plain chest radiograph' below and "Noninvasive ventilation in adults with acute respiratory failure: Benefits and contraindications" and "Mechanical ventilation of adults in the emergency department".)

The following alveolar processes that present with hypoxic respiratory failure have targeted interventions:

- **Acute cardiogenic pulmonary edema (ACPE)** – The sudden onset and rapid progression of pulmonary edema can be caused by ischemia, arrhythmia, or hypertensive crisis (ie, "flash pulmonary edema"). Patients typically present with acute-onset dyspnea, severe hypertension, diaphoresis, respiratory distress, and diffuse rales. Respiratory effort typically improves quickly with NIV and administration of agents to reduce afterload. (See "Noninvasive ventilation in adults with acute respiratory failure: Benefits and contraindications" and "Evaluation and treatment of hypertensive emergencies in adults", section on 'Cardiac emergencies'.)

- **COVID-19 pneumonia** – In patients with hypoxemic respiratory failure due to suspected or confirmed infection with COVID-19, awake pronation and high-flow oxygen delivered via nasal cannulae prior to mechanical ventilation often corrects hypoxia. (See "COVID-19: Respiratory care of the nonintubated hypoxemic adult (supplemental oxygen, noninvasive ventilation, and intubation)".)

- **Organophosphate poisoning** – Acute toxicity from organophosphorus agents presents with manifestations of cholinergic excess (bradycardia, salivation, lacrimation, vomiting, diarrhea, bronchorrhea, miosis) and should be recognizable on rapid examination. Management typically involves endotracheal intubation and empiric antidotal therapy with

atropine, pralidoxime, and benzodiazepines. (See "Organophosphate and carbamate poisoning".)

Hypoxia with clear lungs — Causes with targeted interventions include:

- **Pulmonary embolism (PE)** – The initial approach to patients with suspected massive PE should focus upon stabilizing the patient while clinical evaluation and definitive diagnostic testing are ongoing. Anticoagulation should be initiated even prior to confirming the diagnosis if risk-benefit regarding suspicion of PE and risk of bleeding appear favorable**.** Computed tomography pulmonary angiography (CTAP) is the first-choice diagnostic test and can discover alternate causes of hypoxia [8]. In the patient too unstable to travel to radiology, bedside emergency physician-performed ultrasound findings of right heart strain (eg, increased right ventricle size, decreased right ventricle function, McConnell's sign) or proximal deep vein thrombosis support the diagnosis. In the patient with inadequate oxygenate or hemodynamic instability, thrombolytic therapy or embolectomy are indicated. (See "Clinical presentation, evaluation, and diagnosis of the nonpregnant adult with suspected acute pulmonary embolism" and "Clinical presentation and diagnosis of the nonpregnant adult with suspected deep vein thrombosis of the lower extremity", section on 'Diagnostic ultrasonography suspected first DVT' and "Treatment, prognosis, and follow-up of acute pulmonary embolism in adults" and "Approach to thrombolytic (fibrinolytic) therapy in acute pulmonary embolism: Patient selection and administration".)

- **Cardiac tamponade** – Cardiac tamponade can be caused by trauma, malignancy, uremia, drugs, or infection and can present with acute dyspnea. The classic triad of hypotension, distended neck veins, and muffled heart tones suggest the diagnosis but are often absent. The ECG generally shows sinus tachycardia and low voltage and only rarely reveals electrical alternans. Bedside cardiac ultrasound can identify pericardial effusion and often diagnose tamponade (movie 1).

  The unstable patient requires emergency pericardiocentesis. (See "Emergency ultrasound in adults with abdominal and thoracic trauma", section on 'Pericardial and limited cardiac examination' and "Cardiac tamponade" and "Emergency pericardiocentesis".)

- **Methemoglobinemia** – This abnormal hemoglobin variant can present with dyspnea, cyanosis, and an artifactual pulse oximetry reading of 85 to 88 percent that does not improve with supplemental oxygen. The diagnosis is confirmed by methemoglobin blood gas measurement (either arterial or venous) and treated with methylene blue. (See "Methemoglobinemia".)

Wheezing — Wheezing suggests obstruction below the level of the trachea and is found with asthma, anaphylaxis, a foreign body in a mainstem bronchus, or a fixed lesion such as a tumor. Wheezing can also occur from pulmonary edema (ie, "cardiac asthma") and is not pathognomic for bronchospasm.

- **COPD** – Exacerbations of COPD can present with acute shortness of breath, often exacerbated by a respiratory infection.

  Respiratory distress from a COPD exacerbation typically improves quickly with NIV in addition to medications (eg, beta adrenergic agonists, muscarinic antagonists, glucocorticoids). In patients who fail to improve with standard COPD treatment, re-evaluate for a PE, which may be responsible for up to 25 percent of apparent "COPD exacerbations." (See "COPD exacerbations: Management" and "Noninvasive ventilation in adults with acute respiratory failure: Benefits and contraindications".)

- **Asthma** – Asthma exacerbations generally present with dyspnea and wheezing. Signs of severe disease include the use of accessory muscles, brief fragmented speech, profound diaphoresis, agitation, and failure to respond to aggressive treatment. The chest examination may be silent in the face of severe bronchospasm. Extreme fatigue, cyanosis, and depressed mental status portend imminent respiratory arrest.

  Management includes beta adrenergic agonists, muscarinic antagonists, glucocorticoids, and other therapies discussed in detail elsewhere. (See "Acute exacerbations of asthma in adults: Emergency department and inpatient management" and "Airway management in acute severe asthma for emergency medicine and critical care".)

ECG changes, tachycardia, or associated chest pain — Causes with targeted interventions include:

- **Acute coronary syndrome (ACS)** – Older adults and patients with diabetes who have a myocardial infarction may present with dyspnea as their sole symptom.

  An ECG showing ST-elevation myocardial infarction requires rapid reperfusion therapy (eg, percutaneous coronary intervention, fibrinolysis). (See "Diagnosis of acute myocardial infarction" and "Overview of the acute management of ST-elevation myocardial infarction".)

- **Cardiac arrhythmia** – Cardiac rhythm abnormalities, such as atrial flutter, atrial fibrillation, and tachyarrhythmias (eg, supraventricular tachycardia [SVT] and ventricular tachycardia) can result in dyspnea. Such abnormalities may stem from underlying disease,

including myocardial ischemia or cardiac decompensation due to the elevated heart rate or uncoordinated contractions.

The subjective feeling of dyspnea often improves with slowing down the rate of a tachycardic rhythm. (See "Overview of the acute management of tachyarrhythmias" and "Atrial fibrillation: Overview and management of new-onset atrial fibrillation" and "Atrioventricular nodal reentrant tachycardia" and "Wide QRS complex tachycardias: Approach to management".)

EVALUATION OF STABLE AND STABILIZED PATIENTS

In dyspneic patients who are stable on presentation, obtain history, physical examination, and diagnostic testing while concurrently providing therapeutic interventions as indicated. In those who were unstable on presentation, additional history, physical examination, diagnostic testing, and therapeutic interventions are generally necessary after initial stabilization.

History — The history is critical to the evaluation of the acutely dyspneic patient but can be difficult to obtain when the patient has difficulty speaking and the clinician must concentrate on ensuring that the patient maintains adequate oxygenation and ventilation. Relevant history can be obtained from the patient, emergency medical services (EMS) providers, family and friends, pharmacists, and primary care clinicians. The following details should be obtained whenever possible:

- **General historical features** – Ask about the events leading up to the episode, particularly any recent symptoms or specific triggers for the acute dyspnea. As examples, noncompliance with medications or diet may lead to an episode of acute decompensated heart failure (ADHF), exposure to cold or an allergen may trigger an asthma flare, acute dyspnea immediately following a meal suggests an allergic reaction, a new productive cough suggests a pulmonary infection, recent surgery or immobilization increases the risk for pulmonary embolism (PE), and recent trauma may have caused a pneumothorax or pulmonary contusion.

- **Past history** – Determine whether the problem is new or recurring. Ask about pre-existing medical conditions such as asthma, chronic obstructive pulmonary disease (COPD), prior deep vein thrombosis (DVT) or PE, or ischemic heart disease and whether the patient has experienced similar acute episodes before. Ask about vaccination status, such as against SARS-CoV-2 infection (COVID-19), influenza, and pneumococcus. If dyspnea resembles

prior episodes, the current problem is often an exacerbation of a pre-existing illness. The medical record and medication list can provide important diagnostic clues.

- **Prior intubation** – Patients with a history of endotracheal intubation for medical conditions have a higher risk for severe disease and the need for subsequent intubation. As an example, patients who have required intubation for a severe asthma exacerbation are at increased risk for subsequent episodes of near-fatal asthma attacks. Similarly, prior tracheostomy indicates significant prior illness, in addition to potential anatomic difficulties with future endotracheal intubation. (See "Identifying patients at risk for fatal asthma".)

- **Time course** – Ask whether the dyspnea developed suddenly or gradually since time course can help differentiate etiology (table 3).

- **Severity** – Ask how severe the dyspnea is compared with the patient's baseline. It is important to distinguish an acute presentation from an acute-on-chronic presentation.

- **Chest pain** – Chest pain associated with dyspnea occurs with a number of diseases, including acute coronary syndrome (ACS), pneumothorax, and PE. Pleuritic chest pain suggests PE or pleural-based disease while non-respirophasic pain suggests ACS. Patients with ACS or PE can complain of dyspnea alone. (See "Evaluation of the adult with chest pain in the emergency department".)

- **Trauma** – Ask about any injury sustained the day of presentation to several weeks prior. Injury to the airway, neck, chest wall, lungs, heart, mediastinal structures, or abdomen can lead to dyspnea. Acute symptoms may not manifest until a day or longer following trauma. (See "Initial evaluation and management of blunt thoracic trauma in adults".)

- **Fever** – Fever can be associated with an infection, hypersensitivity pneumonitis, aspiration pneumonitis, PE, or poisoning (eg, aspirin overdose can cause hyperthermia and tachypnea). (See "Salicylate (aspirin) poisoning: Clinical manifestations and evaluation".)

- **Paroxysmal nocturnal dyspnea (PND)** – Keep in mind that PND is **not** specific for ADHF. Patients with COPD may present with a similar history.

- **Hemoptysis** – Hemoptysis is associated with a number of conditions, including PE, severe valvular disease (eg, mitral stenosis), tuberculosis, malignancy, bronchitis, pneumonia, pulmonary contusion, and the effects of anticoagulants (eg, alveolar hemorrhage). (See "Evaluation and management of life-threatening hemoptysis".)

- **Cough and sputum** – Purulent sputum suggests bronchitis or pneumonia, and pink frothy sputum suggests ADHF. A nonproductive cough is a nonspecific symptom and may be associated with asthma, heart failure, respiratory infection, or PE.

- **Medications** – A medication list provides information about chronic or acute illness. Ask about recent dosing changes, new medications, and adherence (in a nonjudgmental manner). Oral combined estrogen-progestin contraceptive use and estrogen replacement therapy increases risk of PE. (See "Menopausal hormone therapy: Benefits and risks", section on 'Venous thromboembolism'.)

- **Family history** – A family history of hypercoagulable disorders, deep vein thrombosis, PE, or premature atherosclerotic disease should increase suspicion for these illnesses.

- **Tobacco and drugs** – Ask about tobacco and drug use in a nonjudgmental manner. Current and prior tobacco use increases the risk for many chronic conditions (eg, COPD, malignancy). People who inject drugs are at increased risk of pulmonary complications such as pneumonia, empyema, septic emboli, and noncardiogenic pulmonary edema. Smoking crack cocaine can cause diffuse alveolar damage and hemorrhagic alveolitis (ie, "crack lung") or pneumothorax. E-cigarettes and other vaping products have been associated with "**E**-cigarette or **v**aping product use-**a**ssociated **l**ung **i**njury (EVALI)." Acute respiratory distress syndrome (ARDS) can occur following overdose of various drugs (eg, aspirin, cocaine, opioids, phenothiazines, and tricyclic antidepressants). (See "Overview of pulmonary disease in people who inject drugs" and "E-cigarette or vaping product use-associated lung injury (EVALI)" and "Pulmonary complications of cocaine use" and "Acute respiratory distress syndrome: Epidemiology, pathophysiology, pathology, and etiology in adults", section on 'Drugs and alcohol'.)

- **Psychiatric conditions** – Ask about existing psychiatric diagnoses, history of panic attacks, and history of similar episodes that may have had unrevealing evaluations. Psychogenic causes of acute dyspnea are diagnoses of exclusion in the ED; organic causes **must** be thoroughly considered first. Nevertheless, among patients younger than age 40 with no medical conditions, psychogenic dyspnea (eg, anxiety, panic attack) may be the cause in a sizable minority of patients [9,10].

- **Pregnancy** – A number of physiologic changes occur during pregnancy that affect respiratory function, including an increase in minute ventilation, a decrease in functional residual capacity, a decrease in hematocrit, and elevation of the diaphragm. Approximately two-thirds of pregnant individuals experience dyspnea during the course of normal pregnancy.

However, pregnancy also increases the risk for several potentially life-threatening conditions that may manifest with dyspnea, notably PE. Pulmonary edema may be identified in the setting of a number of diseases associated with pregnancy, including preeclampsia, amniotic fluid embolism, and cardiomyopathy. (See "Maternal adaptations to pregnancy: Dyspnea and other physiologic respiratory changes" and "Deep vein thrombosis in pregnancy: Epidemiology, pathogenesis, and diagnosis" and "Eclampsia" and "Amniotic fluid embolism" and "Peripartum cardiomyopathy: Etiology, clinical manifestations, and diagnosis" and "Management of heart failure during pregnancy".)

Physical examination — Perform a more thorough physical examination after the initial rapid screen and any necessary stabilization is completed. Important signs are discussed below and in the accompanying table (table 4). An unremarkable pulmonary and cardiac examination does **not** rule out significant disease. As examples, the sensitivity and specificity of the pulmonary examination are limited for making the diagnosis of pneumonia or ADHF [11-15].

- **Respiratory rate** – Patients with serious underlying disease may have a fast, normal, or slow respiratory rate. As an example, patients with PE may have a respiratory rate in the normal range. Measurements of the respiratory rate obtained during triage may not be accurate [16,17].

- **Pulse oximetry** – Pulse oximetry provides crucial information about arterial oxygenation and should be checked upon presentation to the ED in all patients complaining of dyspnea. Examining the waveform, if feasible, can help ensure accuracy since many conditions can cause erroneous readings (table 5). Standard pulse oximeters are **not** accurate in the setting of hypothermia, shock, carbon monoxide poisoning, and methemoglobinemia. Pulse oximetry may miss hypoxia, especially in patients with darkly pigmented skin [18]. (See "Pulse oximetry".)

  In general, healthy individuals demonstrate an oxygen saturation (SpO_2) of 95 percent or greater. Older adults and patients with obesity or who smoke heavily often maintain saturation between 92 and 95 percent, while patients with severe chronic lung disease may have baseline saturation below 92 percent. In the setting of acute dyspnea, saturations that are lower than expected or lower than a patient's known baseline should be investigated and explained.

  In a patient who complains of dyspnea on exertion, especially who is not hypoxic at rest, obtaining pulse oximetry while ambulating often provides useful data that can inform further testing and disposition. Worsening oxygen desaturation with ambulation can often change an anticipated discharge to an admission.

- **Breath sounds during audible and lung auscultation**
  - **Stridor** is a sign of airway obstruction. Inspiratory stridor suggests obstruction above the vocal cords (eg, foreign body, epiglottitis, angioedema). Expiratory stridor or mixed inspiratory and expiratory stridor suggests obstruction below the vocal cords (eg, croup, bacterial tracheitis, foreign body).
  - **Wheezing** represents bronchoconstriction, bronchospasm, or obstruction below the level of the trachea and is found with asthma, anaphylaxis, a foreign body in a mainstem bronchus, ADHF, or a fixed lesion such as a tumor.
  - **Crackles (rales)** are found in the presence of interalveolar fluid, as seen with pneumonia or ADHF. They can also occur with pulmonary fibrosis. However, the absence of crackles does not rule out the presence of pneumonia, ADHF, or pulmonary fibrosis [11].
  - **Diminished breath sounds** are caused by processes that prevent air from entering the lungs, such as severe COPD, severe asthma, pneumothorax, tension pneumothorax, pleural effusion, hemothorax, and others.
- **Cardiovascular signs**
  - **Irregular heart rhythm** – A dysrhythmia may be a response to the underlying disease (eg, tachycardia in the setting of PE) or the cause of dyspnea itself (eg, atrial fibrillation).
  - **Heart murmurs** – A murmur may be physiologic or represent ADHF or diseased or otherwise compromised cardiac valves. Acute-onset dyspnea with evidence of cardiac ischemia and new systolic murmur suggests papillary muscle or interventricular septal rupture and warrants emergency echocardiogram since surgical correction is needed. (See "Auscultation of cardiac murmurs in adults" and "Acute myocardial infarction: Mechanical complications".)
  - **Third and fourth heart sounds** – The S3 heart sounds is heard with left ventricular systolic dysfunction, especially in the setting of ADHF. The S4 heart sound is heard with left ventricular dysfunction and may be present with severe hypertension, aortic stenosis, hypertrophic cardiomyopathy, ischemic heart disease, or acute mitral regurgitation. (See "Auscultation of heart sounds", section on 'Third (S3) and fourth (S4) heart sounds'.)

- **Muffled or distant heart sounds** – Quieter heart sounds occur with pericardial effusions but must be interpreted in the context of the overall clinical setting.

- **Jugular venous distension** – Elevated jugular venous pressure may be present with ADHF, tension pneumothorax, cardiac tamponade, or any other cause of elevated intrathoracic pressure that prevents right heart filling. It can be assessed by observing the veins of the neck (movie 2) or examining hepatojugular reflux. (See "Examination of the jugular venous pulse", section on 'How to examine the jugular venous pulse'.)

- **Pulsus paradoxus** – Pulsus paradoxus can occur when right heart function is compromised such as in severe asthma, PE, or cardiac tamponade (table 6). It is obtained by measuring the patient's systolic blood pressure after a normal exhalation, then having the patient inhale normally and determining the systolic pressure when the lungs are expanded. Pulsus paradoxus is present if the difference in systolic pressures is greater than 10 mmHg. In many settings, emergency physician proficiency in recognizing signs of pericardial tamponade on bedside cardiac ultrasound has replaced routine measurement of pulsus paradoxus in patients with dyspnea, but serial measurements can be useful when monitoring a patient with a pericardial effusion. (See "Pulsus paradoxus in pericardial disease" and "Emergency ultrasound in adults with abdominal and thoracic trauma", section on 'Pericardial and limited cardiac examination'.)

- **Airway assessment** – Perform a brief airway assessment to identify a potentially difficult airway in case endotracheal intubation is ultimately needed (figure 1 and picture 2). (See "Approach to the difficult airway in adults for emergency medicine and critical care", section on 'Identifying the anatomically difficult airway'.)

- **Abdominal distension, masses, or peritoneal signs** – A number of conditions such as peritonitis, ruptured viscous, or bowel obstruction can cause severe pain that affects respiration and may manifest as acute shortness of breath, although this is generally not the patient's primary complaint [19]. (See "Evaluation of the adult with nontraumatic abdominal or flank pain in the emergency department".)

  - Ascites secondary to malignancy or liver disease can distend the abdominal cavity, placing pressure on the diaphragm and thereby increasing the work of breathing [20]. In such cases, dyspnea often improves after large-volume paracentesis. (See "Evaluation of adults with ascites".)

  - Obesity is a risk factor for obstructive sleep apnea and obesity hypoventilation syndrome, which can present with chronic dyspnea on exertion in addition to daytime

hypersomnolence, hypoxemia, and hypercapnia. Additionally, increased abdominal pressure on the diaphragm can affect pulmonary function, and obesity has been associated with an increased risk of DVT and PE. (See "Clinical manifestations and diagnosis of obesity hypoventilation syndrome" and "Overweight and obesity in adults: Health consequences", section on 'Respiratory system'.)

- **Skin inspection** – Examine the skin for discoloration (eg, cyanosis, pallor, mottling) suggesting hypoxia or poor perfusion, signs of an allergic reaction such as erythema or urticaria, and evidence of trauma.

- **Extremities** – Peripheral edema may not occur with acute left heart failure but, if present, suggests ADHF as the cause of dyspnea. Clubbing is associated with conditions causing chronic hypoxemia.

General measures — Most patients with acute dyspnea should have the following general measures (if not performed during initial stabilization):

- Provide supplemental oxygen if needed. (See 'Supplemental oxygen' below.)

- Establish intravenous (IV) access (and obtain blood for laboratory measurements). (See 'Complete blood count, serum chemistries' below and 'Directed studies based on clinical suspicion' below.)

- Place on continuous cardiac and pulse oximetry monitoring.

- Maintain appropriate infection control measures – If tuberculosis, COVID-19, or other highly transmittable infections are a concern, appropriate infection control measures are crucial (table 2). (See "COVID-19: General approach to infection prevention in the health care setting" and "COVID-19: Infection prevention for persons with SARS-CoV-2 infection", section on 'Infection prevention in the health care setting'.)

Supplemental oxygen — Most patients with acute dyspnea should receive supplemental oxygen, an important and readily available treatment for many causes of dyspnea. Some patients with normal oxygen saturation (95 percent or greater) and normal vital signs may not need supplemental oxygen and the above general measures (eg, young patients with no comorbidities and patients with typical exacerbations of chronic disease such as asthma). Supplemental oxygen can be started at 2 liters per minute (LPM) via nasal cannula with a target oxygen saturation of 94 to 98 percent. Other oxygen delivery systems and targets are discussed elsewhere. (See "Evaluation and management of the nonventilated, hospitalized adult patient with acute hypoxemia", section on 'Oxygen and respiratory support'.)

Supplemental oxygen should be used cautiously in patients with COPD exacerbation or ACS, although this is not an exhaustive list.

- COPD exacerbation – Do **not** withhold oxygen from patients with COPD, but lower the target saturation to 90 to 94 percent, with the understanding that this may reduce ventilatory drive and cause hypercarbia. However, failure to oxygenate the patient may have profoundly adverse consequences. If a clinician determines that a COPD patient requires endotracheal intubation, oxygen delivery should be maximized without regard to the target oxygen saturation or hypercarbia. (See "COPD exacerbations: Management" and "The evaluation, diagnosis, and treatment of the adult patient with acute hypercapnic respiratory failure".)

- ACS – Excessive oxygen may be harmful in those with ACS. Administer oxygen for ACS only when the oxygen saturation is consistently below 94 percent. (See "Overview of the acute management of ST-elevation myocardial infarction", section on 'Therapies of unclear benefit'.)

Often, response to supplemental oxygen and other therapies or provocative maneuvers (eg, obtaining pulse oximetry while ambulating) assists in diagnosis and disposition. The following are examples:

- An improvement in SpO_2 immediately after the administration of low-flow oxygen indicates a ventilation-perfusion (V/Q) mismatch. (See "Measures of oxygenation and mechanisms of hypoxemia", section on 'Ventilation-perfusion mismatch'.)

- Rapid improvement following treatment with bronchodilators strongly suggests bronchoconstriction.

- Failure to improve with oxygen administration may indicate a right-to-left shunt or presence of methemoglobinemia. (See "Methemoglobinemia" and "Measures of oxygenation and mechanisms of hypoxemia", section on 'Right-to-left shunt'.)

- In a patient with HIV (especially CD4 counts < 200 cells/microL) who presents with fever, cough, and dyspnea progressing over days to weeks, a profound drop in SpO_2 associated with ambulation is characteristic of *Pneumocystis* pneumonia. (See "Epidemiology, clinical presentation, and diagnosis of Pneumocystis pulmonary infection in patients with HIV".)

Diagnostic testing

Overview — Testing should be performed in the context of the history and examination findings. Random testing without a clear differential diagnosis can mislead the clinician and

delay appropriate management. A plain chest radiograph (CXR), an electrocardiogram (ECG), and laboratory tests are routinely obtained in most ED patients with acute dyspnea. Bedside emergency physician-performed ultrasound is an effective and rapid tool for investigating several important causes of acute dyspnea but requires equipment and operator proficiency. (See 'Studies for almost all patients' below and 'Role of bedside ultrasound' below.)

The routine use of dyspnea panels (ie, biomarker panels that include troponin, myoglobin, creatinine kinase, D-dimer, and B-type natriuretic peptide) does not appear to improve accuracy beyond clinical assessment and focused testing [21,22]. Arterial blood gas and chest multidetector computed tomography (MDCT) should not be performed routinely and reserved for specific circumstances.

- **Arterial blood gas** – The role of arterial blood gas in the diagnosis and treatment of the acutely dyspneic patient is limited. The decision to perform tracheal intubation is based on clinical assessment and not on the arterial blood gas. Oxygenation is easily assessed using transcutaneous pulse oximetry. If there is any concern about pulse oximetry accuracy (table 5), we obtain an arterial blood gas. Acid-base status can be assessed using a venous blood gas and the serum bicarbonate. We do not routinely use arterial or venous blood gas for monitoring unless there is a discrepancy with pulse oximeter or capnography. (See "Arterial blood gases" and "Venous blood gases and other alternatives to arterial blood gases".)

- **Chest MDCT** – Even though chest MDCT will diagnose multiple problems, including malignancy, pneumonia, and pulmonary edema, it is most useful in dyspneic ED patients with trauma or concern for PE and when plain chest radiograph is inconclusive. MDCT does entail some risks, including contrast-induced nephropathy, allergic reaction to contrast, and radiation-related malignancy. MDCT should be performed when it is needed to establish an important diagnosis, even in patients with compromised kidney function since contrast-related kidney injury is relatively uncommon. (See "Prevention of contrast-induced acute kidney injury associated with computed tomography" and "Radiation dose and risk of malignancy from cardiovascular imaging".)

Studies for almost all patients

Plain chest radiograph — A CXR is obtained for most ED patients with acute dyspnea (an exception being otherwise asymptomatic asthma patients who respond well to treatment).

Diseases identified by CXR

- **ADHF** – Signs of ADHF that may appear on a CXR include cardiomegaly, cephalization of blood vessels, interstitial edema (eg, "Kerley B" lines, peribronchial cuffing), vascular congestion (image 1), and pleural effusions. Radiographic findings can lag behind the clinical picture, and approximately 20 percent of patients admitted with ADHF have a nondiagnostic CXR [23]. ADHF and pneumonia can have a similar appearance on CXR, but ADHF is generally associated with hypertension (except in cardiogenic shock), often responds to aggressive early therapy, and is associated with an elevation in brain natriuretic peptide (BNP). Lung ultrasound is more sensitive than CXR in diagnosing ADHF and should be used if the operator is proficient and resources are available [24,25]. (See "Heart failure: Clinical manifestations and diagnosis in adults".)

  Symptomatic ADHF can be caused by volume overload, heart failure with preserved ejection fraction (HFpEF [previously known as "diastolic dysfunction"]), heart failure with reduced ejection fraction (HFrEF [previously known as "systolic dysfunction"]), or outflow obstruction (eg, aortic stenosis, hypertrophic cardiomyopathy, severe systemic hypertension). Myocardial ischemia and arrhythmia are common precipitants. Symptoms range from mild dyspnea on exertion to severe pulmonary edema. Common findings include tachypnea, pulmonary crackles, jugular venous distension (movie 2), S3 gallop, and peripheral edema. ADHF is among the most common causes of acute respiratory failure among patients over 65 years of age. (See "Examination of the jugular venous pulse", section on 'How to examine the jugular venous pulse' and "Approach to diagnosis and evaluation of acute decompensated heart failure in adults".)

- **Cardiomyopathy** – The physiologic derangements associated with cardiomyopathy (primarily dilated cardiomyopathy) may result in pulmonary edema and manifest as dyspnea. CXR can appear similar to ADHF or just show cardiomegaly. Potential causes include cardiac ischemia, hypertension, alcohol abuse, cocaine abuse, and a number of systemic diseases (eg, sarcoidosis, systemic lupus erythematosus). (See "Causes of dilated cardiomyopathy".)

- **High-output heart failure** – High-output heart failure may be precipitated by a number of conditions, including severe anemia, pregnancy, beriberi (thiamine deficiency), and thyrotoxicosis. Signs include tachycardia, bounding pulses, a venous hum heard over the internal jugular veins, and carotid bruits. An elevated troponin may be seen secondary to demand ischemia. (See "Causes and pathophysiology of high-output heart failure".)

- **Pneumonia** – Although an infiltrate on CXR is considered the "gold standard" for diagnosing pneumonia (image 2), radiographs obtained early in the clinical course may be nondiagnostic [26]. Volume depletion may also lead to a negative initial CXR. Contrary

to past teaching, the appearance of the CXR (lobar versus diffuse disease) does not accurately predict the nature of the pneumonia (eg, "typical" versus "atypical" organisms). Pneumonia and ADHF can have a similar appearance on CXR, but pneumonia is more often associated with a normal or low blood pressure, does not respond to early therapy, and is generally **not** associated with an elevation in BNP. (See "Clinical evaluation and diagnostic testing for community-acquired pneumonia in adults".)

Infection with COVID-19 can cause pneumonia. Common abnormal radiographic findings are consolidation and ground-glass opacities with bilateral, peripheral, and lower lung zone distributions. Lung involvement peaks in severity at 10 to 12 days after symptom onset. (See "COVID-19: Clinical features", section on 'Imaging findings' and "COVID-19: Diagnosis".)

- **Noncardiogenic pulmonary edema (ARDS)** – ARDS can complicate a wide range of conditions and is characterized by rapidly progressive dyspnea, hypoxia, and bilateral infiltrates on CXR. It can be difficult to distinguish from ADHF purely on clinical grounds; BNP and echocardiography can be helpful in differentiation. Potential causes of ARDS include sepsis, shock, severe trauma, toxic inhalations (eg, aspiration, thermal injury, anhydrous ammonia, chlorine), infections (eg, COVID-19, hantavirus, severe acute respiratory syndrome [SARS], dengue, Middle East respiratory syndrome), blood transfusion, and drug overdose (eg, cocaine, opioids, aspirin). (See "Acute respiratory distress syndrome: Epidemiology, pathophysiology, pathology, and etiology in adults" and "Acute respiratory distress syndrome: Clinical features, diagnosis, and complications in adults".)

- **High-altitude pulmonary edema (HAPE)** – HAPE is a form of noncardiogenic pulmonary edema that typically occurs between two to four days after rapid ascent to elevations over 2500 meters (8000 feet). Symptoms often begin with a nonproductive cough, dyspnea on exertion, and fatigue and may progress quickly to dyspnea at rest. Pulmonary symptoms are often accompanied by a headache and occasionally cerebral edema. (See "High-altitude pulmonary edema" and "Acute mountain sickness and high-altitude cerebral edema".)

- **Pneumothorax** – A pneumothorax sufficient to cause acute dyspnea is usually visible on CXR (image 3 and image 4), while an additional expiratory view may be helpful [27]. In addition to trauma, vigorous coughing, and medical procedures (eg, central venous catheter placement, intubation, and barotrauma), a number of medical conditions (eg, COPD, cystic fibrosis, tuberculosis, Marfan syndrome, *Pneumocystis* pneumonia) increase the risk for developing a pneumothorax. (See "Clinical presentation and diagnosis of

pneumothorax" and "Pneumothorax in adults: Epidemiology and etiology" and "Initial evaluation and management of blunt thoracic trauma in adults" and "Treatment of primary spontaneous pneumothorax in adults" and "Treatment of secondary spontaneous pneumothorax in adults".)

- **Pleural effusion, hemothorax** – A moderate to large pleural effusion or hemothorax, which appear similar on CXR, can cause acute dyspnea. Common etiologies include infection, ascites, pancreatitis, cancer, heart failure, pneumonia, iatrogenic vessel transection, PE, and trauma. These are evaluated by cross-sectional imaging studies (eg, blood has high attenuation/Hounsfield units on CT) or by analysis of the pleural fluid. (See "Imaging of pleural effusions in adults" and "Pleural fluid analysis in adults with a pleural effusion".)

- **COPD and asthma** – Large lung volumes and a flattened diaphragm on CXR suggest air trapping, which occurs with COPD (image 5) or asthma. However, many patients with mildly or moderately severe COPD and most patients with asthma have an unremarkable CXR. (See "Acute exacerbations of asthma in adults: Emergency department and inpatient management", section on 'Chest radiograph' and "Chronic obstructive pulmonary disease: Diagnosis and staging", section on 'Additional testing'.)

- **Airway foreign body** – Unilateral air trapping suggests a bronchial foreign body (image 6), but many organic foreign bodies are radiolucent and not evident on CXR [28]. Additionally, the absence of unilateral air trapping does not rule out a foreign body. (See "Airway foreign bodies in adults", section on 'Imaging'.)

- **Acute chest syndrome** – This potentially life-threatening complication of sickle cell disease typically has new pulmonary infiltrates on CXR. Patients generally complain of severe chest pain and acute dyspnea and have a fever. It is easily confused with pneumonia but should be considered in any patient with sickle cell disease. (See "Overview of the clinical manifestations of sickle cell disease" and "Overview of the pulmonary complications of sickle cell disease".)

- **Chest wall and direct pulmonary injury** – Rib fractures, flail chest, or a pulmonary contusion or laceration are possible sources for acute dyspnea in any patient with chest trauma. (See "Initial evaluation and management of chest wall trauma in adults" and "Initial evaluation and management of blunt thoracic trauma in adults".)

- **Diffuse alveolar hemorrhage** – Hemorrhage from an injury, underlying disease (eg, malignancy, tuberculosis), or as an adverse effect of anticoagulation can cause acute

dyspnea. CXR can demonstrate diffuse opacities. (See "Evaluation of nonlife-threatening hemoptysis in adults", section on 'Chest imaging showing diffuse opacities'.)

- **EVALI** – Initially described in 2019, EVALI is caused by e-cigarette use, especially among males younger than 35 years of age. It is primarily associated with vaping tetrahydrocannabinol (THC) products and strongly correlated with the presence of vitamin E acetate in the formulations [29,30]. EVALI is characterized by shortness of breath, cough, chest pain, and gastrointestinal complaints and diffuse hazy or consolidative opacities on CXR. (See "E-cigarette or vaping product use-associated lung injury (EVALI)".)

- **Toxins and exposures** – As examples, pneumonitis can result from ingestions of various agents such as hydrocarbon (eg, petroleum distillates) or paraquat (an herbicide). Inhalation of typically low-water solubility agents (eg, oxides of nitrogen, chlorine, phosgene) can also cause a delayed pneumonitis. (See "Common occupational chemical exposures: General approach and management of selected exposures" and "Acute hydrocarbon exposure: Clinical toxicity, evaluation, and diagnosis" and "Paraquat poisoning" and "Inhalation injury from heat, smoke, or chemical irritants".)

- **Lung cancer** – Shortness of breath is a common symptom in patients with lung cancer at the time of diagnosis, occurring in approximately 25 percent of cases. Dyspnea may be due to extrinsic or intraluminal airway obstruction, obstructive pneumonitis or atelectasis, lymphangitic tumor spread, tumor emboli, pneumothorax, pleural effusion, or pericardial effusion with tamponade. (See "Clinical manifestations of lung cancer".)

  CXR findings that should raise the suspicion for lung cancer include new lesions larger than 3 cm, measurable growth in any nodule or mass, pleural nodularity, and asymmetric or significantly enlarged hilar or paratracheal nodes. Concerning but less specific findings include pleural effusions and nondependent or substantial atelectasis. (See "Overview of the initial evaluation, diagnosis, and staging of patients with suspected lung cancer", section on 'Initial evaluation'.)

Electrocardiogram — An ECG with ST segment changes is strong evidence for cardiac ischemia; however, the initial ECG is normal in approximately 20 percent of patients subsequently diagnosed with a myocardial infarction, and only 33 percent of initial ECGs are diagnostic. The ECG may also reveal signs of PE (right heart strain), pericardial effusion (diffuse low voltage (waveform 1), electrical alternans (waveform 2)), and other disease processes. However, the sensitivity and specificity of the ECG for PE is limited. Diffuse ST segment elevations, along with PR depression, may be seen in pericarditis. (See "Diagnosis of acute myocardial infarction" and "Clinical presentation, evaluation, and diagnosis of the nonpregnant

adult with suspected acute pulmonary embolism" and "Pericardial effusion: Approach to diagnosis".)

Complete blood count, serum chemistries — A complete blood count and serum chemistries are commonly obtained tests that are useful to screen for infection, anemia, metabolic acidosis, hyperglycemia, and kidney failure.

- Acute anemia may result in dyspnea due to the lack of oxygen-carrying capacity. (See "Diagnostic approach to anemia in adults".)

- Diseases that cause metabolic acidosis (eg, diabetic ketoacidosis) can cause tachypnea and dyspnea from the respiratory compensation. Patients with diabetic ketoacidosis may give a history of polyuria, polydipsia, polyphagia, and progressive weakness; signs of severe disease include hyperventilation, altered mental status, abdominal pain, and vomiting. (See "Diabetic ketoacidosis and hyperosmolar hyperglycemic state in adults: Clinical features, evaluation, and diagnosis".)

Role of bedside ultrasound — Bedside emergency physician-performed ultrasound is an effective and rapid tool and can help identify the following illnesses [31]. It does not require transporting unstable patients and can be repeated when clinical status changes.

- **ADHF** (image 7) (see "Indications for bedside ultrasonography in the critically ill adult patient", section on 'Thoracic ultrasonography' and "Approach to diagnosis and evaluation of acute decompensated heart failure in adults" and "Emergency ultrasound in adults with abdominal and thoracic trauma", section on 'Ultrasound assessment of volume status')

- **Pericardial tamponade** (movie 1) (see "Emergency ultrasound in adults with abdominal and thoracic trauma", section on 'Pericardial and limited cardiac examination')

- **Clinically significant pneumothorax** (image 8) (see "Bedside pleural ultrasonography: Equipment, technique, and the identification of pleural effusion and pneumothorax", section on 'Evaluation for pneumothorax' and "Clinical presentation and diagnosis of pneumothorax", section on 'Diagnostic imaging')

- **Pneumonia** (ultrasound may be more sensitive for pneumonia than the traditional CXR, but accuracy is operator dependent) (see "Clinical evaluation and diagnostic testing for community-acquired pneumonia in adults", section on 'Ultrasound and other studies')

- **Pleural effusion** (image 9) (see "Bedside pleural ultrasonography: Equipment, technique, and the identification of pleural effusion and pneumothorax", section on

'Identification of pleural effusion using ultrasonography' and "Imaging of pleural effusions in adults")

- **Cardiac wall motion abnormalities** (supports ischemia in the appropriate clinical setting) (see "Role of echocardiography in acute myocardial infarction", section on 'Use of echocardiography')

- **Right heart strain or McConnell's sign** – The presence of right heart strain is approximately 50 percent sensitive and 83 percent specific for PE in the proper clinical setting but can also be seen with right heart failure, ARDS, pulmonary hypertension, volume overload, tricuspid valve disease, and others [32]; McConnell's sign, which is regional wall motion abnormalities that spare the right ventricular apex, appears to be more specific for PE than global right ventricular dysfunction [33] (see "Clinical presentation, evaluation, and diagnosis of the nonpregnant adult with suspected acute pulmonary embolism", section on 'Echocardiography' and "Echocardiographic assessment of the right heart")

- **Proximal deep vein thromboses** (supports diagnosis of PE in patient with dyspnea) (see "Clinical presentation and diagnosis of the nonpregnant adult with suspected deep vein thrombosis of the lower extremity", section on 'Diagnostic ultrasonography suspected first DVT')

Directed studies based on clinical suspicion — In most instances, the emergency clinician can determine the diagnosis based upon a thorough history, physical examination, CXR, and ECG. Signs associated with dyspnea, their clinical significance, and possible diagnoses are presented in the table (table 4). If needed, further testing is directed based on suspicion for disease processes and detailed below.

Upper airway obstruction — Angioedema, anaphylaxis, and tracheal foreign bodies are discussed above. (See 'Stridor' above.)

Oropharyngeal infections, including epiglottitis, pertussis, Ludwig's angina, severe tonsillitis, peritonsillar abscess, and retropharyngeal abscess, can cause acute dyspnea [34-37]. Management depends on the etiology and is reviewed separately. (See "Epiglottitis (supraglottitis): Clinical features and diagnosis" and "Pertussis infection in adolescents and adults: Clinical manifestations and diagnosis" and "Deep neck space infections in adults" and "Lemierre syndrome: Septic thrombophlebitis of the internal jugular vein".)

Airway trauma can present with acute dyspnea. Blunt or penetrating injuries of the head or neck can cause hemorrhage, swelling, and anatomic distortion. A laryngeal fracture should be

suspected in patients complaining of dyspnea in the setting of severe neck pain and dysphonia following blunt trauma. Patients who have sustained facial burns or smoke inhalation are at risk for rapidly progressive airway edema and compromise. Management depends on the etiology and is reviewed separately. (See "Penetrating neck injuries: Initial evaluation and management" and "Emergency care of moderate and severe thermal burns in adults" and "Inhalation injury from heat, smoke, or chemical irritants".)

Once the airway is stabilized, evaluation can proceed with the following studies as indicated:

- **Neck radiographs** – Plain neck radiographs are of limited utility in adults because they are neither sensitive or specific. Neck radiographs may reveal changes associated with retropharyngeal abscess, epiglottitis, or a radiopaque foreign body. (See "Airway foreign bodies in adults", section on 'Imaging' and "Assessment of stridor in children", section on 'Neck radiographs'.)

- **Neck CT** – CT is generally the imaging modality of choice to diagnose and evaluate the site and extent of deep neck space infections as well as radiopaque foreign bodies [38]. CT angiography may be indicated if clinical features suggest involvement of the neurovascular compartment within the carotid sheath (eg, Lemierre syndrome). (See "Deep neck space infections in adults", section on 'Clinical suspicion and urgent imaging' and "Lemierre syndrome: Septic thrombophlebitis of the internal jugular vein".)

- **Fiberoptic laryngoscopy/bronchoscopy** – If clinical suspicion remains high for an airway foreign body aspiration with a negative CT scan, visual airway inspection is recommended as many organic foreign bodies are radiolucent [28]. Flexible laryngoscopy is used to identify a foreign body in the upper airway (above the vocal cords), and bronchoscopy (flexible or rigid) is used for the identification of foreign bodies located in the lower airway (below the vocal cords). (See "Airway foreign bodies in adults", section on 'Diagnosis' and "Airway foreign bodies in adults", section on 'Flexible bronchoscopy'.)

Acute coronary syndrome/heart failure

- **Cardiac biomarkers** – Elevated biomarkers support the diagnosis of cardiac ischemia. However, the initial cardiac biomarkers (eg, troponin I) obtained in the ED are frequently normal. Risk scores and serial measurements of cardiac biomarkers are generally used to rule out ACS. Cardiac biomarkers have limited specificity and may be elevated in the setting of PE, sepsis, pericarditis, myocarditis, renal failure, and interference with the assay (generally from monoclonal antibodies or rheumatoid factor). (See "Troponin testing: Clinical use" and "Evaluation of emergency department patients with chest pain at low or

intermediate risk for acute coronary syndrome", section on 'Initial evaluation' and "Elevated cardiac troponin concentration in the absence of an acute coronary syndrome".)

- **BNP** – The measurement of BNP is helpful when the diagnosis of ADHF is in question. However, it can be elevated secondary to many causes of fluid overload, such as kidney failure. BNP testing is not helpful when used indiscriminately in patients with acute, severe dyspnea, although it may be helpful in patients with a suspected cardiac origin [39]. In a metanalysis of ED patients with acute dyspnea of suspected cardiac origin (5 trials, 2513 patients), routine BNP testing decreased hospital length of stay by about one day, and there was a trend towards reduced admission rates [40]. BNP is interpreted as follows (see "Natriuretic peptide measurement in heart failure"):
  - Less than 100 pg/mL – Negative predictive value of over 90 percent for ADHF.
  - Between 100 pg/mL and 500 pg/mL – Cannot differentiate between ADHF and other causes of elevated BNP. Causes of a false-positive BNP (generally between 100 pg/mL and 500 pg/mL) include PE, fluid overload states (eg, renal failure, liver failure), critical illness, and other causes of right ventricular distension (eg, cor pulmonale, pulmonary hypertension).
  - Greater than 500 pg/mL – Positive predictive value over 90 percent for ADHF.

Infection — Pulmonary infections such as community-acquired pneumonia (CAP) can present with productive cough, fever, dyspnea, hypoxia, and pleuritic chest pain, although these are insensitive signs. Dyspnea is generally subacute in onset unless underlying chronic pulmonary disease is present. Mild CAP deemed appropriate for outpatient treatment does not need any microbiologic testing (with exception of COVID-19 testing during the pandemic and possibly testing for respiratory viruses, particularly for influenza, during respiratory virus season). Selection of appropriate tests is discussed elsewhere. (See "Clinical evaluation and diagnostic testing for community-acquired pneumonia in adults", section on 'Approach to testing'.)

Pulmonary embolism — The diagnosis of PE should be considered in any patient with acute-onset dyspnea. Risk factors include a history of deep venous thrombosis or PE, prolonged immobilization, recent trauma or surgery (particularly orthopedic), pregnancy (algorithm 1), malignancy, stroke or paresis, oral contraceptive or other estrogen use, smoking, and a personal or family history of hypercoagulability. Many patients have no known risk factor at the time of diagnosis. Presentation varies widely, but dyspnea at rest and tachypnea are the most common signs. Other embolic phenomena include fat embolism, especially after a long bone fracture or associated with the acute chest syndrome of sickle cell disease, and amniotic fluid

embolism. Although most patients with dyspnea secondary to a PE demonstrate some abnormality on CXR or ECG, there are no pathognomonic findings in either test. (See "Epidemiology and pathogenesis of acute pulmonary embolism in adults" and "Clinical presentation, evaluation, and diagnosis of the nonpregnant adult with suspected acute pulmonary embolism" and "Fat embolism syndrome" and "Acute chest syndrome (ACS) in sickle cell disease (adults and children)" and "Amniotic fluid embolism".)

- **D-dimer** – This test is **not** part of the workup of every patient with chest pain or shortness of breath and depends upon the patient's pretest probability for PE. Patients with a negative Pulmonary Embolism Rule-out Criteria (PERC) score (table 7) (calculator 1) can be ruled out for PE without D-dimer testing. Patients at low or moderate risk for PE according to a validated scoring system (eg, modified Wells criteria for PE) (table 8) and a negative enzyme-linked immunosorbent assay (ELISA) D-dimer can be ruled out for PE without further testing (algorithm 2 and algorithm 3). It is **not** appropriate to use a D-dimer to screen patients at high risk for thromboembolic disease; they require a CT angiogram or ventilation perfusion (V/Q) scan (algorithm 4). (See "Clinical presentation, evaluation, and diagnosis of the nonpregnant adult with suspected acute pulmonary embolism" and "Pulmonary embolism in pregnancy: Clinical presentation and diagnosis".)

- **Computed tomography pulmonary angiography (CTAP)** - CTAP (ie, chest CT angiogram with contrast) is the first-choice diagnostic imaging modality for diagnosis of PE because it is sensitive and specific and can discover alternate diagnoses [8]. (See "Clinical presentation, evaluation, and diagnosis of the nonpregnant adult with suspected acute pulmonary embolism", section on 'Computed tomography pulmonary angiography'.)

- **Ventilation perfusion (V/Q) scan** – In clinically stable patients with concern for PE who can not undergo CTAP, V/Q scan is an alternative method. V/Q scan and other alternative imaging approaches are discussed elsewhere. (See "Clinical presentation, evaluation, and diagnosis of the nonpregnant adult with suspected acute pulmonary embolism", section on 'Alternate imaging approaches'.)

Valvular or pericardial process

- **Echocardiogram** – Echocardiogram can diagnose cardiomyopathy, aortic stenosis, mitral regurgitation, or ruptured chordae tendinae, which can present with acute dyspnea and a new murmur. Since a murmur is not always present with valvular disease, echocardiogram can be helpful in patients with acute dyspnea after other nonvalvular etiologies have been excluded. (See "Valvular heart disease in older adults".)

Metabolic acidosis — Tachypnea and dyspnea can occur from respiratory compensation for metabolic acidosis, which is suggested by a low bicarbonate concentration (often defined as <23 mEq/L) on the serum chemistries. Evaluation includes confirmation with a blood gas and testing for specific causes (table 9).

A venous blood gas is generally sufficient (as compared with arterial blood gas) to confirm presence and assess degree of metabolic acidosis. Laboratory evaluation for specific causes is discussed elsewhere. (See "Approach to the adult with metabolic acidosis", section on 'Evaluation'.)

Asthma

- **Peak flow and pulmonary function tests (PFTs)** – The peak expiratory flow rate (PEFR) can be helpful in distinguishing pulmonary and cardiac causes of dyspnea and determining the severity of bronchoconstriction in cases of severe asthma. Normal PEFR varies with sex, height, and age (table 10 and table 11); and accuracy depends upon patient cooperation.

  Small observational studies suggest PEFR is generally higher in patients with a cardiac cause of dyspnea [41,42]. During acute asthma exacerbations, PEFR measurements provide a screening tool for the presence of hypercapnia and obviate the need for routine blood gases. In the absence of respiratory depressant medications (eg, opioids or sedatives), hypercapnia is rarely present until the PEFR falls below 25 percent of normal or 200 L/minute. (See "Peak expiratory flow monitoring in asthma".)

Neuromuscular disease

- **Negative inspiratory force (NIF)** – NIF and forced vital capacity measurements can be obtained at the bedside to assess dyspneic patients with possible neuromuscular disease (eg, myasthenia gravis, Guillain-Barré) or musculoskeletal disease (ankylosing spondylitis, severe scoliosis, kyphosis). If the NIF is less than 30 centimeters of water (cm H_2O) or the forced vital capacity is less than 15 to 20 mL/kg, the patient should be admitted to an intensive care unit in anticipation of the need for mechanical ventilation [43]. (See "Tests of respiratory muscle strength".)

Cause of dyspnea not established — In some instances, the cause of dyspnea cannot be determined with certainty in the ED. Treatment should still be provided based upon available data (eg, broad-spectrum antibiotics when serious infection is suspected). In a patient who is going to be admitted, consultation with the appropriate specialist (eg, cardiologist, intensivist, pulmonologist), which can occur during the hospitalization, may help establish a diagnosis.

Hyperventilation from anxiety is a diagnosis of exclusion in the ED. Even among young, healthy patients with a known anxiety disorder, it is prudent to perform a history and physical examination to screen for medical causes of dyspnea. To complicate matters, anxiety is common among patients with severe medical disease. As an example, COPD patients have a threefold increase in the prevalence of anxiety disorders compared with the general population [44]. In such patients, it is best to assume that an exacerbation of their underlying medical disease is the cause of dyspnea until proven otherwise.

DISPOSITION

The patient's condition, preliminary diagnosis, and risk assessment determine disposition. Patients at risk of rapid deterioration require close monitoring and should be admitted to an intensive care setting. Patients who do not improve sufficiently during ED treatment, have significant comorbidities, or are deemed high-risk should be admitted to the appropriate hospital ward. High-risk dyspneic patients include older adults, those with immunocompromise, severe underlying lung or heart disease, or those who demonstrate unexplained abnormal vital signs.

Stable patients whose evaluation has ruled out significant disease or are deemed to have acceptably low risk for such disease may be discharged. Patients being discharged must have a clear understanding of their discharge diagnosis, written discharge instructions, and planned follow-up with clear instructions to return to the emergency department if their condition worsens.

SOCIETY GUIDELINE LINKS

Links to society and government-sponsored guidelines from selected countries and regions around the world are provided separately. (See "Society guideline links: Adult with chest pain in the emergency department".)

INFORMATION FOR PATIENTS

UpToDate offers two types of patient education materials, "The Basics" and "Beyond the Basics." The Basics patient education pieces are written in plain language, at the 5th to 6th grade reading level, and they answer the four or five key questions a patient might have about a given condition. These articles are best for patients who want a general overview and who prefer short, easy-to-read materials. Beyond the Basics patient education pieces are longer, more

sophisticated, and more detailed. These articles are written at the 10th to 12th grade reading level and are best for patients who want in-depth information and are comfortable with some medical jargon.

Here are the patient education articles that are relevant to this topic. We encourage you to print or email these topics to your patients. (You can also locate patient education articles on a variety of subjects by searching on "patient info" and the keyword(s) of interest.)

- Basics topic (see "Patient education: Shortness of breath (The Basics)")
- Beyond the Basics topic (see "Patient education: Shortness of breath (dyspnea) (Beyond the Basics)")

SUMMARY AND RECOMMENDATIONS

- **Background** – Dyspnea is the perception of an inability to breathe comfortably and is a common chief complaint among emergency department (ED) patients. Dyspnea can be caused by abnormalities affecting the upper airway, lungs, heart, chest wall, diaphragm, and nervous system. (See 'Introduction' above and 'Pathophysiology' above.)
- **Epidemiology** – The most common diagnoses among older adult patients presenting to an ED with a complaint of acute shortness of breath and manifesting signs of respiratory distress (eg, respiratory rate >25 breaths per minute, oxygen saturation [SpO_2] <93 percent) are decompensated heart failure, pneumonia, chronic obstructive pulmonary disease (COPD), pulmonary embolism (PE), and asthma. (See 'Epidemiology' above.)
- **Overview of management** – The primary goals for the emergency clinician faced with an acutely dyspneic patient are the following (see 'Overview of Approach' above):
  - Optimize arterial oxygenation
  - Determine the need for emergency airway management and ventilatory support
  - Establish the most likely causes of dyspnea, initiate treatment, and stabilize if critically ill
- **Common life-threatening causes of acute dyspnea** – These are presented in the table (table 1). (See 'Overview of Approach' above.)
- **Initial rapid assessment** – The clinician should perform a screening physical examination looking for signs of imminent respiratory arrest or significant respiratory distress in all patients complaining of acute dyspnea.

- **Clinical danger signs** – Signs that portend imminent respiratory arrest include (see 'Clinical danger signs' above):

  - Depressed mental status
  - Inability to maintain respiratory effort
  - Cyanosis

  Signs suggestive of severe respiratory distress include:

  - Retractions and the use of accessory muscles
  - Significant tachypnea
  - Appearing anxious; sitting bolt upright or in a tripod position
  - Audible stridor or wheezing
  - Brief, fragmented speech
  - Inability to lie supine
  - Profound diaphoresis; dusky skin
  - Agitation or other altered mental status

- **Emergency stabilization and general measures** – After the screening examination, in a patient with any vital sign abnormalities or danger signs, perform an assessment of airway patency and need for ventilatory support, establish the following general measures, and search for rapidly reversible causes (tension pneumothorax, pericardial tamponade, upper airway foreign body). (See 'Emergency stabilization of patients with danger signs' above.)

  - Provide supplemental oxygen
  - Establish intravenous (IV) access and obtain blood for laboratory measurements
  - Place on continuous cardiac and pulse oximetry monitoring
  - Maintain appropriate infection control measures

  In a patient with respiratory distress:

  - Assemble and check airway management equipment
  - Request respiratory therapist to the bedside
  - Perform bedside ultrasound to assess for etiology and reversible causes

- **Options for oxygenation and ventilatory support** – The following are options for oxygenation and ventilatory support (see 'Options for oxygenation or ventilatory support' above):

- **High-flow nasal cannulae (HFNC)** – Provides a relatively high fraction of inspired oxygen (FiO_2) to patients with severe nonhypercapnic hypoxemic respiratory failure, and is often used in patients with COVID-19. (picture 1)

- **Noninvasive ventilation (NIV)** – Delivers continuous positive airway pressure (CPAP) or bilevel positive airway pressure (BiPAP) via a mask and is best for patients with acute decompensated heart failure (ADHF) or a COPD exacerbation.

- **Mechanical ventilation** – Endotracheal intubation and controlled mechanical ventilation should be pursued aggressively when ventilatory support is needed and NIV is **not** expected to improve outcomes, such as for acute exacerbations of asthma and diseases that do not respond rapidly to medical therapy (eg, pneumonia and acute respiratory distress syndrome [ARDS]).

- **Extracorporeal membrane oxygenation (ECMO)** – In patients with hypoxemic respiratory failure who are mechanically ventilated and not responding to conventional measures, ECMO is an option (if available). Appropriate services must be consulted early to mobilize resources.

- **Diagnostic testing**

  - In most ED patients with acute dyspnea, obtain a plain chest radiograph (CXR), an electrocardiogram (ECG), and complete blood count and serum chemistries. (See 'Studies for almost all patients' above.)

  - After a history and examination generates clinical suspicion for various diagnoses, obtain studies directed to diagnose or rule out specific illnesses. (See 'Directed studies based on clinical suspicion' above.)

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Topic 292 Version 44.0

GRAPHICS

Differential diagnosis of acute dyspnea

HEENT
Angioedema
Anaphylaxis
Pharyngeal infections
Deep neck infections
Foreign body
Neck trauma
Chest wall
Rib fractures
Flail chest
Pulmonary
COPD exacerbation
Asthma exacerbation
Pulmonary embolism
Pneumothorax
Pulmonary infection
ARDS
Pulmonary contusion or other lung injury
Hemorrhage
Cardiac
ACS
ADHF
Flash pulmonary edema
High output failure
Cardiomyopathy
Arrhythmia
Valvular dysfunction
Cardiac tamponade

Neurologic
Stroke
Neuromuscular disease
Toxic/metabolic
Organophosphate poisoning
Salicylate poisoning
CO poisoning
Toxic ingestion
Diabetic ketoacidosis
Sepsis
Anemia
Acute chest syndrome
Miscellaneous
Hyperventilation
Anxiety
Pneumomediastinum
Lung tumor
Pleural effusion
Intra-abdominal process
Ascites
Pregnancy*
Massive obesity*

HEENT: head, eyes, ears, nose, and throat; COPD: chronic obstructive pulmonary disease; ARDS: acute respiratory distress syndrome; ACS: acute coronary syndrome; ADHF: acute decompensated heart failure; CO: carbon monoxide.

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*While these conditions do not cause acute dyspnea directly, they can exacerbate symptoms or contribute to other underlying causes.

Graphic 52926 Version 7.0

Types of precautions for infection control[1]

Type of precaution	Selected patients	Major specifications
Standard	All patients	Perform hand hygiene before and after every patient contact.* Gloves, gowns, eye protection as required. Safe disposal or cleaning of instruments and linen. Cough etiquette: Patients and visitors should cover their nose or mouth when coughing, promptly dispose used tissues, and practice hand hygiene after contact with respiratory secretions.
Contact¶	Colonization of any bodily site with multidrug-resistant bacteria (MRSA, VRE, drug-resistant gram-negative organisms) Enteric infections (Norovirus, *Clostridioides difficile**, *Escherichia coli* O157:H7) Viral infections (HSV, VZV, RSV$^{\Delta}$, parainfluenza, enterovirus, rhinovirus$^{\diamond}$, certain coronaviruses [eg, SARS-CoV-2, MERS-CoV]) Scabies Impetigo Noncontained abcesses or decubitus ulcers (especially for *Staphylococcus aureus* and group A *Streptococcus*)§	**In addition to standard precautions:** Private room preferred; cohorting allowed if necessary. Gloves required upon entering room. Change gloves after contact with contaminated secretions Gown required if clothing may come into contact with the patient or environmental surfaces or if the patient has diarrhea. Minimize risk of environmental contamination during patient transport (eg, patient can be placed in a gown). Noncritical items should be dedicated to use for a single patient if possible.
Droplet¶	**Known or suspected:** *Neisseria meningitidis* *Haemophilus influenzae* type B *Mycoplasma pneumoniae* *Bordetella pertussis* Group A *Streptococcus*§ Diphtheria	**In addition to standard precautions:** Private room preferred; cohorting allowed if necessary. Wear a mask when within 3 feet of the patient. Mask the patient during transport. Cough etiquette: Patients and visitors should cover their nose or mouth when coughing,

	Pneumonic plague Influenza Rubella Mumps Adenovirus Parvovirus B19 Rhinovirus◇ Certain coronaviruses¥	promptly dispose used tissues, and practice hand hygiene after contact with respiratory secretions.
Airborne	**Known or suspected:** Tuberculosis Varicella Measles Smallpox Certain coronaviruses¥ Ebola‡	**In addition to standard precautions:** Place the patient in an AIIR (a monitored negative pressure room with at least 6 to 12 air exchanges per hour). Room exhaust must be appropriately discharged outdoors or passed through a HEPA filter before recirculation within the hospital. A certified respirator must be worn when entering the room of a patient with diagnosed or suspected tuberculosis. Susceptible individuals should not enter the room of patients with confirmed or suspected measles or chickenpox. Transport of the patient should be minimized; the patient should be masked if transport within the hospital is unavoidable. Cough etiquette: Patients and visitors should cover their nose or mouth when coughing, promptly dispose used tissues, and practice hand hygiene after contact with respiratory secretions.

This system of isolation precautions is recommended by the United States Healthcare Infection Control Practices Advisory Committee.

MRSA: methicillin-resistant *S. aureus*; VRE: vancomycin-resistant enterococci; HSV: herpes simplex virus; VZV: varicella-zoster virus; RSV: respiratory syncytial virus; SARS-CoV: severe acute respiratory syndrome coronavirus; MERS-CoV: Middle East Respiratory Syndrome coronavirus; AIIR: airborne infection isolation room; HEPA: high-efficiency particulate aerator.

* Alcohol-based hand disinfectant is an acceptable alternative to soap and water in all situations EXCEPT in the setting of norovirus and *C. difficile* infection, for which soap and water should be used.

¶ Many hospitals favor simplifying the approach to isolation precautions for viral respiratory pathogens by placing all patients with suspected viral illness on both contact and droplet precautions.

Δ RSV may be transmitted by the droplet route but is primarily spread by direct contact with infectious respiratory secretions. Droplet precautions are not routinely warranted but are appropriate if the infecting agent is not known, if the patient may be coinfected with other pathogens that require droplet precautions, and/or if there is a chance of exposure to aerosols of infectious respiratory secretions.

◇ The most important route of transmission for rhinovirus is via droplets; contact precautions should be added if copious moist secretions and close contact are likely to occur (eg, young infants).

§ Patients with invasive group A streptococcal infection associated with soft tissue involvement warrant both droplet precautions and contact precautions. Droplet precautions alone are warranted for patients with streptococcal toxic shock or streptococcal pneumonia, as well as for infants and young children in the setting of pharyngitis or scarlet fever. Droplet and contact precautions may be discontinued after the first 24 hours of antimicrobial therapy.

¥ Refer to UpToDate topics on coronaviruses, including SARS-CoV, SARS-CoV-2, and MERS-CoV, for specific information on infection control precautions.

‡ Refer to the UpToDate topic on prevention of Ebola virus infection for full discussion of infection control issues.

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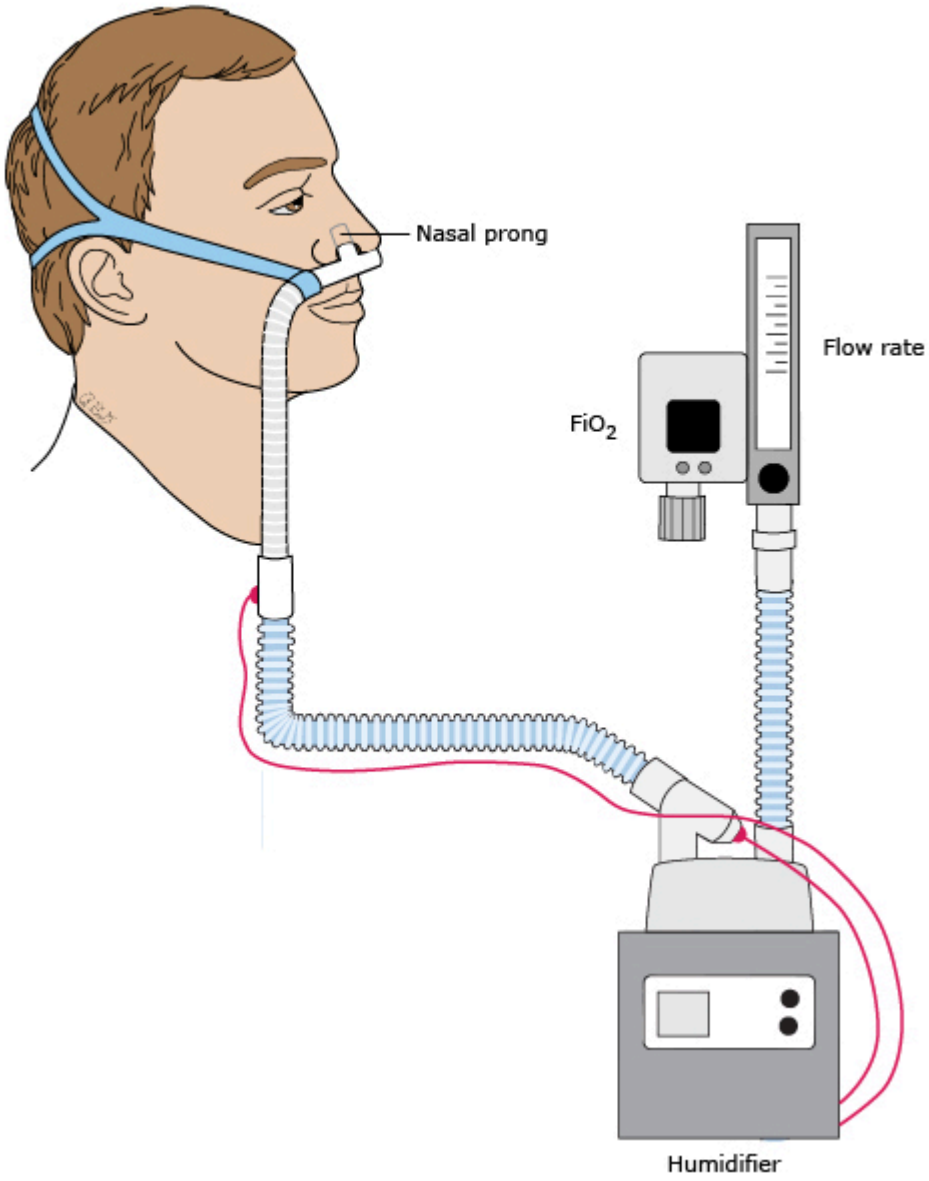
Graphic 50001 Version 26.0

High flow nasal cannulae: Equipment-patient interface



Shown is a typical set up for oxygen delivery through high flow nasal cannulae.

FiO_2: fraction of inspired oxygen.

Graphic 116804 Version 1.0

Dyspnea onset

Acute onset dyspnea	Gradual onset dyspnea
Aspirated foreign body	Pharyngeal/neck infections
Airway trauma	Pulmonary infection
Direct pulmonary injury	Sepsis
Anaphylaxis	High output heart failure
Angioedema	HF exacerbation
Flash pulmonary edema	COPD exacerbations
Pulmonary embolism	Asthma exacerbations*
Pulmonary hemorrhage	Pleural effusion
Pneumothorax	Lung tumor
Cardiac arrhythmia	ARDS
Acute coronary syndrome	Anemia
Cardiac tamponade*	Neuromuscular disease
Organophosphate poisoning	Carbon monoxide poisoning
Hyperventilation	Diabetic ketoacidosis
Anxiety attack	Salicylate poisoning or other toxin-related acidosis
	Pregnancy
	Cardiomyopathy
	Valvular dysfunction
	Ascites
	Acute chest syndrome
	Intraabdominal process (eg, infection, obstruction, ischemic bowel)
	Massive obesity

*May be acute or gradual in onset.

Graphic 64683 Version 1.0

Dyspnea signs

Physical examination finding	Clinical significance	Possible diagnosis
Absent/diminished breath sounds	Decreased air movement	COPD Severe asthma Pneumothorax Tension pneumothorax Hemothorax
Accessory muscle use	Muscle weakness, fatigue	Respiratory failure Severe COPD Severe asthma
Expiratory/mixed stridor	Air flow obstruction below vocal cords	Croup Foreign body Bacterial tracheitis
Inspiratory stridor	Air flow obstruction above vocal cords	Foreign body Epiglottitis Angioedema
Hyperventilation		Acidosis Sepsis Salicylate poisoning Anxiety
JVD with clear lungs	Right heart failure	Cardiac tamponade Pulmonary embolism
JVD with crackles	Right and left heart failure	ADHF ARDS
Heart murmur	Valvular disease	Valvular dysfunction
Hepatojugular reflux	Right heart failure	ADHF
Pulsus paradoxus	Poor right heart filling	Right heart failure Pulmonary embolism Cardiogenic shock Pericardial tamponade

		Asthma exacerbation
Crackles (Rales)	Interalveolar fluid	ADHF ARDS Pneumonia
Wheezes	Obstruction below trachea	Asthma exacerbation Foreign body ADHF COPD

ADHF: Acute decompensated heart failure; ARDS: Acute respiratory distress syndrome; COPD: Chronic obstructive pulmonary disease; JVD: Jugular venous distension;

Graphic 76513 Version 2.0

Causes and troubleshooting erroneous pulse oximetry readings

Problem and potential errors	Solution
Inadequate waveform	
Malposition of probe	Reposition probe, alternate site
Motion artifact	Reposition probe, alternate site
Hypoperfusion	Reposition probe, alternate site, warming
Hypothermia	Use ear or forehead probe, warming
Skin pigment	Measure ABG
Falsely normal or elevated oximetry reading	
Carboxyhemoglobin (eg, carbon monoxide poisoning)	Co-oximetry
High levels of glycohemoglobin A1c	Measure ABG
metHb, sulfHb*	Multiwavelength co-oximetry (metHb), biochemical analysis (sulfHb)
Ambient light	Remove ambient light source
Skin pigment	Measure ABG
Falsely low oximetry reading	
Inadequate waveform	Reposition probe, alternate site
metHb*	Multiwavelength co-oximetry
sulfHb*	Biochemical analysis
HbS and inherited forms of abnormal Hb	Measure HbS and abnormal Hb levels
Severe anemia	Measure ABG
Venous pulsations or congestion	Loosen probe, reposition patient or probe, measure ABG
Ambient light	Remove ambient light source
Nail polish	Remove polish or change site
Vital dyes	Usually transient, measure ABG

ABG: arterial blood gas; metHb: methemoglobin; sulfHb: sulfhemoglobin; HbS: sickle hemoglobin; Hb: hemoglobin; SpO_2: peripheral arterial oxygen saturation; SaO_2: true arterial oxygen saturation.

* In these conditions, SpO_2 is low; when levels of metHb or sulfHb are mildly elevated, SpO_2 underestimates the SaO_2, but when levels are high, the SpO_2 automatically trends towards 85% such that pulse oximetry can overestimate SaO_2.

Adapted from: Chan ED, Chan MM, Chan MM. Pulse oximetry: understanding its basic principles facilitates appreciation of its limitations. Respir Med 2013; 107:789.

Graphic 107546 Version 4.0

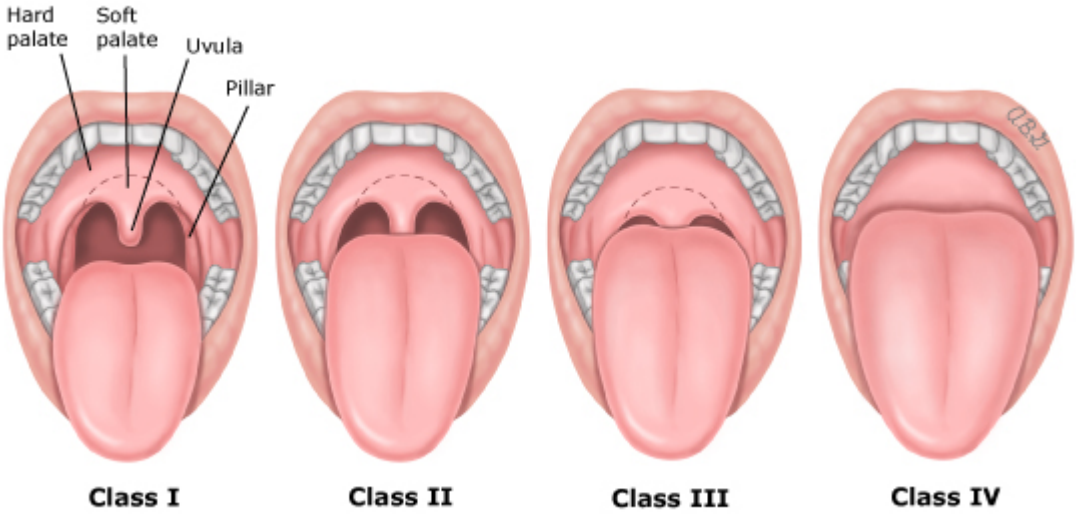
Causes of pulsus paradoxus

Cardiac
Pericardial
▪ Cardiac tamponade
▪ Constrictive pericarditis
Nonpericardial
▪ Right ventricular myocardial infarction
▪ Restrictive cardiomyopathy
Noncardiac
Pulmonary
▪ Asthma and COPD
▪ Obstructive sleep apnea
▪ Pulmonary embolism
▪ Tension pneumothorax
▪ Bilateral pleural effusions
Other
▪ Marked obesity
▪ Hypovolemic shock
▪ Pectus excavatum
▪ Extrinsic cardiac compression

COPD: chronic obstructive pulmonary disease.

Graphic 115612 Version 1.0

The modified Mallampati classification for difficult laryngoscopy and intubation



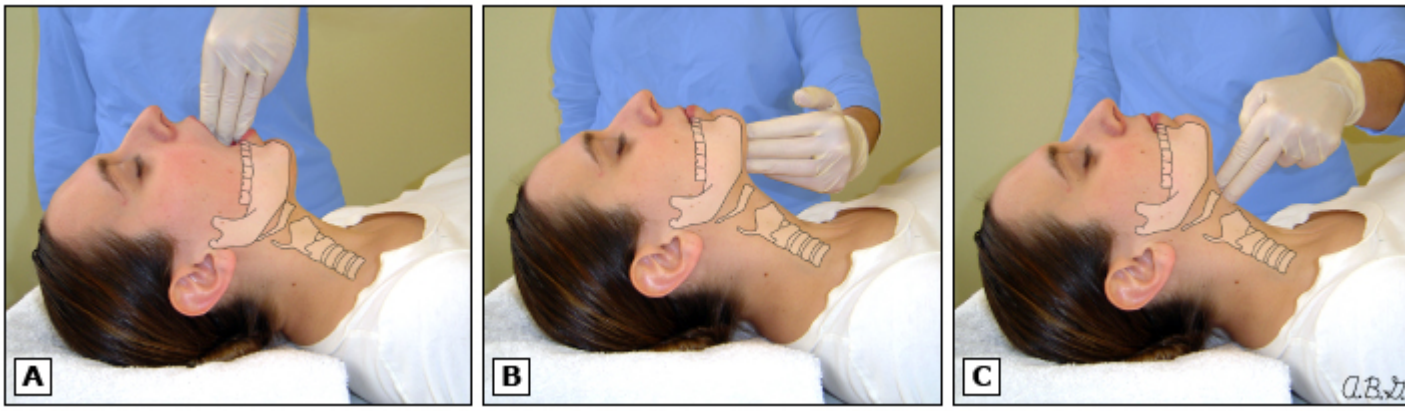
The modified Mallampati classification[1] is a simple scoring system that relates the amount of mouth opening to the size of the tongue and provides an estimate of space available for oral intubation by direct laryngoscopy. According to the Mallampati scale, class I is present when the soft palate, uvula, and pillars are visible; class II when the soft palate and the uvula are visible; class III when only the soft palate and base of the uvula are visible; and class IV when only the hard palate is visible.

Reference:

1. *Samsoon GL, Young JR. Difficult tracheal intubation: a retrospective study. Anaesthesia 1987; 42:487.*

Graphic 75229 Version 10.0

The 3-3-2 rule for identifying a difficult airway



The spatial relationships described here are important determinants of successful direct laryngoscopy. The normal distances shown suggest laryngoscopy would not be difficult for this patient.

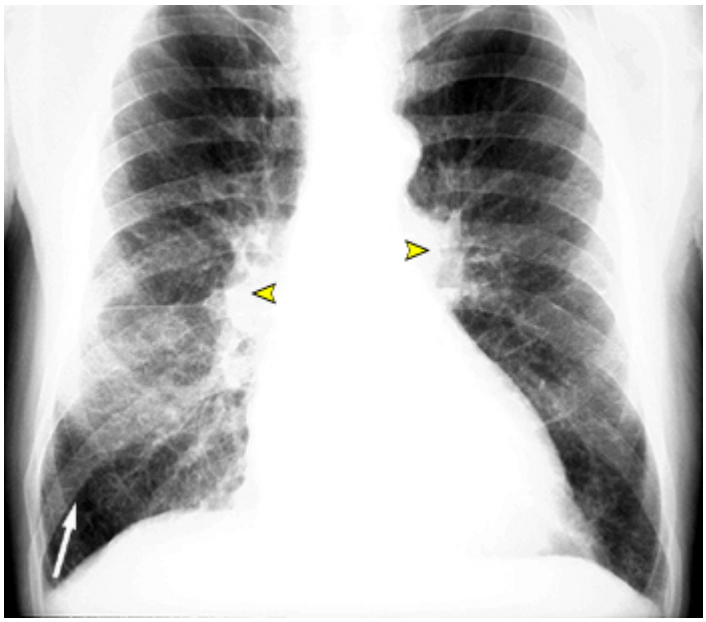
(A) The patient can open their mouth sufficiently to admit 3 of their own fingers.

(B) The distance between the mentum and the neck/mandible junction (near the hyoid bone) is equal to the width of 3 of the patient's fingers.

(C) The space between the superior notch of the thyroid cartilage and the neck/mandible junction, near the hyoid bone, is equal to the width of 2 of the patient's fingers.

Graphic 60507 Version 7.0

Heart failure

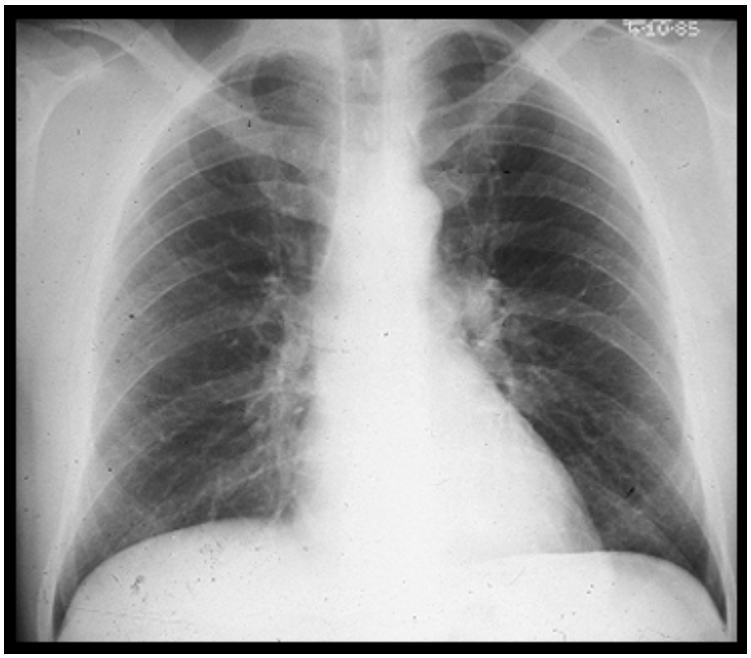


This chest radiograph of a 65-year-old male with dyspnea and orthopnea demonstrates mild pulmonary vascular congestion, septal lymphatic distention (arrow), interstitial veiling, and enlarged hilar shadows (arrowhead), indicative of left ventricular decompensation.

Courtesy of Jonathan Kruskal, MD.

Graphic 61783 Version 3.0

Normal chest radiograph

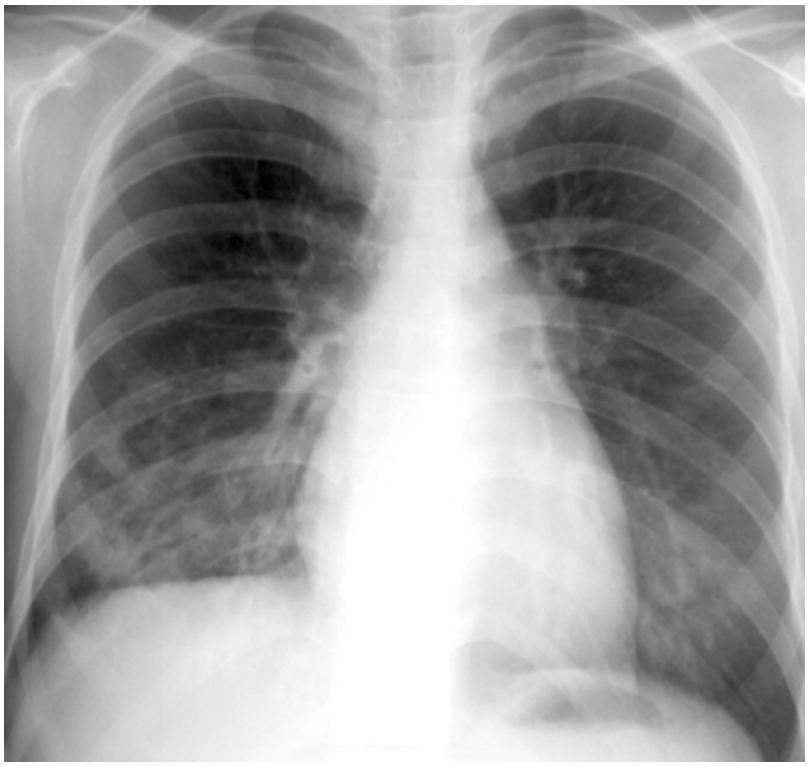


Posteroanterior view of a normal chest radiograph.

Courtesy of Carol M Black, MD.

Graphic 65576 Version 5.0

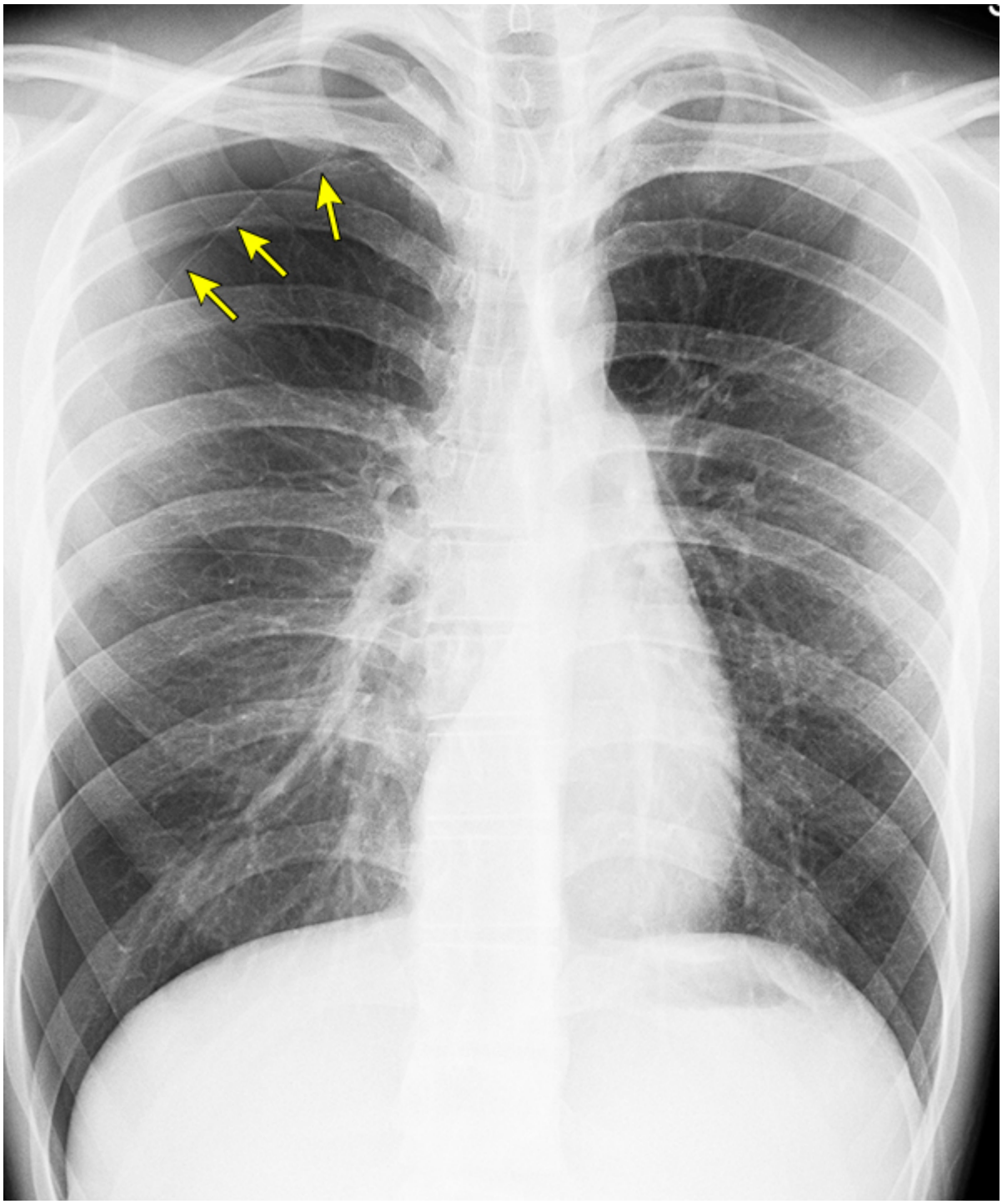
Mycoplasma pneumonia



Chest radiograph shows poorly defined nodular opacities in the right lower lung zone. The patient was a 30-year-old woman with *Mycoplasma pneumoniae* pneumonia.

Graphic 51752 Version 10.0

Chest radiograph of spontaneous pneumothorax

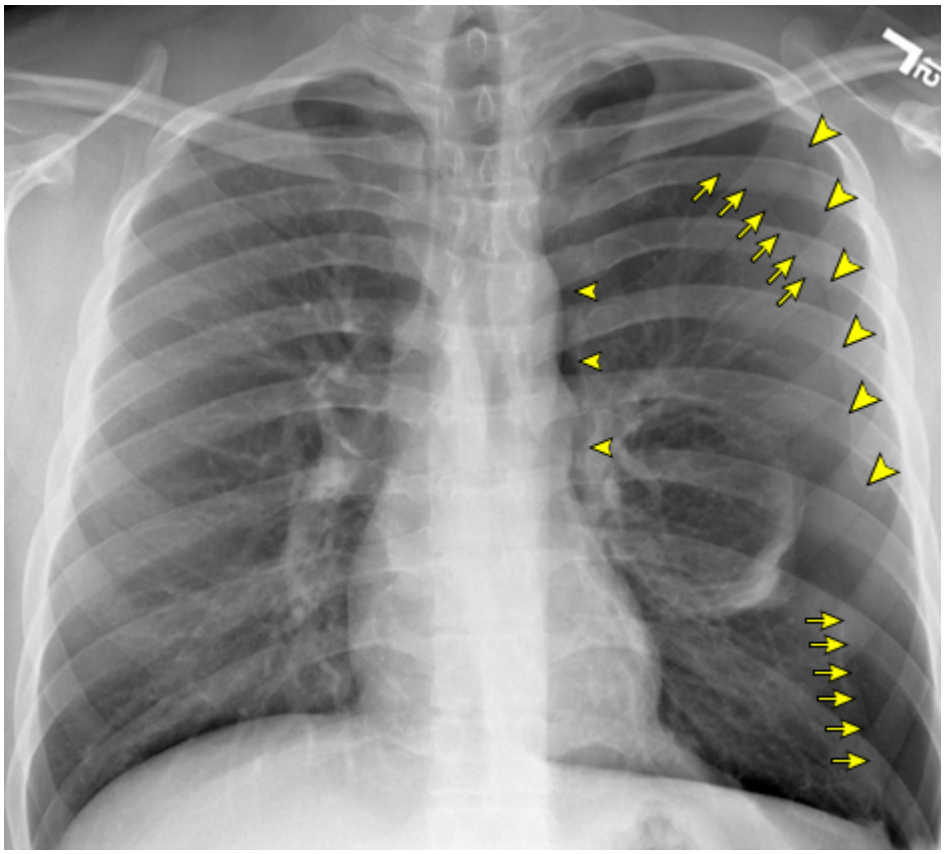


Chest radiograph of a 20-year-old male with small spontaneous right pneumothorax demonstrates the characteristic convex right white visceral pleural line (arrows).

Courtesy of Nestor L Muller, MD, PhD.

Graphic 139477 Version 1.0

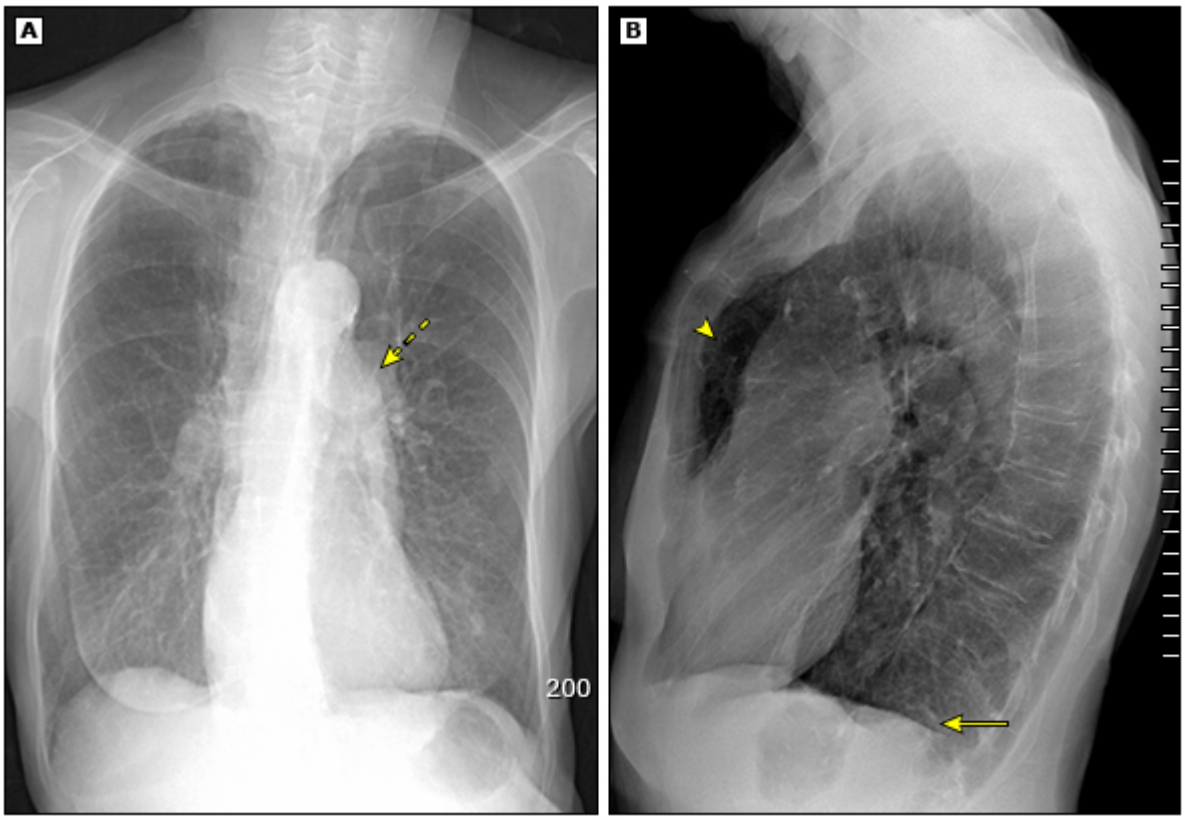
Pneumothorax



This posterior-anterior (PA) radiograph of the chest reveals a left pneumothorax. The lateral border of the lung (arrows) no longer lies adjacent to the chest wall, and the lung parenchyma is contracted, accounting for the lung's abnormal appearance. Note the air and absence of lung markings along the left lateral border of the heart (small arrowheads) and the inferior and medial displacement of the left mainstem bronchus. The border of the scapula (large arrowheads) is sometimes mistaken for a pneumothorax.

Graphic 53428 Version 4.0

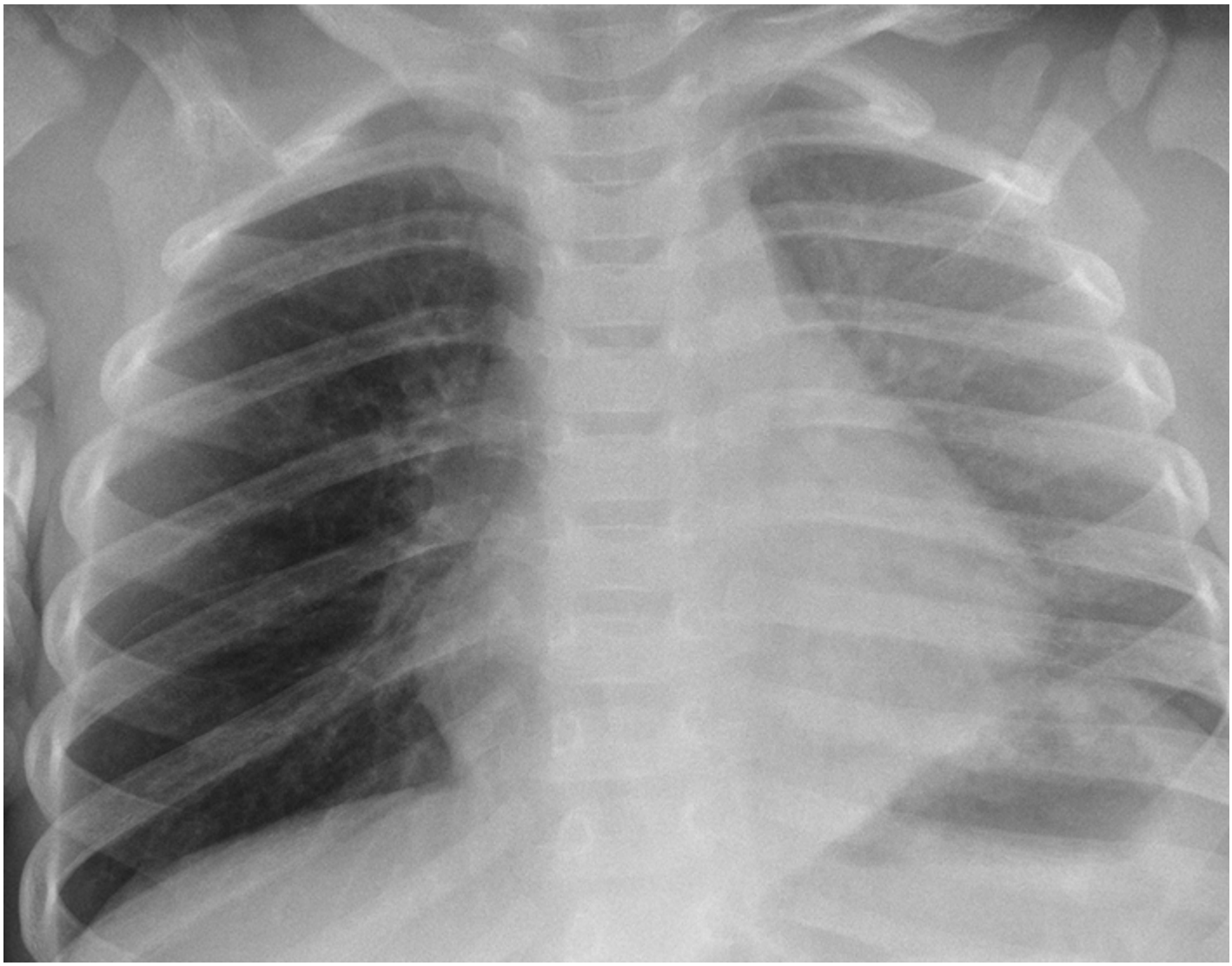
Chest x-ray emphysema



The posteroanterior (A) and lateral (B) chest x-rays of a 71-year-old female with emphysema show increased lung volumes with flattened hemidiaphragms on the lateral examination (arrow) and increase in the retrosternal space (arrowhead). The normal retrosternal airspace is less than 2.5 cm. A prominent pulmonary artery on the posteroanterior view (dashed arrow) reflects secondary pulmonary hypertension.

Graphic 87221 Version 2.0

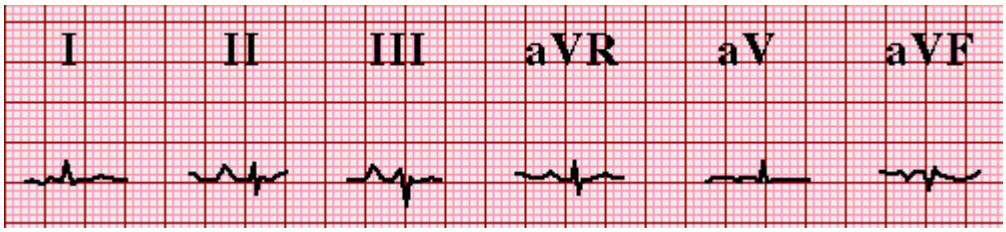
Airway foreign body



An expiratory chest radiograph showing right lung hyperinflation and contralateral underinflation, suggestive of foreign body aspiration. This child's bronchoscopy revealed a nut in the right main bronchus.

Graphic 113198 Version 2.0

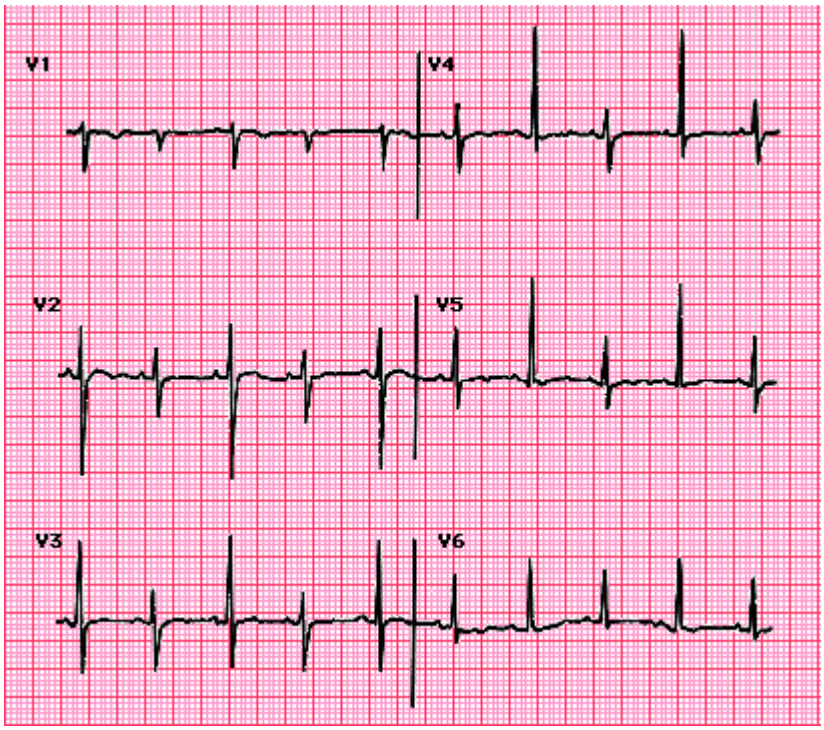
Low voltage in the limb leads



Low voltage of the limb leads is present when the amplitude of the QRS complex in each of the three standard limb leads (1, 2, and 3) is <5 mm. Each large box represents 5 mm (calibration: 1 mV = 10 mm).

Graphic 74856 Version 2.0

Electrical alternans



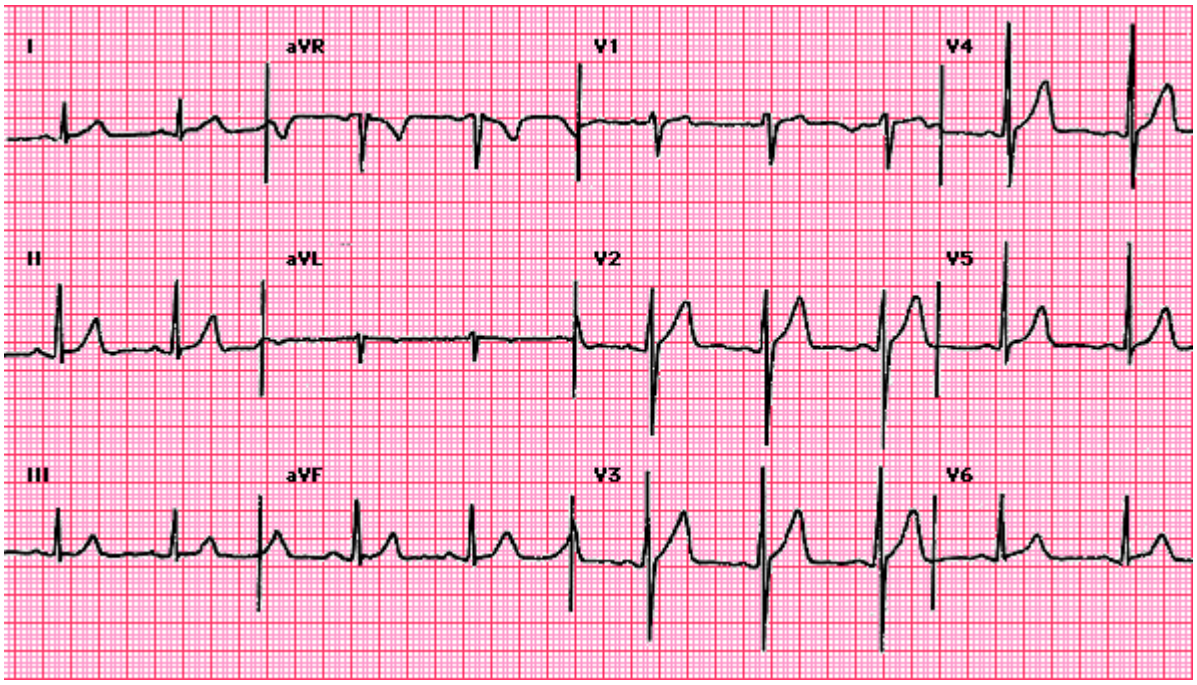
Sinus tachycardia with electrical alternans which is characterized by beat-to-beat alternation in the QRS appearance (best seen in leads V2 to V4). These findings are strongly suggestive of pericardial effusion, usually with cardiac tamponade. The alternating ECG pattern is related to back-and-forth swinging motion of the heart in the pericardial fluid.

ECG: electrocardiogram.

Courtesy of Ary Goldberger, MD.

Graphic 72525 Version 5.0

Normal ECG

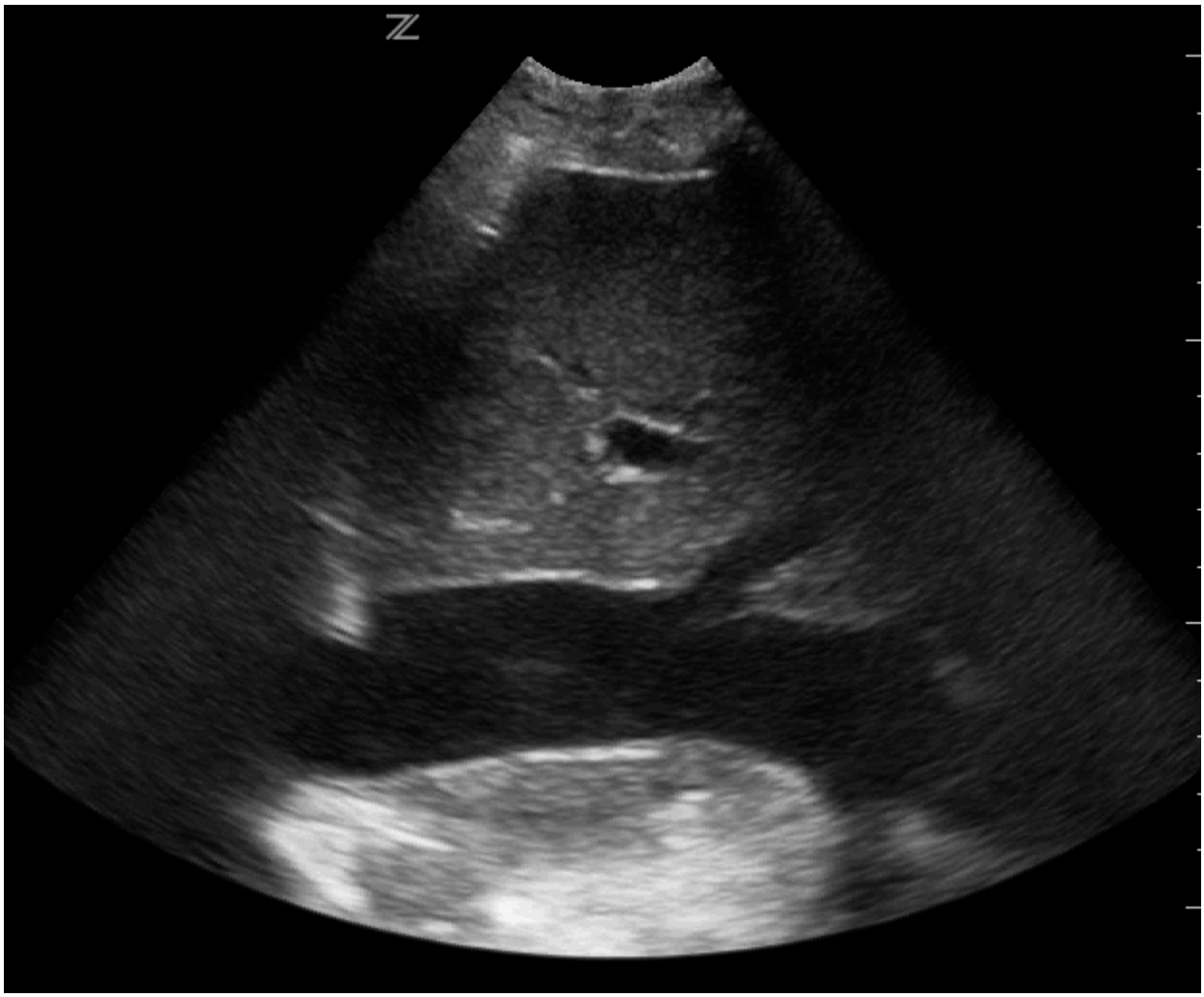


Normal electrocardiogram showing normal sinus rhythm at a rate of 75 beats/minute, a PR interval of 0.14 seconds, a QRS interval of 0.10 seconds, and a QRS axis of approximately 75°.

Courtesy of Ary Goldberger, MD.

Graphic 76183 Version 4.0

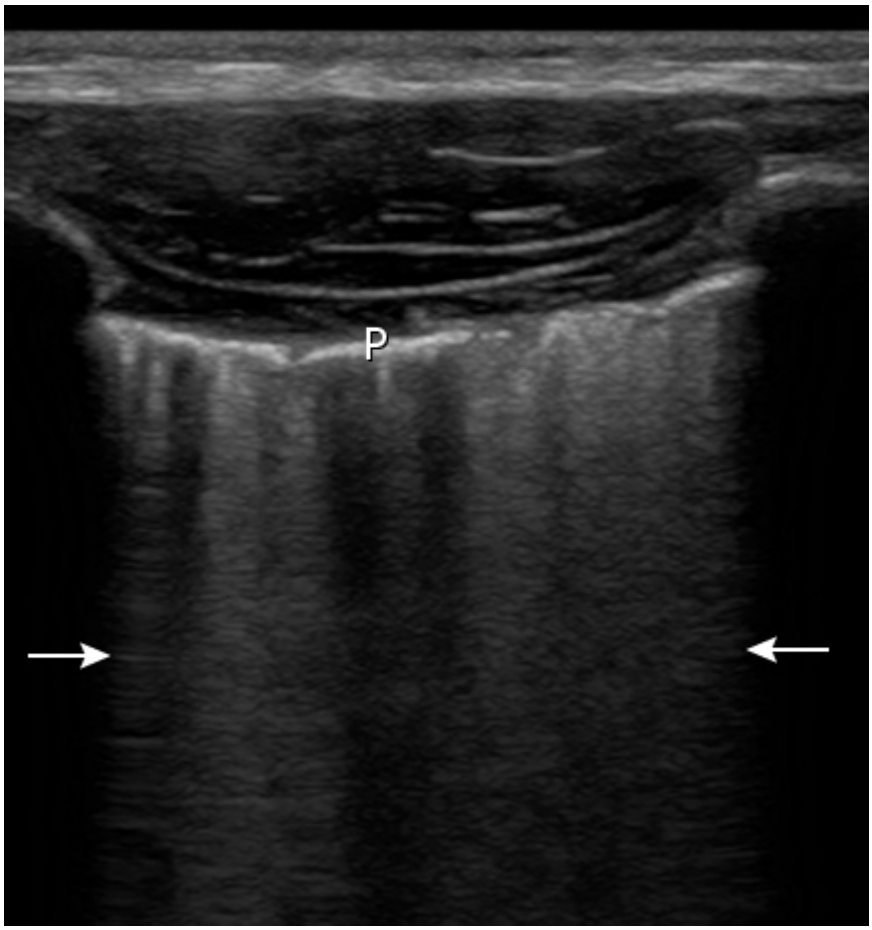
Bedside ultrasound close-up view dilated IVC



This close-up ultrasound image shows a grossly dilated inferior vena cava (IVC). Possible causes include fluid overload, cardiac dysfunction (eg, acute decompensated heart failure), and obstruction (eg, pulmonary embolism, pericardial tamponade, pneumothorax).

Graphic 132356 Version 1.0

FAST: Evaluation for pneumothorax



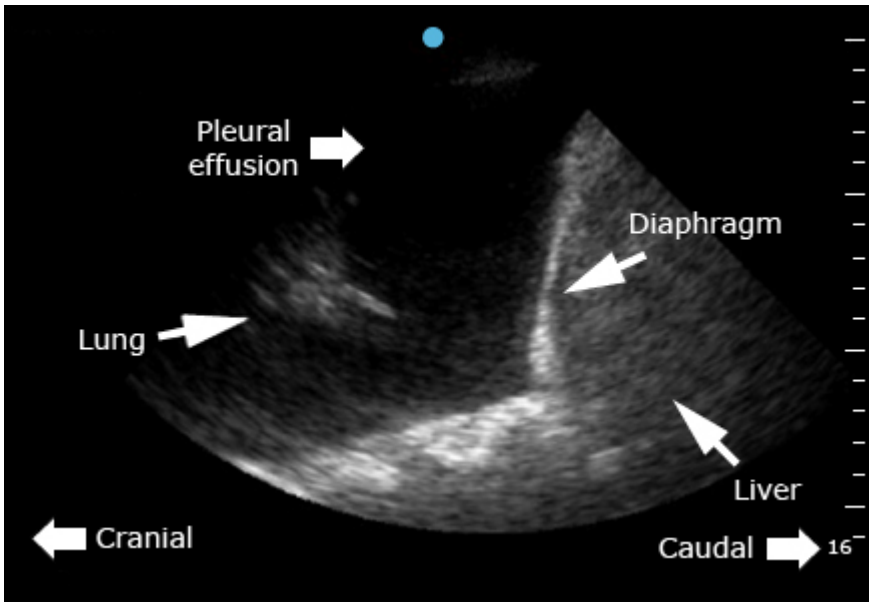
This image of the chest, obtained during a FAST examination, reveals comet tail artifacts (indicated by arrows) extending down from the visceral pleura (P). The presence of these artifacts is one sign indicating the absence of a pneumothorax.

FAST: Focused Assessment with Sonography for Trauma.

Courtesy of Greg Snead, MD.

Graphic 59301 Version 4.0

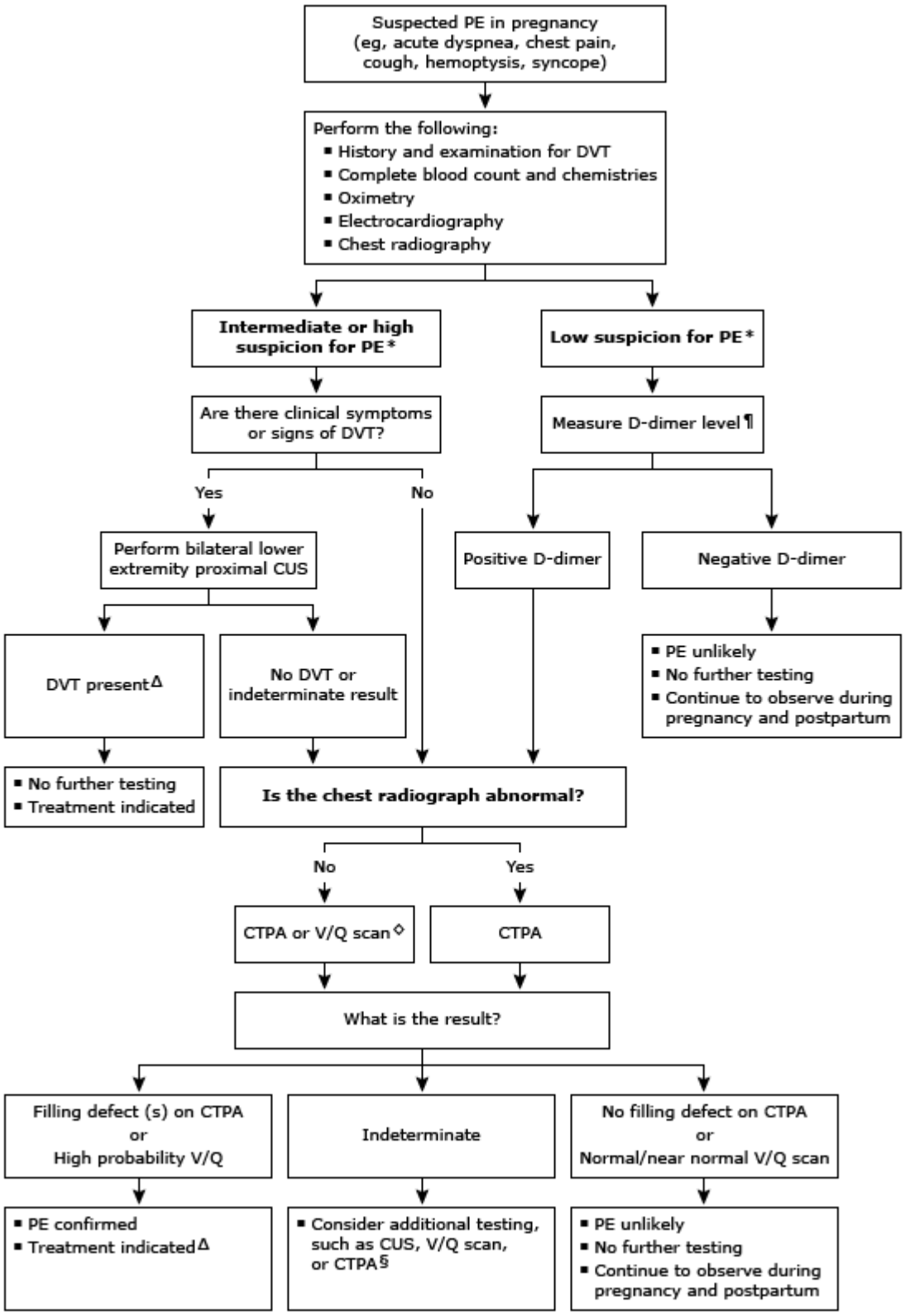
Pleural effusion and diaphragm on thoracic ultrasound



Typical orientation of ultrasound views of a large anechoic pleural effusion on the right. The transducer marker is seen in blue at the top of the image. The effusion is located immediately above the diaphragm, which appears hyperechoic relative to the liver.

Graphic 73746 Version 3.0

Diagnosis PE in pregnancy



PE: pulmonary embolism; DVT: deep venous thrombosis; CUS: compressive ultrasonography; CTPA: computed tomographic pulmonary angiography; V/Q: ventilation/perfusion.

* The suspicion for PE is made by gestalt estimate rather than using preprobability rules (eg, Wells, revised Geneva score, PE rule out criteria [PERC]), which perform poorly in pregnant patients. Similar to

nonpregnant patients, any one or combination of acute-onset dyspnea, pleuritic pain, hemoptysis, syncope, or hypoxemia, particularly in the setting of a normal chest radiograph, should raise the clinical suspicion for PE during pregnancy.

¶ D-dimer becomes less useful with gestational age, such that fewer than 5% of pregnant patients with suspected PE in the third trimester have a negative D-dimer. There are no agreed-upon pregnancy-adjusted reference ranges, further limiting its use in this setting. Thus, D-dimer is less sensitive and specific than in nonpregnant patients, particularly when traditional cutoff values are used (eg, <500 ng/mL, which is the cutoff we use). When measured, we prefer high sensitivity D-dimer assays.

Δ Refer to UpToDate content on treatment of PE.

◇ While in the past V/Q scan was the preferred mode for imaging patients with suspected PE, we now use CTPA as the primary imaging modality. This preference is based upon the practical rationale that CTPA is more readily available than V/Q scanning, may provide an alternate diagnosis in 12 to 13% of cases, is associated with lower doses of radiation than in the past and has better interobserver agreement for radiologists than nuclear scans.

§ Indeterminate results are a diagnostic challenge. We either repeat testing or perform imaging that has not already been done. Magnetic resonance or contrast pulmonary angiography can be considered on a case-by-case basis but are rarely performed and poorly validated in pregnancy. In many cases, clinical judgement is required to inform therapeutic options.

Graphic 140996 Version 1.0

The pulmonary embolism rule out criteria (PERC rule)*[1]

Age <50 years
Heart rate <100 bpm
Oxyhemoglobin saturation ≥95%
No hemoptysis
No estrogen use
No prior DVT or PE
No unilateral leg swelling
No surgery/trauma requiring hospitalization within the prior four weeks

DVT: deep venous thrombosis; PE: pulmonary embolus; bpm: beats per minute.

* This rule is only valid in patients with a low clinical probability of PE (gestalt estimate <15 percent). In patients with a low probability of PE who fullfil all eight criteria, the likelihood of PE is low and no further testing is required. All other patients should be considered for further testing with sensitive D-dimer or imaging.

Reference:

1. *Kline JA, Courtney DM, Kabrhel C, et al. Prospective multicenter evaluation of the pulmonary embolism rule-out criteria. J Thromb Haemost 2008; 6:772.*

Graphic 94941 Version 2.0

Wells criteria and modified Wells criteria: Clinical assessment for pulmonary embolism

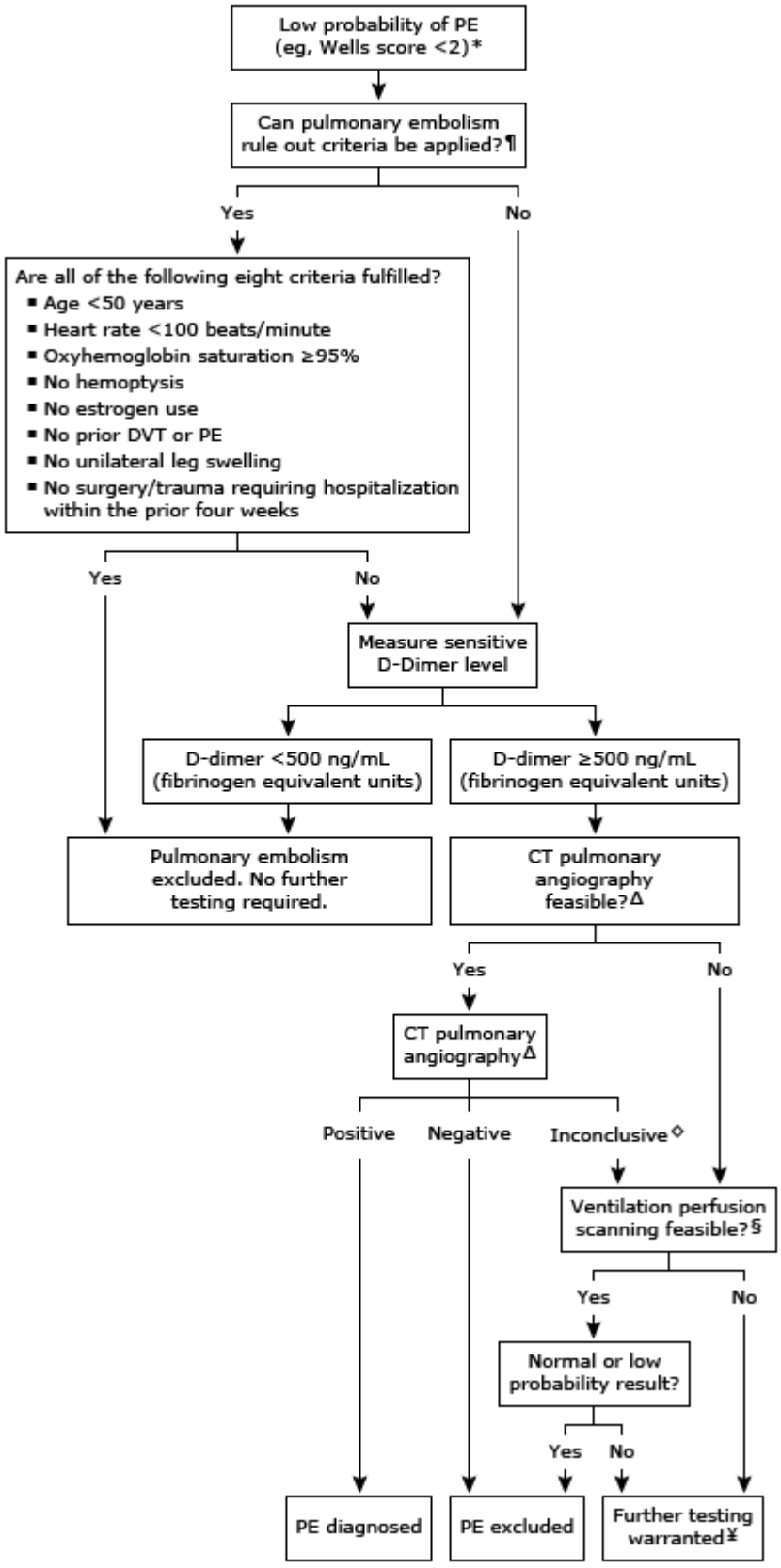
▪ Clinical symptoms of DVT (leg swelling, pain with palpation)	3.0
▪ Other diagnosis less likely than pulmonary embolism	3.0
▪ Heart rate >100	1.5
▪ Immobilization (≥3 days) or surgery in the previous four weeks	1.5
▪ Previous DVT/PE	1.5
▪ Hemoptysis	1.0
▪ Malignancy	1.0
Probability	**Score**
Traditional clinical probability assessment (Wells criteria)	
High	>6.0
Moderate	2.0 to 6.0
Low	<2.0
Simplified clinical probability assessment (Modified Wells criteria)	
PE likely	>4.0
PE unlikely	≤4.0

DVT: deep vein thrombosis; PE: pulmonary embolism.

Data from van Belle A, Buller HR, Huisman MV, et al. Effectiveness of managing suspected pulmonary embolism using an algorithm combining clinical probability, D-dimer testing, and computed tomography. JAMA 2006; 295:172.

Graphic 54767 Version 4.0

Evaluation of the nonpregnant adult with low probability of pulmonary embolism



CT pulmonary angiography is also called chest CT angiogram with contrast and is tailored for to evaluate the pulmonary arteries. A conventional chest CT with contrast is not adequate to exclude PE.

PE: pulmonary embolism; DVT: deep vein thrombosis; CT: computed tomography; PERC: pulmonary embolism rule-out criteria.

* We prefer the Wells criteria to determine the pretest probability of PE, although the modified Geneva score or clinical gestalt is also appropriate. Refer to UpToDate text for details.

¶ PERC is best used in the emergency department in patients with a low clinical pretest probability of PE and is not suitable for other clinical settings or in those with an intermediate or high pretest probability of PE. Some experts choose not to use PERC and proceed directly to sensitive D-Dimer testing.

Δ Feasibility requires adequate scanner technology. Also the patient must be able to lie flat, to cooperate with exam breath-holding instructions, have a body habitus that can fit into scanner, and no contraindications for iodinated contrast.

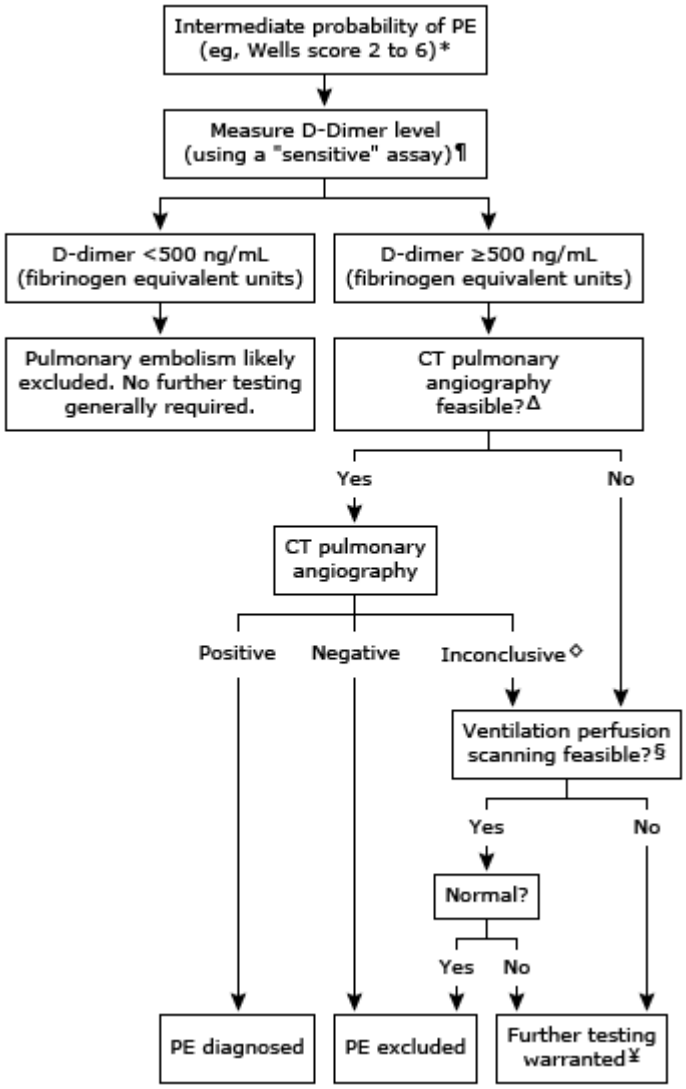
◇ Repeat CT pulmonary angiography for more definitive results may be worthwhile if the factor causing poor image quality can be mitigated (eg, patient more capable of cooperating with positioning and breath-holding instructions). Repeat imaging is unlikely to prove useful if exam was nondiagnostic due to factors such as scanner technology, body habitus, or indwelling metallic foreign bodies.

§ Feasibility requires a chest radiograph demonstrating clear lungs and a patient able to lie still for >30 minutes.

¥ Further testing first involves revisiting the feasibility of CT pulmonary angiography. If CT pulmonary angiography is still not feasible then lower extremity compression ultrasonography with Doppler is appropriate.

Graphic 113963 Version 1.0

Evaluation of the nonpregnant adult with intermediate probability of pulmonary embolism



CT pulmonary angiography is also called chest CT angiogram with contrast and is tailored for to evaluate the pulmonary arteries. A conventional chest CT with contrast is not adequate to exclude PE.

PE: pulmonary embolism; CT: computed tomography.

* We prefer the Wells criteria to determine the pretest probability of PE, although the modified Geneva score or clinical gestalt is also appropriate. Refer to UpToDate text for details.

¶ Some experts proceed directly to CT pulmonary angiography in patients on the higher end of the intermediate risk spectrum (eg, Wells score 4 to 6) or in those with limited cardiopulmonary reserve.

Δ Feasibility requires adequate scanner technology. Also the patient must be able to lie flat, to cooperate with exam breath-holding instructions, have a body habitus that can fit into scanner, and no contraindications for iodinated contrast.

◇ Repeat CT pulmonary angiography for more definitive results may be worthwhile if the factor causing poor image quality can be mitigated (eg, patient more capable of cooperating with positioning and

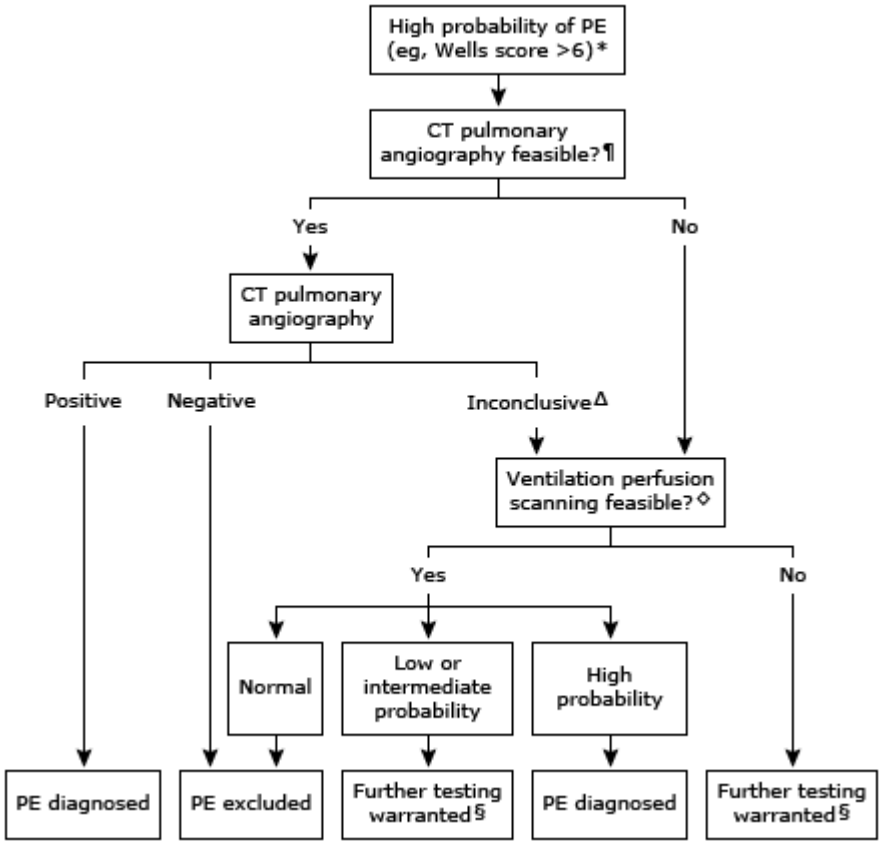
breath-holding instructions). Repeat imaging is unlikely to prove useful if exam was nondiagnostic due to factors such as scanner technology, body habitus, or indwelling metallic foreign bodies.

§ Feasibility requires a chest radiograph demonstrating clear lungs and a patient able to lie still for >30 minutes.

¥ Further testing first involves revisiting the feasibility of CT pulmonary angiography. If CT pulmonary angiography is still not feasible then lower extremity compression ultrasonography with Doppler is appropriate.

Graphic 113962 Version 1.0

Evaluation of the nonpregnant adult with high probability of pulmonary embolism



CT pulmonary angiography is also called chest CT angiogram with contrast and is tailored for to evaluate the pulmonary arteries. A conventional chest CT with contrast is not adequate to exclude PE.

PE: pulmonary embolism; CT: computed tomography.

* We prefer the Wells criteria to determine the pretest probability of PE, although the modified Geneva score or clinical gestalt is also appropriate. Refer to UpToDate text for details.

¶ Feasibility requires adequate scanner technology. Also the patient must be able to lie flat, to cooperate with exam breath-holding instructions, have a body habitus that can fit into scanner, and no contraindications for iodinated contrast.

Δ Repeat CT pulmonary angiography for more definitive results may be worthwhile if the factor causing poor image quality can be mitigated (eg, patient more capable of co-operating with positioning and breath-holding instructions). Repeat imaging is unlikely to prove useful if exam is nondiagnostic from factors such as scanner technology, body habitus, or indwelling metallic foreign bodies.

◇ Feasibility requires a chest radiograph demonstrating clear lungs and a patient able to lie still for >30 minutes.

§ Further testing first involves revisiting the feasibility of CT pulmonary angiography. If CT pulmonary angiography is still not feasible then lower extremity compression ultrasonography with Doppler is appropriate.

Graphic 113961 Version 1.0

Major causes of metabolic acidosis

Mechanism of acidosis	Increased AG	Normal AG
Increased acid production	Lactic acidosis	
	Ketoacidosis	
	Diabetes mellitus	
	Starvation	
	Alcohol associated	
	Ingestions	
	Methanol	
	Ethylene glycol	
	Salicylates	
	Toluene (if early or if kidney function is impaired)	Toluene ingestion (if late and if kidney function is preserved; due to excretion of sodium and potassium hippurate in the urine
	Diethylene glycol	
	Propylene glycol	
	D-lactic acidosis	A component of non-AG metabolic acidosis may coexist due to urinary excretion of D-lactate as Na and K salts (which represents potential HCO_3)
	Pyroglutamic acid (5-oxoproline)	
Loss of bicarbonate or bicarbonate precursors		Diarrhea or other intestinal losses (eg, tube drainage)
		Type 2 (proximal) RTA
		Posttreatment of ketoacidosis
		Carbonic anhydrase inhibitors
		Ureteral diversion (eg, ileal loop)
Decreased renal acid excretion	Severe kidney dysfunction (eGFR <15 to 20 mL/min/1.73 m^2)	Moderate kidney dysfunction (eGFR >15 to 20 mL/min/1.73 m^2
		Type 1 (distal) RTA (hypokalemic)
		Hyperkalemic RTA

		Type 4 RTA (hypoaldosteronism)
		Voltage defect
Large volume infusion of normal saline		Diffusion acidosis

AG: anion gap; RTA: renal tubular acidosis.

Graphic 77464 Version 15.0

Predicted peak expiratory flow (PEF; liters/minute) for females age 20 to 70 years

Age (years)	Height				
	55 inches/140 cm	60 inches/152 cm	65 inches/165 cm	70 inches/178 cm	75 inches/190 cm
20	333	372	418	468	517
25	340	379	425	475	524
30	344	383	429	479	528
35	344	383	430	479	529
40	342	381	427	477	526
45	336	376	422	471	521
50	328	367	413	463	512
55	316	323	401	451	501
60	301	341	387	436	486
65	283	323	369	419	468
70	263	302	348	398	447

For patients who do not know their personal best PEF, this table can help estimate an expected "personal best." This table uses a prediction equation for White females age 20 to 70 years. Refer to UpToDate calculator for values for additional age, height, and race/ethnicity parameters.

Reference:

1. *Hankinson JL, Odencrantz JR, Fedan KB. Spirometric reference values from a sample of the general U.S. population. Am J Respir Crit Care Med. 1999; 159(1):179.*

Graphic 62839 Version 10.0

Predicted peak expiratory flow (PEF; liters/minute) for males age 20 to 70 years

Age (years)	Height				
	60 inches/152 cm	65 inches/165 cm	70 inches/178 cm	75 inches/191 cm	80 inches/203 cm
20	477	539	606	678	748
25	484	546	613	685	756
30	488	550	616	688	759
35	487	549	616	688	758
40	483	545	611	683	754
45	474	536	603	675	746
50	462	436	591	663	733
55	446	508	575	646	717
60	426	488	554	626	697
65	402	464	530	602	673
70	374	436	503	574	645

For patients who do not know their personal best PEF, this table can help estimate an expected "personal best." This table uses a prediction equation for White males, age 20 to 70 years. Refer to UpToDate calculator for values for additional age, height, and race/ethnicity parameters.

Reference:

1. *Hankinson JL, Odencrantz JR, Fedan KB. Spirometric reference values from a sample of the general U.S. population. Am J Respir Crit Care Med. 1999; 159(1):179.*

Graphic 57257 Version 10.0