

Acute Dyspnea: A Sign of Underlying Disease

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Dyspnea, defined as an uncomfortable awareness of breathing,¹ is a subjective sensation for which there is no accurate objective measurement. Because the word itself is not typically used by patients, the physician is left to interpret the patients' verbalized symptoms and decide what qualifies as dyspnea and what does not. Dyspnea usually is either cardiac or pulmonary in origin, although healthy individuals may experience dyspnea with exercise. Acute dyspnea is defined as dyspnea arising over the course of 24 to 48 hours. This article discusses the pathophysiology and common causes of acute dyspnea and reviews the evaluation of the patient with acute dyspnea.

PATHOPHYSIOLOGY

The pathophysiology of dyspnea is complex, and the neural pathways underlying the symptom are not well understood. Regions of the cerebral cortex have been mapped for visual, olfactory, and other sensations, but no region for dyspnea has been isolated. Additionally, there is no known cortical lesion that will abolish the sensation of dyspnea. The study of dyspnea is further complicated by the inability to define the precise physical stimulus that causes it.

The subjective sensation of dyspnea is modified by a complex interaction of feedback loops that include receptors in the chest wall, respiratory muscles, lung parenchyma, upper airway, larynx, chemoreceptors, mechanoreceptors, and possibly the face. These receptors send and receive input to and from the motor and sensory cortex as well as the brain stem.¹ Several other receptors are known to have a role in the generation of the sensation of dyspnea.^{2,3} Intuitively, one would think that hypoxia has a clearly described role in the generation of dyspnea; however, this is not the case. There are few studies that formally examine the relationship between hypoxia and dyspnea. Furthermore, some patients experience notable hypoxia without dyspnea and many patients experience dyspnea without hypoxia.

CAUSES OF ACUTE DYPNEA

- Pulmonary disease
- Cardiac disease
- Acute blood loss
- Metabolic acidosis
- Anxiety
- Poor physical condition

This situation may be partially explained by the fact that mechanoreceptors located in the face, upper airway, lung, and chest wall are all thought to affect the individual perception of dyspnea.¹

EVALUATION OF THE PATIENT WITH ACUTE DYPNEA

Work-up of acute dyspnea begins with a complete and thorough history and physical examination. Studies have shown that a medical history and clinical examination alone predicts the final diagnosis in 70% to 80% cases.⁴⁻⁶ Causes of acute dyspnea are multifactorial in up to one third of cases. The most common etiologies are asthma, congestive heart failure (CHF), chronic obstructive pulmonary disease (COPD), pneumonia, cardiac ischemia, and psychogenic.⁵ Hospitalization of patients with acute dyspnea should be considered in those who are hemodynamically unstable (eg, hypotensive and tachycardic), who have hypoxia (eg, oxygen saturations below 90%), or who will require rapid diagnostic procedures or aggressive therapeutic regimens to control their symptoms.

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Table 1. Differentiating History and Physical Examination Findings for Cardiac and Pulmonary Causes of Dyspnea

	Cardiac Disease	Pulmonary Disease
History	Dyspnea on exertion	Dyspnea with rest and exertion
	Paroxysmal nocturnal dyspnea	Tobacco use
	Orthopnea	Cough
	Associated chest pain	Sputum production Wheezing Pleuritic chest pain
Physical examination	Jugular venous distention	Expiratory wheezes Decreased air movement
	Peripheral edema	Resonance to percussion
	Ascites	Barrel-chested physique
	Pleural effusions	
	Pulmonary edema	
	Cardiomegaly	
	S ₃ gallop	

Adapted from Sherman DL, Ryan TJ. Differentiating cardiac and pulmonary causes of dyspnea. *ACC Curr J Rev* 1995;4:65 with permission from Elsevier.

History

History taking should be tailored to include information pertinent to evaluating potential etiologies for dyspnea. Pulmonary and cardiac disease are responsible for the vast majority of cases; therefore, special attention should be given to these possibilities. **Table 1** outlines the differentiating history and physical examination findings for cardiac and pulmonary disease. **Table 2** lists the major causes of acute dyspnea.

Pre-existing pulmonary or cardiac disease should be noted because the presence of either makes recurrence or exacerbation of the underlying process more likely. Acute dyspnea in a patient with underlying COPD is likely to indicate an acute exacerbation of COPD. Information about the onset, timing, associated symptoms, severity, and exacerbating and relieving factors obviously are important. Physicians should specifically ask about the presence of pedal edema, paroxysmal nocturnal dyspnea, orthopnea, angina, or palpitations; these symptoms may indicate occult heart disease. Wheezing, cough, sputum production, hemoptysis, recent upper respiratory infection, or a history of smoking may suggest a pulmonary etiology for the dyspnea. In addition, the occupational history may point to a pulmonary cause (eg, sandblasting and silicosis or pipefitting and asbestosis).

Additional medical history findings that may be helpful include the presence of fever, chills, night

sweats, weight loss, change in appetite, chest pain/pleurisy, recent trauma, or symptoms of gastroesophageal reflux disease. A detailed medication history always should be taken. Drugs may cause hemolytic anemia (quinidine and penicillin); methemoglobinemia (nitrites and nitrates); sulfhemoglobinemia (dapsone and sulfonamides); and acute or chronic fibrosis (nitrofurantoin or amiodarone). Aspirin sensitivity is a cause of asthma in a significant number of patients.

Acute myocardial infarction (MI) is an important cause of acute dyspnea. A study evaluating the frequency of symptoms among 88 patients who presented with acute MI found that 47% reported shortness of breath.⁷ The most common symptoms of acute MI in this study were chest pain and diaphoresis, occurring in 64% and 78% of patients, respectively.⁷ Angina pectoris is defined as chest pain of cardiac origin due to an imbalance between myocardial oxygen supply and demand. Patients frequently describe the discomfort as a heavy pressure or squeezing that usually is brought on by exertion. Associated symptoms include dyspnea, diaphoresis, nausea, vomiting, and weakness.

Details in the past medical history may be useful for establishing a diagnosis. History of proximal deep venous thrombosis should prompt a search for pulmonary thromboembolism or pulmonary hypertension. Previous history of cancer, particularly breast or bronchogenic, should raise the suspicion of a malignant pleural effusion. Although usually associated with chronic dyspnea, previous thoracic radiation and chemotherapy with busulfan or other agents are known to be associated with pulmonary fibrosis, which may present with acute dyspnea.

Physical Examination

Results of the physical examination can direct the physician toward a specific diagnosis. General assessment of the patient may yield such information as severity of dyspnea, presence of tachypnea, central or peripheral cyanosis, presence of pursed lip breathing (indicative of severe obstructive lung disease), central obesity consistent with obstructive sleep apnea, or extreme cachexia (suggestive of malignancy). Pallor raises anemia as a consideration. The pharynx should be examined for signs of obstruction or enlarged tonsils, which may predispose to obstructive sleep apnea. Stridor, indicative of an upper airway obstruction from laryngospasm, tumor, or vocal cord dysfunction, should be sought.

Auscultation of the lung fields can elicit further evidence of a pulmonary etiology. A localized wheeze may be evidence of foreign body or tumor. Decreased or

absent breath sounds unilaterally may represent pneumothorax (when accompanied by hyperresonance to percussion) or pleural effusion (when accompanied by dullness to percussion). Crackles or rhonchi are indicative of pneumonia, pulmonary fibrosis, or pulmonary edema. Wheezing, a prolonged expiratory phase of respiration, increased lung fields by percussion, or palpable liver without hepatomegaly is indicative of obstructive airway disease. Pleural friction rubs may be a sign of pleurisy or pulmonary infarction.

CHF is suggested by the presence of an S₃ gallop, bibasilar crackles, elevated jugular venous pressure, a laterally displaced point of maximal cardiac impulse, and/or peripheral edema. S₃ gallop, a sign of left ventricular failure, can be heard in conditions resulting in rapid ventricular filling and volume overloading, such as mitral or aortic insufficiency. An S₃ gallop is low pitched and is heard best at the apex with the bell of the stethoscope. An increased pulmonic S₂, right ventricular heave, and elevated jugular venous pressure are indicative of pulmonary hypertension.

S₄ (atrial gallop) in a patient presenting with acute dyspnea is suggestive of decreased left ventricular compliance. S₄ is a presystolic, low-pitched sound that occurs just before S₁ and is heard best at the apex with the bell of the stethoscope. S₄ is encountered in conditions that cause decreased ventricular compliance such as hypertension, aortic stenosis, coronary artery disease, acute MI, and hypertrophic cardiomyopathy. These conditions cause increased resistance to ventricular filling following atrial contraction.

Evidence of deep venous thrombosis may indicate the presence of a pulmonary thromboembolism. Most pulmonary embolisms arise from venous thromboembolisms of the lower extremity. The possibility of pulmonary embolism is suggested by the acute onset of dyspnea, pleuritic chest pain, severe hypoxia, and risk factors such as recent surgery, underlying malignancy, and a bedridden or sedentary state. One study found that the most common symptoms of pulmonary embolism include dyspnea (73%), pleuritic pain (66%), cough (37%), lower extremity edema (28%), and hemoptysis (13%).⁸ Physical signs included crackles on lung auscultation (51%), and tachycardia (30%).⁸

Laboratory Examination

The laboratory work-up of dyspnea is directed by the results of the history and physical examination. Chest radiograph, oxygen saturation, arterial blood gas (ABG) analysis, electrocardiogram (ECG), echocardiogram, and cardiac enzyme levels may help differentiate between an acute pulmonary versus an acute cardiac

Table 2. Causes of Acute Dyspnea

Pulmonary

- Airway obstruction
 - Foreign body, tumor/secretion/edema/inflammation
 - Acute exacerbation of chronic obstructive pulmonary disease
- Parenchymal disease
 - Infection
 - Malignancy
 - Primary
 - Metastatic
 - Atelectasis
 - Vascular disease
 - Thromboembolus/tumor embolus/fat embolus
 - Pulmonary hypertension
 - Vasculitis
 - Pulmonary edema
 - Noncardiogenic
- Pleural disease
 - Effusion
 - Pneumothorax

Cardiac

- Ischemic disease
- Valvular disease
- Septal defects
- Cardiomyopathy
- Myxoma
- Pericardial disease
 - Effusion
 - Pericarditis

Other

- Respiratory muscles/thoracic cage
 - Systemic neuromuscular disease
 - Phrenic nerve dysfunction
- Anemia/hemoglobinopathy
 - Decreased/abnormal hemoglobin
 - Carboxyhemoglobin
- Metabolic acidosis
- Hyperthyroidism
- Ascites
- Gastroesophageal reflux disease
- Anxiety/psychogenic

etiology. A ventilation-perfusion scan and/or a lower extremity Doppler study should be performed if pulmonary thromboembolism is suspected.

In patients presenting with an exacerbation of



Figure 1. Chest radiograph for patient 1, revealing a right upper lobe infiltrate.

COPD or asthma, measurement of oxygen saturation is essential. Pulse oximetry is an effective screening tool for detecting hypoxia, and it has the advantage of lower cost and discomfort to the patient compared with ABG sampling. Pulse oximetry also is easy to use, and it provides immediate results and continuous assessment. However, an ABG analysis may be needed in the initial assessment of a chronic smoker with acute dyspnea because of elevated carboxyhemoglobin levels caused by smoking. Carbon monoxide causes the pulse oximeter to overestimate the arterial oxygen saturation, especially when carboxyhemoglobin levels exceed 2%.⁹

For patients presenting with acute chest pain, the ECG is the most important initial laboratory examination. A prospective study of 247 patients who presented to an emergency department with acute chest pain found that the initial history, physical examination, and ECG are the most important predictors of cardiac events, with a 96% sensitivity of predicting a cardiac event.¹⁰ The addition of cardiac marker data, including serum troponin I levels, did not improve the positive predictive value in this patient population beyond that of the history, physical examination, and ECG.¹⁰

CHF is a very common clinical problem among the elderly and often is misdiagnosed in an urgent care setting because of nonspecific symptoms. B-type natriuretic peptide (BNP), a cardiac neurohormone, recently has been identified to have diagnostic potential in patients with left ventricular dysfunction.¹¹ BNP is released by the ventricles in response to increased end-diastolic pressure or volume expansion. A BNP level of 100 pg/mL is highly indicative of decompensated heart failure.¹¹

ILLUSTRATIVE CASES

Case 1 Presentation

A 75-year-old man with a past medical history of coronary artery disease presents with a 1-day history of dyspnea, right-sided chest pain, and cough with rust-colored sputum. Further history reveals subjective fever and chills. His physical activity level has diminished over the last 2 days. Physical examination reveals the patient to be mildly tachypneic and afebrile but in no acute distress. Cardiac examination is without significant findings. There are crackles and a friction rub in the right anterior lung field. Laboratory examination demonstrates a mild leukocytosis and a Pao_2 of 60 mm Hg.

Discussion

In this patient, the differential diagnosis includes pulmonary thromboembolism, CHF, acute MI, COPD exacerbation, pneumothorax, pleural effusion, and pneumonia. Findings of new onset dyspnea, chest pain, hypoxemia, and friction rub all are consistent with a diagnosis of pneumonia and pulmonary thromboembolism. However, a new cough with sputum production would be unusual in pulmonary thromboembolism. Neither the history nor the physical examination yields signs or symptoms of acute cardiac decompensation that would indicate a cardiac etiology. There are no findings consistent with pneumothorax or pleural effusion, and the unilateral findings make a COPD exacerbation unlikely. A chest radiograph is ordered and is consistent with pneumonia (**Figure 1**).

Case 2 Presentation

A 50-year-old man presents with complaints of chest pain and dyspnea for the past 6 hours. He describes the pain as a tightness that is substernal in location, mainly on the left side. The pain radiates to his left arm and left jaw. Physical examination reveals blood pressure of 150/90 mm Hg and pulse of 110 bpm. Cardiac examination reveals an S_4 gallop but no murmurs. Lung examination reveals crackles at the bases bilaterally. An ECG is ordered (**Figure 2**).

Discussion

In this patient, the main diagnostic consideration is an acute coronary event because dyspnea frequently accompanies cardiac symptoms. The dyspnea seen in patient 2 is caused by pulmonary edema related to acute left ventricular dysfunction. The crackles detected on lung examination are consistent with CHF. The ECG confirms acute MI as the cause. Further studies, such as cardiac catheterization and echocardiogram, also should be considered.

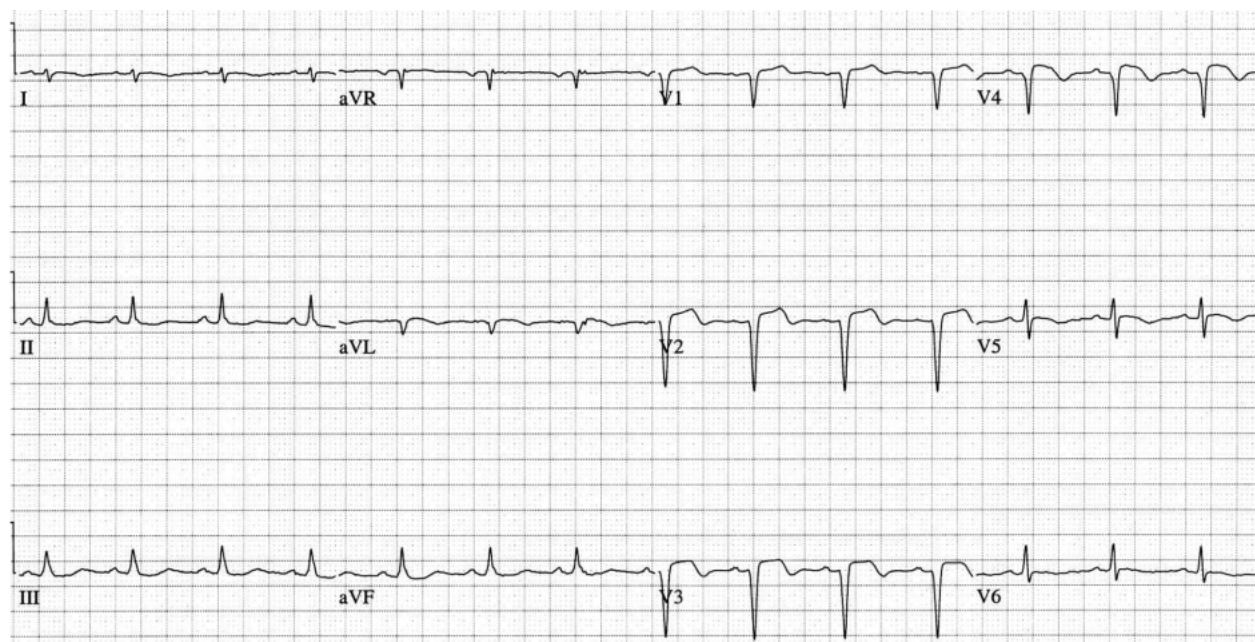


Figure 2. Electrocardiogram for patient 2, showing an ST-segment elevation in the anterior segment (V₁-V₄) that is consistent with acute anteroseptal myocardial infarction.

CONCLUSION

Acute dyspnea is a common symptom seen in clinical practice. It is most commonly pulmonary or cardiac in origin. A thorough history and physical examination are important for effectively directing the work-up. **HP**

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