“Pointing you in the right direction”

Dr Bronwyn Avard
Ms Heather McKay
Ms Nicole Slater
Dr Kathryn Daveson
Dr Paul Lamberth
Dr Tony Lafferty
Ms Susan Chen
Mr John Darvill
Dr Imogen Mitchell

COMPASS © 2008

All forms of copyright in relation to the manual and CD are held by the Australian Capital Territory.
For enquiries about the COMPASS course or copies of the manual contact
Heather McKay
Program Manager
ACT Health
compass@act.gov.au
02 6244 3885

Disclaimer

The authors, the Australian Capital Territory or ACT Health cannot be held responsible for any loss, damage, or injury incurred by any individual or groups using this manual

First Edition April 2009
All rights reserved: no part of this publication may be photocopied, recorded or otherwise reproduced, stored in a retrieval system or transmitted in any form by any electrical or mechanical means, without the prior permission of:
ACT Health
CONTENTS

Introduction 3
Modified Early Warning Scores / Children’s Early Warning Tool 5
Oxygen Delivery 15
Airway and Breathing 19
Circulation 32
Central Nervous System & Urine Output 45
Communication, Teamwork, Management Plans 54
Introduction

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

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

Paediatric Patients

This symbol will indicate the important differences between adult and paediatric patients in the presented material. The paediatric specific information will be presented at the end of each section.

It has become increasingly apparent that many clinicians are unable to manage a deteriorating patient in an appropriate, timely fashion. This is often as a result of the inability of recognising that the patient is deteriorating. Delaying resuscitation and treatment increases the likelihood of organs failing due to inadequate oxygen delivery to these tissues. This in turn can lead to unexpected death, unexpected cardiac arrests and unplanned admissions to the intensive care unit.

It is important to understand the key components that lead to appropriate management.

a) Absence of observations
   - Equipment not available
   - Equipment malfunctioning
   - Inability to use equipment due to lack of knowledge
   - Inadequate time to perform observations
   - Inability to make time for performing observations
   - Lack of understanding of why observations are important
   - General culture that observations are not important

b) Inability to understand observations recorded
   - Unable to trend results and interpret the meaning
   - Lack of knowledge

c) Failure to trigger timely appropriate response
   - Absence of observations to make an interpretation
   - Inability to understand observations recorded
   - Inability to develop a diagnosis
   - Inability to develop a treatment plan

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

An example of what can go wrong

A 60 yr old male was admitted with pancreatitis.

- Over a period of twenty-four hours there were thirteen occurrences of hypotension, with the systolic blood pressure ranging from 64 to 86 mmHg during this time. The patient met the Medical Emergency Team (MET) criteria but the MET was not called.
- Respiratory Rate was inconsistently documented.
- The patient was being reviewed and treated by a junior medical officer without real improvement seen in the patient over time and staff failed to escalate the issue to a more senior doctor.
The patient was seen by a registrar 15 ½ hours after the first occurrence of low blood pressure however by this stage it was too late to correct the physiological deterioration and the patient died 14 hours later.

The System issues identified were:
- Failure to follow hospital policy in calling a MET
- Inadequate documentation of observations, particularly respiratory rate
- Failure to change the management plan of the patient despite its inadequacy
- Failure to escalate the level of medical review despite the seriousness of the situation

This education package, in conjunction with a clearly formatted observation chart and utilisation of a track and trigger system, aims to prevent such cases

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

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

How it works
There are three phases to the package to be completed in the following order:
- An online training program
- An online quiz
- A 3 hour face-to-face session

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

Once you have completed the case study you will proceed to a multiple-choice quiz to test your knowledge. You will be unable to skip ahead in the CD, however you will be able to go back to any area that you have already completed if you choose to.

The online training program MUST be completed prior to coming to the face-to-face session you have been scheduled for.

You MUST also access the online quiz. This again is multiple choice and MUST be completed prior to the final session.

OK LET'S GET STARTED
Modified Early Warning Scores (MEWS)

Children’s Early Warning Tool (CEWT)
Modified Early Warning Scores

Learning Objectives

- Be able to calculate a Modified Early Warning Score (MEWS).
- Be aware of your responsibilities when a trigger score is met.
- Understand how to complete the new observation chart.

A “vital” sign is a sign that pertains to life without which life would not exist. Derangements in pulse and blood pressure measurements can reflect an increased risk of life not existing and so can be considered a “vital sign”.

If derangements in pulse and blood pressure measurements reflect an increase in the risk of death, it is important that these signs are detected early and appropriate treatment is delivered to not only normalise these signs but also decrease the risk of the patient dying.

Background

A MEWS is a simple bedside score and track and trigger system that is calculated by nursing staff from the observations taken, to try and indicate early signs of a patient’s deterioration. It is a valuable additional tool to facilitate the detection of deteriorating patients, in particular in acute wards where patients are often quite unwell and there may be many inexperienced staff. The MEWS looks at all the observations together, not just a single observation in isolation. It includes respiratory rate, oxygen saturations, temperature, blood pressure, heart rate, sedation score, and urine output.

Trigger Score:
A score of 4 is the initial trigger point for action, with escalated notification at 6 and 8.

The MEWS policy includes:

- Direction to nurses as to which doctor needs to be notified based on the score.
- Direction for nurses on the frequency of observation once a trigger score is reached
- Direction on the personnel required for escorting a patient to another clinical area, (e.g. medical imaging) once a trigger is reached

MEWS is beneficial as it:

- Provides a point in time for communicating the changes in vital signs and empowers nurses to take action. It does not replace MET
- Assists doctors in prioritising the management of their patients
- Prompts more timely review and treatment of patients as it has an inbuilt escalation policy if the patient has not been reviewed in the required time frame
MEWS does **NOT** replace calling the Medical Emergency Team (MET). If the patient meets the MET criteria a MET should be called as per the MET protocol.

**Adult MET criteria**
- All respiratory and cardiac arrests
- Threatened airway
- RR <5 or >36
- A new reduction in O₂ sats of <90% despite oxygenation
- Heart rate <40 or >140
- Systolic BP <90 mmHg
- Fall in GCS of more than 2 points
- New onset, prolonged or repeated seizures

---

**Adult MEWS calculation**
To obtain the MEWS each individual observation is scored according to the criteria in table below.

<table>
<thead>
<tr>
<th>MEWS 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>Resp Rate</td>
<td>≤ 8</td>
<td>9-20</td>
<td>21-30</td>
<td>31-35</td>
<td>≥36</td>
<td></td>
<td></td>
</tr>
<tr>
<td>SpO₂</td>
<td>≤ 84</td>
<td>85-89</td>
<td>90-92</td>
<td>≥93</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Temperature</td>
<td>≤ 34</td>
<td>34.1-35</td>
<td>35.1-36</td>
<td>36.1-37.9</td>
<td>38-38.5</td>
<td>≥38.6</td>
<td></td>
</tr>
<tr>
<td>Heart Rate</td>
<td>≤ 40</td>
<td>41-50</td>
<td>51-99</td>
<td>100-110</td>
<td>111-130</td>
<td>&gt;130</td>
<td></td>
</tr>
</tbody>
</table>

**Blood pressure**

<table>
<thead>
<tr>
<th>Sedation Score</th>
<th>0-1</th>
<th>2</th>
<th>3</th>
<th>4</th>
</tr>
</thead>
<tbody>
<tr>
<td>UOP for 4 hrs OR UOP for 24 hours</td>
<td>&lt;80</td>
<td>80-119</td>
<td>120-800</td>
<td>&gt;800</td>
</tr>
<tr>
<td></td>
<td>&lt;480</td>
<td>480-714</td>
<td>720-4800</td>
<td>&gt;4800</td>
</tr>
</tbody>
</table>

**MEWS score**

To obtain the blood pressure score the patient’s usual systolic blood pressure **MUST** be determined in and written on the front of the observation chart. The usual can then be circled on the table on the back of the chart.

The current systolic blood pressure reading is then compared in the table below to establish the blood pressure score. For example a patient with a blood pressure of 140mmHg who has a current reading of 100mmHg would score a 2
The total MEWS is calculated and documented on the new observation chart. If a different chart is being used the MEWS score is still required to be calculated and recorded.

Additional considerations relating to the MEWS for the individual patient can be documented in the space provided at the top of the blood pressure table. An example might be for a dialysis patient, the medical team may decide to exclude the urine output score from the total MEWS. Another example is for a chronic lung disease patient the medical team may only want to trigger a score for an oxygen saturation of less than 90%. These alterations need to be documented by the medical team, and are aimed at preventing false readings for observations that are normal for that patient.

**Track and Trigger Procedures**

If the total MEWS reaches an initial trigger point of 4 the activation protocol is to be initiated.

**MEWS ≥ 4**
- Increase the frequency of observations
- Notify medical staff
- Patient requires and escort

**Increase Frequency of Observation**

If the score is equal to or greater than 4, the frequency of observations is escalated to:
- ½ hourly for the first hour (or more frequently if the patient’s condition dictates).
- then hourly for the next four hours if MEWS is <4
- then 4/24 for the next 24 hours if MEWS is < 4

**Communicate Score Appropriately**

Nurses must notify the CNC/Team leader when a patient meets a trigger score. Nurses must notify the relevant medical officer depending on the MEWS as outlined in the notification and escalation flow chart:
Type of Escort Required Out of Ward

If the trigger score is reached, the following guide for who should accompany the patient is to be used if the patient requires escort out of the ward area.

- MEWS ≥ 4 Registered Nurse
- MEWS ≥ 6 Registered Nurse & Intern
- MEWS ≥ 8 Registered Nurse & Registrar

Adult Summary

- Trigger Score: A total MEWS score of 4 or more is the trigger point for action, with escalated notification at 6 and 8 or if the patient is not improving.
- MEWS does NOT replace calling the Medical Emergency Team (MET). If the patient meets the MET criteria a code blue/MET should be called if there is no documentation describing otherwise.

- MEWS ≥ 4
  - Increase the frequency of observations
  - Notify medical staff
  - Patient requires an escort

- At the time of reaching a trigger score the nurse MUST always notify the team leader or CNC/NUM.
Paediatric Warning Scores

Learning Objectives
- To recognise that paediatric patients deteriorate more rapidly than adults
- Be able to calculate an early warning score using the Children’s Early Warning Tool (CEWT).
- To be aware of your responsibilities when a trigger score is met.

Introduction
The Children’s Early Warning Tool (CEWT), like the adult MEWS, is a bedside tool that is calculated by nursing staff to indicate early signs of a patient’s deterioration.

The CEWT tool is a multi parameter system which incorporates many of the same features as the MEWS chart as well as paediatric specific observations such as capillary refill and respiratory distress.

The following chapters will highlight, where applicable, the differences in the physiology, assessment and management of the paediatric patient.

The CEWT policy includes:
- A flow chart to direct nurses regarding who needs to be notified and at what point
- A structure for increasing the frequency of observations once a trigger score is reached
- A guide to what escort is required if the patient is to be transferred to another clinical area e.g. medical imaging

Age Specific Observation Charts
The CEWT system has 4 age related observation charts:
- <1 year
- 1 – 4 years
- 5 – 11 years
- > 12 years

MEWS does NOT replace calling the Medical Emergency Team (MET). If the patient meets the MET criteria a MET should be called as per the MET protocol.

Paediatric MET criteria
- All respiratory and cardiac arrests
- Threatened airway, cyanosis or grunting
- RR:
  - <1 yr <10 or >55
  - 1 – 4 yr <5 or >50
  - 5 – 11 yr <5 or >45
  - > 12 yr <5 or >40
- HR & BP:
  - <1 yr <60 or >190 SBP <50 mmHg
  - 1 – 4 yr <60 or >170 SBP <55 mmHg
  - 5 – 11 yr <60 or >170 SBP <55 mmHg
  - > 12 yr <50 or >150 SBP <70 mmHg
- Lethargy, extreme agitation, sudden loss of consciousness
- Unexpected, repeated or prolonged seizures
- Any patient a nurse, doctor or parent is worried about that does not fit the above criteria
- If managing a premature infant the corrected age should be utilised up to the age of 12 months.
- The CEWT charts also include a section to complete for pain management

**Paediatric Early Warning Score Calculation**
To obtain a total CEWT score each individual observation is scored according to the criteria in the tables below.
Blood pressures should be recorded as per the local paediatric unit policy however a blood pressure must be recorded on admission and if the CEWT score is $\geq 4$.
A corrected age should be used for premature infants until the age of 12 months

<table>
<thead>
<tr>
<th>Respiratory Rate</th>
<th>&lt; 1 year</th>
<th>1 – 4 years</th>
<th>5 – 11 years</th>
<th>&gt;12 years</th>
</tr>
</thead>
<tbody>
<tr>
<td>56 – 60</td>
<td>MET</td>
<td>MET</td>
<td>MET</td>
<td>MET</td>
</tr>
<tr>
<td>51 – 55</td>
<td>2</td>
<td>MET</td>
<td>MET</td>
<td>MET</td>
</tr>
<tr>
<td>46 – 50</td>
<td>1</td>
<td>2</td>
<td>MET</td>
<td>MET</td>
</tr>
<tr>
<td>41 – 45</td>
<td>0</td>
<td>2</td>
<td>2</td>
<td>MET</td>
</tr>
<tr>
<td>36 – 40</td>
<td>0</td>
<td>1</td>
<td>1</td>
<td>3</td>
</tr>
<tr>
<td>31 – 35</td>
<td>0</td>
<td>0</td>
<td>1</td>
<td>2</td>
</tr>
<tr>
<td>26 – 30</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>1</td>
</tr>
<tr>
<td>21 – 25</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>16 – 20</td>
<td>2</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>11 – 15</td>
<td>3</td>
<td>2</td>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td>6 – 10</td>
<td>MET</td>
<td>3</td>
<td>2</td>
<td>2</td>
</tr>
<tr>
<td>0 - 5</td>
<td>MET</td>
<td>MET</td>
<td>MET</td>
<td>MET</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Respiratory Distress</th>
<th>&lt; 1 year</th>
<th>1 – 4 years</th>
<th>5 – 11 years</th>
<th>&gt;12 years</th>
</tr>
</thead>
<tbody>
<tr>
<td>Severe</td>
<td>3</td>
<td>3</td>
<td>3</td>
<td>3</td>
</tr>
<tr>
<td>Moderate</td>
<td>2</td>
<td>2</td>
<td>2</td>
<td>2</td>
</tr>
<tr>
<td>Mild</td>
<td>1</td>
<td>1</td>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td>Nil</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>O2 l/min</th>
<th>&lt; 1 year</th>
<th>1 – 4 years</th>
<th>5 – 11 years</th>
<th>&gt;12 years</th>
</tr>
</thead>
<tbody>
<tr>
<td>$\geq 15$</td>
<td>3</td>
<td>3</td>
<td>3</td>
<td>3</td>
</tr>
<tr>
<td>11 - 14</td>
<td>2</td>
<td>2</td>
<td>2</td>
<td>2</td>
</tr>
<tr>
<td>2 - 10</td>
<td>1</td>
<td>1</td>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td>&lt; 2</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>O2 Saturations (%)</th>
<th>&lt; 1 year</th>
<th>1 – 4 years</th>
<th>5 – 11 years</th>
<th>&gt;12 years</th>
</tr>
</thead>
<tbody>
<tr>
<td>$&gt; 93$</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>90 - 93</td>
<td>1</td>
<td>1</td>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td>85 – 89</td>
<td>2</td>
<td>2</td>
<td>2</td>
<td>2</td>
</tr>
<tr>
<td>$&lt; 85$</td>
<td>3</td>
<td>3</td>
<td>3</td>
<td>3</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Temperature (C)</th>
<th>&lt; 1 year</th>
<th>1 – 4 years</th>
<th>5 – 11 years</th>
<th>&gt;12 years</th>
</tr>
</thead>
<tbody>
<tr>
<td>$&gt; 40.5$</td>
<td>2</td>
<td>2</td>
<td>2</td>
<td>2</td>
</tr>
<tr>
<td>40.1 – 40.5</td>
<td>2</td>
<td>2</td>
<td>2</td>
<td>2</td>
</tr>
<tr>
<td>39.6 – 40</td>
<td>2</td>
<td>2</td>
<td>2</td>
<td>2</td>
</tr>
<tr>
<td>39.1 – 39.5</td>
<td>2</td>
<td>2</td>
<td>2</td>
<td>2</td>
</tr>
<tr>
<td>38.6 – 39</td>
<td>1</td>
<td>1</td>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td>Heart Rate (bpm)</td>
<td>&lt; 1 year</td>
<td>1 – 4 years</td>
<td>5 – 11 years</td>
<td>&gt; 12 years</td>
</tr>
<tr>
<td>-----------------</td>
<td>----------</td>
<td>-------------</td>
<td>--------------</td>
<td>-----------</td>
</tr>
<tr>
<td>&gt;190 MET</td>
<td>MET</td>
<td>MET</td>
<td>MET</td>
<td>MET</td>
</tr>
<tr>
<td>181 - 190</td>
<td>2</td>
<td>MET</td>
<td>MET</td>
<td>MET</td>
</tr>
<tr>
<td>171 – 180</td>
<td>2</td>
<td>MET</td>
<td>MET</td>
<td>MET</td>
</tr>
<tr>
<td>161 – 170</td>
<td>1</td>
<td>2</td>
<td>2</td>
<td>MET</td>
</tr>
<tr>
<td>151 – 160</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>MET</td>
</tr>
<tr>
<td>141 – 150</td>
<td>0</td>
<td>1</td>
<td>1</td>
<td>2</td>
</tr>
<tr>
<td>131 – 140</td>
<td>0</td>
<td>0</td>
<td>1</td>
<td>2</td>
</tr>
<tr>
<td>121 – 130</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>1</td>
</tr>
<tr>
<td>111 – 120</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>101 – 110</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>91 – 100</td>
<td>1</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>81 – 90</td>
<td>2</td>
<td>1</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>71 – 80</td>
<td>3</td>
<td>2</td>
<td>1</td>
<td>0</td>
</tr>
<tr>
<td>61 – 70</td>
<td>3</td>
<td>3</td>
<td>2</td>
<td>0</td>
</tr>
<tr>
<td>51 – 60</td>
<td>MET</td>
<td>MET</td>
<td>MET</td>
<td>1</td>
</tr>
<tr>
<td>&lt; 50</td>
<td>MET</td>
<td>MET</td>
<td>MET</td>
<td>MET</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Systolic Blood Pressure (mmHg)</th>
<th>&lt; 1 year</th>
<th>1 – 4 years</th>
<th>5 – 11 years</th>
<th>&gt; 12 years</th>
</tr>
</thead>
<tbody>
<tr>
<td>&gt;150</td>
<td>2</td>
<td>2</td>
<td>2</td>
<td>2</td>
</tr>
<tr>
<td>146 – 150</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>141 – 145</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>136 - 140</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>131 – 135</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>126 – 130</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>121 – 125</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>116 – 120</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>111 – 115</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>105 – 110</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>101 – 105</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>96 – 100</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>91 – 95</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>1</td>
</tr>
<tr>
<td>86 – 90</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>1</td>
</tr>
<tr>
<td>81 – 85</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>1</td>
</tr>
<tr>
<td>76 – 80</td>
<td>0</td>
<td>1</td>
<td>1</td>
<td>3</td>
</tr>
<tr>
<td>71 – 75</td>
<td>1</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>66 – 70</td>
<td>1</td>
<td>2</td>
<td>2</td>
<td>MET</td>
</tr>
<tr>
<td>61 – 65</td>
<td>2</td>
<td>3</td>
<td>3</td>
<td>MET</td>
</tr>
<tr>
<td>56 – 60</td>
<td>2</td>
<td>3</td>
<td>3</td>
<td>MET</td>
</tr>
<tr>
<td>50 – 55</td>
<td>3</td>
<td>MET</td>
<td>MET</td>
<td>MET</td>
</tr>
<tr>
<td>&lt; 50</td>
<td>MET</td>
<td>MET</td>
<td>MET</td>
<td>MET</td>
</tr>
</tbody>
</table>
### Capillary Refill Time

<table>
<thead>
<tr>
<th></th>
<th>&lt; 1 year</th>
<th>1 – 4 years</th>
<th>5 – 11 years</th>
<th>&gt;12 years</th>
</tr>
</thead>
<tbody>
<tr>
<td>&gt; 2 sec</td>
<td>1</td>
<td>1</td>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td>≤ 2 sec</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
</tbody>
</table>

### Level of Consciousness

<table>
<thead>
<tr>
<th></th>
<th>&lt; 1 year</th>
<th>1 – 4 years</th>
<th>5 – 11 years</th>
<th>&gt;12 years</th>
</tr>
</thead>
<tbody>
<tr>
<td>Alert</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Verbal</td>
<td>1</td>
<td>1</td>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td>Pain</td>
<td>3</td>
<td>3</td>
<td>3</td>
<td>3</td>
</tr>
<tr>
<td>Unresponsive</td>
<td>MET</td>
<td>MET</td>
<td>MET</td>
<td>MET</td>
</tr>
</tbody>
</table>

### Track and Trigger Procedures

If the total CEWT reaches an initial trigger point of \(\geq 4\) the activation protocol is to be initiated.

**MEWS \(\geq 4\)**
- Increase the frequency of observations
- Notify medical staff
- Patient requires and escort

**Increase Frequency of Observation**

If the score is \(\geq 4\), the frequency of observations is escalated to:
- \(\frac{1}{2}\) hourly for the first hour (or more frequently if the patient’s condition dictates).
- then hourly for the next four hours if CEWT is \(<4\)
- then 4/24 for the next 24 hours if CEWT is \(<4\)

**Communicate Score Appropriately**

Nurses must notify the CNC/Team leader when a patient meets a trigger score. Nurses must notify the relevant medical officer depending on the CEWT score as outlined in the notification and escalation flow chart:

---

**CEWT 4 – 5**
- Contact RMO to review patient within 30 min
- Contact registrar to review the patient within 30 minutes
- If patient not reviewed within 30 minutes
- Contact consultant for review

**CEWT 6 - 7**
- Contact registrar & RMO to review the patient within 15 minutes
- Notify consultant of patient's condition
- If patient not reviewed within 30 minutes or clinically concerned
- Call MET call

**CEWT \(\geq 8\)**
- Register to attend urgently
- Notify the consultant of patient's condition and ask for urgent review
- Call MET call
Type of Escort Required Out of Ward
If the trigger score is reached, the following guide for who should accompany the patient is to be used if the patient requires escort out of the ward area.

- MEWS ≥ 4 Registered Nurse
- MEWS ≥ 6 Registered Nurse & Intern/JHO
- MEWS ≥ 8 Registered Nurse & Registrar

Paediatric Summary

- A CEWT score of ≥ 4 is the point at which the escalation policy takes effect, with further escalations at a score of 6 and 8.
- CEWT does not replace the calling of a MET. If the patient meets MET criteria call a MET.
- CEWT score ≥ 4
  - Increase the frequency of observations
  - Notify medical staff
  - Escort required if travelling outside of ward
  - Notify the team leader or CNC
Oxygen Delivery
Oxygen Delivery

Learning Objectives:
- To understand the importance of oxygen delivery at the tissue level
- To understand the factors that affect adequate oxygen delivery

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

Aerobic Metabolism (i.e. with oxygen)

\[
\begin{align*}
1 & \text{Glucose} \\
\text{CYTOPLASM} \\
\text{Glucose-6-Phosphate} \\
\downarrow \\
2 & \text{Pyruvate} \\
\text{Mitochondrial membrane} \\
\text{pyruvate dehydrogenas} \\
\text{3Pyruvate} \quad \text{Acetyl CoA} + 2\text{ATP} \\
\text{MITOCHONDRIA} \\
\text{4Citrate, TCA cycle/Kreb’s} \\
\text{NADH} \quad \text{NAD}^+ \\
\text{+} \\
\text{H} + \text{O}_2 \quad \text{H}_2\text{O} \\
\text{ADP} + \text{P}_i \quad 36 \text{ATP}
\end{align*}
\]

Aerobic Metabolism (i.e. with oxygen)
If there is inadequate oxygen supply, ATP production falls, and cellular function is then depressed, through lack of energy. This can lead to organs failing and may result in unplanned admissions to ICU or death.

Oxygen supply to the cells can be described by the “oxygen delivery chain”

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

Thus oxygen delivery requires

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

B. Cardiac output

**Summary**

- Oxygen is essential for the adequate production of adenosine triphosphate (ATP)
- If there is inadequate oxygen supply, ATP production falls and cellular function is then depressed
- Oxygen delivery = Cardiac Output × Arterial Oxygen Content
Airway and Breathing
Airway and Breathing

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

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

Airway

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

Causes of Airway Obstruction
- Functional airway obstruction - may result from decreased level of consciousness. Whereby the muscles relax and allow the tongue to fall back and obstruct the pharynx.
- Mechanical airway obstruction - may be through aspiration of a foreign body or swelling/bleeding in the upper airway (e.g. trauma, allergy, and infection). It may also be caused by oedema or spasm of the larynx.

Examination of the airway
Recognition of airway obstruction is possible using the “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. With incomplete obstruction, breathing will be noisy and breath sounds reduced (stridor = inspiratory wheeze)
- Feel: placing your hand immediately in front of the patient’s mouth allows you to feel if there is any air moving

Management of the obstructed airway
In the majority of cases in hospital, airway obstruction is functional, i.e. due to depressed level of consciousness. Simple manoeuvres may be required to open the airway:
- Head tilt / Chin lift
- Jaw thrust
- Insertion of an oropharyngeal (Guedel’s airway) or nasopharyngeal airway
Suctioning of the airway using a yankauer sucker may be required to remove any vomitus or secretions, which could be contributing to airway obstruction. If the patient continues to have a depressed level of consciousness and is unable to protect their own airway endotracheal intubation may be required, which needs to be performed by experienced staff.

In all patients with an airway obstruction or if they are unable to maintain an adequate airway, a MET should be called. In rare cases 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 and a Code Blue/MET should be called.

A surgical airway may be required if intubation is not possible (cricothyroidotomy). This should only be attempted by experienced staff.

**BREATHING**

Breathing is required to move adequate oxygen in and carbon dioxide out of the lungs. Breathing requires
- An intact respiratory centre in the brain
- Intact nervous pathways from brain to diaphragm & intercostal muscles
- Adequate diaphragmatic & intercostal 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. When you walk into a room and the first thing that you notice is the patient’s breathing, there is a significant problem with the patient.

Look for signs of inadequate ventilation:
- Use of accessory muscles
- Sweating/pallor
• Central cyanosis
• Abdominal breathing
• Shallow breathing
• Unequal chest movement

**Listen**
Initially listen at the “end of the bed”, for:
• Noisy breathing, which may indicate secretions in the upper airway
• Stridor or wheeze which may indicate partial airway obstruction

Then auscultate with a stethoscope to assess
• Quiet or absent breath sounds may indicate the presence of a pneumothorax or a pleural fluid
• Bronchial breathing may indicate the presence of consolidation

**Feel**
Palpate the trachea and chest wall:
• Tracheal deviation indicates a mediastinal shift, which may be due to:
  - a pneumothorax or pleural fluid – tracheal deviation away from the side of the lesion
  - lung collapse - tracheal deviation toward the side of the lesion
• Chest wall crepitus (subcutaneous emphysema) is highly suggestive of a pneumothorax or oesophageal / bronchial rupture
• Asymmetrical chest wall movement may indicate unilateral pathology

Percuss the chest wall
• Hyper-resonance indicates pneumothorax
• Dullness indicates consolidation or pleural fluid

**Why is respiratory rate important?**
An increased respiratory rate can reflect either a drop in arterial saturation 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 of the deteriorating patient. As oxygen delivery to the tissues is reduced, cells revert to anaerobic metabolism. This increases lactate production, resulting in build up of acid. The accumulation of lactic acid stimulates an increase in respiratory rate (tachypnoea).

---

**Inadequate oxygen delivery at the tissue level**

1. Anaerobic metabolism
2. Lactate production
3. Metabolic acidosis
4. Stimulates respiratory drive
5. Increases the respiratory drive

---

Metabolic Acidosis can increase the respiratory rate even though the arterial oxygenation saturation may be normal
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.

**A normal arterial saturation and tachypnoea**
There can be falling oxygen delivery despite normal arterial oxygen saturation. Increased respiratory rates can occur in patients with normal or low arterial oxygen saturation and may well be a better indicator of a deteriorating patient than arterial oxygen saturation.

**Respiratory rate SpO2 MEWS and MET**
The respiratory rate and arterial oxygen saturations score for the Adult MEWS are as follows:

<table>
<thead>
<tr>
<th>MEWS</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>Resp Rate</td>
<td>&lt; 8</td>
<td>9 - 20</td>
<td>21 - 30</td>
<td>21 - 35</td>
<td>&gt;36</td>
<td></td>
<td></td>
</tr>
<tr>
<td>SpO2 %</td>
<td>&lt; 84</td>
<td>85 - 89</td>
<td>90 - 92</td>
<td>&gt;93</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Adult respiratory MEWS

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

**MET criteria RR <5 or >36**

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

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

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

In a small subgroup of patients who have Chronic Obstructive Pulmonary Disease (COPD) and are “CO2 retainer” or patients who have risk factors for hypercapnic respiratory failure (e.g. morbid obesity, chest wall deformities or neuromuscular disorders), high concentrations of oxygen can be disadvantageous by suppressing their hypoxic drive. However, these patients will also suffer end-organ damage or cardiac arrest if their blood oxygen levels fall too low. The aim in these patients is to achieve PaO2 of 60mmHg, or saturation of 90% on pulse oximetry. So in a patient with COPD who has a pCO2 >60mmHg but is also hypoxic, pO2 <60mmHg, do not turn the inhaled O2 down, however do not leave the patient unattended. If their pO2 is >60 mmHg, then you can turn the inhaled O2 down to maintain SaO2 > 90%

**Oxygen Delivery Systems**
The oxygen delivery systems available are classified into fixed and variable performance devices which are capable of delivering a wide range of oxygen concentrations.
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 e.g. the Venturi mask.

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


<table>
<thead>
<tr>
<th>Diluter Colour</th>
<th>Inspired oxygen (diluter setting)</th>
<th>Suggested oxygen flow rate (L/min)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Blue</td>
<td>24%</td>
<td>3</td>
</tr>
<tr>
<td>White</td>
<td>28%</td>
<td>6</td>
</tr>
<tr>
<td>Orange</td>
<td>31%</td>
<td>8</td>
</tr>
<tr>
<td>Yellow</td>
<td>35%</td>
<td>10</td>
</tr>
<tr>
<td>Red</td>
<td>40%</td>
<td>12</td>
</tr>
</tbody>
</table>

Relationships between inspired oxygen and oxygen flow rate with Venturi masks

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

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

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

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

Hudson facemask – reservoir volume of oxygen is increased above that achieved by the nasopharynx thus higher oxygen concentration can be achieved in inspired gas (max 50-60%).
**Non-rebreathing mask** – simple facemask with the addition of a reservoir bag with one or two-way valves over the exhalation ports which prevent exhaled gas entering the reservoir bag (permits inspired oxygen concentration up to 90%). The mask requires an oxygen flow rate of 12-15 L/min.

**Non-rebreather 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 (ABG). The advantage of measuring arterial blood gases is that they measure oxygen, carbon dioxide levels and metabolic status (including lactate).

Oxygen needs to be prescribed by a medical officer. If oxygen is applied or the amount increased, a medical officer must review the patient.

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 pO₂ 60mmHg.

Patients do not die from raised CO₂ alone: they die from hypoxaemia.

In an acute setting, when taking an arterial blood gas sample, do not remove the oxygen. It is unnecessary, and may precipitate sudden deterioration.
As long as the concentration of oxygen being delivered is recorded, the degree of hypoxaemia can be calculated using the alveolar-air equation and A-a gradient. The blood gas machine will calculate this for you as long as the correct inspired oxygen concentration is recorded.

\[
P_aO_2 = F_iO_2 - \frac{PaCO_2}{0.8}
\]

\(P_aO_2\) should be close to \(PaO_2\) in normal lungs

\(A = \text{alveolar} \quad a = \text{arterial} \quad (A-a = \text{age}/3)\)

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

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

If the pulse oximeter does not give a reading, do not assume it is broken ... the patient may have poor perfusion.

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

Oxygen saturation may be “normal” but the \(pCO_2\) may be high which reflects inadequate minute ventilation and hence respiratory failure.

Arterial oxygen saturation being “normal” does not rule out acute respiratory failure.

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

ABG’s should be measured in patients who:

- Are critically ill
- Have deteriorating oxygen saturations or increasing respiratory rate
- Require significantly increased supplemental oxygen to maintain oxygen saturation
- Have risk factors for hypercapnic respiratory failure who deteriorate
- Have poor peripheral circulation and therefore unreliable peripheral measurements of oxygen saturation
**ADULT SUMMARY**

- An increase in respiratory rate will 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 so easily treated, e.g. airway swelling, post-operative haematoma, infection. This is a medical emergency. A Code Blue/MET should be called.
- In a small subgroup of patients who have Chronic Obstructive Pulmonary Disease (COPD) and are “CO₂ retainers”, high concentrations of inspired oxygen can be disadvantageous by suppressing their hypoxic drive. However, these patients will also suffer end-organ damage or cardiac arrest if their blood oxygen levels fall too low. The aim in these patients is to achieve PaO₂ of 60mmHg, or saturation of 90% on pulse oximetry. So in a patient with COPD who has a pCO₂ > 60mmHg but is also hypoxic, pO₂ < 60mmHg, do **not** turn the inhaled O₂ down. If their pO₂ is ≥ 60 mmHg, then you can turn the inhaled O₂ down to maintain SaO₂ ≥ 90%.
- Oxygen flow rates less than 6L/min for Hudson’s mask should not be used due to carbon dioxide retention in the mask.
- When taking an arterial blood gases, **do not remove oxygen mask**. It is unnecessary, and may precipitate sudden deterioration.
- If the pulse oximeter does not give a reading, do not assume it is broken...the patient may have poor perfusion!
- 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!
- MET criteria RR< 5 or > 36
Paediatric Airway Differences

The paediatric airway differs from the adult airway in several important ways.

1. Anatomy
   - The tongue is relatively large compared to the oral cavity
   - The larynx is relatively high and anterior in position compared to the adult airway
   - The epiglottis is “U” shaped and protrudes into the pharynx
   - The trachea is short and soft and can become compressed if the neck is hyper extended
   - The narrowest section of the airway in children under the age of 8 years is at the level of the cricoid cartilage
   - The vocal cords are short and concave
   - The diaphragm plays a more significant role in the generation of tidal volume in infants and young children as the more compliant chest wall and horizontal ribs contribute less to chest expansion than in the older child and adult.
   - The lower airways are smaller and so are at greater risk from being obstructed by mucous, oedema or active constriction
   - Minor decreases in diameter of the smaller airways creates a dramatic increase in the resistance to airflow

2. Physiology
   - Infants have a relatively higher metabolic rate and oxygen consumption which accounts for their higher respiratory rates. This also means that the infant and child are at higher risk of hypoxaemia in times of stress.
   - Chest wall compliance leads to the development of prominent recession in the child with reduced lung compliance

Breathing
Breathing is required to move adequate oxygen in and carbon dioxide out of the lungs. Breathing requires
   - An intact respiratory centre in the brain
   - Intact nervous system from the brain to the diaphragm and intercostal muscles
   - Adequate diaphragmatic and intercostal muscle function
   - Unobstructed airflow (large and small airways)

Recognition of potential respiratory failure
A rapid assessment of an infant or child’s respiratory function is an important step in determining whether there may be inadequate oxygen delivery to the cells.
The degree of increase in the effort and efficacy of breathing allows the clinician to assess the severity of respiratory compromise.

**Assess response** – An unresponsive child will require immediate life support interventions to be undertaken

**Airway**
- Assess for the child’s ability to vocalise. This suggests that the child has at least some airway patency
- Assess airway patency by looking for:
  - Chest and/or abdominal movement
  - Symmetry
  - Recession
- Listen for:
  - Breath sounds and other airway noises
- Feeling for:
  - Expired air, chest expansion

**Breathing**

**Effort of breathing**
- Respiratory rate – increased respiratory rate at rest indicates a need for increased ventilation in response to either lung or airway disease, or metabolic acidosis. A slow respiratory rate indicates fatigue, cerebral depression or a pre terminal state.
- Recession – indicates increased effort of breathing.
- Inspiratory or expiratory noise – inspiratory noise (stridor) indicates either laryngeal or tracheal obstruction; wheezing indicates lower airway narrowing. Beware the quite chest as the volume of the noise is not an indication of severity of obstruction.
- Grunting – occurs in an effort to generate “positive end expiratory pressure” at the end of expiration
- Accessory muscle use
- Flaring of nares
- Gasping

It is important to note that breathlessness and cyanosis in infants and children despite oxygenation may be a sign of serious cardiac pathology. Other signs and symptoms which may suggest a cardiac cause for respiratory inadequacy may include: a tachycardia which is out of proportion with respiratory difficulty; raised JVP, an enlarged liver or cardiac murmur.

**Efficacy of Breathing**
- Observation of the degree of chest expansion
- Auscultation of the chest – listen for reduced, asymmetrical or bronchial breath sounds.
- Pulse oximetry
- Effects of decreased oxygenation on other organs – tachycardia initially with children becoming bradycardic when severely hypoxic; pallor or cyanosis; confusion, agitation or drowsiness.

**Management of the paediatric patient in respiratory distress**
Specific management will depend on the cause of respiratory distress.
It is important to identify and manage life threatening conditions immediately e.g. foreign body inhalation, severe asthma, epiglottis. It is important to note that progressive respiratory compromise which results in acidosis and hypoxia can lead to cardiac arrest in children.

If the airway is not patent an airway opening manoeuvre should be used. If required a formal airway should be secured is further airway management is required with an LMA or intubation.

All deteriorating infants and children should receive high flow supplemental oxygen via a face mask with an appropriate flow rate to achieve and oxygen saturation of > 95%.

If the child is hypo ventilating then respiration should be supported with oxygen and bag-valve mask ventilation.

There are a small group of children who have an arterial oxygen saturation range that is normal for them but far below the normal range for a well child (chronic lung disease, cystic fibrosis, heart defects). In this case the CEWT criteria may be changed to reflect the “normal” for these children. This must be decided by a senior medical officer and documented in the modifications section on the CEWT observation chart.

**Paediatric Oxygen Delivery Systems**

There are many oxygen delivery systems used in paediatrics that are age and/or size specific.

**Fixed Performance**

Devices such as venturi masks are used only occasionally in paediatrics.

**Variable performance**

Devices such as nasal prongs, simple face masks, partial re-breather and non re-breather masks come in different sizes to suit the size of the child.

Paediatric Hudson masks have a minimum flow rate of 4L/min

There are three types of nasal prongs used in paediatrics:

- Micro flow nasal prongs – can be used for neonates and premature infants. These can deliver oxygen flow rates of up to 0.1 L/min
- Low flow nasal prongs – can deliver oxygen flow rates of < 2L/min. Infants are often placed on flows form 0.1 – 1.0L/min via a low flow meter. Rates of 1 – 2L/min can be delivered via a regular flow meter.
- High flow nasal prongs – use higher flow rates of oxygen which increases the amount of air remaining in the lungs at end expiration, preventing closure of the alveoli and stabilising the airway. These systems can be run with oxygen flow rates of 2 – 8L/min.

This type of therapy is generally suitable for patients with:

- bronchiolitis (usually up to 3 years of age)
- bronchial asthma (usually up to 6 years of age)
- other conditions deemed suitable by the consulting paediatrician

Once the child requires 2L/min or more oxygen flow it is recommended to change to warmed humidified oxygen. This helps improve tolerance by decreasing discomfort and irritation. Humidification helps loosen secretions and improve mucociliary transport; prevent nasal obstruction from hard dry secretions and reduces discomfort and irritation. The type of humidification device selected will depend on the oxygen delivery system in use and the patient’s oxygen requirements. Cold dry air may increase heat and fluid loss in infants.

**Paediatric Summary**

Children’s metabolic rates are higher than those of adults leading to a higher rate of oxygen consumption. This means that further increases in metabolic rate can result in hypoxaemia. It is important to note that breathlessness and cyanosis which does not improve with oxygenation may be a sign of serious cardiac pathology.

Any alteration to the CEWT parameters should be decided by a senior medical officer and documented in the modifications section on the CEWT observation chart.
Circulation

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

The Importance of Oxygen
Oxygen reaching the cells and mitochondria is dependent upon adequate amounts of oxygen being delivery. 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

\[
\text{Oxygen delivery} = \frac{\text{Cardiac output} \times \text{Arterial Oxygen Content}}{\text{Stroke Volume} \times \text{HR} \times \text{Hb} \times \text{SaO}_2}
\]

Blood Pressure

Blood Pressure, Pulse and Oxygen Delivery
Blood pressure is the product of cardiac output and total peripheral resistance (TPR).

\[
\text{Blood Pressure} = \frac{\text{Cardiac Output} \times \text{Peripheral Vascular Resistance}}{\text{Stroke Volume} \times \text{HR} \times \text{Hb} \times \text{SaO}_2}
\]

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

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

Blood pressure and maintenance of organ function

- There are some organs that also 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.
- The body’s organs adapt over time to a person’s “normal” blood pressure. If blood pressure is always elevated, e.g. chronic hypertension; the brain and kidneys adapt and will require a greater blood pressure in order to function normally. Therefore it is important to know what your patient’s “normal” or “usual” blood pressure was prior to their current illness. There
Calculation of adult MEWS for heart rate:

<table>
<thead>
<tr>
<th>MEWS</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 (Beats per minute)</td>
<td>&lt; 40</td>
<td>41 - 50</td>
<td>51 - 99</td>
<td>100 - 110</td>
<td>111 - 130</td>
<td>&gt; 130</td>
<td></td>
</tr>
</tbody>
</table>

A heart rate of less than 40 beats per minute or greater than 140 beats per minute requires a MET call.

Calculation of adult MEWS for blood pressure:

<table>
<thead>
<tr>
<th>Usual SBP</th>
<th>190</th>
<th>180</th>
<th>170</th>
<th>160</th>
<th>150</th>
<th>140</th>
<th>130</th>
<th>120</th>
<th>110</th>
<th>100</th>
<th>90</th>
<th>80</th>
</tr>
</thead>
<tbody>
<tr>
<td>Current Blood Pressure</td>
<td>200s</td>
<td>0</td>
<td>0</td>
<td>1</td>
<td>1</td>
<td>2</td>
<td>2</td>
<td>2</td>
<td>3</td>
<td>3</td>
<td>4</td>
<td>5</td>
</tr>
<tr>
<td></td>
<td>190s</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>1</td>
<td>1</td>
<td>2</td>
<td>2</td>
<td>2</td>
<td>3</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td></td>
<td>180s</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>1</td>
<td>1</td>
<td>2</td>
<td>2</td>
<td>3</td>
<td>3</td>
</tr>
<tr>
<td></td>
<td>170s</td>
<td>1</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>1</td>
<td>1</td>
<td>2</td>
<td>2</td>
<td>3</td>
<td>3</td>
</tr>
<tr>
<td></td>
<td>160s</td>
<td>1</td>
<td>1</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>1</td>
<td>1</td>
<td>2</td>
<td>2</td>
</tr>
<tr>
<td></td>
<td>150s</td>
<td>1</td>
<td>1</td>
<td>1</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>1</td>
<td>1</td>
<td>2</td>
</tr>
<tr>
<td></td>
<td>140s</td>
<td>2</td>
<td>1</td>
<td>1</td>
<td>1</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>1</td>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td></td>
<td>130s</td>
<td>2</td>
<td>2</td>
<td>1</td>
<td>1</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td></td>
<td>120s</td>
<td>2</td>
<td>2</td>
<td>2</td>
<td>1</td>
<td>1</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td></td>
<td>110s</td>
<td>3</td>
<td>2</td>
<td>2</td>
<td>2</td>
<td>1</td>
<td>1</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td></td>
<td>100s</td>
<td>3</td>
<td>3</td>
<td>3</td>
<td>2</td>
<td>2</td>
<td>1</td>
<td>1</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td></td>
<td>90s</td>
<td>4</td>
<td>3</td>
<td>3</td>
<td>3</td>
<td>2</td>
<td>2</td>
<td>2</td>
<td>1</td>
<td>1</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td></td>
<td>80s</td>
<td>MET</td>
<td>MET</td>
<td>MET</td>
<td>1</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>70s</td>
<td>MET</td>
<td>MET</td>
<td>MET</td>
<td>1</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
</tbody>
</table>

Definition of Hypotension

The generally acceptable definition of hypotension is:
- Systolic blood pressure of less than 100mmHg OR
- A drop of more than 20% from “usual” blood pressure

It is important to remember that someone who is normally hypertensive may be relatively hypotensive even when their systolic blood pressure is above 100mmHg.

Do not always use 100mmHg as your CRITICAL Systolic Blood Pressure cut off

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

- A fall in cardiac output **OR**
- A fall in peripheral vascular resistance.

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

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

**Factors affecting stroke volume:**
1. **Contractility**
   - The ability of the heart to contract in the absence of any changes in preload or afterload – it reflects the strength

   **Major negative influences include:**
   - Myocardial ischemia
   - Acidosis
   - Drugs (beta-blockers, anti-dysrhythmic)

   **Major positive influences 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 increases 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:

   **Intravascular blood volume**
   - **Absolute:**
     - A decrease in intravascular blood volume (bleeding, electrolyte and water loss), water loss (diabetes insipidus) will cause a decrease in stroke volume
   - **Relative**
     - There is no loss of intravascular blood volume but with vasodilatation and pooling of blood (vasodilators, epidurals, sepsis) will cause a decrease in venous return to the heart and hence a decrease in stroke volume.

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

   **Intrathoracic pressure**
   - Increases in intrathoracic pressure (asthma, positive pressure ventilation) will restrict the amount of blood returning to the heart and therefore reduce stroke volume
3. **After-load**
   This is the resistance to ejection of blood from the ventricle. This resistance can either be caused by an outflow resistance from the heart (aortic stenosis) or resistance to flow in the systemic circulation. This resistance is determined by the diameter of the arterioles and per-capillary sphincters. As resistance rises, stroke volume is reduced.

---

**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. vagal response, parasympathomimetics
- **sympathetic stimulation**: INCREASES the heart rate via the sympathetic cardiac fibres e.g.: stress response, temperature, sympathomemetics

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

Any changes in heart rate can change the cardiac output. A faster heart rate can increase the cardiac output and this often occurs when the stroke volume is falling and any reductions 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 where an increase in heart rate may reduce the cardiac output. If the ventricle does not have adequate time to fill with blood there is a reduction in the end diastolic volume and therefore stroke volume. Cardiac output reduces as a result and may cause a drop in blood pressure. A good example is atrial fibrillation with a rapid ventricular response.

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

Sometimes when the heart slows there may be no reduction in cardiac output. As the ventricle has a longer time to fill the end diastolic volume is increased with each beat. This stretches the myocardial fibres and increases the stroke volume per beat which may then compensates 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 is a very healthy athlete.

---

**Fall in cardiac output**

- Fall in stroke volume
  - Decreased contractility
  - Decreased preload
  - Increased afterload
- Fall in heart rate
- Fall in peripheral vascular resistance (PVR)

**Peripheral Vascular Resistance**

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

1. **Increase in peripheral vascular resistance**
Autonomic Nervous System

- **Stimulation of Sympathetic Receptors**: Sympathetic stimulation (α₁) of the arterioles can cause vasoconstriction and a subsequent increase in blood pressure. This often occurs in response to a fall in blood pressure, which is detected by baroreceptors situated in the carotid sinus and aortic arch. In response to this reduction in pressure these baroreceptors send fewer inhibiting messages to the vasomotor centre which in turn sends more messages through the sympathetic nerves to the vessels causing constriction. e.g. Sympathomimetics that stimulate the α₁ receptor will cause vasoconstriction of arteriole, examples include noradrenaline, adrenaline.

- **Direct action on arteriole smooth muscle**: examples include metaraminol, vasopressin, angiotensin and methylene blue

2. Decrease in peripheral vascular resistance

- **Blockade of Autonomic Sympathetic Nervous System**
  Anything that causes a reduction in the sympathetic stimulation of the arterioles will result in vasodilatation, reducing vascular resistance and blood pressure.
  Influences include:
  - increasing the stimulation of the baroreceptors from a rise in blood pressure, which causes a reduction in the sympathetic outflow causing vasodilatation
  - any drug that blocks the sympathetic nervous system can cause vasodilatation and a fall in blood pressure e.g. α2 agonists – clonidine

- **Direct action on arteriole smooth muscle**
  Molecules and drugs can have a direct effect on the vascular smooth muscle in arteriole causing vasodilatation
  Examples include:
  - Vasodilating Drugs: calcium channel blockers, ACE inhibitors
  - Vasodilating Molecules: nitric oxide (infection/sepsis)
  - Vasodilating conditions: acidosis, increases in temperature

Compensatory Mechanisms for Hypotension

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

**Causes**

1. **Reduction in Cardiac Output (CO=SV×HR)**
   **Reduction in Stroke Volume**
   - There will be a compensatory increase in heart rate (tachycardia) and a compensatory increase in peripheral vascular resistance (cool, blue peripheries).
   - While this compensation can return BP to normal values. If CO has not been restored, there may be evidence of persistent inadequate oxygen delivery.

**Clinical Features of a reduction in Stroke Volume:**

- **Reduction in Pre-Load**: Hypotension with a postural drop, tachycardia and cool, blue peripheries
- **Reduction in Contractility**: Hypotension, tachycardia and cool, blue peripheries with signs of heart failure
Reduction in heart rate
- There will be a compensatory increase in total peripheral vascular resistance to try and maintain blood pressure

2. Reduction in peripheral vascular resistance
There will be a compensatory increase in cardiac output. Cardiac output will increase by increasing heart rate (tachycardia) and increasing the contractility of the heart to improve stroke volume. Cardiac output may be temporarily supported by infusion of intravenous fluids to increase circulating volume.

Clinical Features of a fall in Peripheral Vascular Resistance: Hypotension, tachycardia and warm peripheries

Consequences of Hypotension

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.

\[ \text{DO}_2 = \text{cardiac output} \times \text{arterial oxygen content} \]
\[ \text{Blood Pressure} = \text{cardiac output} \times \text{peripheral vascular resistance} \]

The following are possible reasons why hypotension may represent shock:

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.

Inadequate Pressure Gradient
- Clearly without a pressure gradient across the vasculature (from high pressure to low pressure) there can be no flow of blood and its constituents including oxygen.
- Some organs are able to maintain blood flow through organs despite changes in blood pressure (autoregulation) eg: brain and kidney. However, there reaches a point when this can no longer occur and this in turn reduces blood flow and hence the amount of oxygen reaching the tissues.
- Inadequate blood flow to the organs results in inadequate oxygen delivery to the organs resulting in reduced generation of ATP and the formation of lactate. This will lead to organ failure (oliguria and altered mentation), lactate formation and shock.

When is hypotension not shock?
In order to demonstrate that there is shock there needs to be evidence that organs are failing and/or that there is evidence of anaerobic respiration by the presence of lactate.

For example: If a patient is hypotensive post anaesthetic and has warm hands (suggesting good flow to the hands i.e. good cardiac output), is not confused, has a good urine output with no signs of heart or respiratory failure and no lactate is found, then the patient is currently not shocked. However, in these situations it is important to continue regular monitoring of the vital signs and continually look out for evidence of organ failure.
Can a patient with normal or high blood pressure have shock?
The key components to adequate oxygen getting to the tissues are cardiac output and arterial oxygen content. If either of these two are reduced can this result in a fall in oxygen getting to the tissues and this results in shock. Sometimes, the compensatory mechanisms associated with a drop in cardiac output, such as increase in total peripheral resistance, can result in there being a normal or even high blood pressure measurement. So, despite there being a “normal” blood pressure, there are signs of organ failure and anaerobic respiration i.e. the patient is shocked with a seemingly normal blood pressure.

For example: An elderly lady presents with an inferior myocardial infarction and complete heart block. On examination she has navy blue fingers, a heart rate of 40 beats per minute, her blood pressure is 210/100 mmHg and she has evidence of pulmonary oedema and oliguria. Her lactate measurement is 10mmol/L (normal < 2 mmol/L). So, despite a high blood pressure due to an enormous increase in vascular tone to try and compensate for the fall in cardiac output and blood pressure, there is evidence of not only organ failure but also anaerobic respiration. This patient IS shocked despite the high blood pressure.

The Initial Management of Hypotension
It is important to remember what generates a blood pressure:
- Cardiac Output (stroke volume x heart rate)
- Peripheral Vascular Resistance

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

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

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

- In the presence of tachycardia, but absence of organ failure and lactate formation
  Important improve venous return and stroke volume to maintain adequate cardiac output and oxygen delivery to the tissues:
  - Administer intravenous fluid bolus (500 – 1000ml of 0.9% Saline)
  - Continue to perform frequent vital signs to document any trends (e.g. 1/2hrly for 1 hr, followed by hourly for 4 hours, then subsequently 4-hourly if stable)
- If there is an improvement in tachycardia and blood pressure, then the fluid bolus has been adequate to restore venous return.
- If the tachycardia remains repeat the fluid challenge
- Continue to observe response
- If the patient continues to have hypotension, tachycardia and warm hands, further fluid can be administered particularly if there are no signs of heart failure
- An intensive care review should be requested once three litres of fluid have been administered and the tachycardia and hypotension are still present.

- **Hypotension and evidence of organ failure**
  - Administer intravenous fluid bolus (500 -1000ml 0.9% Saline)
  - Continue to perform frequent vital signs to document any trends (1/2hrly x 1 hr, 1/24 x 4, then 4/24 if stable)
  - If there is an improvement in tachycardia and blood pressure, then the fluid bolus has been adequate to restore venous return.
  - If the tachycardia, hypotension and organ failure remains, repeat the fluid challenge
  - Call for an intensive care review
  - A maximum of three litres of fluid should be administered before an intensive care review.
  - Continue to perform hourly observations to ensure that the trend of blood pressure, pulse and mental state are being monitored.

**Fall in Cardiac Output**
There are two predominant common causes of fall in cardiac output, both having very different presentations:

1. **Fall in Pre-Load**
   - Common causes include bleeding, loss of fluids and electrolytes.
   - **History**
     - Will describe histories relevant to bleeding, loss of fluid and electrolytes [diarrhoea, vomiting, 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, maelena, nasogastric losses)
     - Cool, blue hands, tachycardia, hypotension with a postural drop (drops more than 10mmHg from lying to sitting)
   - **Laboratory Investigations**
     - Evidence of bleeding (fall in haemoglobin)
     - Evidence of renal dysfunction (rising creatinine)
     - Evidence of lactate formation (metabolic acidosis on arterial blood gas sampling [negative base excess], lactate rising)
   - **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 rapidly administer 500 – 1000mls 0.9% Saline via a blood pump set through a large bore cannula
- Observe response (tachycardia should be reduced and blood pressure increase)
- Continue to administer fluid rapidly until there is the desired response:
  - Blood pressure returning to normal
  - Heart rate returning to normal
  - Improvement in organ function, particularly urine output
  - Intensive care should be alerted if there are no signs of improvement despite administering 3L fluid
  - Continue to perform regular observations (hourly or as directed by MEWS protocol)

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

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

Adult Summary
   - Blood Pressure = Cardiac Output x Peripheral Vascular Resistance
   - Hypotension:
     - High pulse and low blood pressure may reflect low oxygen delivery
     - It is important to remember that someone who is normally hypertensive may be relatively hypotensive even when their systolic blood pressure is above 100mmHg.
     - Hypotension can be a marker of a deteriorating patient who is at greater risk of death. A “shocked” patient has signs of organ failure which may or may not accompany hypotension.
   - Decreases in cardiac output can be caused by:
     - Decreased intravascular volume
     - Increased intrathoracic pressure
- Increased peripheral vascular resistance
- The greatest concern is that hypotension may suggest that there is an inadequate amount of oxygen getting to the tissues, which is described as SHOCK.
- Management of hypotension in adults
  - Hypotension and warm hands: Administer fluids
  - Hypotension, cool hands, no signs of heart failure: Administer fluids
  - Hypotension, cold hands, signs of heart failure: Cease fluids, refer to CCU/ICU for inotropes
- **Remember to incorporate all the vital signs in your assessment!**
- **A SYSTOLIC BLOOD PRESSURE OF LESS THAN 90mmHg requires a MET call to be made**

---

**Paediatric Specific Points for Circulation**

The paediatric airway differs from the adult airway in several important ways.

1. **Anatomy**
   - A child’s circulating blood volume is higher in children than in adults (70 – 80 ml/kg) but the overall volume is smaller therefore relatively small blood losses can be critically important

2. **Physiology**
   - In the infant, stroke volume is small and relatively fixed and therefore cardiac output is directly related to heart rate. Clinically this is important as infants responses to fluid challenges may be blunted as stroke volume cannot increase to improve cardiac output. By the age of 2 years the child’s cardiac function and response to fluid is much the same as in an adult.
   - Blood pressure is related to systemic vascular resistance as can be seen to rise as systemic vascular resistance increases over time.

**Recognition of Potential circulatory failure**

**Heart rate**

Heart rates gradually decrease as the child’s age increases. Heart rates will initially increase in the shocked child and the rate, especially in small children, may be extremely high (up to 220 bpm). A slow (<60bpm) or rapidly falling heart rate with accompanying signs of systemic failure is extremely worrying in the deteriorating child as it is a pre terminal sign.

Normal heart rates for children are outlined below.

<table>
<thead>
<tr>
<th>Age (years)</th>
<th>Heart Rate (beats/min)</th>
</tr>
</thead>
<tbody>
<tr>
<td>&lt;1</td>
<td>110 – 160</td>
</tr>
<tr>
<td>1 - 2</td>
<td>100 – 150</td>
</tr>
<tr>
<td>2 – 5</td>
<td>95 – 140</td>
</tr>
<tr>
<td>5 – 12</td>
<td>80 – 120</td>
</tr>
<tr>
<td>&gt;12</td>
<td>60 - 100</td>
</tr>
</tbody>
</table>

**Pulse Volume**

Blood pressure is often maintained in a child until the late stages of shock therefore is not a good indicator of perfusion. To obtain a clearer picture of perfusion both peripheral and central pulses should be palpated and assessed for volume and regularity. Poor pulse volumes provide a good indication of poor perfusion.
**Capillary refill**
Capillary refill in the infant and child should be assessed centrally and over a period of 5 seconds. Capillary refill should be brisk, returning after 2 – 3 seconds. A slow refill time is an indication of poor perfusion and in the presence of other signs of shock is a sign of poor tissue perfusion. In children with pigmented skin, capillary refill may be difficult to assess. The nail bed, palms of the hands or soles of the feet in young children may be useful areas to assess capillary refill time.

**Blood pressure**
Blood pressure in children increases with age with the normal ranges according to age outlined below.

<table>
<thead>
<tr>
<th>Age (years)</th>
<th>Systolic blood pressure (mmHg)</th>
</tr>
</thead>
<tbody>
<tr>
<td>&lt;1</td>
<td>70–90</td>
</tr>
<tr>
<td>1–2</td>
<td>80–95</td>
</tr>
<tr>
<td>2–5</td>
<td>80–100</td>
</tr>
<tr>
<td>5–12</td>
<td>90–110</td>
</tr>
<tr>
<td>&gt;12</td>
<td>100–120</td>
</tr>
</tbody>
</table>

All children over the age of 3 years must have their blood pressure taken, on admission, preoperatively and on return to the ward postoperatively. Children should also have their blood pressures measured:

- If coarctation of the aorta is suspected or if there are signs of poor perfusion (increased heart rate, increased respiratory rate, decreased urine output).
- Any child or adolescent of any age with symptoms of shock, unexplained tachycardia, renal or cardiac disease, diabetes or adrenal disorders, head injury or trauma
- Critically ill children
- If the CEWT score is 3 or greater without the inclusion of a BP

**Correct cuff use**
Use of the correct sized cuff is vitally important in obtaining an accurate blood pressure. The width of the cuff should be no more than 80% the length of the upper arm and the bladder more than 40% of the arm’s circumference

A child’s pain, anxiety, anatomy or condition may hinder blood pressure measurements. Blood pressure readings should be interpreted with this in mind

Hypotension is a very late sign in children due to their excellent cardiovascular compensatory mechanisms.
Hypovolaemia causing circulatory collapse is one major precipitant of cardiac arrest in infants and children and is most likely to occur because of fluid loss from conditions such as gastroenteritis, intussusception or haemorrhage.

The seagull sign is not relevant in the paediatric patient as the heart rate in infants and children may be normally higher than the systolic blood pressure. This remains true until the child reaches adolescence

**Management of circulatory failure**
- All children with signs of inadequate circulation (shock) require the delivery of high flow oxygen irrespective of oxygen saturations.
- Venous or intraosseous access should be established if not already present.
Intraosseous access should be considered if intravenous access is unable to be obtained after 2 attempts. The preferred site for intraosseous access in children is the anterior surface of the tibia 2-3 cm below the tibial tuberosity.

- Immediate infusion with crystalloid or colloid of 20ml/kg should be given to the child in shock. A further bolus of 20ml/kg should be considered if there is not sustained haemodynamic improvement after the first bolus.
- If a third bolus is required – expert help should be sought as intensive care management may be required.

**Paediatric Summary**

- Infants have a relatively fixed stroke volume until the age of 2 years therefore cardiac output is critically dependant on heart rate.
- This fixed stroke volume also means that response to fluid resuscitation may be blunted in the less than 2 year old group as they are unable to increase stroke volume greatly to improve cardiac output.
- Decompensation can occur rapidly following a long period of compensation and if not treated rapidly can lead to death. Hypotension in infants and children is a pre terminal sign.
Central Nervous System & Urine Output
Central Nervous System

Learning Objectives:
- Identify common causes of depressed level of consciousness (LOC)
- Describe how to assess a patient’s level of consciousness
- Describe how to manage a patient with depressed level of consciousness

Introduction

Depressed level of consciousness is a common finding in acute illness. It can occur due to intracranial disease or as a result of systemic insults (table 6).

<table>
<thead>
<tr>
<th>Intracranial disease</th>
<th>Meningitis, encephalitis</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Epilepsy</td>
</tr>
<tr>
<td></td>
<td>Cerebrovascular disease, SAH</td>
</tr>
<tr>
<td></td>
<td>Head injury</td>
</tr>
<tr>
<td></td>
<td>CNS infection</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Systemic conditions</th>
<th>Hypoxia, hypercapnia</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Hypotension, hypo/hyperosmolar</td>
</tr>
<tr>
<td></td>
<td>Hypoglycaemia, hyponatraemia</td>
</tr>
<tr>
<td></td>
<td>Hypo/hyperthermia</td>
</tr>
<tr>
<td></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>

Common causes of decreased level of consciousness

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

Causes of depressed level of consciousness

1. Inadequate oxygen delivery

   Neurones in the central nervous system, like all other cells in the body, are highly dependent on oxygen. Adequate oxygenation allows the formation of large amounts of ATP “energy packets” which are required for all cellular functions. When oxygen supply is inadequate, insufficient ATP is produced, which leads to failure of some cellular functions. This causes the symptoms of confusion or depressed level of consciousness.

   Oxygen supply to the cells in the brain 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.

1. Decreased cardiac output

   - decreased stroke volume
   - decreased heart rate

   (This may be indicated by a decreased blood pressure)
2. Decreased arterial oxygen content
   - decreased haemoglobin
   - decreased arterial saturation
3. 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 substrate for energy production. However, neurones can only use glucose as their substrate for energy production. Therefore if serum glucose levels fall too low, neurones will stop producing ATP and cellular function will be compromised. Thus confusion or depressed level of consciousness could also result from hypoglycaemia.

---

Checking the **BGL** is one of the first things, which should be checked on an unconscious, or fitting patient, whether they are diabetic or not. The BGL should be >3 mmol/L

**Assessment of CNS**

1. **Level of consciousness**
   The sedation score used in the MEWS observation chart grades the degree of drowsiness according to the criteria below and should be done on every patient. This is added to the other vital signs, and the total is calculated to give an overall MEWS.

<table>
<thead>
<tr>
<th>MEWS</th>
<th>3</th>
<th>2</th>
<th>1</th>
<th>0</th>
<th>1</th>
<th>2</th>
<th>3</th>
<th>4</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sedation score</td>
<td></td>
<td></td>
<td></td>
<td>0 - 1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
</tr>
</tbody>
</table>

**Sedation Score:**

0 = Awake & alert
1 = Normally asleep, responds to stimuli
2 = Mild, occasionally drowsy, easy to rouse
3 = Moderate, frequently drowsy easy to rouse but unable to maintain wakeful state
4 = Severe, somnolent, difficult to rouse

Another common method of measuring CNS function is the “Glasgow Coma Scale”. The GCS is not included in the MEWS calculation but may be indicated for specific patients or in specific wards. The GCS is divided into three sections – best motor response, best verbal response, and best eye-opening response.
Patients with GCS <8 will almost certainly require intubation, as they are unable to protect their own airway. Further assistance will be required with anyone who has this level of consciousness. A MET call is required if the patient suddenly drops their conscious level two or more GCS points.

If there is a sudden fall in consciousness, or a fall in GCS >2 a MET call is indicated.

2. **Pupillary Size**

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

   Bilateral pupillary dilatation
   - Sympathetic overactivity, e.g. fear, stress, anxiety, hypoglycaemia
   - Sympathomimetic administration, e.g. administration of adrenaline in an arrest situation
   - Anticholinergic activity e.g. atropine, tricyclics antidepressants

   Bilateral pin point pupils
   - Opioids/opiates
   - Cholinergic drugs , organophosphates
   - Brain stem CVA

   Unequal pupils
   - Previous surgery
   - Prosthetic eye
   - Eye drops
   - Brain lesion, aneurysms, infections
   - Glaucoma

**Management of decreased level of consciousness**

- Check airway and breathing; ensure airway is patent
  - head tilt, jaw thrust
  - give high-flow oxygen
- Guedel’s or nasopharyngeal airway
- Measure blood glucose, and correct if <3mmol/L (administer 30mls of 50% glucose intravenously)
- MET call if patient meets the criteria (GCS fallen >2 points)
- If respiratory rate or arterial oxygen saturation is decreased, may need ventilatory assistance using self-inflating bag and mask
- Ensure intravenous access; 500 ml intravenous fluid bolus may be required if patient is hypotensive
- Reverse any drug-induced CNS depression, e.g. naloxone for opioid overdose
- If the airway is patent, and the patient is breathing, place patient supine in the lateral recovery position

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

**Adult Summary**

- Checking the **BGL** is one of the first things, which should be checked on an unconscious, or fitting patient, whether they are diabetic or not.
- If there is a sudden fall in consciousness, or a fall in GCS >2 a MET call is indicated

**Paediatric Specific Points for Central Nervous System**

In children coma is most commonly caused by a diffuse metabolic problem (95%) rather than a structural lesion (5%). The most common causes include:
- Hypoxic ischemia – following respiratory or cardiac arrest
- Seizure disorders
- Head trauma
- Cerebral infections
- Poisoning
- Metabolic disorders
- Vascular lesions

**Recognition of potential central neurological failure**

**Conscious level**
A rapid assessment of conscious level can be made by utilising the AVPU score.

<table>
<thead>
<tr>
<th>A – Alert</th>
<th>V – responds to Voice</th>
<th>P – responds to Pain</th>
<th>U – unresponsive</th>
</tr>
</thead>
</table>

This can be particularly useful in infants where a traditional coma score is difficult to apply.

When using a full coma score it is important to use a developmentally appropriate tool in infants and children. It can be difficult to assess the early signs of neurological deterioration in the infant/young child. Often the parent can provide valuable information as they know when their child is “just not right”. They may also be able to provide information about other symptoms such as fever, headache or episode of trauma which can help guide diagnosis. The child who does not recognise their parent is significantly compromised.
Posture
Children suffering from serious illness display hypotonic posturing. Stiff posturing such as decorticate and decerebrate is a late sign and a sign of serious neurological dysfunction.

Pupils
As in adults both cerebral lesions and drugs may have effect on pupil size and reactivity. The most important changes which may indicate a serious brain disorder include pupil dilatation, unreactivity and inequality.

Assessment of fontanelle
The anterior fontanelle is useful clinically. Examination of an infant includes palpating the anterior fontanelle. A sunken fontanelle indicates dehydration, whereas a very tense or bulging anterior fontanelle indicates raised intracranial pressure. However, this is not a certain indicator for raised pressure as prolonged crying by the baby may produce the same effect.

Management of central neurological failure
- Consider intubation in order to protect and stabilise the airway in the child with a conscious level assessment of P or U (responding only to pain or unresponsive).
- Measure blood glucose level and administer 2 – 5ml/kg of 10% dextrose intravenously
- Intravenous diazepam or midazolam, PR diazepam or IM midazolam should be given for prolonged or recurrent seizures
• Consider the likely causes of a decreased level of consciousness and manage as symptoms suggest
• If there is evidence of raised intracranial pressure (decreasing consciousness, abnormal posturing and abnormal papillary responses the child should be intubated and strategies implemented to reduce the intracranial pressure.

Urinary Output

At the end of this section you will 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
Poor 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 poor urine output is correctly diagnosed.

Pathophysiology
Normal urine flow requires
• Adequate oxygenation of the kidneys
• Adequate perfusion pressure
• Normal function of kidneys
• No obstruction to urine flow e.g. prostatomegaly, renal calculus, blocked catheter

1. Oxygen delivery
In order to function, renal cells require adequate oxygen delivery, just as all the other cells in the body. Oxygen delivery depends on cardiac output and arterial oxygen content.
If oxygen delivery to the kidney falls, 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.

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

\[
\text{MAP} = \frac{(2 \times \text{Diastolic BP}) + \text{Systolic BP}}{3}
\]
If mean arterial blood pressure falls below the lower limit of autoregulation, renal perfusion pressure will decrease and thus urine output will fall.

<table>
<thead>
<tr>
<th>MEWS</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>Urine for 4 hours (mls)</td>
<td>&lt;80</td>
<td>80 - 119</td>
<td>120 - 800</td>
<td>&gt;800</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>For 24 hours</td>
<td>&lt;480</td>
<td>480 - 719</td>
<td>720 - 4800</td>
<td>&gt;4800</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Management
The cause of decreased urine output needs to be determined.
- Decreased renal blood flow in the face of decreased blood pressure, cardiac output or tissue oxygen delivery
- Obstructed urine flow – needs to be urgently corrected if this is the case, thus it is important to diagnose early

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

Decreased Renal Blood Flow
This can be due to decrease in Cardiac Output, as a result of
- Decreased stroke volume
  - Decreased pre-load
  - Decreased contractility
  - Decreased after-load
- Alteration in heart rate
- Change in peripheral vascular resistance

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

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 500ml of normal saline. Frusemide is not to be given
unless you have ruled out other possible reasons for low urine output, and the patient is clinically fluid overloaded.

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

**Management of post-renal oliguria**

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

- Assess bladder size
- Check catheter patency
- If there is no catheter in-situ, the patient may need one inserted

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

**Adult Summary**

- Adult urine output should be > 0.5 ml/kg/hr i.e. 35ml/hr for a 70 kg person
- 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

**Paediatric differences for Urine Output**

**Physiological differences**

- At birth the total body water is higher than in the adult. This gradually decreases over the first year of life until it reaches the adult level of 60%. Extracellular water content also decreases over the first year of life to the adult level of 20 – 25%.
- Glomerula filtration rate in the newborn is 25% of that of an adult. This gradually increases over time until it reaches adult levels at around at the age of 2 years. Despite this infants are able to handle large fluid loads due to their low concentrating capacity.
- Urine concentrating capacity is lees than in the adult patient; this means that infants have less capacity to concentrate their urine in response to dehydration.

**Urine output in children should be maintained at 1 – 3 ml/kg/hr**

**Management of paediatric urinary output**

It is important to maximise renal perfusion and maintain renal tubular patency by maintaining adequate urinary output. This may be maximised by providing:

- Adequate oxygenation
- Maintaining good circulation through fluid resuscitation and use of inotropic support
- Careful use of diuretics to maintain a urinary output of at least 1ml/kg/hr
- Monitoring and normalising electrolyte levels and acid base levels in both the blood and urine.
Communication, Team Work and Management Plans
Communication, Teamwork and Management Plans

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

One of the most important factors in determining an acutely ill patient’s outcome is the quality of communication among the clinicians involved. In each team, each member has their strengths and weaknesses, varying skills and different levels of knowledge. The aim of managing the deteriorating patient is to determine the role of each member of the team, identifying their comfort zones and working together in this knowledge to affect the best outcome for the management of the particular patient. The steps for optimising the management are outlined below

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

1. Gathering Information

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

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 event to a medical officer who has reviewed a patient for the first time. This may significantly alter the doctor’s interpretation of what is wrong with the patient in the acute setting. The team physio may have noticed that a patient’s exercise tolerance or arterial oxygen saturations on exercise have significantly deteriorated. This may alert the team to lower respiratory tract infections or perhaps even pulmonary emboli. This should be communicated and documented in the notes.

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

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

2. Integration of the information

The next step is to integrate the information they have gathered to fully understand the current situation of the patient e.g. the need to understand why a BP has fallen or why a heart rate or respiratory rate has risen.

3. Communicating Information

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

The important thing is to recognise when there is an abnormality in vital signs and make sure someone more senior knows about it and that someone is attending the patient appropriately.

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

This means health professionals need to:
- Identify that there is a problem
- Attempt to interpret the problem in the context of the patient we are caring for
- Communicate the trigger to the appropriate people for further actioning.
“ISBAR” Communication
The Identify, Situation, Background, Assessment and Recommendation (ISBAR) technique is an easy, structured and useful tool to help communicate concerns and call for help or action.

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

**Situation:** What is the current situation, concerns, observations, MEWS?

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

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

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

For example:
A 75-year-old lady with a history of IHD is admitted with a fractured neck of femur. Twelve hours post-operatively, she complains of chest pain and her arterial oxygen saturation has fallen to 88% on 2L nasal prongs and a MEWS score of 4. You as the person nursing her are concerned that she is unwell and needs attention. The SBAR 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 MEWS score of 4 due to her dropping her oxygen saturations to 88% on 2L, tachycardia and tachypnoea. She is also complaining of chest pain”

**BACKGROUND**
“She is twelve hours post-op for a fractured neck of femur and she has a history of ischaemic heart disease”

**ASSESSMENT**
“I think she is acutely unwell and may have………”

**RECOMMENDATION**
“I think this patient requires an urgent medical review. I have increased her inspired oxygen to 15l/min via a non re-breather mask.”

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

In the worst case scenario if you are not getting the attention that you think this patient deserves or you are not sure what to do next, then you can end with – “This a significant change in the medical status of the patient and this patient needs a medical review urgently.”
**Documentation**

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

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

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

**When communicating information you must:**

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

**4. Adequate response to information/concerns**

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

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

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

*For example:*

*A nurse is told to take the blood pressure in the left and right arm for a person with central chest pain. However, this particular patient may have a fistula or have had a mastectomy and cannot have bilateral blood pressure measurements performed. The doctor comes back to review all the collected information and review the management plan only to find out this has not been done. If the doctor had specifically asked if the nurse had any problems with the plan, the issue would have been identified much earlier, saving everyone time and allowing the appropriate observations to be measured in a timely fashion.*
The primary responsibility of the doctor is to stabilise the patient. However, the needs of the ward and the nursing staff need to be integrated into this plan. The nursing staff may feel that the patient cannot be managed in a general ward because of level of nursing care required, but the doctor feels that there is no medical reason that they need step up of their care. This needs to be discussed and a plan endorsed by all members, and agreed on.

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

Use the SBAR strategy when communicating at a MET call as well

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

5. Formulating, Documenting and Communicating Management Plans

The make or break of patient care is often in the formulation of management plans. To allow successful flow of information from one team, one shift and one ward to the next, plans MUST be documented. They must be thorough, yet concise and most importantly understandable, both legible and logical.

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

- **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 which has fallen from 150/90 to 98/50 may need the frequency of their observations changed so that vital signs are done every half an hour until the blood pressure is above a certain level and stable without intervention.

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

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

- **Change in Therapy Orders**
  This may include changing antibiotics from oral to intravenous, or adding a diuretic.
• **Investigation/intervention orders**
  If it has been decided that the patient needs their electrolytes checked then this must be documented, as well as whose responsibility it is to check the results. It is often useful to write what is expected and what to do about abnormal results if this is predictable. You may now realise that the patient requires IV access for antibiotics that have been ordered.

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

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

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

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

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

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

When documenting a medical entry always document:
- H-history
- E-examination
- I-impression/diagnosis
- P-management plan

Management Plans should include:
- Observation orders
- Nursing orders
- Allied health orders
- Change in therapy orders
- Investigation/intervention orders
- Notification orders
SUMMARY

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


Eather, B. (2006). *St.George Hospital Modified Early Warning Score policy*.

Greater Ormond Street Hospital – Children’s Early Warning Score


