I CAN'T BREATHE

DETECTING DETERIORATION, EVALUATION, TREATMENT, ESCALATION, AND COMMUNICATING IN TEAMS

2ND EDITION 2009

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AIM

The aim of this Chapter is to give you the knowledge to:

- understand the causes of breathlessness, cyanosis and hypoxia;
- understand the significance of oxygen therapy and pulse oximetry;
- understand the simple principles of underlying pathophysiology that lead to breathlessness.

Early Warning Signs 	
Pulse rate 40–49 or 121–140/min	SpO_2 90–95%
Alteration in mentation	PaO_2 50–60 mmHg
Partial airway obstruction	$PaCO_2$ 50–60 mmHg
RR 5–9 or 31–40 breaths/min	pH 7.2–7.3

Table 7

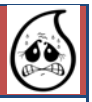
Late Warning Signs 	
*Pulse rate <40 or >140/min	PaO_2 <50 mmHg
*Unresponsive to verbal command	$PaCO_2$ >60 mmHg
*Airway obstruction/stridor	pH <7.2
*RR <5 or >40 breaths/min	* Failure to reverse variable within 1hr
SpO_2 < 90%	

Table 8 ** = common MET call criteria*

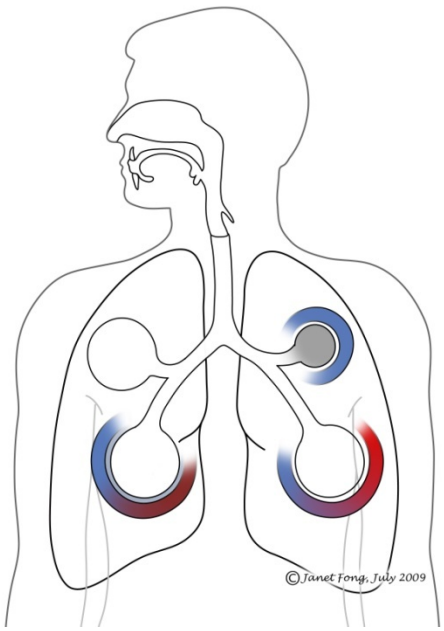
Chapter One has given you the knowledge for rapid and systematic assessment of the airway and breathing. Simple manoeuvres to clear the airway are provided in the BLS and ALS algorithms and Airway Resuscitation chapter (Chapter Nine, p 91).

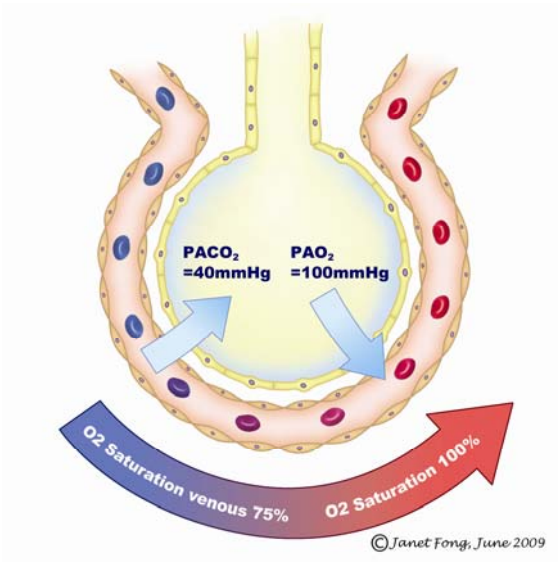
SIMPLE PATHOPHYSIOLOGY

The physiological importance of our practical approach to a patient who is having trouble breathing is to ensure oxygenation of the blood and elimination of carbon dioxide from the blood.

There are four important causes of low oxygen levels in the arterial blood (measured via arterial blood gases as PaO_2 in mmHg). They are:

1. Hypoxia
2. Hypoventilation
3. Ventilation and perfusion mismatch
4. Diffusion abnormalities (uncommon).





This is a practical approach based on the signs and symptoms of hypoxia and cyanosis and pulse oximetry abnormalities. An understanding of the Alveolar Gas Equation and Oxygen Haemoglobin Dissociation Curve will help you monitor and treat hypoxia and respiratory failure. The other physiological equation often discussed in this context is the Shunt Equation which is beyond the scope of this programme.

The Alveolar Gas Equation describes how the partial pressure of oxygen in the alveoli (P_AO_2) changes with the $PaCO_2$ (partial pressure of CO_2 in the arterial system) and the FiO_2 (fraction of inspired

oxygen). The P_AO_2 is closely related to the PaO_2 (partial pressure of oxygen in the arterial system). It should help you understand why hypoventilation and rising $PaCO_2$ leads to hypoxaemia and why taking oxygen off for such things as doing a blood gas can precipitate profound hypoxia. PiO_2 refers to the partial pressure of inspired oxygen.

ALVEOLAR GAS EQUATION

$$P_AO_2 = PiO_2 - \frac{PaCO_2}{0.8}$$

Where the $P_iO_2 = Fi0_2 (P_b – P_{H20})$

Therefore, at sea level, breathing room air (FiO_2 0.21 with a normal $PaCO_2$ of 40 mmHg):

- $P_AO_2 = 0.21(760\text{-}47)–40/0.8 \cong 100$ mmHg
- If the $PaCO_2$ doubles to 80 mmHg:

  $P_AO_2 = 0.21(760–47)$ to $80/0.8 \cong 50$ mmHg

Any increase in the $PaCO_2$ will reduce the alveolar oxygen concentration and lead to hypoxaemia. The higher the $PaCO_2$ rises the lower the P_AO_2 falls and hence the $Pa0_2$ falls.

OXYGEN HAEMOGLOBIN DISSOCIATION CURVE

The oxygen haemoglobin dissociation curve (Figure 1) describes the relationship between oxygen saturation (the amount of oxygen bound to haemoglobin as a percentage of maximum haemoglobin oxygen carrying capacity) and PaO_2 (partial pressure of oxygen in the arterial system). Normally at a PaO_2 of 85 mmHg haemoglobin is 95% saturated and for further increases in PaO_2 the amount of extra oxygen carried by haemoglobin is not significant (the flat part of the sigmoid shaped dissociation curve). As the PaO_2 falls, haemoglobin releases oxygen and binds it less readily. Below an oxygen saturation of 90% the slippery slope of the curve occurs and a sharp fall in oxygen saturation will occur with small decreases in PaO_2. The ability of haemoglobin to bind and release oxygen changes in certain circumstances. If there is a lower pH (acidosis) or higher $PaCO_2$ or fever for example, more oxygen is released by haemoglobin and it binds oxygen less readily, i.e. the curve shifts to the right. So oxygen delivery to the tissues depends on: oxygen supply and respiratory function; haemoglobin concentration and its ability to bind and release oxygen; and cardiac output.

RELATIONSHIP BETWEEN SAO_2 & PAO_2 (AT 37 DEGREES)

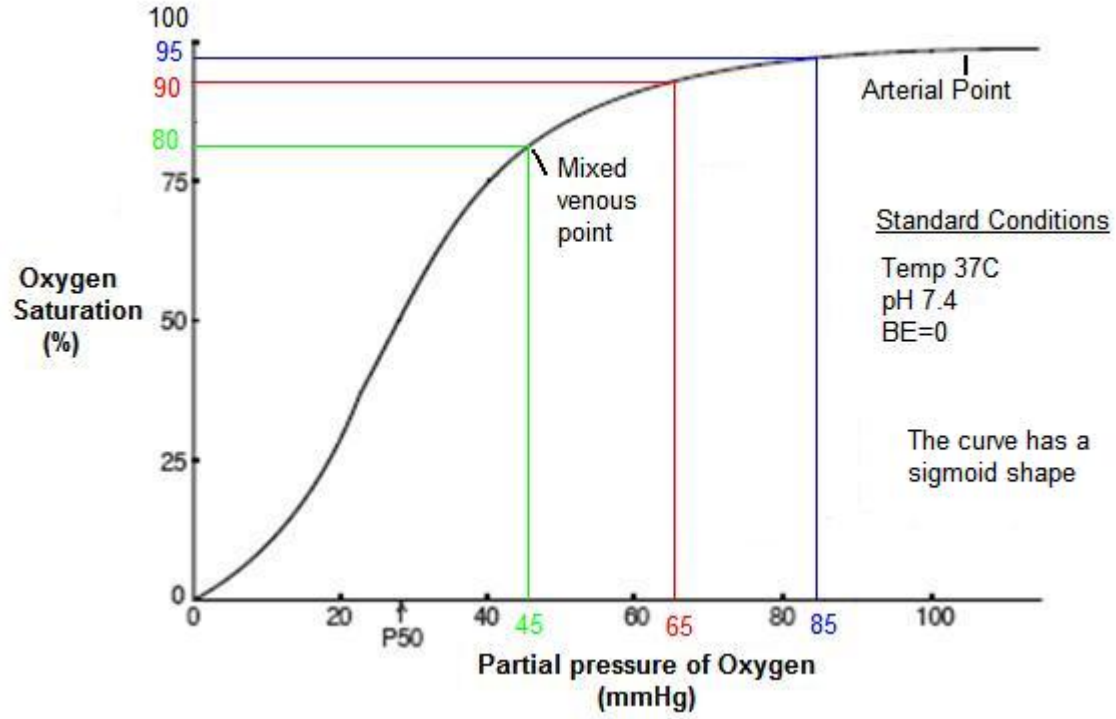


Figure 1: Oxygen Haemoglobin Dissociation Curve

ASSESSMENT OF RESPIRATORY FAILURE

Respiratory failure can be divided into hypoxaemic (PaO_2 ≤60 mmHg) and hypercapneic ($PaCO_2$ ≥50 mmHg). Deteriorating patients may have acute or acute-on-chronic lung disease. The relevant early and late signs of *When to Worry* are provided at the beginning of this chapter (Tables 3 and 4). When making your clinical assessment of the respiratory system, start with the airway patency as per the ABCDEFG algorithm. Determine the type, cause and severity of the respiratory failure and you may have to initiate respiratory treatment prior to continuing through the ABCDEFG algorithm.

BREATHLESSNESS

Breathlessness is a symptom of respiratory crisis. Breathlessness can have various causes. These are presented in a systematic way to help you come to the potential diagnosis when your patient becomes breathless. The patient's respiratory rate will go up, they may appear laboured in their breathing, have difficulty speaking, have signs of using accessory respiratory muscles and they may change their posture to make breathing easier (e.g. sitting up and leaning forward). Nasal flaring, intercostal recession, suprasternal and supraclavicular retraction are signs of significant increase in respiratory effort.

Beware of hypoxia and breathlessness as a cause in the confused and combative patient.

CAUSES OF BREATHLESSNESS INCLUDE:

1. Increased ventilatory demands
    - Hypoxaemia
    - Hypercapnia
    - Acidosis
    - Anaemia
    - Pulmonary embolus
    - Pulmonary oedema
    - Exercise.
2. Decreased ventilatory capacity
    - Thoracic deformity
    - Pneumothorax
    - Airflow obstruction such as asthma and exacerbation of COPD
    - Pleural effusion
    - Pneumonia
    - Respiratory muscle weakness
    - Fractured ribs/sternum.
3. Increased subjective sensitivity
    - Anxiety
    - Hysteria.

SIMPLE MEASURES FOR BREATHLESSNESS:

- Sit the patient up
- Reassure them
- Give supplemental oxygen
- Consider bronchodilators
- Consider opiates to relieve severe dyspnea or pain
- Call for help if not improving

All patients who are acutely unwell with breathlessness or hypoxia should have supplemental oxygen.

CYANOSIS

Cyanosis is blue discolouration of the skin, lips or nail bed. It is secondary to low oxygen tension in arterial blood (PaO_2). It occurs in two forms, central and peripheral. It is subject to observer error and a range of patient factors.

CENTRAL CYANOSIS

- Blue discolouration of the lips.
- Requires more than 5 g/dL deoxygenated haemoglobin in capillary blood.
- Usually detectable when SaO_2 <85%.

Common conditions which may cause central cyanosis

Acute	Chronic
• Pneumonia • Acute asthma • Pulmonary oedema • Pneumothorax	• COPD • Pulmonary fibrosis • R -> L cardiac shunt • Polycythaemia

PERIPHERAL CYANOSIS

- Blue discolouration of the fingers, toes and nail beds.
- Occurs with adequate oxygenation of circulating blood but abnormalities of local circulation in the extremities.
- May indicate hypovolaemia, low cardiac output state, arterial or venous obstruction, or simply exposure to cold!

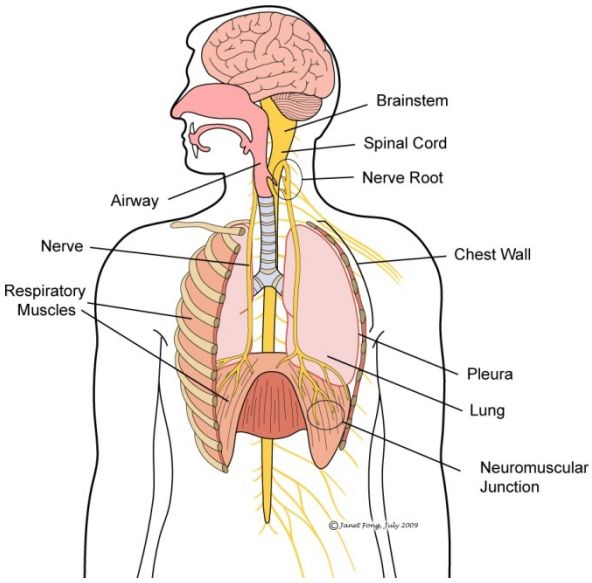
Central Cyanosis (blue lips) and Peripheral Cyanosis (blue fingertips)

HYPOVENTILATION

The cardinal feature of hypoventilation is hypercapnia. This is because elimination of CO_2 from the body depends on adequate minute ventilation. (Minute Ventilation = RR x Tidal Volume). Counting the Respiratory Rate is an important and simple clinical monitor. Progressive hypoventilation leads to hypoxaemia (unless FiO_2 is increased) as the oxygen is being used but not replaced and the CO_2 is rising. Flap (rhythmic movement of hands with arms outstretched), bounding pulses, hypertension and peripheral vasodilation can indicate hypercapnia.

CAUSES OF HYPOVENTILATION

- Airway obstruction
- Severe chronic obstructive lung disease
- Central Nervous System (CNS) depressant drugs
- Sedatives, opiates, anaesthetic agents, antidepressants and others
- CNS disease
- Head injury, meningitis, stroke, encephalopathy
- Peripheral neuromuscular disease, e.g. myasthenia gravis, muscle relaxant
- High spinal cord injury
- Chest wall disease: kyphoscoliosis, ankylosing spondylitis
- Exhaustion
- Severe pulmonary fibrosis.



TREATMENT OF HYPOXAEMIA DUE TO HYPOVENTILATION

- Give oxygen in sufficient concentration to raise PaO_2 above 60 mmHg (SaO_2 >90%)
- Provide assisted ventilation using a manual resuscitator
- Reverse any drug-induced CNS depression
- Call for help
- Position the patient to improve ventilation (this will depend in part on conscious state)
- Consider non-invasive or invasive ventilation
- Do not remove or reduce oxygen therapy.

VENTILATION-PERFUSION MISMATCH

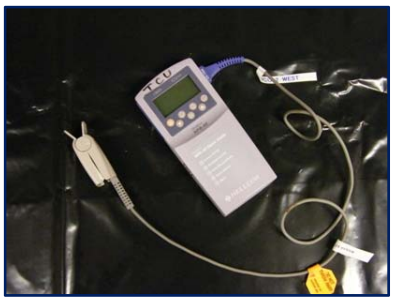
Ventilation-perfusion mismatch occurs when the alveolar ventilation and pulmonary blood flow to the alveoli do not match. There are several permutations possible. Either decreased ventilation with normal blood flow, or normal ventilation with reduced blood flow, or absent blood flow to ventilated areas of the lung (increased physiological dead space), or pulmonary blood flow without exposure to ventilated alveoli (shunt). The commonest causes of hypoxaemia with ventilation-perfusion mismatch in patients with acute respiratory failure are:

- Pneumonia
- Pulmonary oedema
- Severe acute asthma
- Pulmonary embolism (increased dead space)
- Chronic obstructive lung disease
- Pulmonary haemorrhage
- Adult Respiratory Distress Syndrome

- Anatomical shunting due to intracardiac shunts (e.g. Fallot's tetralogy)
- Hypovolaemia and low cardiac output, which can cause increased physiological deadspace.

Management of ventilation-perfusion mismatch depends on the underlying cause. Use the ABCDEFG algorithm and DETECT assessment to determine the cause and early treatment. Oxygen therapy should be applied. In general, ventilation-perfusion mismatch and hypoventilation will be temporarily improved by sitting the patient up.

PULSE OXIMETRY



Pulse oximetry is a widely used monitor in the wards. It measures oxygen saturation and pulse rate by placing a detector probe on the patient's finger tip, ear lobe or bridge of the nose. Look for a good wave form on the monitor when applying the probe. It is a convenient way of measuring arterial oxygen saturation continuously and non-invasively. It removes the observer error in detecting cyanosis.

Pulse oximeters work by measuring the differential absorption of two wavelengths of light by oxyhaemoglobin and total haemoglobin. The waveform on the monitor is pulsatile and reflects arterial absorption so it is subject to errors when there is poor peripheral perfusion and movement. It is unreliable if the patient is very cold, has nail varnish, is shivering or having seizures. Fluctuations in ambient light can give false pulsatile signals. Methaemoglobin causes error, carboxyhaemoglobin (caused by carbon monoxide poisoning and heavy smoking) causes the oximeter to over read. However even with these limitations pulse oximetry is a useful measure of systemic oxygenation. We know from the *When to Worry* early and late signs that an oxygen saturation less than 90% is associated with increased risk of cardiac arrest and death. At less than 90% a small fall in PaO_2 results in a large fall in SaO_2. (Note the slippery slope of the oxygen dissociation curve Figure 1, p 23, 90% oxygen saturation and a PaO_2 of 60 mmHg).

Normal oxygen saturation value is 98–100%. Apply oxygen if saturation is less than or equal to 97%.

Other caveats: Pulse oximetry gives **no** information regarding adequacy of ventilation and is not helpful in detecting hypoventilation, cardiac output, blood pressure, or cardiac rhythm and should **not** exclude the use of others simple monitors such as counting respiratory rate, using an ECG trace and blood pressure measurement and measuring ABGs.

Remember, the force or gradient driving oxygen from the lung to the tissues depends on partial pressure, and not saturation. Do not dismiss borderline low SaO_2 levels (90–96%) and assess the patient to exclude hypoventilation, rising $PaCO_2$ and eventually falling PaO_2. The fall in oxygen saturation in this case is a **very late** sign of hypoventilation, especially if the patient is receiving oxygen therapy.

OXYGEN THERAPY

- Most patients with respiratory distress can safely be given high-flow oxygen.
- Aim for SaO_2 of at least 97%.
- The cardinal rule is frequent clinical re-evaluation, including blood gas analysis if appropriate.
- Consider ventilator assistance, e.g. BiPAP to maximise effectiveness of oxygen therapy.

RISING $PACO_2$ WITH OXYGEN THERAPY

In patients with a breathing problem, if the $PaCO_2$ was initially normal (or more commonly, low) and rises with oxygen therapy, this suggests exhaustion, and the patient cannot sustain the work of breathing. Reduction of inspired oxygen at this stage, thinking the ventilatory drive will increase, could lead to worsening of hypoxaemia and cardiorespiratory arrest. The treatment is ventilatory support, not reduction or removal of oxygen therapy, and you should call for help.

OXYGEN DELIVERY SYSTEMS

Oxygen supply systems can be divided into variable and constant performance systems. By performance we refer to the actual oxygen concentration that the patient will end up receiving, i.e. the inspired oxygen fraction or FiO_2 often expressed as a percentage of inspired oxygen concentration. The variable performance systems are most widely used in the ward setting. Be familiar with the performance of each of the devices. You will need to prescribe or dial up a fresh oxygen gas flow for each device you choose. The ability of the device to match the patient's inspiratory flow requirements will determine how much air is entrained and therefore how much dilution of the inspired oxygen with entrained air occurs. Hence the device is *variable* in its performance.

VARIABLE PERFORMANCE SYSTEMS

- **Nasal cannulae**

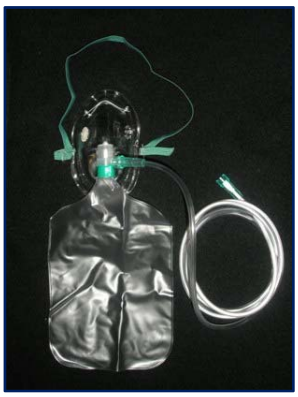
  2–4 L/min is all that can be tolerated without causing nasal mucosal drying, irritation and damage. At these low flows nasal cannulae are usually well tolerated. The maximum tracheal oxygen concentration is variable and usually <40% (0.4).

- **Simple facemasks**

The most common mask is called the Hudson mask. This is a vented enclosed mask applied over the nose and mouth. Air is entrained with each inspiration according to each patient's inspiratory gas flow requirements and hence the air oxygen mix varies and thus the FiO_2. Use of Hudson masks with too low flow (<5 L/min) can result in rebreathing of CO_2 and increasing $PaCO_2$. If the mask is fogging this suggests you need a higher fresh oxygen flow rate. At 4–10 L/min the FiO_2 can be 0.35 to 0.55 but this is variable and depends very much on the patient's inspiratory flow requirements.

- **Non-rebreathing facemask**

This has a reservoir bag attached to it which is filled with oxygen. The delivered oxygen flow rate is adjusted so that the bag remains distended throughout the respiratory cycle. Some air is entrained as the seal is not tight. At 15 L/min the FiO_2 is about 0.6–0.7.



FIXED PERFORMANCE SYSTEMS

- **Venturi masks**

These can provide 24%, 28%, 31%, 35%, 40%, and 50% oxygen according to the size of the venture and required oxygen flow. They are colour coded. They allow much higher total fresh gas flows.

Venturi masks can provide sufficient (high) gas flow to meet patient's minute ventilation. The FiO_2 depends on fixed ratio of air entrainment, at high flow, determined by venturi valve and oxygen flow. You need to read the required gas flow usually written on the side of the venture device or the package insert.

- **CPAP & Bi-level Systems**

CPAP refers to continuous positive airway pressure delivered by a machine throughout the respiratory cycle. BiPAP refers to bi-level positive pressure support, where the machine delivers two levels of positive pressure, a higher inspiratory pressure support and lower pressure during expiration increasing functional residual capacity. It serves to reduce work of breathing and improve ventilation-perfusion matching. It increases tidal volume and minute ventilation. CPAP can be used for patients with low lung volumes such as alveolar collapse, pulmonary oedema (cardiogenic or non-cardiogenic), pneumonia and atelectasis.

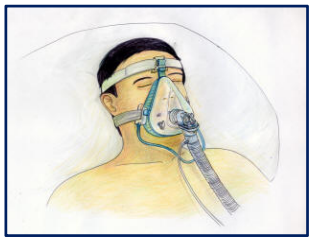


Figure 1: BiPAP Face Mask

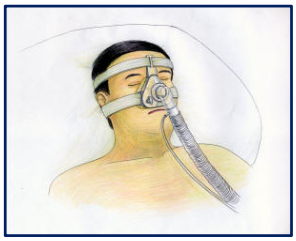


Figure 2: BiPAP Nasal Mask

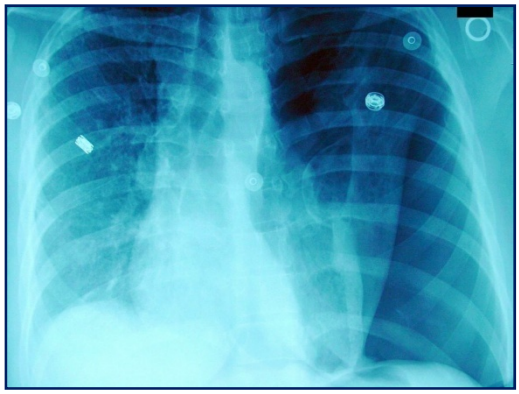
CPAP and BiPAP via face or nasal masks:

- Provide positive pressure to the airway throughout the respiratory cycle
- Increase functional residual capacity and recruit collapsed alveoli
- Prolong time for gas exchange to take place
- Shift position on Pressure:Volume curve at end-expiration and improve lung compliance
- Reduce work of breathing
- Reduce alveolar-arterial oxygen gradient
- Provide high flow oxygen and allow administration of 100% oxygen (FiO_2 = 1.0)
- CPAP will improve oxygenation (PaO_2), but not (in general) ventilation (i.e. $PaCO_2$)
- Both modes have haemodynamic effects that may be beneficial in cardiac failure.

The disadvantages are that CPAP/BiPAP can hide the severity of the patient's condition, discourage coughing and clearing of sputum, cause gastric distension and increase risk of aspiration. The patient can find the mask claustrophobic and uncomfortable. Pressure areas can occur with tight masks. A beard or facial trauma can make application difficult. A decreased conscious level (a decreased GCS) or oesophageal surgery contraindicate use.

If you think a patient requires BiPAP or CPAP you should call for help.

TENSION PNEUMOTHORAX



Tension pneumothorax is the accumulation of air under pressure in the pleural space. This condition develops when injured tissue forms a one-way valve, allowing air to enter the pleural space and preventing the air from escaping naturally. The lung collapses. Arising from numerous causes, this condition rapidly progresses to respiratory insufficiency, cardiovascular collapse, and, ultimately death if unrecognised and untreated. So look, listen and feel for signs of respiratory distress and cardiovascular deterioration. Favourable patient outcomes require prompt diagnosis and management.

AETIOLOGY

- Trauma
- Acute Respiratory Distress Syndrome
- Central venous cannulation
- Tracheostomies
- Laparoscopic surgeries
- Barotrauma (maybe related to high pressure ventilation).

The most common etiologies are either iatrogenic or related to trauma.

SIGNS AND SYMPTOMS

- **Early**
  - Chest pain
  - Dyspnea, breathlessness
  - Tachypnoea
  - Tachycardia
  - Hyper-resonance of the chest wall on the affected side
  - Diminished breath sounds on the affected side.
- **Late**
  - Decreased level of consciousness
  - Tracheal deviation toward the contralateral side
  - Hypotension
  - Distension of neck veins (may not be present if hypotension is severe)
  - Cyanosis.

DIAGNOSIS

The diagnosis should be suspected in any patient who has a deterioration post procedure or a condition which has the possibility to progress to tension pneumothorax, e.g. central venous cannulation.

If the patient is extremely unwell (e.g. with severe hypotension and cyanosis) then clinical diagnosis based on signs and symptoms should be established quickly and urgent needle decompression considered.

A chest X-ray is the confirmatory test but should not delay emergency decompression.

TREATMENT

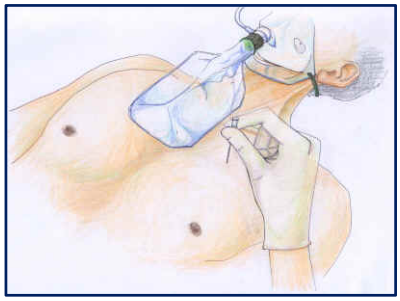
- Administer high flow oxygen via face mask
- Needle decompression
- Following needle decompression insert an intercostal catheter (thoracostomy tube).

- **Procedure for needle decompression**

Explain to the patient what you are going to do. Insert a large-bore 14–16 G needle with a catheter into the second intercostal space at the midclavicular line. Hold the needle at 90 degrees to the chest wall when inserting.

Once the needle is in the pleural space, you will hear the hissing sound of escaping air. Remove the needle while leaving the catheter in place. Put a three way tap on the catheter.

After needle decompression, insert a thoracostomy tube and under water seal drain or call for help for someone with the skills to do so. This is the definitive treatment for tension pneumothorax.



Inserting needle at 90 degrees into second intercostal space

ACUTE ASTHMA

Asthma is a complex disorder of the airways that is characterised by variable and recurring symptoms related to airflow obstruction, bronchial hyper-responsiveness, and an underlying inflammation of the airways. The interaction between these features determines the clinical manifestations, the severity of asthma and the response to treatment.

Beware of the asthmatic unable to speak in sentences due to breathlessness.

Asthma is characterised by one or more of the following:

- Shortness of breath
- Wheeze or silent chest
- Cough
- Chest tightness.

The rate of progression of symptoms is important. Deterioration can be rapid and some patients may progress rapidly to respiratory arrest.

FACTORS ASSOCIATED WITH HIGH RISK

- History of previous severe exacerbations requiring hospitalisation
- Current use or recent withdrawal of corticosteroids therapy
- Frequency of hospitalisations
- ICU admissions requiring intubation

PATHOPHYSIOLOGY

- Increased airway resistance results in reduced expiratory air flow.
- Airway resistance increases due to inflammation, excess secretions and bronchospasm.
- Due to this the expired gas gets trapped in the lungs causing hyperinflation.
- Hyperinflation causes over stretching of respiratory muscles and increases the work of breathing.
- There is an increase in the physiological dead space. Initially an increased respiratory rate compensates but fatigue may set in and hypercarbia results.
- Severe air trapping produces extreme intrathoracic pressures, reduced venous return to the right heart, reduced cardiac output and can result in loss of cardiac output.
- Thick mucous secretions can cause ventilation-perfusion mismatch.

MANAGEMENT

Asthmatic patients can rapidly deteriorate. Repeated and close monitoring is necessary. Frequent clinical observations including respiratory rate, heart rate and blood pressure should be performed and ECG and pulse oximetry monitors applied.

Acute severe asthma is a medical emergency and treatment and assessment should be carried out simultaneously using the ABCDEFG algorithm:

- Treat hypoxia
- Assess severity
- Treat bronchospasm and inflammation
- Prevent complications.

HYPOXIA

Usually asthma patients are not significantly hypoxic and hypoxia is often seen late in severe asthma or with complications such as pneumothorax. Hypoxia should respond well to supplemental oxygen. These patients should be supplemented with oxygen by Hudson mask to keep their saturations >95%. If oxygen saturation does not improve suspect things like added infection, worsening asthma despite treatment with rising CO_2, and pneumothorax. A CXR may be helpful.

Warning Signs of Severe Asthma

- The presence of persistent hypoxia despite oxygen therapy
- Cyanosis
- Inability to speak in short sentences
- Extensive wheeze or silent chest
- Fatigue/ restlessness /confusion /exhaustion
- Rising $PaCO_2$
- Extreme tachycardia (>150) or bradycardia and hypotension.

These patients should be closely monitored in a high dependency or intensive care unit with staff skilled in management of severe respiratory failure and ability to manage the airway and institute mechanical ventilation.

If the asthma is severe by the above criteria you should invoke your local Rapid Response System and call for help.

TREATMENT OF BRONCHOSPASM AND INFLAMMATION

- **Oxygen Therapy**

  High flow oxygen should be started immediately in all patients. Initial hypoxia can become worse temporarily after bronchodilators. Nasal cannulae, although more comfortable, deliver variable oxygen and may not be sufficient.

- **Beta2 -Agonists**

  Inhaled high dose beta2-agonists are bronchodilators of choice in acute asthma. Doses of salbutamol 5 mg by nebuliser every 15 minutes up to a maximum dose of 20 mg are recommended to gain an effective response. Continuous delivery by nebuliser is only required for some patients.

- **Corticosteroids**

  All adult patients presenting with severe acute asthma should receive intravenous hydrocortisone 100 mg stat or oral prednisolone 50 mg stat. Hydrocortisone is continued six-hourly. For mild to moderate asthma oral prednisolone is effective unless they cannot swallow the tablet safely.

- **Ipratropium**

  Routine use of ipratropium is not recommended in patients with acute asthma unless there is a definite indication for it.

- **Antibiotics**

  If the presentation is consistent with an infective component then antibiotics are recommended.

- **Adjunctive measures**

  Take a detailed history looking for precipitants such as drug reactions, viral or bacterial infection symptoms and recent illicit drug use. Remove any potential precipitants, position patient and reassure.

Remember if the patient doesn't settle call for help and transfer to a monitored area where they can receive salbutamol or adrenaline infusions and ventilatory support if required.

PREVENTION OF COMPLICATIONS

The most important aspect of asthma management is frequent assessment of the patient to ensure improvement and prevent complications. The complications can be severe and life threatening. Some of the complications are respiratory failure requiring ventilatory support, pneumothorax and in severe cases, cardiac arrest.

CHRONIC OBSTRUCTIVE PULMONARY DISEASE

Chronic Obstructive Pulmonary Disease (COPD) is characterised by airflow limitation secondary to chronic bronchitis or emphysema. There is often a long history of smoking.

- Chronic bronchitis is a clinical term defined as productive cough for three months of two consecutive years.
- Emphysema is an anatomical term defined as the abnormal permanent dilatation of the airways with no obvious fibrosis.

The disease process is progressive and may be partially reversible; however with the progression reversibility diminishes.

PATHOPHYSIOLOGY

The changes in COPD occur in large and small bronchioles resulting in lung tissue destruction and loss of elastic recoil of the lung.

CLINICAL HISTORY

- Most patients of COPD are smokers or have been smokers in the past.
- Alpha1-antitrypsin deficiency is the only known genetic deficiency and accounts for less than 2% of all cases.
- Pollution and airway hyper-responsiveness may contribute to the progression of disease.

- Look for a history suggestive of an infective exacerbation.

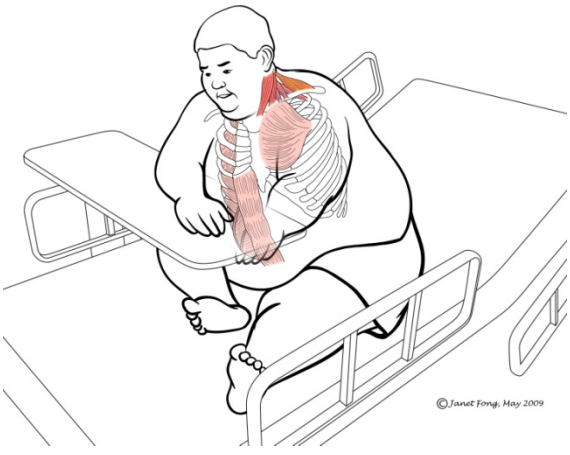
CLINICAL SIGNS AND SYMPTOMS

- Breathlessness is the most common symptom and often the first to be noticed.
- Wheezing occurs in most patients particularly during exacerbations.
- Productive cough which may be related to infection causing exacerbations.
- Cyanosis.
- Cor-pulmonale is the advanced form of right heart failure secondary to lung disease.
- Anorexia.
- Significant weight loss is associated with very poor prognosis.

- **Signs**

Use the ABCDEFG algorithm and DETECT assessment to look for:

- Increase in respiratory rate
- Use of accessory muscles
- Paradoxical 'drawing-in' of intercostal muscles
- Peripheral oedema
- Raised jugular venous pressure
- Cyanosis
- Barrel shaped chest
- Hyper-inflated lung fields
- Diffuse breath sound
- Wheeze
- Crackles often representing infection or failure.



Use of accessory muscles and drawing in of intercostal muscles

LABORATORY, DIAGNOSTICS AND IMAGING

- Detailed investigations are beyond the scope of this programme.
- Simple tests include ECG suggestive of right ventricular strain with P–pulmonale, polycythaemia, increased white cell count on full blood count, sputum culture, and CXR.

MANAGEMENT

- **Oxygen therapy**: Institute oxygen supplementation as these patients have increased oxygen demands during the exacerbation. A secondary increase in $PaCO_2$ and decrease conscious level is not of primary importance and will be detected with close observation and monitoring of ABGs. Oxygen therapy is very important. Consideration of the risks of CO_2 narcosis is less important than treating hypoxia. It is safer to give oxygen than not. If the patient stops breathing then ventilate.

  In a patient with known chronic lung disease where it is known the $paCO_2$ is chronically high they may be labeled as "running on hypoxic drive." Be careful about putting all patients with chronic lung disease in this group. Such patients often carry a card stating their arterial gases and they have usually seen a specialist medical officer. It may also be documented in the medical record. If in doubt contact the senior supervising medical officer. In these patients, a slightly lower oxygen saturation (around 90%) may be acceptable. And remember if in doubt always give a hypoxic patient oxygen and, if necessary, support ventilation.
- **Steroids:** Patients admitted to hospital will often benefit from oral corticosteroids but only around half of COPD patients benefit from long term therapy.
- **Bronchodilators:** Beta2 agonists (e.g. salbutamol) have been shown to be extremely effective in treatment and also prevention of exacerbation and remain a simple treatment to institute until help arrives.
- **Anticholinergic agents:** Ipratropium bromide is useful in most patients and may be more effective than salbutamol in some patients.
- **Antibiotics:** When infection is the cause of exacerbation the common organisms seen are *Streptococcus pneumoniae, Haemophilus influenzae,* and *Moraxella catarrhalis.* The penicillin group, cephalosporins and the macrolide antibiotics are the drugs of choice.
- **Non-invasive/invasive ventilation:** Hypercarbia is often well tolerated by COPD patients due to the chronic compensation. However acute decompensation leads to respiratory fatigue and an acute rise in $PaCO_2$. These patients can benefit from bi-level ventilation (BiPAP) or in some extreme cases may need invasive mechanical ventilation.

Note: If the disease is end stage, escalation of medical treatment may not be efficacious and focus may need to change to comfort care. It is important to consider the ethical aspects of end- of-life care (Chapter 7, p 76) and seek help in such decision making.

KEY KNOWLEDGE AND SKILLS

- Be able to demonstrate systematic assessment of breathing and oxygenation.
- Understand the causes of central and peripheral cyanosis.
- Describe the indications and rationale for safe oxygen therapy in the deteriorating patient.
- List the common causes of breathlessness.
- Describe the clinical signs and treatment of a tension pneumothorax.
- Describe how to recognise and initiate treatment of an acute asthma attack.
- Describe how to recognise and initiate treatment for an acute exacerbation of chronic obstructive lung disease.
- Describe the principles and limitations of pulse oximetry.

CASE STUDIES

CASE 1

A 37 year old female, with a history of asthma, presents to the Emergency Department with tachypnoea (RR 35 bpm), and acute shortness of breath with audible wheezing. The patient has taken her prescribed medication of salbutamol at home with no relief of symptoms prior to coming to the ED. The triage nurse calls you to see the patient.

1. Outline your initial assessment

A physical examination revealed the following:

- HR 110, RR 40 with signs of accessory muscle use.
- Auscultation revealed decreased breath sounds with inspiratory and expiratory wheezing and the patient is coughing up small amounts of yellow sputum.
- SaO_2 is 93% on room air.
- An arterial blood gas (ABG) is ordered with the following results: pH 7.5, $PaCO_2$ 27, PaO_2 75.

2. Outline your initial treatment measures

She is given nebulised salbutamol 5 mg every 15 min for the next 30 min.

You reassess her.

Auscultation reveals loud expiratory wheezing and better airflow.

3. What is your follow up treatment?

A second treatment is given with the above medications and on auscultation there is clearing of breath sounds and much improved airflow. RR is 24 at this time and HR 108.

She is given 50 mg of prednisolone orally and continued on intermittent nebulisations.

4. Where would you place her and what sort of monitoring and instructions do you give?

Over the next hour she starts to worsen again and progressively becomes wheezier. Her oxygen saturations drop to 90%. She improves her oxygenation with a Hudson mask at 10 L/min. However you note she is using accessory muscles to breathe and looks more fatigued.

5. What is your plan?

IV hydrocortisone 100 mg stat is given and followed by continuous nebulisation with salbutamol. The Rapid Response Team is called.

She is commenced on non-invasive ventilation, intravenous salbutamol and continued on intravenous hydrocortisone. A chest X-ray is suggestive of hyper-inflated lung fields but no evidence of pneumothorax. She is transferred to intensive care where she improves over the next 48 hours and is discharged to the ward.

CASE 2

A 62 year old male is admitted to the ward with a three day history of increased sputum production and shortness of breath. His background history is of COPD and hypertension. He is on regular steroids prednisolone 5 mg daily, salbutamol puffs three times a day and Prazosin 2 mg BD.

In the last hour he has been feeling increasingly short of breath. He presses the call button to tell you this.

1. Outline your approach to his call to the bedside.

He has a 60-pack/year history of smoking and says he quit one month ago. He has had numerous admissions to hospital in the past but never needing intubation. His last admission was one month ago.

On examination you find the following:

A thin male sitting bolt upright in bed, with pursed lips using accessory muscles of breathing. He is speaking in only short sentences and he appears distressed. You note he is diaphoretic and has cool peripheries, but is not cyanosed. He has bruised skin.

HR is 108/min, BP is 160/106 mmHg, RR is 28/min . You place a pulse oximeter probe on him. His oxygen saturation is 84% on oxygen 2 L/min. There is pedal pitting oedema, a normal JVP and apex impulse. There is decreased excursion of diaphragm on percussion. On auscultation there are very distant breath sounds with diffuse wheeze and bilateral basal crackles. On listening to the heart sounds there is a loud P2. The abdomen is soft and no organomegaly or abnormal enlargement of organs.

2. What is your management plan?

Simple treatment measures

- Oxygen – enough to maintain saturation >95%
- Fluids – he will need IV fluids given the increased effort to breathe
- Cultures – sputum cultures and blood cultures if febrile
- Antibiotics
- Steroids – intravenous hydrocortisone or oral prednisolone
- NIV – if he continues to be hypercarbic or has increased work of breathing, call for help as he may benefit from non-invasive ventilation.

3. What instructions do you give and to whom?

Six hours later you review him and note he is now improved and has reduced effort to breathe.