



compass

'Pointing you in the right direction'

ADULT/PAEDIATRIC/MATERNITY

Dr. Bronwyn Avar
Ms. Heather McKay
Ms. Nicole Slater
Dr. Paul Lamberth
Dr. Kathryn Daveson

Dr. Tony Lafferty
Mr. Jon Darvil
Ms. Sue Chen
Ms. Raelene Garret-Rumba
Dr. Imogen Mitchell

COMPASS © 2016

ISBN 978-0-9750076-1-7

All forms of copyright in relation to the manual and application are held by the Australian Capital Territory.

For enquiries about the COMPASS course or copies of the manual contact:

Early Recognition of the Deteriorating Patient Program Manager

ACT Health

compass@act.gov.au

+ 61 2 62076827

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 November 2006; Second edition September 2010;

Third edition May 2014; Fourth edition July 2016

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.

Application design, layout, and publication by Publishing Services,
ACT Government.

CONTENTS

Introduction	1
Modified Paediatric/Maternity Early Warning Scores	5
Oxygen Delivery	25
Airway and Breathing	31
Circulation	55
Central Nervous System and Urine Output	85
Communication, Teamwork, Management Plans	103
References	119
Quiz Questions	123



Introduction

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

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

Paediatric Patients



This symbol will indicate the important differences between adult and paediatric patients in the presented material.

Maternity Patients



This symbol will indicate the important differences between adult, paediatric and maternity patients in the presented material. The Maternity specific information will be after the Paediatric specific information.

Preamble

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

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

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

a. Absent or inaccurate observations

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

b. Inability to understand observations recorded

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

c. Failure to trigger timely appropriate response

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

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

An example of what can go wrong

A 60 yr old man was admitted with pancreatitis.

- On day 3 of his stay, his systolic blood pressure fell into the 80s, and whilst fulfilling the Medical Emergency Team (MET) criteria no code blue was called. Over the next 24 hours the patient filled Code Blue/ MET criteria thirteen times, still with no activation of MET. The patient died 14 hours later.
- The system issues identified were:
 - failure to follow hospital policy in calling a MET

- inadequate documentation of observations, particularly respiratory rate
- failure to change the management plan despite its inadequacy
- failure to escalate the level of medical review despite the seriousness of the situation.

The Australian Commission on Safety and Quality in Healthcare has developed National Safety and Quality Health Service Standards. Standard 9 - Recognising and Responding to Clinical Deterioration in Acute Health Care, outlines clear requirements for acute healthcare settings in regards to a minimum standard for documenting vital signs, education and escalation systems to assist clinicians in recognising and responding to clinical deterioration. This education package, in conjunction with a clearly formatted observation chart and the utilisation of a track and trigger system, aims to prevent missed cases of deterioration and guide clinicians in recognising and responding to clinical deterioration.

Our Aim

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

Our Objectives

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

How it Works

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

- The application and manual to be worked through independently
- An online quiz
- A 3-hour face-to-face session.

With COMPASS online learning you will be guided through a case study.

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

At the completion of the **online learning** application there is a short multiple choice quiz. You will be unable to skip ahead, however you will be able to go back to any area that you have already completed if you choose to. The application **MUST** be completed prior to coming to the

face-to-face session you have been scheduled for. You must also go to an internet terminal and access an online quiz. This again is multiple choice and **MUST** be completed prior to the final session. Your results will be available to the program coordinator.

OK LET'S GET STARTED!



Modified/Paediatric/Maternity Early Warning Scores

Early Warning Scores

Learning Objectives

- Be able to calculate a Early Warning Score
- Be aware of your responsibilities when a trigger score (see text box 1) is met
- Understand how to complete the track and trigger observation chart.

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

If derangements in respiratory rate, heart rate 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

An Early Warning Score¹ (EWS) is a multiparameter 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, particularly in acute wards where patients are often quite unwell and where there may be many inexperienced staff. Classic vital signs include Heart Rate, Blood Pressure, Temperature, and Respiratory Rate; however the EWS takes into account other observations as well.

The EWS 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.

Trigger Score:

A score of ≥ 4 is a trigger point for action, with escalated notification at 6 and 8.

Text box 1: Trigger Score

The Modified Early Warning Score (MEWS) policy includes:

- direction for nurses regarding who needs to be notified and at what point
- direction for nurses on the frequency of observation measurement 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 observations and empowers nurses and junior doctors to take action. It does not replace MET (text box 2)
- assists doctors in prioritising the management of their patients
- prompts more timely medical review and treatment of patients as it has an inbuilt escalation policy if the patient has not been reviewed in the required time frame.

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

Adult MET Criteria:

- All respiratory & cardiac arrests
- Threatened Airway, RR < 5 or > 36 breaths per minute (bpm)
- Pulse < 40 or > 140 bpm
- Systolic BP < 90 mmHg
- Sudden fall in level of consciousness, fall of GCS > 2, repeated or prolonged seizures
- Any patient you are seriously worried about that does not fit the above criteria.

Text box 2: Medical Emergency Team Criteria

² Reproduced with permission of The Australian Society of Anaesthetists

Adult MEWS Calculation

To obtain the total MEWS each individual observation (table 1) is scored according to the criteria in table below.

MEWS	4	3	2	1	0	1	2	3	4
Resp Rate per min	≤4	5-8			9-20	21-24	25-30	31-35	≥ 36
Oxygen saturation	≤84	85-89	90-92	93-94	95-100				
Temperature		≤ 34	34.1-35	35.1-36	36.1-37.9	38-38.5	≥ 38.6		
Heart Rate	<40			40-49	50-99	100-109	110-129	130-139	≥140
Sedation score					Awake		Mild	Mod	Severe

Table 1: Adult Modified Early Warning Score for each variable

² Reproduced with permission from Taylor & Francis Ltd

To obtain the blood pressure score for adults the patient's usual systolic blood pressure **MUST** be determined and written on the front of the observation chart. The usual BP can then be circled on the BP table on the chart.

The current systolic blood pressure reading is then compared in the table to establish the blood pressure score.

					USUAL BP >	190	180	170	160	150	140	130	120	110	100	90	80	CURRENT SYSTOLIC BP
> < Blood Pressure & Heart Rate (+)	200 & more				200 & more	0	0	1	1	2	2	2	3	3	4	5	5	
	190-199				190-199	0	0	0	1	1	2	2	2	3	3	4	4	
	180-189				180-189	0	0	0	0	0	1	1	2	2	3	3	4	
	170-179				170-179	1	0	0	0	0	1	1	2	2	3	3	3	
	160-169				160-169	1	1	0	0	0	0	1	1	1	2	2	2	
	150-159				150-159	1	1	1	0	0	0	0	0	1	1	2	2	
	140-149				140-149	2	1	1	1	0	0	0	0	0	1	1	1	
	130-139				130-139	2	2	1	1	0	0	0	0	0	0	0	1	
	120-129				120-129	2	2	2	1	1	1	0	0	0	0	0	0	
	110-119				110-119	3	2	2	2	1	1	0	0	0	0	0	0	
	100-109				100-109	3	2	2	2	1	2	1	1	0	0	0	0	
	90-99				90-99	4	3	3	3	2	2	2	2	1	1	0	0	
	80-89				80-89											1	0	
	70-79				70-79													
	60-69				60-69													
	50-59				50-59													
	40-49				40-49													
	30-39				30-39													
	20-29				20-29													

Figure 1: Scoring a MEWS for BP using Usual BP.

The total MEWS is calculated and documented on the observation chart. If another chart is being used the MEWS is still required to be calculated and recorded.

Variance

Additional considerations relating to the MEWS for the individual patient can be charted as a variance. An example would 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 falsely high readings for what is considered usual for that patient.

A variance should only be used for chronic conditions and not for acute conditions, as the physiological changes in these cases are indications of failure of oxygen delivery and compensation. To ignore review in these cases would be detrimental to the patient.

Track and Trigger Procedures

If the total MEWS reaches a trigger point of 4 or more, the activation protocol (text box 3) is to be initiated:

A. Increase Frequency of Observations

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.

B. Communicate Score Appropriately

Nurses must notify the Clinical Nurse Consultant (CNC/CMC)/ Nurse/Midwife Unit Manager (NUM) when a patient meets a trigger score (text box 3). Nurses must notify the relevant medical officer depending on the MEWS as outlined in the notification and escalation flow chart (figure 2).

MEWS ≥ 4

- **Increase the frequency of observations**
- **Notify nursing/midwife team leader**
- **Patient requires an escort**
- **Notify the correct level medical officer to review patient.**

Text box 3: Activation protocol

If the patient is not improving escalation should also occur.

MEWS	Notify	Escalate	Observation	Intra-hospital escort
MEWS 4-5	<ul style="list-style-type: none"> • Team Leader • RMO to review within 30 mins 	After 60 mins. If nil review or improvement. Escalation per MEWS 6-7.	Vital signs: ½ hourly for 1 hour Commence fluid balance chart If patient improves, decrease vital sign frequency to: <ul style="list-style-type: none"> • Hourly 4 hours • 4 hourly for 24 hours 	RN/RM
MEWS 6-7	<ul style="list-style-type: none"> • Team Leader • RMO to review within 30 mins 	After 60 mins. If nil review or improvement. Escalation per MEWS ≥ 8		RN/RM & RMO
MEWS ≥ 8	<ul style="list-style-type: none"> • Team Leader • Contact Registrar to review immediately • Contact Consultant 	Consider MET if nil review or improvement.		RN/RM & REG

Figure 2: Adult notification flowchart

C. Type of Escort Required Out of Ward (Adult)

The following guide determines which personnel should accompany the patient if a patient is required to go to another clinical area such as the medical imaging department.

MEWS \geq 4 Registered Nurse /Midwife

MEWS \geq 6 Registered Nurse/Midwife & Resident Medical Officer

MEWS \geq 8 Registered Nurse/Midwife & Registrar

Adult Summary

- Trigger Score: A total MEWS of 4 or more is the trigger point for action, with escalated notification at \geq 6 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 unless there is a documented and clear management plan in place that indicates appropriate/alternate actions for these events.
- MEWS \geq 4
 - Increase the frequency of observations
 - At the time of a patient reaching a trigger score of four or more the nurse/midwife MUST always notify the team leader or CNC/NUM.
 - Notify medical staff
 - Patient requires an escort.



PAEDIATRIC EARLY WARNING SCORES

Learning Objectives

- Be able to recognise that children deteriorate more rapidly than adults
- Be able to calculate a Paediatric Early Warning Score
- To be aware of responsibilities when a trigger score is met (text box 4)
- Be able to complete the age appropriate observation chart.

Introduction

The Paediatric Early Warning Score (PEWS) like the adult MEWS is a bedside score that is calculated by nursing staff to indicate early signs of a patient's deterioration.

The PEWS looks at all the observations together, not just a single observation in isolation. It includes respiratory rate and effort, oxygen use and saturations, heart rate, blood pressure, and level of consciousness.

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

Trigger Score:

A score of 4 or more is a trigger for action, with escalated notification at ≥ 6 and ≥ 8 .

Text box 4: Trigger Score Paediatrics

The PEWS procedure includes:

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

To obtain the PEWS, each individual observation is scored on the appropriate chart for age according to the criteria in the tables below. Use the age specific observation charts available.

Age Specific Observation Charts

Use corrected age for premature babies up until the age of 12 months:

- < 3 months observation chart
- 3 - < 12 months observation chart
- 1 – 4 years observation chart
- 5 – 11 years observation chart
- 12 to 17 years observation chart.

Additional observations relating to the PEWS are documented on the observation chart. These observations include: Capillary refill, BGL, and Pain Score.



The following tables outline an example of a PEWS. The Paediatric MET criteria is age specific and is included in the PEWS tables.

Resp Rate	< 3 mths	3 - < 12 mths	1 – 4 yrs	5 – 11 yrs	12 – 17 yrs
≥81	4/MET	4/MET	4/MET	4/MET	4/MET
75-80	3	4/MET	4/MET	4/MET	4/MET
70-74	3	4/MET	4/MET	4/MET	4/MET
65-69	2	4/MET	4/MET	4/MET	4/MET
60-64	1	4/MET	4/MET	4/MET	4/MET
55-59	0	3	4/MET	4/MET	4/MET
50-54	0	2	4/MET	4/MET	4/MET
45-49	0	1	3	4/MET	4/MET
40-44	0	0	2	3	4/MET
35-39	0	0	1	2	4/MET
30-34	0	0	0	1	2
25-29	1	0	0	0	1
20-24	4/MET	1	0	0	1
15-19	4/MET	2	1	1	0
10-14	4/MET	4/MET	2	2	0
5-9	4/MET	4/MET	4/MET	4/MET	3
0-4	4/MET	4/MET	4/MET	4/MET	4/MET

Table 2: PEWS for respiratory rate.

¹³ Reproduced with permission of Greater Ormond Street Hospital (GOSH)



Heart Rate	< 3 mths	3- < 12 mths	1 – 4 yrs	5-11 yrs	12- 17 yrs
≥190	4/MET	4/MET	4/MET	4/MET	4/MET
180-190	3	4/MET	4/MET	4/MET	4/MET
170-179	2	3	4/MET	4/MET	4/MET
160-169	1	2	3	4/MET	4/MET
150-159	0	1	2	3	4/MET
140-149	0	0	1	2	4/MET
130-139	0	0	0	1	3
120-129	0	0	0	0	2
110-119	0	0	0	0	2
100-109	0	0	0	0	1
90-99	0	0	0	0	0
80-89	1	1	1	0	0
70-79	2	2	2	1	0
60-69	3	3	3	2	1
50-59	4/MET	4/MET	4/MET	4/MET	2
< 50	4/MET	4/MET	4/MET	4/MET	4/MET

Table 3: PEWS for Heart Rate.

¹³ Reproduced with permission of GOSH



Systolic Blood Pressure	< 3 Months	3 - < 12 Months	1 – 4 Years	5 – 11 Years	12 – 17 Years
>150	4/MET	4/MET	4/MET	4/MET	3
145-150	4/MET	4/MET	4/MET	3	3
140-144	4/MET	4/MET	3	3	3
135-139	4/MET	4/MET	3	3	2
130-134	4/MET	2	2	3	2
125-129	4/MET	2	2	2	1
120-124	3	2	2	1	0
115-119	3	1	1	0	0
110-114	2	1	1	0	0
105-109	2	0	0	0	0
100-104	1	0	0	0	0
95-99	0	0	0	0	0
90-94	0	0	0	0	0
85-89	0	0	0	1	3
80-84	0	0	0	2	4/MET
75-79	0	0	2	4/MET	4/MET
70-74	1	2	4/MET	4/MET	4/MET
65-69	2	4/MET	4/MET	4/MET	4/MET
60-64	3	4/MET	4/MET	4/MET	4/MET
55-59	4/MET	4/MET	4/MET	4/MET	4/MET
50-54	4/MET	4/MET	4/MET	4/MET	4/MET
<45-49	4/MET	4/MET	4/MET	4/MET	4/MET

Table 4: PEWS for Blood Pressure.

¹³ Reproduced with permission of GOSH



Oxygen delivery			
PEWS	0	1	2
< 12 Years	Room Air Nasal Prongs Hudson Mask 4L/min	HFNP ≤ 1.5 L/kg or HFNP with $\text{FiO}_2 \leq 40\%$ or Hudson Mask $>4\text{L}$	HFNP ≥ 1.6 L/kg or HFNP with $\text{FiO}_2 > 40\%$
12-17 Years	Room Air Nasal Prongs Hudson Mask 6L/min	HFNP ≤ 1.5 L/kg or HFNP with $\text{FiO}_2 \leq 40\%$ or Hudson Mask $>6\text{L}$	HFNP ≥ 1.6 L/kg or HFNP with $\text{FiO}_2 > 40\%$
High Care Charts			CPAP

Table 5: PEWS for oxygen delivery.

¹³ Reproduced with permission of GOSH



Oxygen saturation %	0	1	2	3	4
	≥95	90-94	87-89	85-86	≤ 84

Table 6: PEWS for oxygen saturation.

¹³ Reproduced with permission of GOSH



Temperature C	0	1	2	3
< 3 months	36.5-37.4	36- 36.4, 37.5-37.9	< 35.5-35.9	≥ 38.0
≥3 months	36.1-37.9	≤ 36 38-39	≥ 39.1	

Table 7: PEWS for temperature.

¹³ Reproduced with permission of GOSH

Effort of breathing:

- normal = nil APLS criteria
- mild = presence of 1 APLS criteria
- moderate = presence of 2 APLS criteria
- severe = presence of 3 or more APLS criteria.

APLS criteria for effort of breathing: ¹⁴, used with permission of APLS

- stridor
- accessory muscle use
- recession
- wheeze
- nasal flaring
- grunting
- gasping.

Level of Consciousness (AVPU):

- A – Alert
- V – Voice (responds to voice)
- P – Pain (responds to pain)
- U – Unresponsive.

Blood pressure is to be recorded as per existing paediatric policy and scored for PEWS. Blood pressure should also be recorded if the PEWS is 6 or greater based on the other parameters.

For patients with a PEWS ≥ 4

- Review by CNC or Team Leader
- Frequency of vital signs
 - ½ hourly for the first hour (more frequently if the patient's condition dictates). If the patient's condition improves they may then progress to:
 - 1/24 for the next four hours
 - 4/24 for the next 24 hours.

- Escort off ward area

Consideration needs to be given to the requirement for off ward procedures and discussion initiated with treating team consultant. If the patient requires movement to another clinical area, the following escort should accompany the patient.

- PEWS ≥ 4 Registered Nurse/Midwife
- PEWS ≥ 6 Registered Nurse/Midwife
- PEWS ≥ 8 Registered Nurse/Midwife.

PEWS Escalation Table			
PEWS	Notify	Escalate	Intra hospital escort
PEWS 4-5	Team Leader. RMO review within 30 minutes	After 60 minutes if no review and/or no improvement escalate per PEWS 6-7	RN
PEWS 6-7	Team Leader. Registrar review within 30 minutes	After 60 minutes if no review and/or no improvement escalate per PEWS ≥ 8	RN and RMO
PEWS ≥ 8	Team Leader. Registrar review immediately. Contact Consultant	Consider MET if no review and/or no improvement	RN and Registrar
Alteration to calling criteria → Patient meeting urgent review criteria → Registrar review within 15 minutes		Consider MET if no review and/or no improvement	RN and Registrar
<u>Vital sign frequency and actions for PEWS ≥ 4:</u> → ½ hourly for 1 hour → Commence fluid balance chart → If PEWS ≥ 6 BP must be measured with each set of vital signs If patient improves decrease frequency of vital signs to: → Hourly for 4 hours → 4 hourly for 24 hours			

Figure 3: An example of a PEWS notification flowchart

**PEWS does NOT replace calling the Medical Emergency Team (MET).
If the patient meets the MET criteria a Code Blue/MET should be
called as per MET protocol.**



PAEDIATRIC SUMMARY

- Trigger Score: A score of 4 or more is a point for action, with further escalation of the level of review at a score of ≥ 6 or if the patient is not improving.
- PEWS does NOT replace calling the Medical Emergency Team (MET). If the patient meets the MET criteria a paediatric or neonatal code blue/MET should be called.
- PEWS ≥ 4 :
 - Increase the frequency of observations
 - At the time of reaching a trigger score the nurse MUST always notify the team leader or CNC
 - Notify medical staff
 - Patient requires an escort for transport off the ward.

In the next few sections you will be taken through each observation individually. Always remember though, that you must look at all the observations when assessing a patient and not just a single parameter in isolation.



MATERNITY MODIFIED WARNING SCORES

Learning Objectives

- Identify the normal physiological changes in pregnancy and therefore facilitate the recognition of the abnormal
- Demonstrate awareness of when it is appropriate and necessary to take observations on antenatal and postnatal women
- Demonstrate how to calculate a Maternity Modified Early Warning Score (Maternity MEWS)
- Demonstrate understanding of responsibilities when a trigger score is reached.

The following chapters will discuss the differences in physiology due to pregnancy, and the assessment and management of the pregnant or postnatal woman.

Introduction

The Maternity Modified Early Warning Score, like the MEWS, is a clinical tool which provides a score which is calculated by staff to indicate the early signs of a pregnant woman's clinical deterioration.

The Maternity MEWS looks at a complete set of observations, not just one observation in isolation. It includes the scoring of respiratory rate, oxygen saturation, temperature, heart rate, blood pressure (both systolic and diastolic) sedation and urine output.

A Maternity MEWS is to be calculated each time a set of observations is performed.

Australian Commission for Safety and Quality in Healthcare requires that all inpatients receive a minimum of 8 hourly observations. Unless otherwise documented in the medical management plan.

Trigger Score

A maternity MEWS of ≥ 4 is a trigger for action, with escalated notification at ≥ 6 and ≥ 8

Text box 5: Trigger Score Maternity MEWS

The standard operating procedure includes:

- A flow chart to direct midwives regarding who to notify and when
- A structure for increasing the frequency of observations once a trigger score is reached
- A guide as to what escort is required if the patient is to be transferred to another clinical area e.g. Fetal Medicine Unit.

The Maternity MEWS does not replace calling the Medical Emergency Team (MET). If a woman meets the MET criteria, a Code Blue/MET should be called as per Medical Emergency Policy.

Maternity MEWS	4	3	2	1	0	1	2	3	4
Resp rate (breaths per min)	≤ 4	5-8			9-20	21-24	25-30	31-35	≥36
SpO2 (%)	≤84	85-89	90-92	93-94	95-100				
Temperature °C		≤ 34	34.1-35.9		36-37.4	37.5-37.9	38-38.9	≥39.0	
Heart Rate (beats per min)	≤39			40-49	50-99	100-109	110-129	130-139	≥140
Systolic Blood Pressure mmHg¹	<90				90-129	130-139	140-169	≥170	
Diastolic Blood Pressure mmHg¹	<45				45-89	90-99	100-109	≥110	
Sedation Score					Awake		Mild	Mod	Severe

Maternity MEWS¹

Table 8: Maternity MEWS for each variable

Maternity Summary

- Maternity MEWS score is calculated by adding the score generated by Respiratory Rate, Oxygen Saturation, Temperature, Heart Rate, Systolic BP & Diastolic BP and Sedation score.
- Trigger score: A total Maternity MEWS of ≥ 4 is a point for action and further escalation of the level of review at a scores ≥ 6 or if the patient is not improving.
- Maternity MEWS does not replace calling the MET. If patient meets MET criteria a MET should be called.
- A Maternity MEWS of ≥ 4 :
 - Increase frequency of observations
 - Notify the team leader or Clinical Midwife Consultant (CMC)
 - Notify Medical staff
 - Patient requires escort for transport off ward.



Oxygen Delivery

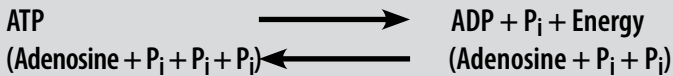
Oxygen Delivery

Learning Objectives

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

Background

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



Text box 6: Energy release

If there is inadequate oxygen supply, ATP production falls, and cellular function is then depressed (figure 5), through lack of energy. This can lead to organs failing and may result in an unexpected admission to ICU or even unexpected deaths. Therefore if oxygen delivery is maintained, this may reduce the incidence of unplanned ICU admissions and unexpected deaths.

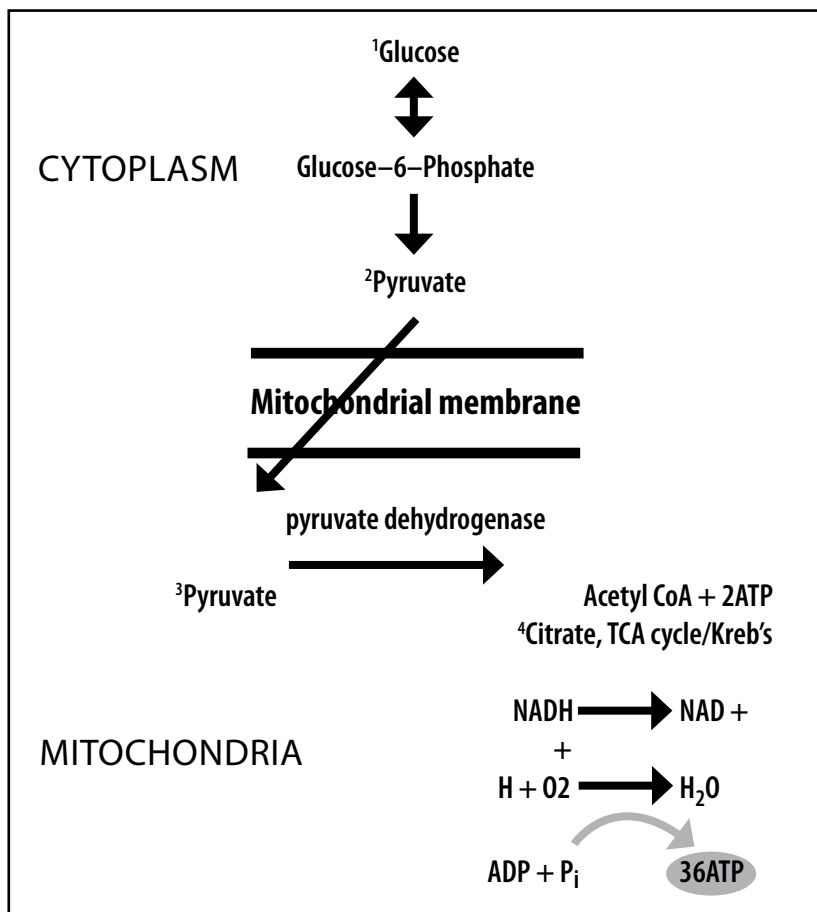


Figure 4: Aerobic Metabolism (i.e. with oxygen)

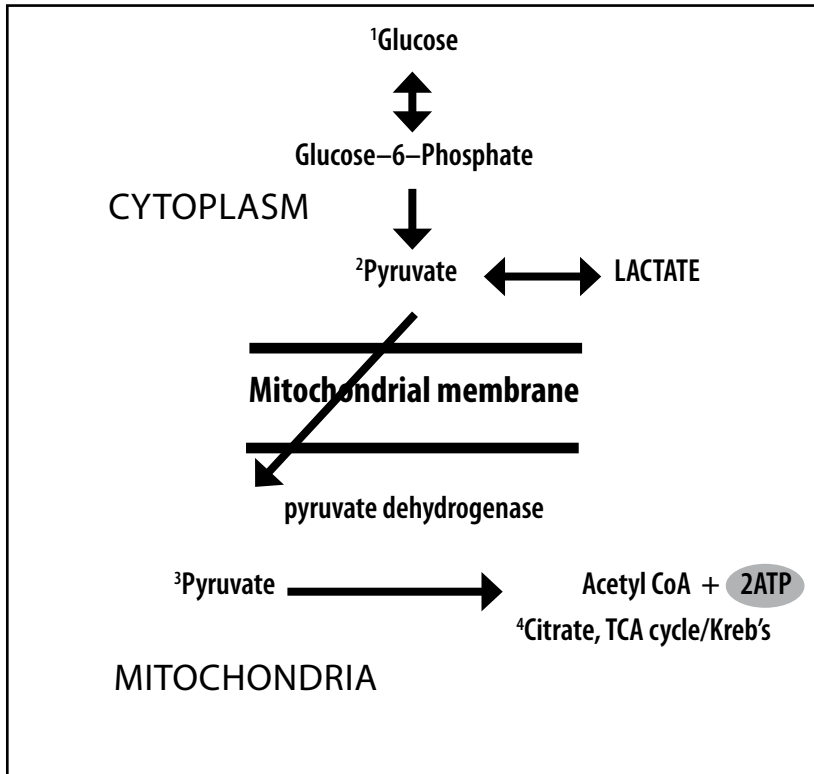


Figure 5: Anaerobic Metabolism (i.e. in the absence of oxygen)

Oxygen supply to the cells can be described by the “oxygen delivery chain” (figure 6).

Oxygen Delivery = Cardiac Output x Arterial Oxygen content.

Thus oxygen delivery requires:

- A. Arterial oxygen content:
 - haemoglobin concentration ($[Hb]$)
 - haemoglobin oxygen saturation (SaO_2)
 - partial pressure of oxygen (PaO_2).

See Section on “Airway and Breathing”.

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

See Section on “Circulation”.

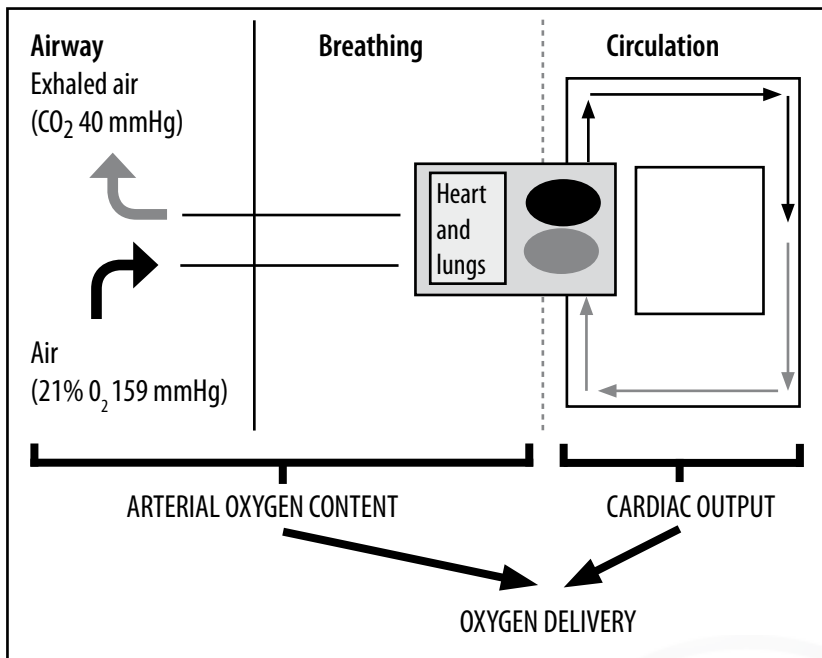


Figure 6: “ABC” and the Oxygen Delivery Chain



MATERNITY OXYGEN DELIVERY

Physiological changes in the oxygen delivery caused by pregnancy include:

- Oxygen consumption increases by 16-20% due to the increased demand from both the woman and the fetus
- PaO_2 increases and PaCO_2 decreases during pregnancy
- There is a respiratory alkalosis due to hyperventilation this is usually compensated by renal excretion of bicarbonate in the kidneys.

SUMMARY

- Oxygen is essential for the adequate production of adenosine triphosphate (ATP).
- If there is inadequate oxygen supply, ATP production falls, and cellular function is then depressed, organs start to fail and puts patient at risk of critical illness and death.
- Oxygen Delivery = Cardiac Output x 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 manage appropriately a patient with impaired arterial oxygenation
- To understand why the respiratory rate is such an important marker of the deteriorating patient.

INTRODUCTION

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

AIRWAY

Adult Airway

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

Causes of airway obstruction

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

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

Examination of the airway

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

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

Management of the obstructed airway

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

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

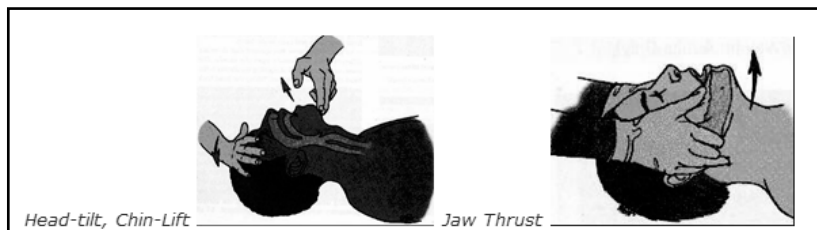


Figure 7: Simple Airway Manoeuvres

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

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

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

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

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

Text box 7: Airway obstruction

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



PAEDIATRIC AIRWAY DIFFERENCES

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

A. Anatomically

- 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 part of the airway in infants and children < 8 years of age is below the vocal cords at the cricoid cartilage
- The vocal cords are short and concave
- Large tongue in proportion to the oral cavity.

B. Physiologically

- The lower airways are smaller and so are at greater risk from being obstructed by mucous, oedema or active constriction
- Minor decreases in the diameter of the small paediatric airway creates a large increase in the amount of resistance to airflow
- The diaphragm plays a more significant role in the generation of tidal volume of infants and children as the cartilage supporting the ribs is more flexible allowing for paradoxical movement of the chest wall
- The metabolic rate of children is higher than adults, so oxygen consumption is higher and hypoxia can occur more rapidly.

Like adults, airway obstruction can either be mechanical or functional.



Figure 8: Infant airway with permission from the European Resuscitation Council

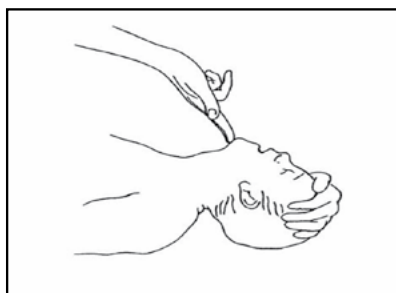


Figure 9: Chin Lift in children. With permission from APLS¹⁴



MATERNITY AIRWAY

Physiologic changes caused by pregnancy affect both airway and breathing.

Airway

Oedema of the airway due to hyperaemia and increased secretions can make intubation more difficult.

There is also a greater risk of aspiration due to the effect of pregnancy on the smooth muscle in the gastrointestinal tract.

Always treat a pregnant woman as if she has a full stomach

BREATHING

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

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

Examination of Breathing

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

Look

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

Look for signs of respiratory failure which can include:

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

Listen

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

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

Then auscultate with a stethoscope to assess breath sounds:

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

Feel

1. Palpation

Palpate the trachea and chest wall:

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

2. Percussion:

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

Why Respiratory Rate is Important

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

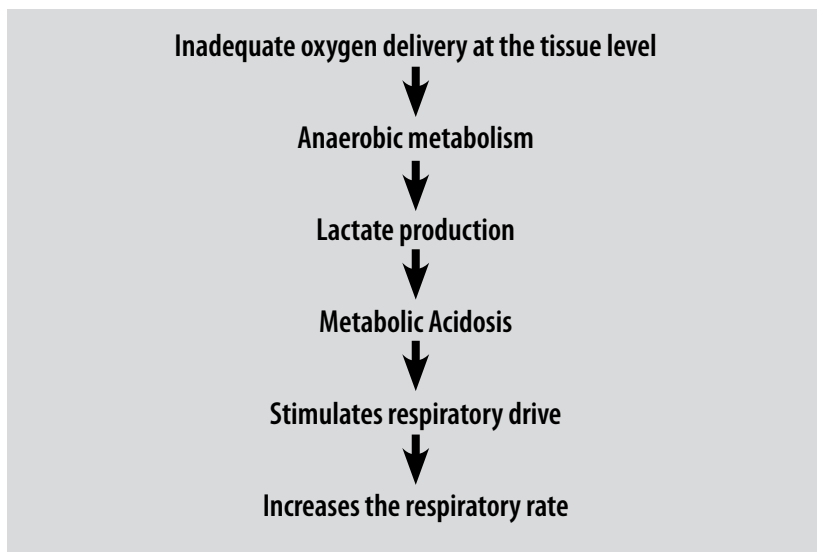


Figure 10: Importance of Respiratory Rate

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

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

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

A Normal Arterial Saturation and Tachypnoea

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

Respiratory Rate, SpO2 MEWS and MET

The respiratory rate and arterial oxygen saturations score (table 9) for the Adult MEWS are as follows:

MEWS	4	3	2	1	0	1	2	3	4
Resp Rate per min	≤4	5-8			9-20	21-24	25-30	31-35	≥ 36
Oxygen saturation	≤84	85-89	90-92	93-94	95-100				

Table 9: Adult Respiratory MEWS

* Reproduced with permission from Taylor & Francis L

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 but their oxygen demands have increased (that is they need more oxygen to maintain the level) the patient is deteriorating.

MET criteria RR < 5 or > 36

Text box 9: Adult Respiratory Rate MET criteria

² With permission from the Australian Society of Anaesthetists

Management

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

All deteriorating patients should receive oxygen before progressing to any further assessment. The aim is to deliver supplemental oxygen to achieve a SpO₂ of 94-98% in those patients not at risk for hypercapnic respiratory failure, and the PaO₂ as close to 100mmHg as possible, but at least 60mmHg (SaO₂ 90%) is essential. In most patients, this can be achieved by sitting them upright, and applying 12-15 litres/min of oxygen via a non-rebreather mask (figure 14).

If the patient does not improve they will require an ICU review (or in paediatric patients NICU/ICU/ Paediatrician review).

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

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

Text Box 10: Oxygen delivery in COPD

OXYGEN DELIVERY SYSTEMS

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

A. Fixed performance devices

Provide gas flow that is sufficient for all the patient's minute ventilation requirements. In these devices, the inspired oxygen concentration is determined by the oxygen flow rate and attached diluter (see table 10), e.g. the Venturi mask (figure 11).

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



Figure 11: Venturi mask system

Image reproduced with permission of mayohealthcare.com.au

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

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

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

B. Variable performance devices

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

The inspired oxygen concentration will depend on:

- 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-rebreather masks.

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

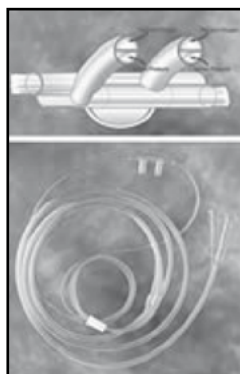


Figure 12: Nasal prongs Image reproduced with permission of mayohealthcare.com.au

- c) **Adult face mask** (figure 13) – reservoir volume of oxygen is increased above that achieved by the nasopharynx (text box 11), thus higher oxygen concentration can be achieved in inspired gas (max 50-60%).

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

Text box 11: Adult face mask



Figure 13: Adult face mask

Image reproduced with permission of mayohealthcare.com.au

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



Figure 14: 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.

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

If the carbon dioxide tension rises in someone with acute respiratory failure, it can be a sign that they are tiring and may require ventilatory support. If CO_2 begins to rise in a patient with COPD, it may be prudent to reduce the inspired oxygen concentration, however always remember that the arterial oxygen tension should not be allowed to fall below pO_2 60mmHg.

Patients do not die from a raised CO_2 alone: they die from hypoxaemia, (text box 12).

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

Text box 12: Arterial blood gases

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

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

PAO_2 should be close to PaO_2 in normal lungs

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

Text box 13: Ideal alveolar gas equation

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

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

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

Text box 14: Pulse oximetry warning

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

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

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

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

ABGs should be measured in patients who¹⁵:

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

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

Text box 16: Vital signs

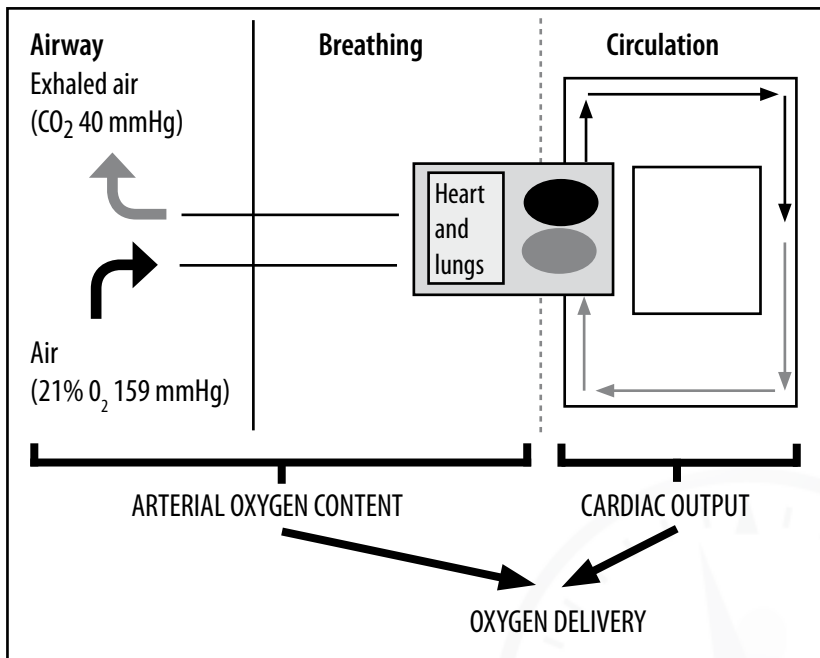


Figure 15: ABC-Oxygen Delivery Chain



PAEDIATRIC BREATHING DIFFERENCES

In children an increasing respiratory rate maybe an early sign of inadequate oxygen delivery. It is important to note that the metabolic rate of children is higher than adults. This means oxygen consumption is higher and hypoxaemia can occur more rapidly.

Examination of Breathing

Look

The normal respiratory rate in children varies with age and it is important to recognise the MET criteria for each age group (See tables 2, 3 & 4).

Clinical signs of inadequate breathing include:

- use of accessory muscles
- pallor
- central cyanosis
- tachycardia or bradycardia in extreme hypoxia
- head bobbing or nasal flaring.

It is important to note that breathlessness and cyanosis in infants and children may be a sign of serious cardiac pathology.

Text box 17: Breathlessness and cardiac pathology

Listen

Timing and nature of airway noises can assist in determining the site of the airway problem. Hearing inspiratory noises like stridor, snoring, and bubbling sounds would suggest upper airway obstruction (croup, foreign body inhalation, excessive nasal and oral secretions). Expiratory noises like wheezes suggest a lower airway obstruction (asthma). If cough is present, identify its characteristics: moist, barky, dry or paroxysmal.

A paroxysmal cough followed by an inspiratory “whoop” is suggestive of pertussis. A barky cough is associated with croup. The early stages of asthma may present with a dry cough especially at night. Grunting is an attempt to provide Positive End Expiratory

Pressure (PEEP). Auscultate with a stethoscope to assess depth and equality of breathing and quality of breath sounds. Absent or decreased breath sounds could indicate foreign body obstruction, consolidation, pleural fluid or pneumothorax.

Feel

The thin chest wall of the infant and young child enables the palpation of vibrations, called fremitus. Increased fremitus may indicate fluid accumulation, while decreased fremitus may indicate chronic obstructive pulmonary disease.

Management

Specific treatment will depend on the cause, and it is vital to diagnose life threatening conditions immediately, e.g. acute asthma, severe croup, epiglottitis and foreign body inhalation.

All deteriorating infants and children should receive oxygen to maintain saturations at least 93%.

Text box 18: Oxygen for deteriorating infants

It is important to remember that high concentrations of oxygen in pre-term infants up to 34 weeks can cause retinopathy and so should be avoided. The method of delivery needs to be determined by taking into consideration the cooperation of the infant/child and the concentration needed to maintain adequate arterial oxygen saturation.

A period of progressive respiratory compromise, which leads to hypoxia and acidosis can lead to a cardiac arrest in an infant and child.

There are a small group of children (chronic lung disease, cystic fibrosis, heart defects) who have an arterial oxygen saturation range that is normal for them but far below normal range for well children. Any alteration to the PEWS should be decided by a senior medical officer and documented in the variances section on the observation chart.

Text box 19: Chronic disease and arterial oxygen saturation

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 in paediatrics, however only occasionally.

Variable performances devices such as nasal prongs, simple facemasks, partial rebreather and non-rebreather masks come in different sizes to suit the size of the child. Paediatric face masks have a minimum flow rate of 4L/min. Nasal prongs come in neonatal, infant, paediatric and adult sizes.

There are three types of flow meters used in paediatrics with nasal prongs:

- **Micro flow via nasal prongs** can be used for neonates and pre mature infants. These are placed on oxygen flow rates of up to 0.1 L/min.
- **Low flow via nasal prongs** for rates less than 2 L/min. Infants are often placed on flows from 0.1 – 1.0 L/min via a low flow meter. Rates of 1- 2 L/min can be via a regular flow meter. However, once the infant/child requires 2L/min or more of oxygen, it is advised to change to warmed humidified nasal prong oxygen. This will improve the tolerance of the nasal prongs and decrease discomfort and irritation.
- **High flow nasal prongs** utilises high flow of oxygen which increases the amount of air remaining in the lungs at the end of expiration, prevents lung closure at the end of respiration and stabilises the airway. They can be run from 2-8 L/min.

Humidification helps to loosen secretions and improve mucocilliary transport, prevent nasal obstruction from hard dry secretions and decrease discomfort and irritation. The type of humidification device selected will depend on the oxygen delivery system in use and the patients requirements. Cold dry air may increase heat and fluid loss in infants.

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.



MATERNITY BREATHING

Breathing:

Pregnancy is a state of relative hyperventilation.

- The tidal volume increases by up to 50% with the chest diameter increasing by up to 2 cm.
- There is a reduction in respiratory reserve (amount that can be breathed out after a normal expiration)
- There is a reduction in residual volumes (the volume of air in the lungs after maximum expiration)
- Respiratory rate remains unchanged during pregnancy.

Oxygen Therapy in Pregnancy

Oxygen therapy may be indicated in the care of an unwell woman. If a woman acutely deteriorates oxygen is indicated and should be applied. Ongoing management of oxygen therapy should be reviewed as part of the management plan once the woman is stabilised and as per other relevant standard operating procedures.

Oxygen Therapy Postpartum

Women who are unwell or who have had a surgical birth may require oxygen therapy; this is usually delivered via nasal prongs as per adult protocols.

ADULT SUMMARY

- An increase in Respiratory Rate can occur even though the arterial oxygen saturation may be normal.
- In rare cases, an airway obstruction may be due to mechanical factors, which may not be so easily treated, e.g. airway swelling, post-operative haematoma, infection. This is a medical emergency. A Code Blue/MET should be called.
- In a small subgroup of patients who have Chronic Obstructive Pulmonary Disease (COPD) and are “CO₂ retainers”, high concentrations of inspired oxygen can be disadvantageous as it suppresses their hypoxic drive. However, these patients will also suffer end-organ damage or cardiac arrest if their blood oxygen levels fall too low. The aim in these patients is to achieve a PaO₂ of 60mmHg, or oxygen saturation of 90% on pulse oximetry. So, in a patient with COPD who has a pCO₂ > 60mmHg but is also hypoxic, pO₂ < 60mmHg, do NOT turn the inhaled O₂ down however do not leave them unattended. If their pO₂ is > 60 mmHg, then you can turn the inhaled O₂ down to maintain SaO₂ > 90%.
- Oxygen flow rates less than 6L/min for adult face mask and 4L/min for a child face mask should not be used due to carbon dioxide retention in the mask.
- When taking an arterial blood gas sample, do not remove the oxygen mask, it is unnecessary and may precipitate sudden deterioration.
- If the pulse oximeter does not provide a reading, do not assume it is broken; the patient may have poor perfusion and be very unwell!
- Oxygen saturation may be “normal” but the pCO₂ may be high, reflecting inadequate minute ventilation and respiratory failure.
- Remember to incorporate all the vital signs in your assessment!
- Adult MET criteria RR < 5 or > 36 breathes per minute.



PAEDIATRIC SUMMARY

- The metabolic rate of children is higher than adults. This means oxygen consumption is higher and hypoxaemia can occur more rapidly.
- It is important to note that breathlessness and cyanosis in infants and children may be a sign of serious cardiac pathology.
- Any alteration to the PEWS should be decided by a senior medical officer and documented in the variances section on the observation chart.



MATERNITY SUMMARY

- Due to relaxation of smooth muscle there is a greater risk of aspiration during pregnancy
- Oxygen Therapy may be indicated in the care of the unwell woman and should be applied and reviewed as part of the management.



Circulation

Circulation

Learning Objectives

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

The Importance of Oxygen

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

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

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

Stroke Volume x Heart Rate **Haemoglobin x SaO₂**

Figure 16: Oxygen delivery

BLOOD PRESSURE

Blood Pressure, Heart Rate and Oxygen Delivery

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

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

Text box 20: Blood pressure

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

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

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

Blood Pressure and Maintenance of Organ Function

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

Therefore it is important to know what your patient’s “normal” or “usual” blood pressure was prior to their current illness. There is a place to record this on the Observation Chart, and the MEWS for BP is adapted to reflect the patient’s usual blood pressure.

Adult Heart Rate MET Triggers

A heart rate of less than 40 beats per minute or greater than 140 beats per minute requires a Code Blue (MET referral)

Text box: 22

Calculation of Adult MEWS for blood pressure:

Usual SBP		190	180	170	160	150	140	130	120	110	100	90	80
Current Blood Pressure	200s	0	0	1	1	2	2	2	3	3	4	5	5
	190s	0	0	0	1	1	1	2	2	3	3	4	4
	180s	0	0	0	0	0	1	1	2	2	3	3	4
	170s	1	0	0	0	0	1	1	2	2	3	3	3
	160s	1	1	0	0	0	0	0	1	1	2	2	2
	150s	1	1	1	0	0	0	0	0	1	1	2	2
	140s	2	1	1	1	0	0	0	0	0	1	1	1
	130s	2	2	1	1	0	0	0	0	0	0	0	0
	120s	2	2	2	1	1	0	0	0	0	0	0	0
	110s	3	2	2	2	1	1	0	0	0	0	0	0
	100s	3	3	3	2	2	2	1	1	0	0	0	0
	90s	4	3	3	3	2	2	2	2	1	1	0	0
	80s	4	4	4	4	4	4	4	4	4	4	1	0
	70s	4	4	4	4	4	4	4	4	4	4	4	4

Table 11: Adult Blood pressure MEWS

³ Reproduced with permission of Wiley-Blackwell Publishing

Definition of Hypotension

The generally acceptable definition of hypotension in adults is:

- A drop of more than 20% from “usual” blood pressure **OR**
- Systolic blood pressure of less than 100mmHg.

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

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

Text box 23: Critical Blood pressure

Possible Causes of Hypotension

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

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

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

A. Cardiac output

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

Factors affecting stroke volume:

1) **Contractility**

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

Major negative influences (negative inotropy) include:

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

Major positive influences (inotropy) include:

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

2) **Pre-Load**

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

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

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

a) Intravascular blood volume

Absolute:

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

Relative:

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

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

Text box 24: Relationship between intravascular blood volume and blood pressure

b) Intrathoracic pressure

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

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

Text box 25: Relationship between intrathoracic pressure and blood pressure

3) After-Load

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

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

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

B. Heart rate

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

- Parasympathetic stimulation: SLOWS the heart rate via the vagus nerve e.g. vasovagals response, parasympathomimetics e.g. anticholinesterases (neostigmine)
- Sympathetic stimulation: QUICKENS the heart rate via the sympathetic cardiac fibres e.g. stress response, temperature, Sympathomemetics (adrenaline, noradrenaline, isoprenaline).

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

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

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

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

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

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

Calculation of Adult MEWS for heart rate:

MEWS	4	3	2	1	0	1	2	3	4
Heart Rate	< 40			40-49	50-99	100-109	110-129	130-139	≥140

Table 12: Heart rate Adult MEWS

* Reproduced with permission from Taylor & Francis Ltd

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

Text box 27: Causes of a fall in blood pressure

C. Peripheral Vascular Resistance

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

1. Increase in peripheral vascular resistance

Autonomic Nervous System

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

2. Decrease in peripheral vascular resistance

a. **Blockade of Autonomic Sympathetic Nervous System**

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

Influences include:

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

b. Direct action on arteriole smooth muscle

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

Examples include:

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

Compensatory Mechanisms for Hypotension

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

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

Text box 28: Components of BP

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

Causes

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

1. Reduction in Stroke Volume

Response:

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

While this compensation can return BP to normal values. If CO has not been restored, there may be evidence of persistent inadequate oxygen delivery (text box 29).

Seagull Sign

- Normally the heart rate value is below the Systolic BP
- If the Heart rate goes above the Systolic BP then this is referred to as a Seagull Sign (See Figure 17)
- BP will only fall once the compensatory mechanisms have failed
- Visually this may be represented on the chart as a Seagull sign where the heart rate can be seen above the Systolic BP this is a sign of deterioration and medical review should be requested.

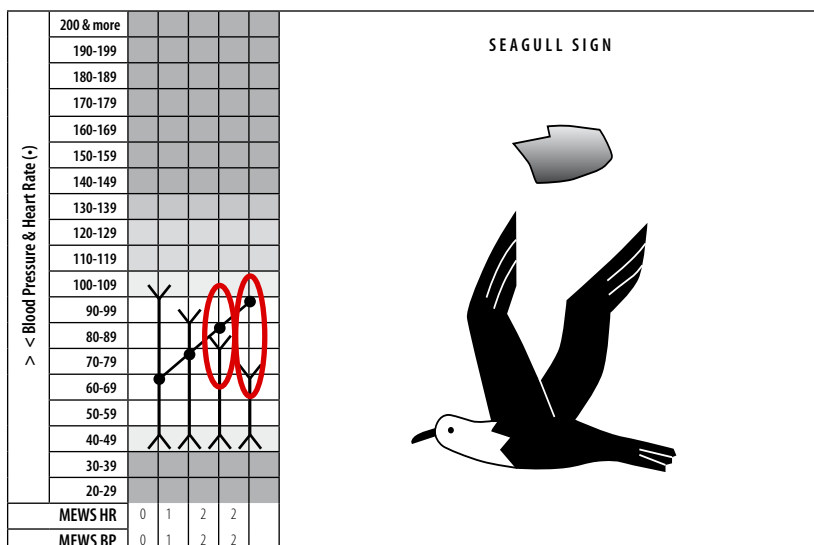


Figure 17: Seagull Sign

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

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

2. *Reduction in Heart Rate*

Response:

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

Hypotension, bradycardia and cool, mottled peripheries

Text box 30: Clinical features of Reduction in Heart Rate

B. Reduction in Peripheral Vascular Resistance

Response:

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

Hypotension, tachycardia and warm peripheries

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

Consequences of Hypotension

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

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

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

Text box 32: Relationship between blood pressure and oxygen delivery

A. Inadequate Cardiac Output

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

B. Inadequate Pressure Gradient

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

When is hypotension not shock?

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

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

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

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

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

The Initial Management of Hypotension

It is important to remember what generates a blood pressure:

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

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

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

Text box 33: Adult MET criteria for BP

A. Fall in peripheral vascular resistance

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

Management Plan

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

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

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

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

- Administer intravenous fluid bolus (500 -1000 mls of Normal Saline [0.9% NaCl] for adults)

- Continue to perform frequent vital signs to document any trends (e.g. 1/2hrly for 1 hr, followed by hourly for 4 hours, then subsequently 4-hourly if stable)
 - If there is an improvement in tachycardia and blood pressure, then the fluid bolus has been adequate to restore venous return (NOTE- this may only be a compensatory response so continue to monitor frequently)
 - If the tachycardia remains repeat the fluid challenge
 - Continue to observe response
 - If the patient continues to have hypotension, tachycardia and warm hands, further fluid can be administered particularly if there are no signs of heart failure
 - An intensive care review should be requested once three litres of fluid have been administered and the tachycardia and hypotension are still present.
- ***Hypotension and evidence of organ failure***
 - Administer intravenous fluid bolus (500 - 1000 mls of Normal Saline [0.9% NaCl] for adults)
 - Continue to perform frequent vital signs to document any trends (1/2hrly x 1 hr, 1/24 x 4, then 4/24 if stable)
 - If there is an improvement in tachycardia and blood pressure, then the fluid bolus has been adequate to restore venous return.
 - If the tachycardia, hypotension and organ failure remains, repeat the fluid challenge
 - Call for an intensive care review particularly if the patient has received three litres of fluid or if the signs of organ failure persist
 - Continue to perform hourly observations to ensure that the trend of blood pressure, pulse and mental state are being monitored.

B. Fall in Cardiac Output

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

1. Fall in Pre-Load

- Common causes include bleeding, loss of fluids and electrolytes
- History
 - Will describe histories relevant to bleeding, loss of fluid and electrolytes (diarrhoea, vomiting, and polyuria from hyperglycaemia), loss of water (diabetes insipidus).
 - Look at fluid balance chart and determine recent fluid balance.
 - Can also describe symptoms of postural hypotension (feels faint when standing up, has actually "fainted").
- Examination
 - Signs that are relevant to the fluid lost (bleeding into drains, melaena, nasogastric losses)
 - Cool, mottled hands, tachycardia, hypotension with a postural drop (a drop more than 10mmHg in Systolic BP from lying to sitting).
- Laboratory Investigations
 - Evidence of bleeding (fall in haemoglobin)
 - Evidence of renal dysfunction (rising creatinine)
 - Evidence of lactate formation (metabolic acidosis on arterial blood gas sampling [negative base excess], rising lactate).
- Management
 - Correct cause of loss of fluid (call surgeon for ongoing bleeding, may need to correct coagulopathy)
 - Replace whatever fluid has been lost (blood if bleeding, saline if gut losses, 5% Glucose if diabetes insipidus)

- Estimate how much has been lost by looking at the fluid balance chart, how much is in the drains, how far has the haemoglobin fallen
- In the first instance in adults rapidly administer 500 - 1000 mls of Normal Saline via a blood pump set through a large bore intravenous cannula
- Observe response (tachycardia should be reduced and blood pressure increase).
- Continue to administer fluid rapidly until there is the desired response:
 - Blood pressure returning to normal
 - Heart rate returning to normal
 - Improvement in organ function, particularly urine output.
- Intensive care should be alerted especially if there are no signs of improvement despite administering 3L of fluid
- Continue to perform regular observations (hourly or as directed by MEWS protocol).

2. Fall in contractility

- Common causes include myocardial ischaemia or infarction.
- History
 - May describe history of chest pain suggesting ischaemia
 - May describe previous symptoms of heart failure (orthopnoea, swollen ankles, breathlessness)
 - Describe palpitations (suggesting a tachycardia- atrial fibrillation, ventricular tachycardia) or symptoms related to causes of cardiomyopathy.
- Examination
 - Cool, blue hands, tachycardia and hypotension.
 - Signs of right heart failure (swollen ankles, raised jugular venous pressure)

- Signs of left heart failure (tachypnoea, fine inspiratory crackles that do not clear on coughing, third heart sound, low arterial oxygen saturation).
- Investigations
 - Evidence of renal dysfunction (rising creatinine)
 - Evidence of lactate formation (metabolic acidosis on arterial blood gas sampling [negative base excess], rising lactate).
- ECG - signs of ischaemia, infarction, dysrhythmia.
- Management
 - If the patient is hypotensive and has signs of organ failure including heart failure (cardiogenic shock), the patient will require inotropic support and referral to either the coronary care unit or intensive care unit
 - Stop all intravenous fluids as the patient is by definition fluid overloaded.

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

Text box 34: oxygen delivery

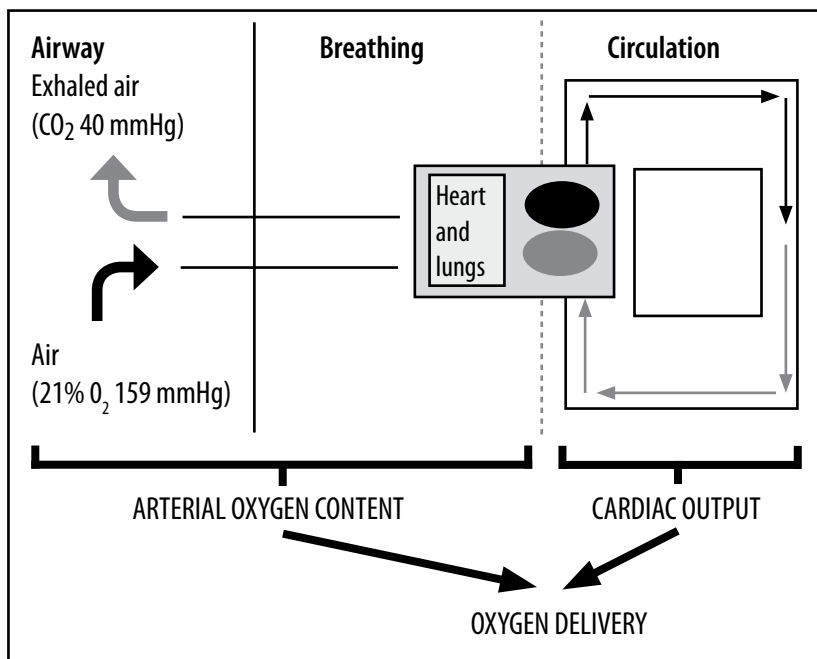


Figure 18: Oxygen Delivery chain



Paediatric Circulation

Paediatric Blood Pressure

Blood pressure in children increases with growth and maturation. The range for normal blood pressure for each age group is outlined in the PEWS standard operating procedure.

Blood Pressure measurement in children

All children must have their blood pressure measured with an appropriate sized cuff:

- on admission
- when the PEWS is ≥ 6
- pre-operatively, and
- on return to ward post-operatively.

Blood pressure should be measured 4 – 8 hourly, or more frequently if indicated, for a child/adolescent of any age who:

- is unwell
- shows clinical signs of deterioration and/or shock
- is requiring intravenous therapy bolus (≥ 10 mls/kg)
- has renal or cardiac disease
- has diabetes or adrenal disorders,
- has neurological presentations such as head injury, encephalopathy, suspected raised intracranial pressure, and focal neurological signs
- has suspected sepsis
- has significant trauma including burns (≥ 10 %).
- has suspected poisoning/ingestion, and

In addition BP should be measured as directed by a medical officer and if the PEWS is ≥ 6 without the inclusion of the BP score, blood pressure should be measured.

What size cuff to use

Use an appropriate size cuff based on the manufacturer's instructions.

ALERT: A child's pain, anxiety, anatomy or condition may hinder blood pressure measurements. The first BP readings should be interpreted with this in mind.

Hypotension in Paediatrics

Hypotension is a very late sign in children due to their excellent cardiovascular compensatory mechanisms. Hypovolemia causing circulatory collapse is one major precipitant of cardiac arrest in infants and children.

NOTE: The Seagull sign is not relevant to Paediatric patients as the normal heart rate is higher than the normal systolic blood pressure until they reach the adolescent age group.

Text box 35: Hypotension in children

Hypotension in infants and children is most likely to occur because of fluid loss from conditions such as gastroenteritis, intussusception, or haemorrhage. Other reasons for hypotension can be from vasodilatation associated with sepsis, anaphylaxis, or poisoning.

Infants and children have a greater percentage of body water compared to body weight than adults. They have the potential for greater insensible losses due to their relatively large surface area to volume ratio. This may result in more water loss and temperature loss.

Hypotension in infants and children is a late sign. Decompensation often happens quickly and if not treated immediately can quickly lead to death.

Text box 36: Hypotension in children

Paediatric Compensatory Mechanisms for Hypotension

Cardiac output

Cardiac output is calculated the same way for infants and children as it is for adults. It is the product of stroke volume and heart rate (i.e. flow is the volume per unit time).

Infants have a smaller stroke volume relative to size than children ≥ 2 years of age. This stroke volume is relatively fixed so that cardiac output is directly related to heart rate. Practically this means that increasing fluid volume only works up to a point as stroke volume cannot be significantly increased.

So an increasing heart rate in infants and children is often an early sign of falling stroke volume, shock and/or inadequate oxygen delivery. This is accompanied by peripheral vasoconstriction to maintain blood pressure.

As the ability of the body to compensate for inadequate oxygen delivery decreases the signs include:

- altered mental state, in infants fatigue may be an early indication
- capillary refill > 3 secs
- heart rate and respiratory rate trending down to normal levels without the infant/child looking any better.

Alert: Hypotension in infants and children is a late sign. Decompensation often happens quickly and if not treated immediately can quickly lead to death.

The Initial Management of Hypotension in Children

1. All children in shock should receive oxygen irrespective of oxygen saturation as they all have tissue hypoxia
2. IV access should be established if not already present. If unable to cannulate within 1-2 minutes, obtain intraosseous access. The preferred site for intraosseous access is the medial anterior surface of the tibia 2-3 cm below the tibial tuberosity. Alternative locations include the medial distal tibia above the medial malleolus and the anterolateral surface of the distal tibia 3cm above the lateral condyle
3. If it has been determined that the child is hypovolaemic, give a fluid bolus of 20 mls/kg Normal Saline (0.9% NaCl). Reassess. If necessary another 20mls/kg can be given. Inotropic support needs to be considered after boluses reach 40-60 mls/kg.



MATERNITY CIRCULATION

Physiological changes caused by pregnancy effect circulation.

- The peripheral vascular resistance (PVR) is decreased from about 6 weeks gestation and peaks at half way through the pregnancy.
- Due to the decreased PVR the afterload is reduced
- The decreased PVR results in decreased diastole and subsequently both systole and diastole should be closely monitored.
- Blood pressure is reduced in the first trimester then steadily increases
- The heart rate increases by 10 – 15 beats per minute
- To compensate for the reduction in PVR, there is an increase in blood volume, up to 50-60%
- The cardiac output is increased by 35-50% due to the increased heart rate and increased blood volume
- Red blood cells increase by 20-30%
- But due to the increase in plasma volume there is a reduction in red blood cell, haematocrit and haemoglobin concentrations
- White blood cells increase but due to the suppression of some lymphocytes pregnant women are more susceptible to viral infections
- At any time the uterus has 30% of the circulating blood
- Aortocaval compression in the supine position significantly reduces venous return and hence reduces cardiac output significantly.

Laying a pregnant patient on their back can cause aortocaval compression from the weight of the gravid uterus on the abdominal aorta and inferior vena cava. This can result in reduced cardiac output and hypotension.

Best position is 30 degrees back from the left lateral position.

Venous Thromboembolism (VTE)

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

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



SUMMARY

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

**A SYSTOLIC BLOOD PRESSURE OF LESS THAN 90mmHg in adults
REQUIRES A CODE BLUE (MET REFERRAL).**

PAEDIATRIC SUMMARY

- Infant's have a smaller stroke volume relative to size than children ≥ 2 years of age.
- Stroke volume is relatively fixed so that cardiac output is directly related to heart rate.
- Increasing fluid volume only works up to a point as stroke volume cannot be increased significantly.

Alert: Hypotension in infants and children is a late sign. Decompensation often happens quickly and if not treated immediately can quickly lead to death.

MATERNITY SUMMARY

- Peripheral vascular resistance is decreased from 6/40 gestation
- This results in changes in diastole so that both Systolic and Diastolic BP is included in the early warning score.
- Heart rate is increased by 10-15 bpm
- Aortocaval compression in the supine position significantly reduces cardiac output therefore a pregnant patient should not be positioned flat on her back.



Central Nervous System and Urine Output

Central Nervous System (CNS)

Learning Objectives

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

Introduction

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

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

Table 13: Common causes of decreased level of consciousness

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

Causes of Depressed Level of Consciousness

1. Inadequate Oxygen delivery

Neurons in the central nervous system, like all other cells in the body, are highly dependent on oxygen. Adequate oxygenation allows the formation of large amounts of ATP “energy packets” which are required for all cellular functions (figure 4).

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

Oxygen supply to the cells in the brain depend on the same factors as oxygen supply to all other tissues in the body (figure 18).

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

a) decreased cardiac output

- decrease stroke volume
- decrease heart rate.

(This may be indicated by a decreased blood pressure)

b) decreased arterial oxygen content

- decreased haemoglobin
- decreased arterial haemoglobin saturation.

c) decreased blood pressure

- decrease in cardiac output
- decrease in peripheral vascular difference.

2. Inadequate Substrate Delivery for Metabolism

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

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

Text box 37: BGL and decreased level of consciousness

Assessment of CNS

A. Level of consciousness

The sedation score used in the MEWS grades the degree of drowsiness according to the criteria in table 14, and should be done on every patient. This is added to the other MEWS for the other vital signs, and the total is calculated to give an overall MEWS.

MEWS	0	2	3	4
Sedation Score	Awake	Mild	Moderate	Severe

Table 14: Adult Sedation MEWS

⁵ Reproduced with permission from Oxford University Press

Sedation Score:

0 = Awake

2 = Mild, Easy to rouse

3 = Moderate, Constantly drowsy, easy to rouse but unable to stay awake

4 = Severe, Somnolent, difficult to rouse

Text box 38: Criteria used for the sedation MEWS

⁵ Reproduced with permission from Oxford University Press

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

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

Table 15: Glasgow Coma Scale

⁶ (Used with permission of Elsevier Limited)

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

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

Text box 39: MET Criteria for CNS

² (With permission from The Australian Society of Anaesthetists)

B. Pupillary Size

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

BILATERAL PUPILLARY DILATATION

Causes:

- sympathetic overactivity, e.g. fear, stress, anxiety, hypoglycaemia
- sympathomimetic administration, e.g. administration of adrenaline in an arrest situation
- anticholinergic activity e.g. atropine, tricyclic antidepressants, ipratropium nebuliser.

BILATERAL PIN POINT PUPILS

Causes:

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

UNEQUAL PUPILS



Causes:

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

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

Text box 40: Pupil size

Management of Decreased Loss of Consciousness

1. Check airway and breathing; ensure airway is patent
 - Head tilt, jaw thrust
 - Insert an oropharyngeal (Guedel's) or nasopharyngeal airway.
2. Apply high-flow oxygen
3. Measure blood glucose, and correct if $<3\text{mmol/L}$ (administer 50mls of 50% glucose intravenously—this requires a medical order).
 (In children administer 2 mls/kg of 10% glucose intravenously—this requires a medical order)
4. MET call if patient meets the criteria (GCS fallen >2 points)
5. If respiratory rate or arterial oxygen saturation is decreased, the patient may need ventilatory assistance using self-inflating bag and mask
6. Ensure intravenous access; 500 mls intravenous fluid bolus may be required if patient is hypotensive.
-  In a child 20 mls/kg NS intraosseous access should be used if an IV cannula cannot be inserted into a child.
7. Reverse any drug-induced CNS depression, e.g. naloxone for opioid overdose (requires a medical order)
8. If the airway is patent, and the patient is breathing, place patient supine in lateral recovery position.

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



PAEDIATRIC DIFFERENCES IN CENTRAL NERVOUS SYSTEM

Assessment of Level of Consciousness

It is important to choose the developmentally appropriate tool when assessing the level of consciousness in infants and children. It can be difficult to assess the early signs of neurological deterioration (reduced attention and dulled affect) in the infant/ young child. Often the parent is the best resource as they know their child is “just not themselves”. The child who does not recognise their parent is significantly compromised.

In infants and young children the “AVPU” scale is most often used as it is more developmentally adaptable than the Glasgow Coma Scale.

A - Alert

V - Responds to voice

P - Responds to pain

U - Unresponsive

Causes of Loss of Consciousness

Poisoning is a common cause of neurological deterioration especially in the toddler age range. Depending on the substance ingested the pupils may be small (opiates, organophosphates) or large (amphetamine, atropine, tricyclic antidepressants).

Checking the Blood Glucose Level is one of the first things, which should be checked on a patient with decreased level of consciousness, or fitting patient, whether they are diabetic or not. The BGL should be > 3.0 mmol/L. In children administer 2 mls/kg of 10% glucose intravenously —this requires a medical order.

Assessment of Fontanelle

The anterior fontanelle is normally flat. It usually closes by 18 months of age but can close as early as 9 months of age. It is best assessed when the infant is quiet. Crying vigorously can make the fontanelle more prominent. A bulging fontanelle is a sign of increased intracranial pressure from such causes as meningitis, an intraventricular bleed, and hydrocephalus. A sunken fontanelle usually indicates dehydration.



MATERNITY CENTRAL NERVOUS SYSTEM

CENTRAL NERVOUS SYSTEM

Changes in the CNS during pregnancy or in the early postnatal period are most commonly related to pre-eclampsia or eclampsia.

Pre-eclampsia

Pre-eclampsia manifests in hypertension arising after the 20th week gestation (and up to 6 weeks postpartum) and the NEW onset of one or more of the following:

- Proteinuria
- Renal insufficiency
- Liver disease
- Neurological problems
- Haematological disturbances
- Fetal growth disturbances.

Severe Pre-eclampsia

Severe pre-eclampsia will manifest by CNS changes such as headache or blurred vision

Any development or worsening of symptoms is indicative of a change in the woman's condition. It is important to remain alert to the development of symptoms even if the woman regularly suffers from headache or migraine.

Headache

- The headache associated with pre-eclampsia can be temporal, frontal, occipital, or diffuse
- It is usually a throbbing/pounding pain, but piercing pain can occur
- If the headache is not relieved by over the counter analgesics or it becomes severe it should be treated as a sign of deterioration.

Treatment for pre-eclampsia

Most women with CNS changes/symptoms attributed to pre-eclampsia are treated with:

- antihypertensive medication to maintain blood pressure in a safe range
- magnesium sulphate to prevent seizures
- analgesia
- birth of the baby if necessary.

ECLAMPSIA

Seizures in a woman with pre-eclampsia signifies a change in diagnosis to eclampsia.

Stroke leading to death or disability is the most serious complication of severe pre-eclampsia/eclampsia, but it is rare.

Treatment for eclampsia

The immediate priorities in caring for a woman with eclampsia include:

- Prevention of maternal hypoxia and trauma
- Management of severe hypertension, if present
 - Hydralazine
- Prevention of recurrent seizures
 - Magnesium sulphate

- Evaluation for prompt delivery of baby

The definitive treatment for eclampsia is the birth of the baby, irrespective of gestational age, to reduce the risk of maternal morbidity and mortality from complications of the disease.

Complications

Complications of eclampsia include increased risk of:

1. Abruptio placenta
2. Preterm delivery
3. Intrauterine growth restriction
4. Perinatal mortality
5. Disseminated Intravascular Coagulation (DIC).

HELLP syndrome is a serious complication of Pre-eclampsia involving a combination of liver and blood disorders.

H - haemolysis - red blood cell damage

EL - elevated liver enzymes

LP - low platelets in the blood.

SUMMARY

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

PAEDIATRIC SUMMARY

- Choose a developmentally appropriate tool to assess level of consciousness on infant or child
- It is important to identify and treat the cause of an altered level of consciousness
- BGL is one the first things that should be checked in a patient with an altered level of consciousness or who is fitting.

MATERNITY SUMMARY

- Severe pre-eclampsia will manifest by symptoms such as headache and blurred vision.
- In addition to the Maternity MEWS signs and symptoms for pre-eclampsia should be monitored and treated.
- It is important to identify and treat the cause of altered level of consciousness
- BGL is one the first things that should be checked in a patient with an altered level of consciousness or who is fitting.

Urine Output

Learning Objectives

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

Introduction

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

Pathophysiology

Normal urine flow requires:

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

A. Oxygen Delivery

In order to function, renal cells require adequate oxygen delivery, just as all the other cells in the body. Oxygen delivery depends on cardiac output and arterial oxygen content (figure 18).

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

B. Perfusion Pressure

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

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

Figure 19: Mean arterial pressure

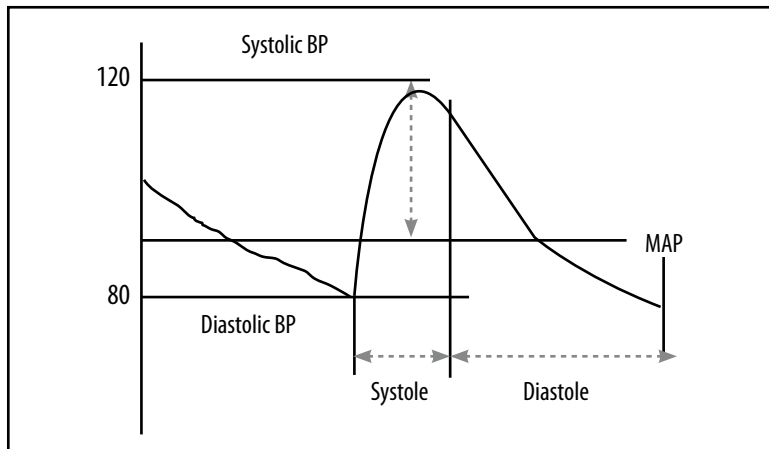


Figure 20: Mean Arterial Pressure Diagram

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

Management of Low Urine Output

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

1. Decreased renal blood flow in the face of decreased blood pressure, cardiac output or tissue oxygen delivery.
2. Obstructed urine flow – needs to be urgently corrected if this is the case, thus it is important to diagnose early.
3. Patients with an escalating MEWS ≥ 4 should be on a strict fluid balance chart.

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

Text box 41: Urine output

Decreased Renal Blood Flow

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

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

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

Text box 42: Oliguria

Management of Pre-Renal Oliguria

When oliguria is due to decreased perfusion i.e. decreased blood pressure or cardiac output, it is potentially reversible. In this circumstance, the most important initial management is to exclude hypovolaemia (decrease in cardiac preload) being the cause. If hypovolaemia is likely (relative or absolute) give an intravenous fluid bolus of 500 mls of Normal Saline (adults). Frusemide is not to be

given unless you have ruled out all other possible reasons for low urine output, and the patient is clinically fluid overloaded.

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

Management of Post-Renal Oliguria

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

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

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

Text box 43: Frusemide



PAEDIATRIC DIFFERENCES FOR URINE OUTPUT

Glomerular filtration rate increases throughout the first two decades of life. This means that infants and children cannot concentrate their urine as efficiently as adults.

Infants and children have a greater percentage of body water compared to body weight than adults. They have the potential for greater insensible losses due to their relatively large surface area to volume ratio. This may result in more water loss and temperature loss.

Newborns have larger extracellular fluid levels than infants and older children. This extracellular fluid decreases over time so that by one year the ratio of extracellular fluid to intracellular fluid is close to adult levels.

Urine output should be 1-2 mls/kg/hr i.e. 12-24 mls/hr for a 12 kg child.

Text box 44: Paediatric urine output



MATERNITY URINE OUTPUT

During pregnancy changes also occur that affect the urinary system. These include:

- The kidneys increase in size
- The bladder's tone decreases
- There is an increase in plasma flow to the kidneys.

Monitoring of urine output is a crucial when recognising and responding to a deteriorating woman.

Renal System

Physiological changes caused by pregnancy effect:

- Elevations in plasma flow and glomerular filtration leading to an elevation in the creatinine clearance
- Blood urea and serum creatinine are reduced by 40%
- Increase in glomerular filtration may affect the ability to reabsorb resulting in glucose and protein losses in the urine
- Mild glycosuria (1-10 gm/day) and/or proteinuria (to 300 mg/day) can occur in normal pregnancy
- Progesterone can induce dilation and atony of the renal calyces and ureters
- The enlarging uterus can compress the ureters as they cross the pelvic brim and cause further dilatation by obstructing flow. These changes may contribute to the frequency of urinary tract infections during pregnancy.

SUMMARY

- Checking the **BGL** is one of the first things which should be checked on an unconscious or fitting patient, whether they are diabetic or not
- Adult urine output should be $> 0.5 \text{ mls/kg/hr}$ i.e. 35mls/hr for a 70 kg adult
- There is a small window of opportunity for reversing oliguria and preventing acute renal failure

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



- Paediatric urine output should be $1\text{-}2 \text{ mls/kg/hr}$ for example 12-24mls/hr for a 12 kg child
- Maternal urine adult will be as for adults $> 0.5 \text{ mls/kg/hr}$ i.e. 35 mls/hr for a 70 kg woman.



Communication, Team Work and Management Plans

Communication, Team Work and Management Plans

Learning Objectives

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

One of the most important factors in determining an acutely ill patient's outcome is the quality of the communication among the clinicians involved. In each team, each member has their strengths and weaknesses, varying skills and different levels of knowledge.

The aim in managing the deteriorating patient is to determine the role of each member of the team, identify their comfort zones and work together with this knowledge to affect the best outcome for the management of the particular patient. The flowchart in figure 21 gives you a basic outline for management.

Optimising the management of the deteriorating patient requires:

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

Text box 45: Optimising management

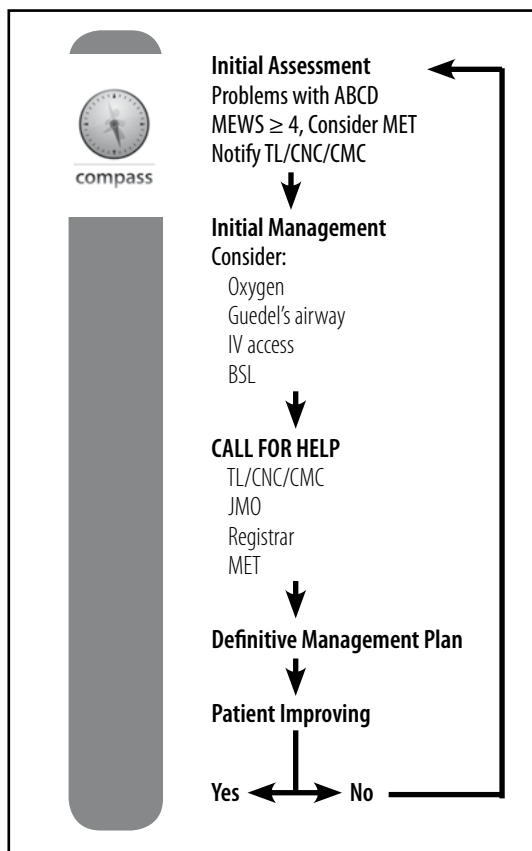


Figure 21: Flow chart of steps in managing a deteriorating patient

1. Gathering Information

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

Examples:

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

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

Information can be obtained from:

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

Text box 46: Sources of information

2. Integration of Information

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

3. Communicating Information

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

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

Text box 47: Abnormalities and appropriate care

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

This means health professionals have to:

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

“ISBAR” COMMUNICATION

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

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

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

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

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

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

Text box 48: ISBAR Communication

⁸ (Reproduced with permission from BMJ Publishing Group Ltd)

For example:

A 75-year-old lady with a history of Ischemic Heart Disease is admitted with a fractured neck of femur. Twelve hours post-operatively, she complains of chest pain and her arterial oxygen saturation has fallen to 88% on 2L nasal prongs and has a MEWS of 4. You as the person nursing her are concerned that she is acutely unwell and needs attention. The ISBAR communication technique would proceed as follows:

IDENTIFY

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

SITUATION

"She is a 75 year old lady who has a MEWS of 4 due to dropping her arterial oxygen saturation to 88% on 2L/min of O₂ via nasal prongs, she is tachycardic and tachypnoeic. She is also complaining of chest pain"

BACKGROUND

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

ASSESSMENT

"I think she is acutely unwell and may have"

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

RECOMMENDATION

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

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



PAEDIATRIC COMMUNICATION

Timely, accurate communication is vital in determining the outcome for an acutely ill infant or child. Infants and children generally deteriorate more rapidly than adults and it is imperative that senior nursing and medical staff are involved early.

Parents will generally be at the bedside and be quite concerned with the changes in their child. Enlist an appropriate person to provide support to the parents while you concentrate on the urgent medical needs.

Paediatric ISBAR example:

An 8 month old male infant with a history of prematurity and extensive bowel resection secondary to Necrotising Enterocolitis (NEC) is admitted to the ward with gastroenteritis. During the morning shift there were large fluid losses through urine and faeces. The baby is tachypnoeic, tachycardic, pale, crying and restless. You, as the nurse caring for the child, are concerned that the baby is acutely unwell.

IDENTIFY

"This is Sarah from Paediatrics calling about David Jones, is this Dr Brown?"

SITUATION

"He is an eight month old male infant who has significant fluid losses and looks unwell" He has a PEWS of 4, his respiratory rate is 70 and he is tachycardic at 180. He has IV fluids prescribed."

BACKGROUND

"He has a short gut from extensive resection following NEC. He came in with gastroenteritis"

ASSESSMENT

"I think he is acutely unwell and may be moderately to severely dehydrated. He has lost 200 grams since admission".

RECOMMENDATION

"I think this patient needs more fluids, electrolyte levels and an arterial blood gas with an urgent medical review."



MATERNITY COMMUNICATION

Prompt concise communication is imperative to the care of a deteriorating maternity patient. Communication is required to alert the Obstetric and Gynaecological (O&G) Team of an obstetric complication. Note that communication of adult MET criteria may also required.

Maternity ISBAR example:

A 35 year old female with a history of pre-eclampsia has undergone an elective caesarian section. She has returned to the postnatal ward and has experiences a significant post partum haemorrhage.

IDENTIFY

"This is Jane RM on postnatal I am calling about Elise Smith, is this Dr Roberts?"

SITUATION

"She is experiencing a post partum haemorrhage of approx 800 mls with a Maternity MEWS of 6 for RR, HR and BP.

BACKGROUND

"She is a G1 P1 who returned from theatre half an hour ago post elective caesarian for pre-eclampsia."

ASSESSMENT

"She has a boggy uterus that is deviated to the left and has soaked 2x pads int he last 5 mins"

RECOMMENDATION

"We have commenced the post partum haemorrhage protocol and I need you to attend for urgent review".

Documentation

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

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

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

When communicating information you must:

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

Text box 49: Communicating

4. Adequate response to information/concerns

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

After communicating with more senior colleagues, an individual may feel as if they were not taken seriously or their particular

concern about a situation wasn't addressed. This can be remedied by specifically asking each member of the team what their concerns are, how they think this can be addressed, and integrating those concerns into their management plan.

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

For example:

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

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

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

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

Text box 50: ISBAR

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

5. Formulating, Documenting and Communicating Management Plans

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

Optimal management plans include action plans for all members of the team and time frames in which things must be actioned. Medical staff must always document their impression, which is the provisional diagnosis (text box 51). When this is done, each member has a clear idea of their roles and responsibilities and no excuses for not following them!

1. Observation Orders

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

2. Nursing Orders

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

3. Allied Health Orders

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

4. Change in Therapy Orders

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

5. Investigation/intervention orders

If it has been decided that the patient needs their electrolytes checked then this must be documented, as well as whose responsibility it is to check the results. It is often useful to write what is expected and what to do about abnormal results if this is predictable. You may now realise that the patient requires IV access for antibiotics that have been ordered.

6. Notification Orders

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

6. Actioning the management plan

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

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

7. Reassess

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

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

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

When documenting a medical entry always document:

H – history

E – examination

I – impression/diagnosis

P – management plan

Text box 51: Documentation

Management Plans should include:

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

Text box 52: Management plans

SUMMARY

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



References

References

1. Morgan, R.J.M, Williams, F. & Wright, M.M. (1997).
'An early warning scoring system for detecting developing critical illness', *Clinical Intensive Care* 8:4, p11
2. Lee, A., Bishop, G., Hillman, K., & Daffurn, K. (1995).
'The Medical Emergency Team', *Anaesth Intensive Care* 23, pp183-186
3. Day, B. (2003).
'Early warning system and response times: an audit', *Nursing in Critical Care* 8:4, pp156-164
4. Australian Resuscitation Council, *Guideline 5- Breathing*
<http://www.resus.org.au/>
5. Malvija, S., Voepel-lewis, T., Tait, A.R., Merkel, S., Tremper, K., and Naughton, N. (2002).
'Depth of sedation in children undergoing computer tomography: validity and reliability of the University of Michigan Sedation Scale(UMSS)', *British Journal of Anaesthesia* 88:2, pp241-245
6. Jennett, B. & Teasdale, G. (1974).
'Assessment of coma and impaired consciousness', *Lancet* 2, pp 81-84
7. Pittard, A.J. (2003).
'Out of our reach? Assessing the impact of introducing a critical care outreach service', *Anaesthesia* 58:9, pp 882-885
8. Leonard, M, Graham, S. & Bonacum, D. (2004).
'The human factor: the critical importance of effective teamwork and communication in providing safe care', *Quality and safety in Health Care* 13, ppi85-i90
9. Eather, B. (2006)
St.George Hospital Modified Early Warning Score policy
10. J Tibballs, S Kinney, T Duke, E Oakley, and M Hennessy
'Reduction of paediatric in-patient cardiac arrest and death with a medical emergency team: preliminary results', *Arch. Dis. Child.*, Nov 2005 90, pp1148 - 1152.

11. Duncan, H., Hutchison, J., Parshuram, C.S.
'The pediatric early warning system score: A severity of illness score to predict urgent medical need in hospitalized children', *Journal of Critical Care*, 2006 3:21, pp271-279
12. 'Paediatric life support', *Resuscitation European Resuscitation Council Guidelines for Resuscitation 2005, Section 6*, ppS97-S113
13. *Greater Ormond Street Hospital Children's Early Warning Score*
14. 'Advanced Paediatric Life Support', *The Practical Approach* (4th Ed.) Blackwell Publishing 2005
15. Ward, H., Douglas J., Armstrong, A. *Prince Charles COMPASS manual*
16. Mitchell I.A., McKay H., VanLeuvan C., Berry R., McCutcheon C., Avar, B., Slater N., Neeman T., Lamberth P.
'A prospective controlled trial of the effect of a multi-faceted intervention on early recognition and intervention in deteriorating hospital patients', *Resuscitation* 81 (2010), pp658-666
17. Emergency Management Training Services
<http://emtsstudentpage.com/>
18. O'Loughlin, Hoan, J. Larki, A. Hindle, H. (2007). *The Early Warning Scoring System (EWS) For Use in Maternity*. The Pennine Acute Hospitals, NHS Trust
19. August, P. & Sibai B. 17.3 Sept 2009. *UpToDate. Clinical features, diagnosis, and long-term prognosis of preeclampsia.*
20. Norwitz, E.R. 17.3 Sept 2009. *UpToDate. Eclampsia.*

This manual has been written by:

Dr. Bronwyn Avar

Ms. Heather McKay

Ms. Nicole Slater

Dr. Paul Lamberth

Dr. Kathryn Daveson

Dr. Tony Lafferty

Mr. Jon Darvil

Ms. Sue Chen

Ms. Raelene Garret-Rumba

Dr. Imogen Mitchell

Special thanks to:

2016 edition was edited by Dr Bronwyn Avar, Nicole Slater, Sarah Mamootil, Mary Bodilsen, Christopher Perkins, Penny Giles and A/Prof Imogen Mitchell.

Matt Gladwish of ACT Publishing Services for his assistance in developing the **online learning** application.

Dr Ross Peake for his voice talents on the application.



Quiz Questions

Quiz Questions

An increased heart rate may reflect:

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

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

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

Cardiac Output =

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

Aerobic metabolism:

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

Tachypnoea:

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

Blood Pressure =

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

Oxygen delivery =

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

Urine output depends on the following two things:

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

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

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

The Seagull sign is when:

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

PAEDIATRIC



The most appropriate tool for assessing the neurological state in an infant is:

- a. GCS
- b. AVPU
- c. Palpating the fontanel
- d. Ask the parents



Infants and children can exhibit early signs of compensated shock. This includes:

- a. Increased heart rate secondary to increased stroke volume
- b. Increased heart rate secondary to relatively fixed stroke volume
- c. Decreased blood pressure
- d. Drowsiness



The observations and PEWS are to be recorded on the age specific observation charts. Use corrected age for premature babies up until the age:

- a. 3 months
- b. 6 months
- c. 12 months
- d. 2 years



Infants and children have a greater potential for greater insensible losses due to :

- a. Lower percentage of body water compared to body weight than adults.
- b. Relatively large surface area to volume ratio.
- c. Relatively small surface area to volume ratio.
- d. Inability to concentrate urine

MATERNITY

A woman's oxygen consumption changes due to pregnancy. This change in oxygen consumption is a:

- a. Decrease by 20%
- b. Increase by 20%
- c. Decrease by 50%
- d. Increase by 50%

During pregnancy a woman's plasma volume increases. This is by a value of:

- a. 50%
- b. 20%
- c. 35%
- d. 10%

There are a number of physiological changes that occur with pregnancy, one of these changes is with Peripheral Vascular Resistance.

Is the PVR:

- a. Reduced
- b. Increased
- c. Unchanged

Tachypnoea is an indicator of oxygen deprivation and thus deterioration of a patient. However, it is normal for a pregnant woman to be tachypnoeac.

This is:

TRUE / FALSE

