Acute breathlessness

Introduction

Acute breathlessness is terrifying for the patient and is a symptom often greatly feared by junior medical staff. The sudden development of dyspnoea heralds, in most cases, a significant cardiopulmonary insult. Left untreated, most causes of acute breathlessness can prove rapidly fatal. Thus, physicians presented with an acutely dyspnoeic patient must provide prompt supportive care, an accurate diagnosis and appropriate management of the underlying disease process.

This review discusses major causes of acute breathlessness, their diagnosis and initial management. Acute breathlessness is defined as severe dyspnoea evolving rapidly over minutes to hours. Sub-acute conditions developing over hours to days (e.g. pneumonia, metabolic acidosis, exacerbations of chronic obstructive pulmonary disease) are not discussed. Acute breathlessness is a condition encountered by all doctors. It is a major cause of inpatient and outpatient emergencies and is a frequent cause of emergency hospital admissions.

Initial assessment of the acutely breathless patient

Acute breathlessness is almost invariably the result of severe cardiac or pulmonary embarrassment (*Table 1*). Prompt supportive therapy is crucial in preventing further deterioration while clinical assessment and investigations are performed. The 'ABC' approach, advocated by the Resuscitation Council (2004), provides a simple effective method for providing initial care:

A Airway – should be clear and if necessary secured using a nasopharyngeal or oropharyngeal airway, laryngeal mask or endotracheal tube

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B Breathing – should be assessed and appropriate respiratory support provided, either with supplemental oxygen or, in severe cases, non-invasive or invasive ventilation

C Circulation – pulse and blood pressure should be recorded and urgent supportive treatment commenced as required.

In parallel, the physician must identify the cause of acute dyspnoea through focussed history taking, examination and key investigations.

Pulmonary oedema

Pulmonary oedema may arise from many causes (*Table 2*) but most commonly results from left ventricular failure. Patients may report profuse sweating, marked anxiety, the expectoration of pink frothy sputum and cardiac chest pain. On examination, patients with acute pulmonary oedema are grey, clammy, tachypnoeic, tachycardic and hypoxic. Peripheral pulses are often thready and difficult to palpate. The jugular venous pressure is elevated and on cardiac auscultation there is a gallop rhythm – care should be taken to listen for murmurs of valvular stenosis or incompetence. Crackles are audible throughout the chest. Frequently, wheeze – 'cardiac asthma' – is also audible and should not be mistaken for true asthma.

Acutely, the key investigation in pulmonary oedema is the chest X-ray, which classically demonstrates a bat's-wing appearance of diffuse, hazy alveolar infiltrate. Other X-ray signs include upper lobe venous distension, Kerly B lines and fluid visible in the horizontal fissure. The electrocardiogram (ECG) may show cardiac ischaemia or even acute myocardial infarction.

Table 1. Causes of acute breathlessness

Pulmonary oedema
Pulmonary embolism
Pneumothorax
Acute asthma attack
Airway obstruction
Psychogenic breathlessness 'panic attack'

Management involves placing the patient in a sitting position and providing high flow supplemental oxygen. Intravenous diamorphine acts as an analgesic, anxiolytic and venodilator. Intravenous diuretic therapy (furosemide or bumetanide) results in an early vasodilatory effect and provides a later diuresis. If the patient's blood pressure allows, a continuous nitrate infusion provides highly effective venodilation and thus reduction of cardiac preload. If these measures fail, continuous positive airway pressure (CPAP) ventilation by facemask improves outcome (Pang et al, 1998). In severe cases not responding to conventional therapy an intra-aortic balloon pump will reduce cardiac after load and improve coronary, cerebral and renal perfusion. If there is evidence of myocardial infarction, reperfusion therapy with thrombolysis or primary coronary angioplasty is imperative.

Table 2. Causes of pulmonary oedema

Cardiogenic	Left ventricular failure
	Tachyarrhythmias or bradyarrhythmias
	Mitral stenosis
	Myocardial ischaemia
	Atrial myxoma
	Acute mitral valve rupture
Drugs	Hydralazine
	Nitrofurantoin
	Intravenous contrast
	Aspirin
	Bleomycin
Inhalational injury	
Fluid overload, e.g. in renal failure or postoperatively	
Severe hypoalbuminaemia	
Neurogenic, e.g. post intracerebral bleed or head injury	
Severe myxoedema	
Acute respiratory distress syndrome	
High altitude	
Reinflation pulmonary oedema, e.g. post-drainage of pneumothorax or pleural effusion	
Renal artery stenosis	

Pulmonary embolism

The incidence of pulmonary embolism (PE) is approximately 70/100 000 (Oger, 2000), with half of cases seen in hospitalized individuals (Heit et al, 2001). Mortality from PE may reach 15% (Heit et al, 1999; Oger, 2000). The diagnosis of PE is notoriously elusive and a high index of suspicion is essential. Sudden onset breathlessness may be the only presenting symptom, although other symptoms include pleuritic chest pain, haemoptysis and symptoms of deep vein thrombosis. Following massive PE, circulatory collapse may occur resulting in syncope or cardiopulmonary arrest.

Bedside investigations are often diagnostically unhelpful. The ECG may show a tachycardia and signs of acute right heart strain (classically a dominant S wave in lead I, a Q wave and inverted T wave in lead III; SI, QIII, TIII). The chest X-ray is often normal but may show wedge-shaped consolidation, small unilateral pleural effusions or loss of vascular markings. D-dimer measurements, used in an appropriate clinical algorithm, can be useful in excluding the diagnosis but not in making a positive diagnosis. A PE can be confirmed by ventilation/perfusion scanning or by computed tomography (CT) pulmonary angiography. In massive PE, the finding of acute right heart strain in the absence of left ventricular failure on echocardiography can be considered diagnostic (Kasper et al, 1997).

Supportive treatment includes oxygen and analgesia and, in circulatory compromise, intravenous plasma expanders and inotropic agents. Massive PE causing acute right heart failure and circulatory collapse, carries a grave prognosis and should be treated with thrombolysis. The British Thoracic Society Standards of Care Committee (2003) guidelines recommend a 50 mg bolus of alteplase. In centres where the expertise exists, clot fragmentation via right heart catheter or surgical embolectomy are alternatives to thrombolysis. If non-massive PE is suspected, immediate anticoagulation with subcutaneous low molecular weight heparin or intravenous unfractionated heparin is necessary. Once the diagnosis is confirmed, an oral anticoagulant should be started.

Pneumothorax

Pneumothoraces may occur spontaneously or following trauma. Primary spontaneous pneumathoraces occur in patients with otherwise healthy lungs. Secondary spontaneous pneumothoraces occur in those with pre-existing lung disease. Large primary pneumothoraces may produce relatively few symptoms. However, patients frequently report sudden onset breathlessness, often preceded by sharp chest pain. Tension pneumothoraces, which may occur spontaneously, must be promptly identified and treated as they may rapidly lead to circulatory compromise and cardiac arrest.

The cardinal clinical findings of pneumothorax are reduced chest wall excursion, hyper-resonant percussion note and reduced or absent breath sounds on the side of the pneumothorax. Signs of a tension pneumothorax include worsening tachypnoea, tachycardia, hypoxia, hypotension and mediastinal shift. Chest X-ray is diagnostic (*Figure 1*).

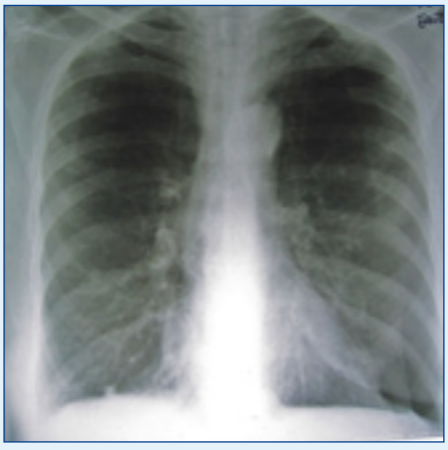
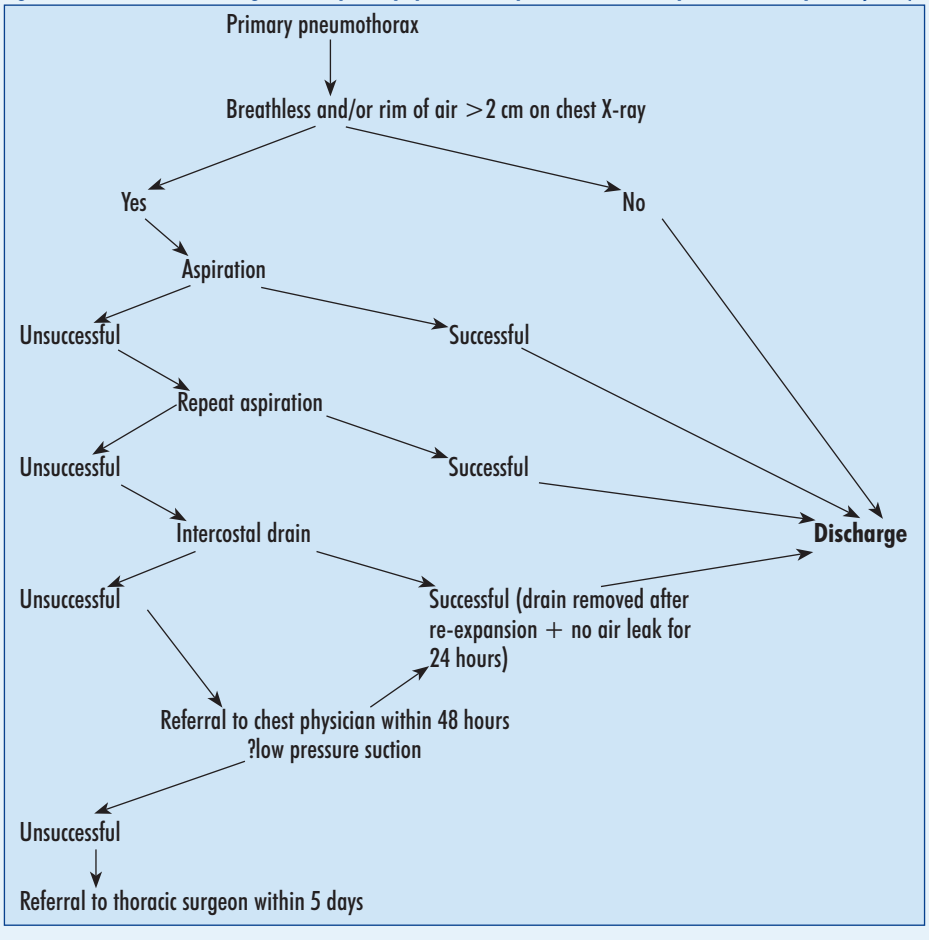


Figure 1. Antero-posterior erect chest X-ray of woman with known emphysema, presenting with acute breathlessness. X-ray shows a spontaneous left-sided pneumothorax.

A tension pneumothorax requires immediate decompression of the intrapleural space. This should be performed by the insertion of a large bore cannula into the second intercostal space in the mid-clavicular line. The patient should also receive high-flow oxygen. For the management of uncomplicated primary and secondary pneumothoraces the British Thoracic Society have provided useful algorithms (*Figures 2* and *3*).

Figure 2. Recommended management of primary spontaneous pneumothorax. Adapted from Henry et al (2003).



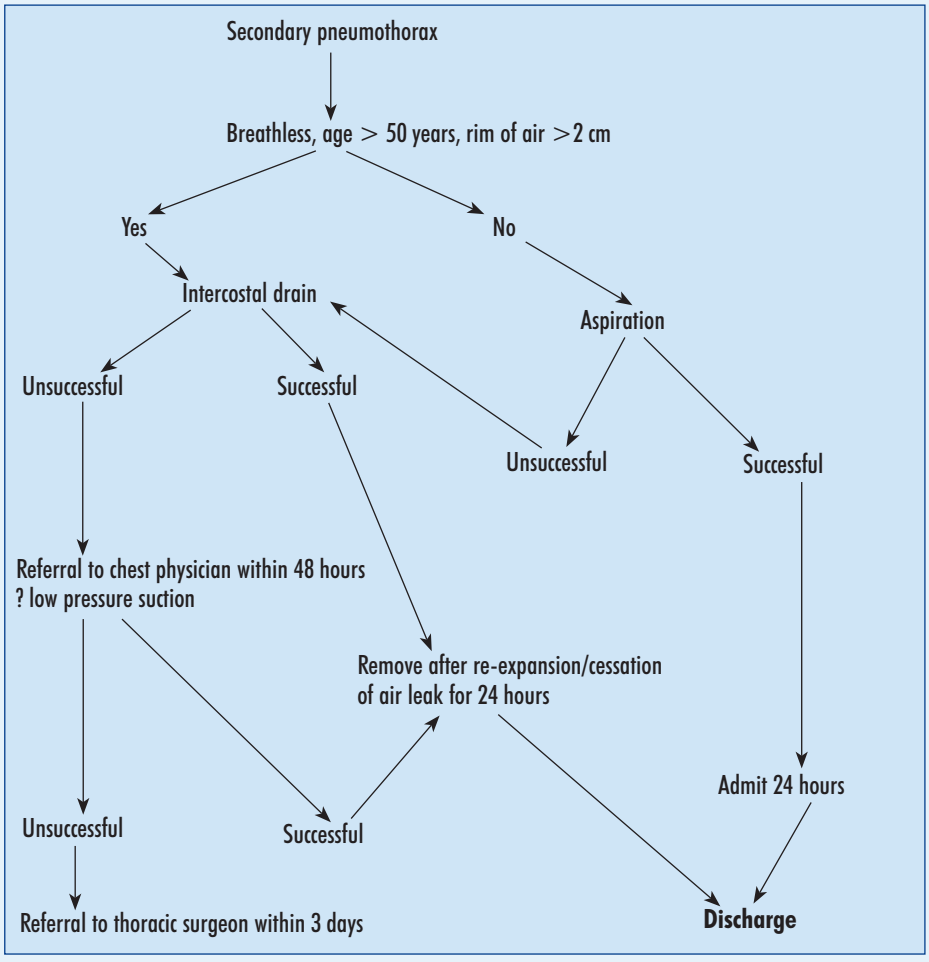


Figure 3. Recommended management of secondary spontaneous pneumothorax. Adapted from Henry et al (2003).

Acute asthma attack

Asthma causes over 1000 deaths in the UK each year (British Thoracic Society/Scottish Intercollegiate Guidelines Network, 2003). Deaths occur most frequently in chronic severe disease but can occur in even mild or moderate disease. Most severe attacks evolve over days, but a small number develop very acutely. It is important that treating physicians promptly identify patients at risk of fatal asthma and recognize severe and life-threatening attacks (*Table 3*).

In acute severe asthma, there is often a history of deteriorating disease control over preceding days or weeks. This usually results in nocturnal cough and an increased requirement for short-acting bronchodilators. Patients may have symptoms suggestive of viral or bacterial infection. On examination patients are tachypnoeic and tachycardic. Auscultation generally reveals diffuse polyphonic wheeze but in a life-threatening attack, the breath sounds may be quiet with an almost total absence of wheeze.

The chest X-ray excludes exacerbating factors (pneumonia, pneumothorax, lobar collapse). Blood tests frequently disclose an eosinophilia and, when infection is a trigger, a neutrophilia and increased inflammatory markers. Peak flow rates are reduced and provide an important tool for risk stratification.

Immediate management is dictated by severity. Severe asthma should be treated with high flow oxygen, nebulized short acting β2 agonist (terbutaline or salbutamol), nebulised ipratropium bromide (all nebulizations should be oxygen driven), and high-dose oral or parenteral steroid therapy. Continued deterioration should prompt the administration of intravenous magnesium. Intravenous aminophylline or salbutamol should be considered in severe cases, with awareness that both may trigger cardiac arrhythmias. Patients with severe or life-threatening asthma may require intubation and mechanical ventilation and should be managed in an environment where intubation can be performed rapidly (in a high dependency or intensive care unit). Other necessary therapies may include antibiotics and intravenous fluid and electrolyte replacement.

Large airway obstruction

Large airway obstruction may arise from many causes (*Table 4*) and often evolves gradually. Obstruction may occur suddenly, however, resulting in acute severe dyspnoea. Important antecedent features include trauma, aspiration and a history of tumour or strictures. Inspiratory stridor is highly suggestive of partial tracheal obstruction, but obstruction of major bronchi may manifest with non-stridorous breathlessness. Examination findings vary with the site of obstruction, but stridor (an inspiratory wheeze audible at the mouth) is a cardinal sign. Lobar collapse may result in reduced chest wall excursion and reduced breath sounds on the side of the collapse. The detection of acute, central airway obstruction depends on clinical acumen, as no readily available test (including the chest X-ray) is diagnostic.

Table 3. Levels of severity of acute asthma exacerbations

Near fatal asthma	Raised $PaCO_2$ and or requiring mechanical ventilation
Life-threatening asthma	Known severe asthma and any of: PEF < 33% predicted, SpO_2 <92%, PaO_2 < 8kPa, normal $PaCO_2$, silent chest, cyanosis, poor respiratory effort, bradycardia, hypotension, confusion, coma
Acute severe asthma	Any of PEF 33–50% predicted, respiratory rate>25/min, tachycardia > 110 bpm, or too breathless to complete sentences
Moderate asthma exacerbation	Patient with increasing symptoms, PEF > 50–75% predicted and no features of acute severe asthma

Adapted from British Thoracic Society/Scottish Intercollegiate Guidelines Network Guidelines (2003). $PaCO_2$ = partial pressure of arterial carbon dioxide; PaO_2 = partial pressure of arterial oxygen; PEF = peak expiratory flow; SpO_2 = arterial oxygen saturations

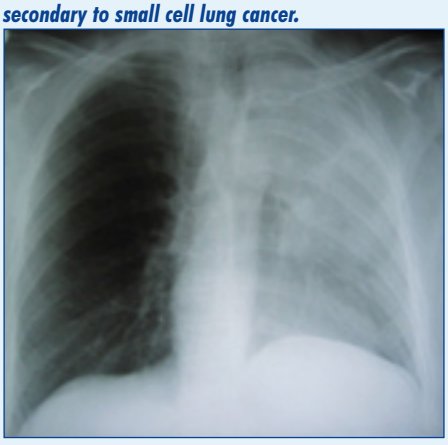
Table 4. Causes of airway obstruction

Aspiration	Blood
	Vomit
	Foreign body
Trauma	
Central nervous system depression	Obstructive sleep apnoea
	Coma
	Drugs, e.g. opiates, benzodiazepines
Epiglottitis	
Tumour (oropharyngeal, laryngeal, tracheal or bronchogenic)	
Stricture	
Extrinsic airway compression	Lymph nodes
	Goitre
	Mediastinal or thoracic outlet tumour
	Surgical emphysema
Laryngospasm	
Retained airway secretions or sputum plugging	

Obstruction of major bronchi with lobar collapse produces characteristic X-ray signs (*Figure 4*).

Treatment of airway obstruction depends on the site of the occlusion. Foreign body aspiration causing total airway obstruction can be cleared by use of the Heimlich manoeuvre. For obstruction above or just below the larynx, endotracheal intubation or, in severe cases, a tracheostomy, enables effective ventilation. Heliox, a mixture of helium and oxygen, decreases airway resistance, and can be an important early supportive treatment. Rigid bronchoscopy may be necessary to remove the cause of lower airway obstruction. In malignant disease, airway stenting can be useful in restoring airway patency. For airway obstruction caused by retained secretions, chest physiotherapy is crucial. Adjuncts to physiotherapy include nebulized bronchodilators, nebulized saline, oral N-acetyl cysteine, and the use of positive end expiratory pressure.

Figure 4. Chest X-ray of 74-year-old woman presenting with sudden onset dyspnoea and left-sided pleuritic chest pain. Chest X-ray demonstrates left upper lobe collapse – subsequently found to be secondary to small cell lung cancer.



Psychogenic breathlessness: 'panic attacks'

Psychogenic breathlessness can be extremely difficult to differentiate from other forms of acute breathlessness. This diagnosis should be made exclusively by an experienced physician after careful exclusion of organic disease. In a panic attack, symptoms are very real and can be terrifying. There is often no obvious trigger. Patients may report prominent anxiety associated with symptoms of hypocarbia (peri-oral tingling and numbness, tingling of hands and feet and light-headedness). Apart from tachypnoea and tachycardia, the examination is normal. Routine investigations are also normal except for arterial blood gas evaluation, which may demonstrate a respiratory alkalosis. It is important to note, however, that early acute asthma and small to moderate size PE can both produce a similar blood gas picture.

Treatment of a panic attack should include reassurance. Re-breathing of expired air, using a paper bag alleviates symptoms of hypocarbia. Benzodiazepines, used judiciously, can be effective in terminating a severe panic episode. The use of breathing control exercises can provide an effective longer-term management strategy for preventing recurrences.

Conclusions

Acute breathlessness typically arises as a consequence of serious cardiorespiratory disease. It is necessary when approaching the acutely dyspnoeic patient to adopt a rigorous approach to providing supportive care, rapidly establishing a diagnosis and instituting appropriate therapy. To do this a detailed knowledge of the causes, and treatment of, acute dyspnoea is crucial. BJHM

Conflict of interest: none.

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KEY POINTS

- Acute breathlessness frequently occurs as a consequence of life-threatening disease.
- Initial supportive therapy should be administered using the 'ABC' approach advocated for the provision of advanced life support.
- A diagnosis needs to be reached through focussed history taking and examination supplemented by the use of key bedside investigations.
- Rapid early treatment of the causes of acute breathlessness can avert cardiopulmonary arrest.