Approach to Adult Patients with Acute Dyspnea

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KEYWORDS

- Dyspnea • Shortness of breath • Asthma • COPD • Respiratory compensation
- Pneumonia • Pulmonary embolism • Angina

KEY POINTS

- The cause of dyspnea is often evident from a complete history and physical examination.
- Rapid determination of the cause of dyspnea saves lives.
- Shortness of breath is not always primarily a pulmonary problem.
- Understanding the pathophysiology of each disease allows clinicians to make rational decisions about testing.

INTRODUCTION

Emergency Medical Services (EMS) calls en route with a 45-year-old woman who has a history of congestive heart failure, chronic bronchitis, morbid obesity, and diabetes. She is breathing 40 times per minute, maintaining oxygen saturations of 94%. She appears mildly confused. You have 5 minutes to consider algorithms before the patient arrives. Perhaps respiratory therapy is paged to supply a ventilator or a biphasic positive airway pressure (BIPAP) machine. Maybe you prepare airway equipment or ask your nursing staff to access medication in advance, or you might use the time to expand your differential and determine what brief information regarding the patient's history and initial physical examination will help you treat her.

Acutely dyspneic patients present in various ways. Are the lungs full of fluid or pus? Did the throat swell shut or is the patient just anxious? Did the patient aspirate a foreign body or have a slow or rapid hemorrhage? Is the patient compensating for a severe metabolic acidosis or did the patient run out of beta agonists at home? This article provides helpful guidelines in the assessment and management of these diverse patients.

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Respiratory distress is responsible for nearly 4 million emergency department (ED) visits each year and is one of the most common presenting complaints in the elderly.[1] When a patient presents with dyspnea, the primary task of the emergency physician is to assess for and ensure stability of the patient's airway, breathing, and circulation. In the case of dyspnea, presentations may range from minor symptoms to extremis. Rapid assessment may necessitate the use of intubation, BIPAP, nebulizations, decompression, or other therapies in the immediate period following the patient's arrival, to treat dyspnea.

PATIENT EVALUATION

The American Thoracic Society suggests that "dyspnea results from a … mismatch between central respiratory motor activity and incoming afferent information from receptors in the airways, lungs and chest wall structures."[2] This dissociation can result from increased metabolic demand, decreased compliance, increased dead-space volume, or many other disorders that are discussed later. Each patient presenting short of breath uses a different set of phrases to describe the symptoms and examination reveals a different combination of disorders. The clinician's ability to interpret these varying constellations is necessary to provide appropriate treatment to these patients, who are often in serious distress.

History

Acute dyspnea, or shortness of breath, is one of the most common chief complaints in the ED. The differential diagnosis includes many disorders that can be divided based on obstructive, parenchymal, cardiac, and compensatory features. A careful history can begin to narrow this wide differential. In addition to common symptoms, consider risk factors such as past medical and family history, trauma, travel, medications, and exposures.

Schwartzstein and Lewis[3] use the analogy of a machine to identify different causes of dyspnea based on pathophysiologic data. Dysfunctions of the respiratory system may be caused by faulty controllers, ventilatory pumps, or gas exchangers (Table 1). This table makes it easier to understand the causes of shortness of breath related to respiratory causes.

Cardiovascular disease manifests as dyspnea by causing disruptions of the system that pumps oxygenated blood to tissues and then transports the carbon dioxide back to the lung. Decreases in cardiac output or increases in resistance limit oxygen delivery. Similarly, decreased oxygen carrying capacity in anemia plays a role in its presentation with dyspnea.

Physical Examination

A detailed physical examination also provides important guidance (Table 2). Respiratory rate and oxygen saturation are obtained with vital signs. The clinician should assess the patient's work of breathing, looking for any tripoding or retractions. Crepitance in the chest may indicate subcutaneous air and pneumothorax. Lung sounds such as wheezing, rales, and rhonchi further guide the differential. Decreased sounds, hyperresonance, or egophony may also provide additional clues.

Jugular venous distension, S3 gallop, and peripheral edema indicate that a patient has fluid overload. Conjunctival pallor, capillary refill, and temperature of extremities can provide clues about blood volume and general circulation. Pulses must also be assessed.

Table 1
A systemic approach to dyspnea by assessing the components of the respiratory process

Part	Description	Manifestations	Examples
Controller	Malfunction presents as abnormal respiratory rate or depth. Often related to abnormal feedback to brain from other parts of the system	Air hunger, need to breathe	Abnormal feedback to brain from other systems. Metabolic acidosis, anxiety
Ventilatory pump	Composed of muscles, nerves that signal the controller, chest wall, and pleura that create negative thoracic pressure, airways and alveoli allowing flow from atmosphere and gas exchange	Increased work of breathing, low tidal volumes	Neuromuscular problems (eg, Guillain-Barré), decreased chest wall compliance, pneumothorax, pneumonia, bronchospasm (COPD, asthma)
Gas exchanger	Oxygen and carbon dioxide cross the pulmonary capillaries in the alveoli. Membrane destruction or interruption of the interface between the gas and capillaries by fluid or inflammatory cells limit gas exchange	Increased respiratory drive, hypoxemia, chronic hypercapnia	Emphysema, pneumonia, pulmonary edema, pleural effusion, hemothorax

Adapted from Schwartzstein RM, Lewis A. Dyspnea. In: Broaddus V, Mason RC, Ernst JD, et al, editors. Murray & Nadel's textbook of respiratory medicine. Elsevier health sciences. 6th edition. Philadelphia: Saunders/Elsevier; 2015.

Testing

Multiple tests are available to narrow the differential diagnosis of acute dyspnea. When using tests to augment clinical decision making, be sure to weigh the information they may provide with any risks involved in performing the tests (**Table 3**).

Ultrasonography provides valuable information about the origin of symptoms, and, often, diagnosis in the initial assessment of an acutely dyspneic patient. These images may be obtained during or shortly after initial assessment, potentially

Table 2
Physical examination findings and correlating diagnoses

Symptom	Differential Diagnosis
Wheeze	COPD/emphysema, asthma, allergic reaction, CHF (cardiac wheeze)
Cough	Pneumonia, asthma, COPD/emphysema
Pleuritic chest pain	Pneumonia, pulmonary embolism, pneumothorax, COPD, asthma
Orthopnea	Acute heart failure
Fever	Pneumonia, bronchitis, TB, malignancy
Hemoptysis	Pneumonia, TB, pulmonary embolism, malignancy
Edema	Acute heart failure, pulmonary embolism (unilateral)
pulmonary edema	Acute and chronic heart failure, end stage renal and liver diseases, ARDS (sepsis)
Tachypnea alone	pulmonary embolism, acidosis (including aspirin toxicity), anxiety

Abbreviations: ARDS, acute respiratory distress syndrome; CHF, congestive heart failure; COPD, chronic obstructive pulmonary disease; TB, tuberculosis.

Table 3
Diagnostic testing for dyspneic patients

Test	General Information	Pros	Cons
Chest radiograph	Often primary imaging	Low radiation, can assess consolidation, pleural fluid, hyperinflation, pneumothorax, and subcutaneous air. Heart size is apparent	Low sensitivity in acute dyspnea. In one series only 8 of 26 pneumonias diagnosed on CT met CXR criteria[37]
Ultrasonography	Multiple protocols to assess acute dyspnea	No radiation, fast, reproducible bedside test, can be done on unstable patients in department and in semirecumbent position	Requires some skill to acquire and interpret bedside images. Patient factors such as subcutaneous air, body habitus, and so forth may limit images
D-dimer	Marker of fibrinolytic activity. When measured by ELISA or second-generation latex agglutination can be used to rule out PE in selected patients	Serum test readily available	Requires risk assessment and clear clinical question. Also increased in consumptive coagulopathy, infection, malignancy, trauma, dissection, preeclampsia, and other cardiovascular disorders
Arterial blood gas	Provides additional information about ventilation (Pa_{CO_2}) to patients with reliable pulse oximetry and bicarbonate level available on BMP	May be faster than general laboratory tests. Useful in assessing anxiety-induced hyperventilation[36]	Limited evidence for routine use in undifferentiated dyspnea
Electrocardiogram	Initial cardiac assessment for assessing dyspnea	Fast and inexpensive. Easy to compare with prior examinations. Specific for dysrhythmias or ACS limiting perfusion	May be nonspecific in findings such as right heart strain and P pulmonale
Troponin	Serum indicator of myocardial damage	Serum test readily available	Can narrow differential to cardiac causes. PE with right heart strain may have increased troponin levels; this finding predicts worse outcomes
BNP and proBNP	Useful in assessing for acute heart failure	Serum test readily available	Limited in obesity, mitral regurgitation, flash pulmonary edema, and renal insufficiency. Context is essential

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Table 3
(continued)

Test	General Information	Pros	Cons
Complete blood count	Provides information about oxygen carrying capacity based on hemoglobin and hematocrit. White blood cell count may indicate infection	Serum test readily available	Nonspecific
CT scan	Provides detailed imaging of cardiorespiratory system. Use is increasing, but practitioners should maintain clinical context and consider whether other modalities can answer the clinical question	Offers sensitive and specific results	Significant radiation exposure, contrast nephropathy, intravenous contrast dye reactions
Ventilation/ perfusion scan	Radiolabeled aerosol and albumin aggregates are used to study ventilation and perfusion. Read as negative or low, medium or high probability for pulmonary embolism	Low in radiation	Limited by underlying pulmonary disease and availability of isotopes

Abbreviations: ACS, acute coronary syndrome; BMP, basic metabolic panel; BNP, B-type natriuretic peptide; CT, computed tomography; CXR, chest radiograph; ELISA, enzyme-linked immunosorbent assay; proBNP, pro–B-type natriuretic peptide.

guiding therapy faster than laboratory tests or other imaging studies would be available. The Bedside Lung Ultrasonography in Emergency (BLUE) protocol offers one approach to differentiate several causes of respiratory failure (**Table 4** and **Figs. 1–9**).

Other protocols include assessments to assess for other cardiac causes of dyspnea.[4–6] Focused evaluation of global left ventricular function, diastolic function, right ventricular size, and any pericardial effusion facilitates rapid assessment for massive myocardial infarction, cardiac tamponade, and massive pulmonary embolism at the bedside. In addition, inferior vena cava measurement can be used to assess for right-sided heart failure and to estimate central venous pressure.

Computed tomography (CT) use to evaluate acute dyspnea has increased in the last decade.[7] Risks include contrast reactions and nephropathy as well as radiation-induced cancers.[8] Recent American College of Physicians recommendations advocate avoidance of CT as an initial test to evaluate patients at low risk for pulmonary embolism (PE).[9] Further, nearly one-fourth of patients undergoing CT for PE evaluation

| | | **Table 4** | | |
| | | **BLUE protocol** | | |
Ultrasonography Finding	**Ultrasonography Approach**	**Description**	**Clinical Meaning**	**Image**
Assess for artifacts: A lines	Anterior	Subpleural air causes repeated linear artifacts parallel to the pleural line (horizontal)	Air in lung: either normal or pneumothorax	Fig. 1
Assess for artifacts: B lines	Anterior	Seven features: hyperechoic, well-defined, hydroaeric comet-tail artifacts arising from the pleural line. They spread upwards indefinitely and obscure A lines. When lung sliding is present, they move with the lung	Represents an interface of 2 widely different transmissions of ultrasound waves: in this case, air and fluid. When 3 or more B lines are in a single interspace, they are B+ lines (or pulmonary rockets), indicating interstitial syndrome	Fig. 2
Assess for lung sliding	Anterior	Absence of lung sliding occurs with a disruption of the normal sliding of viscera on parietal pleura or separation of the two. In M mode, absence of lung slide is seen as the stratosphere sign (also known as bar-code sign)	Absence of lung sliding in the presence of A lines necessitates search for pneumothorax. Lung point is the ultrasonography finding in which lung slide is seen in the same view with the abolished lung slide and A lines in the same location, indicating the tip of the lung	Fig. 3 (stratosphere sign) Fig. 4 (normal lung) Fig. 5 (lung point)
Assess for alveolar consolidation or pleural effusion (posterolateral alveolar and/or pleural syndrome)	Lateral subposterior	The classic anechoic, dependent pattern may be inconsistent. Other findings include (1) quad sign: pleural effusion on expiration noted between the pleural and regular, lower lung lines (viscera). (2) Shred sign: tissuelike pattern seen with alveolar consolidation, with the upper border of lung line (or pleural line when there is no effusion) with an irregular lower border. (3) Sinusoid sign: movement of the lung line toward the pleural line in inspiration	Pleural effusion: sinusoid, plus may have quad sign. Alveolar consolidation: tissuelike appearance or shred sign, absent lung line, absent sinusoidal sign	Figs. 6 and 7 (pleural effusion) Fig. 8 (tissuelike lung) Fig. 9 (sinusoidal)
Deep venous thrombosis	Femoral veins	Visualization of thrombus in the lumen or lack of compressibility is positive test	Consider pulmonary embolus if positive	—

Adapted from Lichtenstein DA, Mezière GA. Relevance of lung ultrasound in the diagnosis of acute respiratory failure: the BLUE protocol. Chest 2008;134(1):117–25; and *Courtesy of* Petra Duran, MD, RDMS, University of Florida College of Medicine-Jacksonville (FL).
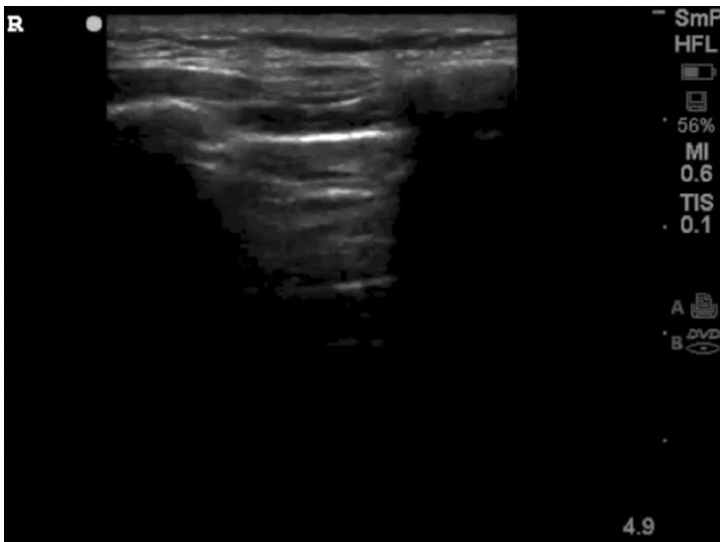
Fig. 1. A lines.

have clinically significant incidental findings. Although CT may provide vital diagnostic information, clinicians must not only consider the scan's necessity but also plan appropriate follow-up for any clinically important incidental findings.[10] Always consider whether CT is necessary or whether less risky modalities, such as chest radiograph or ultrasonography, will answer pertinent questions.
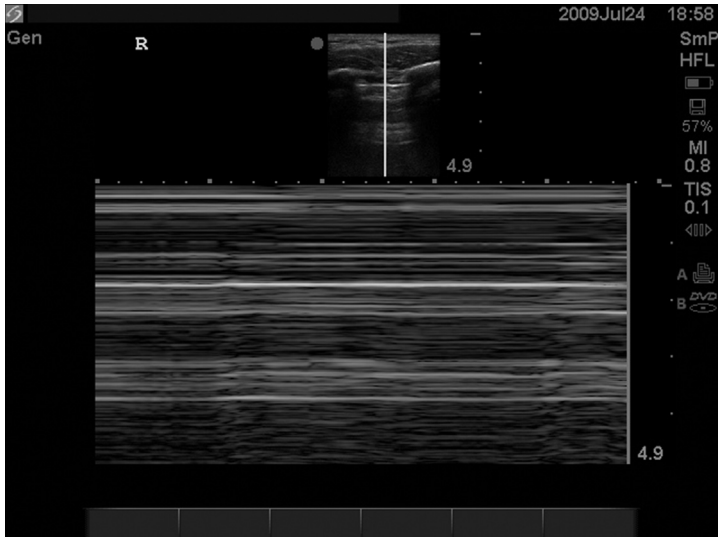

Fig. 2. B lines.

Fig. 3. Stratosphere sign.

DIFFERENTIAL DIAGNOSIS FOR ACUTELY DYSPNEIC PATIENTS
Obstructive Dyspnea

Consider the 35-year-old woman discussed earlier. Medics report tachypnea with very poor air movement during transport. As she rolls through the ambulance bay doors, you are already assessing her. Adept clinicians can spot respiratory distress from across the room. She is diaphoretic, her shoulders are held adjacent to her ears, and she is breathing extremely rapidly with minimal air movement. You decide
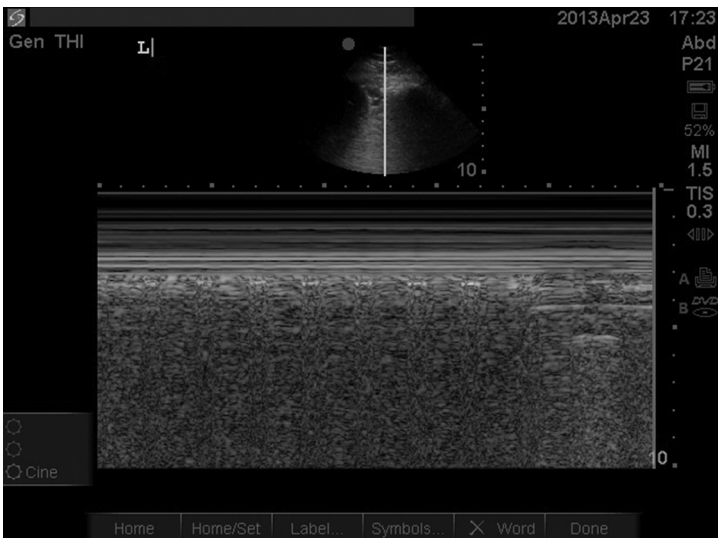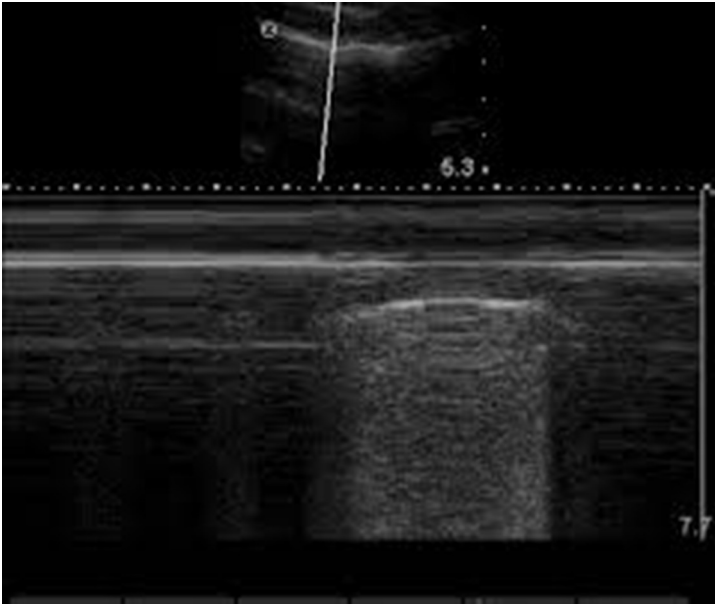

Fig. 4. Normal lung.

Fig. 5. Lung point.

to aggressively treat her for a severe asthma exacerbation, starting BIPAP ventilation with continuous nebulized albuterol and order adjunct therapies including intravenous steroids, intravenous magnesium, and intramuscular epinephrine. After 20 minutes at her bedside, she begins to breathe more comfortably with the BIPAP machine and repeat auscultation reveals diffuse wheezing and improved air movement.

Fig. 6. Pleural effusion.

Fig. 7. Pleural effusion.

As she begins to improve, EMS returns with another patient. His breath sounds are audible to everyone in the resuscitation bay. He appears diaphoretic and panicked. Examination reveals stridor, periorbital edema, tachycardia, and hypotension. Immediate intervention for anaphylactic shock begins and, after 2 rounds of epinephrine, fluid boluses, antihistamines, and steroids, he too begins to look better.

Wheezing, or musical respiratory sounds, typically result from partial airway obstruction.[11] Because this obstruction can result from inflammation, secretions, or even a foreign body, patients with noisy or whistling breathing need close evaluation to
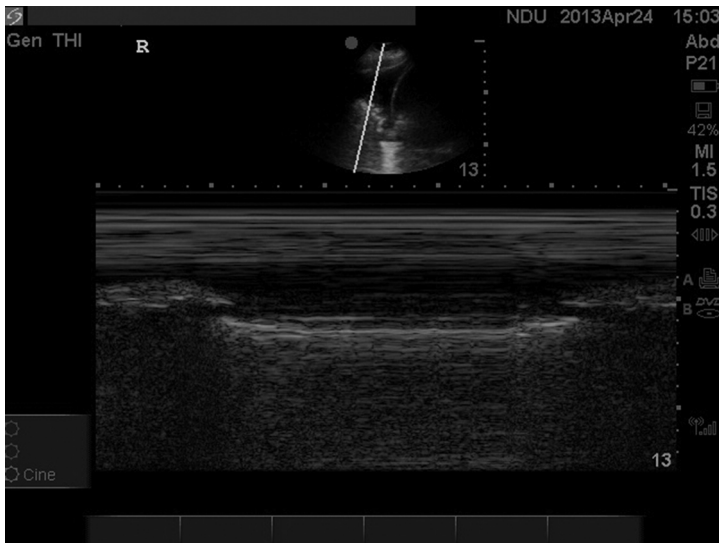

Fig. 8. Tissuelike lung.

Fig. 9. Sinusoidal sign.

determine whether the noise is inspiratory or expiratory, and whether it is from the lower airways or the upper airways. Stridor from a swollen airway, foreign body, or other airway obstruction is imminently dangerous. Although patients in anaphylaxis may benefit from the nebulized beta-agonist treatment used to treat an asthma exacerbation, it is not sufficient to save their lives. As opposed to wheezing, which is a lower airway expiratory sound, stridor is an upper airway sound transmitted when there is obstruction to the inflow of air during inspiration. The obstruction may be fixed (food bolus; **Fig. 10**) or inflammatory (anaphylaxis), but in any situation must be emergently managed.

National and world organizations define asthma "by the history of respiratory symptoms such as wheeze, shortness of breath, chest tightness and cough that vary over time and in intensity, together with variable expiratory airflow limitation."[12] The reversibility of airflow obstruction is the hallmark distinguishing asthma from other obstructive respiratory disorders. In contrast, chronic obstructive pulmonary disease (COPD)/emphysema is defined as "persistent airflow limitation that is usually progressive and associated with enhanced chronic inflammatory responses in the airways and the lungs."[12] These patients also frequently wheeze, but may have a different course of acute and chronic disease. **Table 5** highlights the differences between these similar, at times overlapping, diseases.

Asthma is an obstructive disease resulting from increased airway resistance. It is a reversible but recurrent chronic inflammatory disorder that characteristically causes severe dyspnea, wheezing, and coughing.[13] There are 2 main problems in asthma: chronically inflamed airways and hyperresponsive airways. Intermittent airflow obstruction in symptomatic patients results in decreased ability to expire, leading to hyperinflation, stenting open the alveoli, and increasing the work of breathing. Early in an exacerbation, symptoms are bronchospastic secondary to smooth muscle contraction. As an episode progresses, inflammatory changes in the airways can cause increased airway resistance and lead to VQ mismatch (**Fig. 11**). The severity of an exacerbation can be assessed clinically and should dictate how aggressively a patient is treated (**Table 6**).

Fig. 10. Food bolus in airway.

COPD is defined by the Global Initiative for Chronic Obstructive Lung Disease (GOLD) as "persistent airflow limitation that is usually progressive and associated with an enhanced chronic inflammatory response in the airways and the lung to noxious particles or gases."[12] The pathophysiology in each patient is typically a mix of lung parenchymal destruction, as seen in emphysema, and small airway inflammation with airway obstruction, or obstructive bronchiolitis.[11] An exacerbation of COPD presents as dyspnea, cough, and increased sputum production. In the emergent

Table 5
Features suggesting asthma or COPD

Favors Asthma	Favors COPD
Onset in childhood	Onset in adulthood
Symptoms vary over time	Symptoms persist even with treatment
Symptoms worse at night	Daily symptoms, some days better than others
Symptoms may be triggered by allergens or exercise	Chronic cough and sputum unrelated to triggers
Variable airflow obstruction	Persistent airflow obstruction
Normal lung function when asymptomatic	Abnormal lung function when asymptomatic
Atopy in self or family	Smoker
No progression over time	Progression over time

Adapted from Global Initiative for Asthma. Diagnosis of diseases of chronic airflow limitation: asthma, COPD and asthma-COPD overlap syndrome (ACOS). 2015. Available at: http://www. ginasthma.org/documents/14/Asthma%2C-COPD-and-Asthma-COPD-Overlap-Syndrome-%28AC OS%29. Accessed May 1, 2015.

Pathophysiology

Early - Bronchospastic   **Late - Inflammatory**

Inflammatory Mediators → Smooth muscle contraction → Bronchospasm

- Desquamate
- Mucus plug
- Edema

Inflammatory Cells

Air Trapping
- Increased resistance
- Obstruction

- Decreased compliance/ increased work of breathing
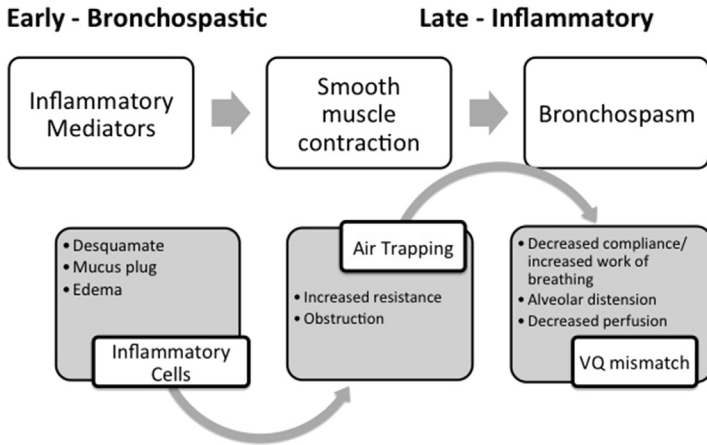- Alveolar distension
- Decreased perfusion

VQ mismatch

Fig. 11. Pathophysiology of symptom development in asthma.

setting, clinicians must treat the airflow limitation. As with asthma, monitoring of pulse oximetry, degree of respiratory distress, and hemodynamic stability can help clinicians anticipate the degree of severity of a particular exacerbation. More specific testing may also have a role, because radiographs and electrocardiograms may help differentiate other causes of shortness of breath from a COPD exacerbation. In addition, an increase in sputum production or the presence of purulent sputum should be treated with antibiotics, regardless of other infectious symptoms.[14]

Anaphylaxis is a sudden, potentially fatal, allergic reaction involving multiple organ systems.[15,16] The Second Symposium on the Definition and Management of Anaphylaxis lists the following clinical criteria for diagnosis of anaphylaxis:

1. Acute onset of an illness (minutes to several hours) with involvement of the skin, mucosal tissue, or both (eg, generalized hives; pruritus or flushing; swollen lips, tongue, uvula)
   And at least 1 of the following:
   a. Respiratory compromise (eg, dyspnea, wheeze-bronchospasm, stridor, reduced peak expiratory flow, hypoxemia)

Table 6
Severity of asthma exacerbation assessment

Symptoms	Mild	Moderate	Severe	Near Death
Breathless	While walking	While talking	At rest	Decreased effort
Speaking	In sentences	In phrases	In words	Unable
Alertness	May be agitated	Usually agitated	Usually agitated	Confused
Respiratory Rate (breaths/min)	Increased	Increased	>30	>30, imminent failure
Accessory Muscle Use	Usually not	Commonly	Usually	Usually
Wheeze	Moderate	Loud	Loud or silent	Silent
Heart Rate (beats/min)	<100	100–120	>120	±
Saturation (%)	>95	92–94	<90	<90

  b. Reduced blood pressure (BP) or associated symptoms of end-organ dysfunction (eg, hypotonia [collapse], syncope, incontinence)
2. Two or more of the following that occur rapidly after exposure to a likely allergen for that patient:
  a. Involvement of the skin-mucosal tissue
  b. Respiratory compromise
  c. Reduced BP or associated symptoms
  d. Persistent gastrointestinal symptoms (eg, crampy abdominal pain, vomiting)
3. Reduced BP after exposure to known allergen for that patient:
  a. Infants and children: low systolic BP (age specific) or greater than 30% decrease in systolic BP
  b. Adults: systolic BP of less than 90 mm Hg or greater than 30% decrease from that person's baseline

It is the respiratory compromise that is relevant to this article, and it is important to recognize that treating the allergic component of these symptoms is necessary to save the patients.

Parenchymal Dyspnea

Now EMS is at the back door with a 60-year-old patient with a history of COPD and congestive heart failure (CHF). He is in respiratory distress with audible wheezing and tripoding. He is diaphoretic, hypertensive, and has pitting edema to his knees. They have given albuterol with no improvement of his symptoms.

Acute dyspnea is the most common symptom of patients presenting with heart failure.[17] Eighty percent of patients with acutely decompensated heart failure present through the ED with a chief complaint of dyspnea.[18] This symptom is related to both pulmonary and systemic fluid overload and also low cardiac output. American College of Emergency Physicians clinical policy makes level B recommendations that standard clinical judgment can be improved with the use of a single B-type natriuretic peptide (BNP) or N-terminal pro–B-type natriuretic peptide measurement to rule in or out the diagnosis of CHF.[19] However, the true utility, may be in patients with dyspnea not expected to have a CHF exacerbation, when finding a positive BNP would change management and allow a faster initiation of treatment.[20]

Carpenter and colleagues[20] found that the classic constellation of symptoms (jugular venous distension, peripheral edema, rales, and S3) were no more predictive of patients with both pulmonary edema on chest radiograph and an increased BNP level greater than 500 pg/dL than any individual finding alone. Although rales were the most sensitive finding tested for either outcome, they had specificity of only about 50% each. Jugular venous distention and S3 gallop were the individual findings most predictive for pulmonary edema on radiograph or increased BNP level. Ultrasonography measurements of the inferior vena cava also improve diagnostic accuracy versus BNP and chest radiograph alone.[21]

The medics are back in your department, this time with a 75-year-old man with cough and fever. His family is worried that he has been eating less and is sleepier than at hospital discharge last week. In pneumonia, the diffusion of oxygen is limited by alveolar infiltrates, leading to shortness of breath. Common complaints and findings in community-acquired pneumonia include fever, cough, pleuritic chest pain, and sputum production, along with dyspnea. However, these clinical criteria may have a sensitivity as low as 50% compared with a chest radiograph.[22] On examination, many patients have crackles or evidence of consolidation. Guidelines from the Infectious Diseases Society of America and the American Thoracic Society,

recommend chest radiograph in patients with suspected pneumonia, which may show lobular consolidation, interstitial infiltrate, or cavitation.[23] Although infiltrate with suggestive symptoms makes the diagnosis, infiltrate may not be visible initially on patients with volume depletion. It is appropriate to treat empirically for 24 to 48 hours in these cases and to reimage when hydration is restored.

The management of pneumonia requires history to allow classification based on the setting in which the illness was acquired. The Infectious Disease Society of America and American Thoracic Society define the types of pneumonia as follows:[24] Hospital-acquired pneumonia (HAP) is "pneumonia that occurs 48 hours or more after admission which was not incubating at the time of admission." Ventilator-associated pneumonia (VAP) arises "more than 48-72 hours after endotracheal intubation." In addition, health care–associated pneumonia (HCAP) is diagnosed in any patient who is "hospitalized in an acute care hospital for two or more days within 90 days of the infection, resided in a nursing home or long-term care facility, received recent IV antibiotic therapy, chemotherapy or wound care within the past 30 days of the current infection or attended a hospital or hemodialysis clinic." Community-acquired pneumonia is not acquired in any of these situations. These classifications identify typical pathogens and guide appropriate initial management. Important historical exposures and risk factors to refine treatment are summarized in **Table 7**.[23]

The American Thoracic Society along with the Infectious Disease Society of America's consensus statement offers 4 important principles in the initial management and evaluation of adult patients with bacterial HAP, VAP, or HCAP; the most important to be accomplished in the ED is to promptly treat with "appropriate and adequate therapy" to decrease mortality.[24]

Circulatory Dyspnea

After a brief delay, you see a 28-year-old woman with shortness of breath and chest pain. She smokes, uses hormonal birth control, and reports that her symptoms started when she came back from a business trip. Pulmonary embolism (PE) interferes with both ventilation and perfusion. It ultimately causes circulatory collapse because of obstruction of right ventricular outflow eventually causing increased pulmonary artery pressure and failure of the right then left ventricles. Before circulatory collapse, echocardiography can show signs of right ventricular (RV) strain, including dilatation of the right ventricle, RV hypokinesis, paradoxic septal wall motion, McConnell sign (hypokinesis of the free RV wall with sparing of the apex), and tricuspid regurgitation.[25]

Dresden and colleagues[26] supported the use of ultrasonography in moderate-risk to high-risk patients to determine whether the patients were appropriate for anticoagulation while awaiting definitive imaging. Early anticoagulation is recommended to improve mortality and there is evidence to support anticoagulation before diagnosis in patients with a Wells score greater than 4 who will have a delay to diagnosis of more than 1 hour and 40 minutes.[27,28]

The assessment of patients with dyspnea and concern for PE requires a series of risk stratification. One common method is to use Wells criteria[29] (**Box 1**) in patients with suspicion for PE; although other stratification tools exist, none has been shown to be clearly superior. When there is low clinical suspicion for PE, PERC (pulmonary embolism rule-out criteria)[30] rules or D-dimer testing may be applied.[27] If PERC (**Box 2**) is negative, or there is intermediate pretest probability for PE with negative high-sensitivity D-dimer, no further testing for PE is required.[27] When further testing is needed (positive D-dimer or high-sensitivity D-dimer not available), negative CT angiogram or low-probability VQ scan may be used to rule out PE.

Table 7 Common pathogens in community-acquired pneumonia	
Condition	**Commonly Encountered Pathogens**
Alcoholism	*Streptococcus pneumoniae*, oral anaerobes, *Klebsiella pneumoniae*, *Acinetobacter* species, *Mycobacterium tuberculosis*
COPD and/or smoking	*Haemophilus influenzae*, *Pseudomonas aeruginosa*, *Legionella* species, *S pneumonia*, *Moraxella catarrhalis*, *Chlamydophila pneumoniae*
Aspiration	Gram-negative enteric pathogens, oral anaerobes
Lung abscess	CA-MRSA, oral anaerobes, endemic fungal pneumonia, *M tuberculosis*, atypical mycobacteria
Exposure to bat or bird droppings	*Histoplasma capsulatum*
Exposure to birds	*Chlamydophila psittaci* (if poultry: avian influenza)
Exposure to rabbits	*Francisella tularensis*
Exposure to farm animals or parturient cats	*Coxiella burnetii* (Q fever)
HIV infection (early)	*S pneumoniae, H influenzae, M tuberculosis*
HIV infection (late)	The pathogens listed for early infection plus *Pneumocystis jirovecii*, *Cryptococcus*, *Histoplasma*, *Aspergillus*, atypical mycobacteria (especially *Mycobacterium kansasii*), *P aeruginosa*, *H influenzae*
Hotel or cruise ship stay in previous 2 wk	*Legionella* species
Travel to or residence in southwestern United States	*Coccidioides* species, Hantavirus
Travel to or residence in southeast and east Asia	*Burkholderia pseudomallei*, avian influenza, SARS
Influenza active in community	Influenza, *S pneumoniae*, *Staphylococcus aureus*, *H influenzae*
Cough>2 wk with whoop or post-tussive vomiting	*Bordetella pertussis*
Structural lung disease (eg, bronchiectasis)	*P aeruginosa*, *Burkholderia cepacia*, *S aureus*
Injection drug use	*S aureus,* anaerobes, *M tuberculosis*, *S pneumoniae*
Endobronchial obstruction	Anaerobes, *S pneumonia*, *H influenzae*, *S aureus*
In context of bioterrorism	*Bacillus anthracis* (anthrax), *Yersinia pestis* (plague), *Francisella tularensis* (tularemia)

Abbreviations: CA-MRSA, community-acquired methicillin-resistant *Staphylococcus aureus*; HIV, human immunodeficiency virus; SARS, severe acute respiratory syndrome.

From Mandell LA, Wunderink RG, Anzueto A, et al. Infectious Diseases Society of America/ American Thoracic Society consensus guidelines on the management of community-acquired pneumonia in adults. Clin Infect Dis 2007;44(Suppl 2):S46; with permission.

In the next bed is a middle-aged woman with diabetes complaining of shortness of breath today. It was associated with some vague nausea and she says that she just does not feel good. Angina pectoris is cardiac chest pain in which oxygen demand outweighs myocardial oxygen supply; in this case caused by occlusion of coronary arteries. Although typically chest pain is a part of the presentation, dyspnea alone may be the initial complaint, termed an anginal equivalent. In one recent large series of patients undergoing stress testing, patients with dyspnea alone were at increased

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Box 1

Wells criteria for pulmonary embolism

- Clinical signs and symptoms of deep vein thrombosis (DVT): +3
- PE is the main diagnosis or equally likely: +3
- Heart Rate greater than 100 beats/min: +1.5
- Immobilization >3 days or surgery in last 4 weeks: +1.5
- History of prior PE/deep venous thrombosis (DVT): +1.5
- Hemoptysis: +1
- Malignancy with treatment within 6 months or palliative: +1

Less than or equal to 1.5 = low risk, 1.3% chance of PE in ED population; 2 to 6 = moderate risk, 16.2% chance of PE in ED population; greater than 6 = high risk, 40.6% chance of PE in ED population

Adapted from Wells PS, Anderson DR, Rodger M, et al. Excluding pulmonary embolism at the bedside without diagnostic imaging: Management of patients with suspected pulmonary embolism presenting to the emergency department by using a simple clinical model and D-dimer. Ann Intern Med 2001;135:99.

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risk of death from cardiac causes. Patients asked simply whether they experienced shortness of breath were considered dyspneic. The subset with no prior known coronary artery disease had more than 4 times the risk of sudden cardiac death versus asymptomatic patients and more than twice the risk of those with typical angina.[31] Clinicians should consider past medical history and risk factors when assessing dyspnea for cardiac causes such as acute myocardial infarction and acute coronary syndrome. Appropriate testing includes bedside electrocardiogram, troponin, and chest radiograph.

The department eventually settles down and you are able to do some charting until a young man comes in with visible respiratory distress. He is tall and thin, smokes regularly, and reports sudden onset of severely painful breathing. Pneumothorax occurs when air enters the plural space between the chest wall and the lung. Typically only a thin serous layer exists between the visceral and parietal pleura. Air enters this potential space only when there is damage to the lung or chest wall, or a gas-producing pleural space infection. The classic risk factors for bleb rupture causing spontaneous

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Box 2

Pulmonary embolism rule-out criteria

- Further work-up recommended if any of the following are present:
  - Age greater than or equal to 50 years
  - Pulse greater than or equal to 100 beats/min
  - Oxygen saturation less than 95%
  - Hemoptysis
  - Unilateral leg swelling
  - History of PE/DVT
  - Exogenous estrogen use
  - Surgery or trauma within 4 weeks that required hospitalization or intubation

- If none are present, probability of PE is less than 2%.

Adapted from Kline JA, Courtney DM, Babrhel C, et al. Prospective multicenter evaluation of the pulmonary embolism rule-out criteria. J Thromb Haemost 2008;6(5):773.

pneumothorax are tall men, although smoking has been suggested to increase the risk of rupture by damaging the pleural layer.

Pneumothoraces may be identified by ultrasonography, chest radiograph, or CT. Treatment may be guided by cause, severity, comorbidities, interventions such as positive pressure ventilation, size of the pneumothorax, and patient's preference. Recent studies suggest that uncomplicated spontaneous pneumothorax in patients not undergoing positive pressure ventilation may be treated as successfully with needle aspiration as with other more invasive chest drains, regardless of size.[32]

Tension pneumothorax is a serious event requiring immediate needle decompression to avert loss of cardiac output and arrest. However, recent review shows that the classic presentation of tension pneumothorax with hypotension, absent breath sounds, and deviated trachea may not be immediately seen in patients with spontaneous, unassisted respiration.[33] Because of the slower development of the accumulation of air and pressure variations, spontaneously breathing patients may compensate much longer and present atypically, as shown in **Table 8**. Thus, clinicians must remain vigilant.

Compensatory Dyspnea

This article focuses on the cardiopulmonary system as the source of the problem in acutely dyspneic patients. It is important to also consider that the appearance of shortness of breath, tachypnea, or other typical symptoms of dyspnea may result from changing metabolic demands. These patients may appear, on the surface, to be in respiratory distress; they may be tachypneic, tachycardic, even pale or diaphoretic. In these cases, the clinician's responsibility is to identify and fix the true problem in order to improve the respiratory symptoms.

Severely anemic patients have limited oxygen carrying capacity. Their bodies therefore experience oxygen hunger, which can manifest as shortness of breath. Patients with dysfunctional hemoglobins secondary to irreversibly bound atoms or toxins may also be functionally anemic with the same symptoms.

People's bodies attempt at all costs to maintain equilibrium. Therefore, in metabolic acidoses (such as diabetic ketoacidosis), chemoreceptors detect acidosis and

Table 8	
Findings in tension pneumothorax	
Unassisted Ventilation	**Positive Pressure Ventilation**
Spontaneous respiration with air passing through 1-way flap	Assisted ventilation forces air through pleural defect into pleural space
Compensatory mechanisms delay collapse: • Tachycardia and accessory muscle use caused by tachypnea, increased tidal volume, and negative movement of the opposite side of the chest • BP is maintained because of limits in the pressure of the pneumothorax on mediastinum and hemithorax	Sudden hemodynamic and respiratory compromise: • Sedation may increase inspiratory pressure • Intrapleural pressure is increased throughout respiratory cycle
Venous siphon resulting from negative intrathoracic pressure in the opposite side of the chest returns blood to the heart	Decreased venous return leads to hypotension and cardiac arrest

Adapted from Roberts DJ, Leigh-smith S, Faris PD, et al. Clinical presentation of patients with tension pneumothorax: a systematic review. Ann Surg 2015;261(6):1069.

stimulate the respiratory center to hyperventilate. Both the rate and the depth of ventilation often increase, leading to both tachypnea and hyperpnea, at times referred to as Kussmaul respirations. This compensatory response is crucial for survival and should not be mistaken for dyspnea. It is equally important to realize that an increase in alveolar ventilation is not always a compensatory response (to acidosis or to primary pulmonary disorders) and hypocapnia may cause primary respiratory alkalosis, from central nervous system compromise, toxins (eg, salicylates), anxiety, or pain.[34] In these patients, imaging rarely reveals a source of dyspnea, but clinical suspicion based on history and examination, including signs such as the fruity breath of ketonemia, the pallor of anemia, or the cyanosis of toxic hemoglobinopathies, directs providers toward appropriate laboratory testing and treatment.

Diagnosis of Exclusion

In addition, sometimes dyspnea is not dyspnea. Acute anxiety and panic disorder can present as shortness of breath, tachypnea, or hyperventilation. Patients with panic disorder often describe symptoms similar to those of patients with true airway obstruction despite their normal pulmonary function. It has been suggested that these patients have abnormal proprioception, experiencing dyspnea without abnormal stimuli.[35] However, patients with a history of pulmonary disease can also have pure panic episodes. Arterial blood gas may be useful in diagnosing anxiety-related hyperventilation.[36]

Severe pain can also induce abnormal respiratory patterns. Like compensatory problems, pain and anxiety can be managed by managing their causes. Treat pain. Reduce stress and anxiety with words, behaviors, or, if necessary, medications. However, air hunger and difficulty breathing also make individuals anxious. Be sure to avoid premature diagnosis of a purely anxiety-based concern without first evaluating for more dangerous disorders.

SUMMARY

Acute dyspnea presents commonly to the ED and it is imperative that emergency physicians be prepared to stabilize patients' oxygenation and ventilation, which requires careful and efficient consideration of the differential diagnosis. Using cues from the history and physical examination, practitioners may guide the work-up and treatment to identify a parenchymal, obstructive, circulatory, or compensatory cause of dyspnea. Early use of bedside testing, including ultrasonography, may limit unnecessary tests and save time in determining the best treatment course. Thus ensuring both the best care for the patient and also the physician's ability to readily respond to the next case.

REFERENCES

1. Prekker ME, Feemster LC, Hough CL, et al. The epidemiology and outcome of prehospital respiratory distress. Acad Emerg Med 2014;21(5):543–50.
2. American Thoracic Society. Dyspnea. Mechanisms, assessment, and management: a consensus statement. Am J Respir Crit Care Med 1999;159:321–40.
3. Schwartzstein RM, Lewis A. Chapter 29: Dyspnea. In: Broaddus V, Mason RC, Ernst JD, et al, editors. Murray & Nadel's textbook of respiratory medicine. 6th edition. Philadelphia: Elsevier Health Sciences, Saunders/Elsevier; 2015. p. 490–1.
4. Kajimoto K, Madeen K, Nakayama T, et al. Rapid evaluation by lung-cardiac inferior vena cava (LCI) integrated ultrasound for differentiating heart failure from

pulmonary disease as the cause of acute dyspnea in the emergency setting. Cardiovasc Ultrasound 2012;10:49.

5. Anderson KL, Jenq KY, Fields JM, et al. Diagnosing heart failure among acutely dyspneic patients with cardiac, inferior vena cava and lung ultrasonography. Am J Emerg Med 2013;31:1208–14.

6. Russell FM, Ehrman RR, Cosby K, et al. Diagnosing acute heart failure in patients with undifferentiated dyspnea: a lung and cardiac ultrasound (LuCUS) protocol. Acad Emerg Med 2015;22(2):182–91.

7. Feng LB, Pines JM, Yusuf HR, et al. U.S. trends in computed tomography use and diagnoses in emergency department visits by patients with symptoms suggestive of pulmonary embolism, 2001-2009. Acad Emerg Med 2013;20(10):1033–40.

8. Huckins DS, Price LL, Gilley K. Utilization and yield of chest computed tomographic angiography associated with low positive D-dimer levels. J Emerg Med 2012;43:211–20.

9. Qaseem A, Alguire P, Dall P, et al. Appropriate use of screening and diagnostic tests to foster, high-value, cost-conscious care. Ann Intern Med 2012;156:147–9.

10. Coco AS, O'gurek DT. Increased emergency department computed tomography use for common chest symptoms without clear patient benefits. J Am Board Fam Med 2012;25(1):33–41.

11. Chapter 8, The thorax and lungs. In: Bickley LS, Szilagyi PG, editors. Bates' guide to physical examination and history taking. 11th edition. Philadelphia: Lippincott Williams & Wilkins; 2013. p. 301.

12. Global Initiative for Asthma. Diagnosis of diseases of chronic airflow limitation: asthma, COPD and asthma-COPD overlap syndrome (ACOS). 2015. Available at: http://www.ginasthma.org/documents/14/Asthma%2C-COPD-and-Asthma-COPD-Overlap-Syndrome-%28ACOS%29. Accessed May 1, 2015.

13. Husain AN. Chapter 15, The lung. In: Kumar V, Abbas AK, Aster JC, editors. Robbins and Cotran pathologic basis of disease. 9th edition. Philadelphia: WB Saunders; 2014. p. 679.

14. Ram FS, Rodriguez-Roison R, Granados-Navarrete A, et al. Antibiotics for exacerbations of chronic obstructive pulmonary disease. Cochrane Database Syst Rev 2006;(2):CD004403.

15. Sampson HA, Munoz-Furlong A, Campbell RL, et al. Second Symposium on the Definition and Management of Anaphylaxis: summary Report – Second National Institute of Allergy and Immunology. Ann Emerg Med 2006;47(4):373–80.

16. Simons FES, Ardusso LRF, Dimov V, et al. World Allergy Organization anaphylaxis guidelines: 2013 update of the evidence base. Int Arch Allergy Immunol 2013; 162:193–204.

17. Pang PS, Collins SP, Sauser K, et al. Assessment of dyspnea early in acute heart failure: patient characteristics and response differences between Likert and visual analog scales. Acad Emerg Med 2014;21(6):659–66.

18. Fonarow GC. The Acute Decompensated Heart Failure National Registry (ADHERE): opportunities to improve care of patients hospitalized with acute decompensated heart failure. Rev Cardiovasc Med 2003;4(Suppl 7):S21–30.

19. Silvers SM, Howell JM, Kosowsky JM, et al. Clinical policy: critical issues in the evaluation and management of adult patients presenting to the emergency department with acute heart failure syndromes. Ann Emerg Med 2007;49(5): 627–69.

20. Carpenter CR, Keim SM, Worster A, et al. Brain natriuretic peptide in the evaluation of emergency department dyspnea: is there a role? J Emerg Med 2012;42(2): 197–205.

21. Miller JB, Sen A, Strote SR, et al. Inferior vena cava assessment in the bedside diagnosis of acute heart failure. Am J Emerg Med 2012;30(5):778–83.
22. Metlay JP, Fine MJ. Testing strategies in the initial management of patients with community-acquired pneumonia. Ann Intern Med 2003;138:109.
23. Mandell LA, Wunderink RG, Anzueto A, et al. Infectious Diseases Society of America/American Thoracic Society consensus guidelines on the management of community-acquired pneumonia in adults. Clin Infect Dis 2007;44(Suppl 2): S27–72.
24. American Thoracic Society, Infectious Diseases Society of America. Guidelines for the management of adults with hospital-acquired, ventilator-associated, and healthcare-associated pneumonia. Am J Respir Crit Care Med 2005;171(4): 388–416.
25. Kucher N, Rossi E, DeRosa M, et al. Prognostic role of echocardiography among patients with acute pulmonary embolism and a systolic arterial pressure of 90 mm Hg or higher. Arch Intern Med 2005;165:1777–81.
26. Dresden S, Mitchell P, Rahimi L, et al. Right ventricular dilatation on bedside echocardiography performed by emergency physicians aids in the diagnosis of pulmonary embolism. Ann Emerg Med 2014;63(1):16–24.
27. Fesmire FM, Brown MD, Espinosa JA, et al. Critical issues in the evaluation and management of adult patients presenting to the emergency department with suspected pulmonary embolism. Ann Emerg Med 2011;57(6):628–52.e75.
28. Blondon M, Righini M, Aujesky D, et al. Usefulness of preemptive anticoagulation in patients with suspected pulmonary embolism: a decision analysis. Chest 2012; 142:697–703.
29. Wells PS, Anderson DR, Rodger M, et al. Excluding pulmonary embolism at the bedside without diagnostic imaging: management of patients with suspected pulmonary embolism presenting to the emergency department by using a simple clinical model and D-dimer. Ann Intern Med 2001;135:98–107.
30. Kline JA, Courtney DM, Babrhel C, et al. Prospective multicenter evaluation of the pulmonary embolism rule-out criteria. J Thromb Haemost 2008;6(5):772–80.
31. Abidov A, Rozanski A, Hachamovitch R, et al. Prognostic significance of dyspnea in patients referred for cardiac stress testing. N Engl J Med 2005;353(18): 1889–98.
32. Zehtabchi S, Rios CL. Management of emergency department patients with primary spontaneous pneumothorax: needle aspiration or tube thoracostomy? Ann Emerg Med 2008;51(1):91–100, 100.e1.
33. Roberts DJ, Leigh-smith S, Faris PD, et al. Clinical presentation of patients with tension pneumothorax: a systematic review. Ann Surg 2015;261:1068–78.
34. Morris CG, Low J. Metabolic acidosis in the critically ill: part 1. Classification and pathophysiology. Anaesthesia 2008;63(3):294–301.
35. Smoller JW, Pollack MH, Otto MW, et al. Panic anxiety, dyspnea, and respiratory disease. Am J Respir Crit Care Med 1996;154:6–17.
36. Burri E, Potocki M, Drexler B, et al. Value of arterial blood gas analysis in patients with acute dyspnea: an observational study. Crit Care 2011;15(3):R145.
37. Syrjala H, Broas M, Suramo I, et al. High resolution computed tomography for the diagnosis of community-acquired pneumonia. Clin Infect Dis 1998;27:358–63.