

## Dyspnea

Joseph R. Shiber, MD<sup>a,b,c,\*</sup>, Jose Santana, MD<sup>b</sup>

<sup>a</sup>*Department of Medicine, East Carolina University, Greenville, NC, USA*

<sup>b</sup>*Department of Emergency Medicine, East Carolina University, Greenville, NC, USA*

<sup>c</sup>*Emergency Medicine–Internal Medicine Combined Residency, East Carolina University, Greenville, NC, USA*

The sensation of breathlessness, dyspnea, is clinically important when it is recognized by the patient as abnormal. The development of shortness of breath (SOB) or the inability to satisfy oxygen requirements is an expected outcome of overexertion, such as occurs after running or heavy lifting. When dyspnea occurs at rest or during exertion that is less than expected, it is considered pathologic and a symptom of a disease state. Multiple organ systems are involved in the differential diagnosis of dyspnea, most commonly the cardiovascular and pulmonary systems. The type and severity of underlying lung or heart disease have been shown to correlate with the description offered by the patient [1,2]. **Box 1** illustrates the extensive differential diagnosis for dyspnea.

The outpatient management of dyspnea has changed recently, with the advent of new diagnostic tests and therapies. These innovations have allowed the outpatient clinician to more accurately diagnose the underlying disorder and initiate appropriate therapy. Now, clinical decisions based on whether to continue outpatient management or admit a patient to the hospital can be augmented by treatment algorithms. This article constructs a decision and management protocol for physicians, allowing for a decision to treat the adult dyspneic patient in the office or transfer the patient to a hospital in pursuit of a confirmed diagnosis and definitive care. The first step is to begin with a rapid initial assessment of the airway, breathing, and circulation, while gathering a focused history and physical examination [3]. Once the initial examination and vital signs have been obtained and an emergent situation has been excluded, the patient can be placed in one of three categories: (1) distress with unstable vital signs; (2) distress with stable

---

\* Corresponding author. Department of Medicine, East Carolina University, 600 Moye Boulevard, Greenville, NC 27834.

*E-mail address:* shiberj@mail.ecu.edu (J.R. Shiber).

**Box 1. Differential diagnosis for dyspnea***Mechanical interference with ventilation*

Abdominal or chest mass  
Asthma, emphysema, bronchitis  
Endobronchial tumor  
Interstitial fibrosis of any cause  
Kyphoscoliosis  
Left ventricular failure  
Lymphangitic tumor  
Obesity  
Obstruction to airflow, central or peripheral  
Pleural thickening  
Resistance to expansion of the chest wall or diaphragm  
Resistance to expansion of the lung  
Thoracic burn with eschar formation  
Tracheal or laryngeal stenosis

*Weakness of the respiratory pump*

Absolute  
Hyperinflation  
Neuromuscular disease  
Obesity  
Pleural effusion  
Pneumothorax  
Previous poliomyelitis  
Relative

*Increased respiratory drive*

Decreased cardiac output  
Decreased effective hemoglobin  
Hypoxemia of any cause  
Metabolic acidosis  
Renal disease  
Stimulation of intrapulmonary receptors

*Wasted ventilation*

Capillary destruction  
Large-vessel obstruction

*Psychologic dysfunction*

Anxiety  
Bodily preoccupation, somatization disorder  
Depression  
Secondary gain, malingering

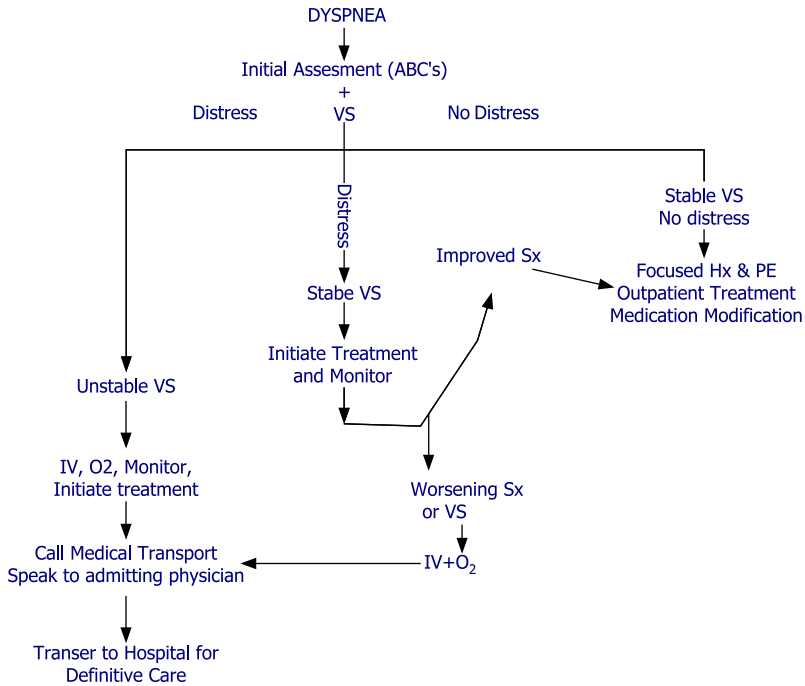


Fig. 1. Algorithm for acute dyspnea management. The initial assessment includes airway, breathing, and circulation (ABC's), a brief history of the present illness, medical history, medications, and allergies. Hx, history; Sx, symptoms; VS, vital signs.

vital signs; or (3) no distress and stable vital signs. Following this algorithm, a patient's disposition can be determined expeditiously (Fig. 1).

Acute myocardial infarction (AMI) and congestive heart failure (CHF) are the most common cardiovascular disorders that lead to dyspnea. Patients who have had an AMI usually describe retrosternal chest discomfort and an inability to catch their breath. This sensation occurs secondary to a reduction in cardiac output and pulmonary perfusion and is caused by reduced contractility of the ischemic or stunned myocardium. The administration of supplemental oxygen, therapy to decrease myocardial oxygen consumption and increase coronary vasodilation, and the use of revascularization strategies such as fibrinolytic therapy or percutaneous coronary intervention may readily relieve the symptoms. Patients who have an exacerbation of congestive heart failure may describe a sensation of SOB with exertion, orthopnea, and paroxysmal nocturnal dyspnea. Symptoms generally resolve or are improved by giving the patient supplemental oxygen, diuretics, therapy aimed at decreasing preload and afterload, and medical or surgical treatments to improve cardiac contractility. Pulmonary dyspnea is commonly caused by chronic obstructive pulmonary disease (COPD), asthma, pneumothorax, and pneumonia. Chronic obstructive pulmonary disease and asthma are considered obstructive processes that may

produce chronic SOB punctuated by sudden acute worsening of symptoms. Often, the treatment of these diseases requires the use of steroids and inhaled bronchodilators to resolve the ensuing exacerbation. Table 1 describes the symptoms of dyspnea according to the disease state. Primary care clinicians should always consider other life-threatening causes of SOB such as pulmonary embolism (PE).

Although many of the causes that lead to dyspnea will be treated ultimately in the causes or inpatient ward, primary care physicians should be knowledgeable about the subtle and atypical presentations of potentially life-threatening diagnoses.

### Congestive heart failure

Congestive heart failure is one of the most common causes of dyspnea seen in health care settings. Approximately 1.2% to 2% of the population in the United States has heart failure, and most patients (75%–80%) are older than 65 years of age. It has been estimated that approximately 20 million people unknowingly have depressed left ventricular function in the absence of symptoms and are likely to become symptomatic within a 1- to 5-year period. Patients who have CHF visit physicians more than 11 million times per year and are responsible for 3.5 million hospitalizations per year, with one third requiring rehospitalization within 3 months after discharge [4].

Patients may present to an outpatient clinic as an established, well-known patient with CHF or as a first-time visitor. Thus, primary care physicians are on the front line when it comes to tentatively diagnosing congestive heart failure. The well-established patient usually has been diagnosed previously with heart failure or has significant predisposing risk factors, such as hypertension or coronary artery disease (CAD). The patient who is newly diagnosed with CHF presents the added challenge of tailoring the outpatient workup and investigation for the cause of the disease. The diagnosis is determined after a full history, physical examination, and diagnostic studies have been performed. Once the diagnosis and severity of illness are determined, the appropriate treatment can be initiated. In many cases, patients who present to an outpatient clinic will be too ill to be taken care of in the office and will require transfer to an emergency department (ED) for a definitive diagnosis, treatment, and possibly admission to the hospital.

Table 1  
Symptoms associated with diagnosis

Diagnosis	Symptoms
CHF	Dyspnea on exertion, paroxysmal nocturnal dyspnea, orthopnea
AMI	Radiating chest pressure, diaphoresis, SOB
PE	Fever, pleuritic chest pain, sudden onset SOB, syncope
COPD/asthma	Cough, smoker, SOB relieved with nebulizer or MDI
Pneumonia	Fever, productive cough, SOB
Pneumothorax	Sudden onset pleuritic chest pain, SOB not relieved with O <sub>2</sub>

### *History*

The patient's history will help determine what elements of the physical examination will aid in the appropriate diagnosis and what diagnostic studies may or may not be indicated. The onset, severity, duration, alleviating, aggravating, and associated symptoms are all pertinent to the history. To illustrate, consider the following case: A 55-year-old man presents to his primary care physician's clinic with a medical history significant for hypertension, with the chief complaint, "I'm unable to walk very far without becoming winded." He states that he now uses three pillows to keep from feeling short of breath and wakes up frequently at night to catch his breath, and he has noticed that his legs are swollen.

This patient is like many of the people seen everyday in outpatient clinics. How is the practitioner to sort out this patient from the other patients seen the same day with similar complaints? Simply, it is all in the history. The important information to obtain includes onset, duration of symptoms, what activities result in SOB, and any other associated symptoms. In addition, the history should focus on the other deadly causes of SOB. Has the patient just taken a 10-hour plane ride? Has there been any associated chest pain, back pain, nausea, or diaphoresis? Has the patient experienced palpitations or any other symptoms?

It is also important to ask the patient about their medical, surgical, social, and family history to determine risk factors and possible causes, and ask about medication use and record the current dosage to initiate or modify treatments.

### *Physical examination*

The physical examination should be given due importance, despite innovations in objective laboratory and radiographic tests. Studies have shown that the physical examination depends on the diagnosis and prognosis of congestive heart failure. The physical examination begins the first moment the patient is encountered, during which their general appearance is observed. A person's appearance speaks volumes about the severity of their dyspnea. A patient who becomes short of breath after a few steps in the office is obviously very different from the one who can walk half of a mile before becoming dyspneic. This categorization can be further described by using the New Heart Association's Functional Classification of Heart Failure. Rating the patient on this functional scale from I to V can be used as a comparison to the previous functional status, determine the acuity of change in an established patient, and the response to treatment [5]. The patient's disposition can often be determined by the initial general appearance; whether the patient can be treated with oral medication or he or she requires transportation to the ED for intravenous (IV) medication administration and hospital admission.

It is imperative to record and review the patient's initial vital signs, including pulse oximetry, and to repeat assessments after interventions.

Obtaining an accurate body weight to compare with previous values, particularly if the patient weighs him/herself regularly, will suggest whether there has been a recent increase in weight. Tachypnea, tachycardia, and hypoxia are expected findings during a CHF exacerbation. Hypertension may be secondary to stress or present as a cause of failure; if hypotension is present, cardiac output is likely to be severely decreased. If hypotension and tachycardia are present, the physician must consider cardiogenic shock with inadequate tissue perfusion. Ideally, this type of patient will be recognized immediately, and stabilization can begin as urgent supervised transportation to the hospital emergency department is arranged.

After the initial observation, the clinician should work through a systematic evaluation. An examination of the neck may show jugular venous distention, indicating increased intravascular volume or right-sided filling pressures. Palpation and auscultation of the carotids may demonstrate signs of atherosclerotic disease, which may indicate that other vascular beds, such as the coronary vessels, are involved with disease. An examination of the lungs may reveal the presence of rales or crackles from pulmonary edema and wheezing from bronchial and interstitial edema. Decreased tactile fremitus on palpation and dullness on percussion indicate pleural effusion. An  $S_3$  gallop found on cardiac auscultation represents left ventricular systolic dysfunction, whereas an  $S_4$  is associated with left ventricular diastolic dysfunction with acute cardiac ischemia being the most worrisome cause. An irregular heart rhythm may be detected that may indicate that an underlying arrhythmia is present, or a new murmur may signal acute valvular dysfunction. Quiet heart sounds may also be detected in some cases and be caused by a pericardial effusion.

An abdominal examination may demonstrate findings secondary to congestion of the hepatic venous system, resulting in hepatomegaly and jugular distension (hepatojugular reflex) when depressing the liver edge. Ascites can also be observed by a fluid wave on palpation. The extremities should be evaluated for perfusion, including an assessment of skin color and temperature, peripheral pulses, and capillary refill. Lower extremity pitting edema is a sign of right-sided heart failure, whereas decreased hair growth on the lower extremity is indicative of chronic edema (Table 2).

### *Diagnostic studies*

Heart failure can be diagnosed by the history and constellation of clinical findings alone, but imaging and laboratory studies may be necessary to help confirm the definitive diagnosis as well as to stratify the severity of the episode. The availability of these tests varies from the primary care office to the urgent care setting. Pulse oximetry should be the first diagnostic modality used while taking the vital signs. Based on these data, the patient's level of oxygenation and the reversibility of the disease process can be determined. If a heart failure patient presents with symptomatic hypoxia that is not improved with supplemental oxygen, this patient falls under the

Table 2  
Physical examination of dyspnea

Diagnosis	Findings
CHF	Edema, jugular venous distension, S3 or S4, hepatojugular reflux, murmurs, rales, wheezing, hypertension
ACS	Tachycardia, heart failure findings
PE	Wheezing, lower extremity swelling, friction rub
COPD/asthma	Wheezing, barrel chest, clubbing, decreased breath sounds, pulsus paradoxical, accessory muscle use
Pneumonia	Fever, crackles, increased fremitus
Pneumothorax	Absent breath sounds, hyperresonance, jugular venous distension, tracheal deviation

category of unstable and is in distress. In this category, the immediate transfer to the hospital is required. Patients who have chronic hypoxia are eligible for home oxygen therapy if their oxygenation on exertion falls below 88%, with coverage by private insurance and government agencies [6].

ECG is an invaluable tool and should be performed early on all patients being evaluated for SOB. The ECG may help determine the cause of heart failure and help define the anatomic location of CAD. Ischemic heart disease may be interpreted on the ECG as an ST-segment depression or a T-wave inversion, whereas an ST-segment elevation or Q waves indicate previous infarction. Patients may also have arrhythmias and signs of electrolyte deficiency or excess. In the case of hypokalemia and hyperkalemia, flattened T waves or U waves, or both, and peaked T waves may be found, respectively. Ventricular and atrial enlargement can be determined as anatomical features of heart failure by using recognized ECG criteria. Low QRS voltages, noted in precordial leads, are caused commonly by pericardial effusion, infiltrative heart disease, COPD, hypothyroidism, and obesity. Chest radiographs (CXR) are used to look for pulmonary vascular congestion, interstitial fluid, and pulmonary edema. The presence of cardiomegaly indicates that the process did not occur suddenly, but differentiating the pericardial silhouette from the cardiac contour can be challenging by chest radiography. Echocardiography is the most important noninvasive modality with which to determine systolic and diastolic function, ventricular size, the presence of valvular disease, and the presence of pericardial effusion. Systolic heart failure is identified by a decreased ejection fraction and dilated chambers, whereas diastolic heart failure is more difficult to diagnose and less common than systolic dysfunction. These patients have a normal or elevated ejection fraction but reduced diastolic filling caused by impaired myocardial relaxation, and they have a specific secondary cause, such as hypertension or aortic stenosis.

Laboratory studies can add to the objective findings. Anemia can be found in chronic heart failure, and polycythemia may be encountered in

those with chronic hypoxic states. A high platelet count can be an acute phase reactant, whereas for older fragile heart failure patients, pneumonia may cause an exacerbation and present with a high white blood cell count. The measurement of serum digoxin level is important in patients taking this drug because of the relatively narrow therapeutic index. Prerenal acute renal failure can be determined by finding an elevated blood urea nitrogen (BUN) level, and increased BUN-creatinine ratio, indicating hypoperfusion of the kidneys. Obtaining serum values of potassium, magnesium, calcium, sodium, chloride, and bicarbonate may assist in determining intravascular volume status, whereas replacing depleted electrolytes may reduce the risk of cardiac arrhythmia. Brain natriuretic peptide (BNP) is a cardiac neurohormone secreted by the cardiac ventricles in response to ventricular wall tension, pressure overload, and increased volume expansion. BNP can now be detected rapidly by a simple laboratory test, which is becoming more readily available in the outpatient setting. This value allows for a more definitive diagnosis of heart failure by narrowing the differential for dyspnea. If this value is elevated by  $\geq 100$  pg/mL, the high sensitivity and specificity of this test allows for accurate diagnosis of heart failure. Values that are  $\leq 100$  pg/mL reliably rule out depressed left ventricular function [7,8]. Brain natriuretic peptide has also been used as a predictor of mortality and can be used to follow improvement after CHF therapy in cases of acute cardiac decompensation and for outpatients. The assessment of BNP values over time allows the clinician to better estimate an exacerbation based on an individualized value. BNP produces vasodilation, diuresis, and natriuresis, and inhibits the renin-angiotensin-aldosterone axis. Although BNP is an available medication, nesiritide (recombinant human BNP), its IV formulation, has been limited to inpatient use for severely decompensated CHF.

### *Management*

The initial management of CHF begins after the patient has been evaluated quickly and the level of acuteness has been determined. Using the categories described earlier, patients are treated according to their specific category. If the patient is in acute distress, they should receive supplemental oxygen and preload reduction with sublingual, transcutaneous, or IV nitroglycerin (NTG). Have them remain with their head elevated but their feet dependent, as in a reverse-Trendelenburg position, to further discourage venous return. Oral or IV furosemide is recommended if the patient has evidence of volume overload. If the blood pressure is still elevated, the reduction in cardiac afterload should be accomplished with an arterial vasodilator, such as an angiotensin-converting enzyme inhibitor or hydralazine. The use of calcium channel blockers should be avoided in patients who have CHF caused by reduced systolic function, and  $\beta$ -blockers should not be used while the patient is in a decompensated episode. Obviously, many of these patients will require transportation to the nearest ED for definitive care.

These patients are further classified into unstable and stable based on vital signs. Unstable patients are initially given therapy while arrangements are made for transfer to an inpatient facility. Stable patients, after initial treatment, can be observed for improvement; but if they become unstable, follow the same algorithm as above. The patients who improve significantly can be managed much in the same way as those that are initially found to be in no acute distress. These patients benefit from home instruction and education, medication modification, and close follow-up (Table 3).

Table 3  
Distress classification and management

Disease	No acute distress	Acute distress	Initial treatment	
			Stable	Unstable
CHF	No dyspnea at rest	Dyspnea at rest or minimal exertion	Furosemide po, Bp control, O <sub>2</sub>	Furosemide IV, NTG, Bp control, O <sub>2</sub>
CAD	Stable angina	Acute chest pain, diaphoresis, tachycardia or bradycardia, hypotension or hypertension, syncope	ASA, NTG, O <sub>2</sub> , analgesia	ASA, NTG, O <sub>2</sub> , analgesia, ± plavix
PE	Asymptomatic silent	Dyspnea at rest, fever, hypoxia, tachycardia, hypertension	NA	LMWH, O <sub>2</sub> , analgesics
Pneumonia	Normal O <sub>2</sub> saturation, mild dyspnea, low PORT score	Decreased O <sub>2</sub> saturation, functional debilitating dyspnea, tachycardia, high/moderate PORT score	O <sub>2</sub> , acetaminophen, antibiotics po	O <sub>2</sub> , acetaminophen, IM or IV antibiotics
Pneumothorax	Spontaneous	Tension	Analgesics	Needle decompression, O <sub>2</sub> , analgesics
COPD/asthma	>80% peak flow	<80% peak flow	O <sub>2</sub> , MDI treatment	O <sub>2</sub> , MDI, or nebulizer treatment and steroids

*Abbreviations:* ASA, aspirin; Bp, blood pressure; IM, intramuscular; LMWH, low molecular weight heparin; NA, not applicable; po, by mouth.

*Data from* Steering Committee and Membership of the Advisory Council to Improve Outcomes Nationwide in Heart Failure. *Am J Cardiol* 1999;83(Suppl 2A):1A-39A.

## Acute coronary syndrome

CAD is the leading cause of death in the United States, caused acutely by myocardial infarction and as a result of chronic problems such as congestive heart failure. It is estimated that 13.7 million people in the United States suffer from CAD, with half having myocardial infarction and the other half suffering from chronic angina [9]. An acute coronary syndrome (ACS) refers to the rupture of an atherosclerotic plaque with the activation of platelets and fibrin, resulting in thrombus formation and either reduced flow or the occlusion of a coronary artery. Many of these individuals have significant risk factors identified before an acute event by their primary medical providers; thus it is important for a physician in an outpatient environment to recognize patients who have acute dyspnea caused by acute coronary syndrome, so that prompt initial treatment can be started and transport initiated as soon as possible. The goal is for no more than 30 minutes to elapse from the time of initial contact until emergency medical services are called or appropriate measures are instituted [10]. Once the patient arrives at the hospital, the goal is 90 minutes from door to reperfusion, in an attempt to limit the myocardial ischemia and injury to 2 hours or less [11]. Another consideration is that some patients, particularly women and the elderly, may present with atypical presentations of AMI, particularly isolated dyspnea. If an acute coronary syndrome is suspected, transport for definitive management should not be delayed.

### *History*

The rapid assessment of dyspnea in acute coronary syndrome is initiated with a brief targeted interview and risk-factor stratification. If the patient has had a syncopal event or has unstable vital signs, the initial assessment starts with an evaluation of airway, breathing, and circulation, while taking a brief history. It is necessary to distinguish an ACS patient from a patient who simply has stable chronic angina, which does not have the same underlying pathophysiology or associated morbidity or mortality and therefore does not require the same aggressive therapies.

Patients who complain of acute dyspnea and have acute coronary syndrome often describe left-sided retrosternal chest pressure, diaphoresis, tachycardia, radiation of pain to the jaw, neck, or left or right arm, and nausea. These symptoms may last several minutes and be alleviated by sublingual NTG and aggravated by exertion. Importantly, a large percentage of cardiac ischemic episodes may not have these specific symptoms [11]. It has also been documented that up to one third of myocardial infarction patients have no chest pain on presentation to the hospital [12]. These patients may not be diagnosed initially or at all, allowing for myocardial damage to exceed that of an otherwise treated patient. Therefore, a thorough history that includes past medical, surgical, social, and family history must be obtained so that those individuals with vague symptoms or several risk factors can be evaluated appropriately.

Risk factors for CAD include male age  $\geq 45$  years, female age  $\geq 55$  years, diabetes, obesity, a family history for CAD, tobacco use, hypertension, a history of atherosclerotic disease, hypertriglyceridemia, high levels of low-density lipoprotein cholesterol, and low levels of high-density lipoprotein (HDL) cholesterol ( $\leq 40$  mg/dL). High HDL ( $\geq 60$  mg/dL) is a negative risk factor, which should be considered [13].

### *Physical examination*

The physical examination should focus on the cardiovascular and respiratory systems. The examination will follow the same general sequence as for congestive heart failure. Specific attention should be devoted to cardiac auscultation. An  $S_4$  sound may indicate ventricular stiffness caused by ischemic stunning, and a new murmur may represent acute valvular dysfunction, causing regurgitation or, less commonly, rupture of the interventricular septum, allowing for a new left-to-right shunt. Quiet heart sounds and other evidence of a symptomatic pericardial effusion may be caused by ventricular free wall rupture, causing acute hemopericardium. Those patients presenting with unstable vital signs in acute distress will first require rapid evaluation of airway, breathing, and circulation.

### *Diagnosis*

The diagnosis of acute myocardial infarction is based on signs and symptoms, diagnostic electrocardiographic criteria, and an elevation of cardiac biomarkers. Because the latter usually can be performed only in the hospital, outpatient criteria are limited to ECG changes and patient presentation. Other diagnostic tools in an inpatient facility may include further blood work, echocardiography, and chest radiography. Relief of chest pain quickly after receiving supplemental oxygen, soluble aspirin, or sublingual NTG, is suggestive but not specific for acute CAD, but it should not be used as diagnostic criteria to confirm or exclude an ACS.

Laboratory tests may demonstrate anemia as the cause of the ischemia or infarction. As described above, electrolytes may be deficient or in excess and predispose to cardiac arrhythmias, which decrease blood flow while increasing demands on the myocardium. Rapid assays of the cardiac isomer of creatine phosphokinase and troponin are becoming more available, and although the assays are very sensitive and specific for cardiac injury, serum levels require a minimum of several hours to rise during an acute coronary event. The ECG may demonstrate many types of changes that indicate ischemia or injury and infarction. Typical changes include hyperacute T waves, T-wave inversion, ST-segment elevation, Q waves, or new onset left bundle branch blocks in infarction and ST-segment depression in ischemia. It is important to identify the region of the myocardium that is affected when evaluating an ECG. An echocardiogram can be used to identify wall motion abnormalities. This information, along with the anatomical description of

myocardial damage by ECG, will provide important information to the cardiac interventionalist when deciding which coronary artery to image first on percutaneous coronary catheterization.

### *Management*

When patients who have acute dyspnea present and ACS is suspected or diagnosed, prompt initial therapy and transfer for further evaluation and definitive treatment are required. An IV line should be started, and the patient should be placed on a cardiac monitor. Therapy begins with the administration of oxygen, rapidly absorbed soluble aspirin (not enteric coated), and sublingual NTG (0.4 mg). If the patient develops hypotension in response to NTG, an IV bolus of crystalloid should be given, and further NTG must be used cautiously [14,15]. A  $\beta$ -blocker should be given to patients without bradycardia or heart block to decrease myocardial oxygen consumption [16]. The transfer of a patient suspected of having ACS should not be impeded by further examinations, studies, or treatment. The goal is to obtain definitive treatment through reperfusion as soon as possible (see Table 3).

One of the most important skills in the outpatient setting is in recognizing and diagnosing an atypical acute coronary syndrome. Patients known to have subtle, atypical, and even silent myocardial infarctions include the elderly and diabetics. Female patients have also been found to present with atypical presentations. Primary care physicians should be on the alert for these types of presentations in their patients.

### **Pulmonary embolism**

PE is one of the most commonly missed lethal diagnoses. Untreated PE has a mortality rate from 18% to 35% and therefore should always be considered in the differential diagnosis of acute dyspnea [17]. In the United States, 1 in 1000 Americans is affected each year, making this disease a common clinical entity that can be treated if identified correctly. Of the 600,000 episodes of PE that occur each year, 50 to 100,000 patients die as a result of the disease [18]. Once the disease is diagnosed and treated, the incidence of recurrence and death is significantly reduced. The strategy in an outpatient clinic should be to immediately send any patient with significant clinical suspicion to the hospital emergency department for a definitive diagnosis and possible treatment. Because the objective data may not be available in the outpatient clinic, clinical suspicion is highly regarded as sufficient evidence to disposition early for definitive management. Considering that most deaths are attributed to the failure to diagnose, a quick, definitive diagnosis and management are imperative.

### *History*

The history in patients who have PE may be misleading because the signs and symptoms are neither sensitive nor specific. An estimated 50% of cases

of PE remain undiagnosed, underscoring the atypical nature of the disease [19]. The frequency of the most common signs and symptoms of PE have been studied. Dyspnea is the most common symptom, whereas tachypnea, pleuritic chest pain, and rales are also common signs and symptoms, at 73%, 70%, 66%, and 51%, respectively [20]. Other frequent symptoms include cough, hemoptysis, syncope, and fever. Although these symptoms are common in PE, they lack diagnostic sensitivity and specificity. It has been shown that patients who do not have PE have a similar frequency of symptoms [21].

It is important to inquire about any recent unilateral leg swelling, warmth, redness, or pain because lower extremity deep venous thrombosis (DVT) is the most common cause of PE. Risk-factor stratification is an important tool in gathering evidence to support suspicion of PE. Factors identified include immobilization, surgery within the past 3 months, stroke, history of venous thromboembolism, and malignancy [20]. Less common risk factors include increasing age, lower extremity trauma, extended travel, oral contraceptive pills, hormone replacement therapy, pregnancy, recent delivery, recent joint replacement, lower extremity fractures, and pelvic fractures. Other studies have added obesity, hypertension, severe CHF, pulmonary hypertension, and cigarette smoking. The medical and family history should also account for genetic or acquired causes of thrombophilia, including antithrombin III deficiency, lupus anticoagulant, protein C and S deficiency, factor V Leiden, and mutation in prothrombin G20210A, as well as nephrotic syndrome and inflammatory bowel disease.

### *Physical examination*

A rapid examination of a dyspneic patient who has possible pulmonary thromboembolism is essential for disposition. The physical examination is used to further support a suspicious history and provides rapid support for the need to transport the patient to the ED. Vital signs may demonstrate fever, tachypnea, tachycardia, hypoxia, and hypotension. Of these findings, hypoxia is the most specific, although this can be masked by hyperventilation from a tachypneic patient. After the vital signs have been evaluated, the patient's general appearance is noted, followed by a focused cardiovascular and pulmonary physical examination. Patients who appear to be in acute distress may have rapid shallow breathing at rest, diaphoresis, and tachycardia. Their airway, breathing, and circulation should be assessed quickly, before they are transported to a hospital.

A focused respiratory examination consists of auscultation of the lungs. Examination findings may include normal, decreased breath sounds, a pleural friction rub, or rales. In patients who have pulmonary consolidation caused by an associated lung infarction, the presence of egophony can be appreciated on auscultation. Dullness to percussion and decreased tactile fremitus on palpation indicate a pleural effusion. Patients may also have reproducible chest pain on deep breathing. The cardiovascular examination

may reveal signs of right-sided heart failure from pulmonary hypertension. Tachycardia, a right-sided  $S_4$ , and an increased pulmonic component of second heart sound may be auscultated. The remainder of the vascular examination consists of examining the lower extremities for signs of deep vein thrombosis as a possible cause. These examination findings include unilateral lower extremity edema, warmth, erythema, a palpable cord, and posterior leg pain on dorsiflexion of the ipsilateral foot.

### *Diagnosis*

The diagnosis of PE is challenging because of vague, nonspecific symptoms or lack of symptoms. PE is diagnosed definitively by imaging studies, once the patient has been sent to the ED for evaluation. Laboratory tests, echocardiograms, and chest radiographs are modalities used to help provide supporting evidence but do not provide concrete evidence of PE. The diagnosis of a deep venous thromboembolism by ultrasonography may allow a clinician to assume PE is present with suggestive pulmonary symptoms or signs. Thus, a patient who is being evaluated for SOB who is found to have a DVT needs no further workup. The diagnosis is PE.

Laboratory test findings may be abnormal but are nonspecific. Leukocytosis can occur as a reactive phenomenon, as well as an elevated sedimentation rate. Respiratory alkalosis with an elevated A-a gradient is often present on arterial blood gas but is also nonspecific. The CXR is abnormal in the majority of patients who have PE with infarction, but these findings are often nonspecific. These findings may include infiltrates, atelectasis, and pleural effusion. More specific radiographic signs include Westermark's and Hampton's sign, although they are uncommon. Westermark's sign is the loss of pulmonary markings distal to the PE, from dilated pulmonary vasculature proximal to the embolus, with oligemia distally. Hampton's "hump" is pleural-based wedge-shaped opacity facing the hilum.

The electrocardiogram is also often abnormal and nonspecific. Sinus tachycardia and nonspecific ST-T wave changes are the most common abnormalities. Signs of right-sided heart strain, such as  $S_1Q_3T_3$ , T-wave inversion in leads  $V_1$  to  $V_3$ , and a new right bundle branch block, are specific but uncommon [20]. Echocardiography is used to assess for signs of right heart strain and to look for thrombus in the right ventricle. Signs of pulmonary hypertension may include increased pressure over the tricuspid valve and the pulmonary artery. The definitive tests are ventilation perfusion imaging, multidetector pulmonary CT angiography, and, in rare circumstances, pulmonary angiography. Any patient who requires this type of examination should be transferred to the hospital to rule out the diagnosis.

### *Management*

The definitive diagnosis cannot be made in most outpatient facilities, thus rapid transfer to a hospital is crucial to effective management. Patients who

appear to be in acute distress should have only their airway, breathing, and circulation examined before transfer measures have begun. Those who appear to be in no acute distress may undergo a more detailed examination. Patients in acute distress should have an IV line started, placed on oxygen, and put on a cardio-pulmonary monitor.

After the history and physical have been completed, the physician should designate the patient's pretest probability for having a PE. The probability may be low, intermediate, or high, although there is no standard method for measurement of pretest probability. Some researchers have based this on objective data not available in the outpatient environment. The outpatient designation should reflect findings on history, examination, and other objective data. This pretest probability can be relayed to the admitting physician as a means to assist in determining what diagnostic test to perform. If the patient is believed to be at high risk for a PE and has no contraindications for anticoagulation, then heparin administration should be considered. From the outpatient view, the most important management point is to immediately transfer any patient suspected of having a PE (see [Table 3](#)).

## Asthma

Asthma is characterized by reversible airflow obstruction, bronchial hyperresponsiveness, airway inflammation, submucosal edema, and increased mucus production caused by hypertrophy and hyperplasia of goblet cells [22–24]. Asthma is the most common chronic lung disease in developed and developing countries, with a current adult prevalence of 5%, and has been increasing in prevalence over the last 20 years [25,26]. It is also the most common cause of respiratory emergency, resulting in almost 2 million ED visits each year, 10% to 20% of which require hospital admission [22–28]. Between 4% and 10% of asthma admissions will require care in the ICU, but the presentations of severe acute asthma requiring ICU care have decreased over the last decade [26–28]. Although the death rate caused by asthma is low, 0.3 per 100,000 people, it is still responsible for approximately 5500 deaths per year, with the overwhelming majority of these patients suffering respiratory arrest before arriving at the hospital [25,27].

## History

During an exacerbation, patients commonly report dyspnea, wheezing, tightness in the chest, and coughing (usually nonproductive). Frequent causes include viral respiratory infections, bacterial infections (most commonly by *Mycoplasma pneumoniae* and *Chlamydia pneumoniae*), exposure to allergens or respiratory irritants (tobacco smoke, perfumes, and fumes from cleaning products), exercise, exposure to cold, emotional distress, medications (aspirin, nonsteroidal anti-inflammatory drugs, or  $\beta$ -blockers), and

noncompliance to medication [22–24,29]. Risk factors for increased morbidity and mortality include two or more ED visits or a hospital admission within the past year, any previous episode requiring mechanical ventilation, chronic steroid use, extremely rapid onset of symptoms, association of anaphylaxis, low socioeconomic and educational status, psychosocial disorders, and language barriers [22–24,29]. A large proportion of the asthma fatalities that occur are believed to be caused by extensive mucous plugging of the airways, with such air trapping that the lungs remain inflated at autopsy after removal from the thorax [22–24]. The failure of the patient or physician to recognize the severity of the asthma attack is also believed to be responsible for many of the deaths [25,30].

### *Physical examination*

Asthma, meaning “panting” in Greek, was described accurately in ancient times for the most obvious clinical feature: tachypnea [22]. Wheezing is often heard diffusely and may be appreciated throughout the respiratory cycle or more prominently with expiration. Because airflow is required to cause turbulence, which produces wheezing, a patient with severe airway obstruction may have minimal airflow and therefore no wheezing. A brief, focused history and physical examination should be performed initially to identify any patient who may be unstable or need immediate airway interventions or assisted ventilation. An initial peak expiratory flow rate (PEFR) should be obtained. A focused examination, including vital signs, mental status, cardiopulmonary examination, and PEFR should be repeated frequently and after interventions to assess response to treatment.

A patient with only a mild exacerbation, defined as an initial PEFR  $\geq 200$  L/min or  $\geq 50\%$  of the predicted best may have only end-expiratory wheezing and a slightly increased respiratory rate without other physical findings. During a moderate asthma exacerbation, a PEFR of 80 to 200 L/min or 25% to 50% of the predicted best, the patient may not be able to speak conversationally without breathlessness, they may not be able to recline on the stretcher and instead choose a more upright posture, respiratory rate will be increased, and wheezing will be more prominent. With a severe exacerbation, a PEFR of  $\leq 80$  L/min or  $\leq 25\%$  of the predicted best, the patient will likely have diaphoresis, supraclavicular retractions, accessory muscle use, halting speech, a tripodding posture (sitting or standing while leaning forward to brace the arms outward on the knees or a fixed object), and severe tachypnea. Beware of agitation or confusion, cyanosis, fatigue, or a quiet chest because these are signs of impending respiratory arrest [22–24,29,30]. An increased pulsus paradoxus,  $\geq 25$  mm Hg, may be found with severe asthmatic exacerbations. Because this abnormality depends on forceful inspiratory and expiratory excursions, the pulsus may diminish as the clinical condition improves, airway obstruction resolves, and respiratory effort normalizes or as the condition worsens and the respiratory effort becomes fatiguing [22–28].

### *Diagnostic studies*

In addition to the assessment of PEFr and oxygen saturation, the physician should consider obtaining a chest radiograph if the patient has not responded adequately to therapy or is in extremis. The film findings may be normal or may show signs of air trapping (increased lung volumes and flattened diaphragms). The actual value of the chest radiograph is to determine whether other serious complications, such as a pneumothorax or pulmonary infiltrate, may also be involved. Laboratory tests, if any, should be ordered specifically to each individual patient. One should consider checking electrolyte levels if the patient is given frequent or continuous  $\beta$ -agonist treatments or if underlying deficiencies of potassium, magnesium, or phosphate are suspected because this therapy will cause an intracellular shift of these ions, thereby further reducing serum levels [25]. Arterial blood gas (ABG) analysis may be performed to confirm a respiratory alkalosis condition caused by tachypnea or follow the resolution of hypercapnea once mechanical ventilation has been instituted, but it should be stressed that the need for an artificial airway and assisted ventilation is based on clinical assessments and must not be delayed while waiting for ABG or other diagnostic results [28].

### *Management*

Oxygen should be administered to keep the arterial oxygen saturation ( $\text{SaO}_2$ ) at  $\geq 90\%$  and  $\geq 95\%$  in pregnant patients or those who have significant CAD [25]. Inhaled  $\beta$ -agonists are the first-line therapy in all asthma exacerbations. They may be delivered with equivalent success by nebulization or by metered-dose-inhaler (MDI) with or without a spacer device [26–28]. A typical nebulized dose of albuterol would be 5 mg every 15 to 20 minutes times three doses. A similar effect can be obtained by giving a continuous nebulized treatment of 15 to 20 mg of albuterol over 1 hour. If a MDI is used, 6 to 8 actuations, 90  $\mu\text{g}$  each, should be given at each treatment to deliver an equivalent dose [26,27,30]. Levalbuterol, the all R-isomer of albuterol (as opposed to the equal mixture of R and S in the standard racemic albuterol) has been shown to be as safe and effective at a dose of 1.25 mg per nebulized treatment and may even be more effective in certain subsets of asthmatics [25]. Ipratropium bromide should also be administered, either by nebulized treatment, 500  $\mu\text{g}$ , or by 4 to 6 MDI actuations. Parenteral  $\beta$ -agonists should be used only if the patient is moribund, coughing excessively, or continues to worsen despite inhaled treatments [26,27,30]. The indicated dosage for epinephrine is 0.2 to 0.5 mg subcutaneously of a 1:1000 solution for three doses every 20 to 30 minutes. If the patient is in shock, it should be given as an IV bolus of 0.2 to 0.5 mg of 1:10,000 solution, followed by an infusion titrated between 1 and 20  $\mu\text{g}/\text{min}$ . An alternative option for the subcutaneous route is terbutaline, 0.25 mg every 20 minutes for three doses [25,28].

Glucocorticoid steroid administration has been shown to decrease admission rates and prevent relapses and should be given to the majority of patients presenting with an asthmatic exacerbation. The one group that does

not require steroids are those with very mild symptoms of short duration, which resolve completely with one inhaled bronchodilator, reflecting only bronchoconstriction without airway inflammation and edema [25,28]. The recommended oral dose of prednisone is 40 to 60 mg (0.5–1.0 mg/kg) daily for 3 to 14 days, with or without a taper. If the patient cannot tolerate the oral administration of prednisone, then methylprednisolone IV, 125 to 250 mg can be given [22–25,27,30].

Dehydration is common, especially if the exacerbation has been ongoing for several days, so that IV fluid administration is recommended by most experts for moderate to severe asthma attacks [3,27]. If an infiltrate is found on chest radiography or empiric antibiotics are given, coverage should include *Mycoplasma* and *Chlamydia* spp [1]. In severe exacerbations, the IV rapid infusion of magnesium sulfate, 2 g over 10 to 20 minutes, has been found to give additional clinical benefit [3,14,16]. There may be some role for the initiation of inhaled steroids or oral leukotriene antagonists in acute exacerbations, but more data need to be collected before these agents can be recommended without reservations [25,26,28]. A mixture of helium and oxygen (heliox) has been found to decrease the work of breathing and improve oxygenation in severe attacks by decreasing airway turbulence and resistance. To be effective, the formulation needs to be 70% to 80% helium and the remainder oxygen, so that heliox is not indicated if the patient requires high oxygen concentrations to maintain an adequate SaO<sub>2</sub> [25,28]. There is no role for sedatives, mucolytics, or opiate cough suppressants in the treatment of acute asthma [25,27].

Most patients will improve after treatments, with only 20% of all asthmatics ever requiring hospitalization [28]. Patients presenting with a PEFr  $\leq 25\%$  of their predicted rate before treatment will likely require admission; if their PEFr remains below 25% despite aggressive therapy, then an ICU stay is warranted. If they improve but PEFr is still  $\leq 40\%$ , then admission is also indicated. Patients who show improvement but not resolution of their symptoms and a PEFr from 40% to 60% should be observed in the office or sent to the emergency department for continued treatment; after an additional 2 to 3 hours, if they have improved, then they can be sent home; but if not, then they should be admitted. Patients who reach a PEFr  $\geq 60\%$  of their predicted best will most likely experience a resolution of subjective symptoms and can be sent home [2,28]. Given the mortality rate associated with asthma, any patient who does not respond to outpatient therapy should be considered for emergency department care to help determine the best course of therapy and to help determine disposition.

### **Chronic obstructive pulmonary disease**

COPD encompasses a group of chronic lung disorders, but emphysema and chronic bronchitis are the disorders most commonly encountered in clinical practice and will be the focus of this discussion. A reduction in bronchial airflow caused by nonreversible disorders is the underlying

pathophysiology. Permanent enlargement of air spaces distal to the terminal bronchiole with wall destruction but without fibrosis defines emphysema. The loss of elastic recoil of lung tissue allows the collapse of distal airways, promoting air trapping [1]. COPD has an incidence of 34.1 per 1000 people over the age 65 years, affecting 14 to 20 million people in the United States, making it the fourth leading cause of death [27,31]. It is responsible for 4 million office visits and 500,000 hospital admissions yearly. COPD patients typically experience one to three exacerbations per year, in which between 3% and 16% of the episodes require hospitalization; the mortality rate is 3% to 10% for each hospital stay [31].

### *History*

The typical baseline symptoms of COPD patients include dyspnea and chronic cough with sputum production. During an exacerbation, the dyspnea will worsen, with an increase in tachypnea, a prolonged expiratory phase, and audible wheezing, and the sputum will often increase in volume while becoming purulent [22–24,31]. Approximately 80% of COPD exacerbations are believed to be caused by respiratory infections, with half being viral and half being bacterial [22–24]. Pulmonary irritants (cigarette smoke, smog, and ozone) and medication noncompliance are also common causes. The four elements responsible for the reduction in airflow during an exacerbation are mucosal edema, increased secretions, bronchial smooth muscle constriction, and airway inflammation [22–24].

### *Physical examination*

COPD patients presenting with an acute exacerbation typically have an increased respiratory and heart rate and pursed-lip breathing with a prolonged expiratory phase of the respiratory cycle. The patient's thorax is often increased in the anteroposterior dimension to accommodate the increased lung volumes. Patients will position themselves to maximize respiratory mechanics, often bracing their hands on their knees to stabilize the upper extremities and recruit accessory muscles. An examination of their lungs may reveal expiratory wheezing or may simply be quiet, with markedly diminished air exchange. Other life-threatening disorders that are associated with COPD should be considered, such as pneumothorax, which may produce asymmetric or absent breath sounds on a hemithorax, distended neck veins, and tracheal deviation, whereas pneumonia may cause signs of focal pulmonary consolidation or pleural effusion. Acute agitation may be a sign of hypoxemia, whereas somnolence or lethargy may indicate an elevated partial pressure of carbon dioxide ( $P_{CO_2}$ ) caused by hypercarbic respiratory insufficiency.

### *Diagnostic studies*

Continuous pulse oximetry is necessary to ensure an adequate level of  $SaO_2$  ( $\geq 88\%$ – $90\%$ ), especially if supplemental oxygen is being

administered, without overshooting and encouraging CO<sub>2</sub> retention by suppression of the hypoxic respiratory drive [22–24]. Chest radiographs typically reveal hyperinflation with flattened diaphragms and may also show bullae and pulmonary oligemia [22–24]. During a presumed COPD exacerbation, the additional value of the CXR is to evaluate for other causes of acute dyspnea, including pneumothorax, pneumonia, and congestive heart failure. Between 16% and 21% of COPD patients have CXR findings that were believed to be helpful by physicians, and treatments were then modified by the findings [31]. Serum electrolytes may be indicated to determine potassium and phosphate level because these are commonly depleted in COPD patients, and serum levels may drop further from intracellular shifts induced by  $\beta$ -agonists. ABG analysis, if available urgently, may be particularly helpful to evaluate for acute CO<sub>2</sub> retention indicated by an elevated PeCO<sub>2</sub> with an acidosis. Although they are not commonly performed at the time of an exacerbation, if pulmonary function tests (PFTs) are obtained, they will confirm an obstructive pattern: a decreased ratio of forced expiratory volume in 1 second (FEV<sub>1</sub>)/forced vital capacity (FVC), an increased total lung capacity and residual volume, and decreased diffusing capacity [22–24].

### *Management*

Oxygen administration is recommended to keep the SaO<sub>2</sub> level at  $\geq 88\%$  to 90% and the PO<sub>2</sub> at  $\geq 60$  mm Hg to maintain tissue oxygenation while preventing pulmonary vasoconstriction and right ventricular strain or pressure overload [22–24,31]. Inhaled  $\beta$ -agonists and anticholinergics should be given by nebulizer or MDI. Albuterol and ipratropium are the most common representatives of these respective classes in the United States. Their effects are additive, so they should be used together, and although albuterol will often give muscular tremor and tachycardia, ipratropium rarely produces any significant side effects [22–24]. Theophylline has been in use for over 70 years and has been found to improve FEV<sub>1</sub> and exercise performance by about 10% in COPD patients by increasing diaphragm strength and endurance and mucociliary clearance. Because theophylline has a narrow therapeutic index, the associated side effects of nausea, vomiting, and tremor are common, whereas life-threatening toxicity includes seizures and cardiac arrhythmias. For these reasons, theophylline has little role acutely but may have a role in outpatient chronic care if used cautiously [22–24,27].

Glucocortical steroids are beneficial acutely and improve outcomes in COPD exacerbations. Either a short course, 4 to 7 days without taper, or a 10- to 14-day course with a taper is effective. Prednisone, 0.5 to 1 mg/kg orally each day, can be used, but an initial dose of IV methylprednisolone or dexamethasone can be given if patients are unable to tolerate oral medications. There is currently not enough evidence to recommend initiating inhaled steroids for acute COPD episodes, but they may also prove to be helpful [22–24,27,31]. Prescribing antibiotics for an exacerbation is believed to decrease bacterial counts in the chronically colonized respiratory tracts of COPD

patients. Currently, the agents recommended for 7- to 10-day outpatient therapy include trimethoprim-sulfamethoxazole, ampicillin, doxycycline, and erythromycin. If the patient has an infiltrate on CXR or has fever or signs of systemic infection, then IV antibiotics effective against *Streptococcus pneumoniae* and *Haemophilus influenzae* should be started [22–24]. Expectorants and mucolytics have been found to provide little or no benefit [22–24,27].

Noninvasive positive pressure ventilation, using either continuous positive airway pressure or bi-level positive airway pressure, may decrease the work of breathing and improve ventilation, but patient cooperation is required to be effective. If acute respiratory failure occurs with hypoxemia ( $PO_2 \leq 50$  mm Hg) and respiratory acidosis despite aggressive medical therapy, tracheal intubation and mechanical ventilation are indicated. These patients should be ventilated cautiously because overzealous correction of acute or chronic hypercarbia may cause severe alkalemia, precipitating seizures or ventricular arrhythmias [22–24].

The majority of COPD patients will improve after therapy and can safely return home. Patients who continue to have dyspnea increased over their baseline will need to be hospitalized if they also experience an inability to walk (if previously ambulatory), loss of appetite or sleep caused by dyspnea, comorbid pulmonary (eg, pneumonia) or nonpulmonary (eg, anemia) conditions, worsened hypoxemia or hypercarbia, new or worsened cor pulmonale, and an inability to manage at home with health care resources (eg, home oxygen) not readily available. Patients will need to be admitted to an ICU if they have alterations in mental status (confusion or lethargy), respiratory fatigue, worsening hypoxemia despite supplemental oxygen, worsening respiratory acidosis ( $pH \leq 7.30$ ), and the need for mechanical ventilation (invasive or noninvasive) [31].

## Pneumonia

Pneumonia is inflammation, most often from infection, affecting the lung parenchyma (respiratory bronchioles and alveolar units) [22–24]. Although there are multiple distinct causes, including viral, fungal, and mycobacterial pneumonias, aspiration pneumonia, ventilator or hospital-acquired pneumonia, and opportunistic infections in immunodeficient individuals, this discussion focuses on community-acquired bacterial pneumonia in immunocompetent adults. An infectious agent is identified in 30% to 40% of the cases of community-acquired pneumonia (CAP), in which the most common bacterial organism is *S pneumoniae*, followed by *C pneumoniae*, *M pneumoniae*, and *H influenzae*. *Legionella pneumoniae* is a rare causative agent in the very elderly (1% in those  $\geq 80$  years old) but is more common in younger adults (8% in those  $\leq 80$  years old). Gram-negative enteric organisms are more common in older patients who have comorbid conditions, particularly diabetes, malignancy, central nervous system disease, and renal, hepatic, or cardiopulmonary diseases [32–34].

The incidence of pneumonia in the United States is 12 cases per 1000 patients per year, resulting in 4 to 5.6 million cases annually. Approximately 25% of these patients will require hospitalization, and 10% of those will require an ICU stay. The risk for developing pneumonia is 10-fold higher in residents of long-term care facilities than for older adults living in the community [27,29,32,35,36]. Pneumonia is the leading cause of infectious deaths and the sixth overall cause of mortality in the United States; in people over age 65, pneumonia moves up to the fifth leading cause of death, resulting in 60 thousand deaths per year. The mortality rate for pneumonia varies depending on the location of treatment, which is a marker for the severity of the disease. For those treated as outpatients, the rate is very low, between  $\leq 1\%$  and 9%, but it is higher for inpatients and approaches 50% in an ICU [32,36,37].

### *History*

Cough, purulent sputum, dyspnea, and pleuritic thoracic pain are reported commonly, localizing symptoms associated with pneumonia; nonspecific symptoms include fever and chills, headache, and myalgias. Elderly patients may have fewer of these symptoms and are more likely than are younger adults to present with a change in mental status. If the lower lobes of the lungs are involved, diaphragmatic irritation can cause upper abdominal pain, referred shoulder or scapular pain, or singultus [32,37,38].

### *Physical examination*

Fever, increased heart rate and respiratory rate, inspiratory crackles, and signs of consolidation (bronchial breath sounds, egophony, increased tactile fremitus) on lung examination are found typically. The patient may appear fatigued, but a rapid evaluation of the mental status is vital to evaluate for possible impending respiratory failure. The skin, mucous membranes, and jugular veins should be examined for evidence of dehydration. Abdominal distension and diminished bowel sounds may be found, caused by paralytic ileus, particularly with lower-lobe pneumonia.  $\text{SaO}_2$  may also be decreased, requiring supplemental oxygen to maintain normal values [37,38].

### *Diagnostic studies*

Chest radiography is necessary to confirm the presence and location of the pneumonia. It may show focal consolidation of air-space pneumonia or a diffuse interstitial pattern; it is also helpful in evaluating for other conditions that may present similarly, such as CHF of a neoplasm. A leukocytosis with an increased percentage of PMNs and immature band forms is common, but leukopenia may occur. ABG analysis, if available, may reveal respiratory acidosis and an elevated  $\text{PaO}_2/\text{FIO}_2$  ratio, showing evidence of respiratory failure caused by severe pneumonia [37,38]. The Infectious

Disease Society of America [34] recommends obtaining a sputum specimen for Gram staining and culture on all inpatients treated for pneumonia, whereas the American Thoracic Society recommends ordering a specimen only if an organism is suspected to be drug resistant or not susceptible to the usual empiric therapy. Blood cultures yield low results and may not need to be ordered routinely, based on studies showing that there were no significant differences in culture rates among patients who have differing pneumonia severity scores and that the rates of bacteremia in pneumonia patients did not significantly influence their hospital stay or mortality rate. Furthermore, blood culture results only rarely offer guidance for clinical decisions or changes in therapy [34,37,39]. A new urinary antigen assay for *S pneumoniae*, which has an 82% sensitivity for diagnosing bacteremia, is now available and may be helpful in identifying these patients [40].

### Management

Most patients diagnosed with pneumonia can be treated with oral antibiotics, but an IV dose should be administered if the patient is unable to take medications orally or is suspected to be bacteremic or acutely ill. The current recommendation by the American Infectious Disease Society of America for the treatment of CAP in adults is a second- or third-generation cephalosporin plus a macrolide or an extended spectrum fluoroquinolone. If the patient is admitted to an ICU, broad-spectrum antibiotics are indicated and should include coverage for *Pseudomonas* organisms if risk factors are present (bronchiectasis, chronic steroid therapy, or recent antibiotic administration) [27,35,37,40,41]. The early initiation of antibiotics (within 8 hours of the patient's arrival) has been shown to decrease the length of stay in the hospital and reduce inpatient and 30-day mortality in patients older than 65 years. The optimum duration of antibiotic therapy is not well defined but is typically from 7 to 21 days [27,35,37,41,42]. Supplemental oxygen should be supplied to maintain a normal SaO<sub>2</sub>, and IV fluids should be administered if the patient has signs of significant dehydration.

Numerous grading systems and scoring scales have been created in an attempt to predict which patients are at higher risk for complications and mortality and require hospitalization or even an ICU stay versus those patients at low risk who can be safely treated as outpatients. Some of these systems include the Pneumonia Severity Index, the Pneumonia Outcome Research Trial (PORT), the British Thoracic Society Scoring System, and the American Thoracic Society Criteria for Severe CAP. These scoring systems generally include similar data (vital signs, comorbidities, CXR, and laboratory studies) to separate patients into different risk classes. Patients are considered to be at high risk and should be hospitalized if they have any of the following: age  $\geq 65$  years, respiratory rate  $\geq 30$  breaths/min, bilateral or multilobar infiltrates, hypotension (systolic  $\leq 90$  mm Hg, diastolic  $\leq 60$  mm Hg), acute renal failure, and altered mental status. If the patient has respiratory failure (defined by PaO<sub>2</sub>/FIO<sub>2</sub> ratio  $\geq 250$  or the need for

mechanical ventilation) or is in septic shock requiring vasopressors, then ICU care will certainly be required. The study result of concern, however, is that in one review of patients in the ICU for CAP, 11% had Pneumonia Severity Index class I scores, and 13% had class II scores (I–V, with I least and V most severe) [35–38,41,43].

### Miscellaneous disorders

Many other less prevalent disorders may lead to dyspnea, some of which can be life threatening and require immediate treatment, whereas others are subacute or chronic conditions for which patients may be followed and treated as outpatients. The history and physical examination often assist in diagnosing these entities, but there may be a need for diagnostic studies (chest radiograph, laboratory studies, referral for pulmonary function studies, laryngoscopy, or bronchoscopy).

Spontaneous pneumothorax can occur without trauma or iatrogenic lung injury and can be further divided into primary (young patients who have normal lung parenchyma but congenital apical blebs) and secondary (older patients who have underlying lung disease such as emphysema or pulmonary fibrosis). The risk is higher for smokers and is greater for men than women. Pleuritic chest pain and dyspnea are the most common complaints. Diminished or asymmetric breath sounds over the affected hemithorax are found on examination. A CXR, specifically an upright expiratory film, is the confirmatory test. Chest tube thoracostomy placement or anterior needle decompression should be performed emergently if the patient appears to have a tension pneumothorax (distended neck veins, tracheal deviation to the opposite side, and hypotension). For small, stable pneumothoraces, simple aspiration is effective but takes longer until resolution than the tube thoracostomy does [27]. These therapies will in most cases require transfer to the ED.

Airway obstruction may be organic, occurring as a result of laryngeal tumors, polyps, or vocal cord paralysis, or be caused by functional vocal cord disorders, such as paradoxical vocal cord movement. Dyspnea and stridor are common features, and visualization of the glottis may be needed to diagnose the obstruction [1,44]. Patients who have neuromuscular diseases resulting in acute weakness, such as Guillain-Barré syndrome or myasthenia gravis, will report dyspnea because they may be using a much greater percentage of their respiratory strength than previously to accomplish thoracic expansion and diaphragmatic excursion. Anxiety disorders often have dyspnea as a central complaint, and hyperventilation syndrome may be identified. Patients who have COPD have a 3-fold increase in the prevalence of anxiety disorders than the general population, so that it may be difficult to readily make this diagnosis [45].

Central nervous system events such as strokes or intracranial hemorrhages will often cause tachypnea and a resultant respiratory alkalosis, but dyspnea is not usually reported [46]. Liver disease and pregnancy are

also associated with an increase in minute ventilation. Moderate to severe anemia will typically cause dyspnea on exertion because of reduced oxygen delivery to peripheral tissues. Metabolic acidosis of any cause (sepsis, increased lactate, or diabetic or alcoholic ketosis) will cause a compensatory increase in minute ventilation that is sensed as SOB by the patient. Toxins, including carbon monoxide and salicylate poisoning, will also cause a symptomatic increase in respiratory rate and tidal volume. These patients, unless a secondary disorder is involved, will all show normal findings on lung examinations.

### Summary

When evaluating a dyspneic patient in the office, a quick initial assessment of the airway, breathing, and circulation, while gathering a brief history and focused physical examination are necessary. Most often, an acute cardiopulmonary disorder, such as CHF, cardiac ischemia, pneumonia, asthma, or COPD exacerbation, can be identified and treated. Stable patients who improve can be sent home, but those in acute distress with unstable or impending unstable conditions need to be transferred emergently to definitive care. Because of the difficult logistics involved in attempting to work up an outpatient for new onset of SOB, some patients will need to be transferred to the nearest ED for a definitive diagnosis.

### Further readings

- Shilon Y, Shitrit AB-G, Rudensky B, et al. A rapid quantitative D-dimer assay at admission correlates with the severity of community acquired pneumonia. *Blood Coagul Fibrinolysis* 2003; 14(8):745–8.
- Stein PD, Terrin ML, Hales CA, et al. Clinical, laboratory, roentgenographic and electrocardiographic findings in patients with acute pulmonary embolism and no pre-existing cardiac or pulmonary disease. *Chest* 1991;100:598.

### References

- [1] Zoorob RJ, Cambell JS. Acute dyspnea in the office. *Am Fam Physician* 2003;68:1803–10.
- [2] Kunitoh H, Wantanabe K, Sajima Y. Clinical features to predict hypoxia and/or hypercapnea in acute asthma attacks. *J Asthma* 1994;31:401–7.
- [3] Hazinski MF, Cummins RO, Field JM. 2000 Handbook of emergency cardiovascular care for healthcare providers. Dallas (TX): American Heart Association; 2000.
- [4] Packer M, Cohn JN; for the Steering Committee and Membership of the Advisory Council to Improve Outcomes Nationwide in Heart Failure. Consensus recommendations for the management of chronic heart failure, II: management of heart failure: approaches to the prevention of heart failure. *Am J Cardiol* 1999;83(Suppl 2A):9A–38A.
- [5] The Criteria Committee of the New York Heart Association. Physical capacity with heart disease. In: *Diseases of the heart and blood vessels, nomenclature and criteria for diagnosis*. 6th edition. Boston: Little, Brown & Co; 1964. p. 110–4.
- [6] Cutaia M. Ambulatory monitoring of oxygen saturation in chronic lung disease: optimizing long-term oxygen therapy. *Clinical Pulmonary Medicine* 2002;9(6):297–305.

- [7] McDonagh TA, Robb SD, Murdoch DR, et al. Biochemical detection of left-ventricular systolic dysfunction. *Lancet* 1998;351:9–13.
- [8] Maisel AS, Koon J, Krishnaswamy P, et al. Utility of B-natriuretic peptide as a rapid, point-of-care test for screening patients undergoing echocardiography to determine left ventricular dysfunction. *Am Heart J* 2001;141:367–74.
- [9] American Heart Association. Heart and stroke facts: 1995 statistical supplement. Dallas (TX): American Heart Association; 1994.
- [10] Tucker NHB, Doty D, Gilmour K, et al, for the Early Diagnosis Steering Committee. Assessment and triage of unstable angina and acute MI. 1998.
- [11] Canto JG, Every NR, Magid DJ, et al, for the National Registry of Myocardial Infarction 2 Investigators. The volume of primary angioplasty procedures and survival after acute myocardial infarction. *N Engl J Med* 2000;342:1573–80.
- [12] Deedwania PC, Carbajal E. Silent myocardial ischemia: a clinical perspective. *Arch Intern Med* 1991;151:2373–82.
- [13] Canto JG, Shlipak MG, Rogers WJ, et al. Prevalence, clinical characteristics, and mortality among patients with myocardial infarction presenting without chest pain. *JAMA* 2000;283:3223.
- [14] Yusuf S, Hawken S, Ounpuu S, et al. Effect of potentially modifiable risk factors associated with myocardial infarction in 52 countries (the INTERHEART study): case-control study. *Lancet* 2004;364:937.
- [15] Antman EM, Anbe DT, Armstrong PW, et al. ACC/AHA guidelines for the management of patients with ST-elevation myocardial infarction—executive summary: a report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines (Writing Committee to Revise the 1999 Guidelines for the Management of Patients With Acute Myocardial Infarction). *Circulation* 2004;110:588.
- [16] Parker JO. Nitrates and angina pectoris. *Am J Cardiol* 1993;72:3C–8C.
- [17] Teo KK, Yusuf S, Furberg CD. Effects of prophylactic antiarrhythmic drug therapy in acute myocardial infarction: An overview of results from randomized controlled trials. *JAMA* 1993;270:1589.
- [18] Calder KK, Herbert M, Henderson SO. The mortality of untreated pulmonary embolism in emergency department patients. *Ann Emerg Med* 2005;45:302–10.
- [19] Fedullo PF, Tapson VF. The evaluation of suspected pulmonary embolism. *N Engl J Med* 2003;349:1247–56.
- [20] Horlander KT, Mannino DM, Leeper KV. Pulmonary embolism mortality in the United States, 1979–1998: an analysis using multiple-cause mortality data. *Arch Intern Med* 2003;163:1711.
- [21] Investigators PIOPED. Value of the ventilation/perfusion scan in acute pulmonary embolism: results of the prospective investigation of pulmonary embolism diagnosis (PIOPED). *JAMA* 1990;263:2753–9.
- [22] Adams L, Stulbarg MS. Dyspnea. In: Murray JF, Nadel JA, editors. *Textbook of respiratory medicine*. 3rd edition. Philadelphia: WB Saunders; 2000. p. 542–9.
- [23] Rennard SI, Shapiro SD, Snider GL. COPD. In: Murray JF, Nadel JA, editors. *Textbook of respiratory medicine*. 3rd edition. Philadelphia: WB Saunders; 2000. p. 1188–230.
- [24] Bushey HA, Burchard EG, Corry DB, et al. Asthma. In: Murray JF, Nadel JA, editors. *Textbook of respiratory medicine*. 3rd edition. Philadelphia: WB Saunders; 2000. p. 1250–77.
- [25] Jaroslaw P, Nowak RM, Zoratti EM. The evaluation and management of acute, severe asthma. *Med Clin North Am* 2002;86:1049–71.
- [26] Cates C, Fitzgerald M. Asthma. In: *Clinical evidence*. Vol. 4. London: BMJ Publishing Group; 2000. p. 828–64.
- [27] Gibbs MA, Camargo CA, Rowe BH, et al. State of the art: therapeutic controversies in severe acute asthma. *Acad Emerg Med* 2000;7(7):800–15.

- [28] Rodrigo GJ, Rodrigo C, Hall JB. Acute asthma in adults: a review. *Chest* 2004;125(3): 1081–102.
- [29] Sherman S. Acute asthma in adults. In: Tintinalli J, Ruiz E, Krome RL, editors. *Emergency medicine: a comprehensive study guide*. 4th edition. New York: McGraw-Hill Co; 1996. p. 430–7.
- [30] Beveridge RC, Grunfeld AF, Hodder RV, et al. Guidelines for the emergency management of asthma in adults. *CMAJ* 1996;155(1):25–37.
- [31] Soto FJ, Varkey B. Evidence-based approach to acute exacerbations of COPD. *Curr Opin Pulm Med* 2003;9(2):117–24.
- [32] Loeb M. Pneumonia in the elderly. *Curr Opin Infect Dis* 2004;17(2):127–30.
- [33] Bjerre LM, Verheij TJM, Kochen MM. Antibiotics for community acquired pneumonia in adult outpatients. *Cochrane Database Syst Rev* 2004;3:CD002109.
- [34] Garcia-Vazquez E, Marcos MA, Mensa J, et al. Assessment of the usefulness of sputum culture for diagnosis of community-acquired pneumonia using the PORT predictive scoring system. *Arch Intern Med* 2004;164(16):1807–11.
- [35] Wilkinson M, Woodhead MA. Guidelines for community-acquired pneumonia in the ICU. *Curr Opin Crit Care* 2004;10(1):59–64.
- [36] Riley PD, Aronsky D, Dean NC. Validation of the 2001 American Thoracic Society criteria for severe community-acquired pneumonia. *Crit Care Med* 2004;32(12):2398–402.
- [37] Alves DW, Kennedy MT. Community-acquired pneumonia in casualty: etiology, clinical features, diagnosis, and management (or a look at the “new” in pneumonia since 2002). *Curr Opin Pulm Med* 2004;10(3):166–70.
- [38] Metlay JP, Fine MJ. Testing strategies in the initial management of patients with community-acquired pneumonia. *Ann Intern Med* 2003;138(2):109–18.
- [39] Ioachimescu OC, Ioachimescu AG, Iannini PB. Severity scoring in community-acquired pneumonia caused by *Streptococcus pneumoniae*: a 5-year experience. *Int J Antimicrob Agents* 2004;24(5):485–90.
- [40] Battleman DS, Callahan M, Thaler HT. Rapid antibiotic delivery and appropriate antibiotic selection reduce length of hospital stay of patients with community-acquired pneumonia. *Arch Intern Med* 2002;162:682–8.
- [41] Alvarez-Lerma F, Torres A. Severe community-acquired pneumonia. *Current Opinion in Critical Care* 2004;10(5):369–74.
- [42] Silber SH, Garrett C, Singh R, et al. Early administration of antibiotics does not shorten time to clinical stability in patients with moderate-to-severe community-acquired pneumonia. *Chest* 2003;124(5):1798–804.
- [43] Oosterheert JJ. Comparison of guidelines for diagnosis of severe community-acquired pneumonia. *Curr Opin Infect Dis* 2003;16(2):153–9.
- [44] McGeehan M, Busse WW. Refractory asthma. *Med Clin North Am* 2002;86:1073–90.
- [45] Brenes GA. Anxiety and chronic obstructive pulmonary disease: prevalence, impact, and treatment. *Psychosom Med* 2003;65(6):963–70.
- [46] Rose BD, editor. *Respiratory acidosis*. In: *Clinical physiology of acid-base electrolyte disorders*. 4th edition. New York: McGraw-Hill Co; 1994. p. 540–603.