



compass

'Pointing you in the right direction'

ADULT

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Introduction

COMPASS is an inter-professional education program designed to enhance our understanding of patients deteriorating and the significance of altered observations. It also seeks to improve communication between health care professionals and enhance timely management of patients.

This education package has been developed in conjunction with the development and implementation of a Modified Early Warning Score and redesigned general observation charts.

Preamble

It has become increasingly apparent that many clinicians are unable to manage a deteriorating patient in an appropriate, timely fashion. This is often as a result of the inability to recognise that the patient is deteriorating.

Delaying resuscitation and treatment increases the likelihood of organs failing due to inadequate oxygen delivery to these tissues. This in turn can lead to unexpected death, unexpected cardiac arrests and unplanned admissions to the Intensive Care Unit.

It is important to understand the key components that lead to the lack of appropriate and timely management.

a. Absent or inaccurate observations

- equipment not available
- equipment malfunctioning
- inability to use equipment due to lack of knowledge
- inadequate time to perform observations
- inability to make time for performing observations
- lack of understanding of why observations are important
- general culture that observations are not important.

b. Inability to understand observations recorded

- unable to trend results and interpret their meaning
- lack of knowledge

c. Failure to trigger timely appropriate response

- absence of, or inaccurate observations, preventing appropriate clinical decision making
- inability to understand observations recorded
- inability to develop a diagnosis
- inability to develop a treatment plan
- failure to escalate if patient not reviewed in a timely fashion
- failure to escalate if no improvement despite intervention

Having identified the key components, it is possible to address those areas that pertain to lack of knowledge.

An example of what can go wrong

A 60 yr old man was admitted with pancreatitis.

- On day 3 of his stay, his systolic blood pressure fell into the 80s, and whilst fulfilling the Medical Emergency Team (MET) criteria no code blue was called. Over the next 24 hours the patient filled Code Blue/ MET criteria thirteen times, still with no activation of MET. The patient died 14 hours later.
- The system issues identified were:
 - failure to follow hospital policy in calling a MET
 - inadequate documentation of observations, particularly respiratory rate
 - failure to change the management plan despite its inadequacy
 - failure to escalate the level of medical review despite the seriousness of the situation.

The Australian Commission on Safety and Quality in Healthcare has developed National Safety and Quality Health Service Standards. Standard 9 - Recognising and Responding to Clinical Deterioration in

Acute Health Care, outlines clear requirements for acute healthcare settings in regards to a minimum standard for documenting vital signs, education and escalation systems to assist clinicians in recognising and responding to clinical deterioration. This education package, in conjunction with a clearly formatted observation chart and the utilisation of a track and trigger system, aims to prevent missed cases of deterioration and guide clinicians in recognising and responding to clinical deterioration.

Our Aim

To enable health care professionals to recognise the deteriorating patient and initiate appropriate and timely interventions

Our Objectives

- For participants to understand the importance and relevance of observations and the underlying physiology.
- For participants to be able to recognise and interpret abnormal observations.
- For participants to be able to communicate effectively to the right people and at the right time.
- For participants to feel confident in recognising and managing deteriorating patients.
- To facilitate teamwork within the multidisciplinary team.
- To enable nurses, doctors, and allied health staff to develop management plans together.

How it Works

There are three phases to the package to be completed in the following order:

- The **online learning application (on the website)** and manual to be worked through independently.
- A **COMPASS** quiz.
- A 3-hour face-to-face session.

- You will have access to the patient's history, the current situation and their observation charts.
- A series of questions will be asked, which will then direct you to information on the specific vital sign in question.
- In order to move on to the next section on a different vital sign you must correctly answer a multiple-choice question.
- If you get any questions wrong you will be directed back to the information just covered, to reinforce the information in that section.
- When you answer the questions correctly you will move on to the next section of the case study.

At the completion of the **online learning application** there is a short multiple choice quiz. You will be unable to skip ahead in the **online learning application**, however you will be able to go back to any area that you have already completed if you choose to. The **online learning application** **MUST** be completed prior to coming to the face-to-face session you have been scheduled for. You must also complete **the COMPASS quiz** prior to the final session. Your results will be available to the program coordinator.

OK LET'S GET STARTED!



Oxygen Delivery

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Oxygen Delivery

Learning Objectives

- To understand the importance of oxygen delivery.
- To understand the factors that affect adequate oxygen delivery.

Background

Oxygen is essential for the adequate production of adenosine triphosphate (ATP) by cell mitochondria (figure 4). Adenosine triphosphate (ATP) is required as a source of energy for all intracellular functions. ATP is formed in the mitochondria via phosphorylation. A phosphate is added to adenosine diphosphate (ADP) via a high-energy bond, thus forming ATP. This stores energy on a temporary basis. When energy is needed by the cell, ATP is dephosphorylated to ADP, releasing the energy from the bond (text box 6).



Text box 6: Energy release

If there is inadequate oxygen supply, ATP production falls, and cellular function is then depressed (figure 4), through lack of energy. This can lead to organs failing and may result in an unexpected admission to ICU or even unexpected deaths. Therefore if oxygen delivery is maintained, this may reduce the incidence of unplanned ICU admissions and unexpected deaths.

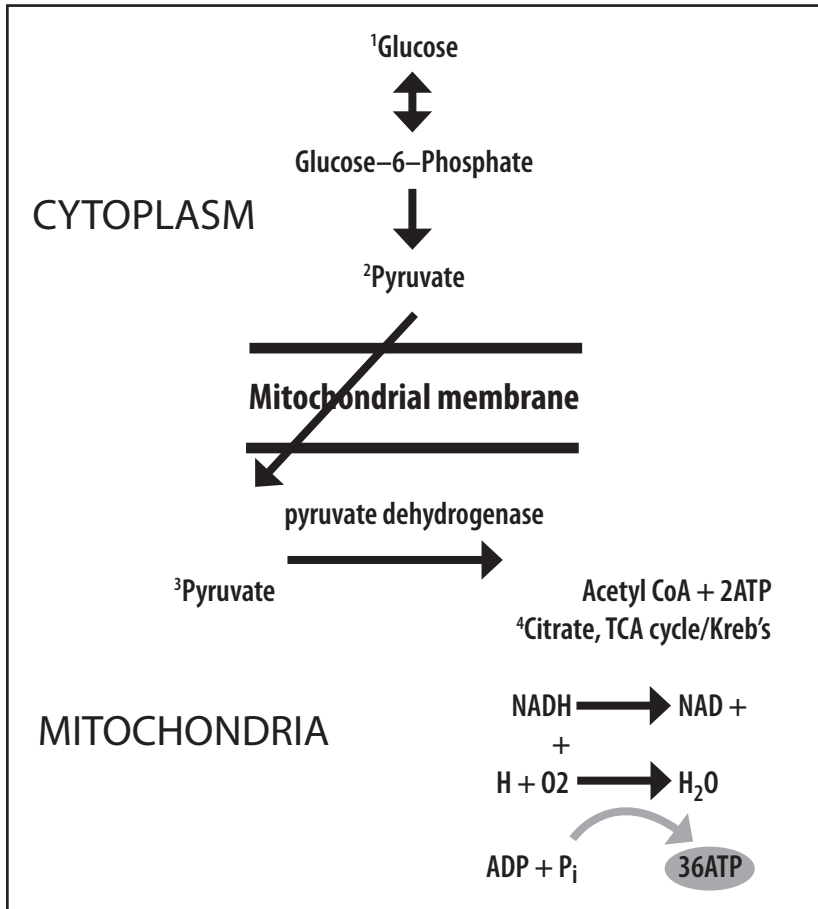


Figure 4: Aerobic Metabolism (i.e. with oxygen)

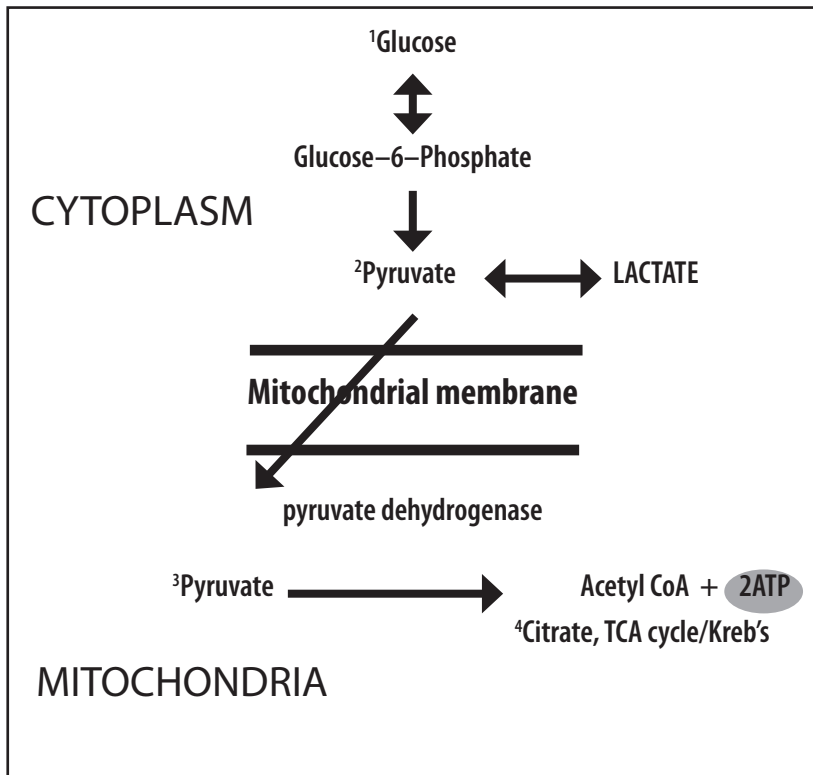


Figure 5: Anaerobic Metabolism (i.e. in the absence of oxygen)

Oxygen supply to the cells can be described by the “oxygen delivery chain” (figure 5).

Oxygen Delivery = Cardiac Output x Arterial Oxygen content

Thus oxygen delivery requires

- A. Arterial oxygen content:
 - haemoglobin concentration ([Hb])
 - haemoglobin oxygen saturation (SaO₂)
 - partial pressure of oxygen (PaO₂)

See Section on “Airway and Breathing”

- B. Cardiac output:
 - Stroke volume (SV)
 - Heart Rate (HR)

See Section on "Circulation"

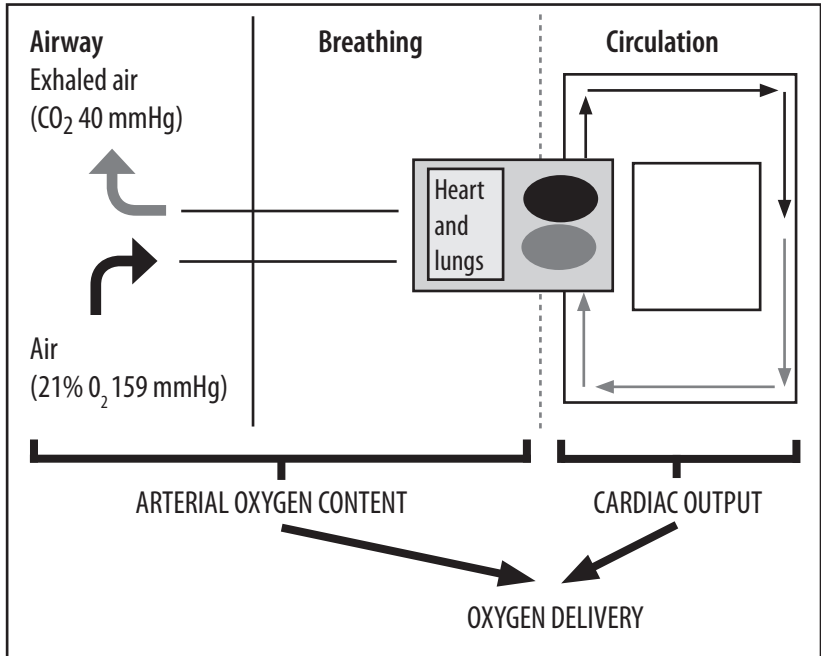


FIGURE 6: "ABC" and the Oxygen Delivery Chain

SUMMARY

- Oxygen is essential for the adequate production of adenosine triphosphate (ATP).
- If there is inadequate oxygen supply, ATP production falls, and cellular function is then depressed, organs start to fail and puts patient at risk of critical illness and death.
- $\text{Oxygen Delivery} = \text{Cardiac Output} \times \text{Arterial Oxygen content}.$



Airway and Breathing

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Airway and Breathing

Learning Objectives

- To recognise when difficulties with airway or breathing may compromise oxygen delivery to the tissues.
- To be able to apply the appropriate oxygen delivery device.
- To be able to manage appropriately a patient with impaired arterial oxygenation.
- To understand why the respiratory rate is such an important marker of the deteriorating patient.

INTRODUCTION

In order for oxygen to reach haemoglobin in the red cells and be transported around the body to the tissues, it needs to pass through the upper airways (nose, mouth, trachea) and lower airways of the lungs (bronchi) to the alveoli. To do this, we need both a patent airway, and the respiratory nerve and muscle function to move air in and out of the lungs. Once oxygen is in the alveoli, it diffuses across the thin alveocapillary membrane, into the blood and attaches to haemoglobin. From here, it is dependent on pulmonary and then systemic blood flow to move oxygen to the tissues and cells where it is required.

AIRWAY

Adult Airway

Oxygen cannot move into the lower respiratory tract unless the airway is patent. Causes of airway obstruction can either be mechanical or functional.

Causes of airway obstruction

- Functional airway obstruction – May result from decreased level of consciousness, whereby the muscles relax and allow the tongue to fall back and obstruct the pharynx.

- Mechanical airway obstruction – May be through aspiration of a foreign body or swelling/bleeding in the upper airway (e.g. trauma, allergy, and infection). It may also be caused by oedema or spasm of the larynx.

Examination of the airway

Recognition of airway obstruction is possible using a “look, listen, feel” approach. With permission of the Australia Resuscitation Council, Guideline 5-Breathing⁴

- **Look:** complete airway obstruction can cause paradoxical chest and abdominal movements (“see-saw” like movement, where inspiration is associated with inward movement of chest and outward movement of the abdomen). Other signs of airway obstruction include use of accessory muscles (neck and shoulder muscles) and tracheal tug.
- **Listen:** in complete airway obstruction, there will be no breath sounds at the mouth or nose; in incomplete obstruction, breathing will be noisy (stridor= inspiratory wheeze) and breath sounds are reduced.
- **Feel:** placing your hand immediately in front of the patient’s mouth allows you to feel if there is any air moving in or out.

Management of the obstructed airway

In the majority of cases in hospital, airway obstruction is functional, i.e. due to depressed level of consciousness. Simple manoeuvres may be required to open the airway:

- chin lift (figure 7)
- jaw thrust (figure 7)
- head tilt (figure 7)
- insertion of an oropharyngeal or nasopharyngeal airway (Guedel’s airway)

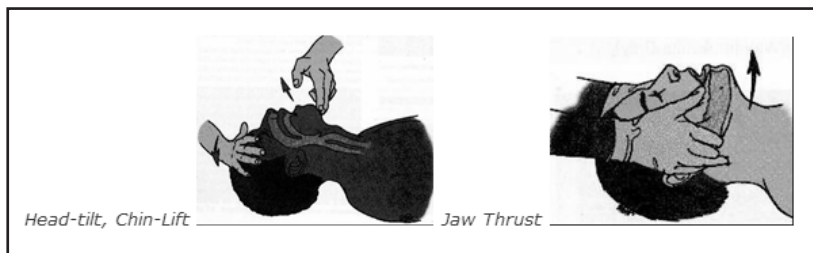


Figure 7: Simple Airway Manoeuvres

With permission from Emergency Management Training Services (<http://emtsstudentpage.com/>)

Suctioning of the airway using a Yankauer sucker may be required to remove any vomitus or secretions which could be contributing to airway obstruction.

If the patient continues to have a depressed level of consciousness and is unable to protect their own airway, endotracheal intubation may be required. This needs to be performed by **experienced** staff.

In all patients with an airway obstruction or who are unable to maintain an adequate airway, a MET should be called.

In rare cases, the airway obstruction may be due to mechanical factors which are not so easily treated, e.g. airway swelling, post-operative haematoma, infection. This is a medical emergency. Again a Code Blue/ MET should be called.

Text box 7: Airway obstruction

A surgical airway may be required if endotracheal intubation is not possible (called a cricothyroidotomy), and this should only be attempted by **experienced** medical staff.

BREATHING

Breathing is required to move adequate oxygen in and carbon dioxide out of the lungs. Breathing requires:

- intact respiratory centre in the brain
- intact nervous pathways from brain to diaphragm and intercostal muscles
- adequate diaphragmatic & intercostal muscle function
- unobstructed air flow (large and small airways).

Examination of Breathing

The “look, listen, feel” approach is a practical method of quickly determining causes for abnormalities in breathing.

Look

Respiratory rate is an important marker of a deteriorating patient (text box 8). When you walk into a room and the first thing you notice is the patient’s breathing, there is a significant problem with the patient.

Look for signs of respiratory failure which can include:

- use of accessory muscles
- sweating/pallor
- central cyanosis
- abdominal breathing
- shallow breathing
- unequal chest movement

Listen

Initially listen at the “end of the bed”, for:

- noisy breathing, which may indicate secretions in the upper airways
- stridor or wheeze which may indicate partial airway obstruction

Then auscultate with a stethoscope to assess breath sounds:

- quiet or absent breath sounds may indicate the presence of a pneumothorax or a pleural effusion or a significant reduction in air entry
- bronchial breathing may indicate the presence of consolidation

Feel

1. Palpation

Palpate the trachea and chest wall:

- tracheal deviation indicates mediastinal shift, which may be due to:
 - a pneumothorax or pleural fluid –tracheal deviation away from the lesion
 - lung collapse-tracheal deviation toward the lesion
- chest wall crepitus (subcutaneous emphysema) is highly suggestive of a pneumothorax, oesophageal or bronchial rupture
- asymmetrical chest wall movement may indicate unilateral pathology eg. consolidation, pneumothorax

2. Percussion:

- hyper-resonance indicates pneumothorax
- dullness indicates consolidation or pleural fluid.

Why Respiratory Rate is Important

An increase in respiratory rate can reflect either a drop in arterial saturation or reflect compensation for the presence of a metabolic acidosis. Respiratory rate may therefore be an important indicator of inadequate oxygen delivery to the tissues and therefore a marker of a deteriorating patient. As oxygen delivery to the tissues is reduced, cells revert to anaerobic metabolism. This increases the lactate production, resulting in build up of acid (see figure 10). The accumulation of lactic acid stimulates an increase in respiratory rate (tachypnoea).

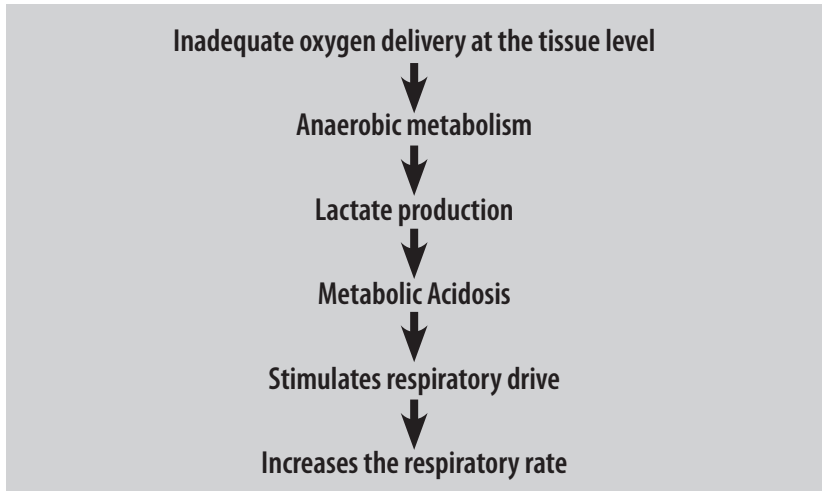


Figure 10: Importance of Respiratory Rate

Metabolic acidosis can increase the Respiratory Rate even though the arterial oxygen saturation may be normal

Text box 8: Metabolic acidosis, respiratory rate and arterial oxygen saturation

The decrease in oxygen delivery to the tissues, which results in tachypnoea, can be due to problems at any point in the oxygen delivery chain (figure 6).

A Normal Arterial Saturation and Tachypnoea

There can be falling oxygen delivery despite normal arterial oxygen saturation. Therefore rises in respiratory rate can occur in patients with a normal or low arterial oxygen saturation and may well be a better indicator of a deteriorating patient than arterial oxygen saturation.

Management

Specific treatment will depend on the cause, and it is vital to diagnose and treat life-threatening conditions promptly, e.g. tension pneumothorax, acute pulmonary oedema, acute asthma and acute pulmonary embolus.

All deteriorating patients should receive oxygen before progressing to any further assessment. The aim is to deliver supplemental oxygen to achieve a SpO₂ of 94-98% in those patients not at risk for hypercapnic respiratory failure, and the PaO₂ as close to 100mmHg as possible, but at least 60mmHg (SaO₂ 90%) is essential. In most patients, this can be achieved by sitting them upright, and applying 12-15 litres/min of oxygen via a non-rebreather mask (figure 12).

If the patient does not improve they will require an ICU review.

There are a small subgroup of patients for which high concentrations of oxygen can be disadvantageous as it suppresses their hypoxic drive, this includes patients with COPD who are CO₂ retainers or those with risk factors for hypercapnoeic respiratory failure (e.g. morbid obesity, chest wall deformities or neuromuscular disorders), high concentrations of oxygen can be disadvantageous by suppressing their hypoxic drive.

However, these patients will also suffer end-organ damage or cardiac arrest if their blood oxygen levels fall too low. The aim in these patients is to achieve PaO₂ of 60mmHg, or saturation of 90% on pulse oximetry. So in a patient with COPD who has a pCO₂ > 60mmHg but is also hypoxic, pO₂ < 60mmHg, do not turn the inhaled O₂ down however do not leave the patient unattended. This patient should be considered for non-invasive or invasive ventilation. If their pO₂ is > 60 mmHg, then you can turn the inhaled O₂ down to maintain SaO₂ > 90%

Text Box 10: Oxygen delivery in COPD

Oxygen Delivery Systems

The oxygen delivery systems available are classified into fixed and variable performance devices. They are able to deliver a wide range of oxygen concentrations.

A. Fixed performance devices

Provide gas flow that is sufficient for all the patient's minute

ventilation requirements. In these devices, the inspired oxygen concentration is determined by the oxygen flow rate and attached diluter (see table 10), e.g. the Venturi mask (figure 11).

In patients at risk of hypercapnia from too high an inspired oxygen, a venturi system is more accurate in delivering the oxygen rate desired.



Figure 11: Venturi mask system

Image reproduced with permission of mayohealthcare.com.au

Diluter Colour	Diluter setting (Inspired Oxygen)	Suggested oxygen flow rate (Litres/min)
Blue	24%	3
White	28%	6
Orange	31%	8
Yellow	35%	10
Red	40%	12

Table 10: Relationship between inspired oxygen and oxygen flow rate with Venturi Masks

Please note that the colours and flow rates vary between companies. Always read the label.

B. Variable performance devices

These do not provide all the gas required for minute ventilation, they entrain a proportion of air in addition to the oxygen supplied.

The inspired oxygen concentration will depend on:

- a) oxygen flow rate
- b) the patient's ventilatory pattern (If the patient has a faster or deeper respiratory rate, more air will be entrained reducing the inspired oxygen concentration).

These devices include nasal prongs, simple facemasks, partial rebreathing and non-rebreather masks.

- a) **Nasal prongs** (figure 12) – The dead space of the nasopharynx is used as a reservoir for oxygen, and when the patient inspires, entrained air mixes with the reservoir air, effectively enriching the inspired gas. Oxygen flow rates of 2 - 4 L/min.
- b) **High flow nasal prongs** – These use warm humidified oxygen at higher flow rates 4-8 L/min.

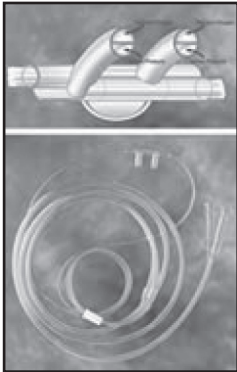


Figure 12: Nasal prongs Image reproduced with permission of mayohealthcare.com.au

- c) **face mask** (figure 13) – reservoir volume of oxygen is increased above that achieved by the nasopharynx (text box 12), thus higher oxygen concentration can be achieved in inspired gas (max 50-60%).

Oxygen flow rates less than 6L/min for an adult face mask mask should not be used due to carbon dioxide retention in the mask.

Text box 11: **face mask**

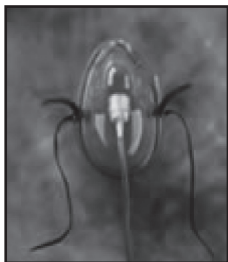


Figure 13: **face mask**

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Non-rebreather mask (figure 14) – A simple facemask with the addition of a reservoir bag, with one or two-way valves over the exhalation ports which prevent exhaled gas entering the reservoir bag (permits inspired oxygen concentration up to 90%). Oxygen flow rate of 12-15 L/min.



Figure 14: Non-rebreather mask

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Monitoring and Titrating Oxygen Therapy

Oxygen therapy can be monitored clinically (patient's colour, respiratory rate, respiratory distress), or by measuring arterial oxygenation with pulse oximetry or arterial blood gas.

The advantage of measuring an arterial blood gas is that both oxygen and carbon dioxide, and metabolic status (including lactate) is measured.

If the carbon dioxide tension rises in someone with acute respiratory failure, it can be a sign that they are tiring and may require ventilatory

support. If CO_2 begins to rise in a patient with COPD, it may be prudent to reduce the inspired oxygen concentration, however always remember that the arterial oxygen tension should not be allowed to fall below pO_2 60mmHg.

Patients do not die from a raised CO_2 alone: they die from hypoxaemia, (text box 13).

In an acute setting, when taking an arterial blood gas sample, do not remove the oxygen. It is unnecessary, and may precipitate sudden deterioration.

Text box 12: Arterial blood gases

As long as the concentration of oxygen being delivered is recorded, the degree of hypoxaemia can be calculated using the alveolar-air equation and A-a gradient (text box 13). The blood gas machine can calculate this for you as long as the correct inspired oxygen concentration is recorded.

$$\text{PAO}_2 = \text{FiO}_2 - \text{PaCO}_2/0.8$$

PAO_2 should be close to PaO_2 in normal lungs

A = alveolar a = arterial (A-a = age/3)

Text box 13: Ideal alveolar gas equation

Pulse oximetry measures how well haemoglobin is saturated with oxygen (oxygen saturation). It uses a probe, which shines light of two wavelengths through the tissues, and detects that which passes through. Oxygenated and deoxygenated haemoglobin absorb different amounts of light, and this information is integrated to determine the arterial oxygen saturation.

Oximeters can be unreliable in certain circumstances (text box 14), e.g. if peripheral circulation is poor, the environment is cold, arrhythmias, or if the patient is convulsing or shivering.

If the pulse oximeter does not give a reading, do not assume it is broken ... the patient may be poorly perfused and/or have a low mean arterial oxygen saturation.

Text box 14: Pulse oximetry warning

Although pulse oximetry provides good monitoring of arterial oxygenation, it does not measure the adequacy of ventilation, as carbon dioxide levels are not measured (text box 15) nor does it determine the adequacy of oxygen delivery to the tissues.

Oxygen saturation may be “normal” but the $p\text{CO}_2$ may be high which reflects inadequate minute ventilation and hence respiratory failure. Arterial oxygen saturation being “normal” does not rule out acute respiratory failure.

Text box 15: Normal SpO_2 does not rule out respiratory failure

Arterial blood gases remain the gold standard for assessing respiratory failure¹⁵ (ABGs). It measures arterial oxygen, arterial saturation and arterial carbon dioxide. It also provides information on the metabolic system (i.e. bicarbonate concentration, base excess and lactate) an approximate haemoglobin, electrolytes and blood glucose.

ABGs should be measured in patients who¹⁵:

- are critically ill
- have deteriorating oxygen saturations or increasing respiratory rate
- requires significantly increased supplemental oxygen to maintain oxygen saturation
- have risk factors for hypercapnic respiratory failure who deteriorate
- have poor peripheral circulation and therefore unreliable peripheral measurements of oxygen saturation.

When assessing a patient remember to incorporate all the vital signs, do not just look at an individual reading. If you suspect failing oxygen delivery, consider where in the oxygen delivery chain may be disordered (see figure 15).

Text box 16: Vital signs

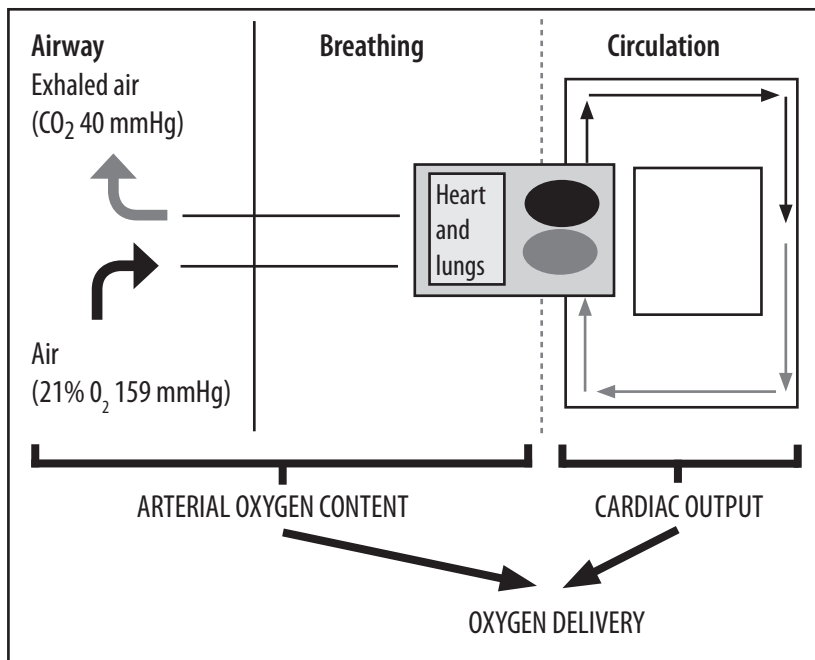


Figure 15: ABC-Oxygen Delivery Chain

SUMMARY

- An increase in Respiratory Rate can occur even though the arterial oxygen saturation may be normal.
- In rare cases, an airway obstruction may be due to mechanical factors, which may not be so easily treated, e.g. airway swelling, post-operative haematoma, infection. This is a medical emergency. A Code Blue/MET should be called.
- In a small subgroup of patients who have Chronic Obstructive Pulmonary Disease (COPD) and are “CO₂ retainers”, high concentrations of inspired oxygen can be disadvantageous as it suppresses their hypoxic drive. However, these patients will also suffer end-organ damage or cardiac arrest if their blood oxygen levels fall too low. The aim in these patients is to achieve a PaO₂ of 60mmHg, or oxygen saturation of 90% on pulse oximetry. So, in a patient with COPD who has a pCO₂ > 60mmHg but is also hypoxic, pO₂ < 60mmHg, do NOT turn the inhaled O₂ down however do not leave them unattended. If their pO₂ is > 60 mmHg, then you can turn the inhaled O₂ down to maintain SaO₂ > 90%.
- Oxygen flow rates less than 6L/min for adult **face mask** mask should not be used due to carbon dioxide retention in the mask.
- When taking an arterial blood gas sample, do not remove the oxygen mask, it is unnecessary and may precipitate sudden deterioration.
- If the pulse oximeter does not provide a reading, do not assume it is broken; the patient may have poor perfusion and be very unwell!
- Oxygen saturation may be “normal” but the pCO₂ may be high, reflecting inadequate minute ventilation and respiratory failure.
- Remember to incorporate all the vital signs in your assessment!





Circulation

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Circulation

Learning Objectives

- To understand why pulse rate and blood pressure are “vital signs” and the importance of measuring them.
- To describe the mechanisms which generate blood pressure, and be able to define, describe causes of, consequences of and compensation for development of hypotension.
- To understand what is meant by shock.
- To manage hypotension in the deteriorating patient.

The Importance of Oxygen

Oxygen reaching the cells and mitochondria is dependent upon adequate amounts of oxygen being delivered (figure 16). Without oxygen being delivered to the mitochondria, inadequate amounts of ATP are generated and cellular dysfunction occurs. Oxygen delivery's key components are:

- Cardiac output = Stroke Volume x Heart Rate
- Arterial oxygen content = Haemoglobin concentration x Arterial Oxygen Saturation

Oxygen delivery = Cardiac output x Arterial Oxygen Content

$$\begin{array}{ccc} & \wedge & \wedge \\ \text{Stroke Volume x Heart Rate} & & \text{Haemoglobin x SaO}_2 \end{array}$$

Figure 16: Oxygen delivery

BLOOD PRESSURE

Blood Pressure, Heart Rate and Oxygen Delivery

Blood pressure is the product of cardiac output and Peripheral Vascular Resistance (PVR).

$$\text{Blood Pressure} = \text{Cardiac Output} \times \text{Peripheral Vascular Resistance}$$

Text box 20: Blood pressure

- A **decrease in blood pressure** can reflect a **decrease in cardiac output** and which can lead to a reduction in the amount of oxygen getting to the tissues.
- An **increase in heart rate** may reflect a **decrease in stroke volume**, which may reflect a **decrease in cardiac output** which may lead to inadequate amounts of oxygen getting to the tissues.
- Hence, the measurement of **pulse and blood pressure** is an important surrogate marker of whether there is **adequate cardiac output** and **hence oxygen delivery** to the tissues and their survival.

High pulse and low blood pressure may reflect inadequate oxygen delivery to the tissues.

Text box 21: Relevance of pulse and blood pressure to oxygen delivery

Blood Pressure and Maintenance of Organ Function

- There are some organs that require an adequate blood pressure as well as adequate oxygen delivery for their optimal function. The brain and kidney are two examples of these organs.
- The body's organs adapt over time to a person's "normal" blood pressure. If blood pressure is always elevated, e.g. chronic hypertension; the brain and kidneys adapt and will require a

greater blood pressure in order to function normally. Therefore it is important to know what your patient's "normal" or "usual" blood pressure was prior to their current illness.

Definition of Hypotension

The generally acceptable definition of hypotension in adults is:

- A drop of more than 20% from "usual" blood pressure OR
- Systolic blood pressure of less than 100mmHg

It is important to remember that someone who is normally hypertensive may be relatively hypotensive even when their systolic blood pressure is above 100mmHg.
Do not always use 100mmHg as your CRITICAL Systolic Blood Pressure cut off!

Text box 23: Critical Blood pressure

Possible Causes of Hypotension

If blood pressure is the product of cardiac output and total peripheral vascular resistance, blood pressure can either fall because of:

- a) a fall in cardiac output
- b) a fall in peripheral vascular resistance.

It is important to understand how cardiac output and total peripheral resistance are determined and what can affect them. Having understood these principles, it is then easier to know what management to put in place.

A. Cardiac output

Cardiac output is the product of stroke volume and heart rate (i.e. flow is the volume per unit time)

Factors affecting stroke volume:

1) **Contractility**

The ability of the heart to contract in the absence of any changes in preload or afterload - it reflects the strength

Major negative influences (negative inotropy) include:

- Myocardial ischaemia
- Acidosis
- Drugs (e.g. beta-blockers, anti-dysrhythmic)

Major positive influences (inotropy) include:

- Sympathetic nervous system
- Sympathomimetics (noradrenaline, adrenaline)
- Calcium
- Digoxin

2) **Pre-Load**

How well filled is the heart at the end of diastole? i.e. the end diastolic volume.

Increases in end diastolic volume will result in an increase in stroke volume although if the end diastolic volume over-stretches the heart muscle, the stroke volume can start to decrease.

The major effect of pre-load is venous return to the heart, which is influenced by:

a) **Intravascular blood volume**

Absolute:

A decrease in intravascular blood volume (bleeding, electrolyte and water loss [diarrhoea, vomiting], water loss [diabetes insipidus]) will cause a decrease in venous return and hence a decrease in stroke volume.

Relative:

There is no actual loss of intravascular blood volume but with vasodilatation and pooling of blood (vasodilators, epidurals, sepsis) a decrease in venous return to the heart occurs and hence a decrease in stroke volume.

Decreases in intravascular blood volume can decrease cardiac output and therefore decrease blood pressure.

Text 24: Relationship between intravascular blood volume and blood pressure

b) Intrathoracic pressure

Increases in intrathoracic pressure (asthma attacks, positive pressure ventilation) will restrict the amount of blood returning to the heart decreasing venous return and therefore reduce stroke volume.

Increases in intrathoracic pressure can decrease cardiac output and therefore decrease blood pressure.

Text box 25: Relationship between intrathoracic pressure and blood pressure

3) After-Load

This is the resistance to the ejection of blood from the ventricle. This resistance can either be caused by an outflow resistance from the heart (aortic stenosis) or resistance to flow in the systemic circulation. This resistance is determined by the diameter of the arterioles and per-capillary sphincters. As resistance rises, stroke volume is reduced.

An increase in peripheral vascular resistance can decrease cardiac output and hence oxygen delivery.

Text box 26: Relationship between total peripheral resistance and oxygen delivery

B. Heart rate

This is determined by the rate of spontaneous depolarisation at the sinoatrial node. The rate can be modified by the autonomic nervous system:

- Parasympathetic stimulation: SLOWS the heart rate via the vagus nerve e.g. vasovagals response, parasympathomimetics e.g. anticholinesterases (neostigmine)
- Sympathetic stimulation: QUICKENS the heart rate via the

sympathetic cardiac fibres e.g.: stress response, temperature, Sympathomemetics (adrenaline, noradrenaline, isoprenaline).

In the absence of conduction through the atrioventricular node (Complete Heart Block), the ventricle will only contract at its intrinsic rate of 30-40 beats per minute.

Any changes in heart rate can change the cardiac output. A faster heart rate can increase the cardiac output and this often occurs when the stroke volume is falling while any reduction in heart rate can cause a decrease in the cardiac output.

Does a fast heart rate always increase cardiac output and blood pressure?

There are situations when an increase in heart rate may reduce the cardiac output. If the ventricle does not have adequate time to fill with blood this reduces the end diastolic volume and therefore stroke volume. Cardiac output reduces as a result, and may cause a drop in blood pressure (text box 27). A good example is atrial fibrillation with a rapid ventricular response.

Does a slow heart rate always decrease cardiac output and blood pressure?

Sometimes when the heart slows there may be no reduction in cardiac output. As the ventricle has a longer time to fill, the end diastolic volume is increased each beat, stretches the myocardial fibres and increases the stroke volume per beat, this may then compensate for the reduction in heart rate. Therefore, there may be no change or even an increase in cardiac output and blood pressure. A good example of this phenomena is a very healthy athlete.

Fall in cardiac output

- **Fall in stroke volume due to**
 - **Decreased contractility (heart muscle)**
 - **Decreased preload (volume)**
 - **Increased afterload**
- **Fall in Heart Rate – e.g. Complete Heart Block**

Fall in Peripheral Vascular Resistance (PVR)

Text box 27: Causes of a fall in blood pressure

C. Peripheral Vascular Resistance

Changes in peripheral vascular resistance (the cumulative resistance of the thousands of arterioles in the body) can increase or decrease blood pressure.

1. Increase in peripheral vascular resistance

Autonomic Nervous System

- Stimulation of Sympathetic Receptors:*** Sympathetic stimulation (α_1) of the arterioles can cause vasoconstriction and a subsequent increase in blood pressure. This often occurs in response to a fall in blood pressure (perhaps as a result of falling cardiac output), which is detected by baroreceptors situated in the carotid sinus and aortic arch, reducing the discharge from them to the vasomotor centre with a resultant increase in sympathetic discharge.
e.g. Sympathomimetics that stimulate the α_1 receptor will cause vasoconstriction of arteriole, examples include noradrenaline, adrenaline.
- Direct action on arteriole smooth muscle:*** Examples include metaraminol, vasopressin, angiotensin, methylene blue (a vasoconstrictor by inhibiting nitric oxide action on the vasculature).

2. Decrease in peripheral vascular resistance

a. *Blockade of Autonomic Sympathetic Nervous System*

Anything that causes a reduction in the sympathetic stimulation of the arterioles will result in vasodilatation, reducing vascular resistance and blood pressure.

Influences include:

- increasing the stimulation of the baroreceptors from a rise in blood pressure, which causes a reduction in the sympathetic outflow causing vasodilatation.
- any drug that blocks the sympathetic nervous system can cause vasodilatation and a fall in blood pressure, e.g. α_2 agonists (clonidine, epidurals)

b. *Direct action on arteriole smooth muscle*

Molecules and drugs can have a direct effect on the vascular smooth muscle of arterioles, causing vasodilatation.

Examples include:

- Vasodilating Drugs:
 - Calcium channel blockers, ACE inhibitors
- Vasodilating Molecules:
 - Nitric oxide (infection/sepsis)
- Vasodilating conditions:
 - Acidosis, increases in temperature

Compensatory Mechanisms for Hypotension

An adequate blood pressure is important for the function of vital organs including the brain, heart and kidneys. Any reduction in blood pressure will trigger responses to maintain homeostasis.

Blood Pressure = Cardiac Output x Total Peripheral Resistance

Text box 28: Components of BP

Depending on the cause of the reduction in blood pressure will depend on the compensatory response.

Causes

A. Reduction in Cardiac Output ($CO=SV \times HR$):

1. Reduction in Stroke Volume

Response:

- There will be a compensatory increase in heart rate (tachycardia) and a compensatory increase in peripheral vascular resistance (cool, blue peripheries).
While this compensation can return BP to normal values. If CO has not been restored, there may be evidence of persistent inadequate oxygen delivery (text box 29).

Seagull Sign

- Normally the heart rate value is below the Systolic BP.
- If the Heart rate goes above the Systolic BP then this is referred to as a Seagull Sign (See Figure 17)
- BP will only fall once the compensatory mechanisms have failed.
- Visually this may be represented on the chart as a Seagull sign where the heart rate can be seen above the Systolic BP this is a sign of deterioration and medical review should be requested.
- Not all observation charts will record Blood Pressure and Heart rate together so this concept may not be seen in all charts.

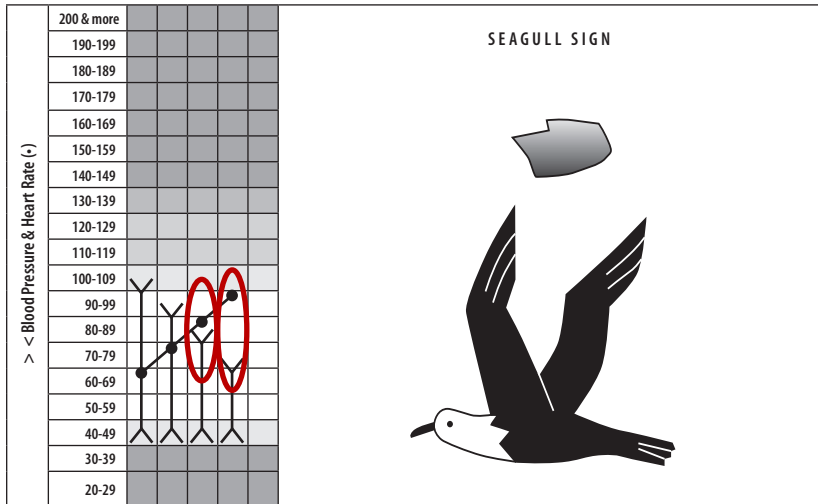


Figure 17: Seagull Sign

- **Reduction in Pre-Load (hypovolaemia):**
Hypotension with a postural drop, tachycardia and cool, mottled peripheries
- **Reduction in Contractility(cardiac failure):**
Hypotension, tachycardia and cool, mottled peripheries with signs of heart failure

Text box 29: Clinical features of a reduction in stroke volume

2. *Reduction in Heart Rate*

Response:

- There will be a compensatory increase in total peripheral vascular resistance to try and maintain blood pressure.

Hypotension, bradycardia and cool, mottled peripheries

Text box 30: Clinical features of Reduction in Heart Rate

B. Reduction in Peripheral Vascular Resistance

Response:

There will be a compensatory increase in cardiac output. Cardiac output will increase by increasing the heart rate (tachycardia) and increasing the contractility of the heart to increase the stroke volume. Cardiac output is further increased following treatment with intravenous fluids to improve venous return.

Hypotension, tachycardia and warm peripheries

Text box 31: Clinical Features of a fall in Peripheral Vascular Resistance

Consequences of Hypotension

The greatest concern is that hypotension may suggest that there is an inadequate amount of oxygen getting to the tissues because of a falling cardiac output, which is described as SHOCK.

$DO_2 = \text{Cardiac Output} \times \text{Arterial Oxygen Content}$

$\text{Blood Pressure} = \text{Cardiac Output} \times \text{Peripheral Vascular Resistance}$

Text box 32: Relationship between blood pressure and oxygen delivery

A. Inadequate Cardiac Output

- Cardiac output is integral to the amount of oxygen being delivered to the tissues. If the cardiac output falls, it is likely that oxygen delivery will fall.
- If there is inadequate oxygen delivery to the tissues, inadequate amounts of ATP can be generated which is vital for cellular function.
- This in turn leads to organ failure, lactate formation and shock.

B. Inadequate Pressure Gradient

- Clearly without a pressure gradient across the vasculature (from high pressure to low pressure) there can be no flow of blood and its constituents including oxygen.
- Some organs are able to maintain blood flow through organs

despite changes in blood pressure (autoregulation) e.g.: brain and kidney. However, there reaches a point when this can no longer occur if the blood pressure is too low. Once this point is reached there is reduced blood flow and hence the amount of oxygen reaching the tissues.

- Inadequate blood flow to the organs results in inadequate oxygen delivery to the organs resulting in reduced generation of ATP and the formation of lactate. This will lead to organ failure (oliguria and altered mentation), lactate formation and shock.

When is hypotension not shock?

In order to demonstrate that there is shock there needs to be evidence that organs are failing and/or that there is evidence of anaerobic respiration by the presence of lactate.

For example: If a patient is hypotensive post anaesthetic and has warm hands (suggesting good flow to the hands i.e. good cardiac output), is not confused, has a good urine output with no signs of heart or respiratory failure and no lactate is found, then the patient is currently not shocked. However, in these situations it is important to continue regular monitoring of the vital signs and continually monitor for evidence of organ failure.

Can a patient with normal or high blood pressure have shock?

The key components to adequate oxygen reaching the tissues are cardiac output and arterial oxygen content. If either of these are reduced there is a fall in oxygen transport to the tissues and this results in shock. Sometimes, the compensatory mechanisms for a fall in cardiac output, such as an increase in total peripheral resistance, can result in there being a normal or even high blood pressure reading. So, despite there being a “normal” blood pressure, there are signs of organ failure and anaerobic respiration i.e. the patient is shocked with a seemingly normal blood pressure.

For example: An elderly lady presents with an inferior myocardial infarction and complete heart block. On examination she has dark blue fingers, a heart rate of 40 beats per minute, her blood

pressure is 210/100 mmHg and she has evidence of pulmonary oedema and oliguria. Her lactate measurement is 10mmol/L (normal < 2 mmol/L). Despite a high blood pressure due to the increase in vascular tone to try and compensate for the fall in cardiac output and blood pressure, there is evidence of not only organ failure but also anaerobic respiration. This patient is shocked despite the high blood pressure.

The Initial Management of Hypotension

It is important to remember what generates a blood pressure:

- Cardiac Output (stroke volume x heart rate)
- Peripheral Vascular Resistance.

It is essential to determine from history and clinical examination, which of these two has decreased leading to a fall in blood pressure.

A Systolic blood pressure of less than 90mmHg in an adult requires a Code Blue (MET referral).

Text box 33: Adult MET criteria for BP

A. Fall in peripheral vascular resistance

- Common causes include infection, and vasodilating drugs.
- History: Chills, fever, symptoms of infection, ingestion/ inhalation of vasodilators.
- Examination: usually accompanied by warm hands (a vasodilated vasculature) and tachycardia. There may be signs of organ failure (confused, oliguria, tachycardia).
- Laboratory Investigations:
 - Evidence of infection (rise or significant fall in white cell count)
 - Evidence of renal dysfunction (rising creatinine)
 - Evidence of lactate formation (metabolic acidosis on arterial blood gas sampling, a negative base excess, a lactate > 2 mmol/L)

Management Plan

- ***In the absence of tachycardia, organ failure, lactate formation***

If there is no evidence of organ failure (not oliguric, not confused), no evidence of anaerobic metabolism (lactate formation) and no associated tachycardia i.e. looks well from the end of the bed. Then there may be no need to do anything other than closely monitor the vital signs (hourly measurements, or according to MEWS protocol triggered) over the next six hours to ensure that there is no downward trend of blood pressure.

- ***In the presence of tachycardia, but absence of organ failure and lactate formation***

The tachycardia could be in response to a fall in venous return (due to pooling in the vasculature) and fall in stroke volume that has not yet affected the amount of oxygen going to the tissues. It is important to improve venous return and stroke volume to maintain adequate cardiac output and oxygen delivery to the tissues:

- Administer intravenous fluid bolus (500 -1000 mls of Normal Saline [0.9% NaCl] for adults)
- Continue to perform frequent vital signs to document any trends (e.g. 1/2hrly for 1 hr, followed by hourly for 4 hours, then subsequently 4-hourly if stable)
- If there is an improvement in tachycardia and blood pressure, then the fluid bolus has been adequate to restore venous return (NOTE- this may only be a compensatory response so continue to monitor frequently)
- If the tachycardia remains repeat the fluid challenge
- Continue to observe response
- If the patient continues to have hypotension, tachycardia and warm hands, further fluid can be administered particularly if there are no signs of heart failure
- An intensive care review should be requested once three litres of fluid have been administered and the tachycardia and hypotension are still present.

- ***Hypotension and evidence of organ failure***

- Administer intravenous fluid bolus (500 - 1000 mls of Normal Saline [0.9% NaCl] for adults)
- Continue to perform frequent vital signs to document any trends (1/2hrly x 1 hr, 1/24 x 4, then 4/24 if stable)
- If there is an improvement in tachycardia and blood pressure, then the fluid bolus has been adequate to restore venous return.
- If the tachycardia, hypotension and organ failure remains, repeat the fluid challenge.
- Call for an intensive care review particularly if the patient has received three litres of fluid or if the signs of organ failure persist
- Continue to perform hourly observations to ensure that the trend of blood pressure, pulse and mental state are being monitored.

B. Fall in Cardiac Output

There are two predominant causes of fall in cardiac output, both having very different presentations:

1. Fall in Pre-Load

- Common causes include bleeding, loss of fluids and electrolytes.
- History
 - Will describe histories relevant to bleeding, loss of fluid and electrolytes (diarrhoea, vomiting, and polyuria from hyperglycaemia), loss of water (diabetes insipidus).
 - Look at fluid balance chart and determine recent fluid balance.
 - Can also describe symptoms of postural hypotension (feels faint when standing up, has actually “fainted”).
- Examination
 - Signs that are relevant to the fluid lost (bleeding into drains, melaena, nasogastric losses)
 - Cool, mottled hands, tachycardia, hypotension with a

postural drop (a drop more than 10mmHg in Systolic BP from lying to sitting)

- Laboratory Investigations
 - Evidence of bleeding (fall in haemoglobin)
 - Evidence of renal dysfunction (rising creatinine)
 - Evidence of lactate formation (metabolic acidosis on arterial blood gas sampling [negative base excess], rising lactate)
- Management
 - Correct cause of loss of fluid (call surgeon for ongoing bleeding, may need to correct coagulopathy)
 - Replace whatever fluid has been lost (blood if bleeding, saline if gut losses, 5% Glucose if diabetes insipidus)
 - Estimate how much has been lost by looking at the fluid balance chart, how much is in the drains, how far has the haemoglobin fallen
 - In the first instance in adults rapidly administer 500 - 1000 mls of Normal Saline via a blood pump set through a large bore intravenous cannula
 - Observe response (tachycardia should be reduced and blood pressure increase)
 - Continue to administer fluid rapidly until there is the desired response:
 - Blood pressure returning to normal
 - Heart rate returning to normal
 - Improvement in organ function, particularly urine output
 - Intensive care should be alerted especially if there are no signs of improvement despite administering 3L of fluid
 - Continue to perform regular observations (hourly or as directed by MEWS protocol)

2. Fall in contractility

- Common causes include myocardial ischaemia or infarction.
- History

- May describe history of chest pain suggesting ischaemia
- May describe previous symptoms of heart failure (orthopnoea, swollen ankles, breathlessness)
- Describe palpitations (suggesting a tachycardia- atrial fibrillation, ventricular tachycardia) or symptoms related to causes of cardiomyopathy
- Examination
 - Cool, blue hands, tachycardia and hypotension.
 - Signs of right heart failure (swollen ankles, raised jugular venous pressure)
 - Signs of left heart failure (tachypnoea, fine inspiratory crackles that do not clear on coughing, third heart sound, low arterial oxygen saturation).
- Investigations
 - Evidence of renal dysfunction (rising creatinine)
 - Evidence of lactate formation (metabolic acidosis on arterial blood gas sampling [negative base excess], rising lactate)
- ECG- signs of ischaemia, infarction, dysrhythmia
- Management
 - If the patient is hypotensive and has signs of organ failure including heart failure (cardiogenic shock), the patient will require inotropic support and referral to either the coronary care unit or intensive care unit
 - Stop all intravenous fluids as the patient is by definition fluid overloaded

When assessing a patient remember to incorporate all the vital signs not just look at an individual reading. Also remember to think about where they sit in the Oxygen Delivery Chain (see figure 18).

Text box 34: oxygen delivery

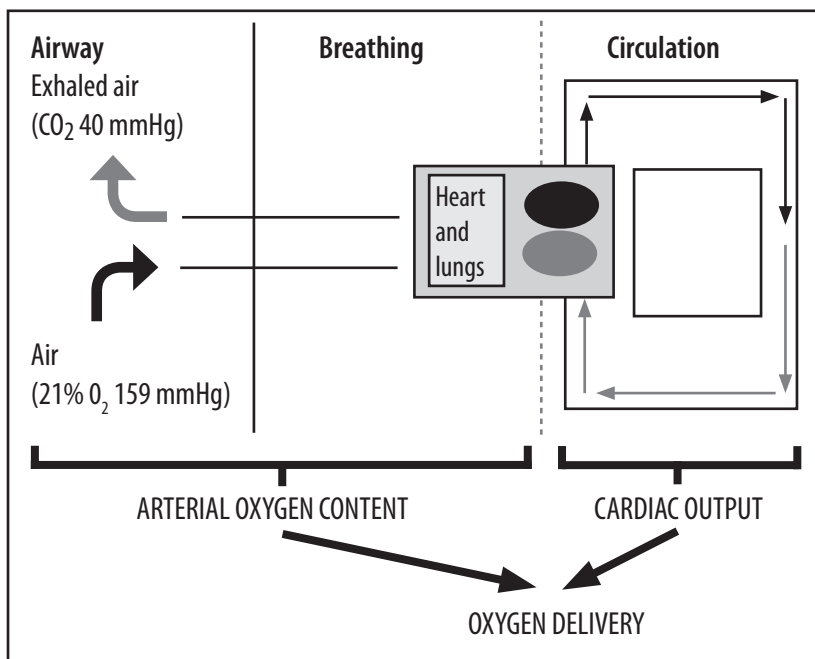


Figure 18: Oxygen Delivery chain

Venous Thromboembolism (VTE)

A hypercoagulable state exists in pregnancy to protect women from haemorrhage but puts them at increased risk of VTE.

Pregnant and postnatal women should be encouraged to be ambulant as much as possible to prevent haemostasis. Women who are unable to ambulate (e.g. post LUSCS) require further protection such as antiembolic stockings and anti coagulant therapy.



SUMMARY

- Blood Pressure = Cardiac Output x Peripheral Vascular Resistance
- Hypotension:
 - High pulse and low blood pressure may reflect low oxygen delivery
 - It is important to remember that someone who is normally hypertensive may be relatively hypotensive even when their systolic blood pressure is above 100mmHg.
 - **In adults do not always use 100mmHg as your CRITICAL Systolic Blood Pressure cut off!**
 - Hypotension can be a marker of a deteriorating patient who is at risk of increased risk of death. A “shocked” patient has signs of organ failure which may or may not accompany hypotension.
- Decrease in cardiac output can be caused by:
 - Decreases in intravascular blood volume
 - Increases in intrathoracic pressure
 - Decrease in peripheral vascular resistance.
- Any decrease in cardiac output can cause a decrease in oxygen delivery.
- The greatest concern is that hypotension may suggest that there is an inadequate amount of oxygen getting to the tissues, which is described as SHOCK.
- Management of hypotension in adults:
 - Hypotension and warm hands:
Administer fluids
 - Hypotension, cool hands, no signs of heart failure:
Administer fluids
 - Hypotension, cold hands, signs of heart failure:
Cease fluids. Refer to CCU/ICU for inotropes.
- **Remember to incorporate all the vital signs in your assessment!**



Central Nervous System and Urine Output

‘Pointing you in the right direction’

Central Nervous System (CNS)

Learning Objectives

- Identify common causes of depressed level of consciousness (LOC).
- Describe how to assess a patient's level of consciousness.
- Describe how to manage a patient with depressed level of consciousness.

Introduction

Depressed level of consciousness is a common finding in acute illness. It can occur due to intracranial disease or as a result of systemic insults (table 13).

Intracranial disease	Meningitis, encephalitis
	Epilepsy
	Cerebrovascular disease, SAH
	Head injury
	CNS infection
Systemic conditions	Hypoxia, hypercapnia
	Hypotension, hypo/hyperosmolar
	Hypoglycaemia, hyponatraemia
	Hypo/hyperthermia
	Hypothyroidism, hypopituitarism, Addison's disease
	Sedative drugs
	Hepatic encephalopathy, uraemic encephalopathy

Table 13: Common causes of decreased level of consciousness

CNS function is an important indicator of adequacy of tissue oxygenation...called "end-organ function". Thus CNS assessment is included in the MEWS. CNS depression in itself can also be associated with life-threatening complications. The most important complication is the associated inability to maintain an adequate airway. Loss of gag or cough reflex is associated with a high risk of aspiration, often resulting in hypoxia and in respiratory failure.

Causes of Depressed Level of Consciousness

1. Inadequate Oxygen delivery

Neurones in the central nervous system, like all other cells in the body, are highly dependent on oxygen. Adequate oxygenation allows the formation of large amounts of ATP "energy packets" which are required for all cellular functions (figure 13).

When oxygen supply is inadequate, insufficient ATP is produced (figure 5), which leads to failure of some cellular functions. This causes the symptoms of confusion or depressed level of consciousness.

Oxygen supply to the cells in the brain depend on the same factors as oxygen supply to all other tissues in the body (text Figure 14).

Thus confusion or decreased LOC can reflect a decrease in oxygen delivery.

- a) decreased cardiac output
 - decrease stroke volume
 - decrease heart rate(This may be indicated by a decreased blood pressure)
- b) decreased arterial oxygen content
 - decreased haemoglobin
 - decreased arterial haemoglobin saturation
- c) decreased blood pressure
 - decrease in cardiac output
 - decrease in peripheral vascular difference

2. Inadequate Substrate Delivery for Metabolism

Cells require a substrate in order to form pyruvate, which enters the Krebs cycle in the mitochondria to produce ATP. Many cells in the body can use glucose, fats or proteins as substrates for energy production. However neurones can only use glucose as their substrate for energy production. Therefore if serum glucose levels fall too low, neurones will stop producing ATP and cellular function will be compromised. Thus confusion or depressed level of consciousness could also result from hypoglycaemia.

Checking the Blood Glucose Level (BGL) is one of the first things, which should be checked on a patient with a reduced level of consciousness, or fitting patient, whether they are diabetic or not. The BGL should be > 3.0 mmol/L.

Text box 37: BGL and decreased level of consciousness

Assessment of CNS

A. Level of consciousness

Sedation Score:

0 = awake & alert

1 = normally asleep, responds to stimuli

2 = mild, occasionally drowsy, easy to rouse

3 = moderate, frequently drowsy easy to rouse but unable to maintain wakeful state

4 = severe, somnolent, difficult to rouse

Text box 38: Criteria used for the sedation MEWS

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Another common method of measuring CNS function is the “Glasgow Coma Scale”. The GCS is not included in the MEWS calculations but may be indicated for specific patients or on specific wards. The GCS is divided into three sections – best motor response, best verbal response, and best eye-opening response (see table 15).

MOTOR	obeys commands	6
	localises to pain	5
	withdraws to pain	4
	abnormal flexion to pain	3
	extension to pain	2
	no response to pain	1
VERBAL	oriented	5
	confused	4
	inappropriate words	3
	incomprehensible sounds	2
	nil	1
EYE	spontaneous	4
	to speech	3
	to pain	2
	nil	1

Table 15: Glasgow Coma Scale

⁶ (Used with permission of Elsevier Limited)

Patients with GCS <8 will almost certainly require intubation as they are unable to protect their own airway. Further assistance will be required with anyone who has this level of consciousness. A MET call is required if the patient suddenly drops their conscious level two or more GCS points (text box 39).

B. Pupillary Size

Pupils should be checked when requested by medical staff, as part of neurological observations, and when there is any reduction in the patient's level of consciousness. A change in the size, equality or reactivity of the patient's pupils is an important clinical sign. This can provide important diagnostic clues (text box 40).

BILATERAL PUPILLARY DILATATION

Causes:

- sympathetic overactivity, e.g. fear, stress, anxiety, hypoglycaemia

- sympathomimetic administration, e.g. administration of adrenaline in an arrest situation
- anticholinergic activity e.g. atropine, tricyclic antidepressants, ipratropium nebuliser

BILATERAL PIN POINT PUPILS

Causes:

- opioids/opiates
- cholinergic drugs-neostigmine, organophosphates
- brainstem CVA

UNEQUAL PUPILS

Causes:

- previous surgery
- prosthetic eye
- eye drops
- brain lesions, aneurysms, infections
- glaucoma

Previous surgery, cataracts or prosthetic eyes can affect pupil size and reaction.

Text box 40: Pupil size

Management of Decreased Loss of Consciousness

1. Check airway and breathing; ensure airway is patent
 - Head tilt, jaw thrust
 - Insert an oropharyngeal (Guedel's) or nasopharyngeal airway
2. Apply high-flow oxygen
3. Measure blood glucose, and correct if $<3\text{mmol/L}$ (administer 50mls of 50% glucose intravenously-this requires a medical order).
4. MET call if patient meets the criteria (GCS fallen >2 points)
5. If respiratory rate or arterial oxygen saturation is decreased, the patient may need ventilatory assistance using self-inflating bag

and mask

6. Ensure intravenous access; 500 mls intravenous fluid bolus may be required if patient is hypotensive.
7. Reverse any drug-induced CNS depression, e.g. naloxone for opioid overdose (requires a medical order)
8. If the airway is patent, and the patient is breathing, place patient supine in lateral recovery position

Once again remember to always incorporate all the vital signs in your assessment.

SUMMARY

- CNS function is an important indicator of adequate oxygenation - "end-organ function".
- It is important to identify and treat the cause of altered level of consciousness
- BGL is one the first things that should be checked in a patient with an altered level of consciousness or who is fitting.

Urine Output

Learning Objectives

- Identify causes of decreased urine output.
- Identify when to be concerned about low urine output.
- Describe the management for low urine output.

Introduction

The kidney is an end-organ, thus poor urine output can be an indicator of patient deterioration due to many causes. This is often one of the earliest signs of overall decline. It is important that the cause of poor urine output is correctly diagnosed.

Pathophysiology

Normal urine flow requires:

1. Adequate oxygenation of the kidneys
2. Adequate perfusion pressure
3. Normal function of kidneys
4. No obstruction to urine flow, e.g. prostatomegaly, renal calculus, blocked catheter, urethral valve disorders, ureterocele

A. Oxygen Delivery

In order to function, renal cells require adequate oxygen delivery, just as all the other cells in the body. Oxygen delivery depends on cardiac output and arterial oxygen content (figure 14).

If oxygen delivery falls to the kidney, urine output will fall. If oxygen delivery is insufficient for renal function, it probably reflects inadequate oxygen delivery to other tissues as well. Therefore urine output can be a sign of the adequacy of whole-body oxygen delivery. The MEWS for adult's urine output is calculated using table 16.

B. Perfusion Pressure

Renal blood flow is autoregulated (i.e. kept constant) throughout a wide range of mean arterial pressures (MAP) (70-170mmHg). The MAP is the perfusion pressure experienced by the organs (figure 19 & 20). This range is increased in chronically hypertensive patients, who then require a higher blood pressure to maintain normal kidney function.

$$\text{MAP} = \frac{(2 \times \text{Diastolic BP}) + \text{Systolic BP}}{3}$$

3

Figure 19: Mean arterial pressure

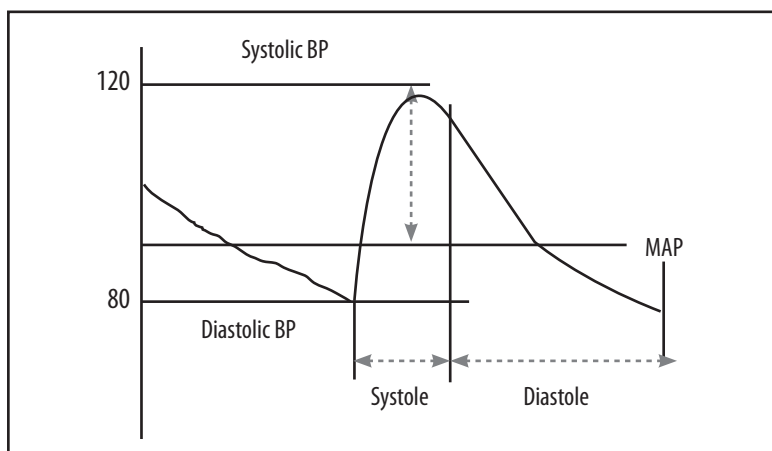


Figure 20: Mean Arterial Pressure Diagram

If mean arterial blood pressure falls below the lower limit of autoregulation, renal perfusion pressure will decrease and thus urine output will fall.

Management of Low Urine Output

The cause of the decreased urine output needs to be determined:

1. Decreased renal blood flow in the face of decreased blood pressure, cardiac output or tissue oxygen delivery.

2. Obstructed urine flow – needs to be urgently corrected if this is the case, thus it is important to diagnose early.

In adults urine output should be > 0.5 mls/kg/hr i.e. 35mls/hr for a 70 kg person.

Text box 41: Urine output

Decreased Renal Blood Flow

This can be due to decrease in Cardiac Output, as a result of

- Decreased stroke volume
 - Decreased pre-load
 - Decreased contractility
 - Decreased after-load
- Alteration in heart rate
- Change in peripheral vascular resistance

There is a small window of opportunity preventing acute renal failure.

Text box 42: Oliguria

Management of Pre-Renal Oliguria

When oliguria is due to decreased perfusion i.e. decreased blood pressure or cardiac output, it is potentially reversible. In this circumstance, the most important initial management is to exclude hypovolaemia (decrease in cardiac preload) being the cause. If hypovolaemia is likely (relative or absolute) give an intravenous fluid bolus of 500 mls of Normal Saline (adults). Frusemide is not to be given unless you have ruled out all other possible reasons for low urine output, and the patient is clinically fluid overloaded.

Giving a fluid bolus will increase circulating volume, thus increase preload, and ultimately increase cardiac output. This will result in increased blood pressure, increased renal perfusion pressure, and ultimately increase the patient's urine output.

Management of Post-Renal Oliguria

Absolute anuria should be seen as a sign of urinary tract obstruction until proven otherwise:

- assess bladder size
- check catheter patency
- if there is no catheter in-situ, the patient may need one inserted.

Do NOT give frusemide to oliguric patients unless you have ruled out all other possible reasons for low urine output, and the patient is clinically fluid overloaded.

Text box 43: Frusemide

SUMMARY

- Checking the **BGL** is one of the first things which should be checked on an unconscious or fitting patient, whether they are diabetic or not.
- Adult urine output should be $> 0.5 \text{ mls/kg/hr}$ i.e. 35 mls/hr for a 70 kg adult.
- There is a small window of opportunity for reversing oliguria and preventing acute renal failure.
- **Do NOT give frusemide to oliguric patients unless you have ruled out all other possible reasons for low urine output, and the patient is clinically fluid overloaded.**





Communication, Team Work and Management Plans

‘Pointing you in the right direction’

Communication, Team Work and Management Plans

Learning Objectives

- To be able to communicate clearly and concisely.
- To understand the use of ISBAR.
- To be able to understand the importance of teamwork.
- To be able to participate in the development of management plans.

One of the most important factors in determining an acutely ill patient's outcome is the quality of the communication among the clinicians involved. In each team, each member has their strengths and weaknesses, varying skills and different levels of knowledge. The aim in managing the deteriorating patient is to determine the role of each member of the team, identify their comfort zones and work together with this knowledge to affect the best outcome for the management of the particular patient. The flowchart in figure 21 gives you a basic outline for management.

Optimising the management of the deteriorating patient requires:

- 1. Gathering as much information as possible**
- 2. Integrating this information into the presentation of the patient**
- 3. Communicating any concerns about a patient to other members of the team**
- 4. Addressing each team member's concerns or respond adequately**
- 5. Formulating, documenting and communicating a management plan with a provisional diagnosis**
- 6. Actioning the management plan**
- 7. Reassessment for possible re-review and escalation of the management plan**

Text box 45: Optimising management

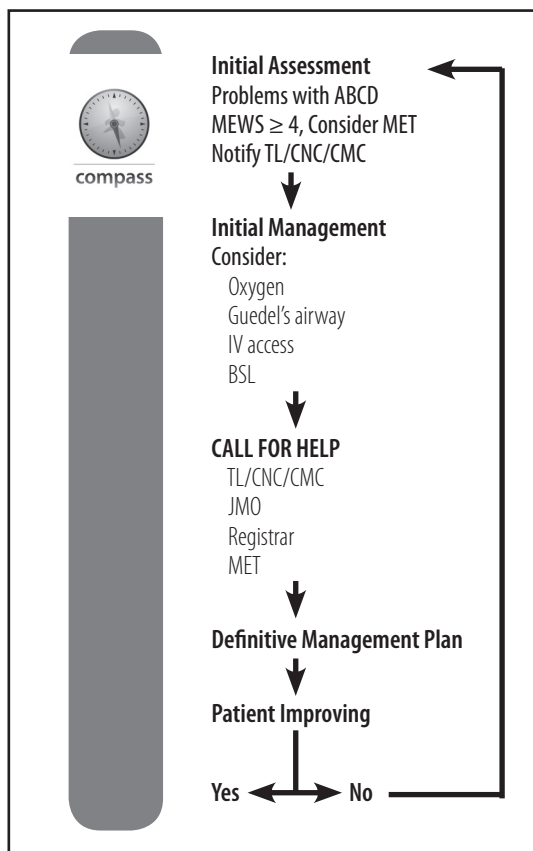


Figure 21: Flow chart of steps in managing a deteriorating patient

1. Gathering Information

Each member of the team provides vital information about the patient's course in hospital and all of this information must be integrated to inform our assessments, decisions and subsequent actions.

Examples:

1. A nurse who has been caring for a patient who is deteriorating will convey significant information about the patient's cognitive state both pre and post deterioration to a medical officer who has reviewed a patient for the first time. This information will further inform the medical officer of the significance of the deterioration.
2. The team physiotherapist may have noticed that a patient's exercise tolerance or arterial oxygen saturations on mobilising have significantly deteriorated. This may alert the team to either a lower respiratory tract infection or pulmonary embolism. This should be communicated to the medical staff and documented in the notes.

It is important in the management of the deteriorating patient to gather as much information from different members of the team as possible.

Information can be obtained from:

- **Verbal contact with members of the team**
- **Reading the daily notes from each different member**
- **Reviewing observation, fluid charts, and medication charts**
- **Comparing current presentation with previous presentations**
- **Family, friends or the patient themselves**

Text box 46: Sources of information

2. Integration of Information

The next step is to integrate the information gathered to fully understand the current situation of the patient. e.g.- the need to

understand why a BP has fallen or why a heart rate or respiratory rate has risen.

3. Communicating Information

Once information has been gathered and thought has been given to what is going on, the next step is working out what to do with the information. This obviously depends on each individual's level of knowledge and understanding. If an enrolled nurse finds an abnormal arterial oxygen saturation, they may refer this information to the registered nurse who is working with them for more guidance about what to do. If a RMO is concerned by a deteriorating patient, then they need to discuss these findings with their registrar and possibly their consultant. The patient must be attended to appropriately.

It is important to recognise when vital signs are abnormal and make sure someone more senior knows about it and that someone is attending the patient appropriately!

Text box 47: Abnormalities and appropriate care

When a high MEWS triggers a communication, describe the observations that have triggered the MEWS (e.g. total MEWS 4 due to Pulse 102, RR 26, Temp 38.7). For a medical officer to be able to appropriately triage and advise on a particular patient, they need to actually know the parameters that have caused the score rather than just a number. We must remember that each member of the team needs to prioritise and attend to many things.

This means health professionals have to:

- identify that there is a problem
- attempt to interpret the problem in the context of the patient we are caring for
- communicate the trigger to the appropriate people for further actioning.

“ISBAR” Communication

The Identify, Situation, Background, Assessment and Recommendation (ISBAR) technique is an easy, structured, and useful tool to help communicate concerns, and call for help or action.

IDENTIFY: Identify yourself, who you are talking to and who you are talking about

SITUATION: What is the current situation, concerns, observations, MEWS, etc

BACKGROUND: What is the relevant background. This helps set the scene to interpret the situation above accurately.

ASSESSMENT: What do you think the problem is? This is often the hardest part for medical people. This requires the interpretation of the situation and background information to make an educated conclusion about what is going on.

RECOMMENDATION: What do you need them to do? What do you recommend should be done to correct the current situation?

Text box 48: ISBAR Communication

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For example:

A 75-year-old lady with a history of Ischemic Heart Disease is admitted with a fractured neck of femur. Twelve hours post-operatively, she complains of chest pain and her arterial oxygen saturation has fallen to 88% on 2L nasal prong. You as the person nursing her are concerned that she is acutely unwell and needs attention. The ISBAR communication technique would proceed as follows:

IDENTIFY

“This is Sarah calling from 7 East about Mrs Smith, is this Dr Jones?”

SITUATION

"She is a 75 year old lady who is dropping her arterial oxygen saturation to 88% on 2L/min of O₂ via nasal prongs, she is tachycardic and tachypnoeic. She is also complaining of chest pain"

BACKGROUND

"She is twelve hours post-op following a fractured neck of femur and she has a history of ischaemic heart disease"

ASSESSMENT

"I think she is acutely unwell and may have"

In this case she may have a pulmonary embolus, a myocardial infarction or pneumonia. If you are not sure what is going on then you can just say that you think she is unwell.

RECOMMENDATION

"I think this patient requires an urgent medical review. I have increased her inspired oxygen in the meantime to 15L/min on a non-rebreather mask"

You have effectively communicated the reason you are calling, given the person some background information that may help them in identifying the cause of the situation, given them an idea of how sick you think the patient is and identified that you feel the patient needs review.

Documentation

Once you have you actioned a particular problem, you always must document what you have done. This may involve documenting low arterial oxygen saturation, and that you have contacted a doctor, or if you are medical officer what treatment you have advised. This documentation has a two-fold purpose:

- it helps the flow of information from one shift to the next and often helps to clarify your own thought processes.
- this is also a medico legal requirement.

You must always identify who needs to know about a deteriorating patient, communicate as much as possible, and document appropriately (text box 46).

When communicating information you must:

- 1. Identify who the most appropriate person is to inform when you encounter a deteriorating patient.**
- 2. Communicate as much information as possible to the next in line to ensure that they have all the information needed to appropriately triage and advise on the situation. Use the SBAR.**
- 3. Document the steps you have taken to remedy the situation and actions taken.**

Text box 49: Communicating

4. Adequate response to information/concerns

After being involved in the management of a deteriorating patient, many people feel that things could have been done better. It might be that they felt the root of the problem was not being addressed and something else was going on, or that they just felt that their particular views were not taken into account. Each member of the team has different priorities with respect to patient management and these need to be integrated into the management plan.

After communicating with more senior colleagues, an individual may feel as if they were not taken seriously or their particular concern about a situation wasn't addressed. This can be remedied by specifically asking each member of the team what their concerns are, how they think this can be addressed, and integrating those concerns into their management plan.

Sometimes people looking after a patient are not sure what an abnormal result means. They feel worried about ringing someone, as they are afraid they might seem stupid or even get scolded for not knowing. This behaviour does not help anyone and there are various communication tools that you can use to overcome this.

For example:

A nurse is told to take the blood pressure in the left and right arm for a person with central chest pain. However, this particular patient may have a fistula or have had a mastectomy and cannot have bilateral blood pressure measurements performed. The doctor comes back to review all the collected information and review the management plan only to find out this has not been done. If the doctor had specifically asked if the nurse had any problems with the plan, the issue would have been identified much earlier, saving everyone time and allowing the appropriate observations to be measured in a timely fashion.

The primary responsibility of the doctor is to stabilise the patient. However, the needs of the ward and the nursing staff need to be integrated into this plan. The nursing staff may feel that the patient cannot be managed in a general ward because of level of nursing care required, but the doctor feels that there is no medical reason that they need step up of their care. This needs to be discussed and a plan endorsed by all members, and agreed on.

Theoretically, in the event of a deteriorating patient (for example at a MET call), all people involved in the patient should be present. ISBAR should be used for communicating during MET calls.

Use the ISBAR strategy when communicating at a MET call as well.

Text box 50: ISBAR

It is the job of the team leader to voice their concerns, pre-empt other people's concerns and integrate that into their management plan. By simply asking what are people's main concerns the team saves time. Often issues are raised that had not been considered and if all team members feel as if their concerns are validated, in the end it benefits the patient's care.

5. Formulating, Documenting and Communicating Management Plans

The make or break of patient care is often in the formulation of management plans. To allow successful flow of information

from one team, one shift and one ward to the next, plans **MUST** be documented. They must be thorough, yet concise and most importantly understandable, legible and logical.

Optimal management plans include action plans for all members of the team and time frames in which things must be actioned. Medical staff must always document their impression, which is the provisional diagnosis (text box 48). When this is done, each member has a clear idea of their roles and responsibilities and no excuses for not following them!

1. Observation Orders

A change in frequency of observations being performed may be needed in a deteriorating patient, for example a person with a blood pressure falling from 150/90 to 98/50 after review, may need their frequency of observations changed so that vital signs are done every half an hour until the blood pressure is above a certain level and stable without intervention.

2. Nursing Orders

More intensive monitoring may be needed if a patient deteriorates, for example changing the bag of an indwelling catheter from a free drainage to an hourly measure bag to monitor urine output more closely.

3. Allied Health Orders

An example of an allied health order is a person who has been diagnosed with hospital-acquired pneumonia. The physiotherapist must know that they now need to do chest physiotherapy intervention on the patient.

4. Change in Therapy Orders

This may include changing antibiotics from oral to intravenous, or adding a diuretic.

5. Investigation/intervention orders

If it has been decided that the patient needs their electrolytes checked then this must be documented, as well as whose responsibility it is to check the results. It is often useful to write what is expected and what to do about abnormal results if this is

predictable. You may now realise that the patient requires IV access for antibiotics that have been ordered.

6. Notification Orders

Guidance from the team as to when to worry, or not to worry in the management of a deteriorating patient is very useful! Notification orders include notifying the doctor when the urine output is less than 0.5ml/kg/hr, or systolic blood pressure less than 100 mmHg. This can alleviate the phone calls from nurse to doctor and also give reassurance to nursing staff about when they need to be concerned in a particular patient. With the MEWS there is the ability to alter the mandatory notification of the medical team in certain situations. These should be documented and communicated verbally to the relevant staff.

6. Actioning the management plan

Everyone must clearly know his or her role and responsibilities in the management plan of the patient. In particular what needs to be done, and then ensuring that it is done!

People must know what to do, must be skilled to do it, must perform the task and then follow-up the results of the task.

7. Reassess

When caring for a deteriorating patient, you must always review them to ensure that your plan or actions have made a difference to the patient. It is NOT adequate to say you have informed someone, discharge your responsibility and forget about the patient. It is as much your responsibility to ensure that something is done, as it the responsibility of the person you informed to come and attend to the patient.

If there is change of shift, then you must ensure that you have verbally conveyed your concerns and outstanding issues with respect to a sick patient to the person taking over the care of the patient so that they will follow them up. If your patient is not improving then you need to reassess them and start at the beginning. Gather the information, initial management, ask for help, and come up with a definitive management plan.

This will be a continuous cycle of review until the patient starts to improve.

When documenting a medical entry always document:

H – history

E – examination

I – impression/diagnosis

P – management plan

Text box 51: Documentation

Management Plans should include:

- a. Observation orders**
- b. Nursing orders**
- c. Allied health orders**
- d. Change in therapy orders**
- e. Investigation/intervention orders**
- f. Notification orders**

Text box 52: Management plans

SUMMARY

- The important thing is to recognise when there is an abnormality in vital signs and make sure someone more senior knows about it and that someone is attending the patient appropriately!
- Use ISBAR when communicating
- When documenting a medical entry always document:
 - H – history
 - E – examination
 - I – impression/diagnosis
 - P – management plan
- Management Plans should include:
 - Observation orders
 - Nursing orders
 - Allied health orders
 - Change in therapy orders
 - Investigation/intervention orders
 - Notification orders





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‘Pointing you in the right direction’

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Quiz Questions

'Pointing you in the right direction'

Quiz Questions

An increased heart rate may reflect:

- a. Failing oxygen delivery
- b. Decreased level of consciousness
- c. Decreased urine output
- d. Rise in stroke volume

Compensation for a decreased oxygen delivery will include which of the following?

- a. Increased urine output
- b. Decreased peripheral vascular resistance
- c. Decreased respiratory rate
- d. Increased heart rate

Cardiac Output =

- a. Blood pressure x heart rate
- b. Stroke volume x blood pressure
- c. Stroke volume x heart rate
- d. Stroke volume x peripheral vascular resistance

Aerobic metabolism:

- a. Generates 2ATP
- b. Produces lactate
- c. Requires oxygen
- d. Causes tachypnoea

Tachypnoea:

- a. Is caused by Morphine
- b. Is caused by metabolic alkalosis
- c. Reflects a deteriorating patient
- d. Is caused by respiratory alkalosis

Blood Pressure =

- a. Peripheral vascular resistance x cardiac output
- b. Stroke volume x heart rate
- c. Stroke volume x peripheral vascular resistance
- d. Cardiac output x heart rate

Oxygen delivery =

- a. PO_2 x cardiac output
- b. O_2 content x cardiac output
- c. PO_2 x blood pressure
- d. Arterial oxygen content x cardiac output

Urine output depends on the following two things:

- a. Frusemide
- b. Oxygen delivery to the kidney
- c. Adequate level of consciousness
- d. pressure in the glomerulus of the kidney

One of the first tests to do for a patient with a decreased level of consciousness is:

- a. Sodium
- b. Potassium
- c. Glucose
- d. Chloride

The Seagull sign is when:

- a. Systolic blood pressure is greater than the heart rate
- b. Heart rate is greater than the diastolic blood pressure
- c. Diastolic blood pressure is greater than the heart rate
- d. Heart rate is greater than the systolic blood pressure

