

UNIT NO. 4

BREATHLESSNESS

Dr Pothiwala Sohil

ABSTRACT

Breathlessness, also known as dyspnoea, is a common presenting complaint in the outpatient family physician clinic. It is most commonly caused by respiratory and cardiac disorders, but there are lots of other important aetiologies that need to be considered. The initial goal of assessment is to determine the severity of the dyspnoea, considering the need for oxygen therapy and intubation. Unstable patients usually present with abnormal vital signs, hypoxia, diaphoresis, and/or altered mental states, and require urgent management. Ongoing care depends on the differential diagnosis established by an adequate history and physical examination, and investigations, if available, can aid definitive diagnosis. The family physicians must be equipped to initiate appropriate therapy, conduct continuous monitoring and stabilise an acutely dyspnoeic patient before determining the final disposition of the patient.

Keywords:

Breathlessness, Dyspnoea, Family Physician, Differential Diagnosis, Management

SFP2015; 41(3): 24-29

INTRODUCTION

Dyspnoea, also known as shortness of breath, is one of the most common presentations in the emergency department or a primary care setting. The differential diagnosis ranges from simple medical conditions to life-threatening causes, and establishing a definitive diagnosis can be challenging.

Dyspnoea is defined as the subjective experience of breathing discomfort that is comprised of qualitatively distinct sensations that vary in intensity. The experience derives from interactions among multiple physiological, psychological, social, and environmental factors, and may induce secondary physiological and behavioral responses.¹

A patient with acute dyspnoea presents challenges in diagnosis and subsequent management. The clinician must consider a long list of differential diagnoses while providing appropriate initial treatment for a potentially life-threatening illness. In this article, we shall review the pathophysiology of dyspnoea, consideration of the differential diagnosis, and management.

This topic review will provide a differential diagnosis of the life-threatening and common causes of dyspnoea in the adult, describe important historical and clinical findings that can help to narrow the differential diagnosis, discuss the use of common

diagnostic studies, and provide recommendations for initial management and disposition.

EPIDEMIOLOGY

A chief complaint of dyspnoea or shortness of breath made up 3.5 percent of the more than 115 million visits to United States EDs in 2003. Other dyspnoea-related chief complaints (cough, chest discomfort) comprised 7.6 percent.²

According to one prospective observational study, the most common diagnoses among elderly patients presenting to an ED with a complaint of acute shortness of breath and manifesting signs of respiratory distress (e.g., respiratory rate >25, SpO₂ <93%) are decompensated heart failure, pneumonia, chronic obstructive pulmonary disease, pulmonary embolism, and asthma.³

PATHOPHYSIOLOGY

The respiratory system is designed to maintain homeostasis with respect to gas exchange and acid-base status. Derangements in oxygenation, ventilation and acidaemia can lead to dyspnoea. The development of dyspnoea is a complex phenomenon generally involving stimulation of a variety of mechanoreceptors throughout the upper airway, lungs, and chest wall, and chemoreceptors at the carotid sinus and the medulla. Respiratory system dyspnoea is related to disorders of the respiratory centre in the brainstem, the respiratory muscles, and the lung and alveoli. The cardiovascular system dyspnoea occurs as a result of cardiac diseases (e.g., acute myocardial ischaemia, systolic or diastolic heart failure, valvular disorders, pericardial diseases, etc.) and anaemia. A combination of these underlying mechanisms can also co-exist.

Evaluation of Dyspnoea

Acute dyspnoea is defined as breathing difficulty that arises over the course of minutes to hours. Consideration needs to be given to relatively few but life-threatening conditions. These entities typically have associated symptoms and signs that provide clues to the appropriate diagnosis, e.g., substernal chest pain with cardiac ischaemia; fever, cough, and sputum with respiratory infections; urticaria with anaphylaxis; and wheezing with acute bronchospasm. However, dyspnoea may be the sole complaint and the physical examination may reveal few abnormalities (e.g., pulmonary embolism, pneumothorax).

Chronic dyspnoea develops progressively over weeks to months. This group of patients are known to have underlying cardiopulmonary disease, the commonest being asthma, COPD, interstitial lung disease, or cardiomyopathy.

POTHIAWALA SOHIL

Consultant

Department of Emergency Medicine

Singapore General Hospital

In one study of 85 patients presenting to a pulmonary unit with a complaint of chronic dyspnoea, the initial impression of the aetiology of dyspnoea based upon the patient history alone was correct in only 66 percent of cases.⁴ Thus, a systematic approach, comprising of adequate history and physical examination, followed by appropriate investigations is necessary.

HISTORY

The history forms a critical component in the evaluation of the dyspnoeic patient, but can be difficult to obtain when the patient has difficulty speaking. Relevant history can be obtained from the patient, paramedics, family, and caretakers.

Present history

Enquire regarding the symptoms, duration, severity, onset whether it is sudden or gradual. Acute dyspnoea following a meal or medication suggests an allergic reaction; a new productive cough suggests chest infection; viral infection or allergen exposure can trigger asthma; recent surgery, immobilisation or long travel time increases the risk for pulmonary embolism (PE); and trauma may cause pneumothorax or pulmonary contusion. Noncompliance with medications or increased fluid intake may lead to acute decompensated heart failure.

Past history

The patient may have pre-existing illness like asthma, COPD, or ischaemic heart disease, and the dyspnoea can be a result of exacerbation of a preexisting illness. The medical records and medication list can provide important diagnostic clues.

Prior intubation

Patients with a history of prior endotracheal intubation have a higher severity risk and the need for subsequent intubation.

Associated Symptoms

Always check for associated symptoms like chest pain (acute coronary syndrome, pneumothorax, PE), fever (infection), trauma (pneumothorax, haemothorax), paroxysmal nocturnal dyspnoea (heart failure), and haemoptysis (tuberculosis, malignancy, PE). The presence of cough and quality of sputum should be enquired as purulent sputum suggests pneumonia, pink frothy sputum suggests heart failure. Similarly, dry cough is a non-specific symptom, and can be associated with asthma, heart failure, respiratory infection, or PE.

Medications

A review of the patient's medications, including recent prescriptions (e.g. antibiotics) and their compliance provides information about the pre-existing illness.

Tobacco/Smoking

Smoking (in pack-years) increases the risk COPD and malignancy.

Psychiatric conditions

Psychogenic causes for acute dyspnoea is a diagnosis of exclusion, and organic causes must be ruled out first before considering this diagnosis (e.g., panic attack).⁵

PHYSICAL EXAMINATION

Signs that suggest severe respiratory distress include tachypnoea/bradypnoea, retractions, use of accessory muscles, inability to speak in full sentences, inability to lie supine or in a tripod position, diaphoresis, agitation or altered mental status, and cyanosis.

Retractions occur with airway obstruction (e.g., asthma, COPD, foreign body) and can be seen in the suprasternal, intercostal, and subcostal areas. They are an ominous sign suggesting extreme respiratory distress. The use of accessory muscles to breathe suggests fatigue of the respiratory muscles and a potential for respiratory failure. Diaphoresis reflects extreme sympathetic stimulation associated with severity of disease process (myocardial infarction, severe asthma, pulmonary oedema). Altered mental status suggests severe hypoxia or hypercarbia, but may also be caused by underlying pathology (e.g., hypoglycaemia, sepsis, poisoning). Cyanosis is a late sign and indicates severe hypoxia.

Vital signs include temperature, heart rate, respiratory rate and pulse oximetry. Patients with serious underlying disease may have a fast, normal, or slow respiratory rate. The extremes indicate the severity of the underlying disease process. Pulse oximetry is an indicator of arterial oxygenation. It may be inaccurate in the setting of hypothermia, shock, carbon monoxide poisoning, methemoglobinaemia, etc.

A thorough systemic examination is an essential part of the evaluation process, and should include inspection, palpation, percussion and auscultation of the cardio-pulmonary, abdominal and neurological systems.

During examination of the respiratory system, the following abnormal breath sounds should be identified:

- Stridor occurs due to upper airway obstruction. Inspiratory stridor suggests obstruction above the vocal cords (e.g., foreign body, epiglottitis, angioedema).
- Wheezing occurs as a result of bronchoconstriction and can be heard in patients with asthma, COPD, anaphylaxis, foreign body obstruction, acute heart failure, or a tumour.
- Crepitations (crackles/rales) suggest the presence of interalveolar fluid seen in heart failure, pneumonia and with pulmonary fibrosis. However, the absence of crepitations does not rule out the presence of these pathologies.
- Diminished breath sounds can be caused by pathology that prevents air from entering the lungs, such as severe asthma or COPD, pneumothorax and haemothorax.

Table 1: Common and life-threatening causes of sudden onset of dyspnoea in an adult

Differential Diagnosis	Key Clinical Features
Upper airway obstruction: - Foreign body - Angioedema - epiglottitis - croup Cardiac: - congestive heart failure - acute pulmonary oedema - acute myocardial infarction - arrhythmia - Cardiac tamponade Pulmonary: - chronic obstructive pulmonary disease - asthma - pneumonia - pneumothorax • tension • simple - pulmonary embolism Trauma: - Pneumothorax/hemothorax/flail chest Psychogenic: - panic attacks/hyperventilation Others - metabolic acidosis - Poisoning - neuromuscular disorders - pain - anaemia	- Choking, stridor - Rash, allergy exposure, oral swelling, wheeze - Drooling, stridor, fever, toxic look - Fever, barking cough, stridor - Tachycardia, fluid retention, cough - Pink frothy sputum, diaphoresis, hypertension - Chest pain, diaphoresis - Tachycardia, +/- hypotension - Hypotension, muffled heart sounds, raised JVP - Cough, sputum change, +/- fever, wheeze - Tachypnoea, wheeze, tripod, diaphoresis - Fever, cough, chest pain - Chest pain, tracheal deviation, hypotension - Chest pain, past history - Pleuritic chest pain, tachycardia, DVT risk - History of trauma, decreased breath sounds - Anxiety, past history, perioral and distal paraesthesia, tetany - Precipitating cause – hyperglycaemia, sepsis - Carbon monoxide, cyanide, aspirin, etc. - Guillain-Barre syndrome, Amyotrophic lateral sclerosis - Precipitating cause of pain - Pallor, fatigue, blood loss

DIFFERENTIAL DIAGNOSIS OF ACUTE DYSPNOEA

Table 1 describes the common and life-threatening causes of sudden onset of dyspnoea in an adult, and their main clinical associated signs and symptoms. In children, the causes are

predominantly respiratory in origin, and can prove to be fatal sometimes.

INVESTIGATIONS

Laboratory and radiological tests should be performed in the

appropriate context of the history, physical examination, and the consideration of differential diagnosis. Random testing without a clear differential diagnosis will delay appropriate management. The use of dyspnoea biomarker panels does not appear to improve accuracy beyond clinical assessment and focused testing.^{6,7}

Blood tests

The initial evaluation usually includes measurement of haemoglobin (to exclude anaemia as a cause of dyspnoea), blood glucose (to rule out Kussmaul's breathing secondary to diabetic ketoacidosis), urea, creatinine and electrolytes.

Chest X-ray

The chest radiograph may provide clues to the cause of dyspnoea.

- Signs of Acute heart failure are cardiomegaly, cephalisation of blood vessels, interstitial oedema ("Kerley B" lines) and pulmonary vascular congestion. Pleural effusion may be present. Around 20 percent of patients admitted with acute heart failure may have a non-diagnostic CXR.⁸

- Infiltrates on CXR are considered the "gold standard" for diagnosing pneumonia. But X-rays obtained early in the clinical course of illness may be non-diagnostic.⁹ Also, the appearance of the CXR (lobar versus diffuse) does not accurately predict the aetiology of the pneumonia (typical versus atypical).

- A pneumothorax is usually visible on CXR as an area with absence of lung markings.

- Hyperinflated lung fields and a flattened diaphragm are suggestive of air trapping that occurs with COPD or asthma. Unilateral air trapping suggests obstruction by a foreign body or mucus plugging.

Electrocardiogram (ECG)

An ECG with ST segment and T-wave changes constitutes strong evidence of cardiac ischaemia. However, clinicians must remember that the initial ECG is normal in approximately 20 percent of patients subsequently diagnosed with a myocardial infarction, and only 33 percent of initial ECGs are diagnostic. The ECG may also reveal signs of pulmonary embolism (right heart strain) and pericardial effusion (low-voltage complexes, electrical alternans).

Bedside Ultrasound

This is increasingly being used to detect important causes of acute dyspnoea like cardiac tamponade, pneumothorax or pleural effusion. It is also being used to detect cardiac wall motion abnormalities suggestive of ischaemia or pulmonary embolism.

Cardiac biomarkers

Elevated cardiac biomarkers support the diagnosis of cardiac

ischaemia. Serial measurements of cardiac biomarkers are necessary as initial results can often be normal.

Brain natriuretic peptide (BNP)

This is used to diagnose heart failure, but it can also be elevated in fluid overload secondary to renal failure. BNP testing is not helpful when used indiscriminately in patients with acute dyspnea.^{10,11}

D-Dimer

Patients at low risk for PE according to a validated scoring system (Wells criteria for PE, PERC rule) and a negative d-dimer can be ruled out for PE without further testing. It cannot be used as a rule-out test in patients with moderate to high pre-test probability of PE.

Arterial and venous blood gas

The role of the arterial blood gas (ABG) in the diagnosis and treatment of the acutely dyspnoeic patient is limited. Venous blood gas can be used as an alternate to determine the acid-base status. The PaCO₂ levels are low in a breathless patient due to hyperventilation. A normal or elevated CO₂ in a breathless patient suggests respiratory failure.

CT Thorax

A multi-detector computed tomography (MDCT) scan of the thorax is not indicated in the initial evaluation, but can aid diagnosis of PE, malignancy, etc. MDCT entails complications like contrast-induced nephropathy, contrast allergy, radiation, and hence must be used judiciously.

Peak flow and pulmonary function tests (PFTs)

The peak expiratory flow rate (PEFR) is useful in determining the severity of asthma and response to treatment.

MANAGEMENT

A quick initial assessment will aid the doctor in determining if the patient is stable or unstable. The management is dependent on his/her presentation and possible diagnosis, but the initial treatment includes administration of supplemental oxygen using various oxygen delivery devices. You need to be careful using oxygen in patients with COPD with type 2 respiratory failure (hypercapnoea) as it may depress their respiratory drive. Aim to maintain oxygen saturation of around 94 percent. Establish intravenous access and start administration of fluids if indicated. Consider the use of oral or nasopharyngeal airway in patients who are unable to maintain their airway due to depressed levels of consciousness. Intubation may be needed if the patient is in severe respiratory failure, gasping or apnoeic. Treatment of a few common conditions is discussed below:

Asthma

The cornerstone of management of an asthmatic patient is bronchodilator therapy and steroids (oral prednisone or intravenous hydrocortisone). Bronchodilator therapy using salbutamol can be delivered through a nebuliser or metered dose

inhaler (MDI). In severe asthma, patients should be transferred to ED for further treatment with nebulised ipratropium bromide, intravenous magnesium, ketamine, IM adrenaline, intubation, and inhalational anaesthesia as needed.

COPD exacerbation

Patients with mild exacerbation are treated with nebulised salbutamol, ipratropium and steroids. Antibiotics are indicated if there is evidence of an infective process suggested by fever, increased sputum production, or purulent sputum. Patients with moderate to severe respiratory distress should be sent to ED for further management, which can include non-invasive ventilation (BiPAP) or endotracheal intubation.

Pneumonia

Patients who are low risk using the CURB-65 score should be commenced on appropriate antibiotic therapy as per the local antibiotic guidelines and discharged with follow-up within 5-7 days for re-assessment. Patients with moderate to high risk should be referred to the hospital for admission and further management. In severe pneumonia, 3rd-generation cephalosporin (ceftriaxone) with a macrolide (azithromycin) is the first-line treatment. In patients with penicillin allergy, a fluoroquinolone should be used.

Acute Pulmonary Oedema

Intravenous furosemide along with sublingual nitroglycerine tablets/spray should be commenced in the clinic. They should then be transferred to the nearest ED for further management.

Pneumothorax

For a patient showing signs of tension pneumothorax, needle thoracocentesis should be urgently performed in the 2nd intercostal space in the mid-clavicular line on the affected side. With the cannula in-situ, the patient can then be transferred by the paramedics to the ED for definitive management with chest-tube insertion. Patients with simple pneumothorax should be commenced on oxygen and referred to the ED for further management.

Pulmonary Embolism

A patient suspected to have PE should be risk stratified using the Wells criteria for PE or PERC rule for PE. Patients who are low risk and PERC positive should have a D-dimer. If the D-dimer is negative, PE is ruled out. The chest X-ray is usually normal, but Hampton's hump or Westermark sign are rare and diagnostic of PE. Patients who are intermediate or high risk, or who have a positive D-dimer, should have advanced imaging like CT pulmonary angiogram. Treatment is largely supportive, with oxygen therapy. Patients who are hypotensive due to massive PE should receive intravenous fluids and vasopressors and commenced on anticoagulant therapy using unfractionated heparin or a low-molecular-weight heparin, followed by warfarin. Haemodynamically unstable patients should be thrombolysed. If thrombolysis is contraindicated, the patient should be referred for embolectomy.

Occasionally, it may be difficult to determine the exact cause of

breathlessness. In such cases, it is prudent to start therapies for multiple conditions in the initial resuscitative phase. For example, for a patient with a past medical history of COPD and congestive cardiac failure, the initial management of sudden onset of dyspnoea may include therapies directed at both these conditions. After initial stabilisation, the final disposition of the patient depends on your consideration of the differential diagnosis. Unstable patients should be transported to the emergency department for further evaluation and treatment. Patients who are stable should be discharged with appropriate medications and advised to return back if their condition persists or worsens.

CONCLUSION

The family physicians must be equipped to perform an initial assessment, initiate appropriate therapy and stabilise an acutely dyspnoeic patient before determining the disposition of the patient, either discharging home or referring to the emergency department for further management.

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LEARNING POINTS

- Acute dyspnoea is most commonly caused by respiratory and cardiac aetiologies, but there are lots of other aetiologies that also need to be considered.
 - The initial goal of assessment is to determine the severity of the dyspnoea and identify unstable patients with a risk of deterioration.
 - The family physicians must initiate appropriate therapy based on the differential diagnosis, stabilise an acutely dyspnoeic patient and decide on appropriate disposition.
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