CHAPTER 22

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]

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 (ABGs) 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 uses to lie in bed (eg, 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 very common presenting complaint among emergency department (ED) patients of every age. Causes vary widely, and range from benign, self-limited conditions to critical pathology that can produce short-term mortality and long-term morbidity.[2,3]

Pathophysiology

The actual mechanisms responsible for dyspnea are only beginning to be specifically described. Normal breathing is controlled both centrally by the respiratory control center in the medulla oblongata and peripherally by chemoreceptors located near the carotid bodies, but there are numerous sensory inputs that affect the feeling of dyspnea, including pulmonary stretch receptors and mechanoreceptors in the diaphragm and skeletal muscles.[4] Imbalances among these inputs can be perceived as dyspnea and may manifest as increased work of breathing, due to increased lung resistance or decreased compliance in asthma or chronic obstructive pulmonary disease (COPD). Alternatively, the imbalances of these inputs may also manifest as increased respiratory drive—ie, resulting from severe hypoxemia, acidosis, or centrally acting stimuli (toxins, central nervous system events).[5]

DIAGNOSTIC APPROACH

Differential Diagnosis Considerations

Dyspnea is subjective and has many different potential causes. The differential diagnosis can be divided into acute and chronic causes, of which many are pulmonary. Other causes include cardiac, metabolic, infectious, neuromuscular, traumatic, and hematologic conditions (Table 22.1).

Pivotal Findings

Symptoms

Patient descriptions of dyspnea vary significantly and generally correlate poorly with severity, although the complaint of dyspnea alone is predictive of mortality.

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.

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 but also occurs in COPD.[6] 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.

Anxiety or overwhelming fear, particularly if it precedes the onset of dyspnea, may point to panic attack or psychogenic dyspnea, but organic causes should be considered first. PE or myocardial infarction may cause isolated dyspnea with or without associated chest pain, particularly if the pain is constant, dull, or visceral.[8] Pain that is sharp and worsened by deep breathing but not by movement may indicate pleural effusion, pleurisy,

TABLE 22.1

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 (CAP score >70)	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 or infectious process	Pregnancy Ascites obesity
Psychogenic			Hyperventilation syndrome Somatization disorder Panic attack
Metabolic or 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 Neurologic injury	Rib fractures
Hematologic	Carbon monoxide or cyanide 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.

or pleural irritation from pneumonia or PE. 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 22.2). For example, fever suggests an infectious cause, somnolence or obtundation may indicate hypercarbia, agitation can be associated with hypoxia, and trauma may produce dyspnea through various injuries. Physical findings found in specific diseases also can be grouped according to presenting patterns (Table 22.3). Some findings have improved predictive value for specific pathologies when combined with laboratory testing in validated risk stratification tools.[9-11]

Ancillary Testing

Specific findings obtained from the history and physical examination should be used to determine which ancillary studies are needed (Table 22.4). 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. In patients with abnormal values, a venous blood gas (VBG) is a less painful alternative to ABG to determine pH.[12] VBG is less reliable for P_{CO_2} or accurate numeric correlation to arterial hypercapnia, although a normal venous P_{CO_2} has a strong negative predictive value, and values greater than 45 mm Hg are highly sensitive in predicting arterial hypercarbia.[13,14] The more invasive ABG is useful when an accurate P_{CO_2} or P_{O_2} is important. An additional resource for quickly assessing ventilatory status is noninvasive waveform capnography. End-tidal carbon dioxide ($ETCO_2$) values correlate well with arterial carbon dioxide (CO_2), and the shape of the capnogram can be helpful in assessing the adequacy of ventilations, as well as underlying causes of the dyspnea (see Chapter 5).[15] An electrocardiogram may be useful if history or physical examination findings suggest heart failure, ischemic cardiac disease, dysrhythmia, or pulmonary hypertension. Bedside ultrasound is useful to rapidly assess multiple parameters that can focus and guide therapy. For example, thoracic ultrasound can quickly visualize pleural effusion, pulmonary edema with B lines, pneumothorax when "sandy beach" and "comet tail" signs are absent, cardiac dysfunction by evaluating myocardial contractility and estimating ejection fraction (EF), or pericardial effusion and tamponade.[16,17] Abdominal ultrasound can assess

TABLE 22.2

Pivotal Findings in Physical Examination

SIGN	PHYSICAL FINDING	DIAGNOSES TO CONSIDER
Vital signs	Tachypnea	Pneumonia, pneumothorax
	Hypopnea	Intracranial insult, drug or 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 or asthma with severe distress
	Traumatic injury	Pneumothorax (simple, tension), rib fractures, diaphragmatic injury, flail chest, hemothorax, pulmonary contusion
Skin and nails	Tobacco stains or odor	COPD, malignancy, infection
	Clubbing	Chronic hypoxia, intracardiac shunts, or pulmonary vascular anomalies
	Pallid skin or 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 or infection, foreign body, traumatic injury, anaphylaxis
	JVD	Tension pneumothorax, COPD or asthma exacerbation, fluid overload or CHF, PE, cardiac tamponade
Lung examination	Wheezes	CHF, anaphylaxis
	Bronchospasm	
	Rales	CHF, pneumonia, PE
	Unilateral decrease	Pneumothorax, pleural effusion, consolidation, rib fractures or contusion, pulmonary contusion
	Hemoptysis	Malignancy, infection, bleeding disorder, CHF
	Sputum production	Infection (viral, bacterial)
	Friction rub	Pleurisy
	Abnormal respiratory pattern (eg, 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, pericardial effusion
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.

TABLE 22.3

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	Pulse oximetry, ABG (A-a gradient), D-dimer ECG (dysrhythmia, right-sided heart strain) CXR (Westermark sign, Hampton's hump), 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			Pulse oximetry Waveform capnography if altered mental status; ABG if capnography unavailable and acid-base derangement or hypercarbia suspected
Viral	Exposure (eg, influenza, varicella)			
Opportunistic	Immune disorder, chemotherapy			
Fungal or parasitic	Exposure (eg, 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 Ultrasound: Pneumothorax, pleural effusion
Simple				Ultrasound positive for pneumothorax
Tension	Decompensation of simple pneumothorax	Diaphoresis	JVD, tracheal deviation, muffled heart sounds, cardiovascular collapse	Clinical diagnosis: Requires immediate decompression. May verify via bedside ultrasound
COPD or asthma	Tobacco use, medication noncompliance, URI symptoms, sudden weather change PMH: Environmental allergies FH: Asthma	Air hunger, diaphoresis	Retractions, accessory muscle use, tripoding, cyanosis "Shark fin" capnograph	CXR: Rule out infiltrate, pneumothorax, atelectasis (mucus plug) Ultrasound: Distinguish from heart failure 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 and/or ultrasound: Pleural effusion, interstitial edema, Kerley B lines, cardiomegaly ECG: Ischemia, dysrhythmia BNP
Anaphylaxis	Abrupt onset, exposure to allergen	Dysphagia	Oral swelling, stridor, wheezing, hives	

A-a, Alveolar-arterial; *ABG,* arterial blood gas; *BNP,* B-type natriuretic peptide; *CBC,* complete blood count; *CHF,* congestive heart failure; *COPD,* chronic obstructive pulmonary disease; *CT,* computed tomography; *CXR,* chest x-ray examination; *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; *PE,* pulmonary embolism; *PMH,* past medical history; *PND,* paroxysmal nocturnal dyspnea; *SH,* social history; *URI,* upper respiratory infection.

TABLE 22.4

Ancillary Testing in the Dyspneic Patient

CATEGORY	TEST	FINDINGS AND 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 (ie, sickling), inclusions Platelets: Thrombocytopenia (marrow toxicity) Chemistry BUN, Cr: Acute or chronic renal failure K, Mg, Phos: Low levels resulting in muscular weakness Glucose: DKA D-dimer: Abnormal clotting activity BNP: Heart failure, PE Troponin: Cardiac ischemia or infarct
Cardiac	ECG	Ischemia, dysrhythmia, $S_1Q_3T_3$ (PE), right-sided 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
	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; *BNP,* B-type natriuretic peptide; *BUN,* blood urea nitrogen; *CO,* carbon monoxide; *CO_2,* carbon dioxide; *COPD,* chronic obstructive pulmonary disease; *Cr,* creatinine; *CT,* computed tomography; *DKA,* diabetic ketoacidosis; *ECG,* electrocardiogram; *Hct,* hematocrit; *Hgb,* hemoglobin; *K,* potassium; *Mg,* magnesium; *MRI,* magnetic resonance imaging; *PE,* pulmonary embolism; *Phos,* phosphate;*WBC,* white blood cell.

intravascular volume by quantifying inferior vena cava size and compressibility.[18] Extremity ultrasound can reveal deep venous thrombosis.[19]

Serum electrolytes may confirm metabolic acidosis or a less common cause, such as hypokalemia, hypophosphatemia, 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.

Expanded availability of specific blood biomarkers relevant to emergent evaluation of dyspnea provides improved immediate decision support and allows for short- and long-term prognostication.[20,21] These include cardiac markers and D-dimer assay, which are useful in pursuing causes, such as cardiac ischemia or venous thromboembolic disease. B-type natriuretic peptide (BNP) analysis adds both diagnostic and prognostic value for several causes of dyspnea, including heart failure, PE, and ischemic cardiac disease.[22]

If venous thromboembolism is suspected, D-dimer testing, with or without chest computed tomographic angiography, duplex venous ultrasonography, or, rarely, ventilation-perfusion scanning, is performed on patients preselected based on clinical decision rules.[23] 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.

DIAGNOSTIC ALGORITHM

The range and diversity of pathophysiologic conditions that produce dyspnea render a simple algorithmic approach difficult. The primary branch point is the determination of whether the dyspnea primarily is cardiopulmonary or toxic-metabolic in origin. After initial assessment, stabilization and symptom relief in critical patients, 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 22.3 presents recognizable patterns of disease for common dyspnea-producing conditions, along with specific associated symptoms.

Critical Diagnoses

Several critical diagnoses should be promptly considered to determine the best treatment options to stabilize the patient. Tension pneumothorax is a critical condition that is diagnosed by history and physical examination. If a dyspneic patient has no breath sounds on one side, ipsilateral hyper-resonance, severe respiratory distress, hypotension, and oxygen desaturation, prompt decompression of presumptive tension pneumothorax is indicated. Jugular venous distension may or may not be apparent and its absence does not rule out the condition. Bedside ultrasonography can confirm pneumothorax in less obvious cases. If dyspnea and stridor indicate upper airway obstruction, early, definitive assessment, and intervention occur in the ED 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 for foreign body removal. Heart failure and pulmonary edema can produce dyspnea and respiratory failure and require prompt intervention to support ventilation and gas exchange if severe. Significant dyspnea and wheezing in anaphylaxis require immediate use of parenteral epinephrine in addition to supportive measures. 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 and steroid therapy.[24] Ultrasound may also be of benefit in rapidly distinguishing between COPD and heart failure, as well as other pathologies.[25,26] As mentioned earlier, waveform capnography is a valuable adjunct for assessing the severity and determining the cause of respiratory distress. Presumptive anticoagulation or even thrombolytics may be appropriate in patients with suspected significant PE even prior to diagnostic testing.

Emergent Diagnoses

Asthma and COPD exacerbations can result in marked dyspnea with bronchospasm and decreased ventilatory volumes.[27] Sudden onset of dyspnea with a decreased oxygen saturation on room air accompanied by sharp chest pain may represent PE. Dyspnea accompanied by decreased breath sounds and tympany on 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. Unilateral rales, cough, fever, and dyspnea usually indicate pneumonia.

Figure 22.1 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. The most critical diagnoses are considered first, and appropriate intervention undertaken.

All patients experiencing dyspnea, regardless of possible cause, should be promptly evaluated in the treatment area. Bedside pulse oximetry readings should be obtained, and the patient placed on a cardiac monitor. If the pulse oximetry result is less than 94% on room air, supplemental oxygen either by nasal cannula or mask should be considered, depending on the degree of desaturation. In patients with somnolence or obtundation, hypercarbia and respiratory failure should be considered as possible etiologies. If necessary, ventilation should be assisted manually or mechanically, either noninvasively for the short term, or with the patient tracheally intubated for airway protection for prolonged ventilation.[28]

Decreased mental alertness, inability to speak in more than one-syllable words, or certain types of body positioning signal the presence of significant respiratory distress and the need for rapid intervention. After the airway has been secured, rapid assessment of the patient's appearance and vital signs can help determine the need for further stabilization and the cause of the dyspnea can be further investigated.

Empirical Management

The management algorithm for dyspnea (Fig. 22.2) 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 with ED management 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 medical follow-up can be arranged. If dyspnea persists despite therapy and no definitive cause has been delineated, the preferred 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 medical follow-up and instructions to return if symptoms recur.

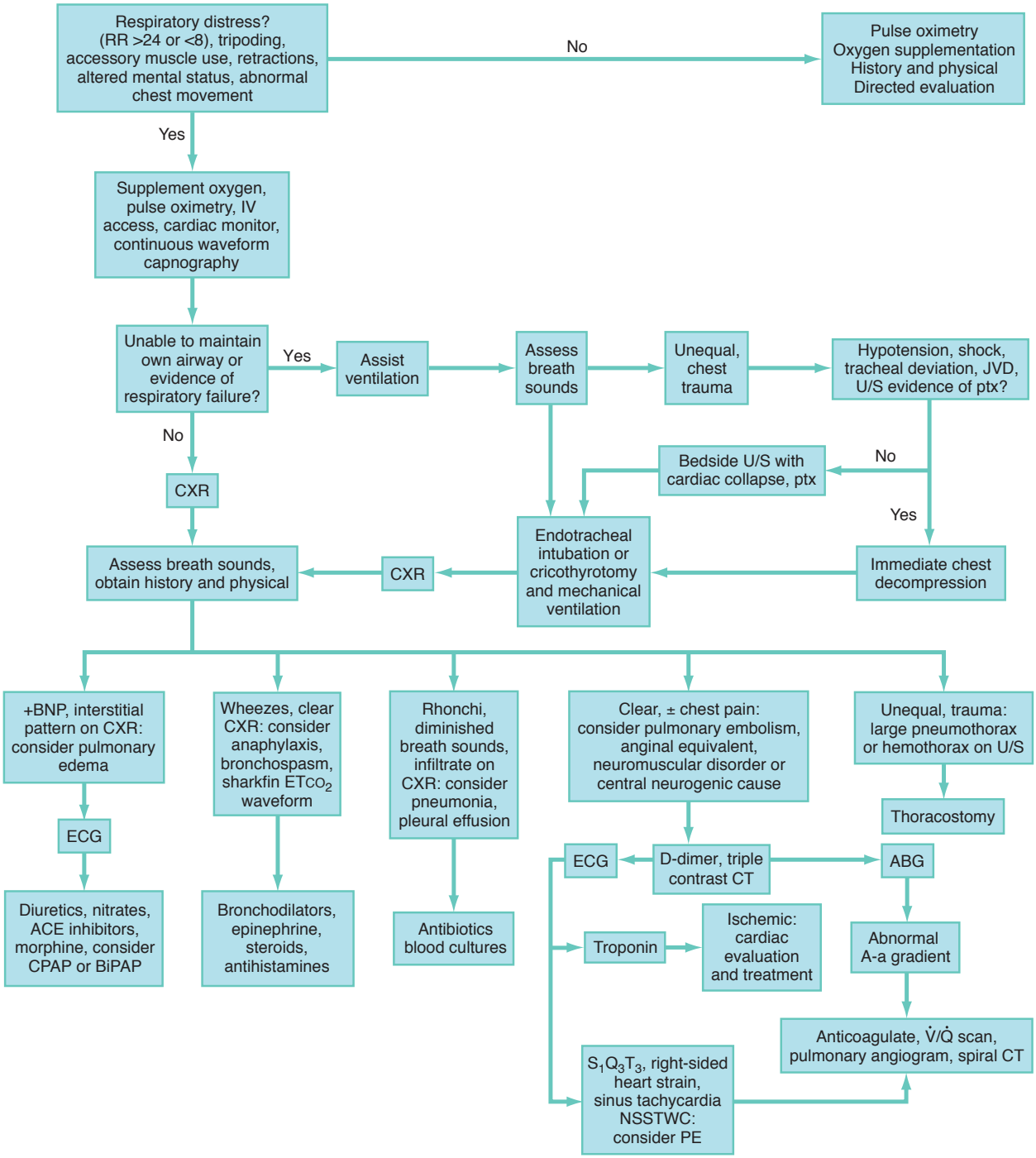

Fig. 22.1. Rapid assessment and stabilization of a dyspneic patient. *A-a,* arterial-alveolar; *ABG,* arterial blood gas; *ACE,* angiotensin-converting enzyme; *BiPAP,* biphasic positive airway pressure; *BNP,* B-type natriuretic peptide; *CPAP,* continuous positive airway pressure; *CT,* computed tomography; *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; *ptx,* pneumothorax; *RR,* respiratory rate; V̇/Q̇, ventilation-perfusion ratio; *U/S,* ultrasound.

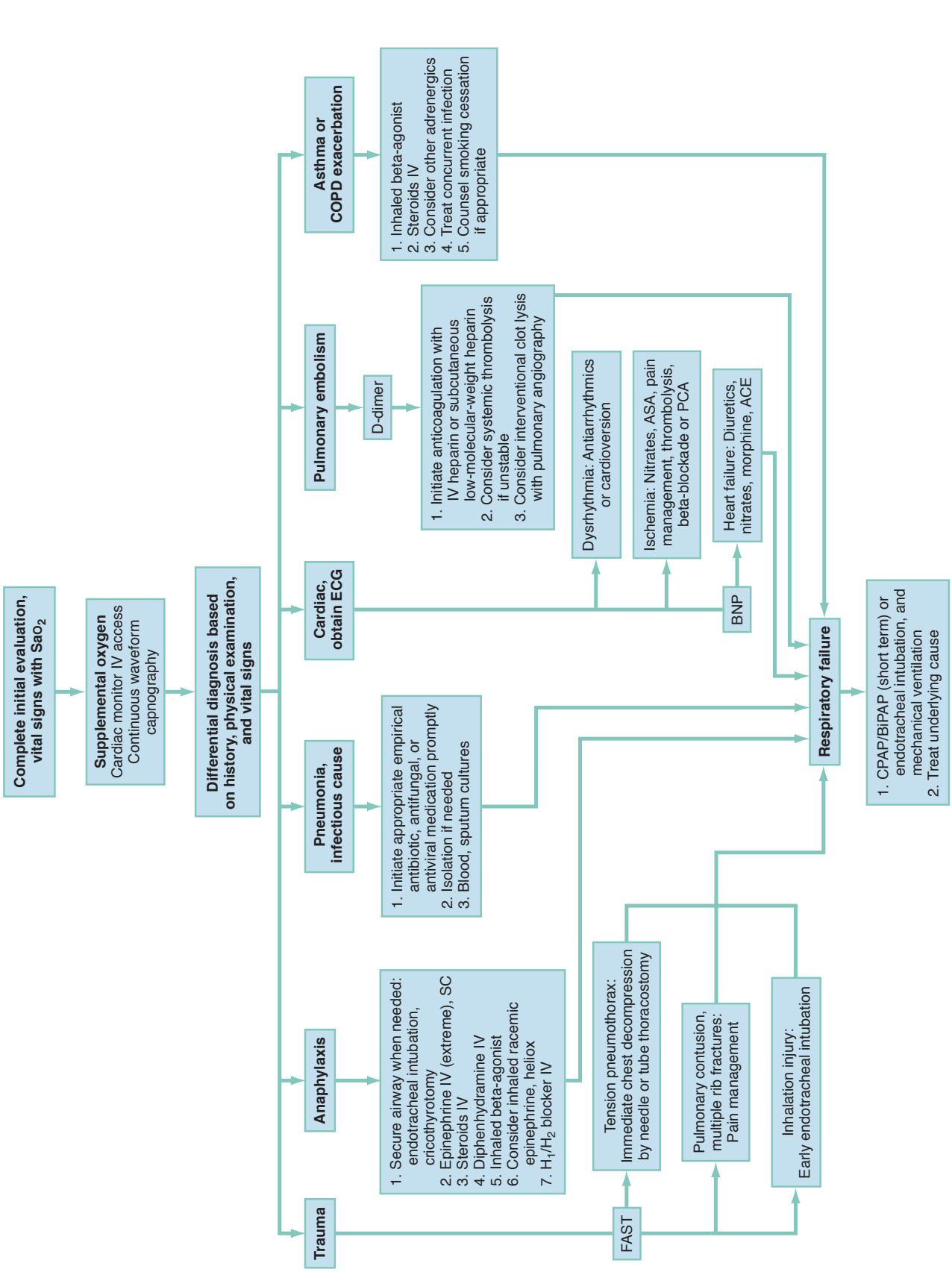

Fig. 22.2. Clinical guidelines for emergency department (ED) management of dyspnea. *ACE*, angiotensin-converting enzyme; *ASA*, acetylsalicylic acid; *BNP*, B-type natriuretic peptide; *COPD*, chronic obstructive pulmonary disease; *CPAP/BiPAP*, continuous positive airway pressure/biphasic positive airway pressure; *ECG*, electrocardiogram; *FAST*, focused assessment with sonography in trauma; *IV*, intravenous; *PCA*, patient-controlled analgesia; SaO_2, arterial oxygen saturation; *SC*, subcutaneously.

KEY CONCEPTS

- 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.
- Dyspnea is subjective and the differential diagnosis can be divided into acute and chronic causes, of which many are pulmonary. Other causes include cardiac, metabolic, infectious, neuromuscular, traumatic, and hematologic conditions.
- Chronic or progressive dyspnea usually denotes primary cardiac or pulmonary disease. Acute dyspneic spells may result from asthma exacerbation; infection; pulmonary embolus; intermittent cardiac dysfunction; psychogenic causes; or inhalation of irritants, allergens, or foreign bodies.
- All patients experiencing dyspnea, regardless of possible cause, should be promptly evaluated in the treatment area. Bedside pulse oximetry readings should be obtained, and the patient placed on a cardiac monitor.
- If the pulse oximetry result is less than 95% on room air, the patient should be placed on supplemental oxygen either by nasal cannula or mask, depending on the degree of desaturation.
- If necessary, breathing should be assisted with manual or mechanical ventilation, either noninvasively for the short term, or with the patient tracheally intubated for airway protection for prolonged ventilation.
- Unstable patients or patients with critical diagnoses must be stabilized and require admission to an intensive care unit. Emergent patients who have improved in the ED may be admitted to an intermediate care unit. Most patients in the nonurgent category can be treated as outpatients if medical follow-up can be arranged.

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