Acute Dyspnea: Is the Cause Cardiac or Pulmonary—or Both?

ABSTRACT: The rate at which acute dyspnea develops can point to its cause. A sudden onset strongly suggests pneumothorax (especially in a young, otherwise healthy patient) or pulmonary embolism (particularly in an immobilized patient). More gradual development of breathlessness indicates pulmonary infection, asthma, pulmonary edema, or neurologic or muscular disease. A chest film best identifies the cause of acute dyspnea; it can reveal pneumothorax, infiltrates, and edema. Pulmonary embolism is suggested by a sudden exacerbation of dyspnea, increased ventilation, and a drop in $PaCO_2$. A normal chest radiograph reinforces the diagnosis of pulmonary embolism, which can frequently be confirmed by a spiral CT scan of the chest. Pneumonia can be difficult to distinguish from pulmonary edema. In this setting, bronchoalveolar lavage and identification of the infectious organism may be necessary to differentiate between the 2 disorders.

Key words: dyspnea, pulmonary disease, cardiac disease

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The survival of a patient with acute dyspnea may depend on your ability to identify and promptly treat the underlying cause. In most cases, a logical approach and basic studies can provide the diagnosis; however, patients with preexisting cardiopulmonary disease may require more extensive investigation.

In this article, I describe the clues in the history, physical examination, and chest film that can alert you to the underlying cause of dyspnea. I also discuss the circumstances in which further workup is warranted.

PATHOPHYSIOLOGY

The precise mechanism underlying dyspnea remains unclear. The generally accepted concept is of a mismatch between CNS neural drive ("motor command") to the respiratory system and the resultant ventilation.[1]

Dyspnea appears to be related to 2 factors:

- The intensity of reflex stimulation of the central motor command output (volitional stimulation does not seem to be a factor).
- The degree to which respiratory muscle activity is hindered by mechanical abnormality (such as obstruction or restriction).[2]

Consequently, dyspnea can result from intense reflex stimulation of breathing by the chemoreceptors from elevated arterial PCO_2, or stimulation of pulmonary or diaphragmatic vagal nerve afferents as a result of pulmonary inflammation (eg, pneumonia), an acute pulmonary vascular abnormality (eg, pulmonary embolism), engorgement (eg, pulmonary edema), respiratory mechanical defects (eg, airways obstruction), or a diaphragmatic defect.[1]

Acute dyspnea may be associated with a pulmonary and/or cardiac abnormality or disease. Cardiac conditions that may precipitate dyspnea include left ventricular systolic or diastolic dysfunction, arrhythmias, valvular disease, pericardial disease, myocardial infarction (MI), pulmonary hypertension, and intracardiac or extracardiac shunts.[3] Dyspnea may also be psychogenic.[4]

Bear in mind that each patient perceives the degree of breathing difficulty differently. You may easily be misled about the severity of the underlying illness if, for example, an extremely stoic patient complains only of mild shortness of breath.

HISTORY

Valuable clues are provided by the history. Pay particular attention to the rate at which dyspnea developed.

Sudden onset of dyspnea. Sometimes pinpointed by the patient almost to the breath, a sudden onset strongly indicates either pneumothorax or pulmonary embolism. Pneumothorax is more likely in a healthy young person with no underlying illness, whereas pulmonary embolism

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Table – Underlying causes of acute dyspnea and diagnostic findings

Cause	Signs and symptoms	Radiologic imaging	ECG findings	Other diagnostic clues
Pneumothorax	Sudden onset of dyspnea, chest pain, mediastinal shift to contralateral side, ipsilateral hyperresonance to percussion, absence of breath sounds	Chest radiograph: diagnostic	Usually normal	—
Pulmonary embolism	Sudden onset of dyspnea; underlying risk factors (eg, bed rest); chest examination may show no abnormalities; jugulovenous pressure may be raised with accentuated pulmonic second heart sound	Chest radiograph: may be normal or may show a variety of abnormalities Spiral chest CT angiogram: highly specific with good sensitivity	May be normal or may show a variety of abnormalities, including tall P waves and QRS abnormalities	May have fever, hemoptysis, or deep venous thrombosis; spiral CT scanning of the chest or pulmonary angiography is diagnostic
Pulmonary edema (left-sided heart failure)	Angina, gallop rhythm	Chest radiograph: pulmonary edema, enlarged cardiac silhouette	Abnormal, may show evidence of left atrial or ventricular hypertrophy and/or ischemic changes	Pulmonary capillary wedge pressure is raised
Pulmonary edema (noncardiac)	Precipitating factor, increasing hypoxia	Chest radiograph: pulmonary edema, normal cardiac silhouette	Usually normal	Pulmonary capillary wedge pressure is usually normal
Pulmonary infection	Cough, fever, sputum; dyspnea may be only symptom in immunocompromised patients	Chest radiograph: infiltrates, changes may be subtle in immunocompromised patients	Usually normal	Leukocytosis, identification of pathogenic organism
Asthma/ exacerbation of chronic bronchitis	History of disease, wheezing, expiratory rhonchi	Usually normal	Usually normal	Spirometry reveals airflow obstruction
Diffuse pulmonary fibrosis	Dyspnea, inspiratory crackles over both lung bases	Chest radiograph: normal or bilateral reticulonodular changes Chest CT scan: bilateral reticulonodular changes	Usually normal	Pulmonary function tests reveal restrictive defect with decreased diffusing capacity
Diaphragmatic paralysis	Underlying neurologic or myopathic disorder or recent thoracic surgery	Chest radiograph: elevated diaphragm Chest fluoroscopy: diagnostic	Normal	Special radiologic/ electrophysiologic studies may be required for diagnosis
Psychogenic	Repeated episodes, usually related to psychological stress; may be accompanied by wheezing	Normal	Normal	Diagnosis of exclusion

must be considered in a patient with risk factors for deep venous thrombosis, such as prolonged bed rest.

Subacute onset of dyspnea. This can result from acute pulmonary infection, asthma, acute exacerbation of chronic bronchitis, diffuse pulmonary fibrosis, pulmonary edema (cardiac or noncardiac), pulmonary hypertension, or diaphragmatic paralysis.

In *pulmonary infection,* the findings of cough and fever are not diagnostically helpful, since either or both may also be present with pulmonary embolism or pulmonary edema (**Table**). The absence of cough and fever makes the possibility of pulmonary infection unlikely. Among immunocompromised patients, however, especially those who are HIV-positive, increasing shortness of breath is often the sole presenting symptom of pulmonary infection.

Asthma or acute exacerbation of chronic bronchitis as the cause of increasing dyspnea can usually be diagnosed by the patient's history and the characteristic wheezing that accompanies the dyspnea.

Diffuse pulmonary fibrosis from any cause usually presents with the gradual onset of dyspnea. This may be associated with the characteristic inspiratory crackles over the lung bases. Other signs of diseases causing pulmonary fibrosis, such as sarcoidosis, scleroderma, and asbestosis, may be present or it may be idiopathic.[5]

Pulmonary edema (cardiogenic or noncardiogenic) is manifested by the subacute onset of shortness of breath. Acute cardiogenic pulmonary edema is frequently accompanied by cough and frothy, blood-tinged sputum. A cardiac cause may be inferred if the presenting symptoms—such as light-headedness, palpitations, chest pain, and syncope—indicate MI.[6]

Noncardiogenic pulmonary edema is usually preceded a few hours earlier by a pulmonary or neurogenic insult and is associated with increasing hypoxemia. For example, look for a history of recent trauma or head injury, lung contusion, or an episode of severe hypotension. A history of light-headedness, paresthesias of the perioral area or distal extremities, and chest pain suggests the hyperventilation syndrome.

Pulmonary hypertension from any cause may be associated with dyspnea. When this is secondary to cardiac or pulmonary parenchymal disease, symptoms and signs of the underlying disease are usually present. However, *pulmonary arterial hypertension* should also be kept in consideration as a cause of dyspnea.

Recent travel or other potential exposure to contagion may indicate an infectious cause. An occupational history may reveal conditions favoring inhalation of organic or inorganic particulates that can precipitate hypersensitivity reaction or acute toxic lung injury. Hoarseness or aphonia points to upper airways impairment.

Underlying neurologic or muscular disease suggests respiratory muscle weakness as a cause of dyspnea. The onset is usually gradual but may occur rapidly.

PHYSICAL EXAMINATION

The chest examination can confirm the underlying diagnosis. It may reveal pulmonary edema, enlarged heart with gallop rhythm, lobar consolidation, pneumothorax, expiratory wheezes—or no abnormalities.

You may recognize clinical signs of bilateral diaphragmatic paralysis. When the patient is supine, breathing becomes more difficult because the abdomen retracts rather than expands during inspiration.

Although you may detect a third heart sound in patients with right or left ventricular failure, this finding does not necessarily implicate heart disease as a cause of dyspnea. A right ventricular third heart sound, with an accentuated pulmonic component of the second heart sound may also be present in persons with primary (arterial) or secondary pulmonary hypertension.

The finding of fine end-inspiratory crackles over both lung bases suggests pulmonary edema, whereas coarse, localized crackles suggest a pneumonic process. Diffuse medium inspiratory crackles over the bases would suggest diffuse pulmonary fibrosis. Expiratory rhonchi indicate airways obstruction; in severe cases, both inspiratory and expiratory rhonchi are present. Unilateral hyperresonance to percussion and absent breath sounds suggest the presence of a pneumothorax.

A fourth heart sound that is transmitted to the suprasternal notch usually originates in the left ventricle. A fourth heart sound transmitted to the right internal jugular vein usually originates in the right ventricle.

IMAGING AND LABORATORY STUDIES

Chest roentgenography. A chest film best identifies the cause of acute dyspnea. In a patient with acute onset of shortness of breath and an underlying risk factor—such as prolonged immobilization (eg, because of acute illness, surgery, or old age) or a clotting abnormality—normal radiographic findings suggest pulmonary embolism. Spiral CT of the chest, which is much less invasive than pulmonary angiography, has become an important tool for diagnosing pulmonary embolism with good sensitivity and specificity.[7,8]

Pneumothorax, pneumonic infiltrates, and pulmonary edema are usually obvious on the chest film (**Figure**). Although the size of the cardiac silhouette may differentiate between cardiac and noncardiac pulmonary edema, this is not a reliable distinguishing feature.

The chest roentgenogram may reveal only subtle abnormalities during the early stages of infection in im-

munocompromised patients. There may be localized or generalized haziness or a fine reticulonodular pattern. A relatively normal appearance does not exclude early infection with, for example, *Pneumocystis carinii*. Suspect diaphragmatic paralysis if the diaphragm is elevated.

ECG. In patients who have no history of heart disease, an ECG helps distinguish cardiac from noncardiac causes of acute dyspnea, and may also indicate pulmonary hypertension.

Arterial blood gas analysis. This is an exclusionary test. Normal values, with no increase in the alveolar-arterial PO_2 gradient, suggest the absence of a significant pulmonary abnormality and point toward a psychogenic cause of dyspnea.

Brain natriuretic peptide levels in plasma. These have been found to be useful in distinguishing the cause of dyspnea. Brain natriuretic peptide (BNP) levels are increased in left heart dysfunction, whereas they are normal in pulmonary disease.[9] However, BNP levels are also increased in right ventricular dysfunction, such as occurs in cor pulmonale or pulmonary embolism.[10,11] Bear this in mind when you consider BNP measurement as a diagnostic test for the cause of dyspnea.

PATIENTS WITH PREEXISTING DISEASE

Diagnostic problems arise in patients with preexisting cardiac or pulmonary disease, most of whom already have some degree of dyspnea at rest or during exercise. The development of new disease exacerbates the dyspnea, and you must identify this new cause.

Because cardiopulmonary disease is a risk factor for pulmonary embolism, always keep this diagnosis in mind. Clues to pulmonary embolism in this setting include sudden intensification of dyspnea, increased ventilation, and a fall in $PaCO_2$. Documentation of deep venous thrombosis (by noninvasive or invasive techniques) makes the diagnosis more likely. Radioisotope ventilation-perfusion lung scans are not helpful because of underlying cardiopulmonary disease. In this setting, spiral CT of the chest is frequently diagnostic and has now become the procedure of choice.[12] Pulmonary angiography is

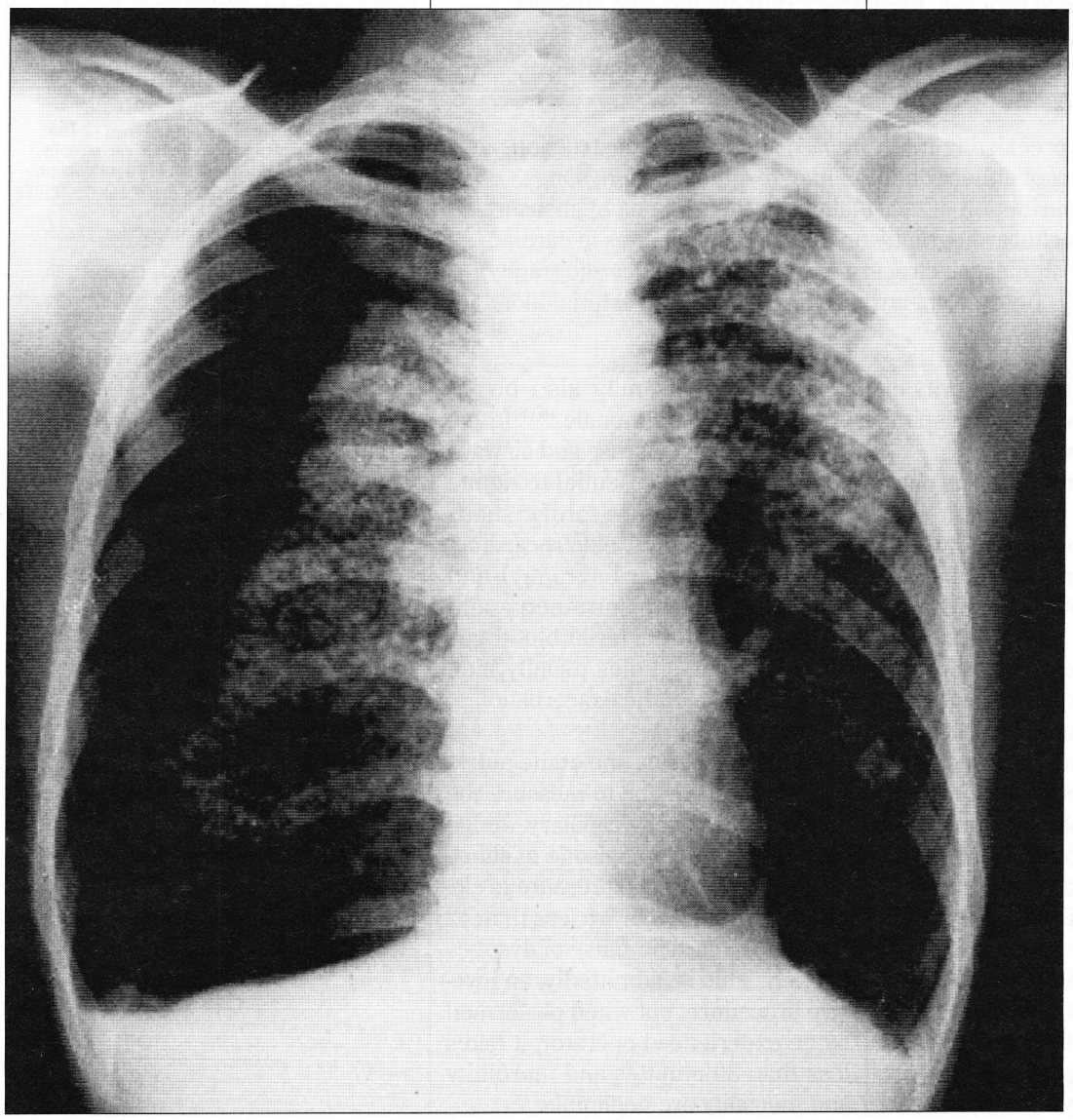

Figure – The pleural line can be seen clearly in this patient with a right pneumothorax. (The nodules scattered throughout the lungs resulted from silicosis.) A chest film is highly valuable in identifying the cause of acute dyspnea.

(Courtesy of Edward Y. Sako, MD, PhD, and Jay I. Peters, MD.)

CLINICAL HIGHLIGHTS

❑ Pulmonary edema is manifested by the subacute onset of dyspnea. Cough and frothy, blood-tinged sputum frequently accompany acute cardiogenic pulmonary edema.

❑ Noncardiogenic pulmonary edema is usually preceded a few hours earlier by a pulmonary or neurogenic insult and is associated with increasing hypoxemia. For example, look for a history of recent trauma or head injury, lung contusion, or an episode of severe hypotension.

❑ Underlying neurologic or muscular disease suggests respiratory muscle weakness as a cause of dyspnea. The onset is usually gradual but may occur rapidly.

❑ In a patient with acute dyspnea, fine end-inspiratory crackles over both lung bases suggest pulmonary edema, medium end-inspiratory crackles suggest pulmonary fibrosis, and coarse, localized crackles suggest a pneumonic process. Expiratory rhonchi indicate airways obstruction; in severe cases, both inspiratory and expiratory rhonchi are present. Unilateral hyperresonance to percussion and absent breath sounds suggest pneumothorax.

❑ In patients with preexisting heart disease and airways obstruction, a severe exacerbation of either condition may result in acute dyspnea. Distinguish the contribution of heart failure to the dyspnea by measurement of BNP and echocardiography and define any changes in cardiac status (by enzyme studies, electrocardiography, and echocardiography) and in lung function (by spirometry and measurement of arterial blood gas levels).

now required only in the minority of patients in whom the results of spiral CT are not conclusive.

Although pneumothorax is obvious on a chest film in a patient with preexisting cardiopulmonary disease, the distinction between pulmonary infection and pulmonary edema may be difficult to make. Purulent sputum, fever, and leukocytosis point toward infection, but diagnosis may require more extensive studies, including bronchoalveolar lavage and protected brush specimens for bacterial, viral, and fungal examination.

Patients with preexisting heart disease and airways obstruction present the most difficult diagnostic problem. A severe exacerbation of either condition may result in acute dyspnea. Measurement of BNP may be very helpful in this setting.[13] An elevated level suggests left heart dysfunction[9]; however, as noted earlier, BNP levels may also be increased in cor pulmonale.[10,11] In patients with heart disease and airways obstruction, it is essential to define clearly any changes in cardiac status (by enzyme studies, electrocardiography, and echocardiography) and lung function (by spirometry and measurement of arterial blood gas levels). These studies usually indicate the cause of the exacerbation of dyspnea.

In some cases, one of the quickest solutions may be to measure the patient's pulmonary capillary wedge pressure during an episode of shortness of breath. If the pressure is elevated (normal levels are less than 12 cm H_2O), a cardiac cause of dyspnea is likely. This is, admittedly, an invasive procedure, but when performed by an experienced operator, it takes less than 30 minutes and morbidity and mortality are negligible.

The best indication of the primary underlying cause is a therapeutic trial, in which therapy for the cardiac and pulmonary conditions is optimized in sequence. For example, you might initially adjust the diuretic and cardiac inotropic or afterload-decreasing therapy to optimal levels. If dyspnea persists, focus on airway drugs, such as bronchodilators and corticosteroids.

In a small proportion of patients, no organic cause of acute dyspnea can be found. In this group, the source is likely to be psychogenic. ■

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