CHAPTER 17 Dyspnea

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PERSPECTIVE

Dyspnea is the term applied to the sensation of breathlessness and the patient's reaction to that sensation. It is an uncomfortable awareness of breathing difficulties that in the extreme manifests as "air hunger." Dyspnea is often ill defined by patients, who may describe the feeling as shortness of breath, chest tightness, or difficulty breathing. Dyspnea results from a variety of conditions, ranging from nonurgent to life-threatening. Neither the clinical severity nor the patient's perception correlates well with the seriousness of underlying pathology and may be affected by emotions, behavioral and cultural influences, and external stimuli.[1,2]

The following terms may be used in the assessment of the dyspneic patient:

Tachypnea: A respiratory rate greater than normal. Normal rates range from 44 cycles/min in a newborn to 14 to 18 cycles/min in adults.

Hyperpnea: Greater than normal minute ventilation to meet metabolic requirements.

Hyperventilation: A minute ventilation (determined by respiratory rate and tidal volume) that exceeds metabolic demand. Arterial blood gases (ABG) characteristically show a normal partial pressure of oxygen (Po_2) with an uncompensated respiratory alkalosis (low partial pressure of carbon dioxide [Pco_2] and elevated pH).

Dyspnea on exertion: Dyspnea provoked by physical effort or exertion. It often is quantified in simple terms, such as the number of stairs or number of blocks a patient can manage before the onset of dyspnea.

Orthopnea: Dyspnea in a recumbent position. It usually is measured in number of pillows the patient must use to lie in bed (e.g., two-pillow orthopnea).

Paroxysmal nocturnal dyspnea: Sudden onset of dyspnea occurring while reclining at night, usually related to the presence of congestive heart failure.

Epidemiology

Dyspnea is a common presenting complaint among emergency department patients of all ages. Causes vary widely and may be due to a benign, self-limited condition or significant pathology that can produce long-term morbidity and premature mortality.

Pathophysiology

The actual mechanisms responsible for dyspnea are unknown. Normal breathing is controlled both centrally by the respiratory control center in the medulla oblongata, as well as peripherally by chemoreceptors located near the carotid bodies, and mechanoreceptors in the diaphragm and skeletal muscles.[3] Any imbalance between these sites is perceived as dyspnea. This imbalance generally results from ventilatory demand being greater than capacity.[4]

The perception and sensation of dyspnea are believed to occur by one or more of the following mechanisms: increased work of breathing, such as the increased lung resistance or decreased compliance that occurs with asthma or chronic obstructive pulmonary disease (COPD), or increased respiratory drive, such as results from severe hypoxemia, acidosis, or centrally acting stimuli (toxins, central nervous system events). Pulmonary stretch receptors also are thought to play a role.

DIAGNOSTIC APPROACH

Differential Considerations

Dyspnea is subjective and has many different potential causes.[5] The differential diagnosis list can be divided into acute and chronic causes, of which many are pulmonary. Other etiologies include cardiac, metabolic, infectious, neuromuscular, traumatic, and hematologic (Table 17-1).

Pivotal Findings

History

Duration of Dyspnea. Chronic or progressive dyspnea usually denotes primary cardiac or pulmonary disease.[6] Acute dyspneic spells may result from asthma exacerbation; infection; pulmonary embolus; intermittent cardiac dysfunction; psychogenic causes; or inhalation of irritants, allergens, or foreign bodies.

Onset of Dyspnea. Sudden onset of dyspnea should lead to consideration of pulmonary embolism (PE) or spontaneous pneumothorax. Dyspnea that builds slowly over hours or days may represent a flare of asthma or COPD; pneumonia; recurrent, small pulmonary emboli; congestive heart failure; or malignancy.

Table 17-1 Differential Diagnoses for Acute Dyspnea

ORGAN SYSTEM	CRITICAL DIAGNOSES	EMERGENT DIAGNOSES	NONEMERGENT DIAGNOSES
Pulmonary	Airway obstruction Pulmonary embolus Noncardiogenic edema Anaphylaxis Ventilatory failure	Spontaneous pneumothorax Asthma Cor pulmonale Aspiration Pneumonia	Pleural effusion Neoplasm Pneumonia (CAP score < = 70) COPD
Cardiac	Pulmonary edema Myocardial infarction Cardiac tamponade	Pericarditis	Congenital heart disease Valvular heart disease Cardiomyopathy
Primarily Associated with Normal or Increased Respiratory Effort			
Abdominal		Mechanical interference Hypotension, sepsis from ruptured viscus, bowel obstruction, inflammatory/infectious process	Pregnancy Ascites Obesity
Psychogenic			Hyperventilation syndrome Somatization disorder Panic attack
Metabolic/endocrine	Toxic ingestion DKA	Renal failure Electrolyte abnormalities Metabolic acidosis	Fever Thyroid disease
Infectious	Epiglottitis	Pneumonia (CAP score < = 70)	Pneumonia (CAP score < = 70)
Traumatic	Tension pneumothorax Cardiac tamponade Flail chest	Simple pneumothorax, hemothorax Diaphragmatic rupture	Rib fractures
Hematologic	Carbon monoxide poisoning Acute chest syndrome	Anemia	
Primarily Associated with Decreased Respiratory Effort			
Neuromuscular	CVA, intracranial insult Organophosphate poisoning	Multiple sclerosis Guillain-Barré syndrome Tick paralysis	ALS Polymyositis Porphyria

ALS, amyotrophic lateral sclerosis; CAP, community-acquired pneumonia; COPD, chronic obstructive pulmonary disease; CVA, cerebrovascular accident; DKA, diabetic ketoacidosis.

Positional Changes. Orthopnea can result from left-sided heart failure, COPD, or neuromuscular disorders. One of the earliest symptoms seen in patients with diaphragmatic weakness from neuromuscular disease is orthopnea.[7] Paroxysmal nocturnal dyspnea is most common in patients with left-sided heart failure,[6] but also can be found in COPD. Exertional dyspnea commonly is associated with COPD, but also can be seen with poor cardiac reserve and abdominal loading. Abdominal loading, caused by ascites, obesity, or pregnancy, leads to elevation of the diaphragm, resulting in less effective ventilation and dyspnea.

Trauma. Dyspnea can result from trauma, causing fractured ribs, flail chest, hemothorax, pneumothorax, diaphragmatic rupture, pericardial effusion, cardiac tamponade, or neurologic injury.

Symptoms

Patient descriptions of dyspnea vary significantly and generally correlate poorly with severity. Fever suggests an infectious cause. Anxiety may point to panic attack or psychogenic dyspnea, if no organic cause can be isolated. PE or myocardial infarction may present with isolated dyspnea or with associated chest pain, particularly if the pain is constant, dull, or visceral.[8,9] If the pain is sharp and worsened by deep breathing but not by movement, pleural effusion and pleurisy or pleural irritation from pneumonia or PE are possible. Spontaneous pneumothorax also may produce sharp pain with deep breathing that is not worsened by movement.

Signs

Physical signs in dyspneic patients may be consistent with specific illnesses (Table 17-2). Physical findings found in specific diseases also can be grouped as presenting patterns (Table 17-3).

Ancillary Studies

Specific findings obtained from the history and physical examination should be used to determine which ancillary studies are needed (Table 17-4). Bedside oxygen saturation determinations, or selective use of ABGs when oximetry is not reliable, are useful in determining the degree of hypoxia and the need for supplemental oxygen or assisted ventilation. An additional resource for quickly assessing ventilatory status is noninvasive waveform capnography. Using both the end-tidal CO_2 value and the shape of the waveform itself can be helpful in assessing the adequacy of ventilations as well as potential causes of the dyspnea (See Chapter 3). An electrocardiogram may be useful if the etiology is cardiac or suggests acute pulmonary hypertension.

Serum electrolytes may suggest less common possible causes, such as hypokalemia, hypophosphatemia, diabetic ketoacidosis, or hypocalcemia. A complete blood count may identify severe anemia or thrombocytopenia associated with sepsis. The white blood cell count is not sufficiently sensitive or specific to be of discriminatory value. Cardiac markers and D-dimer assay may be useful in pursuing etiologies such as

Table 17-2 Pivotal Findings in Physical Examination

SIGN	PHYSICAL FINDING	DIAGNOSES TO CONSIDER
Vital signs	Tachypnea	Pneumonia, pneumothorax
	Hypopnea	Intracranial insult, drug/toxin ingestion
	Tachycardia	PE, traumatic chest injury
	Hypotension	Tension pneumothorax
	Fever	Pneumonia, PE
General appearance	Cachexia, weight loss	Malignancy, acquired immune disorder, mycobacterial infection
	Obesity	Hypoventilation, sleep apnea, PE
	Pregnancy	PE
	Barrel chest	COPD
	"Sniffing" position	Epiglottitis
	"Tripoding" position	COPD/asthma with severe distress
	Traumatic injury	Pneumothorax (simple, tension), rib fractures, flail chest, hemothorax, pulmonary contusion
Skin/nails	Tobacco stains/odor	COPD, malignancy, infection
	Clubbing	Chronic hypoxia, intracardiac shunts or pulmonary vascular anomalies
	Pallid skin/conjunctivae	Anemia
	Muscle wasting	Neuromuscular disease
	Bruising	Chest wall: rib fractures, pneumothorax
		Diffuse: thrombocytopenia, chronic steroid use, anticoagulation
	Subcutaneous emphysema	Rib fractures, pneumothorax, tracheobronchial disruption
	Hives, rash	Allergic reaction, infection, tick-borne illness
Neck	Stridor	Upper airway edema/infection, foreign body, traumatic injury, anaphylaxis
	JVD	Tension pneumothorax, COPD or asthma exacerbation, fluid overload/CHF, PE
Lung examination	Wheezes	CHF, anaphylaxis
		Bronchospasm
	Rales	CHF, pneumonia, PE
	Unilateral decrease	Pneumothorax, pleural effusion, consolidation, rib fractures/ contusion, pulmonary contusion
	Hemoptysis	Malignancy, infection, bleeding disorder, CHF
	Sputum production	Infection (viral, bacterial)
	Friction rub	Pleurisy
	Abnormal respiratory pattern (e.g., Cheyne-Stokes)	Intracranial insult
Chest examination	Crepitance or pain on palpation	Rib or sternal fractures
	Subcutaneous emphysema	Pneumothorax, tracheobronchial rupture
	Thoracoabdominal desynchrony	Diaphragmatic injury with herniation; cervical spinal cord trauma
	Flail segment	Flail chest, pulmonary contusion
Cardiac examination	Murmur	PE
	S_3 or S_4 gallop	PE
	S_2 accentuation	PE
	Muffled heart sounds	Cardiac tamponade
Extremities	Calf tenderness, Homans' sign	PE
	Edema	CHF
Neurologic examination	Focal deficits (motor, sensory, cognitive)	Stroke, intracranial hemorrhage causing central abnormal respiratory drive; if long-standing, risk of aspiration pneumonia
	Symmetrical deficits	Neuromuscular disease
	Diffuse weakness	Metabolic or electrolyte abnormality (hypocalcemia, hypomagnesemia, hypophosphatemia), anemia
	Hyporeflexia	Hypermagnesemia
	Ascending weakness	Guillain-Barré syndrome

CHF, congestive heart failure; COPD, chronic obstructive pulmonary disease; JVD, jugular venous distention; PE, pulmonary embolism.

cardiac ischemia or PE. Amino-terminal pro-B-type natriuretic peptide (NT-proBNP) analysis adds both diagnostic and prognostic value for several causes of dyspnea, including heart failure, PE, and ischemic cardiac disease.[9-11] Combinations of specific serum markers can also help define pathology.[12-14] Specialized tests, such as ventilation-perfusion scans, chest computed tomography, pulmonary angiography, or, rarely, conventional pulmonary angiography, may confirm the diagnosis of PE.[15] If dyspnea is believed to be upper airway in origin, direct or fiberoptic laryngoscopy or a soft tissue lateral radiograph of the neck may be useful.

DIFFERENTIAL DIAGNOSIS

The range and diversity of pathophysiologic states that produce dyspnea make a simple algorithmic approach difficult.[16] After initial stabilization and assessment, findings from the history, physical examination, and ancillary testing are collated to match patterns of disease that produce dyspnea. This process is updated periodically as new information becomes available. Table 17-3 presents recognizable patterns of disease for common dyspnea-producing conditions, along with specific associated symptoms.

Table 17-3 Diagnostic Table: Patterns of Diseases Often Resulting in Dyspnea

DISEASE	HISTORY: (DYSPNEA)	ASSOCIATED SYMPTOMS	SIGNS AND PHYSICAL FINDINGS	TESTS
Pulmonary embolism	HPI: abrupt onset, pleuritic pain, immobility (travel, recent surgery) PMH: malignancy, DVT, PE, hypercoagulability, oral contraception, obesity	Diaphoresis, exertional dyspnea	Tachycardia, tachypnea, low-grade fever	ABG (A-a gradient), D-dimer ECG (dysrhythmia, right heart strain) CXR (Westermark sign, Hampton's hump) $\dot{V}/\dot{Q}$, spiral CT, MRV Pulmonary angiogram Ultrasound positive for DVT
Pneumonia	Fever, productive cough, chest pain	Anorexia, chills, nausea, vomiting, exertional dyspnea, cough	Fever, tachycardia, tachypnea, rales or decreased breath sounds	CXR, CBC, sputum and blood cultures
Bacterial	SH: tobacco use			ABG if hypoxia suspected Waveform capnography if altered mental status
Viral	Exposure (e.g., influenza, varicella)			
Opportunistic	Immune disorder, chemotherapy			
Fungal/parasitic	Exposure (e.g., birds), indolent onset	Episodic fever, nonproductive cough		
Pneumothorax	Abrupt onset ± trauma, chest pain, thin males more likely to have spontaneous pneumothorax	Localized chest pain	Decreased breath sounds, subcutaneous emphysema, chest wall wounds or instability	CXR: pneumothorax, rib fractures, hemothorax
Simple				Ultrasound positive for pneumothorax
Tension	Decompensation of simple pneumothorax	Diaphoresis	Above JVD, tracheal deviation, muffled heart sounds, cardiovascular collapse	Clinical diagnosis: requires immediate decompression. May verify using bedside ultrasound
COPD/asthma	Tobacco use, medication noncompliance, URI symptoms, sudden weather change PMH: environmental allergies FH: asthma	Air hunger, diaphoresis	Retractions, accessory muscle use, tripoding, cyanosis	CXR: rule out infiltrate, pneumothorax, atelectasis (mucus plug) Waveform capnography
Malignancy	Weight loss, tobacco or other occupational exposure	Dysphagia	Hemoptysis	CXR, chest CT: mass, hilar adenopathy, focal atelectasis
Fluid overload	Gradual onset, dietary indiscretion or medication noncompliance, chest pain PMH: recent MI, diabetes, CHF	Worsening orthopnea, PND	JVD, peripheral edema, S_3 or S_4 gallop, new cardiac dysrhythmia, hepatojugular reflux	CXR: pleural effusion, interstitial edema, Kerley B lines, cardiomegaly ECG: ischemia, dysrhythmia NT-proBNP
Anaphylaxis	Abrupt onset, exposure to allergen	Dysphagia	Oral swelling, stridor, wheezing, hives	

ABG, arterial blood gas; CBC, complete blood count; CHF, congestive heart failure; CT, computed tomography; CXR, chest x-ray; DVT, deep vein thrombosis; ECG, electrocardiogram; FH, family history; HPI, history of present illness; JVD, jugular venous distention; MI, myocardial infarction; MRV, magnetic resonance venography; NT-proBNP, amino-terminal pro-brain natriuretic peptide; PE, pulmonary embolism; PMH, past medical history; PND, paroxysmal nocturnal dyspnea; SH, social history; URI, upper respiratory infection.

Table 17-4 Ancillary Testing in the Dyspneic Patient

CATEGORY	TEST	FINDINGS/POTENTIAL DIAGNOSES
Laboratory	Pulse oximetry, selective ABG use	Hypoxia, hyperventilation (muscular weakness, intracranial event)
	Waveform capnography	CO_2 retention (COPD, sleep apnea), obstructive or restrictive pulmonary pattern Metabolic versus respiratory acidosis (DKA, ingestions) A-a gradient (PE) Elevated carboxyhemoglobin (inhalation injury or CO poisoning)
	Complete blood count	WBC Increase: infection, stress demargination, hematologic malignancy Decrease: neutropenia, sepsis Hgb/Hct: anemia, polycythemia Smear: abnormal Hgb (i.e., sickling), inclusions Platelets: thrombocytopenia (marrow toxicity)
	Chemistry	BUN/Cr: acute/chronic renal failure K/Mg/Phos: low levels resulting in muscular weakness Glucose: DKA D-dimer: abnormal clotting activity NT-proBNP: heart failure, PE Troponin: cardiac ischemia or infarct
Cardiac	ECG	Ischemia, dysrhythmia, $S_1Q_3T_3$ (PE), right heart strain
	Echocardiogram	Pulmonary hypertension, valvular disorders Wall motion abnormalities related to ischemia, intracardiac shunts
Radiologic	Chest radiograph	Bony structures: fractures, lytic lesions, pectus, kyphoscoliosis Mass: malignancy, cavitary lesion, infiltrate, foreign body Diaphragm: eventration, elevation of hemidiaphragm, bowel herniation Mediastinum: adenopathy (infection, sarcoid), air Cardiac silhouette: enlarged (cardiomyopathy, fluid overload) Soft tissue: subcutaneous air Lung parenchyma: blebs, pneumothorax, effusions (blood, infectious), interstitial edema, local consolidation, air bronchograms, Hampton's hump, Westermark's sign
	$\dot{V}/\dot{Q}$ scan	PE
	Pulmonary angiogram	PE, intervention (thrombolysis)
	CT	Mass lesion, adenopathy, trauma, PE
	MRI	PE, bony and soft tissue lesions, vascular abnormality
	Soft tissue neck radiograph	Epiglottitis, foreign body
	Ultrasound	Pneumothorax, pleural effusion, impaired cardiac function or pericardial effusion
Fiberoptic	Bronchoscopy	Mass lesion, foreign body Intervention (stenting, biopsy)
	Laryngoscopy	Mass lesion, edema, epiglottitis, foreign body

A-a, alveolar-arterial; ABG, arterial blood gas; BUN, blood urea nitrogen; CHF, congestive heart failure; CO, carbon monoxide; COPD, chronic obstructive pulmonary disease; Cr, creatinine; CT, computed tomography; DKA,.diabetic ketoacidosis; ECG, electrocardiogram; MRI, magnetic resonance imaging; NT-proBNP, amino-terminal pro-brain natriuretic peptide; PE, pulmonary embolism; V/Q, ventilation-perfusion; WBC, white blood cell.

Critical Diagnoses

Several critical diagnoses should be promptly considered to determine the best treatment options to stabilize the patient. Tension pneumothorax is such a critical diagnosis. If a dyspneic patient has diminished breath sounds on one side, ipsilateral hyper-resonance, severe respiratory distress, hypotension, and oxygen desaturation, prompt decompression of presumptive tension pneumothorax is necessary. Bedside ultrasonography may assist in confirming pneumothorax. If obstruction of the upper airway is evidenced by dyspnea and stridor, early, definitive assessment and intervention must occur in the emergency department or operating room. Complete obstruction by a foreign body warrants the Heimlich maneuver until the obstruction is relieved or the patient is unconscious, followed rapidly by direct laryngoscopy. Congestive heart failure and pulmonary edema can produce dyspnea and respiratory failure and should be treated as soon as possible if severe.[17] Significant dyspnea and wheezing can be seen in anaphylaxis and must be treated promptly to prevent further deterioration. Severe bronchospastic exacerbations of asthma at any age may lead rapidly to respiratory failure and arrest and should receive vigorous attention, including continuous or frequent administration of a beta-agonist aerosol.[18] As mentioned earlier, waveform capnography is a valuable tool for assessing the severity and determining the cause of respiratory distress.

Emergent Diagnoses

Asthma and COPD exacerbations can result in marked dyspnea with bronchospasm and decreased ventilatory volumes.[19] Sudden onset of dyspnea with a decreased oxygen saturation on room air accompanied by sharp chest pain may represent PE.[15] Dyspnea accompanied by decreased breath sounds and tympany to percussion on one side is seen with spontaneous pneumothorax. Dyspnea associated with decreased respiratory effort may represent a neuromuscular process, such as multiple sclerosis, Guillain-Barré syndrome, or myasthenia gravis.[14] Unilateral rales, cough, fever, and dyspnea usually indicate pneumonia.

Figure 17-1 provides an algorithm for assessment and stabilization of a dyspneic patient. The initial division is based on the degree of breathing effort associated with the symptoms.

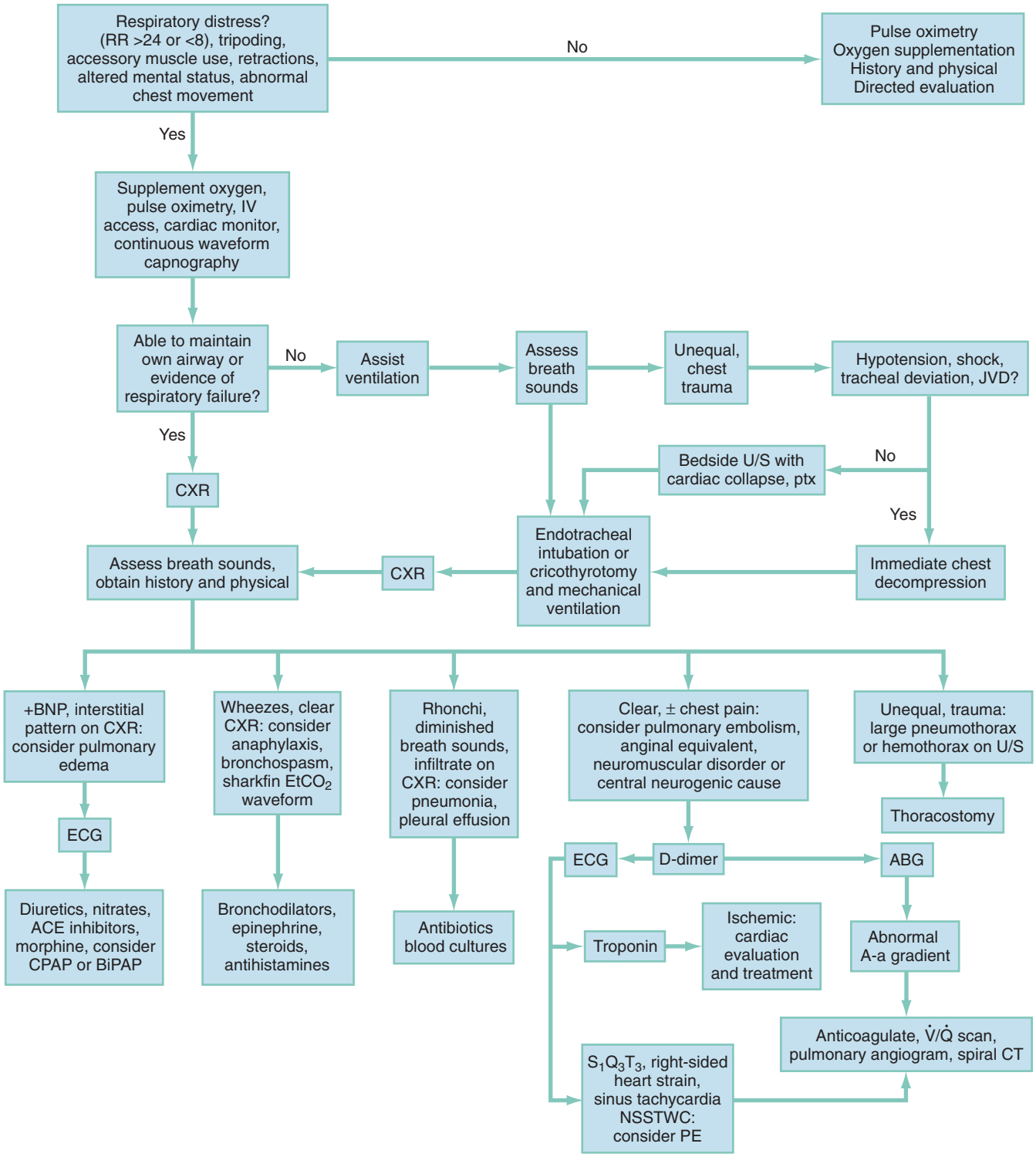

Figure 17-1. Rapid assessment and stabilization of a dyspneic patient. ABG, arterial blood gas; ACE, angiotensin-converting enzyme; BiPAP, biphasic positive airway pressure; BNP, B-type natriuretic peptide; CO, carbon monoxide; CPAP, continuous positive airway pressure; CT, computed tomography; CXR, chest x-ray; ECG, electrocardiogram; $EtCO_2$, end-tidal carbon dioxide; IV, intravenous; JVD, jugular venous distention; NSSTWC, nonspecific ST wave changes (on ECG); PE, pulmonary embolism; RR, respiratory rate; $\dot{V}/\dot{Q}$, ventilation-perfusion ratio; U/S, ultrasound.

The most critical diagnoses must be considered first and appropriate intervention taken as necessary.

All patients experiencing dyspnea, regardless of possible cause, should be promptly transported to the treatment area. Bedside pulse oximetry should be obtained, and the patient should be placed on a cardiac monitor. If the pulse oximetry is less than 98% saturated on room air, the patient should be placed on supplemental oxygen either by nasal cannula or mask depending on the degree of desaturation detected. If necessary, the patient should be intubated, and breathing should be assisted with manual or mechanical ventilation.

When the airway has been secured, rapid assessment of the patient's appearance and vital signs can help determine the need for further stabilization. Decreased mental alertness, inability to speak in more than one-word syllables, or certain types of body positioning, signal the presence of significant

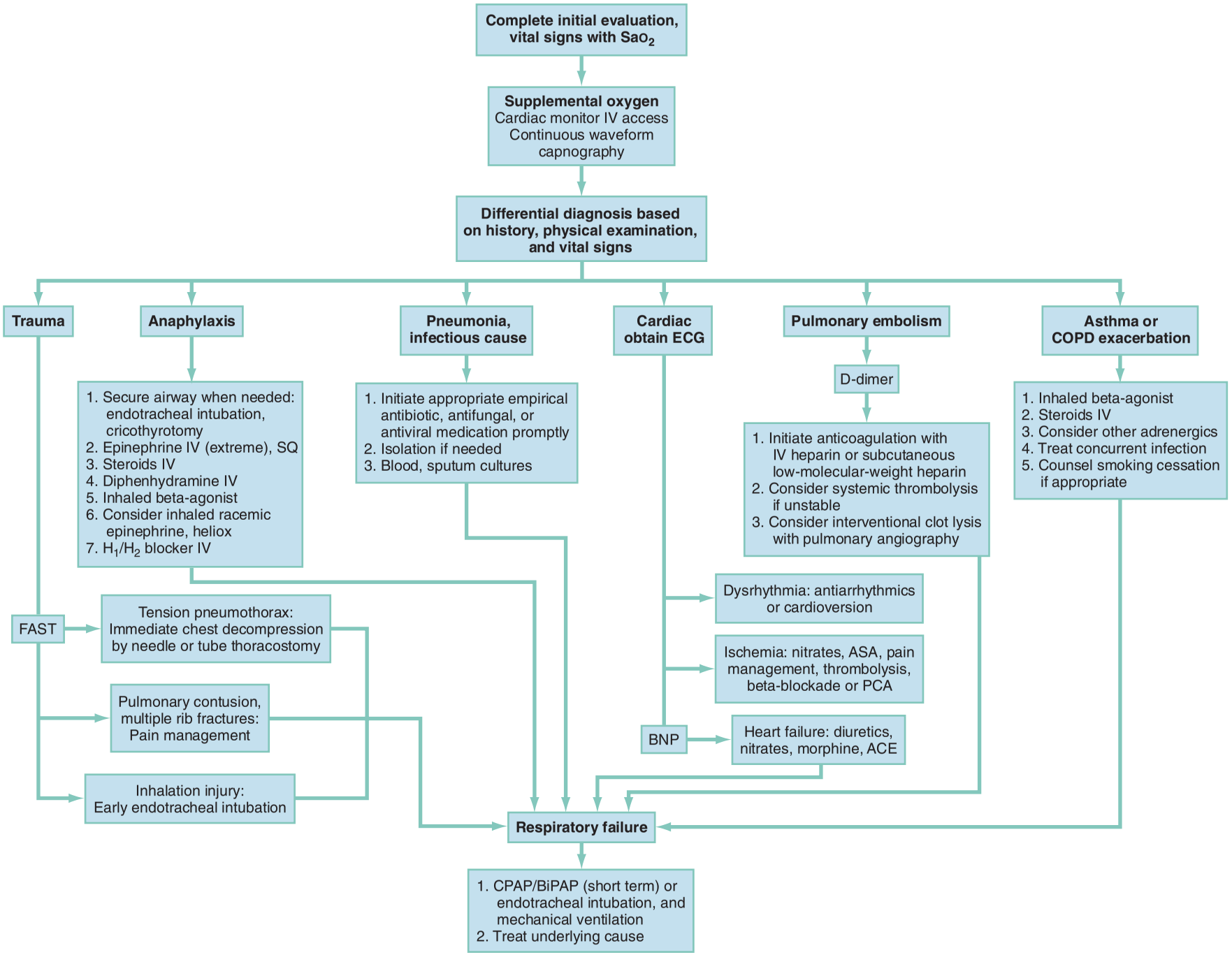

Figure 17-2. Clinical guidelines for emergency department management of dyspnea. ACE, angiotensin-converting enzyme; ASA, acetylsalicylic acid; COPD, chronic obstructive pulmonary disease; CPAP/BiPAP, continuous positive airway pressure/biphasic positive airway pressure; ECG, electrocardiogram; IV, intravenous; PCA, patient-controlled analgesia; SQ, subcutaneous.

respiratory distress and the need for rapid intervention. After stabilization has occurred, the cause of the dyspnea can be further investigated.

EMPIRICAL MANAGEMENT AND DISPOSITION

The management algorithm for dyspnea (Fig. 17-2) outlines the approach to treatment for most identifiable diseases. Unstable patients or patients with critical diagnoses must be stabilized and may require admission to an intensive care unit. Emergent patients who have improved in the emergency department may be admitted to an intermediate care unit. Patients diagnosed with urgent conditions in danger of deterioration without proper treatment or patients with severe comorbidities, such as diabetes, immunosuppression, or cancer, may also require admission for observation and treatment.

Most patients in the nonurgent category can be treated as outpatients if good medical follow-up can be arranged. If dyspnea persists despite therapy and no definitive cause has been delineated, the best course of action is hospitalization for observation and ongoing evaluation. If no definitive diagnosis can be obtained and the symptoms have abated, the patient may be discharged with good medical follow-up and instructions to return if symptoms recur.

The references for this chapter can be found online by accessing the accompanying Expert Consult website.