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Dyspnea

9

Rachel H. Bardowell and Adam V. Meyer

GENERAL PRINCIPLES

- Dyspnea is the sensation of difficulty breathing. Patients use various descriptors (e.g., “tight,” “heavy,” “hard to breathe,” “hunger for air,” “cannot get enough air,” etc.) that may correspond to specific causes.
- Up to 50% of patients admitted to tertiary care hospitals are affected by this often debilitating symptom.[1]
- When patients with dyspnea first present to the hospital, priority should be placed on determining the underlying process leading to the symptom as well as optimizing oxygenation. Many patients will have a previously known cardiac, respiratory, or neuromuscular condition, which can lead to dyspnea. It may be challenging to determine if there is worsening of a previously known condition or the onset of a new condition.
- Dyspnea is subjective and distinct from the laboratory finding of hypoxia.

- The medical consultant is frequently called to evaluate an inpatient who experiences the sudden onset of dyspnea. In this situation, a reasonable, rapid differential includes the following (even before seeing the patient): pulmonary embolism (PE), pneumothorax, pneumonia, airflow obstruction (mainly chronic obstructive pulmonary disease [COPD] and/or asthma), pulmonary edema, angioedema or anaphylaxis, myocardial ischemia/infarction, arrhythmias, and anxiety.
- Of course, once the patient is evaluated, this initial differential should quickly be refined. Anxiety is common in patients with dyspnea. Dyspnea may also be a manifestation of an underlying anxiety disorder. Psychogenic dyspnea is common in patients presenting to the hospital, and there is significant cost associated with their evaluation.[2] These patients may have associated depression or pain. Patients may describe progressive anxiety culminating in dyspnea (rather than progressive dyspnea resulting in

anxiety). They may also describe perioral or extremity numbness.

- Metabolic acidosis is compensated by tachypnea with large tidal volumes and a respiratory alkalosis. This rarely leads to dyspnea unless the acidosis is severe or there is underlying pulmonary pathology.
- Abdominal distention from ascites, pregnancy, or morbid obesity may also lead to dyspnea, but this is less likely to present acutely.
- Table 9-1 reviews the differential diagnosis in more general terms for the acute setting. The list of potential specific causes is enormous. Further details on many of the elements in the differential are discussed in other chapters.

TABLE 9-1 DIFFERENTIAL DIAGNOSIS OF DYSPNEA

Pulmonary: bronchospasm (e.g., COPD, asthma, anaphylaxis), aspiration, pneumonia, ARDS, pneumothorax, pulmonary embolism, pleural effusion, pulmonary hemorrhage, pulmonary hypertension
Cardiac: heart failure, pulmonary edema, right-to-left shunts, cardiac tamponade, acute myocardial ischemia, valvular dysfunction, arrhythmia
Hematologic: significant anemia, toxins resulting in impaired O_2–Hgb association or dissociation (e.g., CO)
CNS or neurologic: increased intracranial pressure (Cushing response), neuromuscular disease (i.e., respiratory muscle weakness)
Miscellaneous: severe metabolic acidosis, deconditioning, abdominal distention (e.g., morbid obesity, pregnancy, ascites), psychogenic (diagnosis of exclusion)

ARDS, acute respiratory distress syndrome; CNS, central nervous system; COPD, chronic obstructive pulmonary disease.

Diagnosis

Clinical Presentation

History

The history should include the following:

- Onset, quality, severity, duration, and ameliorating/exacerbating factors

- Associated symptoms such as fevers, chills, sweats, orthopnea, paroxysmal nocturnal dyspnea, wheezing, edema, chest pain, cough, sputum production, hemoptysis, palpitations, nausea, anxiety, dizziness, orthostasis, and weakness
- History of pulmonary, cardiac, neuromuscular/neurologic, renal, hepatic, and coagulopathic disorders
- Risk factors for deep venous thrombosis/PE[3]
- Ingestion of drugs, medications, toxic substances, and administration of IV fluids
- Smoking and environmental exposures

Physical Examination

The exam should focus on the cardiovascular and respiratory systems.[4–6]

- Respiratory rate, effort, and pattern of breathing should be carefully observed.
- Auscultate for adventitious lung sounds such as rales/crackles, rhonchi, and wheezes as well as for assessing the quality of airflow.
- Auscultate for murmurs, rubs, and gallops as well as note the rhythm and rate.
- Evaluate for signs of pulmonary consolidation, hyperresonance, and pleural effusion (e.g., egophony, changes in tactile fremitus, dullness to percussion).
- Evaluate jugulovenous pulsations and edema.
- Palpate the chest wall for tenderness, heaves, abnormal apical impulse, or crepitus.
- Evaluate for signs of deep venous thrombosis (e.g., asymmetric edema).
- Consider measurement of the pulsus paradoxus.

Diagnostic Testing

Testing should be directed based on results of history, physical exam, and vital signs, including pulse oximetry. The sometimes tenuous correlation between SaO_2 by pulse oximetry and measured PaO_2 must be recognized, particularly in the clinically important range of percentages in the 80s to low 90s.

Laboratories

- Anemia is rapidly ruled out with a complete blood count (CBC).
- Consider checking arterial blood gases (ABG) in all patients being evaluated for dyspnea. The ABG can provide a great deal of information. A patient can have a normal SaO_2, but an ABG may reveal a wide A–a gradient—an indication of lung pathology.
    - A simple but useful approach to interpreting the blood gas is to consider a low PaO_2 to be the result of pulmonary parenchymal or airspace disease, right-to-left shunts, ventilation/perfusion mismatching, or a dramatic increase in oxygen consumption with respect to delivery.
    - An elevated $PaCO_2$ is almost invariably the result of decreased alveolar ventilation or decreased exchange of gas between atmosphere and the alveolus. Most commonly, this is due to disease of the airways (COPD or asthma), but it may also be caused by chest wall disease or weakness of the respiratory muscles.
    - Central causes of elevated PCO_2 include central nervous system lesions, obesity, hypoventilation, and hypothyroidism.
    - With psychogenic causes, the ABG often reveals respiratory alkalosis with normal O_2 transfer.
    - The A–a gradient is calculated as follows: A–a gradient = PaO_2 − PaO_2 where the PA = $[(760 - 47) \times FiO_2] - (PaCO_2/0.8)$.
- Consider where appropriate, but not in all patients: B-type natriuretic peptide (BNP),[7] troponin I (or troponin T), and D-dimer.
    - Maintain a high degree of suspicion for PE, or you will miss the diagnosis. A normal D-dimer can be useful to exclude a PE in patients with low pretest probability[8] but is less useful in older patients and patients who have been hospitalized for more than 3 days.[9]
    - Similarly, a BNP can exclude the diagnosis of heart failure but will not replace clinical and imaging correlation if elevated.[10,11]

Electrocardiography

An electrocardiogram should be obtained.[11]

Imaging

- A chest radiograph should be obtained.[11] Most causes of acute onset

dyspnea are apparent after evaluating with a CXR and ECG.

- Bedside lung ultrasound, when available, can improve diagnostic accuracy in the evaluation of acute onset dyspnea.[12] This requires some degree of specialized training, however.
- Where appropriate, consider additional imaging studies including:
  - If PE is highly suspected, additional imaging including venous Doppler, ventilation–perfusion scanning, or computed tomography scan of the chest may be necessary.[3]
  - Pulmonary function testing with diffusing capacity for CO of the lung.
  - If heart failure is the suspected etiology, then echocardiography may be beneficial.

Diagnostic Procedures

Additional testing may be necessary to confirm a suspected diagnosis. Further diagnostic procedures to evaluate dyspnea can include the following: pulmonary function testing with diffusing capacity for CO (DLCO) of the lung and exercise cardiopulmonary testing and ABGs may reveal abnormalities that are not apparent at rest, particularly in patients with chronic dyspnea.[1]

TREATMENT

Treatment should focus on identifying and treating the underlying cause. Further details regarding management of specific conditions are discussed in other chapters. Psychogenic dyspnea can be controlled acutely with benzodiazepines such as lorazepam, though caution should be exercised especially in patients with concomitant pulmonary disease, due to the risk of respiratory depression and hypercapnia. Haloperidol may also be considered for its anxiolytic effect (which will not cause respiratory depression) in patients in the hospital who are delirious or psychotic.[2]

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