



Feidhmeannacht na Seirbhíse Sláinte
Health Service Executive

Training Manual for The National Early Warning Score and associated Education Programme



**ACUTE
MEDICINE**

The National Early Warning Score Project and associated Education Programme is a work stream of the Acute Medicine Programme, in association with other National Clinical Programmes, Quality & Patient Safety Directorate, the Patient Representative Group, Nursing and Midwifery Services Director, Clinical Indemnity Scheme, the Assistant National Director, Acute Hospital Services – Integrated Services Directorate, Irish Association of Directors of Nursing and Midwifery (IADNAM) and Therapy Professionals Committee





compass

‘Pointing you in the right direction’

ADULT PROGRAMME

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Copyright

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Disclaimer

The authors, the Australian Capital Territory or the Health Directorate, ACT Government, Australia, other contributors to the Programme, those who modified the training manual, the National Early Warning Score Project Governance and Advisory Groups cannot be held responsible for any loss, damage, or injury incurred by any individual or groups using this Programme.

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This Adult COMPASS Programme and National Early Warning Score (ViEWS) is for use in the HSE, Voluntary and Private acute hospital services in the first instance. This Programme is not relevant for Paediatric and Obstetric services.

Introduction

Compass is an interdisciplinary education programme designed to enhance our health care professionals' understanding of patients who are clinically deteriorating, and the significance of altered clinical observations. It also seeks to improve communication between health care professions, and adopt a patient-centred, quality-driven approach, enhancing the timely management of patients. The COMPASS programme has been modified to suit the Irish health care system with the kind permission of the Health Directorate, ACT Government, Australia. The programme has been developed in conjunction with the national Early Warning Score which incorporates the VitalPAC™ Early Warning Score (ViEWS) vital sign parameters, with the kind permission of Professor Gary Smith (UK). In addition a re-designed general vital sign observation chart for clinical practice areas has been designed using an amended version of CYMRU chart with the kind permission of Professor Chris Subbe (Wales) on behalf of the developers. The programme includes:

- The national Early Warning Score (EWS) using ViEWS parameters
- A vital signs observation chart for clinical practice areas
- An escalation protocol for deteriorating patients (slight modifications may be required for individual sites).

Preamble

It has become apparent, that on occasion, some health care professionals do not manage patients who are deteriorating in an appropriate, timely fashion. This is often as a result of delayed recognition that a patient is deteriorating. Delaying resuscitation and appropriate treatment increases the likelihood of a patient's organs failing due to inadequate oxygen delivery to these tissues. This in turn can lead to unexpected death, unexpected cardiac arrest and unplanned admissions to the intensive care unit. It is important for all health care professionals to understand the key components that contribute to the lack of appropriate patient management.

- A. Absent or inaccurate observations
 - Appropriate equipment not available
 - Equipment malfunctioning
 - Inability of staff to use appropriate equipment due to lack of knowledge
 - Inadequate time to perform observations
 - Lack of understanding of why observations are important
 - General culture that observations are not important
- B. Inability of staff to understand the clinical observations recorded
 - Unable to trend results and interpret their meaning
 - Lack of knowledge
- C. Failure of staff to trigger timely, appropriate response
 - Absence of, or inaccurate observations preventing correct interpretation and delaying appropriate clinical decision making
 - Inability to understand observations recorded
 - Inability to develop a diagnosis

- Inability to develop a treatment plan
- Failure to escalate treatment plan if unable to review, or failure of the patient to improve.

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

An example of what can go wrong

A 60-year-old man was admitted with pancreatitis and a past medical history of hypertension

- On day three of his stay, his systolic blood pressure fell below 90mmHg and whilst fulfilling criteria for immediate medical review as BP EWS score = 3 the patient was not reviewed. Over the following 24 hours, the patient fulfilled EWS calling criteria 13 times, however the patient's condition was never reviewed. The patient died 14 hours later.
- On investigation, the system issues identified were:
 - health care professionals' failure to follow hospital policy in activating EWS escalation plan
 - 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.

Hence, this education programme in conjunction with a clearly-formatted observation chart and the utilisation of a track and trigger system, aims to prevent such cases recurring.

Learning Outcomes

On completing the COMPASS education programme the learner will be knowledgeable in the recognition and management of clinically deteriorating patients.

They will be able to utilise their skills and competencies to provide supportive symptom management until a definitive diagnosis has been made and treatment initiated.

Aims and Objectives

1. Prioritise Care, using
 - Clinical judgement – apply prior and acquired knowledge to enable early recognition and management of the deteriorating patient
 - Decision making skills
 - Guidelines and algorithms
 - An appropriate and timely response – escalate care as required.
2. Show Clinical Reasoning
 - Recognise, interpret and act on abnormal clinical observations
 - Understand the importance and relevance of clinical observations and the underlying physiology
 - Interpret results of investigations
 - Recognise own limitations
3. Appropriate referral of patients
 - Assess severity of illness
 - Recognise the need for specialist assistance
 - Identify the most appropriate environment for the patient

4. Use evidence based medicine
 - Utilise most recent scientific evidence agreed with health care colleagues
 - Work within local and national guidelines and protocols
5. Improve communication and teamworking:
 - Promote the use of more focussed communication between healthcare professionals
 - Communicate the patient status effectively with colleagues (to the right people at the right time)
 - Facilitate teamwork within the multi-disciplinary team for enhanced patient outcomes
 - Develop and action management plans for patients in conjunction with colleagues.

How To Complete This Course

This education programme has three phases. You must complete them in the following order:

1. Work through the CD and Training Manual independently.
2. Complete the paper quiz.
3. Attend the mandatory face-to-face session.

On the CD you will be guided through a patient case study

- You will have access to the patient's history, their current health situation and their observation charts.
- You will be asked a list of questions, 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 question wrong, you will be directed back to the information just covered, to clarify the information in that section.
- When you answer the question correctly, you will then move on to the next section of the case study.

Once you have completed the case study, you will proceed to a multiple-choice quiz to test your knowledge and to practise for the assessment. You will be unable to skip ahead in the CD, however you will be able to go back to review any area that you have already completed if you require any clarification on the content. You **MUST COMPLETE** the CD prior to attending the face-to-face education session you have been scheduled for. You must also complete the paper quiz, which is located at the back of the manual. This is also multiple-choice and you **MUST** complete it and return it to the course co-ordinator at least two days prior to the face-to-face education session.

OK ... LETS GET STARTED!

Early Warning Score

Early Warning Score

Learning objectives

When you have successfully completed this section, you should be able to:

- Calculate an Early Warning Score
- Be aware of your responsibilities when a trigger score (see Text Box 1) is identified

A “vital” sign is a sign that pertains to life, without which life would not exist.

Vital signs constitute pulse, blood pressure, respiratory rate and temperature.

Derangements in pulse and blood pressure measurements can reflect an increased threat to life so can be considered a “vital sign”.

Derangements in pulse, blood pressure, respiratory rate and temperature measurements reflect an increased risk of death. It is important to detect, observe and record these vital signs early, and deliver timely treatment not only to normalise these signs, but also to decrease the risk of the patient dying.

Background

An Early Warning Score (EWS) is a bedside score and track and trigger system which nursing staff calculate from the vital signs recorded, and aims to indicate early signs of a patient’s deterioration.

It is a valuable additional tool to facilitate detection of a deteriorating patient, particularly in acute hospital wards where patients are often quite unwell and there may be many inexperienced staff. Vital signs only include Pulse, Blood Pressure, Respiratory Rate and Temperature; however the ViEWS takes into account other observations as well.

The Early Warning Score (using ViEWS parameters) considers all the patient’s recorded observations together, not just a single observation in isolation. It includes pulse, blood pressure, respiratory rate, temperature, oxygen saturation, FiO₂ (inspired O₂) and AVPU (Alert, response to Voice, response to Pain, Unresponsive) score (see Patient Observation Chart).

Trigger Score

A score of 3 in any single parameter is a trigger point for action.

A score of 2 for Heart Rate \leq 40 (Bradycardia) is a trigger point for action.

A total score of 3 is a trigger point for action, with escalated notification at 4-6 & 7 (see Escalation Protocol Flow Chart).

Text Box 1: Trigger Score

The EWS policy includes:

(individual hospitals must review this and adapt as appropriate)

- Direction to nurses as to what level of doctor needs to be notified, based on the EWS score.
- Direction for nurses on the frequency of vital sign observation measurement once a trigger score is reached.

EWS is beneficial as:

- It provides a point in time for communicating the changes in a patient's vital signs and observations and empowers nurses and junior doctors to take appropriate action.
- **It does not replace clinical judgement when staff are concerned about a patient** (see Text Box 2).
- It assists doctors in prioritising the management of their patients.
- Prompts more timely medical review and treatment of patients as it has an inbuilt escalation policy if the patient has not been reviewed within the required time frame.
- Does not replace calling an Emergency Response System (ERS).

1. EWS does NOT replace calling the cardiac arrest team in the event of collapsed adult with suspected no pulse and/or no breathing.
2. EWS does not replace calling the ERS.
3. EWS does not replace contacting the registrar/consultant for immediate review of any patient that the health care professional is seriously worried about, including a patient with a sudden fall in level of consciousness, fall of GCS > 2 (where this score is in use), repeated or prolonged seizures or threatened airway.

Text Box 2: EWS

NOTE: The Emergency Response System (ERS) must be identified in each acute hospital for daytime, out of hours, weekends etc. as appropriate to their local hospital model. See further information on hospital models in Acute Medicine Programme document on www.hse.ie.

Adult EWS Calculation

To obtain the total EWS:

- 1) Record a full set of vital sign observations on the patient
- 2) Each individual observation is scored according to the criteria outlined below (Table 1)
- 3) Total the score for each observation to achieve a total score.

Table 1: Adult Early Warning Score for each variable

Early Warning Score - ViEWS - Key							
SCORE	3	2	1	0	1	2	3
Respiratory Rate (bpm)	≤ 8		9 - 11	12 - 20		21 - 24	≥ 25
SpO ₂ (%)	≤ 91	92 - 93	94 - 95	≥ 96			
Inspired O ₂ (FiO ₂)				Air			Any O ₂
Systolic BP (mmHg)	≤ 90	91 - 100	101 - 110	111 - 249	≥ 250		
Heart Rate (BPM)		≤ 40	41 - 50	51 - 90	91 - 110	111 - 130	≥ 131
AVPU/CNS Response				Alert (A)			Voice (V), Pain (P), Unresponsive (U)
Temp (°C)	≤ 35.0		35.1 - 36.0	36.1 - 38.0	38.1 - 39.0	≥ 39.1	

Note: Where systolic blood pressure is ≥ 200mmHg, request Doctor to review.

Track and Trigger procedures

If any single parameter scores 3 or the total EWS reaches a trigger score of 3, the activation protocol must be initiated (Text Box 3).

If the total EWS reaches a trigger score of 4-6 & 7, escalated notification must be initiated (see Escalation Protocol Flow Chart, Figure 1):

A. Increase Frequency of Vital Sign Observations

When the total EWS score is **2**, the nurse in charge must be notified and observations increased as per Escalation Protocol. The minimum monitoring recommended is 6 hourly. The frequency of observations may be increased at any stage by the nurse.

With improvement of the patient's condition, the Escalation Protocol may be stepped down as appropriate and documented in the management plan.

When the total EWS score is **3**, the nurse in charge and the Team/On-call SHO must be notified and observations increased as per Escalation Protocol.

Total EWS Trigger Point – Score of 3:

Notify Nurse in charge and Team/On-Call SHO, 4 hourly observations, SHO to review patient within 1 hour.

Text Box 3: Activation Protocol

B. Communicate Score Appropriately

The nurse must notify the CNM/nurse in charge, when a patient reaches a total score of 2 (Text Box 4).

The nurse must notify the nurse in charge and the relevant medical personnel, depending on the EWS as outlined in the Escalation Protocol flow chart (Figure 1).

At the time of a patient reaching a score of 2, the nurse must notify the CNM/Nurse in charge. If the patient reaches a score of 3 or above, the nurse must always notify the nurse in charge and the relevant medical personnel.

Text Box 4: CNM Notification

Resuscitation status should be established and documented in the patient's notes by the primary medical team.

National Early Warning Score Escalation Protocol Flow Chart

Note: In the event of respiratory or cardiac arrest activate the cardiac arrest system. If EWS score is 3 in any single parameter or AVPU score is 3 or GCS (where this score is in use) falls > 2 points contact doctor for immediate review and follow escalation plan.

Figure 1: EWS Escalation Protocol

Escalation Protocol Flow Chart

Total Score	Minimum Observation Frequency	ALERT	RESPONSE
1	12 Hourly	Nurse in charge	Nurse in charge to review if new score ¹
2	6 Hourly	Nurse in charge	Nurse in charge to review
3	4 Hourly	Nurse in charge & Team/On-call SHO	1. SHO to review within 1 hour
4-6	1 Hourly	Nurse in charge & Team/On-call SHO	1. SHO to review within ½ hour 2. If no response to treatment within 1 hour contact Registrar 3. Consider continuous patient monitoring 4. Consider transfer to higher level of care
≥ 7	½ Hourly	Nurse in charge & Team/On-Call Registrar Inform Team/On-Call Consultant	1. Registrar to review immediately 2. Continuous patient monitoring recommended 3. Plan to transfer to higher level of care 4. Activate Emergency Response System (ERS) <i>(as appropriate to hospital model)</i>
Note: Single Score triggers			
Score of 2 HR ≤ 40 (Bradycardia)	½ Hourly	Nurse in charge & Team/On-call SHO	1. SHO to review immediately
*Score of 3 in any single parameter	½ Hourly or as indicated by patient's condition	Nurse in charge & Team/On-call SHO	1. SHO to review immediately 2. If no response to treatment or still concerned contact Registrar 3. Consider activating ERS
<p>*In certain circumstances a score of 3 in a single parameter may not require ½ hourly observations i.e. some patients on O₂.</p> <ul style="list-style-type: none"> • When communicating patients score inform relevant personnel if patient is charted for supplemental oxygen e.g. post-op. • Document all communication and management plans at each escalation point in medical and nursing notes. • Escalation protocol may be stepped down as appropriate and documented in management plan. 			
<p>IMPORTANT:</p> <ol style="list-style-type: none"> 1. If response is not carried out as above CNM/Nurse in charge must contact the Registrar or Consultant. 2. If you are concerned about a patient escalate care regardless of score. 			

Adapted from CJKDKU chart

Escort required out of ward area: consider expertise of personnel and equipment required for safe transport.

Where a patient is being continuously monitored using electronic technology, a full set of vital signs data must be documented on the observation chart, as per Escalation Protocol.

Summary EWS Adult

Trigger score: A score of 2 for Heart Rate ≤ 40 (Bradycardia) is a trigger point for action. A score of 3 in any single parameter or total EWS of 3 is the trigger point for action, with escalated notification required at EWS of 4-6 and 7 or if the patient is not improving.

- 1. EWS does NOT replace calling the cardiac arrest team in the event of collapsed adult with suspected no pulse and/or no breathing.**
- 2. EWS does not replace calling the ERS as appropriate to local hospital model.**
- 3. EWS does not replace contacting the registrar/consultant for immediate review of any patient that staff are concerned about, including those who experience a sudden fall in level of consciousness, fall of GCS >2 , repeated or prolonged seizures or threatened airway.**

The Early Warning Score guides the Escalation Protocol.

The Early Warning Score does not replace clinical judgement when staff are concerned about a patient

In the next few sections you will be taken through each vital sign individually. Always remember that you must consider all the clinical observations when assessing a patient, and not just a single parameter in isolation.

Note: The scoring parameters for the physiological signs identified in the Nationally agreed Early Warning Score (ViEWS) in Table 1, must be strictly adhered to.

OXYGEN DELIVERY

Oxygen Delivery

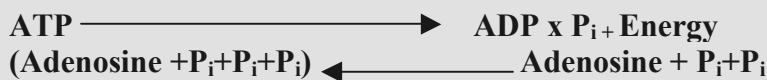
Learning Objectives

When you have successfully completed this section, you should be able to:

- Explain the importance of oxygen delivery to health
- State and explain the factors that affect adequate oxygen delivery

Background

Oxygen is essential for the adequate production of adenosine triphosphate (ATP) by cell mitochondria (Figure 2). 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 5).



Text Box 5: Energy Release

If there is inadequate oxygen supply, ATP production falls, and cellular function is then depressed (Figure 3), through lack of energy. Therefore, reduced levels of oxygen supply at cellular level may cause organ failure, which at best leads to the unplanned admission of a patient to ICU and at worst a patient's death.

Therefore if oxygen delivery is maintained, this may reduce the incidence of unplanned ICU admissions and unexpected deaths.

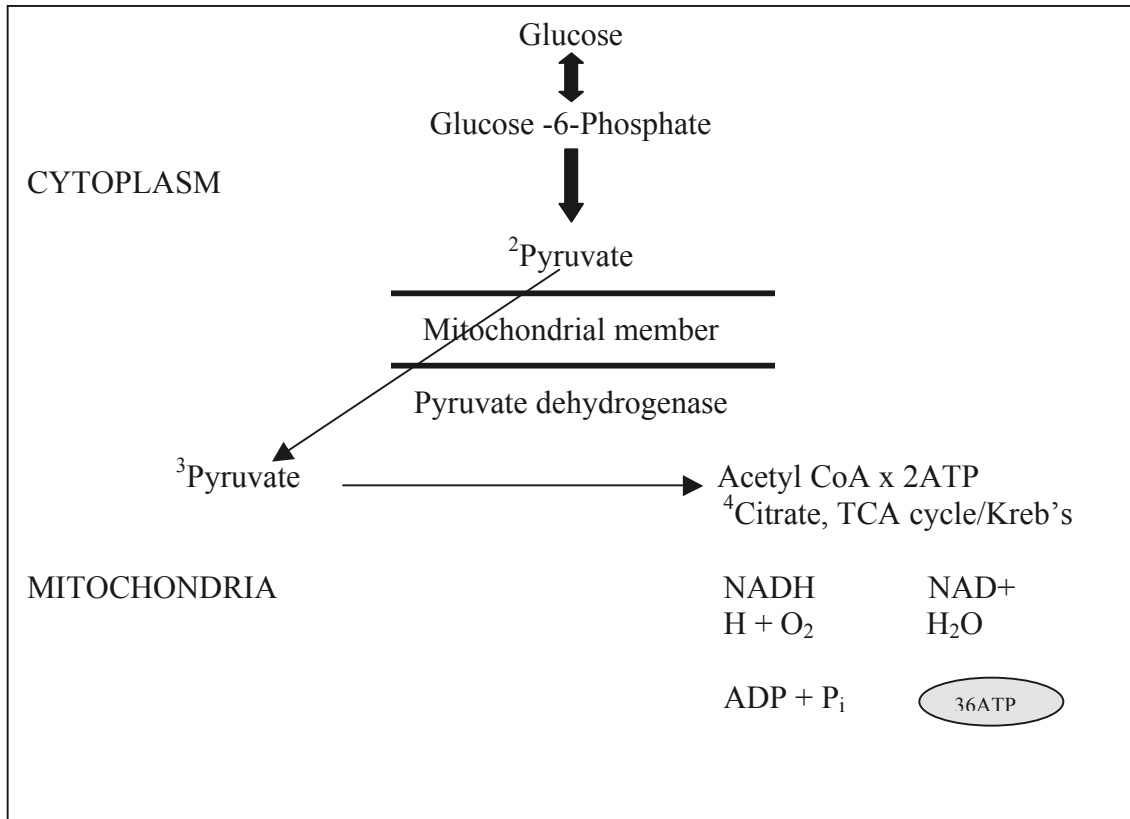


Figure 2: Aerobic Metabolism (i.e. with oxygen)

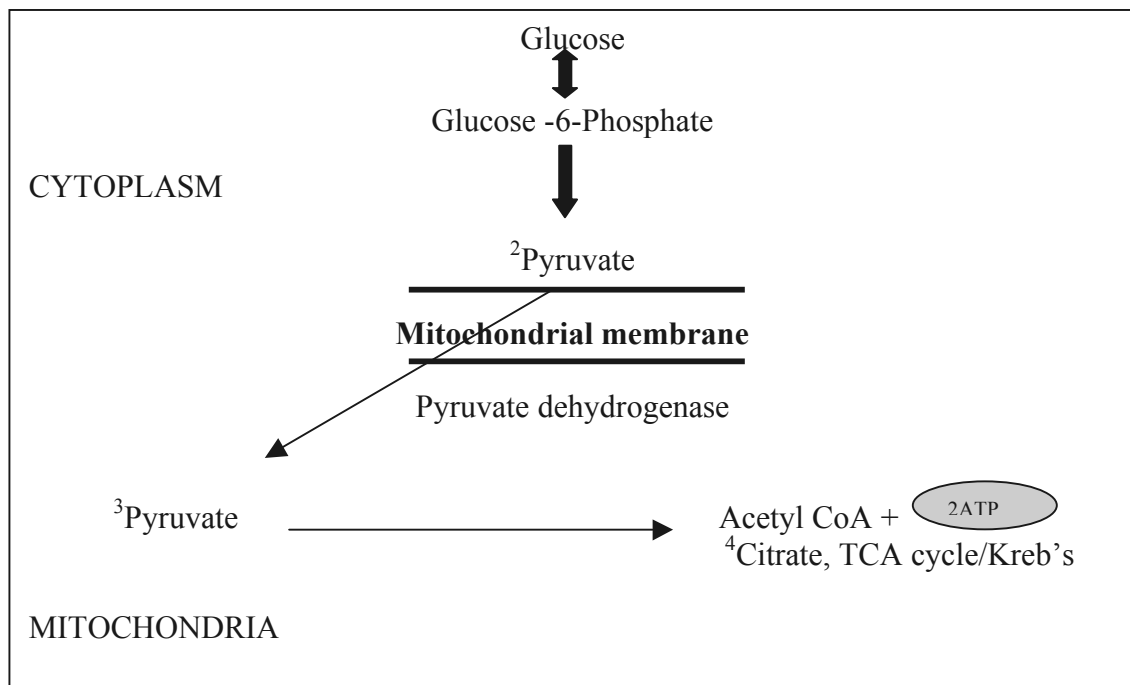


Figure 3: Anaerobic Metabolism (i.e. in the absence of oxygen)

Oxygen supply to the cells can be described by the “oxygen delivery chain” (Figure 4)

$$\text{Oxygen Delivery} = \text{Cardiac Output} \times \text{Arterial Oxygen content}$$

Thus oxygen delivery requires

A. Arterial oxygen content:

- haemoglobin concentration (Hb)
- haemoglobin oxygen saturation (SaO_2)
- partial pressure of oxygen (PaO_2)

SEE SECTION ON “AIRWAY AND BREATHING”

B. Cardiac output – SEE SECTION ON “CIRCULATION”

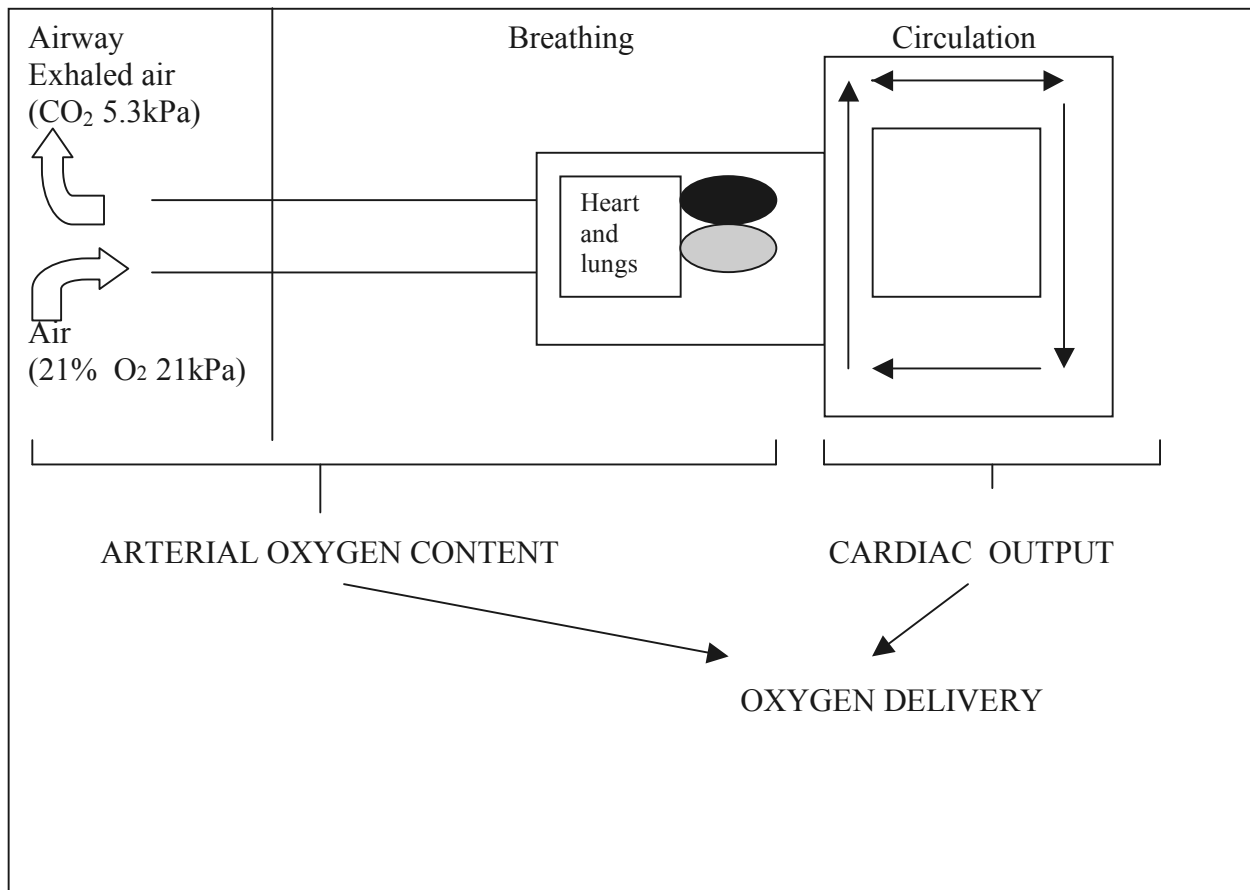


Figure 4: ABC and the Oxygen Delivery Chain

SUMMARY

- Oxygen is essential for the adequate production of adenosine triphosphate (ATP)
- If there is an inadequate oxygen supply, ATP production falls, and cellular function is then depressed.
- $\text{Oxygen Delivery} = \text{Cardiac Output} \times \text{Arterial Oxygen content}$

AIRWAY and BREATHING

Airway and Breathing

Learning Objectives

When you have successfully completed this section, you should be able to:

- Recognise when difficulties with a patient's airway or breathing may compromise oxygen delivery to the tissues.
- Apply the appropriate oxygen delivery device
- Manage appropriately a patient with impaired arterial oxygenation
- Explain why the respiratory rate is such an important marker of the deteriorating patient.

Introduction

In order for oxygen to reach haemoglobin 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 an intact 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 alveolar capillary membrane into the blood, and attaches to haemoglobin. From here, it is dependent on the pulmonary and then the systemic blood circulation to move the oxygen to the tissues and cells where it is required.

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). Mechanical obstruction 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.

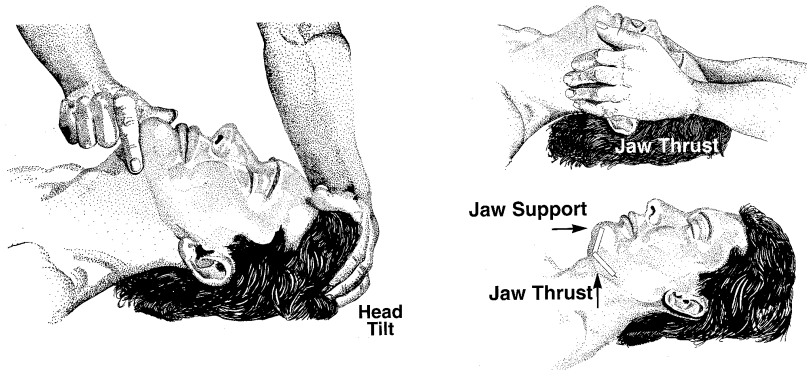
- **Look:** Complete airway obstruction can cause paradoxical chest and abdominal movements (“see-saw” like movement, where inspiration is associated with outward movement of the chest, but inward movement of the abdomen). Other signs of airway obstruction include use of accessory muscles (neck and shoulder 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 of the airway.

Management of the Obstructed Airway

In the majority of patients in hospital, the patient’s airway obstruction is functional, i.e. due to a depressed level of consciousness. Simple clinical manoeuvres may be required to reopen the airway (see Figure 5):

1. Head tilt
2. Chin lift
3. Jaw thrust
4. Insertion of an oropharyngeal or nasopharyngeal airway.

Figure 5: Simple Airway Manoeuvres



Suctioning of the airway using an oropharyngeal sucker may be required to remove any vomitus or secretions, which may be contributing to airway obstruction. Due care must be taken when performing oral suctioning so as not to further compromise the patient’s airway.

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

In all patients with an airway obstruction or patients who are unable to maintain an adequate airway, activate the cardiac arrest system (including anaesthesia) or ERS. 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, therefore activate the cardiac arrest system or ERS.

Text Box 6: Airway Obstruction

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

Breathing

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

- An intact respiratory centre in the brain
- Intact nervous pathways from brain to diaphragm and intercostals muscles
- Adequate diaphragmatic & intercostals 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 of abnormalities in breathing.

Look

Respiratory rate is an important marker of a deteriorating patient (Text Box 6). 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:

- Abnormal respiratory rate
- Use of accessory respiratory muscles
- Sweating/pallor
- Central cyanosis
- Abdominal breathing
- Shallow breathing
- Unequal chest movement

Listen

Initially listen 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
- 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 e.g. consolidation, pneumothorax

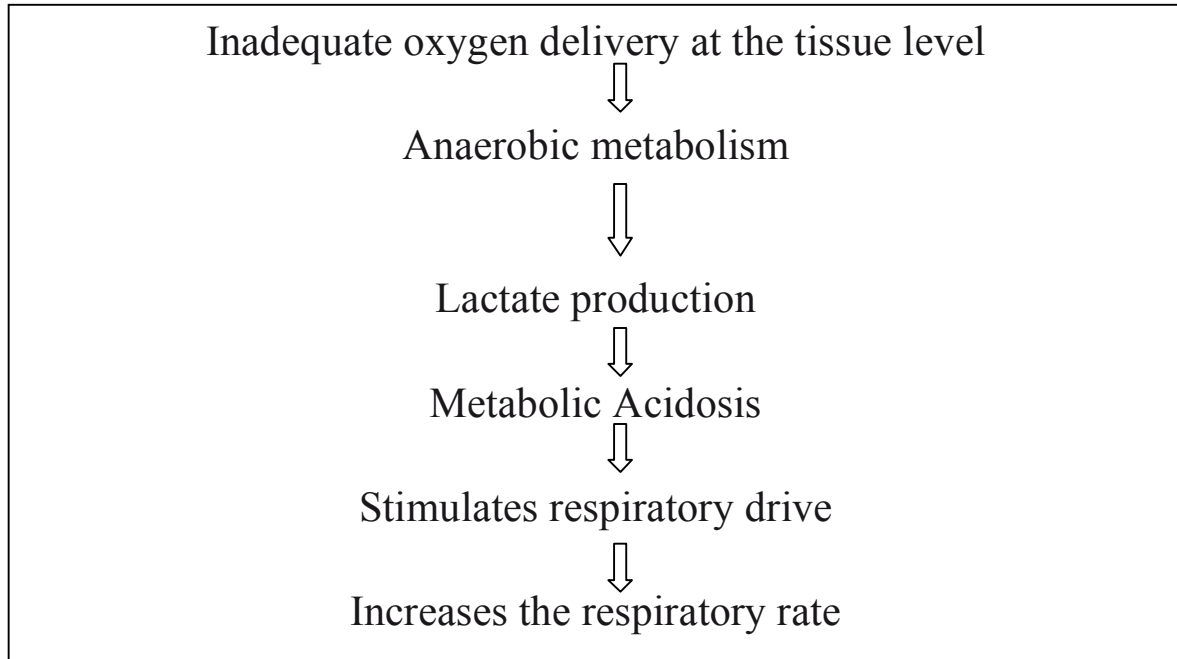
2. Percussion:

- Hyper-resonance indicates pneumothorax
- Dullness indicates consolidation or pleural fluid.

Why Respiratory Rate is Important

An increase in a patient's respiratory rate can reflect either a drop in arterial blood oxygen saturation level or reflect compensation for the presence of 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 the accumulation of acid (see Figure 6). This accumulation of lactic acid stimulates an increase in respiratory rate (tachypnoea).

Figure 6: Importance of Respiratory Rate



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 4).

Metabolic acidosis can increase the respiratory rate even though the arterial oxygen saturation may be normal

Text Box 7: Metabolic Acidosis, Respiratory Rate and Arterial Oxygen Saturation

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 normal or low arterial oxygen saturation levels and may well be a better indicator of a deteriorating patient than arterial oxygen saturation.

Respiratory rate, SpO₂ EWS

The respiratory rate, arterial oxygen saturation and inspired O₂ (FiO₂) score (Table 3) for the Adult EWS are as follows:

Table 3: Adult Respiratory EWS

SCORE	3	2	1	0	1	2	3
Respiratory Rate (bpm)	≤ 8		9 - 11	12 - 20		21 - 24	≥ 25
SpO ₂ (%)	≤ 91	92 - 93	94 - 95	≥ 96			
Inspired O ₂ (FiO ₂)				Air			Any O ₂

The EWS is noted for each individual parameter and will make up part of the total EWS. Be aware that if a patient is maintaining a normal saturation, but their oxygen demands have increased (that is, they need more oxygen to maintain the normal level) then the patient is deteriorating.

Respiratory rate score of 3 requires immediate medical review.

Text Box: 8 Adult Respiratory Rate Notification

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 SpO_2 of $\geq 96\%$ in those patients not at risk for hypercapnoeic respiratory failure and the PaO_2 as close to 13kPa as possible, but at least 8kPa (SaO_2 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 10).

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

In a small subgroup of patients who have Chronic Obstructive Pulmonary Disease (COPD) and are “CO₂ retainers” or their risk factors for hypercapnoeic respiratory failure are increased (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 of care for these patients is to achieve PaO_2 of 8kPa or saturation of greater than 90% on pulse oximetry. Therefore in a patient with COPD who has a $\text{PCO}_2 > 8\text{kPa}$, but is also hypoxic, $\text{PO}_2 < 8\text{kPa}$, do not turn the inhaled O_2 down, however, do not leave the patient unattended. If their PO_2 is $> 8\text{kPa}$ then you can turn the inhaled O_2 down to maintain $\text{SaO}_2 > 90\%$

Text Box 9: 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 4) e.g. the Venturi mask (Figure 7).

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

Figure 7: Venturi Mask System

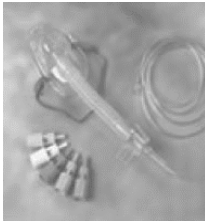


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Table 4: Relationship Between Inspired Oxygen and Oxygen Flow Rate with Venturi

Diluter Colour	Diluter setting (Inspired Oxygen)	Suggested oxygen flow rate (Litres/min)
Blue	24%	2
White	28%	4
Yellow	35%	8
Red	40%	8
Green	60%	15

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 re-breathing and non-re-breather masks.

- A) Nasal prongs (Figure 8) – The dead space of the nasopharynx is used as a reservoir for oxygen, and when the patient inspires, entrained air mixes with reservoir air, effectively enriching the inspired gas. Oxygen flow rates of 2-4L/min

- B) High flow nasal prongs – these use warm humidified oxygen at higher flow rates 4-8L/min

Figure 8: Nasal Prongs

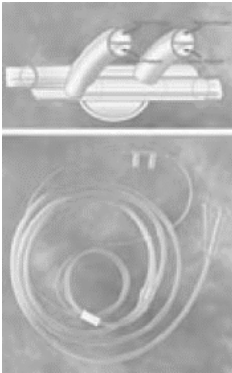


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- C) Oxygen facemasks (Figure 9)– reservoir volume of oxygen is increased above that achieved by the nasopharynx (Text Box 10), thus higher oxygen concentration can be achieved in inspired gas (max 50-60%)

Figure 9: Oxygen Mask

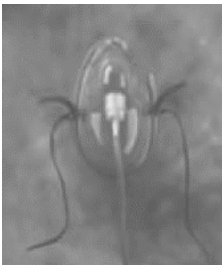


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- D) Non-re-breathermask (Figure10) – A simple face mask with the addition of a reservoir bag, with one or two-way valves over the exhalation ports which prevent exhaled gas entering the reservoir bag (permits inspired oxygen concentration up to 90%). Oxygen flow rate of 12-15L/min.

Figure 10: Non-rebreather Mask

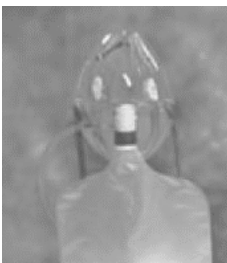


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Monitoring and Titrating Oxygen Therapy

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 are measured, as well as the metabolic status (including lactate).

Oxygen needs to be prescribed by a doctor.

In urgent situations, where oxygen is applied or the amount increased, a doctor must review the patient.

Text Box 10: Oxygen

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 a PO_2 of 8kPa.

Patients do not die from a raised CO_2 alone: they die from hypoxaemia (Text Box 11)

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

Text Box 11: Arterial Blood Gases

As long as the concentration of oxygen being delivered is recorded, the degree of hypoxaemia can be calculated. The blood gas machine can calculate this for you as long as the correct inspired oxygen concentration is recorded.

SaO_2 refers to the directly measured in vitro measurement of arterial oxygenation usually via ABG. SpO_2 refers to the indirect assessment using the oximeter.

Text Box 12: SaO_2 and SpO_2

Directly Measured Arterial Oxygenation

Arterial blood gases can be sampled and analysed in a standard fashion. These provide the gold standard method of measuring oxygenation. The ratio of oxygen carrying haemoglobin compared to the total amount of haemoglobin can be accurately measured. This value can be presented as a percentage which is termed SaO_2 . Arterial blood gas measurement is invasive.

Indirectly Measured Oxyhaemoglobin

Pulse oximetry measures the presence of oxyhaemoglobin indirectly by measuring the absorption of light at certain frequencies. The absorption of light can be related to the presence of oxyhaemoglobin. This is usually done with a finger probe. It is possible to provide an estimate of indirectly measured oxyhaemoglobin concentration as a percentage of total haemoglobin. This is defined as the SpO_2 (Text Box 12). Pulse oximetry is non-invasive but can be affected by variables such as movement, the presence of unusual haemoglobin varieties, including carboxyhaemoglobin, and nail varnishes.

Oximeters can be unreliable in certain circumstances (Text Box 13), 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 that it is broken...the patient may have poor perfusion!

Text Box 13: 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 14) nor does it determine the adequacy of oxygen delivery to the tissues.

**Oxygen saturation may be “normal” but the PCO_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 14: Normal SpO_2 Does Not Rule Out Respiratory Failure

Arterial blood gases remain the gold standard for assessing respiratory failure. 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 who

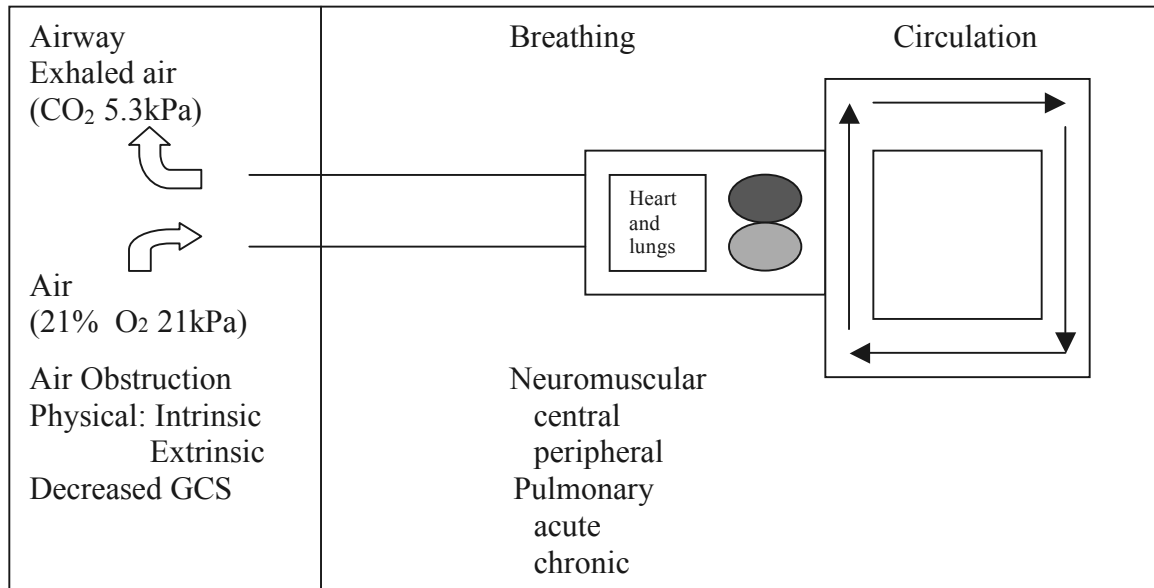
- Are critically ill
- Have deteriorating oxygen saturations or increasing respiratory rate
- Requires significantly increased supplemental oxygen to maintain oxygen saturation
- Have risk factors for hypercapnoeic respiratory failure
- Have poor peripheral circulation and therefore unreliable peripheral measurements of oxygen saturation.

When assessing a patient remember to incorporate all the vital signs, do not just look at an individual reading (Text Box 15). If you suspect failing oxygen delivery, consider where in the oxygen delivery chain may be disordered (see Figure 11).

Remember to incorporate all the vital signs in your assessment!

Text Box 15: Vital Signs

Figure 11: ABC – Oxygen Delivery Chain



Some Questions and Answers for Patients with Chronic Obstructive Pulmonary Disease (COPD)

Q. Will a high concentration of inspired oxygen have an effect on the respiratory drive of my patient with Chronic Obstructive Pulmonary Disease?

A. This is a common concern. Many patients with COPD rely on a “hypoxic” respiratory drive. Their usual PaO_2 may be lower than the normal range e.g. 8kPa (normal 11-15kPa). Delivery of high concentrations of oxygen to achieve a PaO_2 within the normal range may indeed suppress the respiratory drive in these patients causing the PCO_2 to rise. However, when patients with underlying COPD deteriorate, e.g. pneumonia, high concentrations of inspired oxygen may be required to treat hypoxia, the aim being to return the PaO_2 to what is considered an acceptable level for this patient – usually 8kPa. Prompt treatment of severe hypoxia is vital, failure to do so will result in cardiac arrest earlier than a raised PCO_2 . Administer enough oxygen as prescribed to achieve an $\text{SpO}_2 > 90\%$ and a PaO_2 of 8kPa. If the PaO_2 rises above 8kPa, you may turn down the inhaled oxygen as prescribed to achieve an SpO_2 of $> 90\%$. It is very important not to leave the patient unattended during this time.

Q. Should I discontinue oxygen therapy temporarily while the deteriorating patient has a sample of arterial blood taken for Arterial Blood Gas (ABG) analysis?

A. No.

Q. What should I do if a COPD patient develops respiratory acidosis?

(pH < or = 7.35 and respiration > 25 breaths per minute)

A. Consider institution of Early Non-invasive Ventilation

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 and requires activation of the cardiac arrest system (including anaesthesia) or ERS.
- 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 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 a PaO₂ of 8kPa, or oxygen saturation of 90% on pulse oximetry. So, in a patient with COPD who has a PCO₂ >8kPa but is also hypoxic, PO₂ <8kPa do NOT turn the inhaled O₂ down however do not leave them unattended. If their PO₂ is >8kPa then you can turn inhaled O₂ down to maintain SaO₂ >90%.
- When the patient is acutely ill, it is unnecessary to remove the oxygen mask when taking an arterial blood gas sample, as it 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.
- Respiratory score of 3 requires immediate review by doctor.

Circulation

Circulation

Learning objectives

When you have successfully completed this section, you should be able to:

- Explain why pulse rate and blood pressure are “vital signs”, and the importance of measuring them.
- Describe the mechanisms which generate blood pressure.
- Define, describe causes of, consequences of and compensation for development of hypotension.
- Explain what is meant by shock
- Manage hypotension in the deteriorating patient.

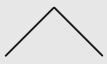
The Importance of Oxygen

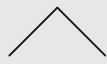
Oxygen reaching the cells and mitochondria is dependent upon adequate amounts of oxygen being delivered via the blood circulation (Figure 12). Without oxygen being delivered to the mitochondria, inadequate amounts of ATP are generated and cellular dysfunction occurs. Oxygen delivery's key components are:

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

Figure 12: Oxygen Delivery

$$\text{Oxygen delivery} = \text{Cardiac output} \times \text{Arterial Oxygen Content}$$


Stroke Volume x Heart rate


Haemoglobin x SaO₂

Blood Pressure

Blood Pressure, Heart Rate and Oxygen Delivery

Blood pressure is the product of cardiac output and total peripheral resistance (TPR)

Blood Pressure = Cardiac Output x Total Peripheral Resistance

Text Box 16: Blood Pressure

- A decrease in blood pressure can reflect a decrease in cardiac output 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 are important surrogate markers of whether there is adequate cardiac output and hence oxygen delivery to the tissues.

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

Text Box 17: Relevance of Pulse and Blood Pressure to Oxygen Delivery

Calculation of Adult EWS for heart rate:

Table 5: Adult Heart rate EWS

Score	3	2	1	0	1	2	3
Heart Rate (BPM)		≤ 40	41 - 50	51 - 90	91 - 110	111 - 130	≥ 131

Adult Heart Rate EWS Triggers

A heart rate of ≤ 40 beats per minute or ≥ 131 beats per minute requires immediate review by Doctor, consider activation of ERS

Text Box 18: Low Heart Rate

Blood Pressure and Maintenance of Organ Function

There are some organs that require an adequate blood pressure for their optimal function as well as adequate oxygen delivery. The brain and kidney are two examples of these organs.

Calculation of Adult EWS for blood pressure:

Table 6: Adult Blood Pressure EWS

SCORE	3	2	1	0	1	2	3
Systolic BP (mmHg)	≤ 90	91 - 100	101 - 110	111 - 249	≥ 250		

Note: Where systolic blood pressure is ≥ 200mmHg, request Doctor to review.

Q. Is the Systolic BP Parameter of 111-249 = Score of 0 correct?

A. Yes. We have been advised that ViEWS is a risk prediction model. Therefore the weightings have been chosen based on achieving the best AUROC* for predicting death within 24 hours of a given observation set. Changing any of the weightings invalidates the score and the performance of the 'modified' system is likely to be harmed. More importantly, it may lead to excessive workload, which is something that should be avoided as it tends to undermine the whole concept of early intervention.

The BP range is weighted based on evidence relating to the chosen outcome. It doesn't mean that extreme BP's are unimportant and do not need a doctor's involvement, in the same way that a nurse who is concerned about a patient should not exclude a review by a doctor.

The approach of placing a note on the chart that if any specific parameter exceeds a given value that a doctor should be called to review the patient is acceptable.

A systolic BP greater than or equal to 200 requires a doctor to review.

* *Area Under a Receiver Operating Characteristic (AUROC) Curve.*

The Area under the Receiver Operating Characteristic is a common summary statistic for the goodness of a predictor in a binary classification task. It is equal to the probability that a predictor will rank a randomly chosen positive instance higher than a randomly chosen negative one.

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 19: 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 plan 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) Cardiac contractility
The ability of the heart to contract in the absence of any changes in preload or after load – it reflects the cardiac muscle 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 overstretches 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, water loss, diarrhoea, vomiting, 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 Box 20: Relationship between Intravascular Blood Volume and Blood Pressure

b) Intrathoracic pressure

An increase in intrathoracic pressure (e.g. asthmatic attack, positive pressure ventilation) will restrict the amount of blood returning to the heart, decreasing venous return and therefore reduce stroke volume.

Increases in intra thoracic pressure can decrease cardiac output and therefore decrease blood pressure

Text Box 21: Relationship between Intrathoracic Pressure and Blood Pressure

3) After load

This is the resistance of 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 precapillary sphincters. As resistance rises, stroke volume is reduced.

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

Text Box 22: 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, sympathomimetics (adrenalin, noradrenalin, 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 change in heart rate can affect 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 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. As a result cardiac output may cause a drop in blood pressure (Text Box 23). 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 rate slows there may be no reduction in cardiac output. As the ventricle has a longer time to fill, the end diastolic volume is increased with each beat, it stretches the myocardial fibres and increases the stroke volume per beat. This mechanism 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 phenomenon 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 23: 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

- a. **Stimulation of Sympathetic Receptors:** Sympathetic stimulation ($\alpha 1$) 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 stimulus discharged from them

to the vasomotor centre with a resultant increase in sympathetic discharge e.g. Sympathomimetics that stimulate the $\alpha 1$ receptor will cause vasoconstriction of the arterioles, examples include noradrenaline, adrenaline.

- b. Direct action on arteriole smooth muscle: Examples include vasopressin, angiotensin, methylene blue (a vasoconstrictor by inhibiting nitric oxide action on the vasculature).

2. Decrease in peripheral vascular resistance

a. Blockage 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. $\alpha 2$ 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, increase 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 the body to respond in order to maintain homeostasis.

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

Text Box 24: Blood Pressure

The body's compensatory response to a fall in blood pressure is dependent on the cause of the drop in blood pressure.

Causes

- A. Reduction in Cardiac Output ($CO=SV \times HR$)
1. Reduction in Stroke Volume

Response

- There will be a compensatory increase in the heart rate (tachycardia) and a compensatory increase in peripheral vascular resistance (cool, blue peripheries).

While this compensation mechanism can return the BP to normal values, if Cardiac Output has not been restored, there may be evidence of persistent inadequate oxygen delivery (Text Box 25).

- **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 25: 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 in an attempt to maintain a near-normal blood pressure

Hypotension, bradycardia and cool, mottled peripheries

Text Box 26: 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 be increased by an increase in the heart rate (tachycardia) and by an increase in the contractility of the heart which results in an increase in the stroke volume. Cardiac output is further increased following treatment with intravenous fluids to improve venous return.

Hypotension, tachycardia and warm peripheries

Text Box 27: 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 28: 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, despite changes in blood pressure (autoregulation) e.g. brain and kidney. However, the body reaches a point when this compensation mechanism can no longer occur, that is, if the blood pressure remains too low. Once this point is reached there is reduced blood flow to the organs resulting in a reduced amount of oxygen being delivered to the tissues and organs.
- Inadequate blood flow to the organs results in inadequate oxygen delivery to the organs resulting in reduced generation of ATP and consequently the formation of lactate. This process will lead to organ failure (oliguria and altered mentation), lactate formation and shock.

Q. When is hypotension not shock?

A. 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 patient is hypotensive post anaesthetic and has warm hands (suggesting good blood 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, it is important to continue regular monitoring of the patient's vital signs and to continually monitor for evidence of organ failure.

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

A. The key components of adequate oxygen reaching the tissues are cardiac output and arterial oxygen content. If either of these two elements 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 for a short period of time. Therefore, despite there being a “normal” blood pressure reading, there are other clinical 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 210/100mmHg and she has evidence of pulmonary oedema and oliguria. Her lactate measurement is 10mmol/l (normal <2mmol/l). Despite a high blood pressure reading due to the increase in vascular tone to try and compensate for the fall in cardiac output, there is evidence of organ failure and 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 the patient’s medical history and clinical examination, which of these two factors have decreased leading to a fall in blood pressure.

A systolic blood pressure of less than 90 mmHg in an adult requires immediate review by doctor.

Text Box 29: Adult ERS Criteria for BP Trigger

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 (confusion, 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 base deficit, a lactate > 2mmol/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. the patient looks well from the end of the bed. Then there may be no need to do anything medically other than close monitoring of the patient’s

vital signs measurements, as per EWS escalation protocol triggered over the following hours to ensure that there is no downward trend in 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 an intravenous fluid bolus (500-1000 mls of normal saline (0.9%NaCl) for adults).
 - Continue to perform frequent vital signs as per escalation protocol, document any trends.
 - If there is an improvement in the tachycardia and blood pressure then the fluid bolus has been adequate to restore venous return at this time. (NOTE – this may only be a compensatory response and therefore temporary, for that reason continue to monitor the patient’s vital signs frequently).
 - Consider Sepsis Six regimen
 - If the tachycardia is not resolved repeat the fluid challenge.
 - Continue to observe response – monitor and record vital signs.
 - If the patient continues to have hypotension, tachycardia and warm hands, further fluid can be administered (check amount with doctor as this will be the third fluid challenge) particularly if there are no signs of heart failure.
 - An intensive care review should be requested when 3 litres of fluid have been administered and the tachycardia and hypotension have not been resolved.
- 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 in the patient’s condition (as per escalation protocol).
 - If there is an improvement in the patient’s tachycardia and blood pressure, then the fluid bolus has been adequate to restore venous return.
 - If the tachycardia, hypotension and organ failure remain, repeat the fluid challenge.
 - Call for an intensive care review particularly if the patient has received three litres of fluid or signs of organ failure persist.
 - Continue to perform observations to ensure that the trend of blood pressure, pulse and level of consciousness are being monitored.

B. Fall in Cardiac Output

There are two predominant causes of fall in cardiac output:

1. Fall in Pre-Load

- Common causes include bleeding, loss of fluids and electrolytes
- History
 - Will describe a history 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 (base deficit, 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 500-1000mls 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 return 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 monitor observations (as per the escalation protocol).

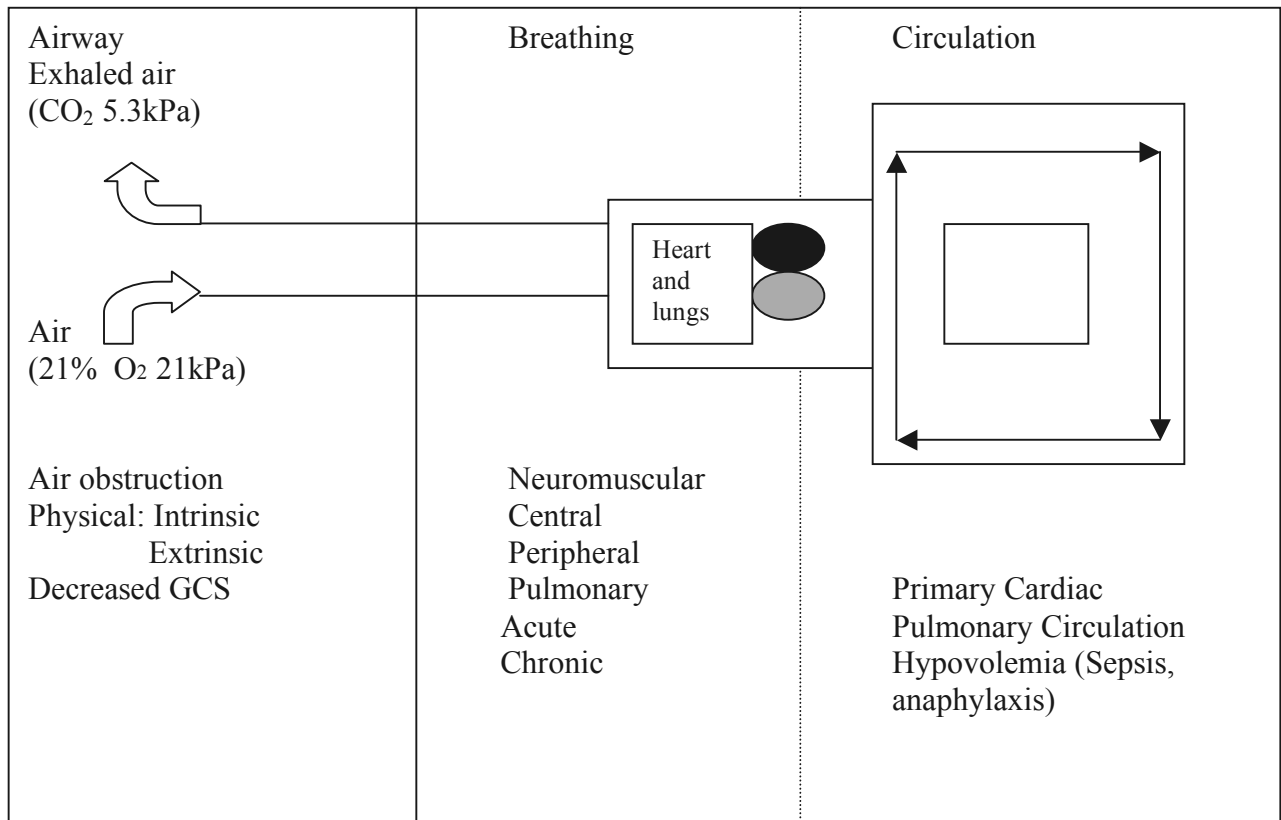
2. Fall in contractility

- Common causes include myocardial ischaemia or infarction.
- Patient 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 cardiomyopathy
- Clinical 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).
- Clinical Investigations
 - Evidence of renal dysfunction (rising creatinine)
 - Evidence of lactate formation (metabolic acidosis on arterial blood gas sampling (base deficit, rising lactate).
 - ECG – signs of ischaemia, infarction, dysrhythmia
- Clinical 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
 - Continue strict monitoring of fluid balance
 - Myocardial infarction protocol if MI confirmed

When assessing a patient remember to incorporate all the vital signs not just look at an individual reading. Remember to think about where they sit in the Oxygen Delivery Chain (see Figure13).

Text Box 30: Oxygen Delivery

Figure 13: Oxygen Delivery Chain



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 a patient 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
 - Increase 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.

- General Management Guidelines for hypotension in adults:
 - Hypotension and warm hands:
Administer fluids
 - Hypotension, cool hands, no signs of heart failure:
Administer fluids
 - Hypotension, cool hands, signs of heart failure:
Cease fluids. Refer to CCU/ICU for inotropes.
- Remember to incorporate all the vital signs in your assessment!
- **A SYSTOLIC BLOOD PRESSURE $\leq 90\text{mmHg}$ in adults requires immediate review by a doctor, consider activation of the ERS.**

Central Nervous System and Urine Output

Central Nervous System (CNS)

Learning Objectives

When you have successfully completed this section, you should be able to:

- 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 7).

Table 7: Common Causes of Decreased Level of Consciousness

Intracranial disease	<ul style="list-style-type: none">• Meningitis, encephalitis• Epilepsy• Cerebrovascular disease, sub-arachnoid haemorrhage• Head injury• CNS infection
Systemic conditions	<ul style="list-style-type: none">• Hypoxia, hypercapnia• Hypotension, hypo/hyperosmolar• Hypoglycaemia, hyponatraemia• Hypo/hyperthermia• Hypothyroidism, hypopituitarism, Addison's disease• Sedative drugs• Hepatic encephalopathy, uraemic encephalopathy

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

Causes of a Depressed Level of Consciousness

1. Inadequate Oxygen Delivery

Neurons 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 2).

When oxygen supply is inadequate, insufficient ATP is produced (Figure 3), which leads to failure of some cellular functions, especially in the brain and CNS. This results in the patient developing symptoms of confusion or a depressed level of consciousness.

Oxygen supply to the cells in the brain depends on the same factors as oxygen supply to all other tissues in the body.

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

- a) Decreased cardiac output
 - Decreased stroke volume
 - Decreased 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 resistance.

2. Inadequate Substrate Delivery for Metabolism

Cells require a substrate in order to form pyruvate, which enters the Krebs's in the mitochondria to produce ATP. Many cells in the body can use glucose, fats or proteins as substrates for energy production. However neurons can only use glucose as their substrate for energy production. Therefore if serum glucose levels fall too low, neurons 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 is one of the first things, which should be checked on an unconscious, or fitting patient, whether they are diabetic or not. The blood glucose should be >3.0mmol/l

Text Box 31: BGL and Decreased Level of Consciousness

A. Level of Consciousness

The level of consciousness score used in the EWS is the AVPU score (Text Box 32). This grades the LOC according to the criteria in Table 8, and should be carried out on every patient. This is added to the EWS for the other vital signs, and the total is calculated to give an overall EWS result for the patient.

Table 8: Adult AVPU EWS

SCORE	3	2	1	0	1	2	3
AVPU/CNS Response				Alert (A)			Voice (V), Pain (P), Unresponsive (U)

AVPU Score (Level of consciousness score)**0= Alert****3= Responds to Voice, to Pain or Unresponsive**

Text Box 32: Criteria used for the AVPU EWS

Another common method of measuring CNS function is the “Glasgow Coma Scale” (GCS). The GCS is not included in the EWS 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 9).

Table 9: Glasgow Coma Scale

• 1 • 2 • 3 • 4 • 5 • 6 • 7 • 8 Pupil scale (m.m.)			Date:	G														
			Time:	C														
				S														
	C O M A S C A L E	Eyes open	Spontaneously	4														
			To Speech	3														
			To Pain	2														
		Best verbal response	None	1														
			Orientated	5														
			Confused	4														
			Inappropriate words	3														
			Incomprehensible sounds	2														
		Best motor response	None	1														
			Obey commands	6														
	Localize Pain		5															
	Flexion to Pain		4															
Abnormal Flexion	3																	
	Extension to Pain	2																
	No Response	1																
	Total G.C.S.																	
PUPILS	right	Size																
	Reaction																	
	left	Size																
	Reaction																	
L I M B M O V E M E N T	A R M S	Normal power																
		Mild weakness																
		Severe weakness																
		Spastic flexion																
		Extension																
	L E G S	No response																
		Normal power																
		Mild weakness																
		Severe weakness																
		Extension																
No response																		
Record right (R) and left (L) separately if there is a difference between the two sides																		
Initials																		

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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.

Where there is a sudden fall in level of consciousness or a fall in GCS >2 (where this score is in use) an immediate medical review or activation of ERS is required

Text Box 33: EWS Criteria for CNS

B. Pupillary Size

Pupils should be checked as part of the neurological observations, and when there is any reduction in the patient's level of consciousness. Any change in the size, equality or reactivity of the patient's pupil is an important clinical sign of a change in the patient's neurological condition. This can provide important diagnostic clues (Text Box 34).

Bilateral pupillary dilatation

Causes:

- Sympathetic over-activity e.g. fear, stress, anxiety, hypoglycaemia
- Sympathomimetic medication administration e.g. administration of adrenaline in an arrest situation
- Anticholinergic medication activity e.g. atropine, tricyclic antidepressants, ipratropium nebulizer

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 for cataracts or prosthetic eyes can affect pupil size and reaction

Text Box 34: Pupil Size

Management of Decreased Level of Consciousness

1. Check airway and breathing: ensure airway is patent
 - Head tilt, chin lift, jaw thrust
 - Insert oropharyngeal or nasopharyngeal airway if required
2. Apply high-flow oxygen
3. Measure blood glucose and correct if $\leq 3\text{mmol/l}$ (administer 50 mls of 50% glucose intravenously)
4. Immediate medical review if GCS falls > 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 the patient supine in lateral recovery position

Reminder: Remember to always incorporate all the vital signs in your assessment.

Urine Output

Learning Objectives

When you have successfully completed this section, you should be able to:

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

Introduction

A reduced urine output (oliguria) is one of the most common triggers for a patient review. The kidney is an “end-organ”; thus poor urine output can be an indicator of patient deterioration due to many different causes, and is often one of the earliest signs of overall decline. It is important that the cause of a reduced urine output is correctly diagnosed.

Pathophysiology

Normal urine flow requires:

- Adequate oxygenation of the kidneys
- Adequate kidney perfusion pressure
- Normal function of the kidneys
- 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 do. Oxygen delivery depends on cardiac output and arterial oxygen content (Figure 12).

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.

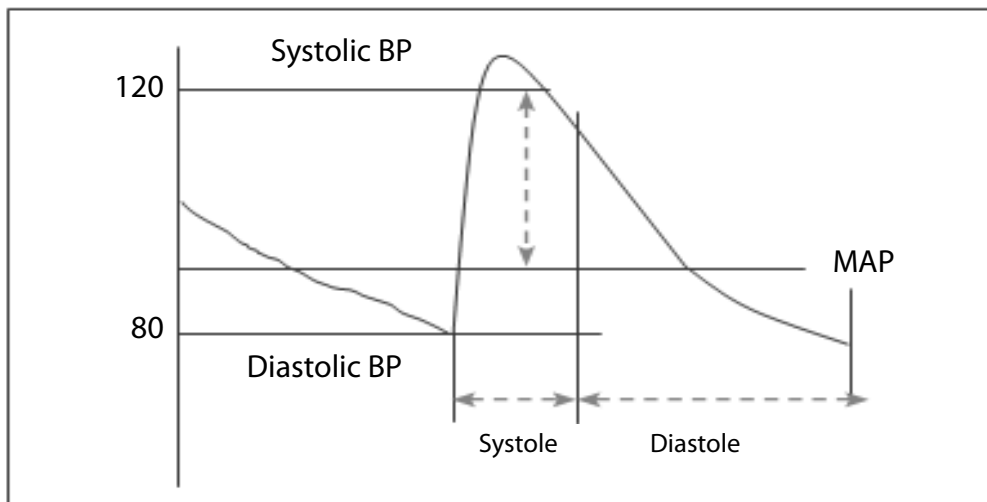
B. Perfusion Pressure

Renal blood flow is auto-regulated (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 14 &15). This range is increased in chronically hypertensive patients, who then require a higher blood pressure to maintain normal kidney function.

Figure 14: Perfusion Pressure Experienced by Organs

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

Figure 15: Mean Arterial Pressure Diagram



If the mean arterial blood pressure falls below the lower limit for auto-regulation, then the 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 – important to diagnose early as it requires urgent correction.

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

Text Box 35: 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 to prevent acute renal failure

Text Box 36: 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 500mls 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 pre-load, 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 37: Frusemide

SUMMARY

- One of the first checks that should be performed on an unconscious or fitting patient is a blood glucose level, whether they are a known diabetic or not.
- Where there is a sudden fall in level of consciousness, or a fall in GCS >2 contact the doctor for immediate medical review or activate ERS if required.
- Adult urine output should be $>0.5\text{mls/kg/hr}$ i.e. 35mls/hr for a 70kg 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.

Sepsis

Sepsis

Learning objectives

When you have successfully completed this section, you should be able to:

- Define what sepsis is and carry out an initial assessment
- Describe the indicators relevant for a diagnosis of sepsis
- Initiate and provide supportive symptom management until a definitive diagnosis has been made using the SEPSIS SIX Regimen

Introduction

Sepsis can occur in any clinical situation as a result of a primary infection or as a consequence of clinical interventions. Sepsis usually originates from a localised infection which progresses into an uncontrolled systemic response.

Sepsis is a hyper-reactive inflammatory response, caused by bacteria, fungi or viruses. Toxic substances released by pathogens trigger the inflammatory mediator's response. This process can lead to multiple organ failure and death. Improved survival can be achieved by early identification of sepsis and appropriate timely intervention. See SEPSIS SIX Regimen (Figure 16).

Sepsis is a medical emergency

'Sepsis Six' is an early goal directed therapy for sepsis, severe sepsis and septic shock.

Morbidity / Mortality

- Up to 135,000 European deaths from sepsis each year
- Severe sepsis is the leading cause of death in the non-coronary ITU

Classifications of Sepsis

- If the systemic inflammatory response is not caused by an infection (i.e pancreatitis, ischaemia or trauma), it is referred to as Systemic Inflammatory Response Syndrome (SIRS)
- Sepsis is a systemic inflammatory response to infection
- Severe Sepsis: If a patient with sepsis has accompanying organ dysfunction, hypoperfusion or hypotension he/she is in severe sepsis
- Septic shock: If the hypotension in a patient with severe sepsis does not respond to adequate fluid resuscitation, the patient is in septic shock

SIRS – Systemic Inflammatory Response Syndrome

2 or more features present:

- Temperature $> 38^{\circ}\text{C}$ or $< 36^{\circ}\text{C}$
- Respiratory rate > 20 breaths per minute or $\text{PaCO}_2 < 4.3\text{kPa}$
- Heart rate > 90 beats per minute
- WCC > 12 or < 4

** American College of Chest Physicians and the Society of Critical care Medicine (ACCP/SCCM) 1990 Consensus Conference*

Sepsis – SIRS and evidence of confirmed infection. Consider the following investigations:

- Bloods: FBC, U&E, Glucose, Clotting screen ABG, C-Reactive Protein
- Chest X-Ray / Urinalysis
- Cultures – Blood / CSF, Urine, Sputum, Wounds
- 12 lead ECG
- Cardiac Echocardiogram
- CT SCAN

Classification of Severe Sepsis

- Sepsis with organ dysfunction
- Hypoperfusion
- Hypotension

Classification of Septic Shock

- Sepsis with refractory hypotension and hypoperfusion in spite of fluid resuscitation
- End Organ Dysfunction: Heart/ Lungs, Kidneys/Liver, Brain

The following contains extracts from the SICS (2006).

Sepsis usually originates from a localised infection which progresses into an uncontrolled systemic response. It can rapidly lead to acute physiological deterioration with the risk of multiple organ failure and death. Early identification of sepsis with appropriate intervention i.e. oxygen, antimicrobials, and more advanced resuscitation, where indicated, has been shown to improve survival.

What is happening around the body?

Sepsis can cause major systemic effects; septic shock is the worst of these. The products of the infecting organism e.g. endotoxin or exotoxin, cause the release and activation of inflammatory mediators such as histamine and kinins. Simultaneously there is an induction of cytokines i.e. interleukins and tumour necrosis factor (TNF).

The net result of these changes is to cause a combination of:

- Hypoxaemia
- Hypovolaemia
- Vasodilation and capillary leak
- Impaired tissue oxygen utilisation

Respiratory Changes

The earliest clinical sign of sepsis is often a rapid respiratory rate.

This may be driven by pyrexia, lactic acidosis, local lung pathology, pulmonary oedema, cytokine-mediated effects on the respiratory control centre or a combination of several of these factors. Hypoxaemia occurs as a result of pulmonary pathology, shunting of deoxygenated blood through the lungs (cytokine mediated) or pulmonary oedema secondary to capillary leak.

Circulatory Changes

The release of bradykinin and production of cytokines cause normally 'tight' endothelial junctions to become loose resulting in increased vascular permeability with accompanying plasma leak i.e. capillary leak. This leads to hypovolaemia and reduced preload. Bradykinin and some cytokines also cause peripheral vasomotor failure: peripheral blood vessels vasodilate and diastolic blood pressure falls as a result.

**The combination of reduced intra-vascular volume
and vasodilatation often produces hypotension.**

The body attempts to compensate by increasing the heart rate and mobilising fluid from the interstitial space or blood from the splanchnic circulation, but this is inefficient due to cytokine mediated effects.

Impaired tissue oxygen utilisation

Although the cardiac output may rise in sepsis there is a mismatch of tissue oxygen delivery and requirements. There may be shunting of blood in the micro-circulation, by-passing cells which become hypoxic. There is also cytokine mediated disturbance of mitochondrial oxygen handling: this blocks the progress of oxygen down the normal cascade. These both lead to lactic acidosis, organ dysfunction and ultimately multiple organ failure.

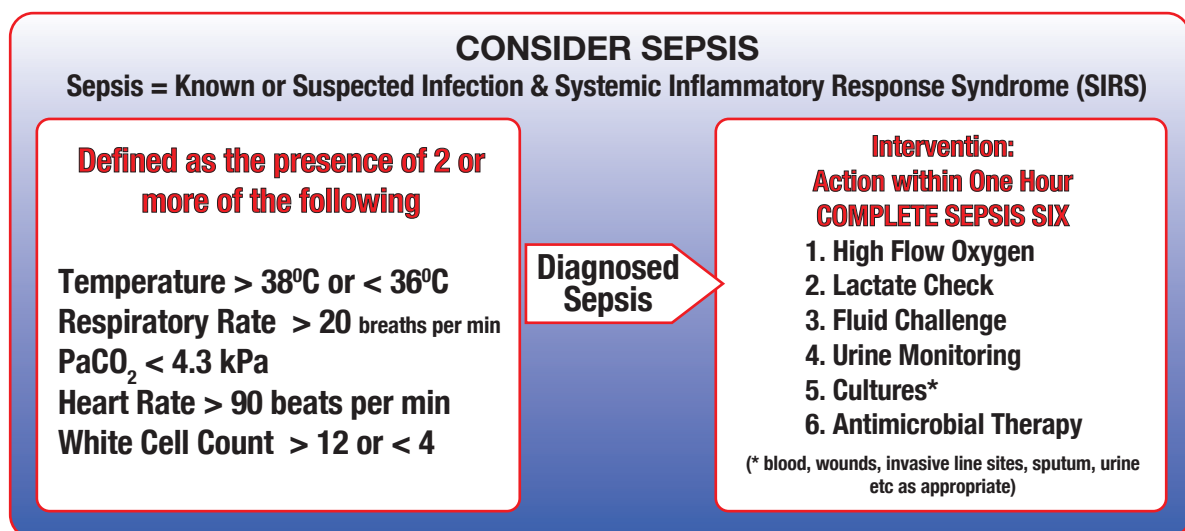
Patient Assessment

- Look.....Listen.....Feel.....ABCDE...
- Altered Mental Status / especially new confusion or agitation / GCS<15
- If the patient's history is suggestive of a new infection, record vital signs, early warning score and bedside capillary blood glucose
- Is there pallor / flushing / cyanosis / rashes / wound / posture
- Can you hear crackles (on chest examination)
- Any complaints of pain / abnormal posture
- Peripheries.....are they warm/cold to touch
- Feel a pulse for rate / quality
- If concerned about the patient / Patient looks unwell

Consider sepsis and contact doctor to review.

Once sepsis/SIRS is diagnosed complete the initial interventions in SEPSIS SIX regimen within one hour.

Figure 16: SEPSIS SIX Regimen (summary)



Following initial diagnosis and intervention within 1 hour, institute organisation's guidelines/protocols/policies for the management of sepsis, severe sepsis and septic shock.

SEPSIS SIX Regimen

Goals to be completed prior to or within 1 hour of Recognition of Sepsis, Severe Sepsis or Septic Shock

Table 10: Sepsis Six Regimen

SEPSIS SIX	Address simultaneously ; Target time 1 hour from recognition
100% Oxygen	<ul style="list-style-type: none"> Give 15L/min via Non Re-breathing Mask unless oxygen restriction necessary (e.g. chronic CO₂ retention: Aim for SaO₂ of 90-92%).
IV FLUIDS	<ul style="list-style-type: none"> Give a 500ml - 1000ml bolus of crystalloid (0.9% Saline or Hartmann's Solution) over 30 minutes. (In a patient with an initial systolic BP < 90 or lactate > 4, give a larger bolus 20-30ml/kg over 30 minutes). If patient improves and stabilises patient is not severely septic – reassess situation. If patient does not stabilise, continue resuscitation and involve a senior doctor (registrar) and above (if not already done). Give additional boluses of 250-500mls of crystalloids and colloids (Voluven or Gelofusin) at a ratio of 3:1, if systolic BP falls to < 90 again.
BLOOD CULTURES	<ul style="list-style-type: none"> Obtain blood cultures before commencing antimicrobials. Do not significantly delay antimicrobial administration. Also send sputum, urine culture / wound swabs etc as appropriate (if not already done).
IV ANTIMICROBIALS	<ul style="list-style-type: none"> Begin IV antimicrobials as early as possible and always within the <u>first</u> hour of recognising sepsis and severe sepsis. Choose antimicrobials according to organisation's antimicrobial guidelines. Contact on-call Microbiologist, if in doubt.
CATHETER	<ul style="list-style-type: none"> Insert a Urinary Catheter. Send urine for C&S if not already done. Monitor urine output hourly. Start fluid balance chart.
LACTATE, Hb, OTHER TESTS & ACTIONS	<ul style="list-style-type: none"> If not already done, request bloods for FBC, U&E, LFTs, blood sugar, coagulation screen, amylase, C-Reactive Protein, ABGs, & lactate levels (Lactate > 4mmol/L indicates severity) Repeat lactate after SEPSIS SIX goals completed. Arrange blood transfusion if Hb 7.0 g/dl. CT scan if indicated Formally evaluate patient for focus of infection. Consider treatment (e.g. abscess drainage, etc). Order appropriate radiological tests.

Summary

- Sepsis is a medical emergency
- Sepsis can occur in any clinical situation as a result of a primary infection or as a consequence of clinical interventions
- Sepsis usually originates from a localised infection which progresses into an uncontrolled systemic response
- SEPSIS SIX is an early goal directed therapy for sepsis, severe sepsis and septic shock

Communication, Teamwork and Management Plans

Communication, Teamwork and Management Plans

Learning Objectives

When you have successfully completed this section, you should be able to:

- Communicate clearly and concisely
- Use ISBAR correctly
- State the importance of teamwork and fully commit to team participation to the best of your ability
- Participate in the development of management plans

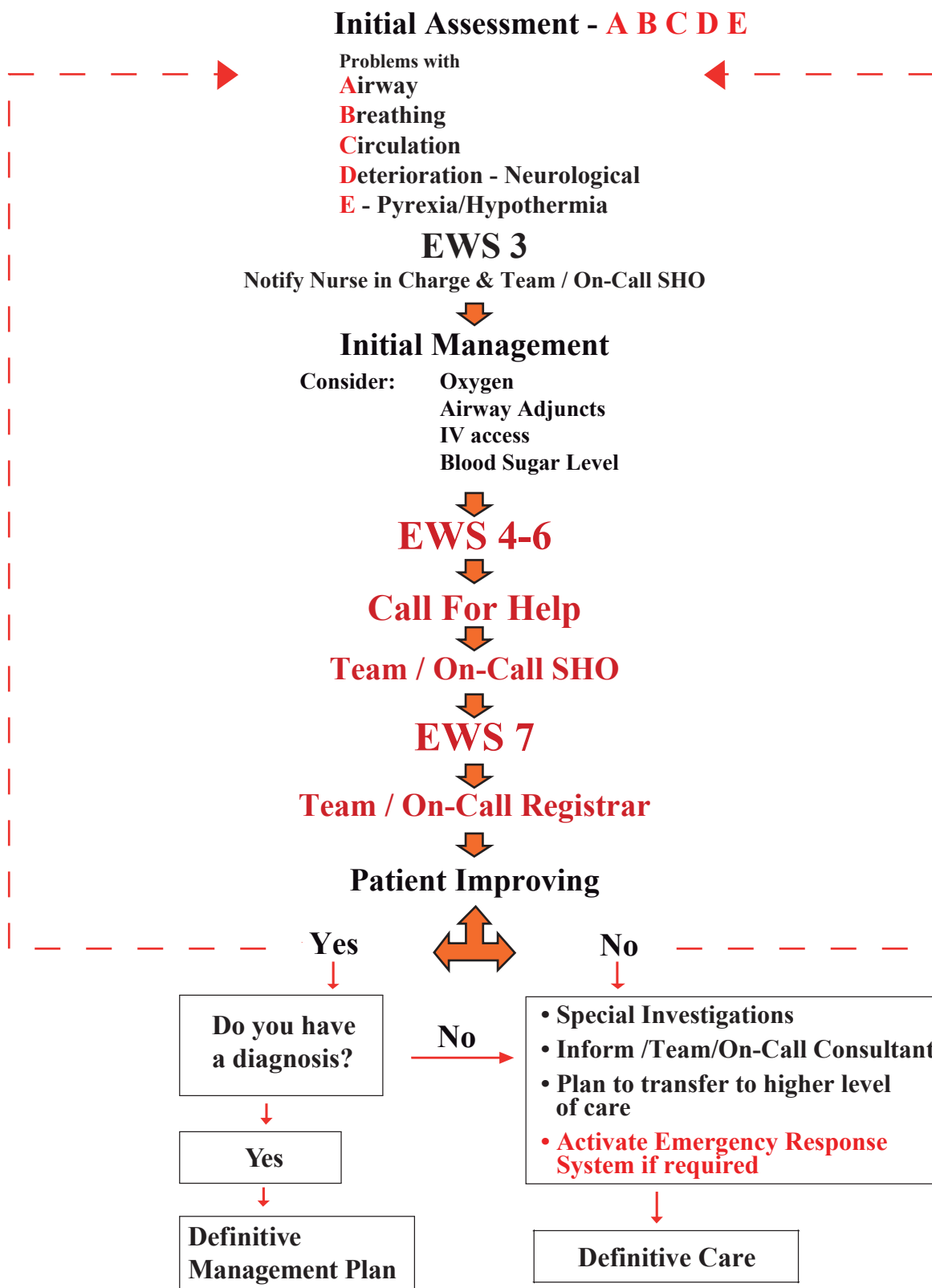
One of the most important factors in determining an acutely ill patient's outcome is the quality of the communication among the clinicians involved. In teams, each member has strengths and weaknesses, varying competencies, skills and different levels of knowledge. The aim in managing the deteriorating patient is to determine the role of each member of the team, identify their comfort zones and work together to achieve the best outcome for the patient. The flowchart in Figure 17 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 and responding adequately**
- 5. Formulating, documenting and communicating a patient management plan with a provisional diagnosis**
- 6. Actioning the management plan**
- 7. Reassessing for possible re-review and escalation of the management plan**

Text Box 38: Optimising Management

Deteriorating Patient Management Flow Chart



Important

1. If response is not carried out as above, CNM/Nurse in charge must contact the Registrar or Consultant.
2. If you are concerned about a patient, escalate care regardless of score.

Figure 17: Flow Chart of Steps in Managing a Deteriorating Patient

1. Gathering Information

Each member of the team provides vital information about the patient's care in hospital and all of this information must be integrated to inform assessment, decision making 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 doctor 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 mobilizing 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/updated notes from each different team member**
- **Reviewing observations, fluid and medication charts**
- **Comparing current presentation with previous presentations**
- **Family, friends or the patient themselves**

Text Box 39: 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

When 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 a student nurse finds an abnormal arterial oxygen saturation, they must refer this information to the registered nurse who is working with them for more guidance on how to act on this. When a junior doctor is concerned about a deteriorating patient, they need to discuss the findings with their registrar and possibly their consultant. The patient must be attended to appropriately and promptly.

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 and promptly!

Text Box 40: Abnormalities and Appropriate Care

When a EWS triggers a communication, describe the observations that have triggered the EWS (e.g. total EWS 5 due to pulse 102, RR26, Temp 38.7). For a doctor to be able to appropriately triage and advise on a particular patient, they need to 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 that problem in the context of the patient.

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.

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

SITUATION: What is the current situation, concerns, observation, EWS, 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 41: ISBAR Communication

For example:

A 75 year old lady with a history of Ischaemic Heart Disease is admitted to hospital 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 oxygen via nasal prongs and has a EWS of 6. As the nurse caring for this patient you are concerned that she is acutely unwell and needs urgent attention (call SHO as per escalation protocol). 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 has a EWS of 6 due to her dropping her arterial oxygen saturation to 88% on 2 L/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, pneumonia or a fat embolus. If you are not sure what the cause of the problem is you can say that you think the patient is unwell and that you are concerned about them.

RECOMMENDATION:

“This patient requires an immediate medical review. I have increased her inspired oxygen in the meantime to 15 L/min on a non-re-breather mask.”

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

Documentation

Once you have actioned a particular problem, you must always document this. This may involve documenting low arterial oxygen saturation, that you have contacted a doctor, giving a time, or if you are a 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.
- Is also a medico-legal requirement.

You must always identify who needs to be informed about a deteriorating patient, communicate as much as possible and document appropriately (Text Box 42).

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 ISBAR.**
- 3. Document the steps you have taken to remedy the situation and actions taken.**

Text Box 42: Communicating

4. Adequate Response to Information/Concerns

After being involved in the management of a deteriorating patient, many people may feel that things could have been done differently or better. They may feel that the root of the problem was not addressed and something else was happening with the patient, or that their particular views were not taken into consideration. Each member of the team has different priorities with respect to patient management and these need to be considered and incorporated into the management plan.

After communicating with more senior colleagues, an individual may feel that they were not taken seriously or their particular concern about a situation was not addressed. This can be remedied by specifically asking each member of the team what concerns they have, how they think they can be addressed and incorporating these concerns into their management plan. Sometimes staff caring for patients may not be sure what an abnormal test result means. They may feel concerned about phoning someone as they are afraid they might seem stupid or even get scolded for not knowing. This behaviour does not help anyone, there are various communication tools that can be used to overcome this.

For example:

A nurse is required 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 returns to review all the collected information and review the management plan only to find that his orders were not followed. If the doctor discussed the plan with the nurse to see if there were any issues with the plan, the issue would have been identified and resolved much earlier, saving 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 requirements 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 the level of nursing care required, but the doctor feels that there is no medical reason that they need to escalate their care. This requires discussion of the plan so that it can be agreed by all members of the team.

Theoretically, in the event of a deteriorating patient, all personnel involved in the care of the patient should be present. ISBAR should be used for all communication.

It is part of the role of the team leader to voice concerns, pre-empt other people's concerns and integrate them into the management plan. By simply asking what each individual's main concerns are, the team may save time. Often issues are raised that had not been previously considered. If all team members feel that their concerns are taken into consideration, ultimately 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, both legible and logical.

Optimal management plans include action plans for all members of the team and time-frames in which actions must be performed. Medical staff must always document their impression, which is the provisional diagnosis (Text Box 38). When this is completed, each member has a clear idea of their roles and responsibilities with 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 the frequency of observation changed so that vital signs are performed every half-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. Physiotherapy Orders

Where the patient is diagnosed with a hospital-acquired pneumonia, the physiotherapist knows that chest physiotherapy as an intervention is required for that patient.

4. Change in Patient Therapy Orders

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

5. Investigation/ Intervention Orders

Where it has been decided that the patient needs their electrolytes checked, this must be documented, as well as identifying who has the responsibility for checking the results. It is often useful to write what is expected and what action to take about abnormal results. It may become evident that the patient requires IV access for antibiotics that have been ordered.

6. Notification Orders

Guidance from the team as to when to be concerned, or not to be concerned in relation to the management of a deteriorating patient is very useful.

Notification orders include notifying the doctor when the urine output is less than 0.5mls/kg/hr, or systolic blood pressure less than 100mmHg. This can reduce phone calls from nurse to doctor and also give reassurance to nursing staff about when they need to be concerned about a particular patient.

6. Actioning the Management Plan

Everyone must be very clear about his or her role and responsibilities in the management plan of the patient. In particular what actions are required, and then ensuring that these are carried out!

Health care staff must know what action to take, must be competent and skilled to carry out that action, must perform the action and then follow-up on the result of the action.

7. Reassess

When caring for a deteriorating patient, health care staff must always review the patient to ensure that the plans 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 is the responsibility of the person you informed to come and attend to the patient.

For a change of shift, all concerns and outstanding issues in relation to patients must be documented and verbally conveyed to the health care professional taking over the care of the patient to ensure continuity of care and follow-up. Where the patient is not improving then they need to be reassessed – start again at the beginning. Gather the information, identify 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 43: Documentation

Management plans should include:

- a. Observation orders
- b. Nursing orders
- c. Physiotherapy orders
- d. Change in therapy orders
- e. Investigations/interventions orders
- f. Notification orders

Text Box 44: Management Plans

SUMMARY:

- It is important 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
 - Physiotherapy orders
 - Change in therapy orders
 - Investigations/intervention orders
 - Notification orders

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Resources required to deliver the National Early Warning Score and associated Education Programme are available on the following webpage: www.hse.ie/go/nationalearlywarningscore/

Please note: this webpage is updated periodically.

To allow for feedback on the programme an issues log has been set up at the following email address: maryn.redmond@hse.ie.

The Quiz Questions may be removed or photocopied from the manual and returned to the course co-ordinator, with answers marked, in advance of the face to face education session.

Date:..... Name:

Organisation: Location:

Quiz Questions

1. An increased heart rate may reflect:
 - a. Failing oxygen delivery
 - b. Decreased level of consciousness
 - c. Decreased urine output
 - d. Rise in stroke volume
2. 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
3. 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
4. Aerobic metabolism:
 - a. Generates 2ATP
 - b. Produces lactate
 - c. Requires oxygen
 - d. Causes tachypnoea
5. Tachypnoea:
 - a. Is caused by Morphine
 - b. Is caused by metabolic alkalosis
 - c. Reflects a deteriorating patient
 - d. Is caused by respiratory alkalosis

6. 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
7. 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
8. Urinary 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
9. 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
10. 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

Appendix 1

National Early Warning Score Project and associated Education Programme Governance Group membership

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Professor Garry Courtney
Professor Shane O'Neill
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Dr. David Vaughan
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Appendix 2

National Early Warning Score and associated Education Programme Advisory Group membership

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Dolores Ryan	Connolly Hospital, Blanchardstown
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Dr. David Vaughan	Dr. Steeven's Hosptial Dublin
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Noreen Curtin	AMNCH, Dublin
Aine Lynch	AMNCH, Dublin
Marina O'Connor	Our Lady of Lourdes Hospital, Drogheda
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