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- Clinicians throughout NSW that were part of the review process for their invaluable feedback prior to its release.

Disclaimer

Any views, recommendations and conclusions expressed in DETECT Junior do not necessarily reflect the views of the New South Wales Ministry of Health. The management strategies outlined in DETECT Junior represent the views of the contributors. The editors, authors and other contributors take no responsibility for any adverse event associated with the use of DETECT Junior. Users of DETECT Junior should check content against local protocols and drug doses against a recognised paediatric formulary. DETECT Junior is not intended to replace local policies or acknowledged sources for administration of medications to children. Clinicians using DETECT Junior to guide them in the management of deteriorating patients should at all times apply clinical judgement regarding appropriate management of the patients in their care. DETECT Junior should not be routinely applied to newborn infants cared for in the neonatal nursery. DETECT Junior material is copyright, and may not be reproduced for commercial usage or sale.
FOREWORD

“Dance there upon the shore;
What need have you to care?
For wind or water’s roar?”

To a Child Dancing in the Wind by W.B. Yeats (1864-1939)

The late Professor Don Harrison and I have focused our research and subsequent education programme on the centre of our working lives, the seriously ill adult patient, and so DETECT was born. With the collaboration of an exceptional editorial team (M Fisher, K Fraser, K Hillman, G Reece) and many learned authors and advisors, DETECT has matured such that the adult product better meets the needs of our broad range of students. The DETECT editors bring together some essential knowledge and skills in the programme using blended learning, believing that the dynamic circumstance of the deteriorating adult cannot be taught by text alone. DETECT emphasises practical vignettes inspired by the wealth of experience of our contributors. DETECT does not replace textbooks or scientific literature and we do not seek to replace the many expert courses that already exist.

We are delighted to welcome DETECT Junior to the DETECT umbrella. DETECT Junior maintains the pragmatic DETECT genre. The target audience remains broad, ranging from those who work predominantly in the “adult world” who may encounter sick children in the emergency department or paediatric wards of community hospitals and are likely to have experienced DETECT, to junior doctors and nurses from paediatric tertiary referral centres who may not.

In the first chapter the DETECT Junior editors, Marino Festa and Joanne Leaver, have set the scene for a practical and simple approach to recognising and treating the sick or deteriorating child. The DETECT acronym — Detect deterioration, Evaluate, Treat, Escalate and Communicate with your Team — has been applied to the different ages of childhood, the familiar DETECT algorithms adapted and the “Worry Face” used to flag paediatric vignettes and caveats. Any controversy about the differences between adults and children becomes irrelevant when it comes to timely detection and intervention for a deteriorating patient of any age. The DETECT editors and DETECT Junior editors hope no child or parent will have ‘need to care’ if the DETECT Junior messages of “When to Worry” are applied by members of their health care team.

DETECT remains a work in motion.
INTRODUCTION

DETECT Junior is a multidisciplinary education and training package that has been developed as a part of the Clinical Excellence Commission’s (CEC’s) Between the Flags (BTF) program. It provides education on the recognition and management of clinical deterioration in paediatric patients and is aimed at all front line clinicians who care for infants and children within NSW healthcare facilities.

The BTF program is designed to improve recognition and response to all patients who deteriorate in NSW public hospitals and facilities and was developed in response to recommendations outlined in a number of reports by the NSW Health Greater Metropolitan Clinical Taskforce (GMCT) Working Party¹, the NSW Health Patient Safety and Clinical Quality Program and the Garling Inquiry.

The BTF program comprises five elements; governance, standard observation charts, escalation systems (CERS), education, and evaluation. The education element incorporates DETECT (Detecting deterioration, Evaluation, Treatment, Escalation and Communicating in Teams), the adult education and training package that focuses on the recognition and management of the deteriorating adult patient, and DETECT Junior.

Although DETECT Junior has been developed as part of the BTF program, its value as an educational resource extends beyond NSW and may easily be adopted by other healthcare facilities, both nationally and internationally.

AIM OF DETECT JUNIOR

The aim of DETECT Junior education is to:

- Provide healthcare practitioners with the knowledge, skills and understanding to recognise and respond to early and late warning signs of clinical deterioration in infants and children.
- Develop a systematic approach to ensure appropriate assessment and timely intervention and escalation.
- Improve non-technical skills such as communication, teamwork, decision making and situation awareness in order to reduce the likelihood of adverse events as a result of human factors.
INSTRUCTIONS

DETECT Junior is a modular learning package that consists of this manual, e-learning and face to face training. The intention of this manual is to provide healthcare practitioners with the theoretical knowledge that will enable successful completion of the other components.

DETECT Junior does not aim to provide training in cardiopulmonary resuscitation (CPR). It is a requirement that participants have current accreditation in CPR training prior to undertaking the face to face training. Resus4Kids is advocated as the CPR training for healthcare practitioners.

DETECT Junior does not aim to provide information regarding the newborn period and does not include parameters from the Standard Newborn Observation Chart used in Special Care Nurseries and Post Natal Wards.

IMPORTANT NOTE

DETECT Junior applies the Clinical Emergency Response System (CERS) outlined in the NSW Health Policy Directive - PD2011_077 Recognition and Management of Patients who are Clinically Deteriorating. It refers to a Clinical Review and Rapid Response as the escalation processes for observations that fall within the yellow and red zones respectively on the Standard Paediatric Observation Charts (SPOC).

A Rapid Response is defined as ‘an immediate review undertaken by an individual or multidisciplinary team of healthcare professionals who have been trained and assessed to hold an advanced level of competency in resuscitation and stabilisation of patients’.

SUMMARY OF DETECT JUNIOR EDUCATION

1. Complete pre-reading: DETECT Junior manual
2. Successfully complete DETECT Junior e-learning
3. Successfully complete appropriate CPR training e.g. Resus4Kids
4. Attend DETECT Junior face to face training

This is the DETECT Worried Face symbol that flags When to Worry signs and key management principles. It shall be used throughout this manual.
CHAPTER ONE

WHEN TO WORRY

MARINO FESTA AND JOANNE LEAVER

AIM

In this chapter you will become familiar with the rationale for *When to Worry*. The chapter aims to enhance your knowledge on a structured approach to assessment, how to assess an infant or child, what to look for, and what physiological signs should be cause for concern. It also highlights the importance of involving the caregiver at all stages of the process.

WHY WORRY?

As in adult healthcare, an important cause of reported adverse events and deaths in children is a delay in response to documented deterioration. As a member of the healthcare team, whether formally trained in adult or paediatric care, you are an important part of the solution to identify “tragic and avoidable” events. By improving our ability to DETECT and manage the deteriorating infant or child’s condition, appropriately listening to concerned caregivers, and communicating and escalating our concerns in a timely manner, undoubtedly we will save lives.

Cardiac arrest in infants and children is often preceded by warning signs.\(^2\)\(^3\) Unlike adults, most arrests in children occur as a result of serious deterioration in either respiratory or circulatory function rather than primary myocardial failure.\(^2\)\(^3\) Respiratory insufficiency leading to respiratory arrest typically precedes cardiac arrest for most children. *If respiratory arrest is treated promptly it may not progress to cardiac arrest.*

The reported outcomes for infants and children following cardiac arrest are poor.\(^4\)\(^5\) Often severe damage has already occurred in the lead up to the cardiac arrest. Early recognition of the signs and symptoms of deterioration prior to cardiac arrest is paramount in preventing death and adverse outcomes.

DIFFERENCES WHEN CARING FOR CHILDREN

Children at different ages and stages of development differ markedly in size, anatomy, physiology, pathophysiology, and psychology. As a child grows and develops major changes occur in weight, body proportions, physical appearance, intellectual ability, emotional response and in body systems and physiological processes.
These different stages of development are commonly grouped as neonate, infant, child and adolescent. An age-appropriate approach to assessment, one based on normal responses, behaviours and stages of development, is essential for an accurate diagnosis of clinical deterioration. A caregiver is often the best person to help you understand what response is normal for their child, particularly in the assessment of ex-premature infants or children with developmental delay. The following are some general guidelines to assist in the assessment of the different age groups.

**NEONATE (UP TO 28 DAYS OF LIFE)**
- No stranger anxiety so your presence will not upset the child.
- No separation anxiety so it’s less essential to assess in the caregiver’s arms.
- They should make eye contact but no big smiles until infancy.

**INFANT (UP TO 12 MONTHS OF AGE)**
- You may console younger infants with comforter/cuddles and older ones may be distracted by noise or objects.
- By 6 months infants start to develop stranger anxiety and later on separation anxiety, so try to ensure the caregiver is present to provide comfort and reassurance.
- Approach gently, do visual assessment first and ‘hands on’ later when the child becomes more trusting of you.
- Engage caregiver in assessments and treatments e.g. they can hold stethoscope on chest, apply oxygen mask, raise the shirt to observe respiratory effort etc.
- It may benefit to have the caregiver hold the child in their arms facing away from you.

**CHILD (1 TO 12 YEARS)**

**Toddler — 1–2 years**
- Toddlers may be scared or distrustful of strangers. Ensure the caregiver is present during the assessment.
- Keep physical contact to a minimum until the child is familiar with you.
- Try to make a game out of the assessment by using toys as distractions and allowing them to touch and play with equipment.
- When healthy, toddlers do not sit still for long and like to be on the move, exploring. If a toddler is quiet reacting little to interventions it often indicates serious illness.
Preschool — 3–5 years

- Young children are typically scared by loss of control and fear being left alone.
- Allow the child to touch equipment and maybe give them a safe object to keep, such as a glove or a bandage.
- Provide reassurance and praise good behaviour.

School Age — 6–12 years

- Children of school age typically fear separation from caregivers, friends and home, and a loss of control, pain and physical disability.
- They may have a limited understanding of illness and hospital so provide simple explanations.
- Involve them in their care and allow time to let them talk in order to give them some control.

ADOLESCENT (13 TO 18 YEARS)

- Adolescents may vary widely in their reaction to illness, from accepting and calm, to tearful and scared, angry, resentful, and uncooperative.
- Treat them like an adult, show respect and empathy and provide reassurance. Speak to the child first and then involve the caregiver.
- Maintain privacy during assessment, uncovering areas only when necessary

Please note: throughout the chapter ‘child’ refers to infant, child and adolescent unless otherwise stated. Neonate is defined separately.

Helpful tips for assessing children

- Have the caregivers present and involve them in the care.
- Think from a child’s perspective and work in a child friendly way.
- Use play to assist children to express themselves.
- Get down to the child’s height and maintain good eye contact.
- Smile and talk in soft, reassuring tones.
- Use child friendly words, such as hurt, sore, tummy, brave, clever.
- Always provide age appropriate explanations before you do anything.
- Always be honest!!
SYSTEMATIC PATIENT ASSESSMENT — THE ABCDEFG ALGORITHM

Systematic assessment procedures involve a structured approach that should be undertaken after a rapid history. The simple assessment and management algorithm below – the ABCDEFG algorithm - provides this structured approach.

<p>| | |</p>
<table>
<thead>
<tr>
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<tbody>
<tr>
<td>A</td>
<td>• Airway</td>
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<tr>
<td>B</td>
<td>• Breathing</td>
</tr>
<tr>
<td>C</td>
<td>• Circulation</td>
</tr>
<tr>
<td>D</td>
<td>• Disability (neurological assessment)</td>
</tr>
<tr>
<td>E</td>
<td>• Exposure</td>
</tr>
<tr>
<td>F</td>
<td>• Fluids</td>
</tr>
<tr>
<td>G</td>
<td>• Glucose</td>
</tr>
</tbody>
</table>

POSITION THE CHILD

GIVE OXYGEN

Call for help
Ensure IV access

Never leave a deteriorating child without a priority management and review plan

Life-threatening conditions must be treated and escalated as they are identified throughout the ABCDEFG assessment.

Please note: this algorithm is for the assessment of the conscious patient and does not aim to replace the DRSABCD approach for use in individuals who are found collapsed or who have sudden collapse. You should be familiar with and competent in the Australian Resuscitation Council (ARC) standards for cardiopulmonary resuscitation.
As you work through the ABCDEFG algorithm, remember to use those simple clinical examination tools — eyes, ears and hands — look at the child, listen to the child and touch the child, always paying particular attention to caregivers concerns. Repeated observations over time are required to ensure subtle signs or cues of illness are detected.

‘Assessment is not a singular event, but a continuous and dynamic process’

You are aiming to detect physiological abnormalities and put history, signs and tests together to make a diagnosis. More importantly, you should be applying some simple treatments to make the child safe and prevent further deterioration, even if you are still unsure of the underlying problem.

**FIRST IMPRESSIONS**

Assessment should begin as you approach the child. The *first impression* of the child takes only moments and provides essential information on their clinical condition. First impression relates to general *appearance* and *behaviour* and is a sensitive indicator to the severity of illness. The TICLS assessment tool is part of the Paediatric Assessment Triangle and can help with this *first impression* – the ‘looks good’ vs. ‘looks bad’. It may assist you in detecting subtle abnormalities.

<table>
<thead>
<tr>
<th>TICLS ASSESSMENT TOOL</th>
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<tbody>
<tr>
<td><strong>Tone</strong></td>
</tr>
<tr>
<td>• An active child grabbing, reaching, or moving is good</td>
</tr>
<tr>
<td>• A still, floppy or quiet child is bad</td>
</tr>
<tr>
<td><strong>Interactivness</strong></td>
</tr>
<tr>
<td>• A child that is interested in the environment, smiling and happy is good</td>
</tr>
<tr>
<td>• A child not interested in the environment is bad</td>
</tr>
<tr>
<td><strong>Consolability</strong></td>
</tr>
<tr>
<td>• A child that can be easily comforted or calmed is good</td>
</tr>
<tr>
<td>• An inconsolable child is bad</td>
</tr>
<tr>
<td><strong>Look/Gaze.</strong></td>
</tr>
<tr>
<td>• A child who looks at caregivers or items of interest is good</td>
</tr>
<tr>
<td>• A child staring and not engaging in eye contact is bad</td>
</tr>
<tr>
<td><strong>Speech/Cry</strong></td>
</tr>
<tr>
<td>• A child who cries or talks is good</td>
</tr>
<tr>
<td>• A child who is moaning, grunting or quiet is bad</td>
</tr>
</tbody>
</table>

*Table 1*
THE ABCDEFG ASSESSMENT

A Start with examining the Airway

Airway obstruction is more common in a child than an adult because of the anatomical differences such as narrow airways and a floppy trachea. In addition, the effects of airway obstruction may manifest more quickly due to reduced physiological reserve and immature respiratory muscles that fatigue more easily. The anatomical and physiological differences are discussed in detail in Chapter 3.

Upper airway obstruction

A patent upper airway is the first priority. Any signs of upper airway obstruction can be determined within moments, without even touching the child. First impressions such as general appearance, behaviour, vocalisation, and the presence of respiratory noises will quickly indicate if the child has any upper airway obstruction.

Specific inspiratory noises can indicate upper airway obstruction:

- **Gurgling** may be heard when fluid or secretions are obstructing the airway. A failure to clear secretions may suggest the child is either very fatigued or has a decreased level of consciousness. It may also arise in children with neuromuscular disease such as cerebral palsy or muscular dystrophy where the ability to clear secretions is limited due to reduced muscle power.

- **Snoring (Stertor)** may be heard in states of decreased consciousness.

- **Stridor** may be heard in conditions that cause partial upper airway obstruction e.g. croup, epiglottitis, laryngomalacia, foreign body aspiration or anaphylaxis. The presence of a stridor requires immediate assessment of the degree of upper airway obstruction present to allow for appropriate treatment and escalation.
  - **Mild obstruction**: Child appears well with a stridor becoming apparent on exertion.
  - **Moderate obstruction**: Stridor at rest with signs of respiratory distress (refer to p 10).
  - **Severe obstruction**: Stridor with increasing tiredness and exhaustion. The stridor may actually decrease in volume as air entry decreases. Agitation or irrational behaviour, decreased level of consciousness, hypotonia, and marked pallor indicate a dangerous airway. **Hypoxaemia is a late warning sign in upper airway obstruction.**
Lower airway obstruction

Obstruction in the lower airways causes an *expiratory wheeze*, which is usually heard only on auscultation with a stethoscope. It is most commonly seen in children with reactive airway bronchoconstriction secondary to asthma, bronchiolitis or anaphylaxis.

Any significant degree of bronchoconstriction is accompanied by an obvious increase in the work of breathing. More severe lower airway obstruction may result in no air entry at all and a “silent” chest without audible air entry on auscultation.

*A silent chest is due to complete lack of air entry and is a medical emergency. Call a Rapid Response, commence oxygen therapy, position, and administer bronchodilators.*

Complete airway obstruction

If the child becomes unresponsive, apply immediate *airway opening manoeuvres* and rapidly assess for breathing using the LOOK, LISTEN, and FEEL approach.

**Airway opening manoeuvres**

The head tilt-chin lift manoeuvre is a simple and effective method to open the airway and is often successful in overcoming upper airway obstruction in children. Place one hand on the forehead and gently apply pressure to tilt the head back whilst lifting the chin upwards with the other hand. **Never overextend:** in an infant aim for a *neutral* position and in a child aim for a *sniffing* position.

**Airway opening manoeuvres in an infant**

Infant’s airways are soft and overextension of the neck can cause airway obstruction. To achieve a neutral position in an infant it is still necessary to perform the head tilt-chin lift manoeuvre, as the
large occiput at the back of the head can cause the chin to fall towards the chest resulting in airway obstruction.

![Figure 1: Head tilt-chin lift manoeuvre in an infant](image)

**Airway opening manoeuvres in a child**

Children's airways are more rigid and can tolerate a sniffing position.

![Figure 2: Head tilt-chin lift manoeuvre in a child](image)

Whilst maintaining the airway open:
- **LOOK** for chest and/or abdominal movement
- **LISTEN** for sounds of breathing
- **FEEL** for expired air

If there is no spontaneous breathing, activate a Rapid Response and commence immediate cardiopulmonary resuscitation (CPR).

**B For assessment of Breathing**

Keeping the child calm and comfortable during an assessment of the respiratory status is an important aspect since anxiety and crying can substantially increase the work of breathing making evaluation more difficult and causing further fatigue. Many clues can be obtained from a distance, as part of your first impressions, particularly their level of alertness and interactivity (*refer to the TICLS assessment tool p 5*).
Do not remove the child from their caregiver for examination unless the child has complete airway obstruction or markedly altered level of consciousness that threatens the airway. Temporary exposure of the child to observe the chest and abdomen is an essential part of the assessment.

A comprehensive respiratory assessment involves:

1. *Rate* of breathing
2. *Effort* of breathing
3. *Efficacy* of breathing
4. *Effects* of respiratory failure

**1. Rate of breathing**

<table>
<thead>
<tr>
<th>Age</th>
<th>Respiratory Rate (per min)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Under 3 months</td>
<td>30-55</td>
</tr>
<tr>
<td>3 - 12 months</td>
<td>30-45</td>
</tr>
<tr>
<td>1-4 years</td>
<td>20-40</td>
</tr>
<tr>
<td>5-11 years</td>
<td>20-30</td>
</tr>
<tr>
<td>12 years and over</td>
<td>15-20</td>
</tr>
</tbody>
</table>

Table 2

An infant's breathing pattern is often irregular (periodic breathing), changing constantly with their state of activity, so care needs to be taken when interpreting a single measurement. Monitoring the trend is an essential aspect and a key feature of the ‘track and trigger’ Standard Paediatric Observation Charts (SPOC), allowing observations to be recorded graphically and trends to be ‘tracked’.

Changes in respiratory rate include:

- **Tachypnoea (or an increasing trend)** indicates a need for increased ventilation due to lung or airway disease, fever or metabolic acidosis.

- **Bradypnoea (or a decreasing trend)** can indicate extreme fatigue or cerebral depression and can be a pre-arrest state. A downward trend in respiratory rate may not necessarily mean the child is improving; it could be an indication of deterioration as fatigue develops.

- **Abnormal pauses** can occur as the child starts to fatigues.
• **Apnoea** (defined as no respiratory effort for more than 20 seconds) can occur in neonates and young infants with an illness that may not always be respiratory in origin, such as sepsis and meningitis. Apnoeas may or may not be accompanied by bradycardia, however, they are always an indication of serious illness and must be escalated appropriately.

Helpful tips for counting respiratory rate

- Count respiratory rate at the beginning – doing temperature and heart rate may upset the child and a representative respiratory rate is virtually impossible.
- A stethoscope may assist - a caregiver can hold the infant against them whilst you place the stethoscope on their back.
- An infant’s breathing is often irregular so count the rate for a full minute as per recommended practice.

2. **Effort of breathing**

Assessing the effort of breathing (‘work of breathing’) is a *quick and efficient* way of determining respiratory inadequacy as it reflects the child’s attempt to compensate for inadequate oxygenation or ventilation. There are several clinical indicators that tell us if a child is breathing with an increased effort:

**Chest Recession**

Recession that reveals the outline of the ribs with each inspiration is an important sign of increased effort of breathing. *In older children it is a sign of severe respiratory distress.* As infants are abdominal breathers the work of breathing is often reflected in the degree of *abdominal excursion.*

*Photo 1: Intercostal recession*  
*Photo 2: Subcostal recession*
Accessory Muscle Use

Children, like adults, recruit accessory muscles when breathing is difficult, i.e. non respiratory muscles in the neck and abdomen. In infants this may be seen as head bobbing and is associated with ineffective breathing. Infants with this degree of respiratory effort are likely to fatigue.

Nasal Flaring

Flaring of the nostrils is especially seen in infants with respiratory distress and may be accompanied by head-bobbing with each breath. It is a sign of moderate to severe respiratory distress.

Tracheal Tug

Downward pull of the trachea, visible in the suprasternal notch during inspiration, often accompanies intercostal or sternal recession. It is a sign of moderate respiratory distress.

Grunting

Grunting is a noise made by neonates and infants during expiration against partially closed vocal cords. It provides increased pressure in the airways in an attempt to prevent airway collapse and improve oxygenation. It is a sign of severe respiratory distress.

Gasping

Gasping is a sign of severe hypoxia and indicates impending respiratory arrest.

Any increase in respiratory effort and/or oxygen requirements warrants a Clinical Review. Any severe recession, grunting or gasping must trigger an escalation to a Rapid Response.

Respiratory deterioration may not always be accompanied with signs of increased respiratory distress. Respiratory deterioration with minimal or no increased effort of breathing may occur in:

- Infants or children who have been in respiratory difficulty for some time and are fatigued (exhaustion is a pre-arrest sign).
- Children with a reduced respiratory drive due to a neurological condition (e.g. raised intracranial pressure), poisoning or over sedation (e.g. post operative opioid administration).
- Children with reduced muscle power due to neuromuscular disease e.g. Muscular Dystrophy, Spinal Muscular Atrophy.

3. Efficacy of breathing

Respiratory efficacy refers to how effectively air is getting into and out of the lungs. Indicators are:
Chest expansion

- Assess if the chest is fully expanding.
- Assess if the rise and fall is equal on both sides.

Air entry

- Auscultate the chest at the level of the nipples, in the axillae or over the upper back. Decreased breath sounds can indicate an overall reduced air entry and poor ventilation.
- Assess air entry for equality in both lungs. Unequal breath sounds can indicate collapse or consolidation to a specific area of the lung. Absent breath sounds on one side (and hyper-resonance to percussion) may indicate a pneumothorax.
- Assess for adventitious (additional) breath sounds. The presence of the following findings should be noted:
  - *Wheeze* heard during expiration is indicative of pulmonary oedema or lower airway obstruction, such as asthma or bronchiolitis.
  - *Crepitations* (crackles or rales) are medium to high pitched crackling or popping sounds heard during inspiration and are indicative of fluid within the lungs. They can be heard in bronchiolitis or pneumonia for example.
  - *Prolonged expiration* indicates lower airway bronchoconstriction, as seen in asthma.

**Helpful tips for auscultation**

- Be systematic when listening to the chest — alternate between right and left sides of the chest to compare upper, middle zone and lower zones (or just upper and lower zones in small infants).
- Ensure a warm stethoscope. A cold instrument can cause an infant to cry making auscultation and respiratory assessment difficult.

Oxygen saturations

- This is a good indicator of the efficacy of breathing.
- Hypoxaemia is defined as oxygen saturations (SpO₂) of less than 95%. Oxygen saturations less than 90% are alarming.
- Pulse oximetry is a valuable tool since cyanosis won’t be detected until SpO₂ is approximately 85%. Cyanosis is a *late and pre arrest sign*. 
Always interpret SpO₂ in context to clinical situation. Oxygen saturations may be normal if the child is receiving high levels of inspired oxygen, even in situations of deterioration.

**ESCALATION RESPONSE TO DECREASED OXYGEN SATURATIONS**

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<table>
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<tr>
<td>95-100%</td>
<td>Normal</td>
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<tr>
<td>90-95%</td>
<td>Clinical Review</td>
</tr>
<tr>
<td>Less than 90%</td>
<td>Rapid Response</td>
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</tbody>
</table>

Some children may still appear ‘happy’ even when oxygenation is poor and can be closer to decompensation than you think.

Helpful tips for monitoring oxygen saturations

- Use age-appropriate pulse oximetry probes to ensure correct fit.
- Conduct continuous pulse oximetry, spot checks are not advocated.
- SpO₂ values are unreliable if they are less than 70%, and in poor perfusion or carbon monoxide intoxication.
- SpO₂ reading may be artificially low if the child is moving or the probe is incorrectly placed - always check the child and the quality of the waveform/amplitude on the pulse oximeter.

4. **Effects of respiratory failure**

Inadequate respiratory function will have an effect on the other systems of the body:

**Cardiovascular status**

Hypoxaemia initially causes tachycardia and, if deterioration continues, bradycardia.

**Neurological status**

Hypoxaemia may cause agitation and restlessness, and later drowsiness and a loss of consciousness. If unsure ask the caregiver how they perceive their child’s behaviour and level of alertness to be.

Bradycardia, central cyanosis and decreasing level of consciousness are late and pre-arrest signs. Escalate to a Rapid Response, ensure the airway is patent, and oxygen is applied.
### Table 3

<table>
<thead>
<tr>
<th>GUIDE TO THE ASSESSMENT OF RESPIRATORY DISTRESS AS PER SPOC</th>
</tr>
</thead>
<tbody>
<tr>
<td>MILD</td>
</tr>
<tr>
<td>---</td>
</tr>
<tr>
<td><strong>Airway</strong></td>
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<tr>
<td></td>
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<tr>
<td><strong>Behaviour &amp; Feeding</strong></td>
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<tr>
<td></td>
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<tr>
<td><strong>Respiratory Rate</strong></td>
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<td></td>
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<tr>
<td><strong>Accessory Muscle Use</strong></td>
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<tr>
<td></td>
</tr>
<tr>
<td><strong>Apnoeic Episodes</strong></td>
</tr>
<tr>
<td><strong>Oxygen</strong></td>
</tr>
<tr>
<td></td>
</tr>
</tbody>
</table>

As a child starts to decompensate they can look deceptively ‘undistressed’ as they tire and breathing becomes shallower.

### For Circulation

Infants and children usually have healthy cardiovascular systems which enables them to compensate well in times of circulatory impairment (e.g. dehydration or hypovolaemia). Tachycardia is the first sign of cardiovascular compensation. Blood pressure is particularly well conserved and will only fall late in the child’s illness, just prior to cardiac arrest.

Recognition of cardiovascular compensation (i.e. tachycardia, poor perfusion, reduced urine output) is important in order to avoid rapid and unexpected deterioration later on. Children may be able to compensate for longer periods than adults but decompensation is rapid.

Circulatory assessment requires a *hands on* approach; pulses need to be palpated for rate and strength, the skin tested for capillary refill, and the hands and feet felt for temperature.
A comprehensive circulatory assessment involves:
1. Heart rate
2. Pulse volume
3. Capillary refill (and skin temperature)
4. Blood pressure
5. Effects of circulatory failure

1. **Heart Rate**

   **RESTING HEART RATE BY AGE AS PER SPOC**

<table>
<thead>
<tr>
<th>Age</th>
<th>Heart Rate (beats per min)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Under 3 months</td>
<td>110-160</td>
</tr>
<tr>
<td>3 -12 months</td>
<td>100-160</td>
</tr>
<tr>
<td>1-4 years</td>
<td>90-140</td>
</tr>
<tr>
<td>5-11 years</td>
<td>80-120</td>
</tr>
<tr>
<td>12 years and over</td>
<td>60-100</td>
</tr>
</tbody>
</table>

   *Table 4*

   - **Tachycardia (or increasing trend)** at rest is an important sign of cardiovascular compensation. Whilst a degree of sinus tachycardia is to be expected in children with fever, or those being treated with sympathomimetic drugs such as salbutamol, it is one of the earliest signs of dehydration or circulatory impairment. In young infants, the sinus rate may increase even as high as 220 beats per minute but if this continues the heart will fatigue. *If the cause of tachycardia is not addressed bradycardia and cardiac arrest will follow.*

   - **Bradycardia** defined as less than 60 beats per minute, or a rapidly falling heart rate, with poor perfusion is a *pre-arrest sign.*

   If the child is unresponsive and breathing is absent escalate to a Rapid Response and commence bag valve mask ventilation and cardiac compressions.

   **Helpful tips for assessing heart rate**

   - Palpation of the pulse and avoidance of automated machines to obtain the heart rate allows assessment of pulse rhythm and strength as well as peripheral temperature.

   - In neonates and young infants, listening to the heart with a stethoscope may be easier than palpating the pulse to accurately ascertain heart rate.

   - Count for a full minute wherever possible as per recommended practice.
2. Pulse Volume

Pulse volume relates to the strength of the peripheral and central pulses. Comparing the volume of the peripheral and central pulses allows assessment of perfusion and cardiac output.

Difficulty or inability to palpate the peripheral pulse at the wrist in the presence of a palpable central pulse may be a sign that the circulation is “shut down”, as compensation for advanced shocked. Very easily palpable or “bounding” pulses may be due to increased cardiac output (such as in toxic shock syndrome or hypercapnia).

Helpful tips for assessing pulse volume

- The peripheral pulses should be palpable in almost all cases unless the infant or child is shocked.
- Central pulses should be felt in the brachial or the femoral artery in under 1yr olds and the femoral or carotid artery in 1yr olds and older.
- Circulation may be inadequate (i.e. weak central pulse) despite hearing a heartbeat or seeing QRS complexes on a cardiac monitor. This is why assessment of pulse volume is important.

![Figure 3: Brachial pulse in an infant](image)

3. Capillary refill time (and skin temperature)

Measurement of the capillary refill time (CRT) and temperature of the hands and feet gives some indication of peripheral perfusion. If the CRT is delayed and the extremities are cool it can indicate poor skin perfusion and may be a sign of shock.

CRT should not be interpreted in isolation and may be abnormally prolonged due to environmental and patient factors other than shock. In cases where it is prolonged due to shock, it will likely be accompanied by pallor and mottling of the skin.
Helpful tips when taking a blood pressure

- Ensure the BP cuff is the correct size and covers at least ⅔ of the upper arm. A cuff that is too small will produce a falsely high reading whilst a cuff too large will produce a falsely low reading.
- Warn the child that the cuff will squeeze and in younger or anxious children demonstrate the cuff inflating on an adult first.
- Young infants may become distressed, so leave until the end of the assessment and gently hold the arm still during the measurement.
- Most automated devices are preset with adult parameters; ensure the neonate/infant or paediatric setting have been selected.
5. Effects of circulatory failure

Inadequate circulation will have an effect on the other systems of the body:

**Respiratory status**

Circulatory inadequacy can lead to raised tissue and blood lactic acid levels (metabolic acidosis) which cause an increase in respiratory rate in an attempt to compensate.

**Neurological status**

Reduced cerebral blood flow can cause agitation or confusion progressing to drowsiness, and, if present, should never be ignored. Some children, however, remain able to talk and interact right up until the point of cardiac arrest, so absence of drowsiness or agitation does not exclude a shocked state.

**Renal status**

Circulatory inadequacy causes a reduction in blood flow to the kidneys which leads to oliguria (decreased urine output) or anuria (no urine output) depending upon duration and severity of circulatory failure.

---

**Hypotension** is a *late and pre-arrest* sign of circulatory failure. Early signs of circulatory failure should be acted on immediately to prevent further deterioration.

**D** For Disability or neurological assessment

Only when A, B and C have been rapidly assessed *and treated*, should neurological function be addressed. A comprehensive neurological assessment involves:

1. Level of consciousness (alertness or arousal)
2. Posture
3. Pupil reaction
4. Effects of neurological failure

**1. Level of consciousness**

The child’s general appearance and behaviour are defining features in detecting altered mental state and/or neurological deterioration. Their level of activity and their interaction and response to the environment and to their caregivers are important factors in the assessment of neurological status. Any reduction in level of alertness or arousal or any increasing agitation or combativeness should be cause for concern and requires appropriate escalation and investigation.
A child’s behaviour should be interpreted with an appreciation of their usual behaviour, routines and level of development. Remember, however, children are often frightened and anxious in unfamiliar surroundings so take this into account during any assessment.

Caregivers know their child best. Ask them how they perceive their child’s behaviour to be and whether they feel there is any change or deterioration. If caregivers are worried take notice.

Neurological Assessment Tools

AVPU

The AVPU tool is commonly used for a rapid assessment of the level of consciousness.\(^2\) It is a simple neurological assessment tool, however, and may be less sensitive in identifying the subtle behavioural changes that the TICLS tool may detect, particularly in the neonate.

<table>
<thead>
<tr>
<th>A</th>
<th>Alert</th>
<th>Normal</th>
</tr>
</thead>
<tbody>
<tr>
<td>V</td>
<td>responds to Voice</td>
<td>Clinical Review</td>
</tr>
<tr>
<td>P</td>
<td>responds to Pain</td>
<td>Rapid Response</td>
</tr>
<tr>
<td>U</td>
<td>Unresponsive</td>
<td>Rapid Response</td>
</tr>
</tbody>
</table>

If there is a deterioration in responsiveness from ‘A’ to ‘V’ look for a cause, discuss with Nurse in Charge and assess whether a Clinical Review is necessary. If the level of consciousness is ‘P’ or ‘U’ call a Rapid Response and obtain an age appropriate Glasgow Coma Scale score.

Helpful tips when using the AVPU tool

- If AVPU is being used as a clinical assessment tool and the child is asleep, you must wake the child to properly assess the neurological status.
- If the child is sleeping but rouses easily to voice and remains awake and alert record this as an ‘A’.
- A score of V on AVPU is warranted if the child is drowsy and not waking fully to stimulation. They may still answer questions and follow commands, but will do so slowly and inattentively.
**Glasgow Coma Scale/ Modified Glasgow Coma Scale**

If a more detailed assessment of neurological function is required the Glasgow Coma Scale (GCS) or a modified Glasgow Coma Scale for under 5 year olds (Child’s Glasgow Coma Scale) should be used. This tool was originally developed and validated for patients with head injuries but has since been adopted for use in the assessment of mental state in any neurological pathology.

Effective assessment with the GCS will enable prompt recognition of deterioration and facilitate potentially lifesaving interventions. *A reduction in the level of consciousness is the most sensitive indicator of neurological deterioration.*

The maximum GCS score is 15 and to score this the child must be fully awake and interactive. A child responding only to Pain is likely to have a GCS score of 8 or less and may not have adequate airway protection reflexes to remain safe without intervention.

*If the GCS score is 8 or less* position the child in the recovery position, provide high flow oxygen via a non-rebreather mask and support the airway with airway opening manoeuvres and adjuncts. Escalate to a Rapid Response immediately.

*Figure 4: Recovery position: Left lateral*
GLASGOW COMA SCALE AND MODIFIED GLASGOW COMA SCALE

<table>
<thead>
<tr>
<th>Glasgow Coma Scale</th>
<th>Modified Glasgow Coma Scale (&lt;5yrs)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Eye opening</strong></td>
<td><strong>Eye opening</strong></td>
</tr>
<tr>
<td>Spontaneously</td>
<td>Spontaneously</td>
</tr>
<tr>
<td>4</td>
<td>4</td>
</tr>
<tr>
<td>To speech</td>
<td>To speech</td>
</tr>
<tr>
<td>3</td>
<td>3</td>
</tr>
<tr>
<td>To pain</td>
<td>To pain</td>
</tr>
<tr>
<td>2</td>
<td>2</td>
</tr>
<tr>
<td>No response</td>
<td>No response</td>
</tr>
<tr>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td><strong>Best verbal response</strong></td>
<td><strong>Best verbal response</strong></td>
</tr>
<tr>
<td>Orientated and converses</td>
<td>Alert; babbles, coos, words to usual ability</td>
</tr>
<tr>
<td>5</td>
<td>5</td>
</tr>
<tr>
<td>Confused and converses</td>
<td>Less than usual words, spontaneous irritable cry</td>
</tr>
<tr>
<td>4</td>
<td>4</td>
</tr>
<tr>
<td>Inappropriate words</td>
<td>Cries only to pain</td>
</tr>
<tr>
<td>3</td>
<td>3</td>
</tr>
<tr>
<td>Incomprehensible sounds</td>
<td>Moans to pain</td>
</tr>
<tr>
<td>2</td>
<td>2</td>
</tr>
<tr>
<td>No response to pain</td>
<td>No response to pain</td>
</tr>
<tr>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td><strong>Best motor response</strong></td>
<td><strong>Best motor response</strong></td>
</tr>
<tr>
<td>Obey verbal command</td>
<td>Spontaneously/obeys verbal command</td>
</tr>
<tr>
<td>6</td>
<td>6</td>
</tr>
<tr>
<td>Localises to pain</td>
<td>Localises to pain/withdraws to touch</td>
</tr>
<tr>
<td>5</td>
<td>5</td>
</tr>
<tr>
<td>Withdraws from pain</td>
<td>Withdraws from pain</td>
</tr>
<tr>
<td>4</td>
<td>4</td>
</tr>
<tr>
<td>Abnormal flexion to pain (decorticate)</td>
<td>Abnormal flexion to pain (decorticate)</td>
</tr>
<tr>
<td>3</td>
<td>3</td>
</tr>
<tr>
<td>Abnormal extension to pain (decerebrate)</td>
<td>Abnormal extension to pain (decerebrate)</td>
</tr>
<tr>
<td>2</td>
<td>2</td>
</tr>
<tr>
<td>No response to pain</td>
<td>No response to pain</td>
</tr>
<tr>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td><strong>Maximum score</strong></td>
<td><strong>Maximum score</strong></td>
</tr>
<tr>
<td>15</td>
<td>15</td>
</tr>
</tbody>
</table>

*Table 6*
2. Posture

Children that are seriously ill will usually be hypotonic (floppy) and less responsive to their surroundings. Abnormal posturing relates to the involuntary flexion or extension of the arms and legs in response to an external stimulus such as pain. It is described as decorticate or decerebrate posturing and indicates severe brain injury and a poor outcome.

- **Decorticate posturing (abnormal flexion)** - the arms are flexed, or bent inward on the chest, the hands are clenched into fists, and the legs extended and feet turned inward.

  ![Decorticate posturing](image)

  *Figure 5: Decorticate posturing*

- **Decerebrate posturing (abnormal extension)** - the head is arched back, the arms are extended by the sides, and the legs are extended, all rotated internally. A hallmark of decerebrate posturing is extended elbows. The patient is rigid, with teeth clenched. The signs may be on one side of the body, on both sides or in the arms only and may be intermittent.

  ![Decerebrate posturing](image)

  *Figure 6: Decerebrate posturing*

The presence of decorticate and decerebrate posturing indicates a severe medical emergency and immediate activation of a Rapid Response is required.

3. Pupils

Pupils should be assessed for size, equality and reaction to light. Pupil reaction to light should be equal and brisk. Any changes in pupil response and/or size should be escalated.

The presence of the following findings should be noted:

- **Pinpoint pupils** may be drug induced e.g. opiate/barbiturate, or may indicate a metabolic disorder.
- **Sluggish pupils** may indicate a raised intracranial pressure. It can also occur post seizure and in situations of drug overdose.

- **Asymmetrical pupils** may imply a space occupying lesion in the brain (e.g. intracranial haemorrhage from a head injury).

- **Changing pupil sizes** may be present during ongoing seizures, even in the absence of tonic-clonic movements.

- **Fixed dilated pupils** may indicate **significantly** raised intracranial pressure. They may also be present in severe hypoxia, hypothermia, post anticholinergic drugs (atropine), during/post seizure, or a late sign of barbiturate poisoning.

  The sudden appearance of fixed and dilated pupils is a neurological emergency that requires the *immediate activation of a Rapid Response*.

4. **Effects of neurological failure**

Inadequate neurological function will have an effect on the other systems of the body:

**Respiratory status**

Raised intracranial pressure or other causes of brainstem dysfunction can cause several abnormal breathing patterns including hyperventilation, irregular respirations, and apnoea.

**Cardiovascular status**

Cushing’s Triad is a classic sign of a raised intracranial pressure. It involves a combination of hypertension, bradycardia and irregular respiration. It is a late warning sign and once present, brainstem herniation is likely.

Changes in vital signs are late warning signs of neurological deterioration. Cushing’s Triad is a medical emergency and requires a Rapid Response.

**E For Exposure**

A thorough assessment of ‘exposure’ should involve an *all over body check* that includes:

- Temperature (axillary is the recommended method)

- Examination for rashes, injury, bleeding or excess drain losses, wound infections, and inflammation around any intravenous or drain sites
• Examination for abdominal distension or tenderness

A thorough assessment must involve appropriate exposure of the skin, removal of bedclothes and possible repositioning.

**Temperature**

Neonates and young infants lose heat more rapidly than adults because they have relatively large heads and greater surface area to body mass ratio, and are less able to compensate for environmental temperature drops.

Fever is very common in childhood, with the majority being caused by mild viral infections that require no treatment. However, any new onset of fevers more than 38.5°C should prompt investigation for the presence of a bacterial infection that does require treatment. It is important to remember that *serious infection may not always present with fever*, particularly in neonates, young infants or immunocompromised children. Hypothermia should be regarded as a warning sign in any child that appears unwell.

*Infants under 3 months old are a high risk group* – any temperature greater than 38.0°C should have a full septic screen and appropriate antibiotics immediately.

Research is now recognising that a fever may be beneficial to the immune response and routine administration of anti-pyretics is not advocated (with the exception of extreme fever of 41.0°C or more as this may be harmful).

Current recommendations for fever management are to dress the child in light clothing, enough to avoid shivering, and administer anti-pyretics only if required as a comfort measure. *Tepid sponging (i.e. sponging with cool water) and the use of fans are not recommended practices.*

**ESCALATION RESPONSE TO TEMPERATURE**

<table>
<thead>
<tr>
<th>Temperature less than 35.5°C</th>
<th>Clinical Review</th>
</tr>
</thead>
<tbody>
<tr>
<td>New onset of fever 38.5°C or more</td>
<td>Clinical Review</td>
</tr>
<tr>
<td>Temperature less than 34.5°C</td>
<td>Rapid Response</td>
</tr>
<tr>
<td>Temperature 41°C or more</td>
<td>Rapid Response</td>
</tr>
</tbody>
</table>

For specific information on assessment and management of fever refer to the Clinical Practice Guideline *PD2010_063 Infants and children: Acute Management of Fever*
Rashes

Rashes can develop from any number of illnesses; infections, allergies, reactions to medication, chemical irritants or insect stings. The child’s appearance and behaviour combined with a thorough history can often determine the type of rash present and whether it is cause for serious concern. If the rash is accompanied by any fever, irritability or lethargy, loss of appetite, or physiological changes (i.e. changes in vital signs) further investigations needs to be carried out. Rashes of particular note include:

- **Petechial (small red/purple spots) or purpuric (larger red/purple areas) rashes** are examples of a non-blanching rash (i.e. does not lose colour when pressed with a glass or finger). They often occur together and although many children can have non-blanching rashes that are not serious they can indicate severe infection, such as meningitis or septicaemia, or leukaemia and must be investigated.

  Petechial or purpuric rashes may initially present as non-specific blanching rashes becoming non-blanching as the disease progresses. **Assess frequently.**

- **Urticarial rash** (also known as hives) suggests an allergic reaction and may be seen in anaphylaxis, although anaphylaxis may occur without any obvious skin rash. An allergic rash is often inflamed and itchy and can come and go.

- **Erythema** simply means ‘redness’ and is the most common type of mild viral rash, and often innocent. If, however, it is accompanied by fever, diarrhoea and unwellness it may be associated with Toxic Shock Syndrome.

**F For Fluids**

**Fluids in**

Infants and young children have higher metabolic rates with relatively high insensible fluid losses and are vulnerable to increased fluid loss from any cause (e.g. diarrhoea). Any interruption in normal fluid intake can quickly lead to dehydration. Daily fluid requirements are calculated on a weight basis; however, this should be regularly assessed and adjusted according to the child’s fluid and electrolyte status.

Any unwell child receiving intravenous fluids is at risk of water retention and *iatrogenic hyponatraemia* due to over hydration. Fluids with low sodium content i.e. less than 0.45% Sodium Chloride must not be used (refer to Chapter 6 p 128).
Never leave a child without an adequate fluid intake. Ensure IV cannulae are restited as soon as possible.

**Fluids out**

**Urine output**

The following is a guide to the minimum expected urine output:

- Neonates & Children less than 30kg = 1mL/kg/hr (or 4 wet nappies in 24 hrs)
- Children 30-60kg = 0.5mL/kg/hr
- Children more than 60kg = 30mL/hr

If you notice a reduction in urine output or unusually dry nappies initiate a Clinical Review. Ask the caregiver if they think their child’s urine output is less than usual.

An excessive urine output (polyuria) defined as more than 3 mL/kg/hr may also be abnormal and should be investigated in all cases (refer to Chapter 6 p 116).

**Blood loss**

The amount of blood per body weight is larger in infants than in adults but the actual volume is small. For example, a blood loss of 75mL in an adult may not be considered significant but it represents ¼ of the circulating blood volume of a neonate and will result in shock.

**AVERAGE BLOOD VOLUME**

<table>
<thead>
<tr>
<th>Age</th>
<th>mL/kg</th>
<th>Total blood volume</th>
</tr>
</thead>
<tbody>
<tr>
<td>Neonate (approx 3.5kg)</td>
<td>85mL/kg</td>
<td>300mL</td>
</tr>
<tr>
<td>Toddler (approx 12kg)</td>
<td>80mL/kg</td>
<td>960mL</td>
</tr>
<tr>
<td>Young Child (approx 20kg)</td>
<td>80mL/kg</td>
<td>1600mL</td>
</tr>
<tr>
<td>Adult (approx 70kg)</td>
<td>70mL/kg</td>
<td>5000mL</td>
</tr>
</tbody>
</table>

Even relatively small volumes of blood loss in an infant may represent a significant proportion of circulating blood volume and can result in significant shock.

**Fluid balance status**

An accurate fluid balance is essential, particularly in infants due to their increased susceptibility to dehydration. Ensure caregivers are aware of the importance of monitoring the input and output in their child, as they are often the ones filling up drink bottles and changing nappies.
The only *objective* measure of acute changes in hydration is body weight\(^{15}\) and this should be established as soon as possible and compared to a recent weight where available (ask the caregiver, or check the Perinatal Health Record (Blue Book) or hospital medical records). *Ensure clothes and nappies are removed in infants.*

It is normal for a newborn baby to lose weight in the first 2 - 3 days of life and to regain and exceed birth weight by 7 – 10 days of life. A rule of thumb for weight gain thereafter is roughly 200g per week, with doubling of birth weight by approximately 6 months.

**Dehydration**

The degree of dehydration is often reflected in the behaviour. An active and alert child will likely only be mildly dehydrated. A more severely dehydrated child will appear pale and lethargic, disinterested and irritable. The more dehydrated the child is the more drowsy they tend to become.

Table 8 categorises the clinical signs and symptoms of dehydration and allows for an estimation of fluid deficit and calculation of appropriate fluid replacement therapy.

### CATEGORISATION OF DEHYDRATION

<table>
<thead>
<tr>
<th>Description of Dehydration</th>
<th>Dehydration (% body weight)</th>
<th>Signs and Symptoms</th>
</tr>
</thead>
</table>
| Mild                       | 3%                          | Dry mucous membranes  
Mild tachycardia  
Reduced urine output  
Thirst |
| Moderate                   | 5%                          | Above signs plus:  
Tachycardia  
Lethargy  
Reduced skin turgor  
Sunken eyes/Sunken fontanelle |
| Severe (without shock)     | 7.5%                        | Above signs plus:  
Poor perfusion - mottled, cool extremities,  
prolonged capillary refill  
Reduced level of consciousness |
| Severe (with shock)        | 10%                         | Above signs plus:  
Absent or thready peripheral pulses with marked tachycardia |

*Table 8*

The most severe cases of dehydration (10% or more) will result in shock. Dehydration *without shock* should be classified as mild, moderate and severe as per table.
Lethargy, irritability, reduced level of consciousness

Lethargy, irritability, reduced level of consciousness

Individual clinical signs of dehydration are unreliable but together provide a reasonable estimation of fluid loss.\textsuperscript{16,17}

Helpful tips for assessing clinical signs of dehydration

- Sunken fontanelle: if the child is crying it will bulge.
- Sunken eyes: difficult to determine, particularly on first contact. Ask the caregiver.
- Dry tongue and lips: be aware oxygen therapy can contribute to dryness.
- Reduced skin turgor: gently pinch the skin on the abdomen or forearm and release. The skin should recoil immediately (less than 1 second) and the more prolonged it is the greater the dehydration. This is unreliable in both overweight and malnourished children.
- Signs of poor perfusion: pale, mottling, prolonged capillary refill, cool peripheries can be affected by environmental temperature.
- Urine output: nappies should be weighed pre and post application to gain an accurate urine output. Difficulties arise when diarrhoea is present. If the diarrhoea is watery it may still be beneficial to weigh nappies. Be aware that dehydration may not always be accompanied by a reduced urine output e.g. diabetes and certain renal disorders (refer to Chapter 6 p 117).
**Fluid overload**

Fluid overload is common in children in hospital. Oedema is the most striking feature of fluid overload usually seen as ‘puffiness’ in the face and eyelids and swelling in the dependant areas (e.g. limbs).

Further assessment may reveal:

- Raised jugular venous pressure: difficult to ascertain in young children
- Hypertension: an important feature of excessive intravascular volume
- Tachypnoea: possibly with accompanying crackles if pulmonary oedema is present

**G For Glucose**

**Hypoglycaemia**

A normal blood glucose level (BGL) in children is 3 to 5mmol/L. Hypoglycaemia (i.e. a BGL less than 3 mmol/L) is more common in infants and young children than in adults, and may be seen in any child with serious illness. It is not usually necessary to measure BGL in children who are alert and orientated, however, any history of a reduced calorie intake, alcohol or drug ingestion, or altered mental state (e.g. lethargy, confusion, delirium, reduced level of consciousness) warrants the measurement of a blood glucose level.

**Signs and symptoms of hypoglycaemia include:**

Hypoglycaemia is often accompanied by signs and symptoms of autonomic (adrenergic) activation and/ or neurological dysfunction (neuroglycopaenia).

**Autonomic signs and symptoms include:**

- Trembling or ‘jitteriness’ in infants
- Pounding heart
- Cold sweatiness
- Pallor

**Neurological signs and symptoms include:**

- Difficulty concentrating
- Blurred/double vision
- Difficulty hearing
- Slurred speech
- Poor judgment and confusion
- Dizziness and unsteady gait
- Irritability/inconsolability
- Erratic behaviour
- Loss of consciousness/seizure

**ESCALATION RESPONSE TO HYPOGLYCAEMIA**

<table>
<thead>
<tr>
<th>BGL 2 to 3 mmol/L</th>
<th>Clinical Review</th>
</tr>
</thead>
<tbody>
<tr>
<td>BGL less than 2 mmol/L</td>
<td>Rapid Response</td>
</tr>
</tbody>
</table>

**Hyperglycaemia**

The recognition of hyperglycaemia is of equal importance, as it could result in potentially life-threatening complications, including a diabetic coma. Hyperglycaemia may be seen in situations of extreme stress, or in newly diagnosed or uncontrolled diabetes mellitus. Left untreated, hyperglycaemia can lead to diabetic ketoacidosis, and eventual coma. Any BGL >10 mmol/L must be investigated promptly.

**Signs and symptoms of hyperglycaemia include:**
- Increased urine output that can lead to dehydration
- Thirst and increased appetite
- Weight loss and fatigue (which may manifest as unusual irritability in infants)

Always check the blood glucose in any lethargic or unwell infant or child, even in the absence of specific symptoms of hypo- and hyperglycaemia.

**GENERAL CONSIDERATIONS**

At this stage, as you have worked through the priorities of the ABCDEFG assessment, you can focus on the potential cause of deterioration, review your assessment findings, take a more detailed history and review the medication charts and any test results available. Remember:

‘Assessment is not a singular event, but a continuous and dynamic process’

As part of your assessment you will need to decide how much deterioration has occurred in the child’s condition in comparison with their usual state and level of function. This can be established by
reviewing the previous history in the medical record and observation chart, asking the parent or caregiver, or checking with on-site staff. Consider the following:

**HIGH RISK INFANTS AND CHILDREN**

Early escalation should be considered for infants and children that are at higher risk of deterioration.

<table>
<thead>
<tr>
<th>High risk infants and children</th>
</tr>
</thead>
<tbody>
<tr>
<td>Under 3 months old</td>
</tr>
<tr>
<td>Chronic or complex conditions</td>
</tr>
<tr>
<td>Post operative</td>
</tr>
<tr>
<td>Pre-existing respiratory or cardiac conditions</td>
</tr>
<tr>
<td>Opioid infusions</td>
</tr>
</tbody>
</table>

*Table 9*

**DISEASE DYNAMIC**

This should include consideration of how long the child has been unwell; what has occurred prior to and since presentation. Consideration of other recent illness in family members may also be important.

**CAREGIVER CONCERN**

Do the caregivers think their child is improving or getting worse? How does the child’s condition and level of responsiveness differ from “normal” for their child?

**CO-MORBIDITIES**

Children with co-morbidities may not follow a predictable course, and are susceptible to complications from standard hospital therapies, e.g. aspiration pneumonia.

Children with chronic conditions are particularly vulnerable to deterioration as abnormal parameters are accepted as ‘normal’ for their condition. *Never assume abnormal observations reflect the child’s normal state.*

**IMMUNOCOMPROMISED**

Immunocompromised children (including those on oral steroids or chemotherapy) are susceptible to opportunistic infections and may be slow to show the normal signs of sepsis.
‘CHILD AT RISK’

If there are concerns (actual or suspected) regarding a child being:

- A victim of violence
- Sexually assaulted
- Neglected
- Non-accidental injury
- Exposed to domestic violence
- At risk of harm (however defined)

Practitioners are advised to use the NSW Mandatory Reporter Guide in all cases where there is a reasonable suspicion of inflicted injury. The guide is available at: www.community.nsw.gov.au/kts/guidelines/documents/mandatory_reporter_guide.pdf.

PAIN

‘Optimal pain management is the right of all patients and the responsibility of all health professionals.’¹⁸

Pain causes increases in respiratory rate, heart rate, blood pressure and oxygen consumption. It can cause oxygen saturations to drop, blood sugar to increase and gastric and gut motility to decrease. It can cause muscle tension and fatigue.

Pain should be assessed and excluded as the primary cause of abnormal observations. The aim of pain management is to identify pain quickly and to relieve it, or better still, to anticipate it and prevent or minimise it. Relieving pain will not only increase comfort, but it will reduce stress and enhance healing.¹⁹

### ESCALATION RESPONSE TO INCREASING PAIN

<table>
<thead>
<tr>
<th>Mild (pain score 1-3)</th>
<th>Increase observations</th>
</tr>
</thead>
<tbody>
<tr>
<td>Moderate (pain score 4-6)</td>
<td>Clinical Review</td>
</tr>
<tr>
<td>Severe (pain score 7-10)</td>
<td>Clinical Review</td>
</tr>
</tbody>
</table>

Increasing pain or pain unresponsive to previous measures should be cause for serious concern and should prompt a Clinical Review or Rapid Response.
Assessment of Pain

The following pain assessment tools (or Pain Rating Scales) have been designed for repeated use to assist healthcare practitioners in the assessment of pain.

Paediatric Pain Assessment Tools

Age-appropriate pain scores should be used to assess and monitor pain in infants and children. The following are a range of pain assessment tools, specific to age.

**Linear Scale**

When asking the older child about their pain do not ask them what their pain is out of 10. Get them to point or draw on the linear scale. In some instances it is more appropriate to use a linear scale without the numbers attached.

**Faces Pain Scale – Revised**

*Use the term “hurt” or “pain” whichever is appropriate for the age of the child.* Show them the faces, explain it to them, and then ask them to point to the face that shows how much they hurt or how much pain they have.

*The scale is intended to measure how children feel inside, not how their face looks.* Score the chosen face 0, 2, 4, 6, 8, 10, counting left to right, so ‘0’ = “no pain” and ‘10’ = “the worst pain”. Do not use words like “happy” and “sad”.

![Linear Scale](image)

![Faces Pain Scale](image)
**FLACC Pain Scale (behaviour assessment)**

This tool is useful in infants and in children who are not able to verbalise the presence or intensity of pain e.g. cognitively impaired children. When children cannot speak or comprehend, behaviour is the primary means by which they communicate their pain.

Observations should occur over at least a minute and if the child is asleep, touch the body to assess for tenseness and tone.

Suggested age is from 2 months to 7 years of age

<table>
<thead>
<tr>
<th>FLACC PAIN SCALE</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Categories</strong></td>
</tr>
<tr>
<td></td>
</tr>
<tr>
<td><strong>Face</strong></td>
</tr>
<tr>
<td><strong>Legs</strong></td>
</tr>
<tr>
<td><strong>Activity</strong></td>
</tr>
<tr>
<td><strong>Cry</strong></td>
</tr>
<tr>
<td><strong>Consolability</strong></td>
</tr>
</tbody>
</table>

Each of the five categories: (F) Face; (L) legs; (A) Activity; (C) Cry; (C) consolability; is scored from 0-2 which results in a total pain score between 0 and 10

*Table 10*

**Neonatal Pain Assessment Tools**

Neonatal assessment of pain is both complex and challenging and a number of tools have been developed as an attempt to address such complexity. To improve validity and reliability tools need to be multidimensional, incorporating behavioural, physiological and contextual factors.

Examples of neonatal pain assessment tools include Pain Assessment Tool (PAT), Premature Infant Pain Profile (PIPP), Crying, Requires O₂, Increased vital signs, Expression and Sleeplessness (CRIES) and Neonatal Infant Pain Scale (NIPS).
The Neonatal/Infant Pain Scale (NIPS) has been incorporated into the Under 3 months Paediatric Emergency Department Observation Chart and is the recommended NSW neonatal pain assessment tool within the emergency department. The NIPS has been shown to be a reliable, valid, clinically feasible and easy-to-use tool for assessing pain in both term and preterm neonates. 24 25 26

NIPS PAIN SCALE

<table>
<thead>
<tr>
<th>Assessment</th>
<th>Score = 0</th>
<th>Score = 1</th>
<th>Score = 2</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Facial expression</strong></td>
<td>Relaxed muscles (Restful face, neutral expression)</td>
<td>Grimace (Tight facial muscles; furrowed brow, chin, jaw)</td>
<td></td>
</tr>
<tr>
<td><strong>Cry</strong></td>
<td>No Cry (Quiet, not crying)</td>
<td>Whimper (Mild moaning, intermittent)</td>
<td>Vigorous Cry (Loud scream; rising, shrill, continuous)</td>
</tr>
<tr>
<td><strong>Breathing patterns</strong></td>
<td>Relaxed (Usual pattern for this infant)</td>
<td>Change in breathing (Indrawing, irregular, faster than usual; gagging; breath holding)</td>
<td></td>
</tr>
<tr>
<td><strong>Arms</strong></td>
<td>Relaxed/Restrained (No muscular rigidity; occasional random movements of arms)</td>
<td>Flexed/Extended (Tense, straight arms; rigid and/or rapid extension, flexion)</td>
<td></td>
</tr>
<tr>
<td><strong>Legs</strong></td>
<td>Relaxed/Restrained (No muscular rigidity; occasional random leg movement)</td>
<td>Flexed/Extended (Tense, straight legs; rigid and/or rapid extension, flexion)</td>
<td></td>
</tr>
<tr>
<td><strong>State of arousal</strong></td>
<td>Sleeping/Awake (Quiet, peaceful, sleeping or alert and settled)</td>
<td>Fussy (Alert, restless, and thrashing)</td>
<td></td>
</tr>
</tbody>
</table>

NIPS interpretation – add the scores from each of the 6 assessments to a total of between 0 – 7
0 = No pain ≤ 2 = Mild discomfort 2 - 4 = Mild to moderate pain 4 - 7 = Moderate to severe pain

Table 11

A stand-alone score, unless very high, is less significant than a series of scores. This is because trends over time, response to painful procedures and response to pain relieving measures, can be observed better with multiple scores. The clinician must be mindful that a falsely low score may be seen in an infant who is too ill to respond, is neurologically impaired or has received a paralysing agent. Clinical judgement and frequent reassessment are very important aspects of assessing pain in neonates.
### When to Worry Early Warning Signs

<table>
<thead>
<tr>
<th>Category</th>
<th>Signs</th>
</tr>
</thead>
<tbody>
<tr>
<td>Airway</td>
<td>- Partial obstruction&lt;br&gt;- Stridor at rest</td>
</tr>
<tr>
<td>Breathing</td>
<td>- Moderate tachypnoea and/or increasing trend&lt;br&gt;- Moderate recession, head bobbing, nasal flaring, tracheal tug&lt;br&gt;- Mild hypoxaemia 90-95% corrected by $O_2$&lt;br&gt;- Increasing oxygen requirements&lt;br&gt;- Difficulty feeding&lt;br&gt;- Difficulty talking</td>
</tr>
<tr>
<td>Circulation</td>
<td>- Moderate tachycardia and/or increasing trend&lt;br&gt;- Pallor, Mottled&lt;br&gt;- Cool extremities&lt;br&gt;- Capillary refill 3 secs or more</td>
</tr>
<tr>
<td>Disability</td>
<td>- Restlessness, irritability/agitation, inconsolability&lt;br&gt;- Poor response to environment, responds only to Voice&lt;br&gt;- GCS/Modified (child) GCS score of less than 15&lt;br&gt;- Any seizure&lt;br&gt;- New, increasing or uncontrolled pain</td>
</tr>
<tr>
<td>Exposure</td>
<td>- New onset of fever more than 38.5 °C&lt;br&gt;- Hypothermia less than 35.5 °C</td>
</tr>
<tr>
<td>Fluids</td>
<td>- Greater than expected fluid loss&lt;br&gt;- Reduced urine output less than 1mL/kg/hr (or less than 0.5mL/kg/hr in older child)</td>
</tr>
<tr>
<td>Glucose</td>
<td>- BGL 2 to 3 mmol/L&lt;br&gt;- BGL more than 10 mmol/L</td>
</tr>
</tbody>
</table>

*Table 12*
<table>
<thead>
<tr>
<th>When to Worry Late Warning Signs</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Airway</strong></td>
</tr>
<tr>
<td>▪ Imminent airway obstruction</td>
</tr>
<tr>
<td>▪ New onset of stridor</td>
</tr>
<tr>
<td><strong>Breathing</strong></td>
</tr>
<tr>
<td>▪ Marked tachypnoea and/or increasing trend</td>
</tr>
<tr>
<td>▪ Decreasing respiratory rate (EXHAUSTION)</td>
</tr>
<tr>
<td>▪ Severe recession, grunting, gasping, cyanosis</td>
</tr>
<tr>
<td>▪ Absent breath sounds (silent chest)</td>
</tr>
<tr>
<td>▪ Hypoxaemia less than 90% in any amount of O₂</td>
</tr>
<tr>
<td>▪ Apnoeas</td>
</tr>
<tr>
<td>▪ Unable to feed</td>
</tr>
<tr>
<td>▪ Unable to talk, difficulty crying</td>
</tr>
<tr>
<td><strong>Circulation</strong></td>
</tr>
<tr>
<td>▪ Marked tachycardia or decreasing heart rate</td>
</tr>
<tr>
<td>▪ Any bradycardic episodes</td>
</tr>
<tr>
<td>▪ Weak or absent peripheral pulses</td>
</tr>
<tr>
<td>▪ Extreme pallor</td>
</tr>
<tr>
<td>▪ Cold extremities</td>
</tr>
<tr>
<td>▪ Hypotension</td>
</tr>
<tr>
<td><strong>Disability</strong></td>
</tr>
<tr>
<td>▪ Drowsiness, hypotonia or confusion</td>
</tr>
<tr>
<td>▪ Responds only to Pain/Unresponsive</td>
</tr>
<tr>
<td>▪ Sudden decrease in LOC of 2 or more GCS points</td>
</tr>
<tr>
<td>▪ New or prolonged seizure</td>
</tr>
<tr>
<td><strong>Exposure</strong></td>
</tr>
<tr>
<td>▪ Fever 41.0 °C or more</td>
</tr>
<tr>
<td>▪ Hypothermia less than 34.5 °C</td>
</tr>
<tr>
<td><strong>Fluids</strong></td>
</tr>
<tr>
<td>▪ Significant/uncontrolled bleeding or fluid loss</td>
</tr>
<tr>
<td><strong>Glucose</strong></td>
</tr>
<tr>
<td>▪ BGL less than 2 mmol/L</td>
</tr>
</tbody>
</table>

*Table 13*
## SUMMARY OF THE DETECT PROCESS

<table>
<thead>
<tr>
<th>D</th>
<th>Detect deterioration</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>• Be systematic - use first impressions and the ABCDEFG algorithm to assess the child</td>
</tr>
<tr>
<td></td>
<td>• Involve the caregivers in the assessment process</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>E</th>
<th>Evaluate your findings</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>• Use your knowledge of late and early warning signs to evaluate if the child is deteriorating</td>
</tr>
<tr>
<td></td>
<td>• Check if there is an adverse trend in the child’s observations</td>
</tr>
<tr>
<td></td>
<td>• Do the caregivers feel their child is getting worse?</td>
</tr>
<tr>
<td></td>
<td>• Consider the diagnosis, any ‘at risk’ factors and the child’s normal observations</td>
</tr>
<tr>
<td></td>
<td>• Discuss with Nurse in Charge</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>T</th>
<th>Treatment</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>• Provide initial treatment.</td>
</tr>
<tr>
<td></td>
<td>• Continue observations at an appropriate frequency</td>
</tr>
<tr>
<td></td>
<td>• Work within your scope of practice, ask for the assistance of senior staff if necessary</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>E</th>
<th>Escalate your concerns</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>• Escalate to a Clinical Review or Rapid Response as per local CERS</td>
</tr>
<tr>
<td></td>
<td>• Stay with the child</td>
</tr>
<tr>
<td></td>
<td>• Be familiar with the NSW Health Policy Directive PD2011_077 Recognition and Management of Patients who are Clinically Deteriorating.</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>C</th>
<th>Communicating in.....</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>• Use the structured communication tool ISBAR</td>
</tr>
<tr>
<td></td>
<td>• Listen to other members of the team and utilise their knowledge and experience</td>
</tr>
<tr>
<td></td>
<td>• Document your assessment findings, treatment, investigations in the clinical records</td>
</tr>
<tr>
<td></td>
<td>• Never leave the child without a priority management and review plan</td>
</tr>
<tr>
<td></td>
<td>• Use appropriate language when providing explanations to the child and caregiver</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>T</th>
<th>Teams</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>• Provide leadership, co-ordinate activities, communicate clearly, use authority appropriately</td>
</tr>
<tr>
<td></td>
<td>• Assign roles according to ability and scope of practice and ensure roles are understood</td>
</tr>
<tr>
<td></td>
<td>• Adopt effective strategies for communicating in teams</td>
</tr>
<tr>
<td></td>
<td>• Identify any other teams or professionals that may need to be involved</td>
</tr>
</tbody>
</table>

Table 14
POLICY DIRECTIVES AND CLINICAL RESOURCES

PD2011_077  Recognition and Management of a Patient who is Clinically Deteriorating
   Author Branch: Clinical Safety, Quality and Governance

PD2011_038  Paediatric Clinical Practice Guideline Recognition of a Sick Baby or Child in the
   Emergency Department

PD2010_063  Paediatric Clinical Practice Guideline Infants and Children: Acute Management of
   Fever

All Paediatric Clinical Practice Guidelines (CPG’s) can be accessed via the NSW Health Intranet
website:
   – Go to Quick Links: Child Health Networks
   – Go to Links & Resources: NSW Health Paediatric Clinical Practice Guidelines

or via the NSW Health Internet website (for ‘off site’ access):
REFERENCES


This chapter emphasises the importance of non-technical skills, such as communication and teamwork, in the quality and safety of the care you deliver to your patient. It aims to equip you with effective tools for communication, both verbal and written, and outline ways of improving teamwork, situation awareness, task management and decision making.

THE IMPORTANCE OF NON TECHNICAL SKILLS

Now that you have the knowledge base of *When to Worry* and are familiar with the ABCDEFG algorithm you need to combine these technical skills with an additional set of skills known as non-technical skills. These non-technical skills can be defined as a set of behaviour markers that encompass both interpersonal skills, such as communication, teamwork and leadership, and cognitive skills, such as situation awareness and decision making.¹

Non-technical skills are often referred to as Crisis Resource Management (CRM), whose origins lie within the aviation industry. After analysing in-flight crises, the aviation industry discovered that 67% of errors occurred due to deficiencies in communication.² These findings resulted in the aviation (and space) industry developing mandatory annual training in Crew Resource Management (CRM) and flight simulation, where trainees were required to display cognitive and behavioural skills, such as teamwork and communication, as well as technical skills. Safety and reliability improved as a result and this method of training became recognised as highly effective in reducing human error.

Emphasis on non-technical skills in healthcare has been slow to evolve despite the strong evidence that supports their importance, and there remains a high incidence of medical errors as a result of human factors.² In 1999, the Institute of Medicine issued a report entitled *To Err is Human: Building a Safer Health System*, which illustrated that 70% of mistakes in medicine are not from gaps in medical knowledge but rather from human errors, such as lack of teamwork and effective communication.²

Data from adverse events in anaesthetics has shown that non-technical skills play a key role in patient safety.¹ Clinicians need to start focusing on enhancing their skills in communication, teamwork, leadership, situation awareness and decision making, and ensure they become an integral part of practice in order to promote overall good practice and reduce the likelihood of ‘human errors’.
Caring for patients in hospital is a team activity, and communication, both verbal and written, is an integral part of this teamwork. Healthcare practitioners can be highly educated and superbly skilled, but if communication breaks down, so does patient care.

Investigations into incidents of sub-optimal patient care have frequently shown that poor communication is a key feature. ‘Of all human errors, suboptimal communication is the number one issue.’ This includes poor written documentation, non reporting of negative trends in observations to the appropriate health care professional, incomplete or inadequate handovers between staff, as well as miscommunication in crisis situations.

“If there were one aspect of health care delivery an organization could work on that would have the greatest impact on patient safety, it would be improving the effectiveness of communication on all levels-written, oral, electronic.”

### Table 1

<table>
<thead>
<tr>
<th>Category</th>
<th>Element</th>
</tr>
</thead>
<tbody>
<tr>
<td>Task management</td>
<td>Planning and preparation</td>
</tr>
<tr>
<td></td>
<td>Prioritisation</td>
</tr>
<tr>
<td></td>
<td>Providing and maintaining standards</td>
</tr>
<tr>
<td></td>
<td>Identifying and utilising resources</td>
</tr>
<tr>
<td>Teamwork</td>
<td>Co-ordinates activities with team members</td>
</tr>
<tr>
<td></td>
<td>Information exchange</td>
</tr>
<tr>
<td></td>
<td>Use of authority and assertiveness</td>
</tr>
<tr>
<td></td>
<td>Assessment of capabilities of team and self</td>
</tr>
<tr>
<td></td>
<td>Supporting others</td>
</tr>
<tr>
<td>Situation Awareness</td>
<td>Gathering information</td>
</tr>
<tr>
<td></td>
<td>Understanding and recognition</td>
</tr>
<tr>
<td></td>
<td>Anticipation</td>
</tr>
<tr>
<td>Decision Making</td>
<td>Identifying</td>
</tr>
<tr>
<td></td>
<td>Balancing risks and selecting options</td>
</tr>
<tr>
<td></td>
<td>Re-evaluation</td>
</tr>
</tbody>
</table>

**THE IMPORTANCE OF COMMUNICATION AND TEAMWORK**

Caring for patients in hospital is a team activity, and communication, both verbal and written, is an integral part of this teamwork. Healthcare practitioners can be highly educated and superbly skilled, but if communication breaks down, so does patient care.

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“If there were one aspect of health care delivery an organization could work on that would have the greatest impact on patient safety, it would be improving the effectiveness of communication on all levels-written, oral, electronic.”

C h a p t e r  T w o  –  C o m m u n i c a t i o n  a n d  T e a m w o r k  |  P a g e  43
BARRIERS TO EFFECTIVE COMMUNICATION AND TEAMWORK

INCREASING WORKLOAD

Stress and fatigue are obvious factors that can disrupt effective communication and teamwork. Modern healthcare facilities are seeing an increase in the number of patients, an increasing acuity and complexity of presenting conditions, decreasing lengths of stay; and advancing technology and treatments that are not always supported with adequate training.

FRAGMENTED PRACTICES

Modern healthcare teams are becoming increasingly fragmented into sub-specialties, with multiple clinicians involved in an individual patient's care. Shift times for the different professional designations aren’t co-ordinated. Rotating medical staff and an increasing reliance on casual staff causes disruption to team building, in addition to a lack of familiarity with hospital internal system and resources. Educational activities are largely kept at ward or department level, with little opportunity for developing inter-departmental communication and collaboration.

LACK OF TRAINING IN COMMUNICATION STRATEGIES

Undergraduates learn about communicating with patients during their course but often don’t learn how to communicate with colleagues, or as part of a team, or in time-pressured situations. Skills in how to communicate in a crisis situation, how to speak up to a senior colleague, how to exchange information, and how to co-ordinate activities with team members are not an integral part of undergraduate, or even post graduate, training. Effective communication in a stressful, chaotic environment requires skills that need to be learned and practised.

TRADITIONAL HIERARCHICAL RELATIONSHIPS

The hospital environment operates as a hierarchical system and, while this facilitates activities such as decision making, it can also act as a significant barrier to communication. Less experienced staff may often feel intimidated by more experienced colleagues and reluctant to ask for assistance or speak up when they identify an error.

DIFFERING PERCEPTIONS AND LANGUAGE

Healthcare workers are predominantly trained within their own disciplines, or ‘silos’. Communicating and working with other disciplines often does not occur until graduates are already looking after patients. This ‘separatism’ continues on into post graduate education, contributing to a lack of understanding, insight and mutual respect into each other’s roles and responsibilities.
COMMUNICATING IN TEAMS

Effective verbal communication is a two way process, relying on the skills of both the sender and receiver:

- The sender must use language that is clear and concise, and convey only the essential information. Excessive or irrelevant details can confuse and detract from the important issues.
- The receiver must dedicate time to actively listen to the sender and then reflect back their interpretation of the information to acknowledge accurate receipt and comprehension. Mutual respect must be present.

STRUCTURED COMMUNICATION

A systematic approach is an important feature of effective communication, allowing information to be succinct and conveyed in order of priority. Structured communication has been promoted by the World Health Organisation (WHO) and the Australian Commission for Safety and Quality in Healthcare as a means of improving patient safety. 6

DETECT Junior promotes ISBAR, a standardised structured communication tool that has been introduced into the healthcare setting over recent years to improve communication between medical, nursing, allied health and other healthcare staff. The strategy has been widely promoted for use in transferring care of patients and can be adopted for face to face or phone communication, providing a simple but effective way of prioritising information when communicating about a patient.

An ISBAR app was jointly developed by SA Health and NSW Health which can be downloaded from the iTunes store here: https://itunes.apple.com/au/app/isbar/id465890292?mt=8.

On 1 July 2011, the Australian Commission on Safety in Quality in Health Care launched the Clinical Communications programme. The programme focuses on the key areas of clinical communication known to influence quality and safety outcomes throughout the patient journey. The Clinical Handover program was amongst the work produced to improve handover communication across a range of healthcare settings.

For further information on the ACSQHC Clinical Communications programme visit http://www.safetyandquality.gov.au/internet/safety/publishing.nsf/content/PriorityProgram-05.
## ISBAR

<table>
<thead>
<tr>
<th>I – Introduction</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>• Introduce yourself and your role in the patient’s care</td>
<td></td>
</tr>
<tr>
<td>• State the unit you are calling from when speaking over the phone</td>
<td></td>
</tr>
<tr>
<td><strong>S – Situation</strong></td>
<td></td>
</tr>
<tr>
<td>• Specify the patient’s name and current condition or situation</td>
<td></td>
</tr>
<tr>
<td>• Explain what has happened to trigger the conversation</td>
<td></td>
</tr>
<tr>
<td><strong>B – Background</strong></td>
<td></td>
</tr>
<tr>
<td>• State the admission date of the patient, his or her diagnosis, and</td>
<td></td>
</tr>
<tr>
<td>pertinent medical history</td>
<td></td>
</tr>
<tr>
<td>• Give a brief synopsis of what has been done so far (e.g. lab tests)</td>
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<tr>
<td><strong>A – Assessment</strong></td>
<td></td>
</tr>
<tr>
<td>• Give a summary of the patient’s condition or situation</td>
<td></td>
</tr>
<tr>
<td>• Note clearly the trend in patient observations</td>
<td></td>
</tr>
<tr>
<td>• Explain what you think the problem is or say, “I’m not sure what the problem</td>
<td></td>
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<tr>
<td>is, but the patient’s condition is deteriorating”</td>
<td></td>
</tr>
<tr>
<td>• Expand upon your statement with specific signs and symptoms</td>
<td></td>
</tr>
<tr>
<td><strong>R – Recommendation</strong></td>
<td></td>
</tr>
<tr>
<td>• Explain what you would like to see done (e.g. lab tests, treatments,</td>
<td></td>
</tr>
<tr>
<td>or “I need you to see the patient now”)</td>
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</tr>
</tbody>
</table>

### CLEAR AND ACCURATE DOCUMENTATION

Poor documentation (i.e. incomplete, inaccurate, illegible or inappropriate), has been attributed to negative outcomes for the patient. It can lead to an inability to provide continuity of care, omission or duplication of treatment, inappropriate decision making, inability to evaluate the effectiveness of treatment, and a lack of response to deterioration in a patient’s condition. Literature highlights a lack of a standard model for documenting and communicating information in medical records, a lack of time, and a reliance on verbal communication, as key reasons for poor documentation.
Documentation is an integral part of safe and appropriate clinical practice, and patient records are amongst the most basic of clinical tools. Its purpose is to provide a clear and accurate picture of the patients care and treatment, to serve as a basis for care planning and continuity of care, to facilitate co-ordination of care, and to comply with legal, regulatory and institutional standards. All practitioners are accountable for maintaining clear and accurate healthcare records.

Patient’s medical record should:

- Be factual, concise and accurate and not include opinions, editorial comments, or meaningless phrases
- Be systematic i.e. use a structured tool such as the ABCDEFG algorithm
- Be written concurrently or as close as possible to the event
- Include a date and time with all entries
- Always be signed and include first initial and last name and professional designation for clear identification
- Be legible
- Ensure corrections are dated, timed and signed and the original entry still legible i.e. original entries should never be erased or obliterated (e.g. with correction fluid)
- Be internally consistent and use only facility approved abbreviations and symbols
- Have entries written in chronological order without any blank spaces between entries

Never leave a deteriorating patient without a medical management plan that is both documented and verbalised to relevant people.

**STRATEGIES TO EFFECTIVE TEAMWORK**

**TWO-MINUTE TRAINING: TRAINING IN TEAMS FOR HOSPITAL EMERGENCIES**

“Two-minute training” for paediatric or adult emergencies aims to provide repeated, standardised opportunities to train team members in the initial responses and interactions required for effective management of the deteriorating patient.

Starting positions determined by role, responsibility and function within any emergency response or arrest team are dependent on available personnel and resources, but should be defined and standardised across healthcare facilities and contain common elements between all facilities. The same starting positions, patterns of behaviour, initial team interactions and individual priority tasks
for team members should be used every time the team responds to a real or simulated emergency event. This will lead to repeated practice opportunities across a prolonged period of time and improved performance in stressful real-life emergencies.

**COMMUNICATION STRATEGIES FOR HOSPITAL EMERGENCIES**

Communicating effectively within a team during an emergency situation requires specific strategies. Many communication strategies that are appropriate in healthcare settings have origins in the aviation industry which, as previously mentioned, is well rehearsed in non-technical skills.

**Leadership**

There needs to be clear role allocation for people to function as a team rather than just a group of people. The role of team leader is central to the team. A common complaint during post resuscitation briefs is that it was unclear who was in charge. ⁹

The team leader must have the skills to be able to assess, evaluate and anticipate the situation (*situation awareness*), identify and utilise all available resources (*task management*), devise a sound plan of action (*decision making*), convey this clearly and precisely to the team and assign appropriate roles and responsibilities (*communicating in teams*) and LISTEN for important cues (*communication*).

Delivery of an instruction or task needs to be directed to a specific person, therefore, as a team leader, it is beneficial to know team members names and levels of expertise to assist with allocations. If you are joining a team, always introduce yourself and ensure they know your level of expertise.

**Avoiding mitigation**

Mitigating speech refers to deferent or indirect language and is often adopted in times of uncertainty. It has been found to be a common occurrence in aviation prior to airline crashes and has been shown to be potentially deadly.

In emergency situations, mitigating speech is inappropriate and, as with aviation, may result in deadly consequences. Communication needs to be clear, brief and unequivocal. Junior staff need to be adequately trained in how to communicate clearly and assertively.

*Examples of combating mitigating speech:*

Perhaps we need the surgeon – *Can you get me the surgeon*

Maybe we should think about intubating this patient – *Lets intubate*
Repeat Back method

The ‘repeat back’ or ‘read back’ model involves communication being read back or repeated back to the sender. It ensures that the message received is the one delivered, it provides the opportunity to recognise and correct mistakes, it brings accountability to the task, and it reinforces what is being said and done to the whole team.

Repeat back is highly effective in telephone conversations to confirm verbal orders, particularly drugs and dosing, instructions or information.

Example of repeat back communication:

Medical officer: *Give a 5mg Salbutamol nebuliser and I will be there in 5 minutes.*
Sarah: *Just to confirm you would like me to give a 5mg Salbutamol nebuliser.*

Closed loop communication

Closed loop communication involves a request or a message being addressed to a specific person by name and that person repeating the message back to the sender. The action is then followed up with confirmation that is has been carried out.

Example of closed loop communication:

Surgeon: *Sarah, could you hang another unit of blood*
Nurse: *Hanging another unit of blood*
Nurse: *Blood has commenced*

GENERAL CONSIDERATIONS

Communication needs to be clear and unambiguous:

- Use generic names for medications
- Reiterate numbers by breaking down to their individual components to ensure they are not misheard e.g. fifteen could be misinterpreted as fifty – clarify by stating ‘fifteen, one-five’.
- Apply caution when using prefixes such as hyper and hypo as these can be misunderstood or misheard. Be literal by using the simple terms ‘high’ and ‘low’.
Avoid the use of jargon or abbreviations as these may vary between hospitals and/or a lack of knowledge may exist.

**CHALLENGING SITUATIONS**

Challenging the actions or decisions of a more experienced colleague can be an extremely daunting task, however, it is the responsibility of every single team member to ensure patient safety. Experience and seniority does not guarantee against errors. It is the responsibility of every team member to challenge colleagues if they are concerned with any decision or action that could potentially harm a patient.

An effective and responsible team leader gives due consideration to concerns raised by other team members and should be sufficiently humble to stand corrected. The aviation industry has a two challenge strategy where, if a senior colleague is questioned more than once about the safety of a given choice of action, they are obliged to come up with, or seriously consider, an alternative plan. Listening is an essential component of leadership.

**Graded assertiveness**

Graded assertiveness is a method that can often assist in situations of conflict or when team members have diverging thoughts. The gradient starts from the least confrontational progressing to the most confrontational as required and as determined by the extent or urgency of the situation. Assertiveness is not the same as aggression, which is based around intimidation and lack of respect.

The use of a gentle cue may be all that is required to effectively communicate a differing opinion allowing for a new perspective. If not, assertiveness can be escalated in a non-threatening manner until each team member is satisfied that their concerns have been addressed.

Example of graded assertiveness:

**Level one: express initial concern with an ‘I’ statement**

*I am concerned about . . .*

**Level two: make an enquiry or offer a solution**

*Would you like me to . . .*

**Level three: ask for an explanation**

*It would help me to understand . . .*

**Level four: a definitive challenge demanding a response**

*For the safety of the patient we need to ......now.*
REFERENCES


CHAPTER THREE

BREATHING HARD OR NOT AT ALL

BRADLEY CEELY
ANDREA CHRISTOFF
TOMAS RATONI

AIM

The aim of this chapter is to provide you with the knowledge to:

- Understand the simple pathophysiology and causes of respiratory deterioration
- Identify, interpret and evaluate the early and late warning signs of respiratory deterioration
- Assess and prioritise immediate and early management of respiratory deterioration
- Escalate concerns to Clinical Review or Rapid Response as appropriate

Throughout the chapter ‘child’ refers to infant, child and adolescent unless otherwise stated.

<table>
<thead>
<tr>
<th>Early Warning Signs of Respiratory Deterioration</th>
</tr>
</thead>
<tbody>
<tr>
<td>Stridor at rest</td>
</tr>
<tr>
<td>Moderate tachypnoea, increasing trend</td>
</tr>
<tr>
<td>Moderate tachycardia, increasing trend</td>
</tr>
<tr>
<td>Restlessness, irritability, agitation, inconsolability</td>
</tr>
</tbody>
</table>

Table 1

<table>
<thead>
<tr>
<th>Late Warning Signs of Respiratory Deterioration</th>
</tr>
</thead>
<tbody>
<tr>
<td>Any new onset of stridor or stridor with drooling of secretions</td>
</tr>
<tr>
<td>Marked tachypnoea or decreasing trend (exhaustion), apnoea</td>
</tr>
<tr>
<td>Marked tachycardia or any bradycardic episode</td>
</tr>
<tr>
<td>Confusion, drowsiness, reduced level of consciousness</td>
</tr>
</tbody>
</table>

Table 2

Concern by any staff or family member irrespective of early or late warning signs should be appropriately escalated.
PATHOPHYSIOLOGY OF RESPIRATORY DETERIORATION

ANATOMICAL AND PHYSIOLOGICAL DIFFERENCES BETWEEN CHILDREN AND ADULTS

There are a number of differences between the airways and lungs of a child and an adult which predispose them to increased risk of respiratory difficulties. For the first 8 years of life in particular, a child’s respiratory system is constantly growing and maturing.

![Diagram of child's respiratory system]

**Figure 1: Anatomy and Physiology of a child’s respiratory system**

<table>
<thead>
<tr>
<th>Difference</th>
<th>Implication</th>
</tr>
</thead>
<tbody>
<tr>
<td>Infants have a large head with a prominent occiput, short neck and soft tracheal cartilage</td>
<td>Obstruction of the airway can occur due to flexion of the neck when the infant is supine or overextension of the head during airway opening manoeuvres.</td>
</tr>
<tr>
<td>Smaller calibre upper and lower airways</td>
<td>Any airway swelling or exudate leads to increased airway resistance and work of breathing.</td>
</tr>
</tbody>
</table>
This brief look at the paediatric respiratory anatomy and physiology allows one to appreciate the significant differences between children and adults. Any child with respiratory distress should be managed with vigilance and caution.

*Infants and children are prone to fatigue, apnoea and rapid respiratory decompensation.*

*Assess frequently and initiate interventions early.*

<table>
<thead>
<tr>
<th>Difference</th>
<th>Implication</th>
</tr>
</thead>
<tbody>
<tr>
<td>Inefficient intercostal respiratory muscles, particularly in infants</td>
<td>Respiratory muscles fatigue quickly leading to respiratory failure and apnoea.</td>
</tr>
<tr>
<td>Infants are “abdominal breathers” relying on the diaphragm as the principal respiratory muscle</td>
<td>Abdominal distension and increased intra-abdominal pressure may cause respiratory failure.</td>
</tr>
<tr>
<td>Infants have a smaller volume of air left in the lungs at the end of each breath (Functional Residual Capacity)</td>
<td>Infant lungs are more prone to alveolar collapse (atelectasis).</td>
</tr>
<tr>
<td>Compliant chest wall with horizontal, short ribs in infants</td>
<td>Chest expansion is limited and use of intercostals muscles for increased respiratory effort may not be effective or sustainable.</td>
</tr>
<tr>
<td>Relatively large tongue, rapidly growing tonsils and adenoids, floppy epiglottis and supraglottic airway structures</td>
<td>Higher risk of airway obstruction. Inflammation can cause significant upper airway obstruction. Airway collapse can occur with increased respiratory effort.</td>
</tr>
<tr>
<td>Infants have relatively narrow nasal passages and may be obligatory nose breathers for the first 4-6 months of life</td>
<td>Nasal secretions can cause significant airway obstruction and respiratory distress.</td>
</tr>
<tr>
<td>Infants, particularly those born preterm or small for gestational age, have an immature respiratory control centre</td>
<td>It is common to see irregular breathing and apnoea as signs of respiratory disease such as in bronchiolitis or pneumonia.</td>
</tr>
<tr>
<td>Infants have a much higher metabolic rate than adults</td>
<td>Infants consume more oxygen and produce more carbon dioxide. The work of breathing can account for up to 40% of cardiac output, particularly in stressed conditions.</td>
</tr>
<tr>
<td>Infants and young children have immature immune systems</td>
<td>They are more susceptible to infections including respiratory infection.</td>
</tr>
</tbody>
</table>

*Table 3*
AIRWAY OBSTRUCTION

As discussed in Chapter one, audible inspiratory or expiratory noises are a sign of airway obstruction and can often provide an indication of where the obstruction lies and what the underlying cause may be.

STRIDOR

Stridor refers to a harsh, vibratory sound of variable pitch caused by turbulent airflow through a partially obstructed upper airway. It indicates upper airway obstruction. More commonly the stridor is inspiratory and suggests airway obstruction above the glottis, as occurs in croup. If the stridor is expiratory it is indicative of an obstruction in the lower trachea but this is much less common. A biphasic stridor (inspiratory and expiratory) suggests a fixed obstruction, usually at the level of the glottis or subglottis.

WHEEZE

Wheeze refers to a prolonged coarse or high-pitched whistling sound during expiration, caused by partial airway obstruction from any cause affecting the lower airways (i.e. below the level of the trachea). It indicates lower airway obstruction. Whilst the diagnosis of asthma should be considered in any child with acute onset of wheeze, it should be remembered that asthma is not the only cause of acute wheeze. Response to bronchodilators, e.g. salbutamol, may help differentiate asthma from other causes of wheezing.

RESPIRATORY FAILURE

Respiratory failure occurs when the respiratory system fails to adequately oxygenate or eliminate carbon dioxide from the blood passing through the lungs leading to a situation of either hypoxaemia or hypercarbia. Respiratory failure may, therefore, be classified as hypoxaemic respiratory failure or hypercarbic respiratory failure.

Infants and young children are more likely to develop respiratory failure than older children due to their anatomical and physiological differences. Other factors that increase the risk of acute respiratory failure include a history of prematurity, immunodeficiency, chronic pulmonary conditions (e.g. chronic lung disease, cystic fibrosis), cardiac conditions (e.g. unrepaired congenital heart disease), and neuromuscular disease with altered muscle tone or power (e.g. muscular dystrophy, spinal muscular atrophy [SMA]).

3
High risk infants and children

| Under 3 months old |
| Chronic or complex conditions |
| Post operative |
| Pre-existing cardiac or respiratory conditions |
| Opioid infusions |

*Table 4*

**HYPOXAEMIC RESPIRATORY FAILURE**

Hypoxaemic respiratory failure is the most common type of respiratory failure and can be associated with most acute respiratory illnesses. It typically occurs in situations of alveolar collapse, oedema, or infection, where normal gas exchange is impaired and insufficient oxygen diffuses from the alveoli into the blood passing through the lungs.

*The first signs of hypoxaemia are tachypnoea and tachycardia.* If deterioration continues, the child will start to show signs of increased respiratory effort, such as recession, nasal flaring and tracheal tug. In severe respiratory distress, an infant will attempt to keep alveoli open and exchange oxygen by breathing against partially closed vocal cords, producing a ‘grunt’ on expiration.

*Children who have been in respiratory distress for a while are at risk of fatigue.* Unfortunately, the period of respiratory distress before exhaustion and impending respiratory arrest is not easily predictable and varies with each child.

As a child fatigues the respiratory rate may start to fall, possibly even to within normal range. However, this will likely be accompanied by other signs of deterioration such as irregular breathing including prolonged pauses or apnoea, marked tachycardia, and changes in behaviour or mental status.

Drowsiness, reduced level of consciousness and worsening hypoxaemia are distinguishing features of fatigue and are pre-arrest signs.

**HYPERCARBIC RESPIRATORY FAILURE**

Hypercarbic respiratory failure occurs when there is a problem with ventilation (i.e. gas flow into and out of the lungs) and not enough carbon dioxide is expired. A state of hypercapnia and respiratory acidosis develops that can result in confusion, convulsions, coma and respiratory arrest.
It can occur as a result of chronic or acute obstructive airway disease, such as asthma or bronchiolitis, or as a result of hypoventilation i.e. abnormally slow and/or shallow respirations.

Hypoventilation can be associated with:

- **Children receiving opioid infusions** who may develop a reduced respiratory drive due to narcosis and central nervous system depression. Abnormally slow respiratory rate, drowsiness and unresponsiveness are important distinguishing signs of respiratory failure in these children, even in the presence of normal saturations.

- **Children with head injuries or neurological illnesses** that could depress the respiratory centre. Again, drowsiness and unresponsiveness are important distinguishing signs of respiratory failure in these children.

- **Children with neuromuscular disease** who have reduced muscle power or chest wall deformities. Attempts to increase ventilation can result in airway or chest wall collapse during inspiration, and marked abdominal excursion. This is often referred to as see-saw breathing.

- **Neonates and young infants** who, in the first months of life, may present with apnoea or hypoventilation rather than the expected increase in respiratory effort due to an immature respiratory centre and general response to systemic illness.

Hypercarbic respiratory failure will lead to hypoxaemia which can be easily corrected with oxygen. However, oxygen therapy may improve oxygen saturations but it will not correct inadequate ventilation, the hypoventilated state, or the risk of a sudden respiratory arrest.

*When normal is bad;* a reduced level of consciousness without tachypnoea or increased respiratory effort may be the only sign of hypercarbic respiratory failure.

**Cyanosis**

Cyanosis is a late and ominous sign, usually visible only when arterial oxygen saturations fall below 85%. Cyanosis may be central or peripheral. Central cyanosis affects the lips and tongue and peripheral cyanosis affects the fingers, toes and nail beds.

*Onset of central cyanosis always signifies hypoxaemic respiratory failure,* however, peripheral cyanosis may be observed in the absence of hypoxaemic respiratory failure, in conditions such as hypovolaemia, low cardiac output state, hypothermia or polycythaemia. Be aware that children with some forms of unrepaired congenital heart disease, may normally have central cyanosis and low
oxygen saturations. Altered calling criteria that reflect appropriate thresholds for treatment and escalation will need to be communicated, documented and reviewed regularly in these children.

Central cyanosis is considered a very late and severe sign in children with respiratory distress. Escalate to a Rapid Response immediately.

**OXYGEN THERAPY**

Oxygen therapy is recommended in all situations of inadequate oxygenation i.e. oxygen saturations (SpO₂) less than 95%; however, *it should never be administered unnecessarily due to the risk of oxygen toxicity*. Oxygen requirements should be reviewed regularly and oxygen weaned if SpO₂ is more than 98%.

Oxygen therapy can be administered a number of ways via:

- Nasal prongs (nasal cannulae)
- Simple facemask
- Partial rebreather mask and non-rebreather mask
- Infant headbox
- Humidified high flow nasal prongs

The most appropriate method of administration of oxygen therapy will depend on:

- Age and size of the child
- Oxygen requirements/therapeutic goals
- Patient tolerance
- Humidification needs

The oxygen supply from the wall delivers an oxygen concentration (FiO₂) of 100%; however, the actual FiO₂ the child receives will vary according to the apparatus used, the flow rate (L/min) and the respiratory status of the child.

It is recommended that a local guideline/policy is in place to augment local practices and expertise with available oxygen delivery equipment.

**NASAL PRONGS**

Nasal prongs deliver *low concentrations* of oxygen. It is difficult to predict exactly how much oxygen the patient actually receives because it varies with the patient’s respiratory rate and pattern, flow rate, prong position and the degree of mouth breathing.
Estimates of oxygen delivery using nasal prongs are:

1 L/min of flow delivers approximately 24% FiO₂
2 L/min of flow delivers approximately 28% FiO₂

If more than approximately 30% oxygen is needed a different delivery system should be used.

There are several sizes of nasal prongs and the appropriate size for an infant and child should be chosen. Nasal prongs should never be trimmed and must sit in the nares. Hydrocolloid or similar skin protective tape can be applied to the face prior to securing nasal prongs to maintain skin integrity.

Nasal prongs are generally well tolerated, however, without humidification and at maximum flow rates they can result in drying of the nasal passages, pain and discomfort.

Maximum nasal prong flow rates should not exceed 2 L/min in infants and 4 L/min in older children to prevent drying of nasal secretions and damage to the nasal mucosa.

SIMPLE FACE MASK

Simple face masks (commonly known as Hudson facemasks) deliver medium concentrations of oxygen. Oxygen delivery is influenced by the size and fit of the mask, the respiratory rate and pattern, and the flow rate.

Estimates of oxygen delivery via simple face masks are:

5-6 L/min of flow delivers 40% FiO₂
6-7 L/min of flow delivers 50% FiO₂
7-8 L/min of flow delivers 60% FiO₂

‘BLOW OVER’ OXYGEN IS NOT A THERAPEUTIC APPROACH TO OXYGEN

If more than 60% oxygen is needed a different delivery system should be used.

Venturi masks deliver a more accurate concentration of oxygen determined by a colour coded venturi valve and oxygen flow as per manufacturer recommendations, but are not commonly used in children.

Minimum flow through a simple face mask is 5 L/min to ensure exhaled carbon dioxide is blown away and not rebreathed.

PARTIAL REBREATHER MASK AND NON-REBREATHER MASK

Partial rebreather masks and non-rebreather masks deliver high concentrations of oxygen because
of the addition of a reservoir bag. Although similar in appearance, the non-rebreather mask has an additional one way valve system on the sides of the mask and between the mask and the reservoir bag in order to deliver an even higher concentration of oxygen. The mask must have a good seal to deliver the highest FiO₂ and the reservoir bag should remain inflated at all times.

*Estimates* of oxygen delivery using a partial rebreather mask are:

- 8-12 L/min of flow delivers 40-70% FiO₂

*Estimates* of oxygen delivery using a non-rebreather mask are:

- 10-15 L/min of flow delivers 80-100% FiO₂

Oxygen flow rate should be regulated so that the bag will not deflate more than one third on inspiration. *Minimum* flow rate is approximately 8-10 L/min.

*Note:* Infants and young children don’t generally tolerate any type of face mask well and ongoing supervision is often required to ensure that the mask is kept in position. Forced application of a face mask may significantly upset the child, thereby, exacerbating respiratory distress and increasing metabolic demand. This should be avoided.

**INFANT HEADBOX**

Headbox oxygen can be used to deliver even relatively high concentrations of humidified oxygen to young infants (up to 80-90%).

An appropriately sized headbox (neonate/infant) is placed over the head of an infant *lying in the supine position*. Some headboxes can be placed over the head and trunk of the infant, allowing for hand to mouth comfort behaviour. A headbox cannot be used in any infant able to sit up by themselves.

*The flow of oxygen must be at least 6-10 L/min* dependant on size of headbox used to ensure effective FiO₂ and prevent accumulation of carbon dioxide. The headbox access ports must be free of obstruction and the seal around the neck or body of the child must not be too occlusive in order to prevent carbon dioxide accumulation. An oxygen analyser should be placed inside the headbox and the relative oxygen/air flows adjusted according to the desired level of inspired oxygen.
HUMIDIFIED HIGH FLOW NASAL PRONGS

Specifically designed high flow nasal prongs (HFNP) deliver high flow humidified oxygen. They may be applied in situations of hypoxaemia (SpO₂ less than 95%) and respiratory distress despite standard flow oxygen therapy. HFNP is tolerated much better than any facemask oxygen therapy and allows delivery of routine cares without interruption of oxygen delivery. It can also be used for prolonged periods without causing drying of secretions.

Humidified HFNP is available in various sizes, and requires a specialised humidifier to allow oxygen concentration to be titrated. Flow rates normally vary from 0.5 L/kg/min up to 2 L/kg/min in the unwell child, although this depends on the size and specifications of the high flow nasal prong being used (always adhere to manufacturer’s guidelines). It is recommended that if flow rates exceed 2 L/kg/min the child should be nursed in a critical care area.

Humidified high flow nasal prongs can be used to deliver inspired concentrations of oxygen more than 80% whilst generating a variable amount of continuous positive airway pressure (CPAP) when used at high flows.

Humidified high flow nasal prongs provide high levels of respiratory support and should only be used under the prescription of experienced clinicians. Local policies must be in place.

<table>
<thead>
<tr>
<th>SUMMARY OF APPROXIMATE OXYGEN CONCENTRATIONS FOR OXYGEN THERAPY</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Device</strong></td>
</tr>
<tr>
<td>-------------</td>
</tr>
<tr>
<td>Room air</td>
</tr>
<tr>
<td>Nasal Prongs</td>
</tr>
<tr>
<td></td>
</tr>
<tr>
<td>Simple Facemask</td>
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<td></td>
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<tr>
<td></td>
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<tr>
<td>Partial rebreather facemask</td>
</tr>
<tr>
<td>Non-rebreather facemask</td>
</tr>
<tr>
<td>Infant Headbox</td>
</tr>
<tr>
<td>Humidified HFNP</td>
</tr>
</tbody>
</table>

Table 5
ASSISTED VENTILATION

When breathing is absent or insufficient to provide adequate ventilation of the lungs, devices that provide positive inspiratory pressure to inflate the lungs are required to assist the infant or child’s ventilation through a supported patent airway. The following devices are commonly used:

**BAG VALVE MASK (‘AMBU BAG’)**

Cheap and readily available, these devices can rapidly re-establish adequate ventilation in an emergency, even without attached oxygen or other gas source. Preferably, however, oxygen should be attached in order to deliver a high concentration of oxygen when required. A neck roll may need to be placed under the shoulders to prevent neck flexion and assist in maintaining a patent airway.

![Figure 4: Bag Valve Mask (BVM) ventilation](image)

Due to the presence of a prominent occiput, a shoulder roll can often assist in positioning an infant in the optimal position to maintain an open airway during bag valve mask ventilation.

There are 3 sizes of self-inflating bags commonly used in infants and children. The exact recommendations for use will vary with manufacturer; however, the following is a guide:

- **250mL** for newborns only
- **500mL** for children up to 30kgs
- **1500mL** for children over 30kgs

The tidal volume should be delivered slowly and must be sufficient to see the chest rise. The ventilation rate for infants should be 20-30 breaths/min and for older children 16-20 breaths/min. Care should be taken not to ventilate too hard or too fast, as this can lead to barotrauma, gastric distension, hypotension, and reduced cerebral blood flow.

Bag valve masks (BVM) do have some limitations; they deliver unpredictable pressures (despite pressure limiting valves incorporated into the neonatal and paediatric sizes). They should not be used in spontaneously breathing children as the negative inspiratory pressure generated by spontaneously breathing infants and young children may be insufficient to open the inspiratory valve.
to allow sufficient fresh gas flow.\textsuperscript{10, 11}

\begin{figure}[h]
\centering
\includegraphics[width=\textwidth]{anaesthetic_bag.png}
\caption{Anaesthetic Bag}
\end{figure}

Self inflating bags should \textit{not be used} to deliver oxygen to a spontaneously breathing child as they do not reliably provide free flow of oxygen.

\begin{figure}[h]
\centering
\includegraphics[width=\textwidth]{infant_resus.png}
\caption{Infant T-piece resuscitator}
\end{figure}

**ANAESTHETIC BAGS (MODIFIED AYRES T-PIECE)**

This is a simple inflatable bag and mask arrangement where the user partially occludes the inflatable bag’s gas outlet with their fingers and squeezes the bag to redirect gas flow to the patient’s lungs in order to provide \textit{positive pressure breaths and CPAP}.

Anaesthetic bags provide the user with a good feel for the patient’s efforts of breathing and allow support of spontaneous breaths as well as the ability to provide fully assisted breaths if required.

Limitations to this device are that it is dependent on a continuous fresh gas supply to inflate the bag and deliver a breath. Lack of expertise can easily result in delivery of dangerously high pressures or lung over-inflation. It is recommended that the device is used only by experienced operators, and usually with a pressure gauge.

**INFANT T-PIECE RESUSCITATOR (NEO-PUFF™)**

This is a simple to operate pressure regulated device and circuit, available in most newborn nurseries and many emergency departments. It delivers a set pressure breath whenever the small hole in the circuit is occluded by the operator’s finger. The Neo-puff™ may be used for the spontaneously breathing infant to deliver CPAP at a set level, as indicated by a dial on the resuscitator.

Limitations to these devices are that it is only suitable for infants up to 10kgs and it relies on a continuous gas supply.
CPAP DEVICES

CPAP devices are used to deliver oxygen under a set pressure in order to optimise lung volume by preventing alveolar collapse on expiration, or to help overcome increased airway resistance. As described above, humidified high-flow nasal prongs provide some degree of CPAP when at the higher acceptable flow rates. If a child requires a greater degree of respiratory support, however, a number of CPAP devices utilising specific nasal or full face masks with circuits to allow exhausting of expired gases can be used under the supervision of appropriately trained clinicians.

In general this should only be done after consultation with senior medical staff with experience in this level of respiratory support and transfer to an intensive care unit should be considered in these children.

CAUSES OF RESPIRATORY DETERIORATION

Identification of the underlying cause of respiratory deterioration allows for immediate specific supportive care. With supportive care and aggressive treatment of the underlying cause most children with respiratory deterioration will recover relatively quickly and uneventfully.

### CAUSES OF RESPIRATORY DETERIORATION

<table>
<thead>
<tr>
<th>Possible Diseases</th>
<th>Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Upper Airway Obstruction</strong></td>
<td></td>
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</tbody>
</table>
| Croup | ▪ Harsh stridor on inspiration with a barking ‘seal’ cough  
▪ Usually occurs 6 months to 3 years of age (unlikely in under 3 month olds) |
| Epiglottitis/Tracheitis | ▪ Quiet or low pitched stridor and absent cough  
▪ Drooling  
▪ Toxic, unwell looking (pale, febrile & poorly perfused)  
▪ ‘Tripod’ position  
▪ Rare usually occurs 2 to 7 years of age |
| Foreign Body | ▪ Sudden onset of stridor often preceded by choking or coughing  
▪ Usually occurs 1 to 2 years of age |
| Anaphylaxis | ▪ Hoarseness and stridor  
▪ Usually accompanied by other symptoms of an allergic reaction (wheezing, facial swelling, urticarial rash)  
▪ Sudden onset, always within 30 minutes or less of exposure to allergen |
<table>
<thead>
<tr>
<th>Possible Diseases</th>
<th>Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Lower Airway Obstruction</strong></td>
<td></td>
</tr>
<tr>
<td>Asthma</td>
<td>- Wheeze, cough and prolonged expiration</td>
</tr>
<tr>
<td></td>
<td>- Usually previous history of similar illness and/or co-existing eczema or hay-fever</td>
</tr>
<tr>
<td>Bronchiolitis</td>
<td>- Nasal congestion and discharge</td>
</tr>
<tr>
<td></td>
<td>- Persistent cough, sometimes with vomiting</td>
</tr>
<tr>
<td></td>
<td>- Rapid, shallow breathing with possible apnoeas</td>
</tr>
<tr>
<td></td>
<td>- Inspiratory crackles and expiratory wheezes</td>
</tr>
<tr>
<td></td>
<td>- Acute feeding problems</td>
</tr>
<tr>
<td></td>
<td>- Usually occurs younger than 1 year of age</td>
</tr>
<tr>
<td><strong>Acute lung disorders</strong></td>
<td></td>
</tr>
<tr>
<td>Pneumonia</td>
<td>- Tachypnoea with expiratory grunt in infants and young children</td>
</tr>
<tr>
<td></td>
<td>- Cough</td>
</tr>
<tr>
<td></td>
<td>- High fever</td>
</tr>
<tr>
<td></td>
<td>- Anorexia</td>
</tr>
<tr>
<td></td>
<td>- Chest pain</td>
</tr>
<tr>
<td>Aspiration pneumonia</td>
<td>- Sudden onset of symptoms – usually following a feed</td>
</tr>
<tr>
<td></td>
<td>- Noisy breathing/wheezing</td>
</tr>
<tr>
<td></td>
<td>- Productive cough</td>
</tr>
<tr>
<td></td>
<td>- Higher risk in children with chronic illnesses</td>
</tr>
<tr>
<td>Pertussis (whooping cough)</td>
<td>- Pronounced cough which may occur in spells or paroxysms</td>
</tr>
<tr>
<td></td>
<td>- Vomiting often follows a coughing spasm.</td>
</tr>
<tr>
<td></td>
<td>- Cough may persist for months</td>
</tr>
<tr>
<td></td>
<td>- Young infants may develop apnoeas</td>
</tr>
<tr>
<td></td>
<td>- Can occur in immunised children but generally less severe</td>
</tr>
<tr>
<td>Pulmonary oedema</td>
<td>- Tachypnoea</td>
</tr>
<tr>
<td></td>
<td>- Wheeze</td>
</tr>
<tr>
<td></td>
<td>- Moist cough and inspiratory crackles</td>
</tr>
<tr>
<td></td>
<td>- Hepatomegaly</td>
</tr>
<tr>
<td>Pneumothorax</td>
<td>- Unequal air entry</td>
</tr>
<tr>
<td>Empyema</td>
<td>- Unequal chest movement</td>
</tr>
<tr>
<td>Pleural effusion</td>
<td></td>
</tr>
</tbody>
</table>

Table 6

It is important to note that respiratory deterioration can also result from **non-pulmonary** causes. Examples include abdominal distension (from acute conditions such as peritonitis and necrotising enterocolitis) that can result in respiratory distress, irregular breathing and apnoeas, particularly in a neonate, and metabolic acidosis such as Diabetic Ketoacidosis that results in rapid and deep (sighing) respirations known as Kussmaul breathing.
RECOGNITION OF RESPIRATORY DETERIORATION

Respiratory illnesses are the most common condition for which children are admitted to hospital. Two thirds of hospital paediatric cardiac arrests are secondary to respiratory failure. It is essential that healthcare practitioners caring for children are able to perform a thorough paediatric respiratory assessment, identify the early and late warning signs of respiratory deterioration, and manage and escalate care appropriately.

It is important to remember that children with respiratory illnesses may initially present with non-respiratory symptoms, such as poor feeding, abdominal pain, vomiting, restlessness and irritability.

Difficulties with feeding may be the first problem noted by caregivers of infants with respiratory deterioration.

<table>
<thead>
<tr>
<th>Assessment</th>
<th>Action</th>
<th>Warning signs</th>
</tr>
</thead>
<tbody>
<tr>
<td>Airway patency</td>
<td>Look, Listen and Feel&lt;br&gt;Observe behaviour, general appearance, posture, chest movement, respiratory effort. Listen for respiratory noises</td>
<td>‘Tripod’ posture&lt;br&gt;Persistent stridor at rest&lt;br&gt;Gurgling/Snoring&lt;br&gt;Drooling</td>
</tr>
<tr>
<td>Rate</td>
<td>Observe for apnoea and count respiratory rate for a full minute. Check the trend</td>
<td>Tachypnoea/increasing trend&lt;br&gt;Abnormal pauses/apnoeas&lt;br&gt;Decreasing trend (fatigue)</td>
</tr>
<tr>
<td>Effort</td>
<td>Assess respiratory effort using the SPOC ‘ASSESSMENT OF RESPIRATORY DISTRESS’ table</td>
<td>Moderate recession, tracheal tug, nasal flaring, head bobbing&lt;br&gt;Severe recession, grunting, gasping</td>
</tr>
<tr>
<td>Efficacy</td>
<td>Observe chest movement&lt;br&gt;Listen to air entry (auscultation)&lt;br&gt;Observe for central cyanosis&lt;br&gt;Monitor SpO₂</td>
<td>Reduced or unequal chest movement&lt;br&gt;Reduced air entry (<em>Absent air entry is a medical emergency</em>)&lt;br&gt;SpO₂ less than 95%&lt;br&gt;Central cyanosis</td>
</tr>
<tr>
<td>Effects</td>
<td>Assess cardiovascular system</td>
<td>Tachycardia&lt;br&gt;Mottled skin</td>
</tr>
<tr>
<td></td>
<td>Assess neurological status</td>
<td>Restlessness/irritability&lt;br&gt;Agitation/combative&lt;br&gt;Drowsiness/hypotonia (floppy)</td>
</tr>
</tbody>
</table>

Table 7

For a detailed assessment of Airway and Breathing refer to Chapter 1 pp 6-14.
INITIAL MANAGEMENT OF RESPIRATORY DETERIORATION

Regardless of the underlying cause, the priorities of initial management are to establish a patent airway (A), administer supplemental oxygen or provide assisted ventilation if indicated (B) and support circulation (C). Once life-threatening conditions have been addressed continue to work through the ABCDEFG algorithm whilst treating the underlying cause.

Remember to keep the caregivers close by and well informed. If possible, assign a member of the team to remain with them to provide continual support and reassurance or request the social worker.

A Start with the Airway

- If the airway is compromised, specific management will depend upon the cause. One or more of the following may be required to open and maintain the airway:
  - Patient repositioning
  - Head tilt-chin lift/jaw thrust airway opening manoeuvres
  - Airway adjuncts
  - Careful suctioning

- Children with marked decreased level of consciousness

  Obstruction of the upper airway can occur due to loss of airway muscle tone. The tongue is also a common cause of obstruction in children. To relieve the obstruction, repositioning with head tilt-chin lift/jaw thrust airway opening manoeuvres can be very effective (refer to Chapter 1 p 8). If airway obstruction persists, oropharyngeal (Guedel) and nasopharyngeal airways are useful adjuncts to improve airway patency.

![Figure 7: Measuring an oropharyngeal airway]
Centre of mouth to angle of jaw ¹¹

![Figure 8: Measuring a nasopharyngeal airway]
Edge of nostril to tragus of ear ¹¹
‘Snuffly’ infants
Infants are obligate nose-breathers and suctioning of nasal passages is often indicated in respiratory illnesses. Use a soft catheter and administer saline drops into the nostrils if secretions are thick.

Children with chronic neuromuscular conditions
Weakness of the respiratory muscles can result in an ineffective cough and inability to clear secretions. Deep suctioning (i.e. below the glottis) by an appropriately trained clinician may be required in order to stimulate a cough. Repositioning and support for the head and trunk may also be indicated.12

Children with stridor
Partial airway obstruction can rapidly progress to complete airway obstruction so escalate concerns early.

– A persisting stridor at rest indicates moderate airway obstruction and must be escalated to a Clinical Review.
– A soft stridor can indicate increasing airway obstruction and, if associated with other signs of severe respiratory distress, should prompt a Rapid Response.
Observe the child closely with *minimal painful or distressing interventions*. Caregivers can play a vital role in providing comfort and reassurance and keeping the child as calm and settled as possible.

Specific initial treatment for new onset or worsening stridor is the administration of an *Adrenaline nebuliser* (0.5 mL/kg of 1:1000 Adrenaline up to max 5 mL). \(^\text{13}\)

For detailed information on the assessment and management of croup refer to Clinical Practice Guideline *PD2010_053 Infants and Children: Acute Management of Croup*.

Any child unable to maintain their own airway must be escalated to a Rapid Response.

**For Breathing**

- Commence appropriate oxygen therapy *(refer to Oxygen Therapy p 58)*. Oxygen administration is recommended for any child with increased respiratory effort and SpO\(_2\) of less than 95% unless Altered Calling Criteria has been done.

- HYPOXAEMIA DUE TO UPPER AIRWAY OBSTRUCTION OR HYPOVENTILATION IS A LATE AND OMINOUS SIGN.

  The commencement of oxygen or any increase in oxygen requirement is a sign of clinical deterioration and requires a Clinical Review.

- Encourage the child to sit upright and provide support for this position. In the case of young infants elevate the head of the cot with appropriate support to prevent any airway obstruction.


- In the event of severe respiratory distress call for a Rapid Response and provide appropriate initial emergency treatment:
  - *Hypoventilation or apnoea*: Provide assisted ventilation using bag valve mask *(refer to p 62)* with high-flow oxygen whilst maintaining the airway open. Call a Rapid Response.
  - *Severe or life threatening asthma*: Commence continuous Salbutamol (Ventolin) nebuliser therapy (load 4 mL of undiluted 0.5% Salbutamol solution [5 mg/mL] into the nebuliser and top up as required) at a min oxygen flow rate of 8 L/min. \(^\text{14}\)
- **Pre-arrest**: Call for a Rapid Response and prepare to administer intramuscular Adrenaline 10 microg/kg i.e. 0.01 mL/kg of 1:1000 (neat) as soon as possible. \(^{14}\)

- **Tension pneumothorax** (indicated by absent air entry, reduced chest movement, tracheal deviation (unless bilateral), and hyper-resonant to percussion, in the presence of increasing respiratory and circulatory compromise): Prepare for needle thoracocentesis (16 gauge or larger cannula, alcohol swabs and 20 mL syringe).

For detailed information on the assessment and management of asthma refer to Clinical Practice Guideline *PD2012_056 Infants and Children: Acute Management of Asthma*.

For detailed information on the assessment and management of bronchiolitis refer to Clinical Practice Guideline *PD2012_004 Infants and Children: Acute Management of Bronchiolitis*.

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**C For Circulation**

- Document and monitor trends in heart rate, blood pressure, capillary refill and presence of peripheral and central pulses. Attach an ECG monitor if there is any suspicion of cardiac involvement.

- Lower airway obstruction, e.g. asthma, may result in hyperinflation, and a raised intrathoracic pressure that can impair cardiac filling. Administer a bolus of 0.9% Sodium Chloride if indicated by marked tachycardia, poor perfusion, weak peripheral pulses, and hypotension.

- Note any change in palpable peripheral pulse intensity during inspiration vs. expiration and consider this a sign of increased severity if present (pulsus paradoxus).

**D For Disability**

- Monitor level of consciousness (alertness and arousal) using the AVPU scale for signs of exhaustion or fatigue.

- Call for a Rapid Response immediately if the child is responsive only to Pain or is Unresponsive or if any concern regarding a deteriorating level of responsiveness.

Uncooperative, and/or combative behaviour may indicate hypoxia. Never assume it is normal age related behaviour.
E For Exposure

- Document and monitor trend in axillary temperature.
- Administration of anti-pyretics is not routinely advocated, however, it can help treat the effects of fever on behaviour (irritability and lethargy) and physiological parameters (respiratory rate and heart rate) allowing for a more accurate assessment of the signs and symptoms caused by the underlying condition.

Any child under 3 months with a new onset of fever more than 38.0°C requires a septic screen, Clinical Review and antibiotic therapy.

F For Fluids

- Cease enteral feeding in any infant or child with severe or worsening respiratory distress and place nil by mouth (NBM).
- Insertion of NG tube or aspiration of existing NG tube may be required if abdomen looks distended due to swallowed air.
- Commence IV fluids as per the recommended NSW guidelines (refer to Chapter 6 Table 8 p 128) and appropriate to latest blood chemistry levels. Maintain a normal hydration state, and avoid dehydration, particularly in asthma.
- Maintain an accurate fluid balance ensuring an infusion pump and burette is used at all times.
- Do not administer fluids with sodium content below 0.45% Sodium Chloride as many infants and children with respiratory illnesses are at risk of iatrogenic hyponatraemia (refer to Chapter 6 p 118). Monitor serum sodium in all cases.

G For Glucose

- Check the blood glucose level (BGL) in any unwell or deteriorating child.
- If hypoglycaemia is present i.e. BGL less than 3.0mmol/L or symptomatic (refer to Chapter 1 p 29) call for a Clinical Review and prepare to administer a bolus of 2 mL/kg of 10% Glucose or IM Glucagon when prescribed.
- Ensure maintenance fluids contain 5 - 10% Glucose.
- Repeat BGL within 5-10 minutes following administration of a glucose bolus.
If BGL is less than 2.0mmol/L call for a Rapid Response and prepare to administer a bolus of 2 mL/kg of 10% Glucose.

**ONGOING MANAGEMENT**

Any child with respiratory deterioration should be escalated to senior members of the local team, and paediatric expertise should be sought. Where appropriate, consideration should be given to consulting with state emergency and/or intensive care services via NETS (Newborn and paediatric Emergency Transport Service) for advice and possible transfer to a tertiary paediatric centre. Refer to Appendix 1 for information on Interhospital transfer.

Investigations may include:

- **Chest X-Ray (CXR)** - This will identify chest wall, pleural and lung parenchymal pathology and should normally be considered part of the respiratory assessment in any acute respiratory deterioration. However, obtaining a CXR should never delay urgent, potentially life-saving treatments, such as oxygen therapy, assisted ventilation or aspiration of a clinically detected tension pneumothorax.

- **Lateral Neck X-Ray** - Rarely if ever indicated in the acute setting and never to be undertaken if likely to cause distress or decompensation on the acute setting of upper airway obstruction.

- **Blood gas analysis** - Blood gas analysis can provide a great deal of information in a short period of time that can assist with diagnosis (if unknown) and treatment. Information obtained includes acid base status, oxygen and carbon dioxide levels, as well as haemoglobin, sodium, potassium, glucose, and ionised calcium.
  - **Capillary blood gas analysis**: May provide a useful estimate of arterial carbon dioxide level and acid-base status. Poor peripheral perfusion or difficulty in obtaining an adequate sample can affect results, arterial sample should be taken to confirm any abnormal results.
  - **Venous blood gas analysis**: This can also provide accurate information on systemic carbon dioxide level, acid-base status, major electrolytes and glucose level, and can be obtained quickly and easily via a peripheral line.
– **Arterial blood gas analysis**: Provides accurate information on arterial oxygen and carbon dioxide levels as well as acid-base balance but it requires expertise and it is a painful procedure.

**SUMMARY OF KEY POINTS**

- Supplementary oxygen should be given to maintain saturations greater than 95% in all children with respiratory distress.
- Hypoxaemia is a late sign of upper airway obstruction and the child is at risk of sudden and complete airway obstruction. Call a Rapid Response.
- Increasing oxygen requirement in order to maintain adequate saturations, is a sign of serious deterioration, and requires a Clinical Review or Rapid Response, depending on severity.
- Decreasing respiratory rate, and/or increasing apnoeas, and a reduced level of consciousness are signs of fatigue. Exhaustion is a pre-arrest sign. Call a Rapid Response call.

**POLICY DIRECTIVES AND CLINICAL RESOURCES**

Detailed information on the assessment and management of specific respiratory conditions and relevant illnesses can be obtained from the following *Clinical Practice Guidelines*:

- PD2010_053  Infants and Children: Acute Management of Croup
- PD2012_056  Infants and Children: Acute Management of Asthma
- PD2012_004  Infants and Children: Acute Management of Bronchiolitis
- PD2010_063  Infants and Children: Acute Management of Fever

All Clinical Practice Guidelines can be accessed via the *NSW Health Intranet* website:

- Go to Quick Links: *Child Health Networks*
- Go to Links & Resources: *NSW Health Paediatric Clinical Practice Guidelines*

or via the *NSW Health Internet* website (for ‘off site’ access):

# REFERENCES


7. Sydney Children’s Hospital, Guideline for the care of paediatric patients receiving humidified oxygen on the ward’, 2011, Reference: SCHR.C.10.08.


AIM

The aim of this chapter is to give you the knowledge to be able to:

- Understand the simple pathophysiology and causes of shock (i.e. cardiovascular deterioration)
- Identify, interpret and evaluate the early and late warning signs of shock
- Assess and prioritise immediate and early management of shock
- Escalate concerns to Clinical Review or Rapid Response as appropriate

Throughout the chapter ‘child’ refers to infant, child or adolescent unless otherwise stated.

### Early Warning Signs of Shock

<p>| | |</p>
<table>
<thead>
<tr>
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<tbody>
<tr>
<td>Moderate tachycardia, increasing trend</td>
<td>Capillary refill 3 seconds or more</td>
</tr>
<tr>
<td>Cool extremities, mottled skin, pallor</td>
<td>Reduced urine output</td>
</tr>
<tr>
<td>Moderate tachypnoea, increasing trend</td>
<td>Lethargy, general malaise or mild agitation</td>
</tr>
</tbody>
</table>

*Table 1*

### Late Warning Signs of Shock

<p>| | |</p>
<table>
<thead>
<tr>
<th></th>
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</tr>
</thead>
<tbody>
<tr>
<td>Marked tachycardia or any bradycardic episode</td>
<td>Weak or absent peripheral pulses</td>
</tr>
<tr>
<td>Cold and clammy, extreme pallor, cyanotic or grey appearance</td>
<td>Hypotension</td>
</tr>
<tr>
<td>Marked tachypnoea Apnoeic episodes</td>
<td>Hypotonia Decreased level of consciousness</td>
</tr>
</tbody>
</table>

*Table 2*

Concern by any staff or family member irrespective of early or late warning signs should be appropriately escalated.
PATHOPHYSIOLOGY OF CARDIOVASCULAR DETERIORATION

ANATOMICAL AND PHYSIOLOGICAL DIFFERENCES BETWEEN CHILDREN AND ADULTS

- The circulating blood volume per kg of a child is higher than that of an adult (70-85mL/kg compared to 60mL/kg) but the actual volume is much smaller. The implication of even a small loss in blood volume in children is significant. A neonate, with an average weight between 3.5 - 4.5kg will only have a total circulating blood volume of 300 – 380mL.

- Compared to adults, cardiac arrest in children is less often a result of a primary cardiac disease, and more often the end result of a prolonged period of decompensation due to respiratory failure or circulatory failure. Ventricular fibrillation is much less common than asystole but does still occur in children.

- Adults have a greater ability to increase stroke volume to meet the demands of the body for an increased cardiac output compared to young children. Infants, in particular, rely on increasing their heart rate to increase cardiac output. Tachycardia at rest is an important sign of cardiovascular compensation and is one of the earliest signs of shock.

Cardiac Output = Stroke volume × Heart rate

Stroke volume depends on preload, contractility and afterload:

Preload = Volume of blood in the left ventricle after diastole
Contractility = Power of the cardiac muscle (myocardium)
Afterload = the force that needs to be generated by the left ventricle to eject blood

CIRCULATORY FAILURE (SHOCK) ¹²³

Circulatory failure or shock is a failure of the cardiovascular system to supply adequate amounts of oxygen to meet the needs of the body’s cells. Regardless of the underlying cause, the inadequate delivery of oxygen and nutrients via the bloodstream to meet the metabolic demands of the cells leads to anaerobic metabolism, lactic acidosis, and the loss of normal cellular and organ function.

Shock is a leading cause of morbidity and mortality in children.⁴ Multiple organ failure and death will occur if shock is not recognised and treated promptly and effectively.
THE 5 TYPES OF SHOCK

Hypovolaemic Shock

Hypovolaemic shock results from inadequate circulating (intravascular) blood volume either due to actual blood or fluid loss (e.g. haemorrhage, severe dehydration) or due to fluid maldistribution where fluid leaks out of the blood vessels into either the interstitial space (capillary leak) e.g. sepsis, burns; or a body cavity (third spacing), e.g. the bowel in intestinal obstruction.

Distributive Shock

Distributive shock is a result of dilation of multiple blood vessels in response to a massive inflammatory stimulus, as occurs in sepsis or anaphylaxis, or due to a loss of normal vasomotor control e.g. following spinal injury. This creates a situation of relative hypovolaemia with reduced cardiac filling (preload) and a fall in blood pressure.

Cardiogenic Shock

Cardiogenic shock occurs due to impairment in cardiac contractility. The most common cause in adults is myocardial infarction, however, in children the primary causes are congenital heart disease in newborns, or acquired heart disease, such as myocarditis or cardiomyopathy, in older infants and children.

Obstructive Shock

Obstructive shock is caused by obstruction of blood flow to or from the heart. It can result from either an acute event, such as tension pneumothorax, or from an obstructive congenital heart defect e.g. Coarctation of the aorta, especially in neonates within the first two weeks of life (refer to Congenital Heart Disease p 79).

Dissociative Shock

Dissociative shock occurs when there is reduced oxygen carrying capacity of the blood to supply the cells with sufficient oxygen to meet their needs (e.g. profound anaemia) or when the cell’s ability to take up oxygen is impaired (e.g. irreversible ‘refractory’ shock).

In some cases, several types of shock may co-exist. For example, a child with sepsis and widespread vasodilation (distributive shock), will likely have intravascular volume depletion from ‘capillary leak’ and/or reduced oral intake (hypovolaemic shock), reduced cardiac function due to myocardial suppression caused by the infection (cardiogenic shock) and, if shock persists, impaired cellular oxygen utilisation from mitochondrial dysfunction (dissociative shock).
STAGES OF SHOCK

Shock is a progressive syndrome which becomes less responsive to treatment the longer it is allowed to persist. It can be divided into 3 phases:

Phase 1: Compensated Shock

- The heart and blood vessels respond to a situation of impaired tissue oxygen delivery and compensate by:
  - increasing heart rate to increase cardiac output
  - altering vascular tone to divert blood from nonessential tissues to the vital organs i.e. brain, heart
- Recognition of compensation and the early warning signs *(Table 1)* and appropriate management at this stage will reverse shock completely.

Phase 2: Decompensated Shock

- Compensatory mechanisms are no longer able to meet cellular demands for oxygen and evidence of inadequate tissue oxygen delivery become apparent.
- If the late warning signs *(Table 2)* are recognised at this stage, shock may still be reversible if treated appropriately.

Phase 3: Irreversible (Refractory) Shock

- Damage to cell apparatus and vital organs begins to occur, and shock is now irreversible even with the restoration of adequate oxygen delivery.

Shock is a medical emergency. The longer it is left untreated, the greater the chance that it will become refractory and lead to death. Early recognition and urgent treatment is vital.

SPECIAL CONSIDERATIONS IN CARDIOGENIC SHOCK (ACUTE HEART FAILURE)

When treating any infant or child with shock, it is important to determine whether the underlying cause is cardiac or non-cardiac in origin. *Failure to improve following a fluid resuscitation bolus may be indicative of cardiogenic shock.* Correct identification of cardiogenic shock is essential to avoiding excess fluid resuscitation and ensuring appropriate ongoing management.

Cardiogenic shock has specific clinical features that are suggestive of a cardiac aetiology. ³

Symptoms may include:

- Shortness of breath with fine crackles and wheezing (pulmonary oedema)
Difficulty feeding in infants with sweating, breathlessness and irritability

- Failure to thrive/anorexia
- Fatigue, weakness, exercise intolerance and peripheral oedema in older children

*Signs* may include:

- Tachycardia *disproportionate to degree of physical exertion*
- Gallop rhythm – an extra 3rd heart sound
- Enlarged heart (cardiomegaly)
- Enlarged liver (hepatomegaly)
- Raised jugular venous pressure (difficult to assess in infants)
- Absent femoral pulses in obstructive congenital heart defects

**Congenital Heart Disease**

Congenital heart disease (CHD) is the most common cause of heart failure in infants. Neonates with a particular subset of congenital heart defects may appear well at birth but can present acutely unwell with signs of shock within the first two weeks of life.

This subset of congenital heart defects is referred to as duct-dependent CHD, where pulmonary or systemic blood flow is reliant on the patency of a blood vessel called the ductus arteriosus. This ‘duct’ is present during foetal life and plays an essential role in foetal circulation. It begins to close in the first few days after birth and it is during this period, that neonates with undiagnosed duct-dependent CHD, will start to develop acute symptoms of heart failure (+/- cyanosis). By the time they present to hospital they may already be in cardiogenic shock.

*Figure 1: Patent Ductus Arteriosus*

Immediate life saving treatment involves Prostaglandin E1 (Alprostadil) to maintain patency of the ductus arteriosus, in addition to routine resuscitation (*refer to p 85*) and should be commenced as soon as the diagnosis of duct-dependent CHD is suspected, or in any newborn infant under 2 weeks of age.
age presenting with severe shock in whom the cause is unknown. Concern must be escalated immediately to a senior paediatrician or neonatologist.

Duct-dependent CHD should be suspected in any neonate less than 2 weeks of age presenting with signs of shock. Prostaglandin E1 (Alprostadil) infusion should be commenced as part of initial resuscitation.

Cardiac arrhythmias

Cardiac arrhythmias can result in an inability of the heart to pump effectively resulting in cardiogenic shock. Arrhythmias can be caused by a number of factors. In adults, they are most often caused by cardiac events, such as a myocardial infarction. In children the cause is much more likely to be non-cardiac. Congenital and acquired heart disease can both predispose to arrhythmias; however, major causes include hypoxia, electrolyte disturbances, and intoxication.

Cardiac arrhythmias may be classified as bradyarrhythmias (slow rhythms) and tachyarrhythmias (fast rhythms).

Bradyarrhythmias

The most common bradyarrhythmia in children is sinus bradycardia defined as less than 60 bpm, or a rapidly falling heart rate. Whilst primary bradyarrhythmia may occur (e.g. complete heart block), bradyarrhythmia in children most often occurs secondary to hypoxia or other serious illnesses and should always be considered a pre-arrest sign.

Tachyarrhythmias

Sinus tachycardia is the body’s normal response to fever, pain, excitement, anxiety and exercise but as discussed above it also occurs as early compensation for a reduced cardiac output. It is important that the cause of any observed tachycardia, particularly if present at rest, is determined so that it may be treated promptly and appropriately.

Supraventricular tachycardia (SVT) is the most common tachyarrhythmia in children. It can occur in very young infants with structurally normal hearts (more than 30% of cases are within the first few weeks of life) or in adolescents and people with underlying heart disease.

SVT can be differentiated from sinus tachycardia as it is typically characterised by a heart rate of more than 220 bpm, often going as high as 250-300 bpm. An infant is likely to be irritable with poor feeding and tachypnoea. Heart failure, and shock, with decreasing level of consciousness will develop if left untreated.
CAUSES OF SHOCK

*Sepsis is the leading cause of shock* in children. Hypovolaemic shock due to *gastroenteritis, intestinal obstruction or haemorrhage* is the second most common cause followed by distributive shock and, least common, cardiogenic shock.  

**Possible Diseases** | **Comments**
--- | ---
**Hypovolaemic shock (reduced intravascular volume)**
Haemorrhage *(Haemorrhagic shock)*  
e.g. Trauma, Surgery, GI bleed  
- A rapid loss of 25% of circulating blood volume (i.e. 20mL/kg) in an infant will cause shock e.g. 60mL in an average 3.5kg neonate

Gastroenteritis  
- Infants and young children have increased susceptibility to gastroenteritis and may lose 10-20% of their intravascular volume within 1-2 hours

Intestinal obstruction  
e.g. Intussusception, Volvulus, Hirschsprungs disease, peritonitis  
- Persistent vomiting with bile, usually with abdominal distention, is a sign of intestinal obstruction and requires immediate resuscitation and urgent surgical review  
- Intussusception is a common cause of intestinal obstruction particularly in children between 3 months and 2 years  
- It typically presents with a triad of colicky abdominal pain (knees typically drawn up and hips flexed whilst crying inconsolably), bilious vomiting, and "redcurrant jelly" stool

Burns  
- Loss of plasma volume after a burn injury is rapid as fluid leaks into the burn area and is lost from the body  
- Assessment of fluid status is difficult as severely burned patients may appear "puffy" or oedematous whilst actually being significantly intravascularly depleted. Presence or absence of oliguria is often a useful sign in these patients

**Distributive shock (widespread vasodilation)**

Sepsis *(Septic shock)*  
- Sepsis is most common in infants and carries significant mortality and morbidity  
- Fever is not always present and children may be normothermic or even hypothermic  
- Non-blanching rashes - petechial (small red/purple spots) or purpuric (larger red/purple areas) may be a feature of some infections
### Possible Diseases

<table>
<thead>
<tr>
<th>Possible Diseases</th>
<th>Comments</th>
</tr>
</thead>
</table>
| **Anaphylaxis** *(Anaphylactic shock)*  
e.g. food, medications, blood, insect stings |  
- A history of previous anaphylaxis may be present  
- Life threatening features can include respiratory difficulties with stridor or wheeze and shock due to vasodilation and fluid loss from capillary leak  
- Typical symptoms of an allergic reaction such as urticaria, angioedema, sweating, nausea, abdominal pain *may not be present* |
| **Spinal injury** *(Neurogenic shock)* |  
- Spinal injury can interrupt sympathetic input to vasomotor neurons resulting in generalised vasodilation  
- Hypotension and bradycardia are characteristic features  
- It is the rarest cause of shock |
| **Cardiogenic Shock (cardiac dysfunction)** |  
- Duct dependent CHD typically present in the first 2 weeks of life as the duct closes  
- Cardiomyopathy & myocarditis are rare but signs include acute onset of heart failure/shock and/or arrhythmias with no previous cardiac history  
- Cardiomegaly is typically seen on CXR |
| **Obstructive Shock (cardiac obstruction)** |  
- Immediate history is important for diagnosis – trauma/cardiothoracic surgery/lung disease  
- Symptoms are *acute and severe* and may include sudden onset of respiratory difficulty, tachycardia, hypotension |
| **Dissociative shock (reduced oxygen carrying capacity)** |  
- Severe anaemia: Hb less than 50g/L  
- Presentation may include cyanosis, dyspnoea, lethargy, headache, dizziness, altered mental status, or stupor  
- $\text{SpO}_2$ may not be reliable in patients with dissociative shock. Arterial blood gas monitoring should be undertaken |

### RECOGNITION OF SHOCK

Early diagnosis of shock may be difficult as symptoms can often be non-specific in nature e.g. general malaise, pallor, fever, poor feeding and reduced urine output. A strong index of suspicion and a focused clinical history to identify key features will assist with early recognition and diagnosis.
Take note of increased respiratory rate and grunting respirations in any child presenting with signs of shock as this may imply the development of pulmonary oedema from either capillary leak, as occurs in sepsis or anaphylaxis, or from cardiac failure (i.e. cardiogenic shock).

SUMMARY OF CARDIOVASCULAR ASSESSMENT

<table>
<thead>
<tr>
<th>Assessment</th>
<th>Action</th>
<th>Warning signs</th>
</tr>
</thead>
<tbody>
<tr>
<td>Heart rate</td>
<td>Palpate pulses and count for a full minute wherever possible</td>
<td>Tachycardia or increasing trend</td>
</tr>
<tr>
<td></td>
<td>Check the trend and any alterations to calling criteria</td>
<td>Any bradycardic episodes</td>
</tr>
<tr>
<td>Pulse volume</td>
<td>Compare the volume of the peripheral and central pulses</td>
<td>Weak /absent peripheral pulses</td>
</tr>
<tr>
<td></td>
<td>In a neonate, ensure femoral pulses are palpable</td>
<td>Absent femoral pulses in a neonate</td>
</tr>
<tr>
<td>Capillary refill time (CRT)</td>
<td>Press for 5 seconds on the centre of the sternum, release and count</td>
<td>CRT 3 seconds or more</td>
</tr>
<tr>
<td></td>
<td>the seconds for skin colour to return</td>
<td>Mottled or pale skin with cool peripheries</td>
</tr>
<tr>
<td>Blood pressure</td>
<td>Take a manual recording wherever possible</td>
<td>Hypotension is a late warning sign</td>
</tr>
<tr>
<td></td>
<td>Use an appropriate size cuff i.e. ⅔ of the upper arm</td>
<td>Wide pulse pressure (diastolic pressure less than 50% of</td>
</tr>
<tr>
<td></td>
<td></td>
<td>systolic value) may be a sign of distributive shock</td>
</tr>
<tr>
<td>Effects of cardiovascular</td>
<td>Assess respiratory system</td>
<td>Tachypnoea</td>
</tr>
<tr>
<td>failure</td>
<td></td>
<td>Wheezing</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Grunting</td>
</tr>
<tr>
<td></td>
<td>Assess neurological status</td>
<td>Lethargy/malaise</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Agitation/confusion</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Reduced level of consciousness</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Hypotonia</td>
</tr>
<tr>
<td></td>
<td>Assess urine output</td>
<td>Oliguria (less than 1 mL/kg/hr in infants</td>
</tr>
<tr>
<td></td>
<td></td>
<td>and less than 0.5 mL/kg/hr in older children)</td>
</tr>
</tbody>
</table>

*Table 4*

For a detailed assessment of cardiovascular status refer to Chapter 1 pp 14-18.

The most sensitive indicator of shock in children is the heart rate. However, as previously mentioned, many factors can elevate a child’s heart rate, therefore, other signs of early compensated shock need to be sought.

Compensated shock is indentified by poor perfusion with the maintenance of an adequate blood pressure. In the later stages, decompensated shock is represented by the development of hypotension.
Hypotension and/or absent peripheral pulses are late signs of shock and indicate a pre-arrest state. Escalate to a Rapid Response call.

INITIAL MANAGEMENT OF SHOCK

Early recognition and urgent resuscitation to reverse the shocked state has been shown to vastly improve mortality rates in infants and children.

A reduction in heart rate is usually the first sign of reversal of shock followed by an improvement in perfusion and urine output.

Caregivers should normally be allowed to remain with their child, and should be kept well informed with appropriate support. If possible, assign a member of the team or request the presence of a social worker.

A Start with the Airway

- If the conscious level is reduced due to a shocked state and the child is having difficulty maintaining their own airway, use simple airway opening manoeuvres to open and maintain the airway (refer to Chapter 1 p 8). Call a Rapid Response and prepare for intubation following initial fluid resuscitation.

Ensure fluid resuscitation is commenced prior to intubation, as anaesthetic agents and mechanical ventilation may cause hypotension in a shocked patient.

B For Breathing

- Commence high flow oxygen via a non rebreather facemask at a flow rate of 10-15 L/min if there are any signs of inadequate oxygenation, as indicated by increased respiratory rate/effort and acute hypoxaemia (SpO₂ of less than 95%).

All children with shock should receive high flow oxygen to maintain SpO₂ above 95%.


- If grunting respirations are present with signs of shock, call a Rapid Response and prepare to provide respiratory support, and possible intubation by an experienced practitioner.
For Circulation

- Attach an ECG monitor. If any abnormal rhythm is noted on the cardiac monitor perform a 12 lead ECG without compromising definitive treatment of shock i.e. fluid resuscitation.

- Document and monitor trends in heart rate, blood pressure, capillary refill and presence of peripheral and central pulses, particularly in response to treatment (i.e. before and after fluid bolus).

- Establish IV access (x 2 wherever possible) and take bloods for blood gas analysis (including lactate), glucose, blood culture and cross-match. If sufficient sample, request Full Blood Count, Urea and Electrolytes, Calcium, Magnesium, Liver Function Tests (LFT’s), and coagulation studies if sepsis is suspected.

- If IV access is difficult due to poor perfusion insert an Intraosseous (IO) needle – do not delay treatment. For information on IO needle insertion refer to Appendix 2.

If IV access is not achieved within a maximum of 2 attempts in a shocked or critically ill child insert an Intraosseous (IO) needle.

FLUID RESUSCITATION FOR SHOCK

1. Administer an IV bolus of 20 mL/kg of 0.9% Sodium Chloride as a ‘push’ (i.e. within 5 minutes).

   A contra-indication to this is cardiovagenic shock – consider 5 -10 mL/kg bolus and evaluate response to fluid bolus prior to any repeat.

2. ASSESS for signs of improvement to circulation i.e. decreased heart rate, more easily palpable peripheral pulses and improved perfusion, urine output and improving level of consciousness prior to any further boluses.

3. Provide a second IV fluid bolus of 20 mL/kg of 0.9% Sodium Chloride if symptoms of shock persist or recur after initial improvement.

4. REASSESS. If there are no signs of improvement following the second bolus of fluid consider:
   - Inotrope infusion via peripheral vein access
   - Colloid i.e. 4.5% albumin (since approximately 50% of the child’s circulating blood volume has already been given)
Anticipate the need for a Red Blood Cell transfusion following repeated fluid boluses due to haemodilution. Ensure a cross match has been sent and monitor the Haemoglobin level (or haematocrit (HCT)) – aim for more than 30%.

Maintain close observation of acid-base status and electrolytes (particularly serum potassium and calcium level).

If two fluid boluses have been administered (i.e. 40 mL/kg) and signs of shock are ongoing escalate to a Rapid Response and prepare for intubation and inotropes.

SPECIAL CONSIDERATIONS IN THE MANAGEMENT OF DIFFERENT TYPES OF SHOCK

Hypovolaemic shock: haemorrhage

- Control the bleeding wherever possible and call a Rapid Response and the surgical/trauma team.
- Always attempt to gain an accurate record of actual blood loss using pads/dressings that can be weighed.
- Consider internal bleeding if signs of shock are present following trauma/surgery with no visible bleeding.

Distributive shock: sepsis

- Administer broad-spectrum antibiotics appropriate for age and likely focus as soon as possible after IV/IO access and within one hour.
- Aggressive resuscitation (i.e. early administration of large volumes more than 40 mL/kg in first hour) has been associated with improved outcomes.

A Paediatric Sepsis Toolkit is due to be released in April 2013. Refer to the CEC Sepsis webpage: Resources via http://www.cec.health.nsw.gov.au/programs/sepsis

Distributive shock: anaphylaxis

- Prepare to administer intramuscular Adrenaline 10 microg/kg i.e. 0.01mL/kg of 1:1000 (neat) as soon as possible. Repeat in 3 - 5mins.

Cardiogenic shock: duct-dependent CHD

- If cyanosis is present, oxygen therapy will not significantly improve SpO₂ – provide enough oxygen to increase saturations and do not use high flow oxygen.
- Commence IV infusion of Prostaglandin E1 (Alprostadil) at an initial dose of 5 nanogram/kg/min and up to 20 nanogram/kg/min to reopen the duct. *Prostaglandin E1 can cause vasodilation and subsequent hypotension* - prepare to give a fluid bolus. It may also cause apnoea - ensure adequate monitoring and appropriate resuscitation equipment is available.

- Perform 12 lead ECG and CXR and seek cardiology advice immediately.

**Cardiogenic shock: acquired heart disease**

- Consider early use of inotropes and diuretics. Dobutamine may be administered via a peripheral IV at a dose of 5-20 microg/kg/min.

- Perform 12 lead ECG and CXR and seek cardiology advice immediately.

**Cardiogenic shock: arrhythmias**

- Conduct urgent analysis of serum potassium, calcium and magnesium levels and prepare to correct levels as appropriate.

- Perform 12 lead ECG and seek cardiology advice immediately.

**D For Disability (neurological)**

- Maintain a continual assessment of the child’s level of consciousness (alertness and arousal) using the AVPU scale.

- Consider spinal shock if there is any history of major trauma and hypotension and/or widened pulse-pressure with bradycardia.

- Consider the possibility of raised intracranial pressure in any child with a history of meningitis (refer to Chapter 5 p 100).

- Call for a *Rapid Response* immediately if the child is responsive only to *Pain* or is *Unresponsive* or if any concern regarding a deteriorating level of responsiveness.

**E For Exposure**

- Document and monitor trend in axillary temperature.

- If the cause of shock is unknown, in conjunction with routine resuscitation:
  - Check for any *blanching or non-blanching rashes* particularly purpuric rashes, even if absent on initial examination (refer to Chapter 1 p 25)
  - Check for any *active bleeding* – expose the child and check any drains for excess fluid loss or
sudden cessation of drainage

– Check for an **acute (surgical) abdomen** - examine the abdomen for signs of distension and tenderness and consider an abdominal X-ray or ultrasound

– Consider a chest X-ray following initial resuscitation to look for **cardiomegaly, pulmonary oedema or pneumothoraces**

| Serious infection may not always be accompanied by fever in neonates and infants. Hypothermia or a normal temperature may be present. |

### **F**or **F**luids

- Cease enteral feeding in any infant or child with shock and place nil by mouth (NBM).

- Following initial resuscitation commence IV maintenance fluids as per the recommended NSW guidelines *(refer to Chapter 6 Table 8 p 128)* and appropriate to latest blood chemistry levels.

- Maintain an accurate fluid balance **ensuring an infusion pump and burette is used at all times.**

- Hypokalaemia may be present, particularly in septic shock, and potassium may need to be added to the maintenance fluids once urine output is established.

| Insert a urinary catheter in any child with persistent (refractory) shock i.e. requires more than 40 mL/kg fluid resuscitation, and monitor hourly urine output. |

### **G**or **G**lucose

- Always check the blood glucose level in any child with shock as hypoglycaemia is common in these children.

- If hypoglycaemia is present i.e. BGL less than 3.0 mmol/L or symptomatic *(refer to Chapter 1 p 29)* call for a Clinical Review and prepare to administer a bolus of 2 mL/kg of 10% Glucose as prescribed.

- Ensure maintenance fluids contain 5 - 10% Glucose.

- Repeat BGL within 5-10 minutes following administration of a glucose bolus.

| If BGL is less than 2.0 mmol/L call for a Rapid Response and prepare to administer a bolus of 2 mL/kg of 10% Glucose. |
ONGOING MANAGEMENT

Without delaying initial resuscitation, any child with shock should be escalated to senior members of the local team, and paediatric expertise should be sought. Where appropriate, consideration should be given to consulting with state emergency and/or intensive care services via NETS for advice and possible transfer to a tertiary paediatric centre. Refer to Appendix 1 for information on Interhospital transfer.

NETS hotline telephone number is 1300 36 2500 - Early referral to NETS (or a local retrieval service) is encouraged for clinical support, advice and/or to activate a medical retrieval.

SUMMARY OF KEY POINTS

✓ Aggressive early fluid resuscitation with septic shock and cautious fluid resuscitation with cardiogenic shock. Always reassess following any fluid bolus.

✓ Give antibiotics early (within one hour) if septic shock is a possibility.

✓ In any neonate with shock of unknown cause, commence IV infusion of Prostaglandin E1 (Alprostadil) and seek urgent cardiology/neonatal review or NETS advice.

✓ Remember to check the blood glucose level as hypoglycaemia is common.

POLICY DIRECTIVES AND CLINICAL RESOURCES

Detailed information on the assessment and management of relevant illnesses can be obtained from the following Clinical Practice Guidelines:

PD2012_065  Infants and Children: Acute Management of Bacterial Meningitis
PD2010_009  Infants and Children: Acute Management of Gastroenteritis
PD2005_384  Infants and Children: Acute Management of Acute Abdominal Pain
PD2010_063  Infants and Children: Acute Management of Fever

All Clinical Practice Guidelines can be accessed via the NSW Health Intranet website:

– Go to Quick Links: Child Health Networks
– Go to Links & Resources: NSW Health Paediatric Clinical Practice Guidelines

or via the NSW Health Internet website (for ‘off site’ access):

### REFERENCES


The aim of this chapter is to provide you with the knowledge to:

- Understand the simple pathophysiology and the causes of neurological deterioration and altered conscious state.
- Identify, interpret and evaluate the early and late warning signs of neurological deterioration.
- Assess and prioritise immediate and early management of neurological deterioration.
- Escalate concerns to Clinical Review or Rapid Response as appropriate.

Throughout the chapter ‘child’ refers to infant, child or adolescent unless otherwise stated.

### Early Warning Signs of Neurological Deterioration

<table>
<thead>
<tr>
<th>Sign</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>Altered mental state</td>
<td>Irritability, agitation, combativeness, inconsolability, increased lethargy</td>
</tr>
<tr>
<td>Decrease in level of consciousness</td>
<td>From alert (A) to rousable only by voice (V) on the AVPU scale</td>
</tr>
<tr>
<td>Glasgow Coma Scale (GCS) or modified (child) GCS score of less than 15</td>
<td>Any seizure, even if short-lived and spontaneously resolved</td>
</tr>
</tbody>
</table>

*Table 1*

### Late Warning Signs of Neurological Deterioration

<table>
<thead>
<tr>
<th>Sign</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>Responding only to pain (P) or unresponsive (U) on the AVPU assessment</td>
<td>New or prolonged seizure activity (defined as longer than 5 minutes) or any seizure with airway compromise</td>
</tr>
<tr>
<td>Sudden decrease in level of consciousness (defined as 2 or more on the GCS or modified GCS) or GCS score 8 or less</td>
<td>Serious concern by any staff member or caregiver regarding mental status</td>
</tr>
</tbody>
</table>

*Table 2*

Concern by any staff or family member irrespective of early or late warning signs should be appropriately escalated.
PATHOPHYSIOLOGY OF NEUROLOGICAL DETERIORATION

ANATOMICAL AND PHYSIOLOGICAL DIFFERENCES BETWEEN CHILDREN AND ADULTS

- At birth the sutures between the cranial bones are open to allow for movement of the bones during childbirth and then to allow for brain growth during infancy.

- The cranial sutures remain unfused for about 12-18 months. Between the bones, at the front and back of the skull, lie two ‘soft spots’ - the anterior fontanelle and the posterior fontanelle.

- The posterior fontanelle usually closes around 1-2 months but the anterior fontanelle remains open for up to 9-18 months.

- The anterior fontanelle is clinically useful. A sunken fontanelle indicates dehydration, whereas a tense or bulging fontanelle can indicate raised intracranial pressure.

RAISED INTRACRANIAL PRESSURE

Intracranial pressure (ICP) is the pressure within the cranial cavity and is influenced by the volume of the three intracranial components: brain mass (80%), blood volume (10%) and cerebrospinal fluid (CSF) (10%), an interrelationship known as the Monro-Kellie doctrine.\(^1\,2\)

The Monro-Kellie doctrine states that, as the skull is a rigid structure and unchangeable, any increase in volume to one of the intracranial components must lead to a reduction in volume of one or both of the other two components in an attempt to maintain a normal ICP (obviously excluding the brain as this cannot reduce in size). Blood volume is reduced through vasoconstriction and CSF is effectively shunted away to the venous system or spinal cord.\(^1\) Raised ICP occurs when an increase in
the volume of one component cannot be offset by a volume reduction in one of the other components.  

Many types of conditions can disrupt the volume equilibrium between these three components:  

Conditions leading to an increase in tissue (brain) volume:

- Cerebral oedema due to a head trauma, hypoxic/ischaemic brain injury, meningitis/encephalitis, metabolic condition such as Diabetic Ketoacidosis, or electrolyte imbalance such as hyponatraemia
- Intracranial haemorrhage or extracerebral haematoma
- Brain lesion such as tumour

Conditions leading to an increase in blood volume:

- Seizure activity
- Hypercarbia/acidosis

Conditions leading to an increase in CSF volume:

- Hydrocephalus

Despite the presence of the open sutures and fontanelle in infants, little protection to raised ICP is afforded by an infant’s cranial and cerebral anatomy.  

Raised ICP in any age is life threatening and can lead to brain injury or herniation. Early recognition and urgent treatment is vital.

**SEIZURES**

Seizures (also known as convulsions or fits) can manifest in many different ways, from simple “absence” seizures to generalised tonic-clonic seizures (stiffening-jerking). More subtle generalised seizures that may not be tonic-clonic may occur, e.g. eye flickering in a baby. This is usually accompanied by autonomic signs such as dilated pupils, tachycardia and hypertension.

Many acute conditions and illnesses, not necessarily restricted to neurological disorders, can trigger a seizure, such as a high fever (febrile convulsion), infection, hypoglycaemia, hypoxia, metabolic disorders, toxins, and certain medications. These forms of seizures often cease when the underlying condition is treated.

A single seizure that results from such a stimulus is called a provoked or non-epileptic seizure, and in
50% of children these are isolated events. However, if seizures continue or re-occur, they may indicate an underlying seizure disorder or epilepsy, and an increased risk of seizures occurring even without an obvious stimulus.

Most seizures in children are brief and stop spontaneously without requiring any treatment. Seizures that last more than 5 minutes are considered prolonged and must be treated promptly with anti-epileptic therapy. It is important to attempt to control the seizure without delay as the longer the seizure continues the more difficult it becomes to control.

Seizures are followed by a postictal state, characterised by altered level of consciousness, such as confusion and drowsiness, nausea, headache and hypertension. This period can last from several minutes up to hours, depending on the length and severity of the seizure. Emergence from this period is often accompanied by memory loss as the brain recovers from the trauma of the seizure. For any unexplained changes in level of consciousness consider a postictal state following an unwitnessed seizure.

**STATUS EPILEPTICUS**

Status epilepticus is either a seizure lasting longer than 30 minutes or repeated seizures with no recovery of consciousness in between. It is most common in children younger than 2 years with the majority being generalized tonic-clonic seizures.

*Nonconvulsive status epilepticus* may also occur. This is a state of prolonged seizure activity that involves behavioural and cognitive changes but no generalized tonic-clonic manifestations. Nonconvulsive status epilepticus can occur as a result of a range of conditions, and diagnosis can often be difficult. An increased awareness should be maintained in children with a history of epilepsy that have any prolonged change in behaviour or personality, any prolonged postictal confusion or any recent onset of psychosis (characterised by confusion, delusions and hallucinations). Suspicion of nonconvulsive status epilepticus should be confirmed with an electroencephalogram (EEG) to allow for urgent management.

Any prolonged convulsion increases the risk of airway obstruction, hypoxia and aspiration, and can cause cardiac arrhythmias and pulmonary oedema. If the convulsion continues it will lead to hypotension and subsequent drop in cerebral blood flow, lactic acidosis and cell death.

Prolonged seizures that are unresponsive to first line treatment, or recurrent short seizures without complete recovery, must be escalated to a Rapid Response immediately.
DELIRIUM

Delirium is a state of acute confusion that can be caused by an underlying disease process such as an infection; metabolic or autoimmune disorders; medications such as anticholinergic agents and opioids; surgery; or trauma. Delirium in children is most often seen with fever and can be expected to improve as the temperature returns to normal.

Delirium is characterised by alterations in level of consciousness, varying degrees of cognitive impairment such as poor concentration, reduced responsiveness and attention deficit, and behavioural changes ranging from restlessness, agitation and aggression to withdrawal and apathy. Delusions and hallucinations may also develop. Often these signs vary, e.g. periods of agitation can alternate with periods of reduced level of consciousness.

Delirium is most common in young children, elderly adults and those with previous brain damage, as the brain is most susceptible when it is first developing, deteriorating or previously damaged. In most cases, depending on the cause, delirium is temporary and recovery is expected when the underlying cause is treated successfully.

The confused state can vary in duration from several days to several weeks depending upon the severity of the underlying illness, the child’s age, and the child’s physical condition. Prognosis depends on successful treatment of the causative disease and the underlying state of the brain.

Delirium without fever is a medical emergency. You must escalate care using your local Rapid Response system.
CAUSES OF NEUROLOGICAL DETERIORATION

Altered level of consciousness includes a spectrum of disorders that range from mild confusion to delirium to deep coma. However, these changes in conscious level are not disease entities in themselves, but are manifestations of a wide variety of conditions that may be acute or chronic, mild or profound. The origins of an altered conscious level can be due to disease, trauma or intoxication and can originate from either intracranial or systemic causes.

**INTRACRANIAL CAUSES OF NEUROLOGICAL DETERIORATION**

<table>
<thead>
<tr>
<th>Disease</th>
<th>Comments</th>
</tr>
</thead>
</table>
| Intracranial infection/inflammation e.g. bacterial meningitis, viral encephalitis | • *This is the most common cause of non traumatic coma* in children accounting for 38% of cases  
  • Classic signs (i.e. neck stiffness, bulging fontanelle, high pitched cry) may often be absent in infants  
  • Less common conditions are acute disseminated encephalomyelitis (ADEM) and other inflammatory conditions causing encephalopathy e.g. systemic lupus erythematosus (SLE) |
| Intracranial haemorrhage/haematoma           | • The main cause of intracranial haemorrhage in children is traumatic brain injury (TBI). If it occurs in early childhood (under 2yrs) a common cause is Non Accidental Injury (NAI)  
  • Spontaneous cerebral haemorrhage may also occur in childhood. Possible causes include abnormalities of the blood vessels e.g. Arteriovenous Malformation (AVM) or aneurysm |
| Hydrocephalus                                 | • This may be *congenital* (present at birth) or acquired following TBI, intracranial haemorrhage or meningitis |
| Space occupying lesion                        | • This is often due to a tumour but can be caused by other pathologies such as an intracranial abscess |
| Seizures                                     | • *The most common cause in children is fever*  
  • Other causes include meningitis, Traumatic Brain Injury (TBI), hypoxia and metabolic conditions |

*Table 3*
# SYSTEMIC CAUSES OF NEUROLOGICAL DETERIORATION

<table>
<thead>
<tr>
<th>Disease</th>
<th>Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td>Systemic infection (septicaemia)</td>
<td>▪ Neurological dysfunction can be an early clinical feature of sepsis and may occur early or as the presenting feature. High fever in itself can cause delirium</td>
</tr>
</tbody>
</table>
| Circulatory Failure             | ▪ Shock from any cause will lead to a decreased brain perfusion and altered conscious level  
▪ Common causes of shock in children include haemorrhage, gastroenteritis, intussusception and sepsis  
▪ Severe hypertension can cause hypertensive encephalopathy |
| Toxins/Poisoning/Overdose        | ▪ Accidental ingestion of medication is most common at the toddler age  
▪ Attempted suicide or recreational use of drugs/alcohol is more likely to occur in the adolescent age group  
▪ Over sedation, often post operatively, leading to respiratory depression is more likely to occur in children with chronic neurological co-morbidities (e.g. cerebral palsy) |
| Metabolic conditions            | ▪ Hypoxia/ Hypercarbia  
▪ Hypothermia/Hyperthermia  
▪ Diabetic Ketoacidosis (DKA)  
▪ Hypoglycaemia  
▪ Electrolyte disturbances (hypo/hyponatraemia, hypercalcaemia)  
▪ Hepatic conditions or Inborn Errors of Metabolism causing hyperammonaemia  
▪ Reye’s Syndrome |

---

Consider hypercarbia, hypoglycaemia, hyponatraemia and narcosis in any child with an unexplained altered level of consciousness.

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**Table 4**
RECOGNITION OF NEUROLOGICAL DETERIORATION

Level of consciousness (LOC) and mental status (i.e. behaviour/level of activity/interaction and response to the environment and parents/caregivers) are the most important parts of neurological assessment. A change in either LOC or mental status is usually the first clue to a deteriorating condition. 16

The following terms are used to describe LOC and may assist in a neurological assessment, however, it is more important to recognise and appropriately describe the change in LOC rather than categorise it. 16

FULL CONSCIOUSNESS
The patient is alert, attentive, and follows commands. If asleep, they respond promptly to external stimulation and, once awake, remain attentive [scoring an A on the AVPU tool].

LETHARGY
The patient is drowsy but awakens—although not fully—to stimulation. They will answer questions and follow commands, but will do so slowly and inattentively [scoring a V on the AVPU tool].

OBTUNDATION
The patient is difficult to rouse and needs constant stimulation in order to follow a simple command. They may respond verbally with one or two words, but will drift back to sleep between stimulation [scoring a V or P on the AVPU tool depending upon the degree of stimulation required].

STUPOR
The patient rouses to vigorous and continuous stimulation; typically, a painful stimulus is required. They may moan briefly but do not follow commands. The only response may be an attempt to withdraw from or remove the painful stimulus [scoring a P on the AVPU tool].

COMA
The patient does not respond purposefully to continuous or painful stimulation and does not make any verbal sounds [scoring a U on the AVPU tool].
SUMMARY OF NEUROLOGICAL ASSESSMENT

<table>
<thead>
<tr>
<th>Assessment</th>
<th>Action</th>
<th>Warning signs</th>
</tr>
</thead>
<tbody>
<tr>
<td>Level of consciousness</td>
<td>Note general appearance, behaviour, level of activity, interaction</td>
<td>Irritability, agitation, combativeness, inconsolability.</td>
</tr>
<tr>
<td>(Alertness and arousal)</td>
<td>(refer to TICLS tool p 5).</td>
<td>Reduced responsiveness, lethargy.</td>
</tr>
<tr>
<td></td>
<td>Complete an appropriate neurological assessment tool: AVPU/GCS/Modified</td>
<td>Responding to Voice only on AVPU Reduction of 2 or more in GCS score</td>
</tr>
<tr>
<td></td>
<td>GCS (refer to p 19 &amp; 21)</td>
<td></td>
</tr>
<tr>
<td>Posture &amp; Tone</td>
<td>Note posture, tone and movement</td>
<td>Floppy (hypotonic) or increased tone (hypertonic)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Decorticate or decerebrate posturing Focal weakness, reduced movement</td>
</tr>
<tr>
<td>Pupil Reaction</td>
<td>Assess pupils for size, equality and reaction to light</td>
<td>Sluggish or unreactive to light Unequal or dilated pupils</td>
</tr>
<tr>
<td>Systemic effects of neurological</td>
<td>Assess respiratory system</td>
<td>Irregular breathing pattern Absent gag and/or cough reflex</td>
</tr>
<tr>
<td>failure</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Assess cardiovascular status</td>
<td>Hypertension/ bradycardia</td>
</tr>
</tbody>
</table>

Table 5

For a detailed assessment of neurological status and details of AVPU and Glasgow Coma Scale tools refer to Chapter 1 pp 18-23.

Caregivers know their child best. Ask them if they perceive any change in behaviour or responsiveness. Always escalate caregivers’ concerns appropriately.

RECOGNITION OF RAISED INTRACRANIAL PRESSURE

It is essential to promptly recognise the signs of raised intracranial pressure (ICP) in order to efficiently manage this life threatening condition.

Specific signs and symptoms may include: 17

- Lethargy/altered conscious state/altered behaviour
- Irritability/high pitched cry
- Poor feeding/ possible vomiting (often projectile)
- Bulging fontanelle
- Headache
- Visual disturbances e.g. blurred vision, blind spot (caused by papilloedema)
- Possible seizures
- Pupillary changes e.g. sluggish response to light, unequal pupils or gradual dilation
- Bradycardia-hypertension-irregular breathing (together known as Cushing’s Triad)*
- Fixed dilated pupils*
- Decorticate or decerebrate posturing* (refer to Chapter 1 p 22)

* Late signs

Helpful tips for assessing the anterior fontanelle
- To assess the anterior fontanelle palpate gently.
- In infants 6 months and older it is preferable to palpate the fontanelle when they are in the sitting position, and ideally when they are settled and content, as crying can cause the fontanelle to bulge.
- The fontanelle may be pulsatile and should appear flat and not bulging or tense

INITIAL MANAGEMENT OF NEUROLOGICAL DETERIORATION

Regardless of the underlying condition, the priorities of initial management are to establish a patent airway (A), administer supplemental oxygen or assisted ventilation if indicated (B) and support circulation (C). Once life-threatening conditions have been addressed, treat the underlying cause and continue to work through the ABCDEFG algorithm.

Life-threatening conditions should be treated as they are identified throughout the ABCDEFG assessment.

Remember to keep the caregivers close by and well informed. If possible, assign a member of the team to remain with them to provide continual support and reassurance or request a social worker if available.

A Start with the Airway

- As described in Chapter 3, any patient with a reduced level of consciousness is at risk of partial or complete airway obstruction. If the airway is compromised carry out airway opening manoeuvres – a head tilt-chin lift and/or jaw thrust (refer to Chapter 1 p 8) and escalate
immediately to a Rapid Response.

- Insert airway adjuncts to maintain a patent airway if necessary. Both an oropharyngeal airway (Guedel) and a nasopharyngeal airway (NPA) prevent the tongue from occluding the airway, a common cause of obstruction in unconscious children (refer to Chapter 3 p 67).

- If there is any history of trauma or any concern about the stability of the cervical spine maintain manual in-line immobilisation (i.e. stabilisation of the C-spine by hand as per Figure 2) with jaw thrust. Once a secure airway has been established, apply a cervical collar. Manual stabilisation must not be released prior to this.

![Figure 2: Manual in-line immobilisation with jaw thrust](image)

**For Breathing**

- Commence high flow oxygen via a non rebreather facemask at a flow rate of 10-15 L/min if there are any signs of inadequate oxygenation, as indicated by increased respiratory rate and/or effort and acute hypoxaemia (SpO₂ of less than 95%). **Rule out airway obstruction in any child with reduced level of consciousness and SpO₂ of less than 95% in room air.**


- If breathing is adequate and the child is unconscious place them in the recovery position (left lateral) and escalate to a Rapid Response.

![Figure 3: Recovery position: Left lateral](image)
If breathing is not adequate, as indicated by a decreased respiratory rate and/or effort (hypoventilation), poor chest movement, and decreased SpO₂, assist breathing with bag valve mask ventilation at a flow rate of 15 L/min and escalate to a Rapid Response.

<table>
<thead>
<tr>
<th>Indications for considering intubation:</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. GCS 8 or less</td>
</tr>
<tr>
<td>2. Loss of protective airway reflexes</td>
</tr>
<tr>
<td>3. Abnormal breathing pattern or hypoventilation</td>
</tr>
<tr>
<td>4. Inability to obtain saturations 92% or more in maximum high flow oxygen</td>
</tr>
</tbody>
</table>

C For Circulation

- Document and monitor trends in heart rate, blood pressure, capillary refill and presence of peripheral and central pulses.
- If there are any signs of shock, as indicated by tachycardia, hypotension, pallor, prolonged capillary refill and cool extremities, administer a bolus of 20 mL/kg of 0.9% Sodium Chloride.
- If sepsicaemia or meningitis is the suspected cause of neurological deterioration administer broad spectrum antibiotics, preferably after blood cultures but do not delay treatment.

D For Disability

- Maintain a continual assessment of the child’s level of consciousness (alertness and arousal) using the AVPU scale.
- If the child is responsive only to Pain or is Unresponsive on the AVPU scale, call for a Rapid Response and commence neurological observations using the GCS or modified GCS.
- Assess for signs of a raised ICP (refer to p100).
- If there are signs of a raised ICP escalate to a Rapid Response immediately. Treatment includes administration of Mannitol 20% IV (0.25 - 0.50 g/kg) over 20 minutes or undiluted 3% hypertonic Sodium Chloride (3 mL/kg) over 20 minutes.

Specific initial management of seizures:
- Note the time the seizure starts and finishes
- Position the child in the recovery position and provide airway support (jaw thrust) if necessary
Commence high flow oxygen via a non rebreather at a flow rate of 15 L/min

- If the seizure is prolonged (more than 5 mins) or involves any airway or haemodynamic compromise administer IV Midazolam 0.15 mg/kg [maximum 5 mg] as prescribed (or Diazepam 0.25 mg/kg IV [maximum 10 mg])
- If IV access is not present administer buccal or intranasal Midazolam 0.3mg/kg [maximum 10 mg] as prescribed (or Diazepam 0.5 mg/kg PR [maximum 10mg]
- Check blood glucose level
- If seizure continues for a further 5 minutes repeat dose of Midazolam (or Diazepam)
- Post seizure – maintain the child in recovery position and increase frequency of observations

If the seizure continues beyond 15 to 20 minutes despite appropriate therapy call a Rapid Response and assemble the team and equipment for possible intubation and ventilation.

For detailed information on the management of seizures refer to Clinical Practice Guideline PD2009_065 Infants and Children: Acute Management of Seizures.

Helpful tips when administering buccal midazolam

Buccal midazolam is administered into the buccal mucosa that lines the cheek.

1. Position the child on their left side in the recovery position.
2. Gently insert the syringe containing the correct midazolam dose into the space between the teeth and the cheek closest to the surface that the child is lying on.
3. Gently depress the plunger on the syringe until the dose has been completely administered into the lining of the cheek.
4. Maintain the child in the recovery position and if clinically appropriate refrain from performing oral suction for several minutes to enable maximum absorption of the dose.

E For Exposure

- Document and monitor trend in axillary temperature.
- Check the child for any new rashes particularly if the neurological deterioration is unexplained or unexpected (refer to Chapter 1 p 25).
Children with fever should be ‘nursed cool’ i.e. dressed lightly. Active measures to reduce the temperature i.e. tepid sponge and fans are not recommended. Avoid shivering.

F For Fluids

- Cease enteral feeding in any infant or child with a reduced level of consciousness and place nil by mouth (NBM).
- Commence IV fluids as per the recommended NSW guidelines (refer to Chapter 6 Table 8 p 128) and appropriate to latest blood chemistry levels.
- Maintain an accurate fluid balance ensuring an infusion pump and burette is used at all times.

G For Glucose

- Ensure a blood glucose level (BGL) is obtained, particularly if the neurological deterioration is unexplained or unexpected.
- If hypoglycaemia is present i.e. BGL less than 3.0 mmol/L or symptomatic (refer to Chapter 1 p 29) call for a Clinical Review and prepare to administer a bolus of 2 mL/kg of 10% Glucose as prescribed.
- Ensure maintenance fluids contain 5 - 10% Glucose.
- Repeat BGL within 5-10 minutes following administration of a glucose bolus.

If BGL is less than 2.0mmol/L call for a Rapid Response and prepare to administer a bolus of 2 mL/kg of 10% Glucose.

ONGOING MANAGEMENT

Any child with neurological deterioration should be escalated to senior members of the local team, and paediatric expertise should be sought. Where appropriate, consideration should be given to consulting with state emergency and/or intensive care services via NETS for advice and possible transfer to a tertiary paediatric centre. Refer to Appendix 1 for information on Interhospital transfer.

NETS hotline telephone number is 1300 36 2500 - Early referral to NETS (or a local retrieval service) is encouraged for clinical support, advice and/or to activate a medical retrieval.
Investigations may include:

- **Blood tests**
  - BGL & blood gas (capillary, venous or arterial)
  - FBC and blood culture - include a C Reactive Protein (CRP) if infection suspected
  - Coagulation studies (especially if cerebral haemorrhage likely)
  - EUC (with particular attention to sodium) and Lactate if available
  - LFTs
  - Plasma Ammonia
  - Calcium
  - Anticonvulsant levels if appropriate

- **Urine test** for culture and drug and metabolic screen

*If the diagnosis remains unclear collect 1-2 mL each of plasma, serum and 10 mL urine to be frozen for potential metabolic studies.*

- **Lumbar puncture** if meningitis is suspected and there are no contra-indications such as:
  - Patient instability i.e. any respiratory or cardiovascular compromise
  - Continuing seizure activity
  - GCS 8 or less
  - Focal neurology with suspicion of space occupying lesion or signs of raised ICP
  - Coagulopathy or increased risks of bleeding

- **A cranial CT scan** should be performed early if the diagnosis is unclear. It may identify intracerebral bleeding, space occupying lesions, cerebral oedema or diffuse brain injury. However, it cannot completely rule out a raised ICP and a normal CT does not exclude subsequent risk of brainstem herniation. The potential risk of transporting and managing a critically ill child in the CT scanner should be considered – always ensure appropriate resuscitation, stabilisation prior to CT, and appropriate observation by experienced staff.

- **MRI** should be considered if a diagnosis is still in doubt after above investigations. ADEM cannot be identified with a CT scan.
SUMMARY OF KEY POINTS

- Actively involve the caregivers in your assessment and ongoing care. Establish a baseline for the child’s normal level of interaction and neurological status.

- The standard Glasgow Coma Scale is unsuitable for children under 5 years. In children less than 5 years use the Child Glasgow Coma Scale.

- For a convulsing child position in the recovery position, commence oxygen, call for help and administer midazolam.

POLICY DIRECTIVES AND CLINICAL RESOURCES

Detailed information on the assessment and management of specific neurological conditions and relevant illnesses can be obtained from the following Clinical Practice Guidelines:

PD2009_065 Infants and Children: Acute Management of Seizures
PD2012_065 Infants and Children: Acute Management with Bacterial Meningitis
PD2011_024 Infants and Children: Acute Management of Head Injury
PD2010_063 Infants and Children: Acute Management of Fever

All Clinical Practice Guidelines can be accessed via the NSW Health Intranet website:

- Go to Quick Links: Child Health Networks
- Go to Links & Resources: NSW Health Paediatric Clinical Practice Guidelines

or via the NSW Health Internet website (for ‘off site’ access):

REFERENCES


The aim of this chapter is to give you the knowledge to be able to:

- Understand the simple pathophysiology and the causes of fluid and electrolyte disturbance.
- Identify, interpret and evaluate the early and late warning signs of fluid and electrolyte disturbance.
- Assess and prioritise immediate and early management of fluid and electrolyte disturbance.
- Escalate concerns to Clinical Review or Rapid Response as appropriate.

*Throughout the chapter ‘child’ refers to infant, child or adolescent unless otherwise stated.*

### Early Warning Signs of Fluid & Electrolyte Disturbances

<table>
<thead>
<tr>
<th>Early Warning Signs</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Sunken eyes, sunken fontanelle or Puffy eyelids, puffy face</td>
<td>Reduced urine output or Increased urine output</td>
</tr>
<tr>
<td>Dry mouth/dry skin, reduced skin turgor or Pitting oedema</td>
<td>Moderate tachycardia or Bounding pulses and hypertension</td>
</tr>
<tr>
<td>Persistent vomiting, watery diarrhoea</td>
<td>Lethargy, headache, irritability</td>
</tr>
</tbody>
</table>

*Table 1*

### Late Warning Signs of Fluid & Electrolyte Disturbances

<table>
<thead>
<tr>
<th>Late Warning Signs</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Significant uncontrolled bleeding or fluid loss</td>
<td>Marked tachycardia, weak peripheral pulses</td>
</tr>
<tr>
<td>No urine output</td>
<td>Cardiac arrhythmia</td>
</tr>
<tr>
<td>Marked tachypnoea or Deep sighing respirations</td>
<td>Drowsiness, hypotonia or confusion</td>
</tr>
</tbody>
</table>

*Table 2*

Concern by any staff or family member irrespective of early or late warning signs should be appropriately escalated.
PATHOPHYSIOLOGY OF FLUID AND ELECTROLYTE DISTURBANCES

ANATOMICAL AND PHYSIOLOGICAL DIFFERENCES BETWEEN CHILDREN AND ADULTS

Total body water content

Neonates and infants have a higher total body water and greater proportion of extracellular fluid which is more easily lost from the body than intracellular fluid, leaving them vulnerable to fluid deficits.

<table>
<thead>
<tr>
<th>Age</th>
<th>Intracellular fluid (mL/kg)</th>
<th>Extracellular fluid (mL/kg)</th>
<th>Total body water (mL/kg)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Neonate</td>
<td>350</td>
<td>450</td>
<td>80</td>
</tr>
<tr>
<td>Infant</td>
<td>400</td>
<td>300</td>
<td>70</td>
</tr>
<tr>
<td>Child</td>
<td>400</td>
<td>250</td>
<td>65</td>
</tr>
<tr>
<td>Adult</td>
<td>400</td>
<td>200</td>
<td>50-60</td>
</tr>
</tbody>
</table>

Higher metabolic rates

Infants and young children have higher metabolic rates that produce larger amounts of water and heat from cellular respiration. It has been estimated that the turnover of fluids and solute can be as much as 3 times that of adults. Any interruption in normal fluid intake or normal kidney function can lead to rapid changes in body fluid composition.

Larger surface to volume ratio

Infants and young children have a higher body surface area-to-volume ratio with proportionally higher insensible losses that is accentuated in disease states (e.g. fever or burns).

Immature kidneys

Kidney function is immature at birth with reduced capacity to reabsorb sodium and water and relative insensitivity to hormonal control. The risk of hyponatraemia is greater as excessive sodium can be lost in the urine. The newborn’s ability to concentrate urine and to conserve sodium improves following birth, particularly over the first 2 months.

Developmental age

Infants and young children are unable to fully communicate their need for fluids, or independently access fluids in response to increased thirst. They are dependent on their caregiver’s ability to offer...
them appropriate intake of food and water and are vulnerable to fluid and electrolyte disturbances for this reason.

**FLUID AND ELECTROLYTE DISTURBANCES**

In states of illness or injury, the normal mechanisms that regulate fluid homeostasis may be disrupted, creating a situation of fluid (and electrolyte) imbalance. Depending upon the underlying condition and the balance of fluid and electrolytes taken in and lost by the body, a state of either fluid deficit or fluid overload can develop. Concurrently, fluid shifts may occur between intracellular and extracellular (includes intravascular, interstitial and body cavity compartments) spaces which can lead to serious clinical consequences with significant implications for management.

**DEHYDRATION**

Dehydration is a loss in *total body water* causing a reduction in both *extracellular* and *intracellular* fluid volumes. Typically, the fluid loss is shared by all fluid compartments and intravascular fluid volume is not compromised until the loss becomes relatively large. If however, dehydration progresses and is severe, intravascular depletion and *hypovolaemic shock* ultimately ensues, resulting in end organ failure and death.

*The only objective measure of acute changes in total body water is body weight and this should be used to determine an accurate fluid deficit wherever possible.*

In the absence of a recently recorded healthy weight, clinical signs of dehydration are used to determine the approximate degree of dehydration present, i.e. mild, moderate or severe, in order to calculate the fluid deficit and appropriate fluid replacement therapy *(refer to Chapter 1 Table 8 p 27).*

It should be remembered that *clinical signs of dehydration can lead to over-estimation of the degree of dehydration.*

As a guide:

Mild dehydration = 2.5% = loss of 2.5 mL /100g body weight *i.e. 25 mL/kg*

Moderate dehydration = 5% = loss of 5 mL /100g body weight *i.e. 50 mL/kg*

Severe dehydration (without shock) = 7.5% = loss of 7.5 mL /100g body weight *i.e. 75 mL/kg*

**Signs of hypovolaemic shock will usually be present at 10% or more dehydration.**
Types of Dehydration

The loss and intake of salt from the body relative to water should also be considered in the assessment and management of fluid and electrolyte abnormalities. Osmolarity describes the concentration of solutes or salts in the blood, and depends mainly on the concentration of sodium, which is usually a good marker of serum osmolarity.

Depending on the relative loss and intake of sodium and water, dehydration can exist as isonatraemic (132 - 145 mmol/L), hyponatraemic (less than 132 mmol/L), or hypernatraemic (greater than 145 mmol/L). Isonatraemic dehydration is the most common (80%). Hypernatraemic and hyponatraemic dehydration each comprise 5-10% of cases and require special consideration.

Isonatraemic dehydration

Isonatraemic dehydration occurs when sodium and water losses are proportional. A common cause in children is gastroenteritis.

Hyponatraemic dehydration

Hyponatraemic dehydration occurs when, compared to normal serum concentrations, the fluid loss contains a greater proportion of sodium than water. The low serum sodium causes water to shift from the intravascular to the extravascular space, exaggerating intravascular volume depletion (and resulting in overestimation of the degree of dehydration).

Rapid or excessive correction of hyponatraemia carries a high risk of fluid shifts out of cells and may cause damage to the central nervous system (central pontine myelinolysis) leading to coma and death. This is more likely to occur if the serum sodium concentration is allowed to rise by any more than a maximum of 10 mmol/L in the first 24 hours during rehydration.

Potassium depletion often accompanies hyponatraemic dehydration, and results from increased loss of potassium in the urine as the kidneys attempt to retain sodium.

Slow correction of hyponatraemia (defined as less than 10 mmol in 24 hours) is necessary to avoid damage to the central nervous system, coma and death.

Hypernatraemic dehydration

Hypernatraemic dehydration occurs when, compared to normal serum concentrations, the fluid loss contains a greater proportion of water than sodium. The high serum sodium causes a relative shift of water from the intracellular space into the intravascular space, and cells become dehydrated and shrink.
To compensate and maintain cellular fluid volume, cells generate osmotically active particles that pull water back into the cell. In this situation, there is a **high risk of cerebral oedema** during rehydration unless the rate of fluid replacement is very slow and the replacement fluid sodium content is adjusted to ensure that the serum sodium concentration returns **slowly** back to normal over 48 to 72 hours.

**FLUID OVERLOAD**

Fluid overload is seen on examination as tissue oedema, as excess fluid is redistributed from the intravascular to the extravascular space. In children this is often first noted as puffiness of the eyelids, face or extremities, as well as in dependent areas of the body where gravity has influenced increased accumulation of extracellular fluid.

In severe fluid overload, oedema may be accompanied by accumulation of fluid in other parts of the body, such as the lungs i.e. pulmonary oedema. Measurement of body weight should be used in the assessment of fluid overload.

Fluid overload most commonly occurs due to over administration of IV fluids (i.e. cause is **iatrogenic**). Children with diabetes or compromised organs, including the heart, kidneys, and liver are most susceptible as their ability to process fluids is impaired, and fluid retention is common.

Fluid overload can result in major electrolyte disturbances, particularly sodium, and can lead to major complications and even death.

**ALTERED URINE OUTPUT**

**OLIGURIA AND ANURIA**

Oliguria defined as less than 1mL/kg/hr urine (or less than 4 wet nappies in 24 hrs or no urination for eight hours in older children) may be a compensatory response to dehydration or hypovolaemia, as the kidney acts to conserve water and maintain intravascular volume, under the influence of several
of the body’s hormones (aldosterone and anti-diuretic hormone). Anuria (no urine output) is never normal and is a result of kidney (renal) injury or failure.

**Acute Renal Failure**

The causes of acute renal failure (ARF) may be described as pre-renal, renal (intrinsic) or post-renal ARF. The presenting feature in all cases of acute renal failure is an altered urine output (usually oliguria, but sometimes polyuria, particularly in cases of chronic renal failure or altered tubular function) together with a rising blood urea nitrogen (BUN) and serum creatinine.

**Pre-renal ARF**

In pre-renal failure the blood pressure and flow to the kidneys is inadequate for them to make normal amounts of urine. This is the most common cause of acute renal failure and may result from any of the causes of shock (*refer to Chapter 4 p 81*). If recognised and treated early, pre-renal failure is reversible, with no long term damage to the kidneys. If the hypoperfused state is allowed to continue, it will lead to permanent damage to the kidneys and intrinsic renal failure.

The most common causes of pre-renal failure include hypovolaemia due to gastrointestinal diseases, and sepsis. Typically in pre-renal ARF the BUN is *disproportionately* raised compared to a mild elevation in serum creatinine.

**Intrinsic ARF**

Intrinsic renal failure results from ischaemic, inflammatory or toxic insults to the kidney that lead to structural and functional damage. A number of drugs are known to cause direct damage to the kidneys. Commonly used nephrotoxic drugs include antibiotics, particularly aminoglycosides (e.g. Gentamicin), but also some penicillins and cephalosporins. Non-steroidal anti-inflammatory drugs (NSAIDs) and diuretics (e.g. Frusemide) may also cause direct toxicity to the kidneys. A urinalysis usually reveals increased protein, sometimes with traces of blood.

Another important cause in childhood is nephritic syndrome (also called post-streptococcal glomerulonephritis), which occurs most commonly secondary to Group A Streptococcus throat infection. Hypertension and oedema typically accompany the findings of blood and protein in the urine in a child with evidence of a recent streptococcal infection (increased anti-streptolysin-O titre (ASOT) in blood). Haemolytic Uraemic Syndrome (HUS) should also be considered in any child with a history of diarrhoea or pneumonia, and with low platelet count.
Post-renal failure

This is the least common form of renal failure but should be considered, particularly in neonates presenting with oliguria. It results from an obstruction to the flow of urine and back-pressure causing damage to the kidneys. The degree of renal failure corresponds directly with the duration and degree of obstruction.

In male infants in the neonatal period, urinary tract obstruction due to posterior urethral valves is the most common cause of post-renal failure. Renal ultrasound should be urgently undertaken in any neonate presenting with ARF in order to exclude obstruction from any cause.

Syndrome of Inappropriate Antidiuretic Hormone (SIADH)

Anti-diuretic hormone (ADH) is secreted from the posterior pituitary gland in response to hypovolaemia or increased serum osmolarity. “Inappropriate” or increased secretion of ADH is known to occur in response to non-osmotic stimuli such as hypoxia, stress, drugs (e.g. Carbamazepine) or potentially in any serious illness. *Increased ADH secretion is often seen in infants with bronchiolitis and other common conditions.*

The effect of increased ADH secretion is greater reabsorption of water in the kidneys, potentially leading to fluid retention and overload and a subsequent fall in serum sodium and osmolarity due to the dilutional effect.

Hence, children admitted with serious illness and requiring IV fluids should routinely have restriction of maintenance IV fluid volumes usually to around two-thirds of normal requirements *once dehydration and hypovolaemia* are excluded, as the risk of fluid overload and hyponatraemia from the action of increased ADH is high.

Restrict normal maintenance IV fluid volumes in sick infants and children without obvious signs of dehydration.

POLYURIA

Polyuria in children may be defined as urine output exceeding 3mL/kg/hr. Unlike primary polydipsia (excessive fluid drinking) which tends to lead to day-time polyuria, pathological causes usually lead to increased urine output both day and night, so night-time wetting in a child with previously dry nights is an important sign. Toddlers and older children may also demonstrate or complain of an increased thirst remember, however, that infants are unable to communicate their needs and are more likely to present with irritability or failure to thrive.
The most common cause of polyuria is *insulin dependent (Type 1) diabetes mellitus (IDDM)*. Poorly controlled or undiagnosed IDDM leads to glycosuria and a glucose-induced osmotic diuresis. Dehydration develops along with ketoacidosis as the body uses fat as an energy source, a condition known as diabetic ketoacidosis (DKA). The clinical picture is one of polyuria, acidosis with tachypnoea, and reduced peripheral perfusion.

*Type 2 diabetes mellitus* is increasingly recognised in obese children, but remains rare compared to adults.

Other pathological causes of polyuria include:

- *Diabetes Insipidus (DI)*, where decreased or absent ADH allows excessive water loss from the kidneys, resulting in severe hypernatraemic dehydration.

- *Cerebral salt wasting syndrome*, usually associated with traumatic brain injury or neurosurgery, is a rare cause of polyuria, and a cause of hyponatraemic dehydration.

- *Intrinsic renal failure*, usually chronic in nature, may lead to inability of the kidneys to concentrate urine resulting in polyuria and excessive sodium and water loss.

- *Adrenal failure (Addison’s disease)* with reduced aldosterone production is a rare cause of polyuria, hypoglycaemia and shock in childhood. In male neonates, a group of congenital disorders of the adrenal gland known collectively as Congenital Adrenal Hyperplasia (CAH) may not be immediately recognisable at birth and typically presents in the first few days of life. Altered responsiveness of the adrenal glands to adrenal stimulating hormones in CAH usually results in severe salt and water imbalance in the first few days of life in an otherwise normal baby boy. Unlike boys, girls are usually diagnosed at birth due to the virilising effects of these stimulating hormones leading to ambiguous genitalia in newborn females.

Hyponatraemia and hyperkalaemia are typically present in intrinsic renal failure, adrenal failure and congenital adrenal hyperplasia.

**ELECTROLYTE DISTURBANCES**

Electrolytes play a critical role in maintaining normal function of the muscles, heart, and nerves. Their functions include:

- Regulation of water distribution
- Regulation of acid-base balance
- Transmission of nerve impulses
- Muscle contraction
- Energy metabolism
- Blood clotting

Any illness or injury that results in altered fluid intake and output can result in or be accompanied by an important electrolyte imbalance. The most common electrolyte disturbances encountered by healthcare practitioners involve sodium and potassium. Other electrolytes to consider include calcium, chlorine, phosphate and magnesium.

Common symptoms of an electrolyte imbalance include weakness, fatigue, faintness, and muscle spasms. Other symptoms can include dry mouth, nausea, vomiting, restlessness and irritability. A severe electrolyte imbalance may cause bradycardia, palpitations, hypotension, confusion, seizures and coma.

**SODIUM** ¹¹

The normal range for serum sodium (Na+) is 135 - 145mmols/L. Sodium regulates the total amount of water in the body and many of the body processes, especially in the brain, nervous system, and muscles.

**Hyponatraemia**

Hyponatraemia is the most common electrolyte disorder encountered in hospitalised patients. *Hospital-acquired hyponatraemia (iatrogenic hyponatraemia) is the most common cause of hyponatraemia in children due to the inappropriate or excessive use of hypotonic fluid.*

Other causes include states of increased total body water as occurs in SIADH *(refer to p 116)*, cardiac failure or renal failure (although this is a late development), loss of sodium via the gastrointestinal tract such as with gastroenteritis, loss of sodium via the kidney from diuretic therapy, or less commonly, cerebral salt-wasting syndrome.

**Hypernatraemia**

Hypernatraemia is uncommon in children, and most often results from dehydration caused from excessive water loss, such as diabetes Insipidus or gastroenteritis. Only rarely does hypernatraemia result from an excess of sodium intake. ¹²

**Symptoms**

The symptoms of hyponatraemia or hypernatraemia are predominantly neurological and severity relates both to the extent of the electrolyte disturbance and the rapidity of onset. ¹³ Symptoms
include nausea and vomiting, headache, and altered mental status such as lethargy, irritability, restlessness and confusion. Seizures, stupor and coma may occur.

### Acute severe hyponatraemia

(defined as less than 125 mmol/L) may result in acute cerebral oedema and a risk of seizures, brain herniation and death.

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### POTASSIUM

Exact values for ranges of serum potassium (K+) are age-dependent, and vary among laboratories but in general, the accepted range is 3.5 mmol/L - 5.0 mmol/L. Normal blood levels of potassium are essential to maintain normal electrical rhythm of the heart and normal muscle function.

#### Hypokalaemia

Hypokalaemia results from excessive loss of potassium from the body in conditions such as Diabetic Ketoacidosis, severe vomiting, diarrhoea, nasogastric losses, and diuretic therapy. It may also occur as a result of potassium shifting from the extracellular to the intracellular space. Such “transcellular” potassium shifts typically occur with acute alkalosis, hypothermia and certain drugs (e.g. insulin, salbutamol).

#### Hyperkalaemia

The most common cause of hyperkalaemia in children are decreased potassium excretion due to renal failure, or increased potassium intake due to inadvertent iatrogenic administration of an excess of potassium in intravenous fluids. Transcellular shift of potassium from the intracellular to the extracellular space may also lead to acute hyperkalaemia which can occur in states of acute acidosis and conditions associated with massive cell death and potassium release (e.g. haemolysis, rhabdomyolysis, tumour lysis syndrome).

#### Symptoms

Hypokalemia can lead to muscle fatigue, cramping, numbness and paralysis. Rarely it may cause respiratory arrest or cardiac arrhythmia.

Hyperkalaemia is a common cause of lethal cardiac arrhythmias. Characteristic early ECG changes that precede ventricular fibrillation and cardiac arrest are peaked or “tented” T-wave, loss of P-wave and widened QRS complex. Cardiac arrhythmias will usually occur when the serum potassium level is more than 6.5 mmol/L and this situation should always be considered a medical emergency.
Hyperkalaemia is a medical emergency and raised serum potassium levels must always be investigated and treated before ventricular fibrillation and cardiac arrest occurs.

**CAUSES OF FLUID AND ELECTROLYTE DISTURBANCES**

**DEHYDRATION**

Dehydration is a common problem in unwell infants and children, occurring as a result of either excess loss of fluid and electrolytes or decreased intake, or more often than not, a combination of both.

Whilst it is true that gastroenteritis is the most common cause of dehydration in infants and children, care should always be taken to consider the possibility of other childhood illnesses which may present with similar symptoms, such as acute appendicitis, intestinal obstruction, gastrooesophageal reflux (GOR), urinary tract infection (UTI) or other serious illness such as sepsis, meningitis or DKA.

**CAUSES OF DEHYDRATION**

<table>
<thead>
<tr>
<th>Common Conditions</th>
<th>Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Gastrointestinal losses</strong></td>
<td></td>
</tr>
<tr>
<td>Diarrhoea and vomiting i.e. gastroenteritis</td>
<td>▪ Dehydration can progress rapidly. Children may lose up to 10-20% of their circulating volume within 1-2 hours. Young infants are most at risk.</td>
</tr>
<tr>
<td></td>
<td>▪ Serum sodium will be normal, high or low depending on the relative loss of water and salt from the body.</td>
</tr>
<tr>
<td>Non bile-stained vomiting:</td>
<td></td>
</tr>
<tr>
<td>– Pyloric stenosis</td>
<td>▪ Vomiting can occur in many conditions (other than gastroenteritis – which is always accompanied with diarrhoea).</td>
</tr>
<tr>
<td>– Acute appendicitis</td>
<td>▪ History and other signs/symptoms can assist with determining cause e.g. projectile vomiting, abdominal pain, fever &gt;39°C, urinary symptoms</td>
</tr>
<tr>
<td>– Severe GOR</td>
<td></td>
</tr>
<tr>
<td>– UTI or other serious illness</td>
<td></td>
</tr>
</tbody>
</table>
### Chapter Six – Sunken eyes or Puffy eyelids

#### Table 4

<table>
<thead>
<tr>
<th>Common Conditions</th>
<th>Comments</th>
</tr>
</thead>
</table>
| Bile-stained vomiting: Intestinal obstruction  
– Volvulus  
– Intussusception  
– Paralytic ileus | ▪ Bile-stained (green) vomiting and abdominal distension are usually present in intestinal obstruction  
▪ Intussusception typically presents with intractable crying and later redcurrant jelly stool  
▪ Bowel obstruction or ileus from any cause may result in fluid maldistribution (as the bowel fills with fluid), hypovolaemia & shock |
| Excess nasogastric losses e.g. post abdominal surgery ileostomy losses | ▪ This typically causes the loss of salt and water resulting in *hyponatraemia* and *dehydration*  
▪ Increased risk of dehydration and electrolyte imbalances exists in children with ileostomies |
| **Excess Urine Output – Polyuria** |  |
| Hyperglycaemia  
Diabetes mellitus | ▪ Osmotic diuresis can quickly result in dehydration  
▪ Measured serum sodium may be falsely lowered by presence of hyperglycaemia |
| Diabetes Insipidus | ▪ Inadequate secretion of ADH as a result of inflammation/injury to the brain (Central DI) or inability of the kidney to respond to circulating ADH (Nephrogenic DI)  
▪ Both types cause excretion of excess dilute urine, resulting in *hyponatraemia* and *hypovolaemia* |
| Cerebral Salt Wasting Syndrome | ▪ Excess loss of sodium in the urine, usually as a result of inflammation/ injury to the brain  
▪ *Hyponatraemia* and *hypovolaemia* accompanying polyuria are characteristic |
| Addison’s Disease or CAH (infants) | ▪ Reduced action of aldosterone may result in increased sodium and water loss in the urine and increased serum potassium level |
| **Increased insensible (skin) losses** |  |
| Excessive heat  
Prolonged fever  
Cystic Fibrosis  
Burns | ▪ The skin is responsible for almost 25% of normal daily fluid loss in infants and young children  
▪ Any alteration in body or ambient temperature or to skin integrity can lead to excess sodium and water loss |
| **Reduced oral intake** |  |
| Acute illness  
Primary herpetic stomatitis  
Iatrogenic | ▪ Any illness or injury can lead to reduced oral intake  
▪ Primary Herpes simplex type 1 infection other causes of stomatitis in young children may result in widespread and painful ulceration of the oral mucosa and reduced oral intake  
▪ Extended periods of nil by mouth due to surgery/procedures may cause dehydration |
FLUID OVERLOAD

The most common cause of fluid overload is *over administration of intravenous fluids* i.e. iatrogenic. The risk of fluid overload is increased in children with pre-existing respiratory or cardiac disease, and in any child with an acute illness.

### CAUSES OF FLUID OVERLOAD

<table>
<thead>
<tr>
<th>Common Conditions</th>
<th>Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Reduced Urine Output - Oliguria</strong></td>
<td></td>
</tr>
</tbody>
</table>
| Acute Renal Failure (ARF): | ▪ Prerenal ARF is commonest and typically results in a disproportionate increase in BUN compared to serum creatinine  
▪ Intrinsic ARF may occur from damage to the kidney from toxins including drugs. Blood and protein are detected on urinalysis. Blood pressure should be measured  
▪ Postrenal ARF should be excluded in any neonate, particularly boys, presenting with ARF |
| — Prerenal  
— Renal (Intrinsic)  
— Postrenal | |
| Syndrome of Inappropriate Antidiuretic Hormone (SIADH) | ▪ Increased ADH production is common in sick or stressed infants and children resulting in fluid overload unless IV therapy is adjusted and closely monitored |
| Maldistribution of fluid and “third spacing” | |
| Cardiac failure  
Chronic inflammation  
Nephrotic syndrome  
Liver failure | ▪ Any persistent or severe infection, injury or disease state can release inflammatory mediators which cause increased capillary leakage leading to tissue oedema  
▪ Urine in children with nephrotic syndrome is typically foamy and contains large amounts of protein on urinalysis  
▪ Liver failure is typically associated with loss of salt, water and bicarbonate into the peritoneal space (ascites) |
| **Increased intake** | |
| Iatrogenic | ▪ Inappropriate IV fluid therapy is a common and potentially fatal cause of hyponatraemia and fluid overload |

*Table 5*

### RECOGNITION OF FLUID DISTURBANCES

A careful history from the caregiver is as important in the assessment of clinical dehydration or fluid overload as the actual examination itself. Information needs to be obtained on the type and amount of fluid intake, urinary and bowel losses including any unusual features of either, and the presence of
any additional symptoms, such as a cough, fever, rash, sore throat, or abdominal pain, as this may assist in clarifying a cause.

**SUMMARY OF FLUID STATUS ASSESSMENT**

<table>
<thead>
<tr>
<th>Assessment</th>
<th>Action</th>
<th>Warning signs</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>First impression</strong></td>
<td>Note general appearance and behaviour</td>
<td>Pale and lethargic, disinterested or unresponsive to environment</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Irritable</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Reduced level of consciousness</td>
</tr>
<tr>
<td><strong>Weight</strong></td>
<td>Record weight wherever possible</td>
<td>Weight loss</td>
</tr>
<tr>
<td></td>
<td>Remove clothes and nappies in infants</td>
<td>Weight gain</td>
</tr>
<tr>
<td><strong>Clinical signs</strong></td>
<td>Assess degree of dehydration using Table 7 Chapter 1 p 27</td>
<td>Dry tongue/lips</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Sunken eyes/sunken fontanelle or puffiness</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Poor perfusion</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Reduced skin turgor</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Tachypnoea/Tachycardia</td>
</tr>
<tr>
<td><strong>Fluid balance</strong></td>
<td>Review fluid balance chart</td>
<td>Reduced fluid intake</td>
</tr>
<tr>
<td></td>
<td>Calculate input vs. output</td>
<td>Reduced urine output</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Polyuria</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Negative or positive fluid balance</td>
</tr>
</tbody>
</table>

*Table 6*

In the presence of *hypernatraemic* dehydration, high serum sodium masks some of the clinical signs of dehydration i.e. sunken fontanelle, sunken eyes, reduced skin turgor.

**URINALYSIS**

A urinalysis is a safe, non-invasive, simple study of urine, providing vital objective information about the patient’s internal functioning. It is important to know what abnormal findings indicate to ensure appropriate management.

Note general appearance of urine:

- Colour and smell may be altered by medications, foods or disease.
- Dark amber may indicate concentrated urine from dehydration, or the presence of blood or urobinogen from jaundice.
- Frothy urine may indicate the presence of glucose or protein.
- Turbid or cloudy urine may result from infection, the presence of blood cells, bacteria or yeast (e.g. Candida).
Protein
Small or moderate amounts usually indicates a UTI or glomerulonephritis. A large amount is typical of Nephrotic syndrome. Leukocytes, Blood and Nitrite may also indicate a UTI.

Specific Gravity
The specific gravity (SG) is a measure of the concentration of dissolved solutes and reflects the effectiveness of the renal tubules to concentrate urine when the body needs to conserve fluid. If no solutes were present the SG would be 1,000, the same as pure water. The SG of urine is around 1,010 but will vary.

Decreased SG indicates dilute urine and causes include:
- Excessive fluid intake (oral or IV fluids)
- Renal failure or acute glomerulonephritis or pyelonephritis
- Diabetes Insipidus

Increased SG indicates concentrated urine and causes include:
- Dehydration
- Prerenal ARF
- Syndrome of Inappropriate Antidiuretic Hormone (SIADH)
- Situations with a high blood solute concentration, e.g. glucose (diabetes or IV glucose) or protein.

Ketones
Not normally found in the urine, ketones are produced during fat metabolism. In children, their presence usually indicates diabetes mellitus or starvation.

Glucose
Not normally found in the urine, glucose usually indicates high blood glucose as occurs in diabetes mellitus or hyperglycaemia from any other cause (e.g. steroids, large glucose load).

Helpful tips for urine collection for urinalysis in infants:
- Insert some cotton wool balls into the nappy. When soaked use a 2mL syringe (without a needle) to draw back urine. This sample is for use with urinalysis only and is not suitable for microbiology testing.
- A urine bag carefully applied to the perineum of an infant is an alternative method and is useful for collecting larger amounts of urine.

NOTE: Specimens sent for culture must be obtained by clean catch, in-out catheter or suprapubic aspiration.
INITIAL MANAGEMENT OF FLUID AND ELECTROLYTE DISTURBANCE

Successful treatment of fluid and electrolyte disturbances requires identification and appropriate management of the underlying disease state. Early assessment of the level of hydration or fluid overload and recognition of existing electrolyte abnormalities is essential.

Caregivers should be encouraged to contribute to observations on their child’s general appearance compared to normal, and where possible, should be given responsibility to help document fluid intake and output with appropriate support from nursing staff.

Always assess and prioritise management using the ABCDEFG algorithm

A Start with the Airway

- If the conscious level is reduced due to severe fluid or electrolyte disturbances and the child is having difficulty maintaining their own airway, use simple airway opening manoeuvres and call for a Rapid Response (refer to Chapter 1 p 8).

B For Breathing

- Commence appropriate oxygen therapy, i.e. nasal prongs, simple face mask, or non-rebreather mask if acute hypoxaemia is present (SpO₂ of less than 95%) and/or increased respiratory distress.


- Auscultate the chest for signs of pulmonary oedema i.e. inspiratory crepes or crackles heard at bases (posteriorly), or for signs of pleural effusions i.e. reduced or absent breath sounds, particularly at bases.

C For Circulation

- Document and monitor trends in heart rate, blood pressure, capillary refill and note volume of peripheral and central pulses.

- If there are obvious signs of hypovolaemic shock or hypotension call for a Rapid Response and administer a rapid IV bolus of 20 mL/kg of 0.9% Sodium Chloride and reassess.
- Attach an ECG monitor and monitor cardiac rhythm. Look for electrocardiograph signs of hyperkalaemia and if suspected, perform a 12 lead ECG and send blood for an urgent serum electrolytes level. This may be immediately available on appropriately calibrated point of care testing devices and the result should always be acted upon prior to the availability of “formal” laboratory results.

- Initial management of severe or symptomatic hyperkalaemia (more than 6.5 mmol/L):
  1. Discontinue any potassium-containing fluids
  2. Commence salbutamol nebuliser (2.5 – 5 mg)
  3. Send a repeat blood sample and take venous blood gas

If severe or symptomatic hyperkalaemia is detected call for a Rapid Response whilst preparing for a salbutamol nebuliser (2.5–5 mg).

D For Disability

- Maintain a continual assessment of the child’s level of consciousness (alertness and arousal) using the AVPU scale.

- If decreased level of consciousness, assess for signs of cerebral oedema and raised intracranial pressure (refer to Chapter 5 p 100). If cerebral oedema is suspected, early intubation is indicated, however, this should not delay administration of more urgent therapy, e.g. osmotherapy.

- Initial management of severe or symptomatic hyponatraemia:
  1. Administer 3 mL/kg hypertonic 3% saline via peripheral or central venous access
  2. Send repeat blood sample to measure serum sodium and observe closely for changes in neurological status
  3. Review intravenous fluids for appropriate sodium content

If there is any altered mental status or seizures check serum sodium, calcium and glucose level urgently.

E For Exposure

- Document axillary temperature.
- Look for evidence of muscle spasm or prolonged twitching and check blood calcium level if present.
- Assess abdomen for distension, discomfort and examine the stool for blood and mucous or jelly.

**F For Fluids**

**IV MAINTENANCE FLUID THERAPY**

- IV maintenance fluid therapy should be adjusted in all unwell children *who are not dehydrated*. Consider reducing to 2/3 full maintenance.

**NORMAL DAILY FLUID REQUIREMENTS FOR CHILDREN**

<table>
<thead>
<tr>
<th>Body Weight</th>
<th>Fluid requirements/day</th>
</tr>
</thead>
<tbody>
<tr>
<td>First 10kg</td>
<td>100 mL/kg</td>
</tr>
<tr>
<td>Second 10kg</td>
<td>1000 mL + 50 mL/kg for each kg over 10kgs</td>
</tr>
<tr>
<td>More than 20kg</td>
<td>1500 mL + 20 mL/kg for each kg over 20kgs</td>
</tr>
</tbody>
</table>

*Table 7*

- Any child on intravenous fluids should have electrolytes and glucose measured on commencement of therapy and a minimum of daily monitoring thereafter. *Assessment of sodium levels and clinical examination are critical to determining safe hydration therapy.*
- For more unwell children, recheck the electrolytes and glucose every 6 - 8 hours and then according to results and the clinical situation (but at least daily).

The detection of any severe electrolyte disturbance should trigger a Clinical Review or Rapid Response, depending on the nature and severity of abnormality and presence of symptoms.

- As per NSW Health policy directive PD2010_034 3.3.10 – An infusion pump and paediatric infusion sets with an inline burette must be used for all children requiring intravenous therapy.
- To minimise risk and promote safety, it is strongly recommended pre-prepared bags of fluids for maintenance, rehydration and neonates should be used with the correct concentrations of glucose and potassium.
- Table 8 below provides a summary of the NSW Working Group recommendations for Paediatric IV fluid content.
### RECOMMENDED NSW PAEDIATRIC IV FLUID CONTENT FOR MAINTENANCE FLUIDS

<table>
<thead>
<tr>
<th>Neonates</th>
</tr>
</thead>
<tbody>
<tr>
<td>0.225% Sodium Chloride + 10% Glucose (+/- 20 mmol/L Potassium Chloride*)</td>
</tr>
<tr>
<td>0.45% Sodium Chloride + 10% Glucose (+/- 20 mmol/L Potassium Chloride*)</td>
</tr>
<tr>
<td>Where there is</td>
</tr>
<tr>
<td>i. pre-existing hyponatraemia (sodium less than reference range)</td>
</tr>
<tr>
<td>ii. increased risk of hyponatraemia - such as sodium losses (e.g. from the gut) or high risk of non-osmotic ADH secretion (e.g. respiratory illnesses or meningitis)</td>
</tr>
<tr>
<td>* once serum potassium known and urine output established</td>
</tr>
<tr>
<td>Use 500mL bags or less</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Infants and children</th>
</tr>
</thead>
<tbody>
<tr>
<td>0.45% Sodium Chloride + 5% Glucose (+/- 20 mmol/L Potassium Chloride*)</td>
</tr>
<tr>
<td>0.9% Sodium Chloride + 5% Glucose (+/- 20 mmol/L Potassium Chloride*)</td>
</tr>
<tr>
<td>Where there is</td>
</tr>
<tr>
<td>i. pre-existing hyponatraemia (sodium less than reference range)</td>
</tr>
<tr>
<td>ii. increased risk of hyponatraemia - such as sodium losses (e.g. from the gut) or high risk of non-osmotic ADH secretion (e.g. respiratory illnesses or meningitis)</td>
</tr>
<tr>
<td>* once serum potassium known and urine output established</td>
</tr>
</tbody>
</table>

*Table 8*

Routine use of hypotonic fluids has been associated with iatrogenic hyponatraemia and subsequent neurological complications and death.

**DO NOT USE ANY FLUID CONTAINING LESS SODIUM THAN 0.45% Sodium Chloride in infants and children even in situation of severe hypernatraemia.**

### IV REHYDRATION FLUID THERAPY 21

- Any IV fluid therapy carries risks and the enteral route (i.e. oral or nasogastric) should be used wherever possible. This will likely be possible for mild-moderate dehydration.

For detailed information on enteral rehydration refer to Clinical Practice Guideline *PD2010_009 Infants and Children: Acute Management of Gastroenteritis*
For moderate to severe dehydration requiring IV replacement, the aim of rehydration therapy is to replace fluid loss whilst maintaining normal daily requirements i.e.

Maintenance + Fluid deficit (+/- ongoing losses)

- If serum sodium is within normal range (135 - 145 mmol/L) initial rehydration can be calculated to commence over 24 hours (unless Diabetic Ketoacidosis when the ‘corrected sodium’ should be used and rehydration conducted over 48 hours).

- If hypernatraemia is present (sodium more than 145 mmol/L) initial rehydration should be calculated to commence over 48 - 72 hours and care should be taken to ensure that the sodium returns to normal range slowly (maximum change in serum Sodium 10 mmol/L or less in 24 hrs).

**Rapid rehydration (e.g. 4 hours), even with a normal serum sodium, is not routinely recommended and should only be undertaken in the ED under close supervision.**

- Rehydration is calculated using the following formula:

\[
\text{Weight (kg) } \times \% \text{ dehydration } \times 10 = \text{mL deficit}
\]

**RECOMMENDED NSW PAEDIATRIC IV FLUID CONTENT FOR REPLACEMENT FLUIDS**

<table>
<thead>
<tr>
<th>Neonates</th>
</tr>
</thead>
<tbody>
<tr>
<td>0.45% Sodium Chloride + 10% Glucose (+/- 20 mmol/L Potassium Chloride)</td>
</tr>
<tr>
<td>For seriously ill dehydrated neonates:</td>
</tr>
<tr>
<td>0.9% Sodium Chloride + 10% Glucose (+/- 20 mmol/L Potassium Chloride)</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Infants and children</th>
</tr>
</thead>
<tbody>
<tr>
<td>0.9% Sodium Chloride + 5% Glucose (+/- 20 mmol/L Potassium Chloride)</td>
</tr>
</tbody>
</table>

*Table 9*

**Example calculation:**

Maintenance and 5% dehydration fluids in a child weighing 12 kg (Sodium = 140 mmol/L):

- Maintenance fluids = 1000 mL + (2 x 50 mL) = 1100 mL (*Table 7*)
- = 1100 mL/24 hr of 0.45% Sodium Chloride + 5% Glucose (*Table 8*)
- +

  Rehydration fluids = 12 kg x 5 % x 10
  = 600 mL/24 hr of 0.9% Sodium Chloride + 5% Glucose (*Table 9*)
Assess the clinical response to fluid rehydration therapy allows for *recalculation of volume and type of fluid* used and should be used as an opportunity to consider early feeding.

Clinical signs of dehydration overestimate the volume of rehydration therapy required. Careful reassessment after 6 - 8 hours is essential.

Reassessment should include:

- Changes in weight
- Clinical signs of dehydration or oedema
- Urine output and fluid balance
- Ongoing losses
- Serum sodium and blood glucose

**ONGOING LOSSES**

- Ongoing losses are not always taken into account; however, if losses are large, they may need to be accurately measured and replaced.
- DO NOT replace excess urinary losses in Diabetic Ketoacidosis.
- Consider replacement of:
  - All drain losses
  - Excess urine loss (replace volumes above 3 mL/kg/hr)
  - Large watery diarrhoea (weigh nappy)

- Replacement can be based on each previous hour or calculated over an agreed period of time depending upon the situation and extent of fluid loss (e.g. a 100 mL loss over previous 4 hours becomes replacement of 25 mL/hr over the next 4 hours).
- Use 0.9% Sodium Chloride to replace ongoing losses unless the composition of the fluid being lost is accurately known.

*Specialist Consultation* is required in **all cases prior** to replacement of ongoing losses and in **all cases** of infants less than 3 months.
For Glucose

- If hypoglycaemia is present i.e. BGL less than 3.0mmol/L or symptomatic (refer to Chapter 1 p 29) call for a Clinical Review and prepare to administer a bolus of 2 mL/kg of 10% Glucose as prescribed.
- Ensure maintenance fluids contain 5 - 10% Glucose.
- Repeat BGL within 5-10 minutes following administration of a glucose bolus.

If BGL is less than 2.0mmol/L call for a Rapid Response and prepare to administer a bolus of 2 mL/kg of 10% Glucose.

ONGOING MANAGEMENT

Any child with severe fluid and electrolyte disturbances should be escalated to senior members of the local team, and paediatric expertise should be sought. Where appropriate, consideration should be given to consulting with state emergency and/or intensive care services via NETS for advice and possible transfer to a tertiary paediatric centre. Refer to Appendix 1 for information on Interhospital transfer.
NETS hotline telephone number is 1300 36 2500 - early referral to NETS (or a local retrieval service) is encouraged for clinical support, advice and/or to activate a medical retrieval.

SUMMARY OF KEY POINTS

- Do not use fluids with sodium content below 0.45% saline in children (excluding neonates).
- Restrict IV fluids to ⅔ maintenance in the absence of dehydration in any unwell infant or child.
- Always consider the serum sodium level when calculating, prescribing and administering fluid replacement. Always, rehydrate SLOWLY in presence of hypernatraemia.
- Never forget to check blood glucose level and to include 5 – 10% Glucose in maintenance fluids in infants and young children.
- Always escalate significant electrolyte abnormalities to senior medical staff.

POLICY DIRECTIVES

Further information on the assessment and management of fluid and electrolyte disturbances and associated conditions can be obtained from the following Clinical Practice Guidelines:

PD2010_009  Infants and Children: Acute Management of Gastroenteritis
PD2005_384  Infants and Children: Acute Management of Acute Abdominal Pain
PD2010_063  Infants and Children: Acute Management of Fever

All Clinical Practice Guidelines can be accessed via the NSW Health Intranet website:

- Go to Quick Links: Child Health Networks
- Go to Links & Resources: NSW Health Paediatric Clinical Practice Guidelines

or via the NSW Health Internet website (for ‘off site’ access):

REFERENCES


Chapter Six – Sunken eyes or Puffy eyelids


APPENDIX 1  INTER-HOSPITAL TRANSFERS

JOANNE LEAVER

AIM

This chapter summarises the key points for the safe inter-hospital transfer of paediatric patients and appropriate involvement of NETS retrieval service.

ROLE DELINEATION OF HEALTH SERVICES

The role delineation level of a service describes the complexity of clinical activity planned to be undertaken by that service. For individual cases, the actual complexity is significantly affected by the presence of medical, nursing and other health care personnel who hold qualifications compatible with the defined level of service.

Levels range from Level 1 (the lowest complexity level of care) to Level 6:

**Level 1** – No planned inpatient paediatric medical service or designated beds. Primary emergency care and stabilisation prior to transfer to a higher level service.

**Level 2** – Designated paediatric inpatient in rural and remote areas for minor medical conditions or convalescence following transfer from higher level unit. Accredited medical practitioner on call and consultative links to paediatrician.

**Level 3** – As per Level 2 + designated paediatric ward/area. Cares for common medical conditions. 24 hour medical officer on site or within 10 mins.

**Level 4** - As per Level 3 + designated Director of Paediatric Medical Services & integrated hospital inpatient unit, family and child health services and community health services. Specialist Paediatrician on call 24 hours

**Level 5** – As per Level 4 + specialised paediatric inpatient unit. Paediatric Registrar on site 24 hours.

**Level 6** – As per Level 5 + most paediatric medical and surgical sub-specialities. Clinical and diagnostic services provided by Paediatric Specialists. Subspecialty consultant on call 24 hours and designated subspecialty registrars.
DETERMINING THE NEED FOR TRANSFER

Inter-hospital transfer of a child is indicated in the following circumstances:

- The child presents in a hospital without inpatient service for children and needs to transfer for inpatient care.
- The child requires a level or type of treatment beyond the capacity of the presenting hospital.

Determining the need for transfer is a joint responsibility between the transferring and receiving hospitals (and NETS as required). The timing and method of transport used should be integral to any treatment discussion and should take into account:

- Clinical needs of the child or infant; including severity, urgency and the potential for deterioration during transport
- Distance between the referring and the destination facilities and the effective time required for a higher level of care to be provided; either by the clinician escort or a retrieval team.
- Appropriate transport (availability, weather considerations etc)
- An appropriate team
- Cost

THE CONSULTATION PROCESS

It is the responsibility of the most senior attending Medical Officer or delegate to assess and determine the need for transfer of a child to a higher level of care, in consultation with the local or network Paediatrician on-call and a Paediatrician and/or Emergency Department physician at the receiving hospital. Where there is no onsite Medical Officer, the most senior nursing staff member should determine and arrange transfer in consultation with a designated covering GP, regional/on call Paediatrician and an appropriate Medical Officer or Paediatric Specialist at a higher level facility. Staff should refer to their local LHD protocol regarding escalation and/or requirements for Medical Officers to attend the patient for assessment.

The senior Medical Officer should:

- Identify children and adolescents who are at risk of deterioration and/or have complex health conditions.
- Identify the level of care required and the most appropriate destination hospital.
Consult with relevant clinicians at the destination hospital and make arrangements for the appropriate timing of transfer [ensuring that consultation occurs with an appropriately senior clinician].

Have complete records available about history, assessment, results and progress to accompany the child.

Involve NETS as part of the clinical consultation in the case of the seriously ill child or infant and identify the most appropriate transport plan, including level of escort (ambulance, regional retrieval, NETS retrieval), degree of urgency and appropriate vehicle type.

Agree on a desirable time-frame for the patient to leave the referring hospital (departure goal) and a plan for any changes if the patient's condition changes pre transfer.

Book the transfer with the ambulance service or patient transport service; indicating a desired time-frame for the ambulance to commence the transfer and/or to be at the destination hospital.

Document the agreed treatment plan required whilst awaiting transfer, and communicate this to nursing staff involved in the child’s care.

Inform the parent/carer and obtain appropriate consent. Review the patient prior to departure; particularly if a significant time has elapsed.

At the time of transfer, document any treatment not commenced or incomplete with respect to the agreed treatment plan [in consultation with a clinician at the receiving hospital].

CONSULTATION WITH THE ON CALL SPECIALIST PAEDIATRICIAN

In accordance with the LHD established networking arrangements, consultation with an on call specialist paediatrician and/or appropriate other specialist should occur if the infant or child has one or more of the following:

- is unstable.
- has no definitive diagnosis.
- has no clear signs of clinical improvement following initial treatment.
- Non-accidental injury is suspected.
- has significant co-morbidity.
CONSULTATION WITH NETS

NETS (Newborn & paediatric Emergency Transport Service) is the state-wide emergency retrieval service for infants and children in NSW and the ACT. Conditions requiring consultation with NETS regarding management and/or transfer include:

Airway

- All intubated patients
- Actual or threatened airway obstruction

Breathing

- Apnoea
- Severe respiratory distress persisting beyond 4 hours
- Cyanosis, despite oxygen therapy
- Oxygen requirement more than 40% to maintain SaO₂ greater than 95%

Circulation

- Shock
- Significant blood loss
- Heart failure or arrhythmia

Disability

- Intractable Seizures
- Severe Burns risk of airway compromise, partial or full thickness burns greater than 10% body surface burnt; special areas burned :genital region ; palms of hands, soles of feet or joints involved – Refer to PD2008_12 Burns Transfer Guidelines
- Major Trauma
- Spinal Injury

In a critical emergency, notification/consultation can occur prior to full patient assessment and investigation.

Calls to NETS usually lead to simultaneous conferencing of appropriate specialists (e.g. NETS consultant, paediatric emergency physicians, intensivists, neurosurgeons, paediatric surgeons) as required in order to provide clinical consultation and management advice. Important and available management interventions should be agreed and instituted by local services prior to arrival of the NETS team.
NETS services include:

- Clinical advice from a paediatric retrieval consultant
- A “one phone call” referral which uses conference call facilities
- Mobilisation of an appropriate retrieval team or ambulance escort
- Liaison with interstate neonatal and paediatric emergency transport services

Any significant change in the condition of a patient should result in further consultation with NETS, even after retrieval has been arranged, to discuss any required modification in management strategy. Such calls are normally ‘conferenced’ with the accepting hospital clinician and the NETS team so that all stakeholders are informed. It can facilitate a change in estimated time of arrival if the child is deteriorating or a reallocation of resources if the child’s condition improves.

**TYPES OF TRANSFERS**

Inter-hospital transfers should be considered either Non-Urgent/Non-Emergency or Urgent/Emergency

**Non-Urgent Transfers**

Non-emergency inter-facility transfer services are used for transporting admitted patients between health facilities. *Transport for Health* policy provides the framework for this mode of transport. All LHDs have established a Health Transport Unit to assist in coordinating non-urgent health related transport, either via non-emergency transport vehicle or Ambulance Service of NSW Ambulance. Referring clinical staff should be familiar with the local procedures for organising non-urgent patient transport through the Health Transport Unit.

Non-urgent health transport is indicated if a child is stable but requires care en route, for example:

- Intravenous therapy, intravenous medication administration or nebulised therapy
- Oxygen therapy
- Patient Controlled Analgesia
- Suctioning
- Alternative transport options (eg by family) are inappropriate.

Non-urgent health transport may or may not require an escort.
URGENT/EMERGENCY TRANSFERS

Urgent/emergency transfer applies to children and adolescents:

- Whose condition is critical, serious or unstable
- Who are at risk of clinical condition deteriorating during before or transport

Retrievals

Medical retrieval is the process of transferring critically or potentially critically ill patients using a team with the necessary skills and equipment to provide critical care during all stages of the transfer process. NETS should be consulted about all children with a triage category of 1 and 2 and all children with a triage category of 3 who are not improving. Whilst awaiting transfer, the child’s condition should be continually monitored and re-evaluated by the local clinicians.

Transfers (without Retrieval)

If retrieval is not indicated, but urgent transfer is still required, this can be provided by Ambulance Service of NSW, using appropriate road or air transport. Children and adolescents may be transported by Ambulance if their condition is serious but stable or has been sufficiently stabilised by staff at the referring facility. Urgent transfers require a medical, nursing or paramedic escort with experience and skill in paediatric resuscitation and airway management. Ambulance officers are graded according to skills and procedural competencies.

Medical and nursing staff should consult with Ambulance officers about transfer decisions and clarify the responsibilities of key staff during the transfer.

Whenever possible and appropriate, a parent/guardian should be offered the option of travelling with their infant/child to the destination hospital.

If uncertain about the type of escort required discuss with NETS.

Transport of a patient by referring hospital staff should not be undertaken if there is a substantial risk of en route deterioration. In rare circumstances, after consultation with NETS and the receiving unit, it may be deemed in the patient’s best interest.
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POLICY DIRECTIVES AND CLINICAL RESOURCES

The following policies provide detailed information that will assist in the decision making process and the safe and appropriate inter-hospital transfer of an infant or child and should be utilised in conjunction with local policies and procedures.

**PD2010_030 CRITICAL CARE TERTIARY REFERRAL NETWORKS (PAEDIATRICS)**

**Summary**
This Policy Directive relates to critically ill children requiring inter-hospital transfer.

**Author Branch:** Statewide and Rural Health Services and Capital Planning

**PD2010_069 CRITICAL CARE TERTIARY REFERRAL NETWORKS (PERINATAL)**

**Summary**
This Policy Directive relates to critically ill neonates and women with high risk pregnancies that require inter-hospital transfer, and should be read in conjunction with complement Policy Directives PD2010_021 Critical Care Tertiary Referral Networks & Transfer of Care (Adult) and PD2010_030 NSW Tertiary Referral Network (Paediatrics).

**Author Branch:** Statewide and Rural Health Services and Capital Planning

**PD2010_031 CHILDREN AND ADOLESCENTS - INTER-FACILITY TRANSFERS**

**Summary**
This Policy Directive provides a framework to facilitate the safe and timely transfer of children and adolescents whose medical condition requires care at a different level from that of the presenting hospital.

**Author Branch:** Statewide and Rural Health Services and Capital Planning

**PD2010_032 CHILDREN AND ADOLESCENTS - ADMISSION TO SERVICES DESIGNATED LEVEL 1-3 PAEDIATRIC MEDICINE & SURGERY**

**Summary**
This Policy Directive provides information and guidance regarding appropriate assessment and admission of presenting patients for paediatric medicine and paediatric surgery in NSW Health facilities with paediatric services designated as levels 1-3 in line within the Guide to the Role Delineation of Health Services, NSW Department of Health, third edition, 2002.
**PD2010_033 CHILDREN AND ADOLESCENTS - SAFETY AND SECURITY IN NSW ACUTE HEALTH FACILITIES**

**Summary**
This Policy Directive provides direction to assist the development of local policies/procedures by LHD to address the safety and security of children and adolescents whilst in NSW acute health facilities or during inter-facility transfers.

**Author Branch:** Statewide and Rural Health Services and Capital Planning

**PD2010_034 CHILDREN AND ADOLESCENTS - GUIDELINES FOR CARE IN ACUTE CARE SETTINGS**

**Summary**
This Policy Directive provides guidelines for the care of children and adolescents in NSW Health acute care facilities.

**Author Branch:** Statewide and Rural Health Services and Capital Planning

**PD2006_068 TRANSPORT FOR HEALTH**

**Summary**
This Policy Directive integrates all non-emergency health related transport service provision throughout NSW into one program. It is aimed at supporting LHD’s to be more strategic in identifying, consolidating and integrating a full range of transport services and resources that can be made available to patients.

**Author Branch:** Primary Health and Community Partnerships

**GL2008_12 BURNS TRANSFER GUIDELINES - NSW SEVERE BURN INJURY SERVICE - 2ND EDITION**

**Summary**
Details emergency treatment and management for patients suffering severe burn injury. This document provides the necessary information for the effective and efficient transfer of burn patients in NSW to specialised burn units.

**Author Branch:** Statewide Services Development
GUIDE TO THE ROLE DELINEATION OF HEALTH SERVICES THIRD EDITION 2002

Summary
This document and the Rural Companion Guide (First edition, 2004), are the tools developed by NSW Health for health service planning, and describe the requirements for designated service levels.

Author Branch: Statewide and Rural Health Services and Capital Planning

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ROLE DELINEATION LEVELS OF EMERGENCY MEDICINE: CONSISTENT WITH THE GUIDE TO THE ROLE DELINEATION OF HEALTH SERVICES THIRD EDITION 2002

Summary
This document provides the current designated role delineation levels for a number of Emergency Services in NSW Public Hospitals as described by the Guide to the Role Delineation of Health Services Third Edition 2002.

Author Branch: Statewide and Rural Health Services and Capital Planning

INTRAOSSEOUS (IO) ACCESS (MANUAL IO NEEDLE OR EZ-IO DRILL SYSTEM) \(^{19, 20}\)

- IO access can be used in any age
- IO access is as a form of “central” access and can be inserted quickly, even in the most poorly perfused patients.
- ANY drug or fluid commonly used in resuscitation and management of shock may be administered via the IO route.
- Most blood tests from bone marrow aspirates are reliable indicators of serum values including glucose, drug levels, haemoglobin, cross match, blood culture and acid-base status
- Contraindications to IO access include:
  - Fracture or infection at the proposed site
  - Previous placement in the same leg/site due to consequent extravasation into soft tissue compartments through the previous puncture site

TYPES OF IO NEEDLES

Manual or automatic IO needles can be used and are equally as efficient.

**Figure 1: Manual IO Needle**  
**Figure 2: EZ-IO Drill and needles**

INSERTION

- The insertion site of choice in children is the proximal tibia as it has a broad flat surface. Alternative sites include the distal tibia and distal femur, however, only a SINGLE attempt per long-bone is recommended.
The EZ-IO system with 15 gauge x 15mm long needle is approved for use between 3 - 39kg. 15 gauge, x 25 mm long needles for patients weighing 40 kg and greater, and longer needles are available for use in obese patients.

Once the IO needle has been inserted, bone marrow should be aspirated and used to check blood glucose level as well as for bone marrow culture and for cross-match. Attach an extension tube and three way tap and manually flush with 0.9% saline.

**Secure the extension line with tape – the IO needle does not require securing. Immobilise the limb with splint to prevent dislodgement of needle.** Frequent site checks for signs of phlebitis or extravasation must be attended and documented.

**REMOVAL**

- The IO needle is usually removed when IV access is established, ideally within 6-12 hours. **Do not leave IO needles in for longer than 24hrs**
- To remove IO needle:
  - Stabilise patient’s extremity
  - Connect sterile Luer lock syringe to IO needle
  - Rotate IO needle clockwise whilst pulling straight back
  - When needle has been removed, immediately place in appropriate biohazard container.

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