

Breathlessness

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Clinical assessment and causes

Physiological mechanisms of breathlessness

Dyspnoea refers to the abnormal and uncomfortable awareness of breathing. Its physiological mechanisms are poorly understood; possible afferent sources for the sensation include receptors in respiratory muscles, juxtacapillary (J) receptors (sense interstitial fluid), and chemoreceptors (sensing $\uparrow\text{CO}_2$ and $\downarrow\text{O}_2$).

Clinical assessment

All patients need a full history and examination. Key points in the assessment are:

- Duration and onset of breathlessness. The box opposite groups the causes of breathlessness by speed of onset, although in practice some variability and overlap exists. Patients often underestimate the duration of symptoms—enquiring about exercise tolerance over a period of time is a useful way of assessing duration and progression
- Severity of breathlessness. Assess the level of handicap and disability by asking about effects on lifestyle, work, and daily activities
- Exacerbating factors. Ask about rest and exertion, nocturnal symptoms, and body position. The timing of nocturnal breathlessness may provide clues to the likely cause: left ventricular failure causes breathlessness after a few hours of sleep, and resolves after about 45 min; asthma tends to occur later in the night; laryngeal inspiratory stridor causes noisy breathlessness of very short duration (<1 min); and Cheyne–Stokes apnoeas result in breathlessness that is recurrent and clears each time in less than 30 s. Orthopnoea is suggestive of left ventricular failure or diaphragm paralysis, although it is also common in many chronic lung diseases. Breathlessness during swimming is characteristic of bilateral diaphragm paralysis. Trepopnoea refers to breathlessness when lying on one side as a result of ipsilateral pulmonary disease
- Associated symptoms, such as cough, haemoptysis, chest pain, wheeze, stridor, fever, loss of appetite and weight, ankle swelling, and voice change. Wheeze may occur with pulmonary oedema, pulmonary embolism, bronchiolitis, and anaphylaxis, in addition to asthma and COPD
- Personal and family history of chest disease
- Lifetime employment, hobbies, pets, travel, smoking, illicit drug use, medications
- Examination of the cardiovascular and respiratory systems. Observe the pattern and rate of breathing. Assess for signs of respiratory distress. Look for paradoxical abdominal movement if the history suggests diaphragmatic paralysis. A useful bedside test is to exercise the patient (e.g. by stepping on and off a 15–20-cm block) until their breathlessness occurs, and then measure oximetry immediately on stopping when the finger is still; a fall in oxygen saturation is expected with organic causes of dyspnoea.

Investigations

Initial investigations typically include resting oximetry, peak flow and spirometry, CXR, and ECG. Further tests depend on clinical suspicion; options include full PFTs with measurement of lung volumes lying and standing, gas transfer and flow-volume loop, bronchial hyperresponsiveness or reversibility testing, maximal mouth or inspiratory sniff pressures, ABGs (with measurement of A–a gradient, see p 793), exercise oximetry, ventilation perfusion scanning and CTPA, HRCT, blood tests (full blood count and TSH), echo, exercise ECG, and cardiac catheterization.

Causes of breathlessness grouped by speed of onset

Instantaneous

- Pneumothorax
- Pulmonary embolism.

Acute (minutes–hours)

- Airways disease (asthma, exacerbation of COPD, upper airways obstruction)
- Parenchymal disease (pneumonia, pulmonary oedema, pulmonary haemorrhage, acute hypersensitivity pneumonitis)
- Pulmonary vascular disease (pulmonary embolism)
- Cardiac disease (e.g. acute myocardial infarction, arrhythmia, valvular disease, tamponade, aortic dissection)
- Metabolic acidosis
- Hyperventilation syndrome.

Subacute (days)

- Many of the above, plus:
 - Pleural effusion
 - Lobar collapse
 - Acute interstitial pneumonia
 - Superior vena cava obstruction
 - Pulmonary vasculitis.

Chronic (months–years)

- Some of the above, plus:
 - Obstructive airways disease (COPD, asthma)
 - Diffuse parenchymal disease (including idiopathic pulmonary fibrosis, sarcoidosis, bronchiectasis, lymphangitis carcinomatosa)
 - Pulmonary vascular disease (chronic thromboembolic disease, idiopathic pulmonary hypertension, veno-occlusive disease)
 - Hypoventilation (chest wall deformity, neuromuscular weakness, obesity)
 - Anaemia
 - Thyrotoxicosis.

Specific situations

Causes of breathlessness with a normal CXR

- Airways disease (asthma, upper airways obstruction, bronchiolitis)
- Pulmonary vascular disease (pulmonary embolism, idiopathic pulmonary hypertension, intrapulmonary shunt)
- Early parenchymal disease (e.g. sarcoid, interstitial pneumonias, infection—viral, PCP)
- Cardiac disease (e.g. angina, arrhythmia, valvular disease, intracardiac shunt)
- Neuromuscular weakness
- Metabolic acidosis
- Anaemia
- Thyrotoxicosis
- Hyperventilation syndrome (p 255).

Causes of episodic/intermittent breathlessness

- Asthma
- Pulmonary oedema
- Angina
- Pulmonary embolism
- Hypersensitivity pneumonitis
- Vasculitis
- Hyperventilation syndrome.

Distinguishing cardiac and respiratory causes of breathlessness

This can be difficult. Many of the clinical features of left heart failure are non-specific and easily confused with respiratory disease (e.g. orthopnoea, wheeze). In chronic cardiac failure, crackles on auscultation and radiological features of pulmonary oedema may be absent even when the pulmonary capillary wedge pressure is significantly raised (due to adaptive changes from vascular remodelling). The presence of emphysema may also render crackles inaudible and lead to atypical CXR appearances of pulmonary oedema. Chronic left heart failure commonly leads to a restrictive ventilatory defect and reduced gas transfer on PFTs, and may also result in pulmonary hypertension. HRCT features of left heart failure include septal and peribronchovascular interstitial thickening, ground-glass shadowing, pleural effusions, and cardiomegaly. Resting ECG is useful—in practice, a cardiac cause of breathlessness is unlikely in the setting of a completely normal ECG. Exercise ECG, echo, and cardiac catheterization may be required. Measurement of B-type natriuretic peptide (BNP) is a recent development that may be of value in the diagnosis of cardiac failure; in patients presenting as an emergency with breathlessness, a serum BNP level <50 ng/L makes cardiac failure very unlikely. Cardiac and respiratory diseases can, of course, coexist.

Further information

Maisel AS, Krishnaswamy P *et al*. Rapid measurement of B-type natriuretic peptide in the emergency diagnosis of heart failure. *N Engl J Med* 2002; **347**: 161–7