'I can't breathe'

Assessment and emergency management of acute dyspnoea

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BACKGROUND Acute dyspnoea is a distressing symptom that can suddenly affect any age group.

OBJECTIVE The article provides an approach to the identification of causes and initial management of acute dyspnoea based on history, physical assessment, appropriate triage and investigation.

DISCUSSION Successful management includes rapid assessment of the extent of the disease process, with emphasis on maintenance or protection of airway integrity and identification of impending respiratory failure. All general practice surgeries must have the appropriate equipment available, and staff trained in its use, to provide immediate relief to any patient who presents with dyspnoea. Urgent transfer from the general practice setting to a more specialised centre is usually necessary.

Dyspnoea literally means 'painful or difficult breathing', and is the sensation of 'air hunger'. It results from inequality between the energy expended by the patient and the physiological result of that exertion. However, because of a significant subjective component in some patients, the degree of dyspnoea can be hard to measure clinically. To overcome this variable, scoring tools have been developed to assist the clinician in the assessment.[1,2] Dyspnoea has many causes, but the majority of cases are due to either a cardiac or respiratory disorder (*Table 1*).

Tachypnoea means 'rapid breathing'. Hyperventilation is a state where the increased ventilation rate causes clinical hypocarbia. Hyperpnoea is increased ventilation without the production of hypocarbia. It should be noted that while hyperventilation and dyspnoea frequently coexist, this relationship is not absolute.[3]

Hypoxia (hypoxaemia) is a reduction of oxygen supply to tissues below their physiological needs. Acute hypoxia causes impaired judgment and motor incoordination and may mimic acute alcohol intoxication. When hypoxia is more longstanding, it produces fatigue, drowsiness, reduced work capacity and delayed reaction time. As it becomes more severe, brainstem centres are affected and death results from respiratory failure. The basic mechanisms of cause include decreased inspired oxygen concentration, inadequate ventilation, inadequate circulation (or a ventilation/perfusion mismatch), reduced oxygen capacity of the blood or cellular hypoxia in which cells are unable to utilise available oxygen (eg. cyanide poisoning). Details are outlined in standard medical texts.[4] Demonstrable clinical effects occur when the arterial partial pressure of oxygen falls below 60 mmHg (8 kPa) (*Table 2*).

The treating doctor/team must act decisively to provide relief to patients with dyspnoea. Treatment options may be simple, such as mechanical clearing of a partially obstructed airway, to advanced interventions such as assisted ventilation. Oxygen administration and pharmacotherapy is usually necessary in all cases.

Assessing the patient with acute dyspnoea

A detailed history can usually identify the process causing the dyspnoea. Many cases result from the worsening of, or complications of, chronic pathology of

Table 1. Causes of dyspnoea with brief clinical features

Airway	
– obstruction foreign body	Afebrile, stridor, choking
– angioedema	Oral swelling, airway obstruction, wheeze
– epiglottitis	Toxic, drooling, upright posture, soft stridor
– other infections, eg. diphtheria	Similar to epiglottitis
Lung	
Rapid onset of dyspnoea	
• Asthma	Hyperinflation, tachypnoea, wheeze
• Pneumonia	Fever, cough, haemoptysis, chest pain
• Croup (laryngotracheobronchitis)	Low grade fever, barking or brassy cough
• Bronchiolitis	Age less than 2 years, tachypnoea, cyanosis, wheeze
• Pulmonary contusion (see 'trauma')	Chest trauma, pain, anaemia
• Adult respiratory distress syndrome	Multifactorial, esp. sepsis
Slower onset of dyspnoea	
• Chronic obstructive pulmonary disease	Smoker, cough, wheeze, poor air entry
• Pneumoconiosis	Slow progression, history of exposure
Chest	
Rapid onset of dyspnoea	
• Pneumothorax – tension	Chest pain, tracheal deviation, hypotension
– simple	Chest pain +/- body habitus, past history
• Pleural effusion, haemo/pneumothorax	Features of the cause, stony dull percussion note
•Trauma – rib fractures	Pain, hypoxia
– flail chest	
Cardiac	
Rapid onset of dyspnoea	
• Congestive cardiac failure	Tachycardia, cough, fluid retention
• Acute pulmonary oedema	Pink frothy sputum, upright posture, anxiety
• Acute myocardial infarction	Chest pain, diaphoresis
• Cardiac arrhythmias	Palpitations, hypotension
Vascular	
Rapid onset dyspnoea	
• Pulmonary embolus	Pleuritic chest pain, collapse, deep vein thrombosis, risk factors
Slower onset dyspnoea	
• Pulmonary hypertension	Slow onset, past history, risk factors
Others	
Rapid onset of dyspnoea	
• Psychogenic hyperventilation	Past history, anxiety symptoms, circumoral and distal paraesthesia, tetany
• Poisoning, eg. carbon monoxide, cyanide	Precipitating causes, eg. exposure to fire, deliberate inhalation
• Metabolic acidosis	Features of precipitating cause, eg. diabetes
Slower onset of dyspnoea	
• Anaemia	Pallor, fatigue, evidence blood loss
• Guillain-Barre syndrome	Worsening ascending paralysis

Table 2. Assessment of respiratory function

Obstruction	Ventilation/perfusion mismatch	Increased work of breathing	Ventilation failure
Wheeze	Hypoxaemia	Tachypnoea	Hypercarbia
Hyperinflation	Central cyanosis	Failure of speech	Restlessness
	Tachycardia	Accessory muscle use	Flushing
	Arrhythmias	SOB and distress	Bounding pulse
		Upright posture	Exhaustion
		Sweating	Altered level of consciousness
		Pulsus paradoxus	Coma and death

which the patient is aware or insightful. Self diagnosis is therefore not uncommon. A useful clinical measure of dyspnoea is the (in)ability of the patient to speak in sentences, phrases or words. Important information can be gained from:

- the time course of the symptoms (sudden onset vs. days or longer)
- associated features such as chest pain, cough and sputum, haemoptysis, stridor, wheeze, orthopnoea
- the possibility of trauma or aspiration, and
- past history.

Evidence of recent infections and exposure to environmental provocation such as metal fumes and volatile gases should be explored. Medication compliance, particularly for underlying cardiovascular and respiratory conditions is important, as are possible drug interactions and known allergies.

Examination

Examination must include identifying imminent respiratory failure (*Table 2*). The well known mantra of '**A**irway, **B**reathing, **C**irculation' is a useful starting point and is often intuitively applied by the treating doctor. The general appearance of the patient is important (cyanosis, pallor, degree of distress). Vital signs (pulse, blood pressure, respiratory rate and pattern, and temperature) must be taken first in all cases as part of a thorough examination of the cardiovascular and respiratory systems. (Normal values for vital signs in children are shown in *Table 3*.)

Pulsus paradoxus, the detectable decrease in blood pressure of 10 mmHg or more with inspiration is an often overlooked but clinically important sign, which if present in asthma indicates significant airway obstruction. It also occurs in other states such as pericardial tamponade. In practice, it may be a difficult sign to elicit and interpret in tachypnoeic, tachycardic

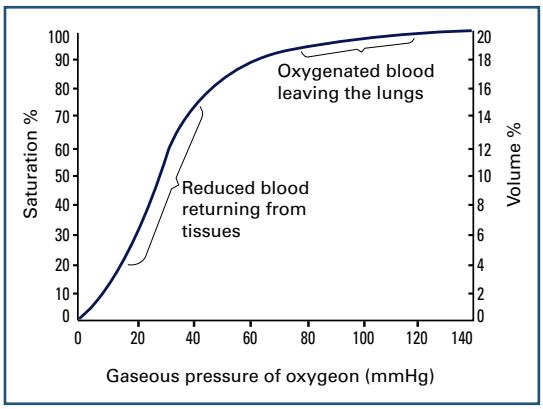

Figure 1. Oxygen dissociation curve

Case studies

Mr OM, aged 80 years

Mr OM tells you he has had increasing shortness of breath over 8 nights. He has been sleeping poorly. He wakes about 2.00 am with shortness of breath. He then gets panicky and is afraid to lie flat. He improves when he walks around the next day. He has little appetite but thinks he has gained weight. Mr OM's past history includes a pacemaker for complete heart block 10 years earlier and an elective endoluminal repair of an abdominal aortic aneurysm 1 year ago. He is a lifelong smoker. He takes perindopril for hypertension. A diuretic was recently ceased because of dizziness.

James B, aged 4 years

James' mother Susan brings James to see you from preschool. James began coughing badly during lunch and since has had coughing bouts in which he appeared to 'choke'. His teacher said he went 'a bit blue' in the face during the early choking episodes. He has an attack of coughing in your presence. The episode lasts only a few seconds but is very distressing to both child and mother. You inquire about James' lunch and find out that he had vegetable sticks and some salted peanuts 'as a treat'.

Susie J, aged 2 years

Susie has had a 'cold' for 3 days with nasal congestion and mild fever. She woke at midnight with noisy breathing and a barking cough. She seemed to have difficulty breathing.

From the above history, the working diagnoses would be cardiac failure for Mr OM, an inhaled foreign body for James, and an infective process for Susie, probably laryngotracheobronchitis.

and dyspnoeic patients.

Breath sounds may be equal, or unilateral indicating localised pathology. The details of respiratory auscultation should be known to all doctors and will not be discussed further here. However, some traps in examination need stating:

- wheeze is a poor indicator of disease severity
- reduction in wheeze may indicate an improvement or a worsening of symptoms
- a 'silent chest' is a grim finding in a sick respiratory patient as it implies poor air movement and imminent respiratory arrest[5]
- the acute appearance of cyanosis is a serious sign.

Tracheal deviation and subcutaneous emphysema are uncommon findings which, when found, have major clinical significance. Pursed lip breathing is seen in some adults and is an example of the patient subconsciously applying positive end expiratory pressure to enhance oxygenation. It is usually associated with accessory muscle use and indicates severe pre-existing respiratory disease. Grunting in expiration and nasal flaring is the paediatric equivalent and has similar serious implications.[6]

Pulse oximetry is the so-called 'fifth vital sign'. Its interpretation relies on knowledge of the oxygen dissociation curve (*Figure 1*). It is a noninvasive indicator of the body's arterial oxygen saturation but does have limitations. For example, it does not give any information about the carbon dioxide (CO2) status of the patient. Also, it can give a normal reading in some acute dyspnoeic states (eg. carbon monoxide poisoning) and is affected by motion artefact (eg. tremor), peripheral vasoconstriction and hypothermia.

By this stage, it should be possible to place the patient in a triage category, which will indicate the level of urgency of the response required[7] (*Table 4*).

Investigations

Time may not permit the full use of investigations if over-riding clinical considerations mandate early intervention. However, the so called 'bedside tests' are powerful tools which can be applied quickly and should be readily to hand in all surgeries. They include peak expiratory flow rate estimation and electrocardiography. Spirometry can also be helpful but may not be appropriate in the setting of severe acute dyspnoea.

Chest X-ray is required in the majority of dyspnoea cases. Exceptions in the general practice setting include a known asthmatic who is well after initial bronchodilator therapy. Special views may sometimes be warranted including lateral decubitus, lordotic chest films, and soft tissue images of the lateral neck. Expiratory chest films have limited but specific indications (*Table 5*).

Haematology and biochemistry to test renal function

Table 3. Normal values for vital signs in children

Age	Weight (kg)	Minimum systolic BP (mmHg)	HR	RR
Term	3.5	50	100–170	40–60
3 months	6	50	100–170	30–50
6 months	8	60	100–170	30–50
1 year	10	65	100–170	30–40
2 years	13	65	100–160	20–30
4 years	15	70	80–130	20
6 years	20	75	70–115	16
8 years	25	80	70–110	16
10 years	30	85	60–105	16
12 years	40	90	60–100	16
14 years	50	90	60–100	16
17+ years	70	90	60–100	16

Source: Royal Children's Hospital, Melbourne. 'Resus card'. Reprinted with permission. Available at www.rch.org.au/clinicalguide/forms/resuscard.cfm

Table 4. Australasian Triage Scale

Category	Treatment acuity	Examples
1	Immediate	Cardiac/respiratory arrest
2	10 minutes	An acute asthmatic speaking only in words
3	30 minutes	An acute asthmatic speaking only in phrases
4	60 minutes	An acute asthmatic speaking in sentences
5	120 minutes	

Treatment acuity = maximum acceptable waiting times. Note: the ATS has been developed for hospital based practice to indicate clinical urgency. Its relevance to general practice has not been established, but as it is a clinical tool first and foremost, it is included here as a guide for the GP

Table 5. Indications for expiratory chest X-ray

- Suspected pneumothorax not seen on conventional inspiratory chest X-ray
- Suspected inhaled radiolucent foreign body that may produce distal lobar or segmental hyperinflation. The comparison with an inspiratory film may reveal the air trapping

Case studies continued

Mr OM

Examination reveals a tachycardia of 106 bpm, BP 115/75 and a respiratory rate of 20. The apex beat is in the fifth space but displaced beyond the midclavicular line. Auscultation of the chest reveals bibasal inspiratory crackles. His jugular venous pressure is not elevated and there is no peripheral oedema.

James B

James is distressed but alert with no evidence of cyanosis. During coughing episodes you note rib recession. Between episodes, James is breathing rapidly with a respiratory rate of 30 and soft expiratory stridor. His temperature is 36.6°C, heart rate is 100 bpm and his BP is 95/60 mmHg. Breath sounds are equal and normal.

Susie J

Susie is flushed but does not look ill. She is not drooling. When crying or distressed you note a harsh biphasic stridor with rib recession but this settles as she calms down. Susie is febrile (38.2°C), with a respiratory rate of 34 and a heart rate of 120 bpm. You feel that it is safe to examine her throat and you will be able to do so without distressing her further. She has an injected throat. The chest sounds clear.

Table 6. Capacity of standard oxygen cylinders and devices

C cylinder	D cylinder	E cylinder	G cylinder
440 L	1500 L	3800 L	7600 L

Device	Oxygen concentration (FiO2)	Oxygen flow/minute
Nasal prongs	24–30%	3 L
Hudson mask	30–40%	6–10 L
Reservoir (nonre-breathing) mask	60–80%	12–15 L
AMBU bag	80–100%	15 L

FiO2 = fraction of inspired oxygen. Room air has an FiO2 of approximately 0.21 or 21% (20.9%). Note: FiO2 does not give any indication of oxygen saturation which requires pulse oximeter readings, or of partial pressure of oxygen in arterial blood which requires blood gas estimation

and electrolyte status are applied as necessary.

Arterial blood gas estimation is used to assess disorders of gas exchange. It offers important information on the acid/base (CO2) status of the patient. It is not readily available at the bedside and requires sophisticated equipment, although recent advances in technology and miniaturisation have made it more accessible. It is an invasive and painful procedure and is associated with morbidity. It too can be normal in the presence of certain pathology.

More complex testing may sometimes be needed including:

- blood and sputum culture and sputum microscopy
- computerised tomography (CT) of the chest with enhancements such as CT pulmonary angiography and contrast studies
- cardiac stress testing, echocardiography and variations such as trans-oesophageal echocardiography
- formal lung functions including provocation testing
- sweat tests, and
- procedures such as laryngoscopy and bronchoscopy.

Management

Oxygen therapy is necessary to reverse hypoxia, aiming to achieve saturations of 92% or above using an oxygen delivery device such as a face mask or nasal prongs (*Table 6*). Some form of airway support may also be required. In conscious patients, this can take the form of noninvasive ventilation such as continuous positive airway pressure as used for acute pulmonary oedema. An obtunded patient on the other hand, may require assisted ventilation via a portable hand held bag-valve-mask unit such as an AMBU bag, laryngeal mask airway, or by formal endotracheal intubation. Obviously these and other advanced modalities are not generally available outside the hospital setting, and when their use is indicated, early transfer of the patient to an appropriate facility is essential. (It should be noted that in some Australian states, selected ambulance service officers are trained and competent to insert the laryngeal mask airway and perform endotracheal intubation under strict clinical indications).

Oxygen delivery can become a complex exercise for the subset of patients with respiratory disease who are chronic retainers of CO2. In the hospital setting, controlled oxygen therapy with a venturi type mask is used, starting with a concentration of 24% and checking blood gases after 30–60 minutes. The exercise becomes even harder for the general practitioner with limited access to investigations such as those in rural or isolated practice. However, in principle, oxygen should not be denied to a hypoxic patient. Details are available in standard texts.[8]

Pharmacological adjuncts such as bronchodilator medication, diuretics, and H1 and H2 antagonists are administered according to the clinical indication and in approved dosages.

Continuous monitoring of the patient's vital signs and general condition is essential until a definitive management and disposition plan has been formulated.

Case studies continued

Mr OM

An electrocardiogram (ECG) reveals left ventricular hypertrophy. You diagnose left ventricular failure with incipient pulmonary oedema. Treatment commenced along conventional grounds and he is admitted to hospital.

James B

A chest X-ray taken in expiration supports the diagnosis and a peanut is removed from his right main bronchus at urgent bronchoscopy.

Susie J

The history suggests a diagnosis of croup rather than epiglottitis. You give her a dose of oral corticosteroid and arrange transfer to the local children's hospital for observation and further treatment.

Equipment

All general practice surgeries must have appropriate equipment on hand and clinical staff competent in its use. All surgery staff should be current to perform basic life support and cardiopulmonary resuscitation. Details of local resources such as children's and adult hospitals, specialist services including radiology and pathology, ambulance communication centres, and poison information centres must be known to all staff. Making these phone numbers part of the surgery speed dial list is good practice. Surgery equipment should ideally include:

- oxygen supply and delivery masks (*Table 6*)
- bag-valve-mask unit
- suction source and suckers (soft and rigid)
- intravenous equipment: cannulae, giving sets, intravenous fluids (N saline)
- spirometer, peak expiratory flow rate (PEFR) meter
- ECG (monitor and defibrillator optional)
- relevant pharmacological agents in appropriate formulations including salbutamol, ipratropium, adrenaline, steroids, frusemide, nitrates, morphine, and
- pulse oximeter (optional).

Conclusion

The extent of the dyspnoea must be assessed and the degree of hypoxia and respiratory failure (if any) determined – initially on clinical evidence. Dyspnoea of sufficient severity to affect respiratory function is always associated with clinical signs and symptoms referrable to the cardiovascular and/or pulmonary systems. Time may not permit the full use of investigations if overriding clinical considerations mandate early intervention. Oxygen therapy is necessary to reverse hypoxia and continuous monitoring of the patient's vital signs and general condition is essential until a definitive management and disposition plan has been formulated. All general practice surgeries must have equipment available and staff trained in its use to provide immediate relief to any patient who presents with dyspnoea.

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