

Chapter 17

Acute Shortness of Breath

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Key Points

- Evaluate patients immediately for signs of clinical instability, e.g. stridor, tachypnoea or altered consciousness.
- The aim of oxygen therapy is to maintain SaO₂ 94–98 % although a target SaO₂ 88–92 % may be appropriate for a patient with COPD.
- PEFr measurement is a valid assessment of airway calibre and should be compared to best or predicted in known asthmatic patients.

Introduction

Acute shortness of breath is a common presenting problem among patients who attend the emergency department. It has been estimated that 3.5 % of patients have attended the emergency department in the United States with shortness of breath in 2003. In 2009, there were a total of 3.7 million emergency department visits with shortness of breath as a primary complaint [1, 2]. Although there is no specific data relating to shortness of breath, the epidemiology of cardiac and pulmonary diseases indicates that the magnitude of the problem is large.

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Shortness of breath is a normal symptom of heavy exertion but becomes pathological if it occurs in unexpected circumstances. In most cases, it is due to asthma, pneumonia, congestive cardiac failure, chronic obstructive pulmonary disease, interstitial lung disease, anaphylaxis, etc. Conditions such as asthma, COPD and PE are already discussed on its own as a separate chapter, and it won't be discussed in this chapter.

Definition

Acute breathlessness is the subjective experience of breathing discomfort for less than 48 h. The American Thoracic Society has defined dyspnoea as a term used to characterise a subjective experience of breathing discomfort that is comprised of qualitatively distinct sensations that vary in intensity [3].

Pathophysiology

The pathophysiology of breathlessness is poorly understood. It is likely that breathlessness arises when there is a mismatch between the neural drive to breathe and the resulting ventilatory response. But whilst hypoxia and hypercarbia may induce 'air hunger', not all patients with abnormalities in gas exchange describe breathlessness. Moreover, most breathless patients have neither hypoxaemia nor hypercarbia. It is more likely that the presence of obstructive lung disease (asthma or COPD) and reduced lung compliance (lung fibrosis, pulmonary oedema, pneumonia) increase the work of breathing giving rise to symptoms.

Causes and Clinical Signs of Shortness of Breath

The duration of the onset of breathlessness usually helps to identify the likely cause of breathlessness (Table 17.1) and general clinical signs as described in Table 17.2.

Investigations

- Oxygen saturation (SaO₂) measured by pulse oximetry can be used to assess the adequacy of oxygen therapy and the need for arterial blood gas (ABG) measurement.
- PEFr measurement is a valid assessment of airway calibre and should be compared to best or predicted in known asthmatic patients.
- Chest X-ray (Fig. 17.1) should be performed looking for the cause of acute breathlessness (infiltrates, consolidation, pneumothorax, pulmonary oedema, etc.).

Table 17.1 Timescale of onset of breathlessness and likely diagnosis

	Minutes to hours	Hours to days	Days to weeks	Weeks to months to years
Respiratory	Pneumothorax Pulmonary embolus Acute asthma	Pneumothorax Pulmonary embolus Acute asthma Pneumonia Pleural effusion	COPD exacerbation Pneumonia Pleural effusion	COPD/emphysema Lung cancer Lung fibrosis Pulmonary Hypertension
Cardiac	Myocardial infarction Acute heart failure Arrhythmias	Myocardial infarction Acute heart failure	Heart failure	Heart failure Valvular heart disease
Other	Anaphylactic shock Inhalation/choking Dysfunctional breathing Aspirin poisoning	Dysfunctional breathing Metabolic acidosis	Dysfunctional breathing Pregnancy Anaemia	Dysfunctional breathing Pregnancy Anaemia Neuromuscular weakness Obesity and deconditioning

Table 17.2 Clinical signs of respiratory distress and respiratory failure

Increased work of breathing	Systemic effects of inadequate respiration
Inspection	Inspection
Use of accessory muscles	Cyanosis
Need to sit upright	Cool clammy skin
Tracheal tug	Confusion or aggressive behaviour
Intercostal recession	Decreased GCS
Inability to speak in whole sentences	Exhaustion
	Chest pain
Auscultation	
Expiratory wheeze or stridor	Observations
Silent chest	Respiratory rate <10 or >25 breaths/minute
	Pulse rate >120 beats per minute
	SaO ₂ <92 % room air
	SaO ₂ <95 % high flow oxygen
	Systolic BP <90 mmHg
	Auscultation
	Silent chest
	Arterial blood gas
	pCO ₂ >6.0 kPa

Breathlessness: Causes and Management

Pneumothorax [4, 4a, 5] (Table 17.3)

Assessment and Treatment of Simple Pneumothorax

Controlled oxygen therapy should be administered with a target SaO₂ 94–98 % unless the patient has known COPD (target SaO₂ 85–92 %).

Fig. 17.1 A large spontaneous primary pneumothorax in a young man

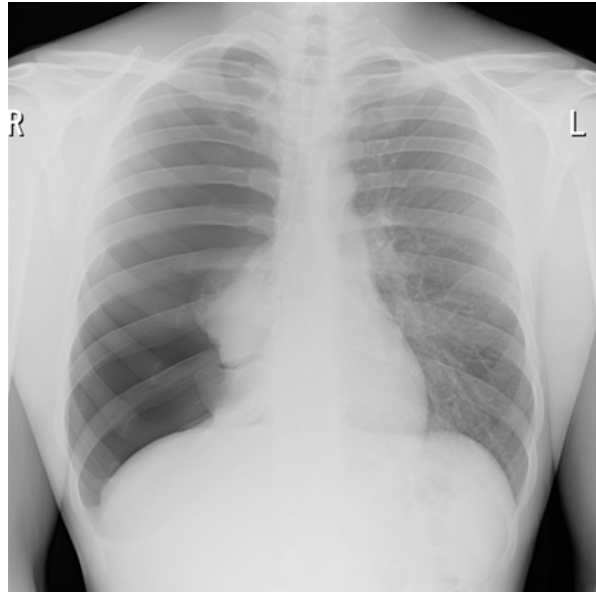


Table 17.3 Classification of pneumothorax [4]

Spontaneous pneumothorax		Traumatic pneumothorax	
Primary (no underlying lung disease)	Secondary (underlying lung disease)	Blunt or penetrating thoracic trauma	Iatrogenic
<p><i>Usually occurs in young healthy adult men</i> 85 % patients are less than 40 years old More common in males Bilateral in 10 % of Cases Usually result of rupture of an acquired subpleural bleb – apical blebs found in 85 % of patients undergoing thoracotomy Increased frequency of spontaneous pneumothorax after each episode, but most recurrences occur within 2 years of the initial episode</p>	<p>10–20 % of spontaneous pneumothorax Can be due to: COPD (70 % cases) Asthma Interstitial lung disease Cancer including pleural metastasis Connective tissue diseases (Ehlers-Danlos syndrome, Marfan’s syndrome) Infection (TB, PCP, necrotising pneumonia)</p>	<p>Associated rib fractures, flail chest and haemothorax</p>	<p>Common and occurs after: Thoracic surgery, thoracocentesis and transbronchial biopsy Other causes include: High-pressure mechanical ventilation Central venous cannulation Tracheobronchial and oesophageal injuries can cause both mediastinal emphysema and pneumothorax</p>

Conservative Treatment

- Not all patients with small spontaneous, traumatic or iatrogenic pneumothorax are symptomatic (Fig. 17.2).
- In the case of post-procedural iatrogenic pneumothorax, a small pneumothorax may only be picked up by a routine post-procedure plain radiograph.
- If no intervention is required, patients must be followed up with a repeat radiograph to ensure that the pneumothorax has resolved and that the lung has fully expanded within 2–4 weeks.
- If the patient becomes increasingly breathless over the next few days, it is likely that the pneumothorax has increased in size and the patient must seek medical attention for an urgent assessment and chest radiograph.
- Perversely, conservative management with close observation during a hospital admission may be safer than undertaking aspiration or intercostal drainage in a patient who has a small secondary pneumothorax.

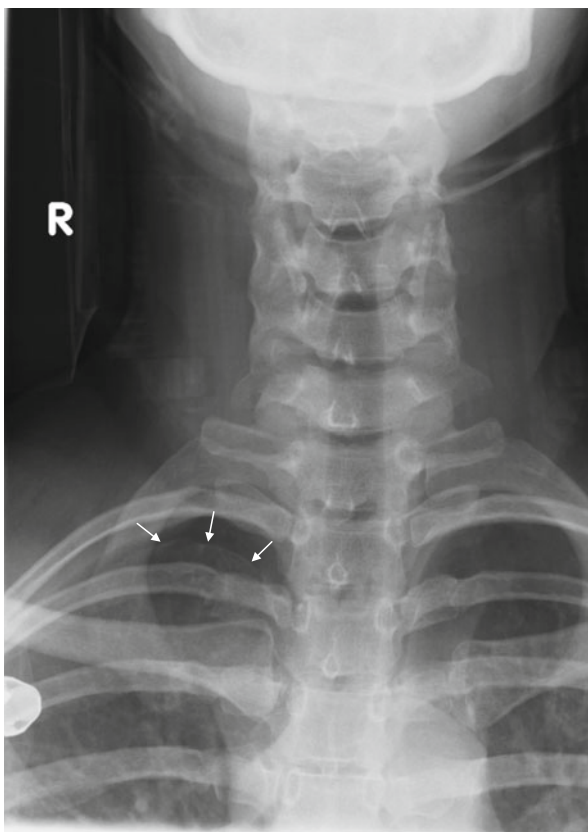


Fig. 17.2 Incidental, small traumatic pneumothorax following cervical neck injury. Pleural line is indicated by *white arrows*. This resolved without any intervention within 7 days

- Patients who have sustained a pneumothorax should be advised that they are not allowed to fly until the lung has been fully re-expanded for 1 week. Recreational sub-aqua diving is not recommended unless there has been corrective surgical repair.

Pleural Aspiration

- Even large spontaneous primary pneumothoraces (>50 %, >2 cm on plain CXR) may be treated with pleural aspiration.
- A large-bore cannula is introduced into the pleural space through a sterile field and using local anaesthesia. A three-way tap is attached to the needle and up to 2.5 L air can be aspirated.
- Aspiration can significantly reduce the size of spontaneous pneumothorax allowing the patient to have a minimally invasive intervention that reduces the risk of morbidity associated with an intercostal drain insertion and hospital admission.
- Aspiration can also be considered in small spontaneous secondary pneumothorax dependent upon the degree of symptoms and the extent of underlying lung abnormality.
- If the lung does not re-expand, it implies that the air leak is larger than the ability to aspirate air from the pleural space and an intercostal drain is required.
- It is advisable to admit patients following successful aspiration for secondary pneumothorax and to repeat the chest radiograph within 24 h.

Intercostal Drainage

- Patients with symptomatic large primary or secondary pneumothorax require intercostal drainage. If the secondary pneumothorax is small, or there is plain radiograph evidence that there is pleural adherence to a part of the chest wall, it is not safe to undertake a 'blind' procedure, and an intercostal drain placed under CT guidance by an interventional radiologist may be required.
- The intercostal drain should be placed in the 'triangle of safety' (pectoralis major anteriorly, latissimus dorsi posteriorly, and a line superior to the horizontal level of the nipple inferiorly).
- A small-bore Seldinger drain (12–18 F) is introduced into the pleural space through a sterile field and using local anaesthesia. A three-way tap is attached to the intercostal drain before being connected to an underwater seal.

Pleurodesis and Thoracic Surgery

- The definitive treatment for pneumothorax is pleurodesis which obliterates the pleural space and adheres the lung to the chest wall. Pleurodesis can be performed medically with talc slurry administered through an intercostal drain.

Other Respiratory Causes of Acute Shortness of Breath

There are some conditions, such as pneumonia, where patients may have had systemic symptoms for a while, but breathlessness occurs late in disease presentation.

Patients with chronic lung disease such as COPD/emphysema, bronchiectasis and fibrotic lung disease have exacerbations where breathlessness worsens suddenly, usually in association with infections.

Pneumonia [7]

Aetiology

- The commonest aetiology is infective, usually bacterial, although viral and fungal pneumonia is increasingly recognised especially in the immunocompromised patient.
- Inflammatory pneumonia such as eosinophilic pneumonia is not infective in aetiology and is treated with oral corticosteroid.
- Likewise, injurious pneumonia such as that caused by aspiration of gastric contents is only in part an infective process.

Risk Factors

- Increasing age
- Smoking
- Pre-existing lung disease
- Diabetes
- Chronic kidney disease
- Alcoholism
- Immunocompromise and recent influenza

Classifications

- Most widely accepted is community versus hospital-acquired pneumonia.

Community-Acquired Pneumonia (CAP) [8]

- The commonest organism identified in this group is *Streptococcus pneumoniae* (approximately 40 %). Other organisms include *Chlamydophila pneumoniae*, *Mycoplasma pneumoniae*, influenza A and B, *Haemophilus influenzae* and *Legionella* species in descending order of frequency.

- The identification of *Staphylococcus aureus* or Gram-negative bacilli is infrequent and accounts for less than 2 % of organisms identified.
- The incidence of CAP is 5–11 per 1,000 population [6] and varies with age with the highest incidence in the very young and the elderly. CAP patients requiring admission have a mortality of 5–15 %, and 1–10 % will require admission to the intensive critical care unit.

Hospital-Acquired Pneumonia (HAP)

Hospital-acquired pneumonia is defined as pneumonia that develops 48 h or more after admission to hospital and was not incubating at the time of admission. HAP affects up to 1.0 % of inpatients and is thought to increase the length of stay by 7–9 days.

- The commonest organisms identified are *Staphylococcus aureus* (20–30 %), *Pseudomonas* spp. (20 %), *Escherichia coli* (5–15 %) and *Klebsiella* spp. (5–10 %). These organisms are also more likely to be highly resistant to antibiotics.

Common Symptoms

- Cough.
- Sputum production.
- Breathlessness.
- Pleurisy and fever.
- Confusion without hypoxaemia is frequently encountered in the elderly patient.
- In the young adult, breathlessness may be a late presenting feature, and myalgia and flu-like symptoms may predominate.

Assessment and Treatment of Pneumonia

Pneumonia can be diagnosed if there are symptoms and signs consistent with acute lower respiratory tract infection and new radiological findings consistent with infection that cannot be explained by another pathology such as pulmonary oedema or infarction.

Radiology

- Plain chest radiograph usually reveals focal or diffuse opacities consistent with airspace shadowing or infiltrates. The hallmark of consolidation is an air bronchogram (air-filled bronchi made visible by surrounding consolidated/infiltrated lung tissue).

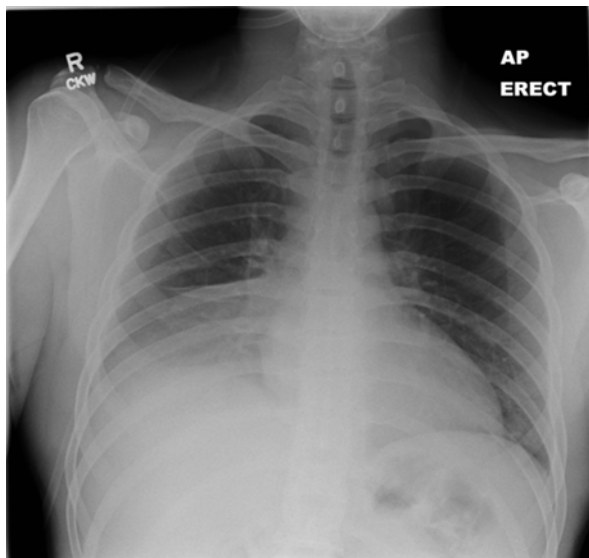
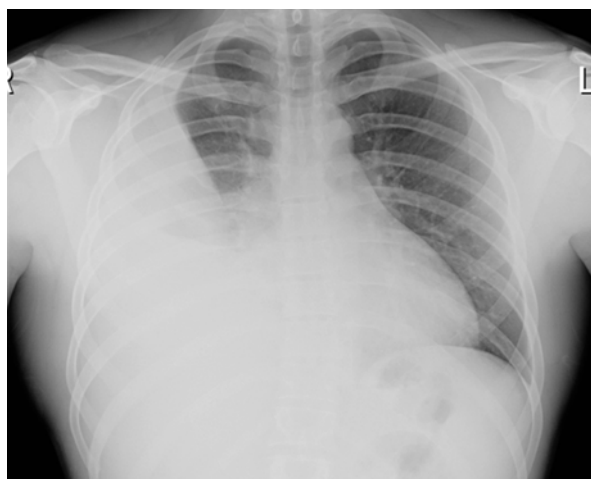


Fig. 17.3 Right lower zone shadowing with loss of the right hemi-diaphragm consistent with consolidation in a young man with community-acquired pneumonia

Fig. 17.4 Parapneumonic effusion secondary to community-acquired pneumonia that required intercostal drainage and surgical debridement



- Other findings on CXR that suggest the presence of pneumonia include silhouette sign (e.g. loss of outline of the right hemi-diaphragm indicates pathology in the right lower lobe) (Fig. 17.3) and pleural effusion (parapneumonic effusion) (Fig. 17.4).
- Complications of pneumonia may be apparent such as atelectasis, lung abscess and, more rarely, pneumothorax.

Laboratory

- Differential white cell count.
- Inflammatory indices such as C reactive protein.
- Electrolytes to look for acute kidney injury and urea to help in assessment of severity of pneumonia.
- Sputum as well as blood cultures to identify aetiological pathogen.
- Serological testing (*Mycoplasma*, *Chlamydomphila*, *Coxiella*, influenza) should be undertaken in all cases of severe CAP.
- Urinary streptococcal and legionella antigen should be undertaken in all cases of moderate and severe CAP.
- It is recommended that HIV serology is undertaken in all adults admitted with CAP.
- If pulmonary tuberculosis is suspected, sputum examination for acid-fast bacilli is essential.

Severity Assessment

The severity of pneumonia is important to assess at presentation. The most widely used severity index for patients with CAP admitted to the hospital is the CURB-65 (Table 17.4), and increasing scores correlate with increasing mortality rates. However, clinical judgement must also be used in the interpretation of this score as younger adults can be very unwell with low CURB-65 values.

Treatment

- Antibiotic treatment for pneumonia is empirical, based on the likelihood of the aetiological pathogen and local guidelines to account for pathogenic resistance.

Table 17.4 CURB-65 [7]

Score 1 for each of the following:	
Confusion (GCS <8 or new disorientation in time, manner, place)	
Urea (>7 mmol/L)	
Respiratory rate >30 per minute	
Blood pressure (systolic <90 mmHg and /or diastolic <60 mmHg)	
Age >65 years	
Score	Mortality (%)
0	0.6
1	3.2
2	3.0
3	17.0
4	41.5
5	57.5

- Different hospitals have antibiotic guidelines specifically for CAP and HAP, including recommendations based on severity, patients with penicillin allergy or intolerance, for co-infection with methicillin-resistant *Staphylococcus aureus* (MRSA) and in the neutropenic or immunocompromised host.
- Once the diagnosis of pneumonia is suspected or confirmed, it is important to treat with empirical antibiotic therapy and fluid resuscitation with crystalloid if appropriate.
- Supportive therapy with CPAP in patients deemed not suitable for mechanical ventilation is now widely accepted best medical practice.

Pleural Effusions

It is unusual for pleural effusions to develop rapidly and cause acute breathlessness. However, conditions such as pneumonia, heart failure and lung cancer may be complicated by pneumonia, and a therapeutic aspiration in the ED may alleviate symptoms.

Acute Aspirin Overdose

In acute aspirin overdose, as well as nausea, vomiting and tinnitus, there may be hyperventilation. This occurs because the uncoupling of cellular oxidative phosphorylation stimulates the respiratory centre in the medulla causing primary respiratory alkalosis. In addition, acute aspirin poisoning induces a metabolic acidosis which may also cause a secondary respiratory alkalosis.

Kussmaul's Breathing or Air Hunger

As metabolic acidosis worsens, respiratory compensation to 'blow off' carbon dioxide occurs and was first described by the German physician Adolph Kussmaul who recognised this phenomenon in patients suffering with severe diabetic ketoacidosis. Air hunger is observed in all forms of severe metabolic acidosis, and the arterial blood gas analysis will reveal decreased PCO₂ with decreased [HCO₃⁻] and a negative base excess.

Hyperventilation Syndrome or Dysfunctional Breathing

Hyperventilation or dysfunctional breathing is a diagnosis of exclusion, and it is important to investigate acute breathlessness to ensure that another cause of breathlessness such as PE has not been missed.

Symptoms

- Breathlessness at rest without any evidence of cardiopulmonary disease
- Tingling in the extremities and perioral paraesthesia
- Chest pain
- Dizziness

Calculating the A-a gap (the difference between the alveolar concentration of oxygen and the arterial concentration of oxygen) ensures that a cause of hypoxaemia is not missed.

To further complicate matters, hyperventilation is common in persons with mild respiratory disease. A good example of this can be seen in patients with mild asthma who can reduce their inhaled therapy when they undertake Buteyko [9, 10] breathing exercises, a form of breathing control.

Treatment

- Removal of the stressor (if possible) and removal of oxygen.
- Rebreathing air (breathing in and out of a brown paper bag) resolves low end-tidal blood carbon dioxide levels. This should only be undertaken for 6–12 breaths at a time as hypoxia can be induced if undertaken for prolonged periods.
- The treatment for dysfunctional breathing is education and physical therapy (exercise and breathing control).

Cardiac Causes of Acute Dyspnoea

Acute changes in cardiac physiology can lead to breathlessness either as a result of decreased cardiac output leading to tissue hypoxia and hypercarbia thus triggering an increased respiratory rate as compensation or as a result of pulmonary oedema, which may also cause tissue hypoxia but also stimulates breathlessness by decreasing lung compliance. The underlying causes of each broad pathophysiological mechanism (i.e. pulmonary oedema or non-oedema) can overlap but can be broadly categorised as acute myocardial ischaemia or dysfunction (e.g. myocarditis), arrhythmia and mechanical valve dysfunction.

Acute Pulmonary Oedema

Any cause of acutely increased left ventricular filling pressure (LVFP) may result in pulmonary oedema. The increase in preload leads to inefficient pump function, hence reduced stroke volume and an increase in total peripheral resistance, which

further reduces stroke volume. Therefore, effective treatment strategies rely on reducing the preload or peripheral resistance.

Chest radiograph usually reveals upper lobe blood diversion with perihilar shadowing and, less commonly, small (bilateral) pleural effusions and/or Kerley B-lines.

Clearly time delayed on a lengthy history and examination approach will lead to the harm of the patient, and therefore, a treatment and simultaneous differential generation approach is advised in all guidelines [11].

Treatment of Acute Pulmonary Oedema

The primary treatment modalities are:

- Oxygen
- Diuretics
- Opiates
- Nitrates

Non-invasive Ventilation (NIV)

It has long been thought that negative intrathoracic pressure reduces left ventricular function by increasing transmural pressures and therefore afterload. Whilst there may be symptomatic benefit in dyspnoea scores using non-invasive ventilation (NIV) such as continuous positive airway pressure (CPAP), there is no definite improvement in overall mortality.

Other Cardiac Causes of Acute Breathlessness

Myocardial Infarction (MI) [12, 13]

Myocardial infarction can also cause breathlessness, likely due to an element of tissue hypoxia. The key management step is identification of the type of myocardial infarction, either with ST segment elevation (STEMI) with high risk of transmural infarction or without ST segment elevation (non-STEMI or NSTEMI).

For a STEMI presentation, the key management step is prompt administration of antithrombotic medications (dual antiplatelet medication and heparin), together with prompt revascularisation. Primary percutaneous intervention (PPCI) has been reliably demonstrated to be superior to thrombolysis for revascularisation both in terms of physiologic indices, incidence of heart failure and mortality, as long as its timing is not too prolonged when thrombolysis is a valid alternative. In addition, in those with shock or pulmonary oedema, PPCI has added benefit.

Arrhythmias

Arrhythmias can lead to decreased cardiac output and tissue hypoxia leading to increased respiratory rate and a sensation of breathlessness. In addition, arrhythmias are also a cause of pulmonary oedema, which must be screened for during the management of acute pulmonary oedema (APO).

When a tachyarrhythmia is present, a low systolic blood pressure or pulmonary oedema requires rapid conversion [14] to sinus rhythm by DC cardioversion in many cases. If more time can be taken, identification of the arrhythmia as broad or narrow and identification of its rhythm will guide further medical therapy [14].

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