



## Chapter 22 – Dyspnea

### EPISODE CONTENT BASED ON ROSEN'S EMERGENCY MEDICINE (9TH ED.)

*Italicized text is quoted directly from Rosen's.*

#### Key Concepts:

- 1. Dyspnea results from a variety of conditions, ranging from nonurgent to life-threatening. Neither the clinical severity nor the patient's perception correlates well with the seriousness of underlying pathology.*
- 2. Dyspnea is subjective and the differential diagnosis can be divided into acute and chronic cases, of which many are pulmonary. Other causes include cardiac, metabolic, infectious, neuromuscular, traumatic, and hematologic conditions.*
- 3. Chronic or progressive dyspnea usually denotes primary cardiac or pulmonary disease. Acute dyspneic spells may result from asthma exacerbation;infection;pulmonary embolism; intermittent cardiac dysfunction; or inhalation of irritants, allergens, or foreign bodies.*
- 4. All patients experiencing dyspnea, regardless of possible cause, should be promptly evaluated in the treatment area. Bedside pulse oximetry readings should be obtained, and the patient placed on a cardiac monitor. If the pulse oximetry result is less than 95% on room air, the patient should be placed on supplemental oxygen either by nasal cannula or mask, depending on the degree of desaturation.*
- 5. If necessary, breathing should be assisted with manual or mechanical ventilation, either non-invasively for the short term, or with the patient tracheally intubated for airway protection for prolonged ventilation.*
- 6. Unstable patients or patients with critical diagnoses must be stabilized and require admission to an intensive care unit. Emergent patients who have improved in the ED may be admitted to an intermediate care unit. Most patients in the non-urgent category can be treated as outpatients if medical follow-up can be arranged.*

#### Core Questions

1. Define the following terms:
  - a. Dyspnea
  - b. Tachypnea
  - c. Hyperpnea
  - d. Hyperventilation
  - e. Dyspnea on exertion



- f. Orthopnea
- g. Paroxysmal Nocturnal Dyspnea
2. What anatomical structures are responsible for controlling respiratory effort?
3. Outline an approach to the history for the dyspneic patient.
4. Detail the physical examination for the dyspneic patient and highlight pivotal exam findings that point to specific pathologies.
5. Outline the differential diagnosis for the patient presenting with dyspnea and highlight 5 critical, 5 emergent, and 5 non-emergent causes of shortness of breath.
6. What ancillary tests are indicated for the dyspneic patient?
7. Detail the utility of point-of-care ultrasound in the assessment of the dyspneic patient.
8. Outline a management algorithm for the acutely dyspneic patient.

## Wisecracks

1. List three findings on chest radiograph suggestive of pulmonary embolism.
2. What is the utility of venous blood gas testing and how do its values correlate with that of an arterial blood gas?

## Rosen's in Perspective

In 2016, ~3.4 million US ED visits (2.4%) were attributed to a primary complaint of dyspnea. The differential diagnosis of dyspnea is broad, and the underlying pathology ranges from benign or slowly progressive conditions to rapidly progressive ones with high rates of morbidity or mortality. Thus, it is important that ED practitioners have a solid, systematic approach to this chief complaint.

Etiologies of dyspnea vary by patient age. The most common etiologies in adult patient's are:

- Decompensated heart failure
- COPD exacerbation
- Asthma exacerbation
- Pulmonary embolism

Anchoring bias plays a large role in the misdiagnosis of dyspnea, so don't be afraid to reexamine the differential diagnosis of a patient who's last note reads "anxiety" as the underlying cause of their complaint.

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## Core Questions:



**[1] Define the following terms:**

<b>Term</b>	<b>Definition</b>
<b>Dyspnea</b>	The sensation of breathlessness and the patient's reaction to that sensation
<b>Tachypnea</b>	A respiratory rate greater than normal. Normal adult ranges from 44 cycles per minute in a newborn to 14-18 cycles per minute in adults
<b>Hyperpnea</b>	Greater than normal minute ventilation to meet metabolic requirements
<b>Hyperventilation</b>	A minute ventilations (determined by respiratory rate and tidal volume) that exceeds metabolic demand. Arterial blood gases characteristically show a normal partial pressure of oxygen with an uncompensated respiratory alkalosis.
<b>Dyspnea on Exertion</b>	Dyspnea provoked by physical effort or exertion. It often is quantified in simple terms, such as the number of stairs or number of blocks a patient can manage before the onset of dyspnea.
<b>Orthopnea</b>	Dyspnea in a recumbent position. It usually is measured in the number of pillows the patient uses to lie in bed (eg, two-pillow orthopnea).
<b>Paroxysmal Nocturnal Dyspnea</b>	Sudden onset of dyspnea occurring while reclining at night, usually related to the presence of congestive heart failure.

*All definitions are taken from Rosen's Emergency Medicine 9th Edition. Please refer to the text for further details and/or clarification.*

**[2] What anatomical structures are responsible for controlling respiratory effort?**

The exact mechanisms that produce the sensation of breathlessness are not precisely known. However, we do know the structures responsible for controlling respiratory effort. These are detailed below.

**Centrally:**

- Respiratory centre located in the medulla oblongata and midbrain
- Most sensitive to alterations in the partial pressure of carbon dioxide in the brain



### Peripherally:

- **Thoracic Neural Receptors**
  - Slowly reacting receptors in the upper airway, trachea, lungs, chest wall, and pulmonary vessels
  - Rapidly responding irritant receptors that respond to changes in lung volumes and the presence of substances such as histamine, prostaglandins, and noxious chemicals in the environment
- **Chemoreceptors located in the carotid bodies**
  - Primarily respond to changes in the partial pressure of oxygen
- **Chemoreceptors in the aortic bodies**
  - Primarily active during infancy and childhood, but largely become inactive during adulthood
- **Mechanoreceptors in the diaphragm and skeletal muscles**

### [3] Outline an approach to the history for the dyspneic patient.

#### Features of Dyspnea:

1. **Duration**
  - a. Chronic or progressive dyspnea usually denotes primary cardiac or respiratory disease
  - b. Acute episodes of breathlessness may result from asthma exacerbation, infection, intermittent cardiac dysfunction, psychogenic causes, or inhalation of allergens, foreign bodies or irritants
2. **Onset of Dyspnea**
  - a. Sudden onset should lead to consideration of PE or PTX
  - b. Slowly building dyspnea may be the result of an acute exacerbation, COPD, pneumonia, small PE, CHF, or malignancy
3. **Provoking Factors of Dyspnea**
  - a. Position changes should be interrogated (think orthopnea, COPD, or neuromuscular disease)
  - b. Interrogate potential PND by asking “do you wake up in the night acutely SOB and need to sit up for a number of minutes to help it resolve?”
    - i. This occurs in both CHF and COPD
  - c. Exertional dyspnea should be questioned, and if present, can hint toward the presence of COPD, abdominal loading, or poor cardiac reserve
  - d. Feelings of anxiety may point toward psychogenic dyspnea but may also point toward an organic cause of a patient’s dyspnea

#### Associated Symptoms:



1. Interrogate for the presence of chest pain, cough, hemoptysis, palpitations, syncope or presyncope
2. Ask about the presence of anginal equivalents
3. Inquire as to the presence of symptoms of systemic infection
4. Seek clarification as to the presence or absence of nausea, vomiting, abdominal pain, changes in stooling, melena, and hematochezia
5. Ask about symptoms of venous thromboembolism
6. Inquire as to the presence of neurologic symptoms, looking for weakness, sensory disturbances, and other symptoms of focal neurological deficit

**[4] Detail the physical examination for the dyspneic patient and highlight pivotal exam findings that point to specific pathologies.**

*The following tables have been adapted from Table 22.2 - Pivotal Findings in Physical Examination in Rosen’s Emergency Medicine, 9th Edition. Please see text for further clarification.*

Vital Signs	
PEx	Dx
Tachypnea	PNA, PTX
Hypopnea	Intracranial insult, drug or toxic ingestion
Tachycardia	PE, traumatic chest injury
Hypotension	Tension PTX
Fever	PNA, PE

General Appearance	
PEx	Dx
Cachexia, weight loss	Malignancy, acquired immune disorder, mycobacterial infection
Obesity	Hypoventilation, sleep apnea, PE
Pregnancy	PE
Barrel Chest	COPD
“Sniffing” Position	Epiglottitis
Tripoding	COPD or asthma with severe distress
Traumatic injury	PTX, rib fractures, diaphragmatic injury, flail chest, hemothorax, pulmonary contusion



<b>Skin and Nails</b>	
<b>PEx</b>	<b>Dx</b>
Tobacco stains or odor	COPD, malignancy, infection
Clubbing	Chronic hypoxia, intracardiac shunts, or pulmonary vascular abnormalities
Pallid skin or conjunctivae	Anemia
Muscle wasting	Neuromuscular disease
Bruising	Chest wall: rib fractures, PTX
Diffuse changes:	Thrombocytopenia, chronic steroid use, anticoagulation
SQ emphysema	Rib fractures, PTX, tracheobronchial disruption
Hives, rash	Allergic reaction, infection, tick-bourne illness

<b>Neck</b>	
<b>PEx</b>	<b>Dx</b>
Stridor	Upper airway edema or infection, foreign body, traumatic injury, anaphylaxis
JVD	Tension PTX, COPD or asthma exacerbation, fluid overload, CHF, cardiac tamponade

<b>Chest Examination</b>	
<b>PEx</b>	<b>Dx</b>
Crepitance or pain on palpation	Rib or sternal fractures
SQ emphysema	PTX, tracheobronchial rupture
Thoracoabdominal dyssynchrony	Diaphragmatic injury with herniation, cervical spinal cord trauma
Flail segment	Flail chest, pulmonary contusion



<b>Lung Examination</b>	
<b>PEx</b>	<b>Dx</b>
Wheezes	CHF, anaphylaxis, bronchospasm
Rales	CHF, PNA, PE
Unilateral decrease	PTX, pleural effusion, consolidation, rib fractures or contusion, pulmonary contusion
Hemoptysis	Malignancy, infection, bleeding disorder, CHF, PE
Sputum production	Infection (viral, bacterial)
Friction rub	Pleurisy
Abnormal respiratory pattern (Cheyne-Stokes)	Intracranial insult

<b>Cardiac Examination</b>	
<b>PEx</b>	<b>Dx</b>
Murmur	PE
S3 or S4 gallop	PE
S2 accentuation	PE
Muffled heart sounds	Cardiac tamponade, pericardial effusion

<b>Extremities</b>	
<b>PEx</b>	<b>Dx</b>
Calf tenderness, Homan's Sign	PE
Edema	CHF

<b>Neurologic Examination</b>	
<b>PEx</b>	<b>Dx</b>
Focal deficits (motor, sensory, cognitive)	Stroke, intracranial hemorrhage causing central abnormal respiratory drive; if long-standing, risk of aspiration pneumonia
Symmetrical deficits	Neuromuscular disease
Diffuse weakness	Metabolic or electrolyte abnormality (hypocalcemia, hypomagnesemia, hypophosphatemia), anemia
Hyporeflexia	Hypermagnesemia
Ascending weakness	GBS



**[5] Outline the differential diagnosis for the patient presenting with dyspnea and highlight 5 critical, 5 emergent, and 5 non-emergent causes of shortness of breath.**

*The following table has been adapted from Table 22.1 - Differential Diagnoses for Acute Dyspnea in Rosen's Emergency Medicine, 9th Edition. Please see text for further clarification*

Organ	Critical	Emergent	Non-Emergent
<b>Pulm</b>	Airway obstruction; Pulmonary Embolism; Noncardiogenic Edema; Anaphylaxis; Ventilatory Failure	Spontaneous PTX; Asthma; Cor pulmonale; Aspiration; PNA (CAP>70)	Pleural Effusion; Neoplasm; PNA (CAP <70); COPD
<b>Cardiac</b>	Pulmonary Edema; MI; Cardiac Tamponade	Pericarditis	Congenital Heart Disease; Valvular Heart Disease; Cardiomyopathy
<b>Primarily Associated with Normal or Increased Respiratory Effort</b>			
<b>GI</b>		Hypotension from: sepsis from ruptured viscus, bowel obstruction, inflammatory or infectious process	Pregnancy; Ascites; Obesity
<b>Psych</b>			Hyperventilation syndrome; Somatization disorder; Panic Attack
<b>Metabolic or Endocrine</b>	Toxic ingestion; DKA	Renal failure; Electrolyte abnormalities; Metabolic acidosis	Fever; Thyroid disease
<b>Infx</b>	Epiglottitis	PNA (CAP >70)	PNA (CAP <70)
<b>Trauma</b>	Tension PTX; Cardiac tamponade; Flail chest	Simple PTX or hemothorax; Diaphragmatic rupture; Neurologic injury; Anemia	Rib fracture
<b>Heme</b>	CO or HCN poisoning; Acute chest syndrome		
<b>Primarily Associated with Decreased Respiratory Effort</b>			
<b>Neuromsk</b>	CVA Intracranial insult; Organophosphate poisoning	MS GBS; Tick paralysis	ALS Polymyositis; Porphyria





## [6] What ancillary tests are indicated for the dyspneic patient?

Rosen's offers an extensive battery of tests to consider for a dyspneic patient. Below, we present a distilled list of tests that are ordered or considered for most dyspneic patients. Read through Table 22.4 of Rosen's for a more extensive list of potential evaluations.

Typically Order/Perform	Consider Based on Clinical Context
<ol style="list-style-type: none"> <li>1. EKG</li> <li>2. CXR (PA and Lateral)</li> <li>3. CBC</li> <li>4. Electrolytes</li> <li>5. Extended Electrolytes</li> <li>6. Renal function</li> <li>7. VBG with Lactate</li> <li>8. Coags</li> <li>9. POCUS - cardiac and lung ultrasounds</li> </ol>	<ol style="list-style-type: none"> <li>1. Cardiac Biomarkers</li> <li>2. BNP</li> <li>3. D-dimer</li> <li>4. ABG</li> <li>5. Liver Enzymes and Function Testing</li> <li>6. CT Chest/Pulmonary Angiogram</li> </ol>

*The following tables have been adapted from Table 22.4 - Ancillary Testing in the Dyspneic Patient in Rosen's Emergency Medicine, 9th Edition. Please see text for further clarification*

Ancillary Laboratory Tests	
Test	Findings and Potential Diagnoses
<b>Pulse Ox, selective ABG</b>	Hypoxia, hypoventilation (muscular weakness, intracranial event)
<b>Waveform Capnography</b>	CO <sub>2</sub> retention (COPD, sleep apnea), obstructive or restrictive pulmonary pattern; Metabolic versus respiratory acidosis (DKA, ingestions); A-a gradient (PE); Elevated carboxyhemoglobin (inhalation injury or CO poisoning)
<b>CBC</b>	<b>WBC Increase:</b> infection, stress demargination, hematologic malignancy; <b>WBC Decrease:</b> neutropenia, sepsis; <b>Hgb, Hct:</b> anemia, polycythemia; <b>Smear:</b> abnormal HgB (i.e., sickling), inclusions; <b>Platelets:</b> thrombocytopenia (marrow toxicity)
<b>Chemistries</b>	<b>BUN, Cr:</b> acute or chronic renal failure; <b>K, Mg, PO<sub>4</sub>:</b> low levels resulting in muscular weakness; <b>Glucose:</b> DKA
<b>D-dimer</b>	Abnormal clotting activity
<b>BNP</b>	heart failure, PE
<b>Troponins</b>	cardiac ischemia or infarct



Ancillary Cardiac Tests	
Test	Findings and Potential Diagnoses
EKG	Ischemia, dysrhythmias, S1Q3T3 (PE), right-sided heart strain
Echo	PHTN, valvular disorders; Wall motion abnormalities related to ischemia, intracardiac shunts

Ancillary Radiologic Tests	
Test	Findings and Potential Diagnoses
<b>Chest Radiograph</b>	<b>Bony structures:</b> fractures, lytic lesions, pectus, kyphoscoliosis; <b>Mass:</b> malignancy, cavitary lesion, infiltrate, foreign body; <b>Diaphragm:</b> eventration, elevation of hemidiaphragm, bowel herniation; <b>Mediastinum:</b> adenopathy (infection, sarcoid), air; <b>Cardiac silhouette:</b> enlarged (CMO, fluid overload); <b>Soft tissue:</b> SQ air; <b>Lung parenchyma:</b> blebs, PTX, effusions (blood, infectious), interstitial edema, local consolidation, air bronchograms, Hampton's hump, Westermark's sign
<b>Scan</b>	PE
<b>Pulmonary angiogram</b>	PE, intervention (thrombolysis)
<b>CT</b>	Mass lesion adenopathy, trauma, PE
<b>MRI</b>	PE, bony and soft tissue lesions, vascular abnormality
<b>Soft tissue neck radiograph</b>	Epiglottitis, foreign body
<b>Ultrasound</b>	PTX, pleural effusion, impaired cardiac function or pericardial effusion

Ancillary Fiberscopic Tests	
Test	Findings and Potential Diagnoses
<b>Bronchoscopy</b>	Mass lesion, foreign body; Intervention (stenting, biopsy)
<b>Laryngoscopy</b>	Mass lesion, edema, epiglottitis, foreign body



**[7] Detail the utility of point-of-care ultrasound in the assessment of the dyspneic patient.**

POCUS for the dyspneic patient is essential. There is extensive data surrounding this topic in Emergency Medicine, but there is a good summary of the diagnostic utility presented in [an excellent emDOCs post](#).

**In summary:**

1. POCUS in isolation correlates well with diagnosis made by the ED physician in the patient with undifferentiated dyspnea
2. POCUS diminishes the time to diagnosis in the ED for the dyspneic patient
3. POCUS is highly specific and moderately sensitive for many of the acute causes of dyspnea in the ED

Examinations to perform	Diagnoses that can be elucidated
<ol style="list-style-type: none"><li>1. Lung US</li><li>2. Cardiac US - parasternal long, parasternal short, apical 4-chamber, and subcostal view</li><li>3. IVC US</li></ol>	<ol style="list-style-type: none"><li>1. CHF</li><li>2. Pericardial effusion</li><li>3. PTX</li><li>4. Pleural effusion</li><li>5. ACS</li><li>6. COPD/Asthma</li><li>7. PNA</li><li>8. PE</li></ol>

**[8] Outline a management algorithm for the acutely dyspneic patient.**

*This algorithm is adapted from Figure 22.1 titled “Rapid assessment and stabilization of the dyspneic patient” from Rosen’s 9th Edition. Please refer to the text for further clarification.*

1. Assess for the presence of respiratory distress (defined as RR >24 or <8, presence of tripodding, accessory muscle use, retractions, altered mental status, and/or abnormal chest wall movement)
  - a. If present, apply supplemental oxygen, apply pulse oximetry and continuous waveform capnography, and obtain IV access; proceed to step two after this has been completed
  - b. If not present, obtain pulse oximetry readings, supplement O2 as needed, and perform a directed history and physical examination
2. Determine if the patient is able to maintain their own airway or if there is evidence of respiratory failure
  - a. If yes to any of the above, assist ventilation and proceed to step three



- b. If no, get a CXR and proceed with history and physical examination
3. If assisting ventilation, assess breath sounds and prepare for endotracheal intubation or cricothyrotomy and mechanical ventilation if air entry is equal bilaterally and there are no pathologic movements of the chest or signs of chest wall trauma
4. If assisting ventilation and air entry is unequal bilaterally or there is pathologic movements of the chest/ signs of chest wall trauma, rapidly assess for signs of obstructive shock (hypotension, JVD, tracheal deviation, or US confirmation of PTX), immediately decompress the chest
  - a. If no tension PTX suspected, perform bedside US for cardiac collapse and PTX
5. Once the airway is secured, get a portable CXR, repeat your physical examination, and perform a targeted history and physical examination and draw initial lab investigations
  - a. If there is a + BNP, interstitial pattern on CXR, and clinical signs of CHF, consider getting an EKG and administering diuretics, nitrates, ACEi's, morphine, and NPPV
  - b. If there is wheeze and a clear CXR, and sharktooth capnography, consider anaphylaxis, bronchospasm, and administer bronchodilators/epinephrine/steroids/antihistamines as appropriate
  - c. If there are rhonchi, diminished breath sounds, and an infiltrate on CXR, consider PNA/pleural effusion and administer ABx after obtaining blood cultures
  - d. If there are no adventitious sounds +/- chest pain, consider PE versus anginal equivalent versus neuromuscular disorder or central neurogenic cause of the patients dyspnea
    - i. Get an EKG, ABG, cardiac biomarkers +/- D-dimer +/- CT PE
      1. If troponin +, consider ischemic causes and treatment for ACS
      2. If evidence of right heart strain on EKG, consider PE and treat for same
  - e. If there is unequal chest wall motion, signs of trauma, or evidence of PTX/hemothorax on US, consider thoracostomy

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## Wisecracks:

### [1] List three findings on chest radiograph suggestive of pulmonary embolism.

Answer: Fleischner sign, Hampton hump, Westermark sign, pleural effusion, Knuckle sign, Palla sign, Chang sign

We have broken these signs down for you below. The information on the diagnostic utility of these tests come from this [Radiopedia article](#).

#### 1. Fleischner Sign



- a. Enlarged pulmonary artery
- 2. Hampton Hump**
  - a. Peripheral wedge of airspace opacity that implies lung infarction
    - i. 22% sensitive, 82% specific
    - ii. PPV 29%, NPV 76%
- 3. Westermark Sign**
  - a. Focal or regional oligemia in the lung
    - i. 14% sensitive, 92% specific
    - ii. PPV 38%, NPV 76%
- 4. Pleural Effusion**
  - a. 36% sensitive, 70% specific
  - b. PPV 28%, NPV 76%
- 5. Knuckle Sign**
  - a. Abrupt tapering of the pulmonary artery secondary to embolus
- 6. Palla Sign**
  - a. Enlarged right descending pulmonary artery
- 7. Chang Sign**
  - a. Dilated right descending pulmonary artery with sudden cut-off

Remember, CXR is not diagnostic for PE. You largely order CXR's to rule out other pathologies that can cause the patient to become breathless. The vast majority of these signs are difficult to pick up at baseline, so take these findings with a grain of salt.

## **[2] What is the utility of venous blood gas testing and how do its values correlate with that of an arterial blood gas?**

Answer:

VBG is not perfect, but it is useful for the following parameters:

- 1. pH (GOOD CORRELATION)**
  - a. Large volume of research that indicates that the pH on a VBG adequately correlates well with the pH on ABG
  - b. There is a pooled mean difference of +0.035 pH units
- 2. PaCO<sub>2</sub> (GOOD AT IDENTIFYING NORMOCARBIA)**
  - a. Correlates well in patients with normocapnia (ie., if the VBG shows normocapnia, the patient is likely normocapnic on ABG)
  - b. 100% sensitive in identifying hypercarbia (PaO<sub>2</sub> > 45 mmHg) in AECOPD - although this conflicts with a recent meta analysis published in 2014
  - c. Once the PaCO<sub>2</sub> begins to climb, the VBG begins to become less reliable (ie., higher levels of CO<sub>2</sub> on VBG do not correlate well with levels on ABG)
  - d. Poor correlation in severe shock



**3. HCO<sub>3</sub> (GOOD CORRELATION)**

- a. Mean difference - 1.41 mmol/L

**4. Lactate (POOR CORRELATION AT HIGH LEVELS)**

- a. Dissociates after 2 mmol/L
- b. Mean difference 0.08 mmol/L

**5. PaO<sub>2</sub> (POOR CORRELATION)**

- a. As stated above, poor correlation with ABG - do not rely on results
- b. ABG PaO<sub>2</sub> often 36.9 mmHg higher on ABG with significant variability

**6. Base Excess (GOOD CORRELATION)**

- a. Mean difference 0.089 mmol/L

So, when should you order an ABG?

- Assess PaCO<sub>2</sub> in severe shock
- To determine PaCO<sub>2</sub> when hypercarbic
- To accurately determine arterial lactate levels

For more information, refer to this excellent post from [Life in the Fast Lane](#).