Training Manual for
The COMPASS© Education Programme incorporating the National Early Warning Score (for non-pregnant adult patients)

The COMPASS© Education Programme incorporating the National Early Warning Score is a workstream of the National Acute Medicine Programme, in association with other National Clinical Programmes, Quality & Patient Safety Division, the Patient Representative Groups, Office of the Nursing and Midwifery Services Director, Clinical Indemnity Scheme, Acute Hospital Services The Irish Association of Directors of Nursing and Midwifery (IADNAM) and Health and Social Care Professionals.

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ADULT PROGRAMME
(for non-pregnant patients)

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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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The COMPASS® Education Programme and National Early Warning Score (NEWS) for non-pregnant adults are recommended by the National Clinical Guideline for use in the HSE, Voluntary and Private acute hospital services in the first instance. The programme should be delivered in full (Recommendation 44, National Clinical Guideline) and completed by relevant personnel every two years. This Programme is not relevant for Paediatric or Obstetric patients. The National Early Warning Score, National Clinical Guideline No. 1 is available at the following link: [http://health.gov.ie/patient-safety/hcec/national-clinical-guidelines/](http://health.gov.ie/patient-safety/hcec/national-clinical-guidelines/)

The HSE’s expectation is that healthcare staff will use clinical judgement, medical, nursing and midwifery knowledge in applying the general principles and recommendations contained in the COMPASS® Education Programme and National Early Warning Score, National Clinical Guideline No. 1. Recommendations may not be appropriate in all circumstances and the decision to adopt specific recommendations should be made by the practitioner taking into account the individual circumstances presented by each patient/resident and available resources. The contents of the COMPASS® Education Programme and National Early Warning Score, National Clinical Guideline No. 1 do not replace or remove clinical judgement or the professional care and duty necessary for each specific patient case. Therapeutic options should be discussed with senior clinical staff on a case-by-case basis as necessary.

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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 (NEWS) 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);
• 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 NEWS score = 3 the patient was not reviewed. Over the following 24 hours, the patient fulfilled NEWS 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 NEWS 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.

AIM
The aim of the COMPASS© programme is to facilitate participants to recognise and manage the clinically deteriorating patient, and enable him/her to utilise the skills and competencies required to provide supportive symptom management until a definitive diagnosis has been made and treatment initiated.

LEARNING OUTCOMES
On completion of this programme, participants should be able to:

1. Prioritise Care, using:
   - Clinical judgement to apply prior and acquired knowledge to identify deterioration or improvement in a patient’s condition;
   - National and local Guidelines and protocols to complete the National Patient Observation Chart fully, accurately & consistently over time, including calculating a National Early Warning Score each time the patient vital signs are recorded;
   - Decision making skills to initiate appropriate early interventions for patients who are deteriorating;
   - An appropriate and timely response - Escalate care appropriately using agreed escalation protocol.

2. Use clinical reasoning to:
   - 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. Refer patients appropriately:
   - Assess severity of illness and respond with life sustaining measures in the event of severe or rapid deterioration pending the arrival of emergency assistance;
   - Recognise the need for specialist assistance.
   - Manage patient deterioration in conjunction with the multi-disciplinary team;
   - Identify the most appropriate environment for the patient.
4. Improve communication and team working:
   - Promote the use of more focussed communication between healthcare professionals using the ISBAR communication tool to communicate information in a structured and effective way to the appropriate healthcare professional, patient and family;
   - 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 education programme

You can complete this education programme as follows:
1. Self directed E-learning – available on HSELaND, or,
2. Attend a face to face programme – available in your clinical area.

Before you complete the eLearning programme OR the face to face programme, you must download and read the Training Manual for the COMPASS© Education Programme incorporating the National Early Warning Score (for non-pregnant adult patients).

The face to face programme has three phases that you must complete in the following order:
1. Work through a CD and the Training Manual for the COMPASS© Education Programme incorporating the National Early Warning Score (for non-pregnant adult patients), independently;
2. Complete the paper quiz;
3. Attend the mandatory face-to-face session.

These resources are available at the following link: http://www.hse.ie/eng/about/Who/clinical/natclinprog/acutemedicineprogramme/earlywarningscore/education.html

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.
The National Early Warning Score
Learning outcomes

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

• Calculate a total 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

The National Early Warning Score (NEWS) 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 National 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 ≤40 (Bradycardia) is a trigger point for action

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

Text Box 1: Trigger Score

The NEWS 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 NEWS score;
• Direction for nurses on the frequency of vital sign observation measurement once a trigger score is reached.
NEWS 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. **The NEWS does NOT replace calling the cardiac arrest team in the event of collapsed adult with suspected no pulse and/or no breathing.**
2. **The NEWS does not replace calling the ERS.**
3. **The NEWS 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: NEWS

**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](http://www.hse.ie).

**Adult NEWS Calculation**

To obtain the total NEWS:
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>
<thead>
<tr>
<th>Score</th>
<th>3</th>
<th>2</th>
<th>1</th>
<th>0</th>
<th>1</th>
<th>2</th>
<th>3</th>
</tr>
</thead>
<tbody>
<tr>
<td>Respiratory Rate (bpm)</td>
<td>≤ 8</td>
<td>9 - 11</td>
<td>12 - 20</td>
<td>21 – 24</td>
<td>≥ 25</td>
<td></td>
<td></td>
</tr>
<tr>
<td>$S_2O_2$ (%)</td>
<td>≤ 91</td>
<td>92-93</td>
<td>94-95</td>
<td>≥ 96</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Inspired $O_2$ (FiO$_2$)</td>
<td>Air</td>
<td>Any $O_2$</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Systolic BP (mmHg)</td>
<td>≤ 90</td>
<td>91-100</td>
<td>101-110</td>
<td>111-249</td>
<td>≥ 250</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Heart Rate (BPM)</td>
<td>≤ 40</td>
<td>41-50</td>
<td>51-90</td>
<td>91-110</td>
<td>111-130</td>
<td>≥ 131</td>
<td></td>
</tr>
<tr>
<td>AVPU/CNS Response</td>
<td>Voice (V), Pain (P), Unresponsive (U)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Temp (°C)</td>
<td>≤ 35.0</td>
<td>35.1 – 36.0</td>
<td>36.1 – 38.0</td>
<td>38.1 – 39.0</td>
<td>≥ 39.1</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

**Note:** Where Systolic blood pressure is ≥ 200mmHg, request Doctor to review
Track and Trigger procedures
If any single parameter scores 3 or the total NEWS reaches a trigger score of 3, the activation protocol must be initiated (Text Box 3).

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

<table>
<thead>
<tr>
<th>Total NEWS Trigger Point - Score of 3:</th>
</tr>
</thead>
<tbody>
<tr>
<td>Notify Nurse in charge and Team/On-Call SHO, 4 hourly observations, SHO to review patient within 1 hour.</td>
</tr>
</tbody>
</table>

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 NEWS 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 the NEWS 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: NEWS Escalation Protocol

<table>
<thead>
<tr>
<th>Total Score</th>
<th>Minimum Observation Frequency</th>
<th>ALERT</th>
<th>RESPONSE</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>12 Hourly</td>
<td>Nurse in charge</td>
<td>Nurse in charge to review if new score1</td>
</tr>
<tr>
<td>2</td>
<td>6 Hourly</td>
<td>Nurse in charge</td>
<td>Nurse in charge to review</td>
</tr>
<tr>
<td>3</td>
<td>4 Hourly</td>
<td>Nurse in charge &amp; Team/On-call SHO</td>
<td>1. SHO to review within 1 hour</td>
</tr>
</tbody>
</table>
| 4-6         | 1 Hourly                      | Nurse in charge & Team/On-call SHO | 1. SHO to review within hour  
2. Screen for Sepsis  
3. If no response to treatment within 1 hour contact Registrar  
4. Consider continuous patient monitoring  
5. Consider transfer to higher level of care |
| 7           | 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)  
(as appropriate to hospital model) |

**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"

*In certain circumstances a score of 3 in a single parameter may not require ½ hourly observations i.e. some patients on O₂.

- 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.

**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.

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.
Practical guidance

The National Early Warning should be recorded using the National Patient Observation Chart for non-pregnant Adult Patients for:

- All admitted patients;
- All patients in the ED when a decision has been made to admit
- All patients on discharge from ICU, CCU and Operating Theatre -Recovery room.

Summary NEWS Adult (non-pregnant)

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 NEWS of 3 is the trigger point for action, with escalated notification required at NEWS of 4-6 and 7 or if the patient is not improving.

1. The NEWS does NOT replace calling the cardiac arrest team in the event of collapsed adult with suspected no pulse and/or no breathing.
2. The NEWS does not replace calling the ERS as appropriate to local hospital model.
3. The NEWS 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
Learning Outcomes

*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).

\[
\text{ATP} \quad \text{ADP} \times \text{Pi} + \text{Energy} \\
\text{(Adenosine +Pi+Pi+Pi)} \quad \text{Adenosine + Pi+Pi}
\]

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)

1. Glucose
2. Glucose-6-Phosphate
3. Pyruvate
4. Citrate, TCA cycle/Kreb’s cycle

CYTOPLASM

Pyruvate dehydrogenase

Mitochondrial membrane

Pyruvate

Acetyl CoA x 2ATP

NADH + H⁺ + O₂ → NAD⁺ + H₂O

MITOCHONDRIA

ADP + Pi → 3 6 ATP

Figure 3: ANAEROBIC METABOLISM (i.e. in the absence of oxygen)

1. Glucose
2. Glucose-6-Phosphate
3. Pyruvate
4. Citrate, TCA cycle/Kreb’s cycle

CYTOPLASM

Pyruvate dehydrogenase

Mitochondrial membrane

Pyruvate

Acetyl CoA + 2ATP

MITOCHONDRIA

Lactate

2ATP

ADP + Pi → 3 6 ATP
Oxygen supply to the cells can be described by the “oxygen delivery chain” (Figure 4)

**Oxygen Delivery = Cardiac Output x Arterial Oxygen content**

Thus oxygen delivery requires
A. Arterial oxygen content:
   - haemoglobin concentration (Hb)
   - haemoglobin oxygen saturation (SaO₂)
   - partial pressure of oxygen (PaO₂)
   
   SEE SECTION ON “AIRWAY AND BREATHING”

B. Cardiac output – SEE SECTION ON “CIRCULATION”

---

**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.
- Oxygen Delivery = Cardiac Output x Arterial Oxygen content.
AIRWAY
and
BREATHING
Learning Outcomes

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 a patient with impaired arterial oxygenation appropriately;
- 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.

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 his/her 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

Inadequate oxygen delivery at the tissue level

Anaerobic metabolism

Lactate production

Metabolic Acidosis

Stimulates respiratory drive

Increases the 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, SpO\textsubscript{2} NEWS**

The respiratory rate, arterial oxygen saturation and inspired O\textsubscript{2} (FiO\textsubscript{2}) score (Table 3) for the Adult NEWS are as follows:

<table>
<thead>
<tr>
<th>Table 2: Adult Respiratory NEWS</th>
</tr>
</thead>
<tbody>
<tr>
<td>Score</td>
</tr>
<tr>
<td>Respiratory Rate (bpm)</td>
</tr>
<tr>
<td>SpO\textsubscript{2} (%)</td>
</tr>
<tr>
<td>Inspired O\textsubscript{2} (FiO\textsubscript{2})</td>
</tr>
</tbody>
</table>

The NEWS is noted for each individual parameter and will make up part of the total NEWS. 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\textsubscript{2} of $\geq 96\%$ in those patients not at risk for hypercapnic respiratory failure and the PaO\textsubscript{2} as close to 13kPa as possible, but at least 8kPa (SaO\textsubscript{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 “CO\textsubscript{2} retainers” or their risk factors for hypercapnic 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\textsubscript{2} of 8kPa or saturation of greater than 90\% on pulse oximetry. Therefore in a patient with COPD who has a PCO\textsubscript{2} > 8kPa, but is also hypoxic, PO\textsubscript{2} < 8kPa, do not turn the inhaled O\textsubscript{2} down, however, do not leave the patient unattended. If their PO\textsubscript{2} is >8kPa then you can turn the inhaled O\textsubscript{2} down to maintain SaO\textsubscript{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 a too high inspired oxygen, a Venturi system is more accurate in delivering the oxygen concentration desired.
Table 3: 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-rebreather 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
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%)

![Image of Oxygen Mask](Image reproduced with permission of mayohealthcare.com.au)

D) Non-re-breather mask (Figure 10) – 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.

![Image of Non-re-breather Mask](Image reproduced with permission of mayohealthcare.com.au)

**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).

---

**Text Box 10: Oxygen Prescription**

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 CO₂ 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 PO₂ of 8 kPa.
Patients do not die from a raised CO\textsubscript{2} alone: they die from hypoxaemia (Text Box 11)

<table>
<thead>
<tr>
<th>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.</th>
</tr>
</thead>
</table>

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.

<table>
<thead>
<tr>
<th>SaO\textsubscript{2} refers to the directly measured in vitro measurement of arterial oxygenation usually via ABG. SpO\textsubscript{2} refers to the indirect assessment using the oximeter.</th>
</tr>
</thead>
</table>

Text Box 12: SaO\textsubscript{2} and SpO\textsubscript{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\textsubscript{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\textsubscript{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.

<table>
<thead>
<tr>
<th>If the pulse oximeter does not give a reading, do not assume that it is broken...the patient may have poor perfusion!</th>
</tr>
</thead>
</table>

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.

<table>
<thead>
<tr>
<th>Oxygen saturation may be “normal” but the PCO\textsubscript{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.</th>
</tr>
</thead>
</table>

Text Box 14: Normal SpO\textsubscript{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) and 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

<table>
<thead>
<tr>
<th>Airway</th>
<th>Breathing</th>
<th>Circulation</th>
</tr>
</thead>
<tbody>
<tr>
<td>Exhaled air (CO₂ 5.3kPa)</td>
<td>Heart and Lungs</td>
<td></td>
</tr>
<tr>
<td>Air (21% O₂ 21kPa)</td>
<td>Neuromuscular Central Peripheral Pulmonary Acute Chronic</td>
<td></td>
</tr>
</tbody>
</table>
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 PaO₂ may be lower than the normal range e.g. 8kPa (normal 11-15kpa). Delivery of high concentrations of oxygen to achieve a PaO₂ within the normal range may indeed suppress the respiratory drive in these patients causing the PCO₂ 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 PaO₂ 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 PCO₂. Administer enough oxygen as prescribed to achieve an SpO₂ >90% and a PaO₂ of 8kPa. If the PaO₂ rises above 8kPa, you may turn down the inhaled oxygen as prescribed to achieve an SpO₂ 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 outcomes

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 and describe the causes and consequences of, and compensation for, 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](image)

Oxygen delivery = Cardiac output x Arterial Oxygen Content

\[
\text{Stroke Volume} \times \text{Heart rate} \quad \text{Haemoglobin} \times \text{SaO}_2
\]

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.

![Text Box 17: Relevance of Pulse and Blood Pressure to Oxygen Delivery](image)

Calculation of Adult NEWS for heart rate:

Table 4: Adult Heart rate NEWS

<table>
<thead>
<tr>
<th>Score</th>
<th>3</th>
<th>2</th>
<th>1</th>
<th>0</th>
<th>1</th>
<th>2</th>
<th>3</th>
</tr>
</thead>
<tbody>
<tr>
<td>Heart Rate (BPM)</td>
<td>≤ 40</td>
<td>41-50</td>
<td>51-90</td>
<td>91-110</td>
<td>111-130</td>
<td>≥ 131</td>
<td></td>
</tr>
</tbody>
</table>

**Adult Heart Rate NEWS Triggers**

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

![Text Box 18: Low Heart Rate](image)

**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 NEWS for blood pressure:

Table 5: Adult Blood Pressure NEWS

<table>
<thead>
<tr>
<th>Score</th>
<th>3</th>
<th>2</th>
<th>1</th>
<th>0</th>
<th>1</th>
<th>2</th>
<th>3</th>
</tr>
</thead>
<tbody>
<tr>
<td>Systolic BP (mmHg)</td>
<td>≤ 90</td>
<td>91-100</td>
<td>101-110</td>
<td>111-249</td>
<td>≥ 250</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

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 harm. 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

**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 afterload – 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 over-stretches the heart muscle, the stroke volume can start to decrease.

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

a) Intravascular blood volume

Absolute:
A decrease in intravascular blood volume (bleeding, electrolyte, 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.

Increase in intrathoracic 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, sympathomemetics (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 (\(\mathcal{I}\)) 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 \(\mathcal{I}\) 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. \(\alpha\) 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=SVxHR)
   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): Hypo tension, 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 NEWS 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%NaCI) 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);
  - If the tachycardia is not resolved repeat the fluid challenge;
  - Consider Sepsis Six regimen;
  - 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 Figure 13)
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 ≤ 90mmHg in adults requires immediate review by a doctor, consider activation of the ERS.
Central Nervous System
and
Urine Output
Central Nervous System (CNS)

Learning Outcomes

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 6: Common Causes of Decreased Level of Consciousness

<table>
<thead>
<tr>
<th>Intracranial disease</th>
<th>Systemic conditions</th>
</tr>
</thead>
<tbody>
<tr>
<td>Meningitis, encephalitis</td>
<td>Hypoxia, hypercapnia</td>
</tr>
<tr>
<td>Epilepsy</td>
<td>Hypotension, hypo/hyperosmolar</td>
</tr>
<tr>
<td>Cerebrovascular disease, sub-arachnoid haemorrhage</td>
<td>Hypoglycaemia, hyponatraemia</td>
</tr>
<tr>
<td>Head injury</td>
<td>Hypo/hyperthermia</td>
</tr>
<tr>
<td>CNS infection</td>
<td>Hypothyroidism, hypopituitarism, Addison’s disease</td>
</tr>
<tr>
<td></td>
<td>Sedative drugs</td>
</tr>
<tr>
<td></td>
<td>Hepatic encephalopathy, uraemic encephalopathy</td>
</tr>
</tbody>
</table>

CNS function is an important indicator of adequacy of tissue oxygenation called “endorgan function”. Thus CNS assessment is included in the NEWS. 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 Kreb’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 NEWS 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 NEWS for the other vital signs, and the total is calculated to give an overall NEWS result for the patient.

Table 7: Adult AVPU NEWS

<table>
<thead>
<tr>
<th>Score</th>
<th>3</th>
<th>2</th>
<th>1</th>
<th>0</th>
<th>1</th>
<th>2</th>
<th>3</th>
</tr>
</thead>
<tbody>
<tr>
<td>AVPU/CNS Response</td>
<td>Alert (A)</td>
<td>Voice (V), Pain (P), Unresponsive (U).</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

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

Text Box 32: NEWS Criteria for CNS
Another common method of measuring CNS function is the “Glasgow Coma Scale” (GCS). The GCS is not included in the NEWS 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 8: Glasgow Coma Scale (GCS).

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.

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

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**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 3mmol/l (administer 50mls 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; 500mls 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 Outcomes

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.
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**

**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 $>0.5$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
(in the non-pregnant adult patient)

This chapter incorporates the principles outlined in the *Sepsis Management, National Clinical Guideline No. 6*

This guideline can be accessed at: [http://health.gov.ie/national-clinical-guideline-no-6-sepsis-management/](http://health.gov.ie/national-clinical-guideline-no-6-sepsis-management/)
Sepsis

Purpose
The purpose of this section is to inform and guide healthcare professionals in the recognition, treatment and appropriate referral of patients (non-pregnant adults) with sepsis, ultimately reducing mortality from sepsis in Ireland.

Aim
The aim is to ensure that there is a clear process that links early detection of the deteriorating patient with the requirement for sepsis screening and subsequent action.

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

- Improve sepsis recognition in patients using the National Early Warning Score (NEWS) as a trigger for sepsis screening;
- Describe the indicators relevant for a diagnosis of Systemic Inflammatory Response Syndrome (SIRS), sepsis, severe sepsis and septic shock;
- Ensure that patients with a diagnosis of sepsis have the Sepsis Six Regimen performed within one hour of diagnosis and care escalated appropriately.

Sepsis impact

“Sepsis is a life threatening condition that arises when the body's response to an infection injures its own tissues and organs. Sepsis leads to shock, multiple organ failure and death especially if not recognised early and treated promptly. Sepsis remains the primary cause of death from infection despite advances in modern medicine, including vaccines, antibiotics and acute care. Millions of people die of sepsis every year worldwide.”

Merinoff Symposium 2010: Sepsis

International estimates of incidence vary, but consensus points to approximately 200-337 cases per 100,000 population per annum (Hall et al. 2011). As comparators, myocardial infarction affects 208 patients per 100,000 per year (Yeh et al. 2010) and stroke 223 (Feigin et al. 2009). Mortality from sepsis is currently as high as mortality from acute myocardial infarction was in the 1960s (deVreede et al. 1991)

According to the Centers for Disease Control and Prevention, sepsis affects more than 800,000 Americans annually and is the ninth leading cause of disease-related deaths. The Agency for Healthcare Research and Quality lists sepsis as the most expensive condition treated in U.S. hospitals, costing more than $20 billion in 2011. In the U.K. sepsis is estimated to claim 37,000 lives annually and cost the NHS approximately £2.5 billion (UK Sepsis Trust)

Sepsis mortality ranges between 15% and 37% in the U.S. (and much higher in other jurisdictions) depending on whether the healthcare providers are thoroughly trained in identifying and treating sepsis. Sepsis incidence is predicted to grow at a rate of 1.5% annually. This is partly due to an aging population, increased numbers of invasive procedures and the increasing number of people living with co-morbidities and on long-term immunosuppressive therapies. Multiple studies have shown that programs aimed at early identification and
treatment of patients with sepsis leads to reduced mortality, intensive care unit (ICU) admission, ICU length of stay and hospital length of stay. (Shorr et al. 2007 and Whippy et al. 2004).

Definitions

**Infection** is defined as a pathological process caused by invasion of normally sterile tissue or fluid or body cavity by pathogenic or potentially pathogenic micro-organisms. It is important to point out that, frequently, infection is strongly suspected without being microbiologically confirmed.

**Systemic inflammatory Response Syndrome (SIRS)** is defined as an inflammatory state affecting the whole body, frequently a response of the immune system to infection, but not necessarily so. It is the body's response to an infectious or non-infectious insult.

**Sepsis** is the clinical syndrome defined by the presence of both infection and a systemic inflammatory response syndrome (SIRS). (9) However, since infection cannot be always microbiologically confirmed, the diagnostic criteria are infection, suspected or confirmed and the presence of some of the SIRS criteria (Levy et al. 2001).

**Severe sepsis** refers to sepsis complicated by organ dysfunction. (9) In the 8th Edition of the ICD-10-AM/ACHI/ACS this is extended to include organ failure. This difference does not affect the guideline diagnostic criteria which identify a minimum level of organ dysfunction beyond which severe sepsis is diagnosed.

**Septic shock** is defined as severe sepsis with circulatory shock with signs of organ dysfunction or hypoperfusion in the 8th Edition of the ICD-10-AM/ACHI/ACS. The diagnostic criteria in this guideline are applied after 30mls/kg isotonic fluid has been administered to reverse any hypovolaemia and are persistent systolic blood pressure <90 mmHg or MAP < 65 mmHg; decrease in systolic blood pressure by 40mmHg from normal and/or lactate >4 mmol/l (Dellinger et al. 2013).

The sources of sepsis are very consistent in the industrialised world with respiratory sepsis being the most common with rates between 35% and 50%. Table 1 reports the findings of the IMPRESS trial a 24 hour point prevalence study of severe sepsis/septic shock in Emergency departments and Intensive Care units in Europe, the US and Asia which were presented at the European Society of Intensive Care Medicine Annual Congress September 2014.

<table>
<thead>
<tr>
<th>Respiratory</th>
<th>35%</th>
</tr>
</thead>
<tbody>
<tr>
<td>Urinary</td>
<td>21%</td>
</tr>
<tr>
<td>Intra-abdominal</td>
<td>16.5%</td>
</tr>
<tr>
<td>Catheter-related blood stream infection</td>
<td>2.3%</td>
</tr>
<tr>
<td>Device-related</td>
<td>1.3%</td>
</tr>
<tr>
<td>CNS</td>
<td>0.8%</td>
</tr>
<tr>
<td>Others eg cellulitis, intra-articular</td>
<td>11.3%</td>
</tr>
</tbody>
</table>
Management of Sepsis in the Non-Pregnant Adult Patient

Sepsis Screening Pathway
A Sepsis Screening Pathway is available as a guide for healthcare professionals for the early detection of Sepsis, Severe Sepsis and Septic Shock (see Appendix 1). It is recommended that this pathway be included on the patient observation chart. There are some pathway modifications as follows:

- Not all patients meeting the modified Systemic Inflammatory Syndrome (SIRS) criteria have Sepsis
- OR there may be additional problems requiring different management including the following: current congestive cardiac failure (CCF), diabetic ketoacidosis (DKA), myocardial infarction (MI), gastro-intestinal (GI) bleed etc.
- OR the patient may be receiving chemotherapy, see below for Sepsis High Risk Group: Cancer Patients.
- OR be palliated.

High Risk Group: Cancer Patients
Febrile neutropenia is a common complication of cytotoxic chemotherapy. Progression to neutropenic sepsis can be a fatal complication resulting in hospital admissions, treatment delays and dose reductions. A small number of patients will develop life threatening sepsis as a result of neutropenia, and a mortality rate of approximately 5% has been reported in patients with solid tumours (a).

A number of UK reports highlighted problems in the management of neutropenic sepsis in adults receiving cytotoxic chemotherapy (b, c). These reports recommend the need to develop systems for urgent assessment and organisation-level policies for the management of neutropenic sepsis.

Key Points
- neutropenic sepsis is a medical emergency
- patient presentation may be non-specific and the possibility of infection must be considered in any patient undergoing anti-cancer treatment, particularly chemotherapy, who is unwell and particularly in those who are neutropenic (note: 2 SIRS criteria may not be present)
- suspected neutropenic sepsis must be treated with antibiotics within 1 hour of arrival at the hospital
- diagnose neutropenic sepsis in patients having anti-cancer treatment whose neutrophil count is 0.5 x 10⁹ per litre or lower, or less than 1 x 10⁹ with a downward trend who have either:
  - a temperature higher than 38oC or
  - other signs or symptoms consistent with clinically significant sepsis (d)
- chemotherapy-induced neutropenia is most likely to occur 10-14 days post-chemotherapy but should remain a consideration after this period. These patients are mainly treated in day ward settings.

**Definitions**

Neutropenia - An abnormal decrease in the number of neutrophils in the blood (d). Neutropenia is associated with a profound impairment in the inflammatory response, leading to a lack or minimisation of the usual signs and symptoms of infection. Neutropenia is a common problem in oncology patients either following chemotherapy, or less commonly secondary to radiation treatment or marrow infiltration by malignancy. Neutropenia is most likely to occur 10-14 days post-chemotherapy.

Febrile Neutropenia - occurs when a patient has a fever and a significant reduction in their neutrophils (4). The fever may be caused by an infectious agent, and when it is, rapid treatment is required. A patient with febrile neutropenia needs assessment for the possible source and type of infection and treatment until the cause is found or it subsides. The risk of infection increases directly in proportion to the degree of neutropenia and its duration.

Neutropenic sepsis and septic shock - An abnormal decrease in the number of neutrophils in the blood together with infection (d). Neutropenic sepsis and septic shock are life threatening and should be dealt with urgently. Sepsis describes clinical syndromes resulting from systemic inflammatory response. Diagnosis of sepsis is based on simple clinical criteria. The term septic shock represents an increased degree of the severity of sepsis where altered organ function or compromise occurs (e.g. hypotension, hypoxemia, lactic acidosis, oliguria, altered mental status). Septic shock carries high risk of mortality.

**References for this section**


b. National Confidential Enquiry into Patient Outcome and Death (NCEPOD) (2008), Systemic Anti-Cancer therapy: For better, for worse?


---

A Sepsis Screening Form is available for sepsis screening of individual patients, where appropriate (see Appendix 2). Following the screening process, this document must be filed in the patient’s notes and may be used for auditing purposes.

Management of the septic patient in the first hour is a time critical emergency and requires a team based approach involving all relevant healthcare staff members. This may be adapted for the local context depending on the composition of the team.

There are essentially four steps that should be taken in the management of sepsis in non-pregnant adults as outlined in Fig 3 below.
1. Detection of the Deteriorating Patient
The first step in appropriate management of a deteriorating patient with sepsis is recognition and timely management. The standardised National Early Warning Score in the Irish healthcare system has the advantage of reducing variation in practice thus minimising risk for the patient. The NEWS alerts clinicians that action is required to prevent further patient deterioration.

It is recommended that adult patients (non-pregnant) with a NEWS of 4 (5 on supplementary \( \text{O}_2 \)) should trigger sepsis screening.

2. Communication – ISBAR Communication Tool
Poor communication has been identified as a contributing factor to adverse incidents where clinical deterioration is not identified or properly managed. The recommended communication tool in healthcare is the ISBAR communication tool (Fig 4)

---

**Fig. 17: The four Steps of Sepsis Management (in the Non-Pregnant Adult Patient)**

1. **Detection**
   - NEWS ≥ 4 (or 5 when on \( \text{O}_2 \))

2. **Communication**
   - ISBAR Communication Tool

3. **Recognition**
   - Clinical Evaluation
   - Sepsis Screening Form

4. **Resuscitate & Refer**
   - Sepsis Six within 1 hour
   - Referral to Senior Clinicians & Critical Care

**Fig. 18: SIRS criteria**

**Fig 19: ISBAR Communication Tool**

Reproduced and adopted with permission from Dr L. Marshall, Monash University, Australia.
3. Recognition

Patients with NEWS 4 (5 on supplementary O$_2$) must be reviewed by a Senior House Officer (SHO) or more senior medical staff, within 30 minutes. The patient needs to be evaluated clinically for the likelihood of sepsis and potential sources of infection. Clinical judgment must always be used, as a diagnosis of sepsis is not made simply on an elevated NEWS. A diagnosis is made after taking a history, examining the patient and applying the sepsis diagnostic criteria outlined in Fig 1 above. Common sources of sepsis in adults are respiratory (35%), urinary tract (35%), intra-abdominal (10%), meningitis, septic arthritis, skin, and intra-vascular devices (10%).

Patients in whom severe sepsis or septic shock is suspected should be reviewed by a registrar or consultant immediately. THIS IS A TIME-DEPENDENT MEDICAL EMERGENCY similar to myocardial Infarction, stroke or trauma with a time-critical period within which to maximize the patient’s survival.

Where sepsis is not diagnosed do not proceed with the Sepsis Screening Form and further treatment, but continue to follow the NEWS protocol.

Where a diagnosis of sepsis is made but a decision has been made NOT to escalate care (excluding further treatment) then do not proceed with further treatment. There may be a requirement to exclude patients from specific elements of the Sepsis Six Regimen based on patient-specific clinical criteria.

Patients with a terminal illness should NOT be excluded from further treatment unless a decision not to escalate care clearly excludes further active treatment with antibiotics or IV fluids in the ward setting.

4. Resuscitation & Referral

Once a diagnosis of likely sepsis is made ‘The Sepsis Six Regimen” must be performed within an hour

<table>
<thead>
<tr>
<th>TAKE 3</th>
<th>GIVE 3</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. CULTURES: Take blood cultures <strong>before giving antimicrobials</strong> (if no significant delay i.e. &gt;45 minutes) and consider source control.</td>
<td>1. OXYGEN: Titrate O$_2$ supplementation to saturations of 94 -98% or 88-92% in chronic lung disease.</td>
</tr>
<tr>
<td>2. BLOODS: Check lactate &amp; full blood count.</td>
<td>2. FLUIDS: Start IV fluid resuscitation if evidence of hypovolaemia and/or shock. 500ml – 1000mls bolus of isotonic crystalloid over 15 – 30 minutes &amp; give up to 30ml/kg, reassessing after each bolus for signs of hypovolaemia, euvoelaemia, or fluid overload.</td>
</tr>
<tr>
<td>3. URINE OUTPUT: Assess urine output and consider urinary catheterisation for accurate measurement in patients with severe sepsis/septic shock.</td>
<td>3. ANTIMICROBIALS: Give IV antimicrobials according to local antimicrobial guidelines.</td>
</tr>
</tbody>
</table>
Note: Some groups of patients, such as older people, may not meet the modified SIRS criteria, even though infection is suspected. Where this occurs check for signs of organ dysfunction and raised biomarkers such as C-reactive protein (CRP).

Following a history and examination and in the absence of suspected infection staff may proceed with using the NEWS protocol

The rationale underpinning these interventions is as follows:

1. Cultures
Antibiotic therapy is guided by local antibiotic prescribing guidelines and based on the site of infection (as per clinical history and examination) or protocols for the management of pyrexia of unknown origin or neutropenic sepsis.

The ‘Start Smart and then Focus’ antibiotic prescribing care bundle outlines the principles of empiric antibiotic prescribing (Appendix 3). Broad spectrum antibiotics should be prescribed initially and then changed as appropriate when the culture results are available, thus minimizing antibiotic resistance and the complications of broad spectrum antibiotic use. To this aim blood cultures should be complemented by body fluid samples from apparently infected areas such as sputum, urine, wound swabs, swabs from Intravenous Cannula site (IVC), Central Venous Catheter (CVC), site and Cerebrospinal Fluid (CSF) as appropriate.

In recognition of the complexity of deep seated abscesses, infection and necrotic tissue source control should be attempted as a matter of urgency except in the case of pancreatitis when debridement is not recommended.

2. Lactate
The aim of fluid resuscitation is to restore $O_2$ delivery to the tissues to ensure normal functioning of the cells. In shock there is inadequate $O_2$ delivery due to inadequate perfusion of the tissues. This results in metabolism converting to an anaerobic mechanism to ensure ongoing energy production. The by-product of this process is lactate. The level of lactate corresponds to the level of inadequate cellular oxygenation and conversely the correction in lactate levels with fluid resuscitation correlates with improvements in tissue perfusion and survival. The Australian sepsis database has provided correlation between lactate levels on first presentation and subsequent mortality from sepsis (Fig 6). This data represents a benchmark against which sepsis mortality should be compared.

![Fig. 21: Correlation between lactate levels on first presentation and subsequent mortality from sepsis](Data from CEC HIE, Australia N=3851)

<table>
<thead>
<tr>
<th>Presenting Lactate level</th>
<th>Mortality</th>
</tr>
</thead>
<tbody>
<tr>
<td>Low (0.7 – 1.9)</td>
<td>4.5%</td>
</tr>
<tr>
<td>Intermediate (2 – 3.9)</td>
<td>10.6%</td>
</tr>
<tr>
<td>High (4+)</td>
<td>27.3%</td>
</tr>
</tbody>
</table>

Tissue hypoperfusion is not the only cause of raised lactate and therefore it has to be interpreted in its clinical context, whist a raised lactate does not diagnose severe sepsis, its presence in the context of sepsis does. In patients with irreversible shock lactate levels will remain elevated despite restoration of perfusion and this is associated with a poor outcome.
3. Urinary output Measurement
A urinary output of 0.5mls/kg/hr is one of the goals of resuscitation that guides fluid administration and has been shown to improve outcome in patients with severe sepsis and septic shock. The placement of a urinary catheter may be required to facilitate this measurement. Urinary catheter insertion should be performed as outlined in the HSE guidelines for the prevention of urinary catheter-related infection.

Patients with chronic oliguria/anuria still need early fluid resuscitation using the other markers of adequate perfusion i.e. lactate, haemodynamic variables +/- ScvO₂. As with all patients receiving a fluid bolus, response to therapy and signs of fluid overload need to be monitored and treatment tailored accordingly. An early critical care consult may be required as this group is vulnerable to severe sepsis and inadequate fluid resuscitation may lead to further deterioration.

4. Supplementary O₂ therapy
Patients are placed on O₂ and titrated to SpO₂ of 94-98% or 88-92% in the case of patients with COPD. Oxygen therapy is recommended because oxygen delivery to the tissues depends on oxygen saturation, cardiac output and haemoglobin levels, as described in the O₂ delivery equation (Fig 7)

Fig. 22: The O₂ Delivery Equation

<table>
<thead>
<tr>
<th>Oxygen Delivery Equation</th>
</tr>
</thead>
<tbody>
<tr>
<td>DO₂ = CO x (1.39 x Hb x SaO₂ + (0.003 x PaO₂))</td>
</tr>
</tbody>
</table>

DO₂ = oxygen delivery to the tissues; CO = Cardiac Output; Hb = haemoglobin; SaO₂ = oxygen saturation; PaO₂ = Partial Pressure of O₂
Fig. 23: National Clinical Guideline Sepsis Management Algorithm

**Adult Sepsis Management Algorithm**

**SUSPECTED INFECTION PLUS 2 SIRS CRITERIA = SEPSIS**

**Sepsis 6 in 1 hour**

- **Give**
  1. Oxygen (94-98% SpO2 or 88-92% COPO Patients)
  2. IV Antimicrobials (according to local guidelines)
  3. Fluids (500mls bolus: give up to 30ml/kg & reassess)

- **Take**
  1. Blood Cultures
  2. Lactate and FBC
  3. Urine Output measurement

If MAP* ≥65mmHg and/or Lactate ≤2mmol/L Document Sepsis

- **BP**
  - If MAP <65mmHg and/or Lactate ≥4 mmol/L
    - Fluid resuscitation of 30ml/kg IV in 1 HOUR
  - If Lactate <2mmol/L and/or MAP ≥65mmHg Document Sepsis

If Lactate ≥4mmol/L and/or MAP <65mmHg

- This is SEPTIC SHOCK Please document it.

If Lactate 2-4 mmol/L and/or MAP ≥65mmHg or organ dysfunction

- This is SEVERE SEPSIS Please document it.

Continue Fluid Resuscitation Call Critical Care Medicine

---

*MAP: Mean Arterial Pressure

For more information go to on National Clinical Guideline No 6. Sepsis Management go to: www.health.gov.ie/patient-safety/ncec
5. I.V. Fluid Resuscitation

Patients with sepsis can have absolute hypovolaemia, relative hypovolaemia and/or distributive shock. These are diagnosed by measuring heart rate, blood pressure, lactate and urinary output as well as assessing clinical signs of hypo-perfusion such as capillary refill or mottling. Patients with hypovolaemia without hypotension require fluid resuscitation to restore euvalamia and normal organ perfusion as evidenced by return of normal mentation, skin perfusion, urinary output and lactate levels.

<table>
<thead>
<tr>
<th>Patients presenting with systolic blood pressure (SBP) &lt;90, mean arterial blood pressure &lt;65mmHg, SBP decrease of 40mmHG from normal or serum lactate levels &gt; 4mmols/l should receive 30mls/kg of intravenous (IV) isotonic crystalloid fluid in the first hour unless fluid overload develops.</th>
</tr>
</thead>
</table>

Text Box 41: IV Fluid Resuscitation

If the patient develops signs and symptoms of overload, IV fluid administration should be stopped and vasoactive agents be used to restore SBP to > 90mmHg or mean arterial blood pressure (MAP) to > 65mmHg. After fluid resuscitation with 30mls/kg isotonic crystalloid, hypotension may persist requiring further fluid resuscitation and vasopressors. This is septic shock. Myocardial dysfunction may also occur.

Fluid resuscitation is performed by giving fluid boluses (e.g. 500mls – 1000mls) over a given time period (e.g. 15 – 30 minutes) and the patient’s response assessed by clinical examination after the bolus has been given and a decision made as to whether the patient needs further fluid resuscitation or not. The amount of the bolus and the time period over which it is given depends on the co-morbidities of the individual patient. Serial measurement of lactate and urinary output measurement can help guide fluid resuscitation.

Sepsis represents a clinical continuum ranging from patients presenting haemo-dynamically stable with no overt fluid deficit as indicated by normal organ function, urinary output and lactate who require little or no fluid resuscitation and only maintenance fluids if fasting, through those that have restoration of haemo-dynamics and organ function after fluid resuscitation as demonstrated by normalisation of mental status, skin perfusion, urinary output and lactate and then require only maintenance fluids, if fasting, to those with persistent hypotension and organ dysfunction post initial fluid resuscitation who require critical care input and consideration of invasive monitoring, advanced haemo-dynamic support (e.g. vasopressors) and further guided fluid resuscitation. An example of a fluid resuscitation algorithm is included below.
Fig. 24: Fluid Resuscitation Algorithm for Adults with Sepsis.

MAP: Mean Arterial Pressure, SBP: Systolic Blood Pressure

* Euvolaemia can be difficult to assess in patients with distributive shock. The patients in the ProCESS and ARISE trials received, on average between 4 and 5 litres of isotonic crystalloid fluid in the first 6 hours, of this 30mls/kg and 34mls/kg of IVT was administered in the first hour respectively.

For more information go to on National Clinical Guideline No 6. Sepsis Management go to: www.health.gov.ie/patient-safety/nccg
6. I.V. Antibiotics

| Antibiotic therapy – Check your hospitals antibiotic guidelines |
| Prescribe it |
| Get it |
| Give it ………. NOW! |

It has been demonstrated that there is a 7.6% increase in mortality for each hour delay in the administration of appropriate antibiotics in patients with severe sepsis and septic shock (Kumar 2006).

There are five general principles that should be considered when choosing antibiotic therapy as follows:

1. Clinical assessment - what is the likely site of infection?
2. Is the infection community, healthcare or hospital-acquired?
3. Has the patient been prescribed antibiotics in the recent past, including from other wards/GP/other hospitals?
4. Are there recent microbiology results available, including history of colonisation/infection with antibiotic resistant organisms such as MRSA & ESBL E. coli?
5. Have I consulted my local antibiotic prescribing guidelines?

When prescribing an antibiotic it is important to document the following:

1. Treatment indication;
2. Drug name, dose, frequency and route;
3. Treatment duration (or review date).

Using local antibiotic guidelines is superior to physicians’ own choice of antibiotic. Local antibiotic guidelines must be followed to guide the best choice of empiric antibiotic therapy. This is to ensure that the antibiotic chosen is appropriate for the local epidemiology. If you are in doubt, contact your consultant clinical microbiologist/infectious diseases specialist for advice.

7. Measuring Improvements in Sepsis Management

The Sepsis Steering Group recommends that hospitals should measure the level of compliance with each element of ‘Sepsis Six’ as part of a ward/unit/directorate quality improvement programme for sepsis.

Each ward/unit/directorate should agree a measurement plan for sepsis that is practical and aligns with other measurements for improvement. The measurement strategy needs to reflect the reality that patients can present with sepsis on admission or develop sepsis while in hospital. It should also include consideration of measurement of some balancing measures as outlined below.
Sources of data: The patient’s medical notes, medication chart, NEWS chart, fluid balance chart and Sepsis Screening Form.

AMAU/MAU Sample five patients with sepsis per week. If total patient numbers are less than 20 per month, all patients should be included in the sample. Patients who are identified having suspected sepsis should have interventions completed within one hour of diagnosis of sepsis.

Wards: Identify one medical and one surgical ward to start. Collect data on one day each week. Look at all patients on the ward that day. Identify any with NEWS ≥4 (5 on supplementary O₂) at any time during the past week. Take time zero as the time the patient developed a NEWS score of ≥4 (5 on supplementary O₂) + two or more modified SIRS criteria + suspicion of infection. Patients who have suspected sepsis should have the ‘Sepsis 6 Regimen’ interventions completed within one hour of time zero.

Exclusion Criteria: Patients with a terminal illness should NOT be excluded from further treatment unless a decision not to escalate care clearly excludes further active treatment with antibiotics or IV fluids in the ward setting. There may be a requirement to exclude patients from specific elements of the Sepsis 6 Regimen based on patient-specific clinical criteria.

Text box 42: Measuring Sepsis Improvement

Balancing Measures
Excessive or inappropriate antibiotic use is associated with increased antimicrobial resistance, C. difficile infection and adverse drug reactions. There are at least three potential unintended consequences of implementing sepsis screening as follows:

1. Patients with an elevated NEWS but no evidence of sepsis/potential infective source are commenced inappropriately on antibiotic therapy;
2. Patients with suspected sepsis are commenced on inappropriate antibiotic therapy that is not in line with local guidelines;
3. Empiric antibiotic therapy that has been commenced in a patient with suspected sepsis is not reviewed at 24-48 hours as recommended by the Start Smart & Then Focus Antibiotic Care bundle. At this stage an antimicrobial prescribing decision needs to be made and one of the five prescribing decision options chosen as follows:
   a. Stop antibiotic(s) - no evidence of bacterial infection, or infection resolved;
   b. Switch from intravenous to oral antibiotic(s) - if patient meets criteria for oral switch as outlined in local antibiotic prescribing guidelines;
   c. Change antibiotic(s) to a narrower spectrum agent, if possible;
   d. Continue current antibiotic(s) and review again after further 24 hours;
   e. Consult with local Outpatient Parenteral Antibiotic Therapy (OPAT) team.

All hospitals should have antimicrobial stewardship programmes in place that monitor process or outcome measures to ensure that antibiotics are not being prescribed unnecessarily, due to inappropriate application of Sepsis Six. Examples of process and outcome measures that would be appropriate for use as balancing measures are outlined in Table 1. Ensuring antibiotics are used appropriately for all infections, not just those associated with sepsis, will help to ensure effective antibiotic therapy is available when cases of sepsis occur.
Table 10: Balancing measures for monitoring implementation of the Sepsis 6 Regimen

<table>
<thead>
<tr>
<th>Measure</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Appropriate Antibiotic Use</strong></td>
</tr>
<tr>
<td>- Audit of compliance with the Start Smart &amp; Then Focus antibiotic</td>
</tr>
<tr>
<td>care bundle</td>
</tr>
<tr>
<td>- Audit of proportion of empiric antibiotic prescription that complies</td>
</tr>
<tr>
<td>with local prescribing guidelines</td>
</tr>
<tr>
<td>- Monitoring of hospital antibiotic consumption, with particular</td>
</tr>
<tr>
<td>emphasis on broad spectrum antibiotics (e.g. third generation</td>
</tr>
<tr>
<td>cephalosporins, anti-pseudomonal penicillins, carbapenems,</td>
</tr>
<tr>
<td>fluoroquinolones)</td>
</tr>
<tr>
<td><strong>Consequences of Antibiotic Use</strong></td>
</tr>
<tr>
<td>- Antimicrobial resistance rates – for example</td>
</tr>
<tr>
<td>- Proportion of antimicrobial resistance among bloodstream</td>
</tr>
<tr>
<td>isolates,</td>
</tr>
<tr>
<td>- Rates of colonisation with antimicrobial resistant pathogens</td>
</tr>
<tr>
<td>such as MRSA or multiple drug-resistant Gram-negative bacteria</td>
</tr>
<tr>
<td>- New cases of hospital-acquired <em>Clostridium difficile</em> infection</td>
</tr>
<tr>
<td>- New cases of hospital-acquired Candida infections</td>
</tr>
<tr>
<td><strong>Medication Safety</strong></td>
</tr>
<tr>
<td>- Medication safety incidents associated with antibiotic therapy</td>
</tr>
</tbody>
</table>

Figure 25: Start Smart, Then Focus

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Start Smart, Then Focus
An Antibiotic Care Bundle for Hospitals

**Day 1: Start Smart...**

1. Start antibiotics only if there is clinical evidence of bacterial infection
   - If there is evidence of bacterial infection, prescribe in accordance with your local antibiotic guidelines and appropriately for the individual patient (see notes below)
2. Obtain appropriate cultures before starting antibiotics
3. Document in both the drug chart and medical notes:
   - Treatment indication
   - Drug name, dose, frequency and route
   - Treatment duration (or review date)
4. Ensure antibiotics are given within four hours of prescription
   - Within 1 hour for severe sepsis or neutropenic sepsis

---

**...then Focus (Day 2 onwards)**

At 24-48 hours after starting antibiotics, make an Antimicrobial Prescribing Decision
- Review the clinical diagnosis
- Review laboratory/radiology results
- Choose one of the five options below
- Document this decision

**Options**

1. Stop antibiotic(s)
   - No evidence of bacterial infection, or infection resolved
2. Switch from intravenous to oral antibiotic(s)
   - If patient meets criteria for oral switch
3. Change antibiotic(s)
   - Narrower spectrum, if possible; broader spectrum, if indicated
4. Continue current antibiotic(s)
   - Review again after further 24 hours
5. Outpatient parenteral antibiotic therapy
   - Consult with local OPAT team

When deciding on the most appropriate antibiotic(s) to prescribe, consider the following factors:
- History of drug allergy (document allergy type: minor, major, anaphylaxis, angioedema)
- Recent culture results (e.g. in patient colonized with a multiple-resistant bacteria?)
- Recent antibiotic treatment
- Potential drug interactions
- Potential adverse effects (e.g. difficult infection is more likely with broad-spectrum antibiotics)
- Some antibiotics are considered unsafe in pregnancy or young children
- Dose adjustment may be required for renal or hepatic failure

Consider removal of any foreign body/implanting device, drainage of pus, or other surgical intervention.

For advice on appropriate investigation and management of infections, consult your local infection specialist(s) (microbiologist, infectious disease physician and/or antimicrobial pharmacist)
Communication, Teamwork and Management Plans
Communication, Teamwork and Management Plans

Learning Outcomes

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 43: Optimising Management
Figure 26: Steps in Managing a Deteriorating Patient (Flow Chart)

National Early Warning Score
Deteriorating Patient Management Flow Chart

Initial Assessment - A B C D E
- Problems with Airway
- Breathing
- Circulation
- Deterioration - Neurological
- E - Pyrexia/Hypothermia

EWS 3
- Notify Nurse in Charge & Team / On-Call SHO

Initial Management
- Consider: Oxygen
- Airway Adjuncts
- IV access
- Blood Sugar Level

EWS 4-6
- Call For Help
- Team / On-Call SHO

EWS 7
- Team / On-Call Registrar

Patient Improving

Yes
- Do you have a diagnosis?
  - Yes
  - Definitive Management Plan
  - No
- Special Investigations
- Inform /Team/On-Call Consultant
- Plan to transfer to higher level of care

No
- Definitive Care

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.
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.

<table>
<thead>
<tr>
<th>Information can be obtained from:</th>
</tr>
</thead>
<tbody>
<tr>
<td>• Verbal contact with members of the team</td>
</tr>
<tr>
<td>• Reading the daily/updated notes from each different team member</td>
</tr>
<tr>
<td>• Reviewing observations, fluid and medication charts</td>
</tr>
<tr>
<td>• Comparing current presentation with previous presentations</td>
</tr>
<tr>
<td>• Family, friends or the patient themselves</td>
</tr>
</tbody>
</table>

Text Box 44: 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 45: Abnormalities and Appropriate Care
When a NEWS triggers a communication, describe the observations that have triggered the NEWS (e.g. total NEWS 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 for deteriorating patients

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.

Table 11: ISBAR Communication for patient deterioration.

<table>
<thead>
<tr>
<th>ISBAR Communication Tool SAMPLE</th>
<th>Patient Deterioration</th>
</tr>
</thead>
<tbody>
<tr>
<td>Identify</td>
<td>You</td>
</tr>
<tr>
<td></td>
<td>Recipient of handover information</td>
</tr>
<tr>
<td>Situation</td>
<td>Why are you calling?</td>
</tr>
<tr>
<td></td>
<td>(identify your concerns)</td>
</tr>
<tr>
<td>Background</td>
<td>What is the relevant background?</td>
</tr>
<tr>
<td>Assessment</td>
<td>What do you think is the problem?</td>
</tr>
<tr>
<td>Recommendation</td>
<td>What do you want them to do?</td>
</tr>
</tbody>
</table>

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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 NEWS 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 NEWS 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 46: 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.
a. 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.

b. 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.

c. 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.

d. Change in Patient Therapy Orders
This may include changing antibiotics from oral to intravenous, or adding a diuretic.

e. 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.

f. 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 47: 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 48: 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.
References and Bibliography


Resources required to deliver the COMPASS© Education Programme incorporating National Early Warning Score 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: newsinfo@hse.ie.

Special thanks to: Matt Gladwish of ACT Publishing Services for his assistance in developing the CD. Dr. Ross Peake for his voice talents on the CD.
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: .............................

<table>
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<tr>
<th>Quiz Questions</th>
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<tbody>
<tr>
<td>1. An increased heart rate may reflect:</td>
</tr>
<tr>
<td>a. Failing oxygen delivery</td>
</tr>
<tr>
<td>b. Decreased level of consciousness</td>
</tr>
<tr>
<td>c. Decreased urine output</td>
</tr>
<tr>
<td>d. Rise in stroke volume</td>
</tr>
<tr>
<td>2. Compensation for a decreased oxygen delivery will include which of the following?</td>
</tr>
<tr>
<td>a. Increased urine output</td>
</tr>
<tr>
<td>b. Decreased peripheral vascular resistance</td>
</tr>
<tr>
<td>c. Decreased respiratory rate</td>
</tr>
<tr>
<td>d. Increased heart rate</td>
</tr>
<tr>
<td>3. Cardiac Output =</td>
</tr>
<tr>
<td>a. Blood pressure x heart rate</td>
</tr>
<tr>
<td>b. Stroke volume x blood pressure</td>
</tr>
<tr>
<td>c. Stroke volume x heart rate</td>
</tr>
<tr>
<td>d. Stroke volume x peripheral vascular resistance</td>
</tr>
<tr>
<td>4. Aerobic metabolism:</td>
</tr>
<tr>
<td>a. Generates 2ATP</td>
</tr>
<tr>
<td>b. Produces lactate</td>
</tr>
<tr>
<td>c. Requires oxygen</td>
</tr>
<tr>
<td>d. Causes tachypnoea</td>
</tr>
<tr>
<td>5. Tachypnoea:</td>
</tr>
<tr>
<td>a. Is caused by Morphine</td>
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<tr>
<td>b. Is caused by metabolic alkalosis</td>
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<tr>
<td>c. Reflects a deteriorating patient</td>
</tr>
<tr>
<td>d. Is caused by respiratory alkalosis</td>
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<tr>
<td>6. Blood pressure =</td>
</tr>
<tr>
<td>a. Peripheral vascular resistance x cardiac output</td>
</tr>
<tr>
<td>b. Stroke volume x heart rate</td>
</tr>
<tr>
<td>c. Stroke volume x peripheral vascular resistance</td>
</tr>
<tr>
<td>d. Cardiac output x heart rate</td>
</tr>
<tr>
<td>7. Oxygen delivery =</td>
</tr>
<tr>
<td>a. PO(_2) x cardiac output</td>
</tr>
<tr>
<td>b. O(_2) content x cardiac output</td>
</tr>
<tr>
<td>c. PO(_2) x blood pressure</td>
</tr>
<tr>
<td>d. Arterial oxygen content x cardiac output</td>
</tr>
</tbody>
</table>
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
Appendices
APPENDIX I: Sepsis Screening Pathway

Sepsis Screening Pathway
(ALWAYS USE CLINICAL JUDGEMENT)

Use this Sepsis Screening Pathway if the National Early Warning Score (NEWS) is ≥ 4 (5 on supplementary O₂) and infection suspected

Doctor must review within 30 mins (use ISBAR)

**INFECTION SUSPECTED**

- Respiratory rate > 20 (rpm)
- Heart rate > 90 (rpm)
- WCC < 4 or > 12 x 10⁹/L
- Temperature < 36 or > 38.3°C
- Acutely altered mental status
- Bedside glucose > 7.7mmol/L

OR Patient unwell and at risk of Neutropenia

Note: Some groups of patients, such as older people, may not meet the modified SIRS criteria, even though infection is suspected. Where this occurs check for signs of organ dysfunction and raised biomarkers such as C-reactive protein (CRP)

NO Following a history and examination, and in the absence of suspected infection, staff may proceed with using the NEWS protocol

YES. THIS IS SEPSIS

Sepsis Six Regimen must be completed within 1 hour

Has a decision been made NOT to escalate care (excluding further treatment)?

NO proceed

YES do not proceed

**SEPSIS SIX** – aim to complete within 1 hour

1. **Blood cultures before** giving antibiotics
   - Do not delay antibiotic administration > 1 hour if blood cultures are difficult to obtain. Send samples from potentially infected sites eg. sputum, urine, wounds, IVCVC. Consider source control.
   
2. Lactate and FBC
3. Urine output measurement

Laboratory tests must be requested as EMERGENCY and aim to have results available and **acted on within the hour**

- **Look for signs of organ dysfunction:**
  - Systolic BP < 90 or Mean Arterial Pressure < 65 or Systolic BP more than 40 below patient's normal
  - New need for oxygen to achieve saturation > 90%
  - Lactate > 2 mmol/L (following administration of fluid bolus)
  - Urine output < 0.5ml/kg for 2 hours – despite adequate fluid resuscitation
  - Acutely altered mental status
  - Glucose > 7.7 mmol/L (in the absence of diabetes)
  - Creatinine > 177 micromol/L
  - Bilirubin > 34 micromol/L
  - PT/INR > 1.5 or aPTT > 60s
  - Platelets < 100 x 10⁹/L

Any organ dysfunction: **THIS IS SEVERE SEPSIS**

Registrar or Consultant to review immediately. Reassess frequently in 1st hour.

Consider other investigations and management

- **Look for signs of septic shock** (following administration of fluid bolus)
  - Lactate > 4 mmol/L
  - Hypotensive (Systolic BP < 90 or MAP < 65)

If either present: **THIS IS SEPTIC SHOCK**

Critical care consult required

- Consultant referral
- Consider transfer to a higher level of care
- Critical care consult requested

A critical care review may be requested at any point during this assessment, but is required for patients with Septic Shock. In a hospital with no critical care unit, a critical care consult must be made and transfer to a higher level of care considered, if appropriate, following the consult.

**Pathway Modification**

Not all patients meeting modified SIRS criteria have sepsis. OR there may be additional problems requiring different management (current Congestive Cardiac Failure (CCF), Diabetic Ketonacidosis (DKA), Myocardial Infarction (MI), Gastro-Intestinal (GI) Bleed etc)

OR patient may be receiving chemotherapy

OR be palliated.
APPENDIX II: Sepsis Screening Form

Sepsis Screening Form

[ALWAYS USE CLINICAL JUDGEMENT]

Complete this form and apply if the National Early Warning Score (NEWS) is ≥ 4 (5 on supplementary O2) and infection suspected

CLINICIAN TO COMPLETE THIS SECTION

<table>
<thead>
<tr>
<th>Date:</th>
<th>Time:</th>
</tr>
</thead>
</table>

NEWS:

Name of Doctor contacted:

Clinician's Name:

Clinician's Signature:

MCR/NMBI PIN:

Doctor must review within 30 mins (use ISBAR). DOCTOR TO COMPLETE REMAINDER OF THIS DOCUMENT AS APPROPRIATE

**INFECTION SUSPECTED**

☐ Respiratory rate > 20 bpm (pm)

☐ Heart rate > 90 bpm

☐ WCC < 4 or > 12 x 10⁹/L

☐ Temperature <36 or >38.3°C

☐ Acutely altered mental status

☐ Bedside glucose >7.7 mmol/L (In the absence of diabetes mellitus)

OR  ☐ Patient unwell and at risk of Neutropenia

Note: Some groups of patients, such as older people, may not meet the modified SIRS criteria, even though infection is suspected. Where this occurs check for signs of organ dysfunction and raised biomarkers such as C-reactive protein (CRP)

☐ NO  Following a history and examination, and in the absence of suspected infection, staff may proceed with using the NEWS protocol

☐ YES, THIS IS SEPSIS  Time Zero: Sepsis Six Regimen must be completed within 1 hour

Has a decision been made NOT to escalate care (excluding further treatment)?

☐ NO (proceed)  ☐ YES (do not proceed)

**TAKING 3**

**SEPSIS SIX** – aim to complete within 1 hour

☐ 1. Blood cultures before giving antibiotic

   *Do not delay antibiotic administration >1 hour if blood cultures are difficult to obtain. Send samples from potentially infected sites eg. sputum, urin, wounds, IV/CVC. Consider source control.*

☐ 2. Lactate and FBC

☐ 3. Urine output measurement

Laboratory tests must be requested as EMERGENCY and aim to have results available and acted on within the hour

**GIVING 3**

☐ 4. O₂ (64-99 SpO₂ or 88-92% in COPD patients)

☐ 5. IV fluid resuscitation

   (500ml bolus - give up to 30ml/kg) & reassess (target systolic BP = 90/MMAP = 65)

   *Monitor response to IV fluids and titrate to effect

☐ 6. IV antimicrobials according to local guidelines

   *Time given:

   *Laboratory results:*

   *Signs of organ dysfunction:

   *Look for signs of organ dysfunction:

   ☐ Systolic BP < 90 or Mean Arterial Pressure

   < 65 or Systolic BP more than 40 below patient's normal

   ☐ New need for oxygen to achieve saturation > 90%

   ☐ Lactate > 2 mmol/L (following administration of fluid bolus)

   ☐ Urine output < 0.5 ml/kg for 2 hours – despite adequate fluid resuscitation

   ☐ Acutely altered mental status

   ☐ Glucose > 7.7 mmol/L (In the absence of diabetes)

   ☐ Creatinine > 177 micromol/L

   ☐ Bilirubin > 34 micromol/L

   ☐ PTR > 1.5 or aPTT > 60s

   ☐ Platelets < 100 x 10⁹/L

   *Any organ dysfunction: THIS IS SEVERE SEPSIS

   Registrar or Consultant to review immediately.

   Reassess frequently in 1st hour.

   Consider other investigations and management

   *Look for signs of septic shock (following administration of fluid bolus)*

   ☐ Lactate > 4 mmol/L

   ☐ Hypotensive (Systolic BP < 90 or MAP < 65)

   **If either present: THIS IS SEPTIC SHOCK**

   Critical care consult required

   ☐ Consultant referral

   ☐ Consider transfer to a higher level of care

   **Pathway Modification**

   Not all patients meeting modified SIRS criteria have sepsis. OR there may be additional problems requiring different management (current Congestive Cardiac Failure (CCF), Diabetic Ketoacidosis (DKA), Myocardial Infarction (MI), Gastro-Intestinal (GI) bleed etc) OR patient may be receiving chemotherapy OR be palliated.
APPENDIX III: Start Smart, Then Focus (Antibiotic Care Bundle).

Start Smart, Then Focus
An Antibiotic Care Bundle for Hospitals

Day 1: Start Smart...

1. Start antibiotics only if there is clinical evidence of bacterial infection:
   - If there is evidence of bacterial infection, prescribe in accordance with your local antibiotic guidelines and appropriately for the individual patient (see notes below)
2. Obtain appropriate cultures before starting antibiotics
3. Document in both the drug chart and medical notes:
   - Treatment indication
   - Drug name, dose, frequency and route
   - Treatment duration (or review data)
4. Ensure antibiotics are given within four hours of prescription:
   - Within 1 hour for severe sepsis or neutropenic sepsis

Then Focus (Day 2 onwards)

At 24-48 hours after starting antibiotics, make an Antimicrobial Prescribing Decision
- Review the clinical diagnosis
- Review laboratory/radiology results
- Choose one of the five options below
- Document this decision

Options
1. Stop antibiotic(s)
   - no evidence of bacterial infection, or infection resolved
2. Switch from intravenous to oral antibiotic(s)
   - if patient meets criteria for oral switch
3. Change antibiotic(s)
   - narrower spectrum, if possible; broader spectrum, if indicated
4. Continue current antibiotic(s)
   - review again after further 24 hours
5. Outpatient parenteral antibiotic therapy
   - consult with local OPAT team

When deciding on the most appropriate antibiotics to prescribe, consider the following factors:
- History of drug allergy (document allergy type: minor rash only or major [anaphylaxis, angioedema])
- Recent culture results (e.g. is patient colonised with a multiple-resistant bacteria?*
- Recent antibiotic treatment
- Potential drug interactions
- Potential adverse effects (e.g. C. difficile infection is more likely with broad spectrum antibiotics)
- Some antibiotics are considered unsafe in pregnancy or young children
- Dose adjustment may be required for renal or hepatic failure

Consider removal of any foreign body/indwelling device, drainage of pus, or other surgical intervention

For advice on appropriate investigation and management of infections, consult your local infection specialist(s) (microbiologist, infectious diseases physician and/or antimicrobial pharmacist)
APPENDIX IV: Sample driver diagram for improving care of adult in-patients with sepsis

To improve the recognition and the timely management of Sepsis in X

- Reliable care
  - Reliable care and process
    - Patient and family centered care
  - Reliable education and awareness
    - Education and awareness

- Reliable Sepsis Screening (NEWS)
  - Reliable communication across clinical and at risk patients
  - Reliable Sepsis Screening (NEWS) (N.E.W.S.)

- Reliable processes and systems
  - Reliable processes and systems
    - Develop measurement framework to guide
    - Reliable measurement framework

- Reliable care delivery
  - Reliable care delivery
    - Improve antimicrobial stewardship
    - 3 day improved performance

- Reliable culture of safety and quality improvement
  - Reliable culture of safety and quality improvement
    - Support from senior management
    - Clinical leadership
    - Multidisciplinary team working

- Reliable recognition
  - Reliable recognition
    - Improve knowledge and skills in clinical workforce
    - NEWS - Day 3

- To improve the recognition and the timely management of Sepsis in X
Appendix V: Algorithm for Standard Therapy for Septic Shock (used as part of the ProCESS Trial in the US)

In the event of difficult access an intra-osseous cannula can also be used.

**Abbreviations**

(IVF–intravenous fluids; HCT–hematocrit; SBP–systolic blood pressure; SI–shockindex; CVP–central venous pressure; ScvO₂–central venous oxygen saturation; MAP–mean arterial pressure; PRBC–packed red blood cells).

1. Central line should only be placed and used for venous access. During the 6-hour intervention, CVP and ScvO₂ measurements are discouraged. If time sensitive fluid targets can be achieved with smaller IV cannulae (e.g. one 18g and one 20g), that is acceptable.
2. Only isotonic fluid should be used (e.g., saline, lactated Ringer’s). Colloids are neither encouraged nor excluded.
3. Fluid replete / overload is defined here as a clinical diagnosis by the treating physician. Signs and symptoms of overload include jugular venous distention, rales, and decreased pulse oximetry readings. Discontinue all IV fluids (boluses, background rate) once this occurs, until no longer deemed fluid replete / overload.
4. If patient’s SBP is within 10% of known baseline SBP, AND patient is not deemed to be clinically hypoperfused, the SBP > 100 mmHg target can be deemed fulfilled. Arterial lines allowed if deemed necessary, but not mandatory. Shock index = heart rate / systolic blood pressure.
5. Hypoperfusion is defined here as a clinical diagnosis by the treating physician. Signs and symptoms include, but are not limited to, MAP < 65 despite SBP > 100, lactate > 4, mottled skin, oliguria, and altered sensorium.
6. Transfuse PRBCs for Hgb < 7.5 g/dL.
7. From time of prompt by protocol (i.e., not from time of physician order, or from when intravenous fluid bag is hung).

---

<table>
<thead>
<tr>
<th>Time-sensitive target</th>
<th>Time allowed</th>
<th>Corrective action</th>
</tr>
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<tbody>
<tr>
<td>Fluid bolus (500-1000 ml)</td>
<td>20 minutes</td>
<td>3rd IV or central line</td>
</tr>
<tr>
<td>Initial fluid bolus (2 L)</td>
<td>1 hour</td>
<td>3rd IV or central line</td>
</tr>
<tr>
<td>SBP ≥ 100 mmHg</td>
<td>1 hour</td>
<td>Vasopressors</td>
</tr>
</tbody>
</table>
**Appendix VI:** National Early Warning Score Project and COMPASS Education Programme

Governance Group membership

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Professor Garry Courtney
Professor Shane O’Neill
A/Prof. Michael Shannon
Avilene Casey
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John Kenny
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Carmel Cullen
Dr. Maria Donnelly
Professor Frank Keane
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APPENDIX VII: National Early Warning Score and associated Education Programme
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Marie Horgan  St. Lukes Hospital Kilkenny
Margaret Gleeson  Mid-Western Regional Hospital, Nenagh
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Anne Marie Oglesby  Clinical Indemnity Scheme
Kathleen McMahon  Cavan General Hospital
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Paula McElligott  MRH Mullingar
Siobhan Scanlon  Cork University Hospital
Ann Scahill  Roscommon County Hospital
Dr. John Cullen  AMNCH, Dublin
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