Neonatal Emergency Resuscitation and Stabilisation
Contents

PART I: NEONATES
The Unique Neonate 3
Life on the inside 3
The birthing process 12
Making the Switch 13
The Premature Infant 18
What places the preterm infant at risk? 19

PART II: IMMEDIATE RESUSCITATION OF THE NEW-BORN
Assessment and Stimulation 21
Assessing the neonate in the first few moments 21
Oxygen and Positive Pressure Ventilation 44
Using Positive Pressure 46
Chest Compressions 52
Airway management 58
IV access, medication and fluid administration 75
Medications for the resuscitation of the neonate 80

PART III: NEONATAL SPECIFIC SITUATIONS
The infant with cyanosis/respiratory distress 89
The Vomiting Infant 112
Ethics and Difficult Decisions 116
Team-Work and the Human Factor 126

Part 4: SKILLS 129
The Unique Neonate

Life on the inside

Maternal physiology of importance

The 9 months a foetus spends “on the inside” sets the tone, not only the first few months of life in the world, but also for the rest of the child’s life on earth.

There are several physiological and anatomical changes that occur during pregnancy to allow for the infant’s growth to be supported. A summary of these changes can be found in the resource below:

Maternal Changes during pregnancy
Factors in the maternal history that place the foetus/neonate at higher risk

The factors below are all risks that may result in increased likelihood of resuscitation after delivery:

**Polyhydramnios:** Excessive amniotic fluid present (rare, happens in approx. 1% of pregnancies)

<table>
<thead>
<tr>
<th>Antepartum Factors</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Maternal Diabetes</td>
<td>Premature rupture of the membranes</td>
</tr>
<tr>
<td>Gestational hypertension/pre-eclampsia</td>
<td>Fetal Hydrops</td>
</tr>
<tr>
<td>Chronic Hypertension</td>
<td>Post-term gestation</td>
</tr>
<tr>
<td>Fetal anaemia</td>
<td>Multiple gestation</td>
</tr>
<tr>
<td>Previous fetal/neonatal death</td>
<td>Preterm delivery</td>
</tr>
<tr>
<td>Bleeding in the second/third trimester</td>
<td>Size-date discrepancy</td>
</tr>
<tr>
<td>Maternal infection</td>
<td>Adrenergic agonists</td>
</tr>
<tr>
<td>Maternal cardiac, renal, pulmonary, thyroid or neurological disease</td>
<td>Maternal substance abuse</td>
</tr>
<tr>
<td>Polyhydramnios</td>
<td>Fetal malformation or anomalies</td>
</tr>
<tr>
<td>Oligohydramnios</td>
<td>Decreased fetal activity</td>
</tr>
<tr>
<td>No prenatal care/check-ups</td>
<td>Advanced maternal age (&gt;35 years old)</td>
</tr>
</tbody>
</table>

Oligohydramnios: the opposite of polyhydramnios, there is not enough amniotic fluid

Foetal Hydrops: Abnormal accumulation of fluid in two of more foetal compartments (ascites, pleural or pericardial effusion, skin oedema)

Unique anatomy and physiology at the foetal/Maternal interface

The placenta and umbilical cord

The placenta is an intricate organ specifically adapted to its functions of:

- Delivery of nutrients (oxygen and glucose) to the growing foetus
- Removal of waste products from the foetus
- Protection of the foetus (barrier)
- Endocrine function (secretion of prostaglandin to keep ductus arteriosus open)

The placenta is an organ similar to the lungs, in that the pressure of blood flow through the placenta is very low, and the volume of blood flowing through this system is high. This organ’s major role is exchange of gases and delivery of energy substrate to the foetus.
For more detailed understanding of the placenta structure and physiology, watch the video below:

A YouTube element has been excluded from this version of the text. You can view it online here: https://neonates.pressbooks.com/?p=20

The Umbilical cord

Figure 1: THE HUMAN UMBILICAL CORD

ACTUAL CROSS SECTION

Outer Covering
Cord Lining WJ
Intermediate WJ
Perivascular WJ
The 3 vessels

WJ = Wharton’s Jelly

DIAGRAM TO SHOW COMPONENTS


A YouTube element has been excluded from this version of the text. You can view it online here: https://neonates.pressbooks.com/?p=20
Foetal Circulation

Foetal haemoglobin

Foetal haemoglobin is specifically designed to maximise oxygen extraction from maternal blood. Foetal haemoglobin requires a low FiO2 to achieve maximal saturation. This form of haemoglobin makes it possible for the foetus to flourish in such a hypoxic environment within the uterus. For a detailed look at how foetal haemoglobin is structured and functions see the video below:
As is shown in the diagram to the left, foetal haemoglobin is particularly designed to maximize oxygen carrying even at lower PaO2’s than adults. Before birth, the foetus receives oxygen from the placental surface, from the mother’s arterial system.

Reference: http://11e.devbio.com/wt1803.html

The foetal lungs are not functional prior to delivery, the lungs are collapsed and are very minimally perfused. They are not active yet. The lungs are filled with fluid only, and there is no air in the alveoli at all. The vessels that supply the lungs are constricted due to the reduced requirement for flow through the lungs, and the generally hypoxic environment of the foetal circulation prior to delivery. The flow through the lungs is incredibly low, while the pressure through the lung vasculature is very high.

Before the baby is delivered, the pulmonary circuit in the foetus is a high pressure, low flow circuit with minimal delivery of blood to pulmonary interface.

The right side of the heart pumps most of its blood into the aorta via the ductus arteriosus and through the foramen ovale (an opening in the atrial septum that allows shunting from right to left) in order to allow for a large majority of blood to bypass the lungs.

Reference: https://my.clevelandclinic.org/health/diseases/17326-patent-foramen-ovale-pfo
## Foetal Development (major organ systems)

<table>
<thead>
<tr>
<th>Period</th>
<th>Respiratory</th>
<th>CVS</th>
<th>Neuro</th>
<th>Renal/ Digestive</th>
<th>Integumentary</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Weeks 0-9 Embryonic period</strong></td>
<td>Lungs begin to develop week 3</td>
<td>Liver produces red blood cells Only the heart and circulatory system is functional at this point</td>
<td>End of 9 weeks the major divisions of the brain are present Startle response present from around 7 weeks</td>
<td>Small volumes of urine are produced by the 5th week</td>
<td>Eyes are present but eyelids fused shut</td>
</tr>
<tr>
<td><strong>Weeks 9-12</strong></td>
<td>Breathing movement starts as early as 11 weeks Bronchioles start to develop</td>
<td>Bone marrow begins to produce red blood cells (RBC)</td>
<td>Sensory development starts, foetus has blinking movements, sucking movements starts in lips Foetus can hiccup</td>
<td>Liver secretes bile, swallowing present Urine starts being produced Stomach lining in place but acid not produced (until delivery) Liver stores glycogen</td>
<td>Hair starts to grow on the head</td>
</tr>
<tr>
<td><strong>Weeks 13-16</strong></td>
<td>By week 16 lungs are fully developed but not yet fully functional</td>
<td>Myelination starts in the spinal cord</td>
<td>Meconium present In the lower intestine Loops of Henle are present and able to reabsorb</td>
<td>Sebaceous glands produce vernix and lanugo grows</td>
<td></td>
</tr>
<tr>
<td><strong>Weeks 17-21</strong></td>
<td>Type 1 and 2 pneumocytes are differentiated</td>
<td>Increased movement</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Weeks 21-30</strong></td>
<td>Surfactant is produced by around week 24 Bone marrow completely takes over RBC production</td>
<td>Myelination starts to occur of the axons of brain in the nervous system The major areas of differentiation are present in the brain Sleep and wake cycles begin</td>
<td>Infante has bursts of sucking motion</td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Weeks 31-birth</strong></td>
<td>Systems mature and</td>
<td></td>
<td>Lots of fat is stored and lanugo starts to disappear Nails grow</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

For more information on the specific development can be found at the following site: [http://philschatz.com/anatomy-book/contents/m46316.html](http://philschatz.com/anatomy-book/contents/m46316.html)
The birthing process

What about when a baby is delivered via Caesar?
Making the Switch

Once the infant is delivered, and takes its first breathe, there will be a sudden reversal of the pressure and flow relationships in the foetus and a sudden drop in resistance of the pulmonary circuit. This leads to flow being directed from the right side of the heart into the low pressure, high flow circuit that makes up the pulmonary circulation (when exposed to higher concentrations of oxygen these vessels dilate).

The lungs then take over the work that the placenta was performing, the constriction of the placental vessels (and more importantly the umbilical vessels) results in a natural pressure shift to the circulation system we find in the adult and older neonate.
What big things that must to happen to allow the flow to change?

1. Pressure on the umbilical cord cuts the placenta out of the neonatal circulation (this happens spontaneously in a natural delivery as “Wharton’s Jelly constricts in response to a temperature drop, or it happens mechanically with a cord clamp). This increases resistance in the umbilical cord towards the placenta, absent flow and increased pressure.

2. The cold stimulus and the massive increase in pressure where the placenta used to be causes stimulus that agitates the baby, resulting in a change in blood flow to the pulmonary arteries, the fluid filled alveoli are filled with air as the baby takes its first breath. Fluid is absorbed into the low-pressure pulmonary circuit, this results in increased oxygen levels, and the pulmonary arteries dilate even further.

![After birth diagram](https://www.slideshare.net/golwalkar/neonatal-resuscitation-2012-ag)

The change in pressure with increased left sided pressure results in a closure of the foramen ovale (blood flows against the flap, closing off this communication between the right and left atrium). Similarly, the ductus arteriosus does not sustain flow as the pressure on the left
side is so much higher in the aorta than in the pulmonary circuit. Smooth muscle in the ductus arteriosus is affected by increased oxygen levels, and the removal of the placental prostaglandin, this results in a decrease of flow in this vessel and eventually closure of this vessel.

Because of the dramatic increase in oxygenation (21% on room air, as opposed to the lower levels the foetus is used to in the womb), room air is usually enough for the neonatal pulmonary vessels to dilate.

<table>
<thead>
<tr>
<th>Structure</th>
<th>Before birth</th>
<th>After birth</th>
</tr>
</thead>
<tbody>
<tr>
<td>Umbilical vein</td>
<td>Bring arterial blood and liver to the heart</td>
<td>Obliterated and become round ligament of the liver</td>
</tr>
<tr>
<td>Umbilical arteries</td>
<td>Bring arterio-venous blood to the placenta</td>
<td>Obliterated and becomes the vesicle ligament on anterior abdominal wall</td>
</tr>
<tr>
<td>Ductous venoses</td>
<td>Shunt arterial blood into the IVC</td>
<td>Becomes the ligamentum venosum</td>
</tr>
<tr>
<td>Ductous arteriosis</td>
<td>Shunt the arterial and some venous blood from the pulmonary artery to the aorta</td>
<td>Becomes the ligamentum arteriosus</td>
</tr>
<tr>
<td>Foramen Ovale</td>
<td>Connects right and left atria (auricles)</td>
<td></td>
</tr>
<tr>
<td>Lungs</td>
<td>Contain no air and are not well perfused with blood</td>
<td>Air filled and are well perfused with blood</td>
</tr>
<tr>
<td>Pulmonary arteries</td>
<td>Pump very little blood into the lungs</td>
<td>Pump blood well to the lungs</td>
</tr>
<tr>
<td>Aorta</td>
<td>Receives blood from both ventricles</td>
<td>Receives blood from only the left ventricle</td>
</tr>
<tr>
<td>Inferior vena cava</td>
<td>Brings venous blood from the body and arterial blood from the placenta</td>
<td>Returns venous blood from the body to the right atrium</td>
</tr>
</tbody>
</table>
Summary of specific Organ Systems in the changeover

Cardiovascular

- Loss of umbilical blood flow to and from the placenta (causing increase in systemic vascular resistance with clamping)
- Closure of the ductus venosus
  - This structure closes within a few days of delivery (around 5-7 days)
- Closure of the ductus arteriosus
  - This is a functional not anatomical closure (meaning it is not yet formalised and a change in flow pressures on the right side of the heart can result in this opening up to flow again)
  - This occurs as a result of increased oxygen levels and a decreased carbon dioxide level, which inhibits prostaglandins E1 & E2 and results in vasoconstriction of the vessel
- Large increase in the pulmonary circulation which leads to:
  - Closure of the foramen ovale
    - As a result of the change in pressure when the umbilical cord is clamped off (rise in pressure on the left side and drop in pressure on the right side as the low-pressure pulmonary system opens up)
    - This structure closes within a few breaths after delivery and won’t reopen unless changes in pressure occur
- Increased renal blood flow as the renal arterial vascular resistance decreases
- This results in an increase in glomerular filtration rate
- Changes in flow to the skin (increased flow)
- The oxy-haemoglobin dissociation curve shifts from the left to the right as more oxygen is available

The process of switching over the CVS

The first breath occurs and the lungs expand, pulmonary blood flow increases and there is a drop in PVR (peripheral vascular resistance), there is a bolus of blood from left atrium and left ventricle which causes a reversal of pressure from foetal circulation to neonatal circulation (almost immediate closure of the foramen ovale)
**Respiratory**

- Traveling through the birth canal increases the pressure on the foetal chest, resulting in fluid being forced out of the lungs
- Remaining fluid is reabsorbed into the pulmonary circulation
- Placental gas exchange ends, and the pulmonary system takes over the job of gaseous exchange
- Functional residual capacity is built, and the lungs do all the work
  - Expected functional residual capacity (FRC) @ 10min = 20mL/kg (approx.)
  - Expected FRC 60min FRC = 30mL/kg (approx.)
- The infant has high alveolar ventilation to maintain CO₂ levels within safe norms, this is maintained by increased respiratory rates (norm 40-60b/min)

**Endocrine/Integumentary**

- The infant becomes responsible for its own blood sugar control, temperature regulation and hormone regulation as soon as the placental blood flow is clamped shut. This means places the infant at risk for derangement as it adjusts to life outside the uterus
  - The infant is more susceptible to cold stress and drops in temperature in the first few hours post-delivery as the ambient temperature decreases from internal to external environment (body surface area to weight ratio)
  - The infant has a drop in blood sugar levels in the first 2 hours post-delivery and may need supplementation if not actively feeding yet (if the infant needs resuscitation for instance)

**References:**


Unknownauthor. 2004. Neonatalphysiologyandphysiologicalchangesafterbirth [available online]


The Premature Infant

Preterm infants are at major risk for increased mortality and morbidity, without the right treatment, those that do survive are likely to do so with long term disability and poorer quality of life.

Some definitions

**Preterm birth:** Any delivery before 37 weeks gestation (WHO. 2015), this is the biggest determinant of adverse outcomes.

**Borderline of viability:** this is a term used to describe the infant who is born alive on or before gestational age of 25 weeks and 6 days

**Non-Viable:** Any delivery before 22 weeks and 6 days is considered non-viable and resuscitation efforts should not be made (this is not a legal definition but rather a clinical definition)

**Medical futility** the judged futility of medical care, used as a reason to limit care. Two reasons for making this judgment are (1) to conserve resources and (2) to protect clinician integrity. The types are physiologic futility and normative futility.

- **normative futility** a judgment of medical futility made for a treatment that is seen to have a physiologic effect but is believed to have no benefit.
- **physiologic futility** a judgment of medical futility based on the observation of no physiologic effect of the treatment.

The grey area of the infant born between 22 weeks 6 days and 25 weeks 6 days is a challenge, routine resuscitation measures are often not instituted as the chance of survival decreases in births before 25 weeks. Resuscitation would not normally be started UNLESS the family and parents are determined to resuscitate despite counselling and discussion. In some cases (such as from 24 weeks on, this care and admission to NICU may be offered unless the family and treating practitioner decide that the resuscitation is futile.
What about in South Africa?

Stillborn: Defined as a foetus that had at least 26 weeks of gestation and was born without any signs of life

Viability: is not defined in South African Law, there are two cases (that were seen in two different courts) that came to two different conclusions:

- A foetus is capable of independent existence at 25 weeks gestation
- In terms of concealment of birth, a foetus is only considered viable (or a child) after 28 weeks

A further complication is that a foetus may not be terminated unless there is a medical or “other specific reasons” after 20 weeks gestation.

This makes the legal definition VERY tricky

You can read more about these definitions at the following link:
https://www.researchgate.net/publication/51666989_Ethical_issues_in_neonatal_intensive_care

What places the preterm infant at risk?

The Premature Infant

- Impaired respiration
- Difficult in feeding (inadequate suck reflex)
- Poor body temperature regulation
- High risk of infection as immunity not yet fully developed on birth
Specific management of the premature neonate is covered under the resuscitation chapter, with specific recommendations for care provided with appropriate evidence.
Assessment and Stimulation

Assessing the neonate in the first few moments

The neonatal resuscitation algorithm is a good place to start when trying to determine what needs to be done for a neonate who has just been delivered. This is the algorithm that will be used and then later adapted for the patient who presents post-delivery, and then later in the neonatal period.

### Primary Assessment

#### Airway
- Ensure airway open, maintained and protected (use basic to advanced if needed)

#### Breathing
- Rate: normal for infant 40-60b/min
- A/E: should be clear on both sides
- Colour: central/peripheral cyanosis
- Effort: Nares, accessory muscle use, retractions, tugs, abdominal muscle, head bobbing.
- ETCO2 assessment
- SPO2: pre and post ductal sats NB

#### Circulation
- Rate: 110-180 is around normal
- AVPU
- Colour: mottling, pale, pallor
- End organ perfusion: CRT<2sec, wet nappies in the last 24h
- Systolic BP: reference norms for age

### Disability
- HGT
- Pupils

### Exposure
- Temperature (hot/cold both concerning)
- Rash, trauma, toxins

### Secondary Assessment
- SAMPLE history
- Head to toe assessment
- Mechanism of Injury/illness
- Other assessments/labs
  - Bloods
  - Scans/X-rays
  - ECG
  - Sonor
Neonatal Resuscitation Algorithm—2015 Update

1 minute

Antenatal counseling
Team briefing and equipment check

Birth

Term gestation? Good tone? Breathing or crying?
Yes

Warm and maintain normal temperature, position airway, clear secretions if needed, dry, stimulate

No

Infant stays with mother for routine care: warm and maintain normal temperature, position airway, clear secretions if needed, dry. Ongoing evaluation

Apnea or gasping? HR below 100/min?
Yes

PPV
Spo₂ monitor
Consider ECG monitor

Labored breathing or persistent cyanosis?
Yes

Position and clear airway
Spo₂ monitor
Supplementary O₂ as needed
Consider CPAP

No

Postresuscitation care
Team debriefing

HR below 100/min?
Yes

Check chest movement
Ventilation corrective steps if needed ETT or laryngeal mask if needed

No

HR below 60/min?
Yes

Intubate if not already done
Chest compressions
Coordinate with PPV
100% O₂
ECG monitor
Consider emergency UVC

No

Targeted Preductal Spo₂
After Birth

1 min  60%-65%
2 min  65%-70%
3 min  70%-75%
4 min  75%-80%
5 min  80%-85%
10 min 85%-95%

© 2015 American Heart Association
It is best to read the next part of this page with the neonatal resuscitation algorithm in front of you to reference.

Another resource that can be used in the South African setting is the Resuscitation Council of SA algorithm, this algorithm is a more South African specific approach and will be seen in many of the settings where new-born infants are seen. http://www.resus.co.za/index.php/newborn-resuscitation-algorithm

**Ask the 3 questions:**

1. **Is the infant a term infant?**
   - How many weeks was the baby “inside” for? If pre- or post-date infant, consider that the infant may present unique challenges (more information on this can be found under special circumstances later in this post)

2. **Does the infant have good tone?**
   - Assess the appearance of the infant for movement of the limbs and the muscle tone, the limp infant should set off alarm bells immediately.

3. **Is the infant breathing or crying?**
   - The healthy infant should be active, crying and breathing well (have a quick look to see how the infant is breathing as effort also indicates red flag areas)

See below for some examples of “normal” findings of these three questions as well as “abnormal” or red flag findings.
Question 1: What is the gestational age of the infant?

Notice that the post-date infant is covered in meconium and is large (with good fat deposits), while the premature infant is small and thin.
**Question 2: Does the infant have good tone?**

The healthy infant should be crying and breathing, with a rate of about 60 b/min initially dropping to the normal range of 40-60 b/min.

Initially, the patient may breathe with some increased effort as the fluid is shifting out of the lungs and the infant is creating functional residual capacity and expanding lung tissue for the first time with air, this effort should decrease over time until the infant is restful. Signs of distress include retractions, nasal flaring, accessory and abdominal muscle use to breathe. See the video below for an example of a patient with respiratory distress.

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**Question 3: Is the infant breathing or crying?**
A YouTube element has been excluded from this version of the text. You can view it online here: https://neonates.pressbooks.com/?p=32
**Next steps**

We have already covered the three questions that need to be asked when a neonate is encountered:

1. Is the infant term?
2. Does the infant have good tone?
3. Is the infant breathing or crying?

If the answer to these questions is **yes**, then the infant is likely stable enough not to need immediate intervention and the infant can be assessed more thoroughly.

In the case of a new-born infant presenting, the cord will need to be cut, this can be delayed in the healthy infant. This means that immediate clamping of the cord is not required, and this could be delayed for 30 seconds (AHA. 2017).

If the answer to these questions is **no**, then the infant needs assistance.

As is shown below, most infants only require the most basic assistance to get them breathing adequately. This hierarchy of needs can be described best as the “inverted triangle” of neonatal resuscitation, which indicates the interventions that are needed most often all the way down to the least common interventions.
The numbers that run on the far right of the diagram indicate what percentage of infants will require this intervention.

Simple methods and interventions are the most commonly required, advanced procedures and interventions are not commonly needed and when they are, the chance of positive outcomes for the infant decrease.

The baby should be warmed, dried and stimulated for no more than 30 seconds.
The head and body should be dried, and then new towels placed to wrap the infant in afterward.

The infant can be agitated by rubbing the trunk front and back, without bruising or injuring the infant.
The Airway

The positioning of the airway should be accomplished through a towel placed behind the infant’s shoulders to allow for a natural extension of the neck.

Reference: http://www.ijciis.org/article.asp?issn=2229-5151;year=2014;volume=4;issue=1;spage=65;epage=70;aulast=Harless
The airway should be clear of secretions and should be open
- The airway can be opened using basic manoeuvres (head tilt-chin lift) to maintain the airway in the open “sniffing position”
- Suction the airway if it is obstructed with secretions
- Be careful not to hyper-extend the infant’s head as the soft tissues of the airway may result in closure of the airway in the opposite direction. Consider placing padding behind the infant’s neck.

Important update: The airway **SHOULD NOT BE ROUTINELY SUCTIONED** unless there is an obstruction, not all infants require their airway to be suctioned.

This is for a number of reasons:

1. Decreased stimulation of the vagus nerve (decreased incidence of bradycardia)
2. Decreased risk of removing the residual volume within the lungs, and protection of pulmonary compliance
3. Reduced risk of hypoxia due to prolonged suction attempts

Reference: AHA. 2017
Ok... But what about meconium-stained infants?

If an infant born through meconium stained liquor is NON-VIGOROUS (poor muscle tone and poor respiratory effort) there should be no change to the initial steps of resuscitation (except that these should take place under a warmer).

Routine intubation for deep tracheal suctioning is NO LONGER RECOMMENDED (AHA. 2017) as there is not enough evidence to show benefit, and there may be harm as the delay to first ventilation (and thus oxygenation) is longer.

If there is evidence of an airway obstruction once PPV has been initiated, then the approach may include intubation and suctioning.

Some suctioning rules (if you HAVE to suction)

- Use a bulb syringe if possible, to prevent harm to the infant from high suction pressures
- The pressure on a wall mounted or mechanical suction device should be set to a maximum of 100mmHg (Kattwinkel, J. 2016)
- Suction first the mouth and then the nose
• Don’t suction too deeply or vigorously as the patient by experience a bradycardia
• **DON’T suction for longer than 5 seconds**
• If a bradycardia occurs, stop suctioning immediately and replace oxygen/PPV

**Why no more than 30 seconds?**

The infant that does not respond to stimulation within the first 30 seconds will more than likely **not respond to further stimulation**. Neonates who have undergone extended periods of hypoxia or asphyxia frequently present with apnoea. There are two types of apnoea, which are not distinguishable initially.

**Primary apnoea** occurs initially as a response to distress and hypoxia, the respiratory rate is affected but the heart rate and perfusion of the infant is not.

**Secondary apnoea** occurs after more prolonged hypoxia, and the result is a drop in the respiratory rate, perfusion and heart rate.

These patients will not respond to stimulation alone and will more than likely require PPV (Positive pressure ventilation) as well as possibly chest compression.

More than 30 seconds of warming, drying and stimulating with no response is likely to increase apnoea time and thus hypoxic damage to the neonate’s physiology, thus if no response to stimulation is gained in 30 seconds, care MUST be escalated rapidly. See the graph below for the two types of apnoea:
Most infants with secondary apnoea respond well to PPV (with a bag-valve-mask device) and will recover relatively quickly. Infants who have been exposed to long periods of hypoxia may have an element of myocardial suppression and will require escalation to the next intervention (chest compressions).

Once the basics have been completed and the infant has been stimulated you need to start assessing the infant’s vital signs.

Rapid Assessment Summary

1. 3 questions
2. Breathing rate assessment (count this over 10 seconds anything less than 3 breaths in 10 seconds is worrying)
3. Place 3 lead ECG and assess heart rate (anything less than 100b/min is worrying)
4. Put a saturation probe on the right hand and assess (base decisions on expected norms for time since delivery)
5. Get a weight on the infant ASAP
Important Note: Assessing the Neonatal heart rate

Heart rate is difficult to assess on the new-born (immediately after birth and the older neonate), for this reason, the 2015 updated guidelines for neonatal resuscitation recommend that a three-lead ECG be placed as a matter of urgency as soon as possible in the patient who is not vigorous after being warmed, dried and stimulated.

- A 3 lead ECG is quicker to get on and more accurate than a saturation monitor
- It can get a reading rapidly and shows real-time data (unlike the SPO$_2$ monitor)
- 3 lead ECG is easy to apply and requires little training for accurate reading
**Decision point Number 1**

Once the infant has been warmed and stimulated, the first question becomes about whether the patient is still apnoeic or gasping, and whether the heart rate is below 100b/min.

**If the answer is no,** and the infant is in respiratory distress or has laboured breathing, the infant is more stable and less invasive management is started: this would include positioning of the airway, and clearing it if not already done, placing SPO2 monitoring if not already done, and giving the patient oxygen.

*See below for more information on laboured breathing and signs of increased work of breathing.*

NIV (non-invasive ventilation) or high-flow nasal cannula should be considered at this point for ongoing management

**If the answer is yes,** the infant needs more urgent and advanced intervention
Positive pressure ventilation should be started ASAP *(for information on PPV and how to perform it safely please follow the link: ventilation)*
Expanded Assessment

(this is used when there is time and the patient is not a “resuscitation” patient)

Apgar score can be done at this point, simultaneously with other assessments (if the patient is freshly new-born)

Breathing

- RACES
  - Rate (40-60b/min)
  - Air entry (listen to the chest and confirm that air is moving in all lung fields)
  - Colour (look for signs of cyanosis or poor oxygen exchange)
  - Effort (look at the way the patient is breathing and check for signs of impending failure/respiratory distress)
  - Saturation readings (in the neonate the SPO2 probe should be placed in a spot that is “pre-ductal” and it would be helpful to have a second reading on a limb that represents “post-ductal” see the information below to understand this statement)
    - There are certain acceptable SPO2 limits that apply to the neonate in the delivery room, and these should be adhered to, in order to prevent hyperoxia in the neonate
Expected SPO2 readings after delivery

<table>
<thead>
<tr>
<th>Targeted Preductal SpO₂ After Birth</th>
</tr>
</thead>
<tbody>
<tr>
<td>1 min</td>
</tr>
<tr>
<td>2 min</td>
</tr>
<tr>
<td>3 min</td>
</tr>
<tr>
<td>4 min</td>
</tr>
<tr>
<td>5 min</td>
</tr>
<tr>
<td>10 min</td>
</tr>
</tbody>
</table>

Circulation

RACES approach

- Rate (heart rate should be above 100 to be considered normal and anything less than 60 should be treated with chest compression after ruling out hypoxia as a cause)
  - The best method for assessing the heart rate is to use a 3 lead ECG (AHA. 2017)
- AVPU should be assessed and level of consciousness checked
- Colour should be assessed for circulatory colour changes such as mottling, paleness, jaundice
- End organ perfusion should be assessed, this is assessed in the form of capillary refill in all limbs
- Systolic blood pressure should be assessed (with an appropriately sized cuff)
# Normal Expected Vital Signs for a Neonate

<table>
<thead>
<tr>
<th>Normal Neonatal Vital signs</th>
<th>Resting</th>
<th>Awake</th>
<th>DANGER ZONE</th>
</tr>
</thead>
<tbody>
<tr>
<td>Heart rate</td>
<td>90-160</td>
<td>100-205</td>
<td>&lt;100 look for reason (hypoxia)</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>&lt;60 Start Compressions</td>
</tr>
<tr>
<td>Respiratory Rate</td>
<td>30-53</td>
<td>30-60</td>
<td>&lt;25 consider assisting</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>&lt;20 Ventilate</td>
</tr>
<tr>
<td>Blood Pressure</td>
<td></td>
<td></td>
<td>&lt;Systolic 40-50mmHg</td>
</tr>
<tr>
<td>12 hours or &lt;1000g Neonate</td>
<td>Systolic 39-59</td>
<td>Diastolic 16-63</td>
<td>&lt;Systolic 50mmHg</td>
</tr>
<tr>
<td>12 hours or 3kg Neonate</td>
<td>Systolic 60-70</td>
<td>Diastolic 31-45</td>
<td>&lt;Systolic 60mmHg</td>
</tr>
<tr>
<td>Neonate (96 hours)</td>
<td>Systolic 67-84</td>
<td>Diastolic 35-53</td>
<td></td>
</tr>
</tbody>
</table>


Reference: https://www.pedscases.com/sites/default/files/Vital%20Signs%20Reference%20Chart%201.2_2.pdf Blood Pressure Cuff sizes:
Disability assessment

At this point you need to assess the infant for the following:

- Pupils (this will assist in determining if there is any opioid toxicity, or possible injury intracranially causing increased ICP)
- Blood sugar (though rarely low immediately after birth could be a reason for a “floppy baby”
  - Blood glucose concentrations should be maintained above 2.5mmol/l in the neonate (Osborn, D. 2010) levels below this should be avoided

Exposure

Look at the infant for signs of

- Rashes (this is unlikely in the newly born infant but if the infant has come in from home/ward this is something to check for)
- Trauma (visually scan the infant for signs of trauma, perform a thorough head to rule out trauma if possible)
- Temperature (vital in the assessment of the neonate due to their incredibly large BSA (body surface area) and risk of heat loss especially in the first few hours following birth
  - It is recommended that the temperature of the newly born infant be maintained from 36.5-37.5 degrees Celsius (American Heart Association. 2015.)

![Normal Temperature Range by Method Reference: CPS Position Statement on Temperature Measurement in Pediatrics, 2015](image)

<table>
<thead>
<tr>
<th>Method</th>
<th>Temperature (°C)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Rectal</td>
<td>36.6-38</td>
</tr>
<tr>
<td>Ear</td>
<td>35.8-38</td>
</tr>
<tr>
<td>Oral</td>
<td>35.5-37.5</td>
</tr>
<tr>
<td>Axillary</td>
<td>36.5-37.5</td>
</tr>
</tbody>
</table>

Temperature ranges do not vary with age. Axillary, tympanic and temporal temps for screening (less accurate). Rectal and oral temps for definitive measurement (unless contraindication).

**Weight Calculations and Equipment Sizing**

Where possible **ALWAYS** use an actual weight (measured weight on the day) to determine the weight of the infant for accuracy. Failing this, a length-based measure to determine the infant’s weight is also acceptable (though has not been tested specifically in the neonate population).

Practitioners are discouraged from “guesstimating” the weight of the patient, as this is notoriously unreliable (Wells, M. 2017).

For more equipment sizing, you will receive a “cheat sheet” of the information you need to assist you, and more information is presented in each of the relevant learning areas as you progress. This should be done for all infants requiring resuscitation (weight calculation) to determine the most accurate drug and fluid doses.
Super Important Note: APGAR

APGAR scores are not indicated until the infant has been determined not to need resuscitation!

As the first APGAR is required only at 1 minute, if we used it as a tool to determine the need for resuscitation, we would delay resuscitation for over a minute before we started. For this reason an APGAR is only completed formally in a patient who DOES NOT need resuscitation.

Another Summary of Assessment can be found at the link below:
https://www.openpediatrics.org/assets/video/newborn-exam

References:


Novac, C. 2016. Pediatric Vital Signs. [available online]

Osborn, D. 2010. The Royal Prince Alfred Hospital Newborn Care Guidelines, Neonatal Hypoglycaemia. [available at]

https://www.nhlbi.nih.gov/sites/default/files/media/docs/hbp_ped.pdf

Oxygen and Positive Pressure Ventilation

Oxygen is a medication and is not without its risks and adverse effects. This is no less true about the resuscitation of a neonate.

There is a lot of literature to support the idea that the neonate (even the healthy infant) does not reach “normal” oxygen levels within their blood until about 10 minutes into their lives.

Oxygen saturation readings may present in the 70-80% range for a few minutes following birth as the circulatory and pulmonary systems start functioning more in line with adult norms (AHA. 2017).

There is plenty of evidence that hypoxia in this period is not ideal, and also a lot of evidence that HYPEROXIA in this period is potentially dangerous to the infant.

For this reason, the 2015 update on Neonatal Resuscitation recommends that resuscitation be started with 21% (room air) up to a 30% (if an oxygen blender is available) and that oxygen saturations (preductal) be targeted to the expected norms according to the following table for time since delivery.

<table>
<thead>
<tr>
<th>Targeted Preductal SpO₂ After Birth</th>
<th>1 min</th>
<th>60%-65%</th>
</tr>
</thead>
<tbody>
<tr>
<td>2 min</td>
<td>65%-70%</td>
<td></td>
</tr>
<tr>
<td>3 min</td>
<td>70%-75%</td>
<td></td>
</tr>
<tr>
<td>4 min</td>
<td>75%-80%</td>
<td></td>
</tr>
<tr>
<td>5 min</td>
<td>80%-85%</td>
<td></td>
</tr>
<tr>
<td>10 min</td>
<td>85%-95%</td>
<td></td>
</tr>
</tbody>
</table>

The same recommendation applies to infants who are premature, however a maximum level for resuscitation of the preterm infant has been set at 65%.
Oxygen can be delivered via a number of different devices (where the heart rate is above 100b/min but there is still evidence of respiratory distress)

Reference: http://fn.bmj.com/content/88/2/F84

Oxygen delivered via the wall is routinely dry and cold (non-humidified), as soon as possible, provision for the delivery of warm humified oxygen must be made to prevent the drying effects on the neonatal respiratory structures and possible heat loss from the cool air (convection) (Kattwinkel, J. 2016)

Reference: http://fn.bmj.com/content/88/2/F84
Using Positive Pressure

When should PPV be used?

When the infant is not breathing, or is gasping, or the heart rate is less than 100b/min

Some Definitions

**PIP**: Peak inspiratory pressure is the pressure delivered to the lungs each time the bag is squeezed; this is the maximum pressure within the larger airways at the end of inspiration.

**PEEP**: Positive end expiratory pressure that remains in the airways at the end of expiration. The consensus regarding application of PEEP in the neonate is that all neonates ventilated with PPV should have a PEEP of 5cmH20 applied to the device (AHA. 2017). PEEP applied may reduce the FiO2 required to achieve acceptable saturation readings in the infant. Increasing the PEEP applied (within certain limits), allows the delivery of oxygen to be improved without the administration of a higher percentage of oxygen. Less “drug” with better effect.

Types of devices for PPV

**Flow inflating bag**

This is the kind of bag that is often present in anaesthetic departments, the bag fills with pressure from a compressed gas source and fills when the outlet is occluded. This device is usually capable of mixing gases to a specified mix (to achieve a set FiO2). PEEP and PIP are easily controlled by the flow.

**Self-inflating bag**

This is the kind of device most commonly found in the ED/prepositional environments (BVM) and possibly the only device available in some maternity units/delivery rooms. This bag
inflates spontaneously once the breath has been delivered and does not need a compressed gas source to function.

The risk with a bag like this is that peak inspiratory pressure is often not measurable and may be higher than is safe (unless a pressure manometer can be applied to the device). PEEP can be applied via an external valve that attaches to the device.

Reference: [http://bag.wikicamino.net/flow-inflating-bag/](http://bag.wikicamino.net/flow-inflating-bag/)

**T-Piece resuscitator**

In South Africa also known as the Neo-Puff (registered trademark), this device is not common outside of the specialized neonatal care units and ICU ambulances. This device provides controlled flow and pressure and only works with a compressed gas source. PEEP and PIP are controlled by the machine.

The size of the bag

Infants require only about 4-6ml/kg of tidal volume, a bag should be able to provide this without providing too much tidal volume to the infant. The recommended bag size is 200ml – 750ml (absolute max) for the infant (Kattwinkel, J. 2016). Even a term infant will require no more than 25ml tidal volume per breath.

The Mask and getting a good seal

The best mask is the one that fits

A mask that fits a new-born’s face does not cover the eyes or chin. A seal is important to prevent air escaping from the sides of the mask.

Pressure applied to the face to achieve a good seal should not be excessive as this may lead to trauma to the face and head and may result in injury.

The mask should be placed from the bridge of the nose down to the chin, with the fingers of the hand providing the “jaw thrust” or head tilt to ensure good chest rise.
Try not to apply pressure to the infant’s eyes, this can result in stimulation of the vagus nerve and thus a resultant bradycardia (oculo-cardiac reflex).

**How do I know I am doing a good enough job with ventilation?**

1. The heart rate is the most sensitive indicator that the ventilation is adequate (if the heart rate is increasing then oxygenation is improving)
2. Chest rise and chest wall excursion can be used to determine if enough air is being delivered
3. Air entry to all lung fields is a good indication that air is moving into the alveoli
4. End tidal CO2 concentrations are being read (this confirms absence of occlusion of the airway)
5. If you can measure the pressure (PIP) being delivered, you should aim for a pressure of around 20cmH2O.

If these end points cannot be achieved, consider changing something to improve the ventilation.
The decision to move to advanced airway is covered a little later, a first step is to maximize BVM ventilation: the mnemonic that has been developed for this is:

**MR SOPA**

<table>
<thead>
<tr>
<th>Letter</th>
<th>Stands for</th>
<th>Action</th>
</tr>
</thead>
<tbody>
<tr>
<td>M</td>
<td>Mask</td>
<td>Reapply the mask, change the mask for a better size/fit, consider using two hands and jaw thrust to achieve a seal Consider OPA insertion if not already done</td>
</tr>
<tr>
<td>R</td>
<td>Reposition</td>
<td>Reposition the airway (place the head more neutral or extend slightly more)</td>
</tr>
<tr>
<td>S</td>
<td>Suction</td>
<td>Bulb syringe or wall mounted device to suction mouth and then nose if there are secretions visible</td>
</tr>
<tr>
<td>O</td>
<td>Open Mouth</td>
<td>Jaw thrust and open the mouth of the infant, place OPA if not yet done</td>
</tr>
<tr>
<td>P</td>
<td>Pressure</td>
<td>Increase the pressure of the PPV in small increments (5-10cmH2O) to a maximum of 40cmH2O</td>
</tr>
<tr>
<td>A</td>
<td>Alternate Device</td>
<td>Consider and start planning for placement of ETT (endotracheal tube) or immediately place supraglottic device if clinically indicated</td>
</tr>
</tbody>
</table>

If you are achieving adequate air movement, chest rise and air entry and CO2 readings are positive and there is no change in heart rate, then it’s time to escalate to the next step in resuscitation

**What rate should I ventilate at?**

The rate that is appropriate for the neonate is around 40-60 breaths per minute (AHA. 2017)

That translates to one breath approximately every 2 seconds, one breath every second is probably just too fast, so aim for two breaths every 3 seconds (or one breath every 1.5 seconds). If breaths are given too fast, there is the risk that the alveoli will not be ventilated and so there will be poor gaseous exchange.

**So… how long do I try to ventilate for before I escalate treatment?**

As with everything in neonatal resuscitation, you should give BVM ventilation (or PPV) a trial of 30-60 seconds. If the effect you are looking for is not achieved, then you should escalate care to the next step in the algorithm. Remember the endpoint for monitoring here is a heart rate above 100b/min/spontaneous respiratory effort.
It should also be noted that when BVM ventilation (or PPV) is continued for more than a few minutes, it might be indicated to insert an advanced airway and orogastric tube to assist with distention of the stomach (this will be discussed later under the heading “Advanced Airway”)

References:
Chest Compressions

If the heart rate is less than 60b/min despite adequate BVM ventilation (or an attempt at achieving this with 30-60 seconds and considering MRSOPA), it is time to perform chest compressions!

Airway management is not a fixed point on this algorithm and can come in at any point where it is relevant (this means if the airway is at risk earlier in the patient presentation, it can be considered as a first line management, in tandem with other interventions)

Once chest compressions have started, advanced airway management should be considered as soon as possible to prevent gastric inflation.

For more information on advanced airway management please see the chapter on this later in the book

MOST infants will not need chest compressions as they will respond to the successful application of oxygenation and ventilation. In the unlikely event that oxygen and ventilation doesn’t achieve the desired outcome, compressions will need to be started.
WHY should I do chest compressions…. the baby might have a pulse??

In the infant with a low heart rate, cardiac output is terrible (when the rate is less than 60b/min this is less than half what is normal for the neonate. This means that sometimes even with good oxygenation and ventilation, there is not enough cardiac output to get the oxygen through the system.

Myocardial hypoxia and systemic acidosis mean even further depressed myocardial function, resulting in decreased ability to pump the delivered oxygen to the system.

Chest compressions augment the pumping action of the heart and assist in pumping oxygen to the cells of the system. This also improves cardiac output and coronary perfusion pressure.

Where should I do compressions?

Chest compressions should be performed on the lower third of the sternum (AHA, 2017).

There are two options for chest compressions, when a lone rescuer is present, two fingers are used to apply compressions to the chest along the width of the sternum, whilst standing to the side of the infant.

When a second rescuer arrives, the provider applying compressions moves to the feet of the infant and uses the “thumbs encircling-hands” technique, while the second rescuer applies ventilation from the head of the patient.

The second option is the preferred method for chest compressions as it may increase blood pressure and coronary perfusion pressures (AHA, 2017). This method is also less strenuous on the rescuer, meaning the rescuer can perform compressions of better quality for longer. The two-thumb technique can also be done from the head of the patient, meaning that there is no need to stop compressions for insertion of UV (Umbilical Venous) access.

Reference: http://emj.bmj.com/content/early/2014/11/28/emem-2014-203873

How hard should I push?

The chest of the infant should be compressed approximately 1/3 the anterior posterior diameter of the chest. This means that pressure will change with size and chest wall compliance of the infant.

How fast should I push?

It is recommended that chest compressions be coordinated with ventilation in the care of the neonate, meaning that the ratio and rate of delivery become important. Compression should be delivered in a 3:1 ratio with ventilation, and this should be continued in this ratio EVEN when an advanced airway is in place.
The ratio may be adjusted (15:2 can be considered) if the cause of collapse is thought to be cardiovascular in nature, but as most infants have a drop in heart rate or cardiac arrest as a result of hypoxia, the 3:1 ratio should be the default for management of the neonate.

The rate of delivery is approximately 120 events per minute (or 2 events per second) meaning that there should be 90 compressions and 30 breaths delivered in a minute period. There should be no pause for exhalation after the ventilation, as the first compression will allow this to occur without a delay in compressions.

**How long should I do compressions for?**

Every **30 seconds – 60 seconds**, the patient should be reassessed (for a heart rate above 60 beats per minute), as long as the infant has a heart rate of less than 60b/min, compressions should continue with escalation to medication interventions as needed.

Because delays in chest compressions should avoided, the time taken to assess heart rate should be kept to a minimum (therefore the cardiac monitor application is so important to ensure rapid assessment of heart rate).

When a heart rate of more than 60b/min is detected, compressions should be stopped and the focused shifted back to effective ventilation.

**Other important stuff to remember…**

- Chest compressions should be performed with 100% oxygen, turn the FiO2 up as soon as you start compressions (AHA, 2017)
- Titrate oxygen down as soon as the heart rate improves to avoid unnecessary oxygen administration to the patient
- Full chest recoil must occur after each compression, but the fingers/thumbs should not leave the chest (just release pressure on the chest wall)
What if chest compressions don’t work?

Double check that you are effectively delivering all the interventions you have started:

- Check that ventilation is effective
  - The chest should visibly rise on ventilation
  - Auscultate and ensure that air is moving into the lungs
  - Ensure 100% oxygen is being delivered and that there is enough oxygen in the cylinder
  - Ensure that the reservoir bag inflates after each ventilation
  - If the chest is not rising or air is not moving into the alveoli, attempt MR SOPA
- Check the airway is patent
  - If ETT not in yet, insert one
  - If ETT is already in then assess patency (suction if needed, insert deeper or pull back if needed, make sure it’s not kinked)
- Ensure the quality of chest compressions
- Make sure they are at the correct rate
- Ensure compressions and ventilations are in the right ratios
- Ensure the compressor is pushing hard enough
- Ensure the compressor is allowing full chest recoil

If the heart rate comes up?

There are two possibilities:

- Heart rate is above 60b/min but below 100b/min
  - Continue ventilation at 40 – 60 breaths per minute
  - Reassess patient every 30 seconds to 60 seconds
  - Assess possible reasons for the decreased heart rate
- Heart rate is above 100b/min
• Assess infant’s respiratory rate, if adequate, apply oxygen in lower concentration and monitor patient - If inadequate continue ventilation, consider decreasing FiO2 depending on other clinical findings

And if I can’t get the infant to respond positively?

You need to move into the most uncommon interventions, consider medications (adrenalin and other medications to reverse possible causes of arrest/persistent bradycardia) and consider fluid administration if there are signs of loss of fluid/blood.

This will be discussed in depth in lesson 5: Medications and Fluid administration

Airway management

Important anatomy

In order to place the ETT or other airway devices correctly and with as little trauma to airway structures as possible, the intubator must be comfortable with the relevant airway structures.

The following are important structures within the airway:

Epiglottis: a large flap-like structure that sits over the trachea and prevents passage of fluids or objects (food or other) into the airway. Often this structure must be scooped out of the airway with the laryngoscope blade to gain access to the vocal cords.

Reference: https://kelownavoicelab.com/blog/blog/how-your-voice-works
The vallecula: a blind ending space formed by the base of the tongue and the epiglottis, when using a curved blade, the tip is inserted into this space to pull the epiglottis out of the way of the cords for visualization

Oesophagus: “the food pipe” this muscular collapsible pipe is posterior to the trachea and is usually the first pipe to be visualised when the laryngoscope blade is inserted to the mouth.

Glottis: the opening of the larynx into the top of the trachea, this is bordered internally by the vocal cords, usually visualised as a white/off white line on each side of the airway when looking down the trachea.

Vocal cords: white to off-white ligaments visible in the laryngeal opening into the trachea.

Carina: the bifurcation (split) of the right and left main bronchi into the right and left lung. The Carina is important although it’s not visualised, it is often felt when a tube is inserted too deeply. The right main bronchus comes off the trachea at the carina at a much less sharp angle and the ETT that is too deeply inserted is often inserted into this area.

Reference: http://www.therespiratorysystem.com/trachea/

The airway of the neonate presents a unique set of challenges for the care-provider for a number of reasons, the most important of all is in size.
The neonatal airway is **MUCH** smaller than a child or older infant’s airway, the mouth is much smaller, meaning less space to manoeuvre equipment and the tongue is relatively large for the small space available. The epiglottis is often large, floppy and difficult to control.

The vocal cords are often very anterior and superior in their placement in the airway making a view of the cords more challenging that with the older child or adult. The larger head in relation to shoulders may make views difficult to attain. Tracheal cartilage in the infant is very soft, and hyper-extension of the airway often leads to closure of the air space, hyper-flexion can also close the airway.

Anatomical anomalies may be present without warning in the neonate as the airway will never have been assessed or investigated for anomalies prior to your intervention.

Despite the reported difficulties that can be anticipated with the neonatal airway, there are a number of methods for mitigating against the challenges. These will be discussed as the different interventions are described.

The important point here is that **good planning and careful consideration** are vital before rushing into the management of the neonate who needs an advanced airway inserted.
When should an advanced airway be considered?
What are the options?

There are a few options for the management of the airway in the neonate

**Laryngeal Mask Airway (LMA)**

An LMA is an option in the neonate, provided they are more than 34 weeks gestation and weigh more than 2kg (AHA, 2017)

An LMA is a device that is inserted blindly, it does not require any equipment for the insertion, nor any special skill (such as use of a laryngoscope). Once the mask is inserted and the cuff is inflated, the opening of the LMA covers the opening of the airway, and air can be directed to the trachea much more directly than with BVM ventilation. In preterm
infants, LMA’s have not been tested and as such they are **not recommended for use in the infant less than 34 weeks gestation or less than 2 kg in weight.**

The LMA is not an airway protection device, though it does offer more direct ventilation of the trachea with a possible decrease in the amount of air that enters the stomach, it is not able to prevent aspiration as it does not seal off the airway completely.

**An LMA may be considered a viable alternative to endotracheal intubation in the resuscitation of the neonate, if face-mask ventilation is ineffective in achieving adequate ventilation.**

There are a number of options for devices that fall under the LMA banner, there are some diagrams illustrating these below:

**Traditional LMA**

I-Gel


LMA Sizing

<table>
<thead>
<tr>
<th>LMA Size</th>
<th>Patient Size</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Neonate / Infants &lt; 5 kg</td>
</tr>
<tr>
<td>1 ½</td>
<td>Infants 5-10 kg</td>
</tr>
<tr>
<td>2</td>
<td>Infants / Children 10-20 kg</td>
</tr>
<tr>
<td>2 ½</td>
<td>Children 20-30 kg</td>
</tr>
<tr>
<td>3</td>
<td>Children/Small adults 30-50 kg</td>
</tr>
<tr>
<td>4</td>
<td>Adults 50-70 kg</td>
</tr>
<tr>
<td>5</td>
<td>Large adult &gt;70 kg</td>
</tr>
</tbody>
</table>

Reference: http://slideplayer.com/slide/9129463/
The use of an LMA is recommended when the use of an endotracheal tube (ETT) is not possible or not successful, it may be considered first line if the ETT is not feasible (AHA, 2017).

### Pro’s and Con’s of the LMA

<table>
<thead>
<tr>
<th>Pro</th>
<th>Con</th>
</tr>
</thead>
<tbody>
<tr>
<td>Easy to insert, requires little training</td>
<td>May not achieve the tight seal needed for high pressure ventilation</td>
</tr>
<tr>
<td>Can be used to assist with access to ventilation when ETT is impossible or not easily achieved</td>
<td>There is not much evidence for use of the LMA during compressions, but if ETT is not successful its makes sense to try it</td>
</tr>
<tr>
<td>May improve effectiveness of BVM ventilation</td>
<td>Cant be used in very small infants (no size small enough is less than 2000g)</td>
</tr>
</tbody>
</table>

**LMA insertion**

The insertion of the LMA should follow a similar approach as the ETT insertion (referenced below) in the patient who is not being actively resuscitated. During resuscitation, the insertion is simplified by the fact that preoxygenation and use of medications for the insertion is not recommended (in this case it is being done as an emergency procedure).

**ETT Insertion**

**What equipment will I need?**

Refer to the checklist below for intubation equipment preparation:
<table>
<thead>
<tr>
<th>Equipment Call</th>
<th>Response Check</th>
</tr>
</thead>
<tbody>
<tr>
<td>Laryngoscope with working batteries</td>
<td>Present and working</td>
</tr>
<tr>
<td>Blade (appropriate size for the infant, 0, 00 or 000) straight</td>
<td>Present and working</td>
</tr>
<tr>
<td>ETT Tube (correct size (2 – 3.5) depending on gestational age and weight measurement) Present and checked</td>
<td></td>
</tr>
<tr>
<td>Stylet (appropriate size for the ETT selected)</td>
<td>Inserted into ETT and lubricated</td>
</tr>
<tr>
<td>End-tidal CO2 detector</td>
<td>Plugged in and reading</td>
</tr>
<tr>
<td>Suction with tubing and soft tip catheters (correct size) Pressure checked less than 100mmHg</td>
<td>Working and connected</td>
</tr>
<tr>
<td>Oral Airway (correct size for infant)</td>
<td>Present and measured</td>
</tr>
<tr>
<td>Tape to secure ETT</td>
<td>Present and prepared</td>
</tr>
<tr>
<td>Device to deliver PPV (BVM/self-inflating bag or neo-puff) Pulse oximeter probe connected to patient and reading LMA/alternate device (appropriate size for infant)</td>
<td>Present, connected to oxygen and functional Connected and reading of ..................... Present and lubricated/checked</td>
</tr>
<tr>
<td>Meconium aspirator present</td>
<td>Present and ready</td>
</tr>
<tr>
<td>Stethoscope</td>
<td>Present and ready</td>
</tr>
<tr>
<td>Scissors (to cut tape for securing tube)</td>
<td>Present</td>
</tr>
</tbody>
</table>
Cuffed or uncuffed ETT?

At present there is no evidence for the use of cuffed endotracheal tubes in neonates, the 2015 update recommends the use of uncuffed tubes for the premature and full-term neonate.

Sizing the ETT?

The recommended size for ETT insertion is weight based, as there are not a lot of sizes (and very little variability in the actual size from 3.5 to size 2), there are not many options for the optimal size of tube.

The following table is a guide only and each patient should be assessed individually for the correct size tube.

<table>
<thead>
<tr>
<th>Weight</th>
<th>Gestational age</th>
<th>Tube size</th>
<th>Suction Tube Size (to fit in tube)</th>
</tr>
</thead>
<tbody>
<tr>
<td>&lt;1000g</td>
<td>Below 28 weeks</td>
<td>2.5</td>
<td>5F or 6F</td>
</tr>
<tr>
<td>1000–2000g</td>
<td>28–34 weeks</td>
<td>3.0</td>
<td>6F or 8F</td>
</tr>
<tr>
<td>2000g–3000g</td>
<td>34–38 weeks</td>
<td>3.5</td>
<td>8F</td>
</tr>
<tr>
<td>&gt;3000g</td>
<td>Above 38 weeks</td>
<td>3.0–4.0</td>
<td>8F or 10F</td>
</tr>
</tbody>
</table>

It must be remembered that the smaller the tube diameter, the higher the pressure required for ventilation. This is as a result of the resistance to flow that a smaller tube offers. The biggest ETT that can fit should be placed.

The tube can be cut shorter prior to intubation to make it easier to handle during the intubation attempt. This can be achieved by cutting the tube diagonally to ensure that the connector will still be able to slide into the top of the tube, it should fit snugly so as not to dislodge during ventilation.
Important notes if using a stylet (introducer)... and you should probably use a stylet/bougie

The stylet must be able to move easily through the tube without too much resistance. The stylet should be inserted in such a way that it does not protrude through the end of the tube (this can create damage to the airway tissues and may force separation of tissues to create a false tract), and should be secured to prevent slipping out of the airway during intubation.

Suction Pressure

The wall suction or portable suction should be prepared so as to prevent creation of a suction pressure of more than 100mmHg. The neonate has a very small lung volume and is very compliant, this means too high a pressure applied to the airway can lead to collapse of the terminal air passages and atelectasis.

Positioning the patient for airway access

The infant has a relatively large head when compared to his shoulders and neck, this means that in order to best align the airway axes (the planes of tissue that make up the airway), the patient must be placed onto a towel or something that can assist with elevating the shoulders off the bed or table surface, this brings the ear to sternal notch as illustrated below and makes opening the airway for visualisation and air passage easier. This may assist with getting the patient into “sniffing position”.

The procedure of intubation

Please see the skill sheet below. This is the skill checklist you will be required to pass for course completion requirements.
Neonatal Intubation

The MOST important thing here is ALVEOLAR VENTILATION not insertion of the ETT.
Don’t allow ventilation and oxygenation to become secondary to the insertion of the ETT. Intubation does NOT mean that adequate oxygenation and ventilation is in place, all it translates to is airway protection.

Selecting Medication for sedation (for intubation)

If the patient is being intubated in the setting of active resuscitation (the patient is obtunded most likely due to hypoxia and there is ongoing CPR), there will likely be no need for sedation for the procedure of intubation.

If the patient is being intubated and is not being actively resuscitated, it is very important to consider use of analgesia and sedation for the purposes of intubating the infant.

More information can be found at the following link:
https://emguidance.com/guidelines
Neonatal endotracheal (et) intubation – sedation

Confirming ETT/LMA Placement

There are numerous subjective measures for the checking of ETT placement in the trachea. These measures are not all required to be used but can be used in conjunction with other measures to determine if the ETT in the correct place. The best method for confirming ETT placement is the objectives measures. These are listed and explained below:

**Objective methods:**

**Seeing the ETT pass through the cords (not relevant for LMA)**

- This is a subjective measure of confirmation as the only person who can witness this is the intubator, it remains nevertheless as one method for confirmation of the tube being in the right place (at least for the immediate post intubation period)

**End-Tidal CO2 and waveform monitoring**

- End tidal CO2 monitoring is an effective method for the confirmation of ETT placement in infants and premature (or low birth weight infants) (2017, AHA).
- A colimetric device may be used for the confirmation of tube placement (this device uses litmus paper to detect pH changes in the air escaping the lungs, if CO2 is dissolved in this air, the paper will change colour from purple to yellow on expiration and back to purple on inspiration.
- A positive result in a patient with perfusion that is intact is a good measure of the ETT being present in the trachea, while a negative result is a good indicator that the ETT is inserted into the oesophagus.
- Waveform capnography with a qualitative and quantitative component is best for determining the correct ETT placement, but colorimetric devices are an acceptable alternative for confirmation of ETT placement (provided the correct size device is used for the neonate)

Reference:
http://www.paramedicine.com/pmc/End_Tidal_CO2.html
Limitations for end-tidal CO₂

It is important to note that infants (or any patient) with poor perfusion will not produce a reading of CO₂ on exhalation that is “normal”, much lower levels than normal are expected, and this ETCO₂ could be measured with CPR ongoing to check that the reading is consistent. This factor should then be taken into consideration with other factors (chest rise, improving vital signs, SPO₂ increasing and colour change.

Possible reasons for incorrect readings:

The tube is in, but the colour doesn’t change:

- Could be that the patient has perfusion that is too low to allow CO₂ to be brought to the lung surface (low heart rate/low blood pressure)
- Not enough tidal volume for ventilation
- Collapsed lungs

The tube is not in, but the colour does change:

- Defective colour-metric device (was contaminated before placed on the patient)

Chest wall movement

- This measure on ventilation through an ETT is objective (meaning that is can be seen by more than one person and confirmed with many people at once)
- Good chest wall movement with ventilation is a good indicator that the ETT is in the trachea

Air entry into all lung fields

- This measure is a good indicator of the ETT being present in the trachea, if on auscultation, air entry can be heard on ventilation in lung spaces, and no sounds can be heard over the stomach (no borborygmi)
- A small stethoscope should be used to ensure that the sound captured is isolated from the area under the stethoscope and not captured from other areas if the bell were very large.
If you have any doubt that the ETT is in the oesophagus, **TAKE THE TUBE OUT** and BVM ventilate the patient until the patient is oxygenated again before attempting the intubation again.

*What if I can’t get the ETT into the airway?*

The endpoint here is NOT an ETT in the airway! The endpoint is oxygenation, ventilation and improving perfusion, the ETT is ONE method for improving access to oxygenation and ventilation. It is not the only one!

Consider using any of the “bailout” options, the aim here is ALVEOLAR VENTILATION to ensure that oxygen can get to the terminal lung surface for exchange, it doesn’t matter how that is achieved.

Refer to the Vortex approach in the link below for options on how to achieve this: http://vortexapproach.org/lifelines/#sga
THE VORTEX

FOR EACH LIFELINE CONSIDER:

- MANIPULATIONS:
  - HEAD & NECK
  - LARYNX
  - DEVICE

- ADJUNCTS

- SIZE / TYPE

- SUCTION / O₂ FLOW

- MUSCLE TONE

MAXIMUM THREE ATTEMPTS AT EACH LIFELINE (UNLESS GAMECHANGER)
AT LEAST ONE ATTEMPT SHOULD BE BY MOST EXPERIENCED CLINICIAN
CICU STATUS ESCALATES WITH UNSUCCESSFUL BEST EFFORT AT ANY LIFELINE

Reference: http://vortexapproach.org/
Some possible complications of ETT insertion

<table>
<thead>
<tr>
<th>Hypoxia</th>
<th>Severe Trauma</th>
</tr>
</thead>
<tbody>
<tr>
<td>• Intubation attempt too long</td>
<td>• Perforation trachea or oesophagus</td>
</tr>
<tr>
<td>• ETT not placed in trachea</td>
<td>• Stylet went too far past tube end</td>
</tr>
<tr>
<td></td>
<td>• Rough insertion of ETT</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Bradycardia</th>
<th>Obstruction</th>
</tr>
</thead>
<tbody>
<tr>
<td>• Hypoxia as above</td>
<td>• Mucous, meconium, blood</td>
</tr>
<tr>
<td>• Vagal stimulation through</td>
<td>• Small tube has kinked</td>
</tr>
<tr>
<td>intubation attempt</td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Trauma (mouth)</th>
<th>Ventilation Trauma</th>
</tr>
</thead>
<tbody>
<tr>
<td>• Trauma to the airway through multiple attempts at ETT insertion</td>
<td>• Pneumothorax from too much volume/pressure</td>
</tr>
<tr>
<td>• Rough laryngoscope handling</td>
<td>• Right main bronchus intubation = increased pressure</td>
</tr>
<tr>
<td>• Blade too large or small</td>
<td></td>
</tr>
</tbody>
</table>

Later:

<table>
<thead>
<tr>
<th>Infection</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>• Bacteria enters infant via ETT/laryngoscope/trauma</td>
<td></td>
</tr>
</tbody>
</table>

DOPES Mnemonic for troubleshooting the advanced airway that is giving problems

**D**islodged

**O**bstructed

**P**nuemothorax (think tension)

**E**quipment

**S**tomach/stacking

Adapted from: Kattwinkel, J. 2013. Neonatal Resuscitation. 6th Ed. AAP. Page 187

IV access, medication and fluid administration

“Asphyxia is an impairment of gas exchange that results not only in the deficit of oxygen in blood but also an excess of carbon dioxide causing acidosis.” (NNF Teaching aids, ND)

Effective positive pressure ventilation is usually successful to return the neonate to a better condition. In the unlikely event that the patient infant does not respond to ventilation and compressions, medications and possibly IV fluid should be initiated.

When a new-born is hypoxic, there is often decreased perfusion as a result of poor cardiac contractility. As the new-born who is systemically hypoxic is often also hypercarbic and acidotic, there is likely a drop in the perfusion status (due to the acidosis and hypoxia).

Maintenance of cerebral perfusion pressure in the infant with a possible ischemic injury is vital (auto regulation of cerebral perfusion can be lost with infants who have ischemic brain injury), in the absence of auto-regulation.

The brain is not well perfused, and the risk of further injury to the brain is increased. For this reason, it is recommended that a MAP of a systemic mean arterial BP of at least 45 to 50mmHg is maintained for infants who are born at term, while a MAP of around 35-40 mm for smaller pre-term infants (1000-2000g) be maintained, with 30-35 mm for micro-premmies (<1000g) be maintained (NNF Teaching Aids, ND).

<table>
<thead>
<tr>
<th>Weight at delivery</th>
<th>Acceptable MAP Guide</th>
</tr>
</thead>
<tbody>
<tr>
<td>Full term (normal weight &gt;2000g)</td>
<td>45-55mmHg</td>
</tr>
<tr>
<td>Premature neonate (1000 – 2000g)</td>
<td>35 – 45mmHg</td>
</tr>
<tr>
<td>Micro-premature neonate (&lt;1000g)</td>
<td>30 – 35mmHg</td>
</tr>
</tbody>
</table>
Normal expected blood pressure by gestational age

<table>
<thead>
<tr>
<th>Post-conceptional age</th>
<th>50th percentile</th>
<th>95th percentile</th>
<th>99th percentile</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>44wks</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>SBP</td>
<td>88</td>
<td>105</td>
<td>110</td>
</tr>
<tr>
<td>DBP</td>
<td>50</td>
<td>68</td>
<td>73</td>
</tr>
<tr>
<td>MAP</td>
<td>63</td>
<td>80</td>
<td>85</td>
</tr>
<tr>
<td><strong>42wks</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>SBP</td>
<td>85</td>
<td>98</td>
<td>102</td>
</tr>
<tr>
<td>DBP</td>
<td>50</td>
<td>65</td>
<td>70</td>
</tr>
<tr>
<td>MAP</td>
<td>62</td>
<td>76</td>
<td>81</td>
</tr>
<tr>
<td><strong>40wks</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>SBP</td>
<td>80</td>
<td>95</td>
<td>100</td>
</tr>
<tr>
<td>DBP</td>
<td>50</td>
<td>65</td>
<td>70</td>
</tr>
<tr>
<td>MAP</td>
<td>60</td>
<td>75</td>
<td>80</td>
</tr>
<tr>
<td><strong>38wks</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>SBP</td>
<td>77</td>
<td>92</td>
<td>100</td>
</tr>
<tr>
<td>DBP</td>
<td>50</td>
<td>65</td>
<td>70</td>
</tr>
<tr>
<td>MAP</td>
<td>60</td>
<td>75</td>
<td>80</td>
</tr>
<tr>
<td><strong>36wks</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>SBP</td>
<td>72</td>
<td>87</td>
<td>92</td>
</tr>
<tr>
<td>DBP</td>
<td>50</td>
<td>65</td>
<td>70</td>
</tr>
<tr>
<td>MAP</td>
<td>59</td>
<td>72</td>
<td>71</td>
</tr>
<tr>
<td><strong>34wks</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>SBP</td>
<td>70</td>
<td>85</td>
<td>90</td>
</tr>
<tr>
<td>DBP</td>
<td>40</td>
<td>55</td>
<td>60</td>
</tr>
<tr>
<td>MAP</td>
<td>50</td>
<td>65</td>
<td>70</td>
</tr>
<tr>
<td><strong>32wks</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>SBP</td>
<td>68</td>
<td>83</td>
<td>88</td>
</tr>
<tr>
<td>DBP</td>
<td>40</td>
<td>55</td>
<td>60</td>
</tr>
<tr>
<td>MAP</td>
<td>48</td>
<td>62</td>
<td>69</td>
</tr>
</tbody>
</table>
IV access in the new-born can be a challenge, due to the size of the vessels in the peripheries and the size of the peripheries themselves, gaining access to the venous system of the neonate is difficult.

The first step in medication/fluid administration will be access to the venous system. There are a few options:

1. **Umbilical venous access**
2. Intraosseous access
3. Peripheral venous access

### How to gain IV access in the neonate?

IV access should only be considered when there are more rescuers available to assist with the resuscitation, as it required at least two rescuers to perform good compressions and PPV (positive pressure ventilation). Compression and oxygenation should not be at the expense of IV access.

The easiest method for intravenous access in the freshly born neonate is the umbilical vein, in the setting of neonatal resuscitation this is not a sterile procedure but should be performed as cleanly as possible.

<table>
<thead>
<tr>
<th></th>
<th>30wks</th>
<th></th>
<th>28wks</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>SBP</td>
<td>DBP</td>
<td>MAP</td>
<td>SBP</td>
</tr>
<tr>
<td>30wks</td>
<td>65</td>
<td>40</td>
<td>48</td>
<td>60</td>
</tr>
<tr>
<td>28wks</td>
<td>80</td>
<td>55</td>
<td>65</td>
<td>75</td>
</tr>
<tr>
<td>26wks</td>
<td>85</td>
<td>60</td>
<td>68</td>
<td>60</td>
</tr>
<tr>
<td></td>
<td>55</td>
<td>30</td>
<td>38</td>
<td>72</td>
</tr>
<tr>
<td></td>
<td>72</td>
<td>50</td>
<td>57</td>
<td>77</td>
</tr>
</tbody>
</table>

The umbilical vein may be accessed from delivery of the infant for up to 1 week post-delivery. Once the infant is delivered the umbilical artery spasms shut, but the umbilical vein remains open for a long time (NNF Teaching aids, ND)

**Umbilical vein cannulation**

Important anatomy reminder: the umbilical cord usual has three vessels, two arteries and one vein. The vein is recognizable by the thinner walled vessel, the fact that it usually does not spasm post-delivery and that it is much larger than the other two vessels in the cord.

The umbilical veins may be accessed for up to 7 days post-delivery (Bengton-Bok, 2013).

Infants with only one artery are at higher risk for other congenital anomalies and should be closely investigated for these after initial resuscitation (bear in mind that this could be why the infant requires resuscitation)

Reference: https://first10em.com/umbilical-vein-catheterization/
The procedure for the insertion of the umbilical vein cannula can be found at this link:

For more information on UVC, the following site provides a good summary:
https://first10em.com/umbilical-vein-catheterization/

**Intraosseous Cannulation**

Intraosseous access is a reasonable approach for IV access in the infant who presents in the out of hospital/ED environment as access to UVC access may not be possible (due to closure of the umbilical vein), or because of decreased access to UVC equipment. If in the delivery room or with the newly delivered infant, the umbilical vein is the best option.
Medications for the resuscitation of the neonate

Adrenalin

Fluid

Naloxone

**Adrenaline**

Adrenaline, if given to the neonate in need of resuscitation, should be administered via the IV line if possible. According to the 2015 guideline update for neonatal resuscitation: there is no supportive data for the use of endotracheal adrenalin, the IV route should be used as soon as venous access is established (Class IIb, LOE C), and the ETT route should be avoided if possible (Open Anaesthesia, 2018).

Adrenaline should be administered in a concentration of 1:10000 (100ug/ml) (Katwinkel et al. 2015), and the neonate should receive a dose of adrenalin no greater than 0.01 – 0.03mg/kg per dose.

Higher doses show no benefit and may show harm in both paediatric and animal studies (hypertension, decreased function of the myocardium, worse neurological function).

Adrenalin (and any medication given in arrest) should be followed by a flush of normal saline to ensure that the medication gets through the admin set and into the vessel. This flush should be small enough not to put the infant at risk of fluid overload with multiple medication doses (recommended flush 0.5-1ml)
Adrenalin can be repeated every 3-5 minutes only if the heart rate does not increase above 60b/min.

**How do I dilute this again?**

In South Africa we get adrenalin in a 1:1000 solution, this means that to have 1 g of adrenalin we would need 1000amps of the preparation.

We need to administer adrenalin at a concentration of 1:10 000 for infants, this means we need to make it 10 times less concentrated.

**WAIT! What’s the dose?**

0.01mg/kg, or 0.1ml/kg of a 1:10 000 solution (this can also be given to smaller infants at 1ml/kg of 1: 100 00 solution)

We do this by adding 9ml of diluent to the 1ml (1mg) of adrenalin, we take the solution from 1mg/ml to 1mg/10ml or 0.1mg/ml (also reported as 100ug/ml) or 1: 10 000 solution.

<table>
<thead>
<tr>
<th>Concentration</th>
<th>Volume</th>
<th>Mg/ml</th>
</tr>
</thead>
<tbody>
<tr>
<td>1:1000</td>
<td>1ml</td>
<td>1mg/ml</td>
</tr>
<tr>
<td>1:10 000</td>
<td>10ml</td>
<td>0,1mg/ml</td>
</tr>
<tr>
<td>1:100 000</td>
<td>100ml</td>
<td>0,01mg/ml</td>
</tr>
</tbody>
</table>
Why adrenalin?

It is thought that adrenalin plays a role in

- increased contractility of the myocardium
- increased peripheral vasoconstriction and thus improved venous return
- increased flow of oxygenated blood to the brain and other vital organs

Adrenalin should NEVER be used if there has not been effort to increase oxygenation and ventilation as these are the possible reasons for poor perfusion in the neonate.

Adrenalin also increases the workload of the heart through increased heart rate and force of contraction, this means the delivery of oxygen must be corrected before adrenalin can be used (supply must match demand).
The heart rate should be checked every minute after adrenalin administration to determine if there has been an effect.

**Dextrose**

Blood Sugar monitoring should be started in any infant who has been resuscitated (Weiner, G. 2016)

The sick infant is at higher risk of hypoglycaemia as they are likely unable to ingest nutrients, they may have increased metabolic demand and are unable to alert anyone that they need assistance. Glucose regulatory mechanisms are sluggish at first, this puts the infant at higher risk of hypoglycaemia after delivery. Other sources of energy (ketones/lactate) are not produced in large enough quantities to be used as a primary fuel source, the infant relies heavily on glucose as their source of fuel. There are some neonates at higher risk of hypoglycaemia than others:

- Small for gestational age infants or preterm infants
- Large infants (macrosomic infants born at > 4.5kg)
- Infant of diabetic mothers
- Infants with CNS depression, respiratory distress or septic at birth
- Infants who are nil per mouth (surgical concerns or unable to feed)

Signs of hypoglycaemia are very vague in this population; a summary of findings can be found below:
During resuscitation if the blood glucose level is less than 2.6mmol/l, the recommendation is that dextrose is administered IV to increase the glucose level rapidly and reverse hypoglycaemia as a potential cause of the condition.

The following is an example of how blood glucose should be approached for management in the neonate:

*Bottom Line: IV glucose infusions should be started if needed as soon as practical after birth, with the goal of avoiding hypoglycaemia (AHA, 2017).*
If BSL remains < 2.6mmol/L

- Increase Dextrose concentration to 12.5% then 15% as appropriate
- Central line required for > 12.5% Dextrose

If BSL remains < 2.6mmol/L

- Glucagon bolus IV 0.02mg/kg
- Commence glucagon infusion if hypoglycaemia continues

NB: if hypoglycaemia persists, consider seeking an endocrinology consultation.

Reference: https://www.rch.org.au/rchcpg/hospital_clinical_guideline_index/Neonatal_hypoglycaemia/
**Naloxone**

Although naloxone has long been used in the case of opioid exposure in the neonate, there is not enough evidence to make any recommendation with regard to the administration of this medication to the neonate.

The recommendation at this time is to reverse **HYPOXIA** with good BVM ventilation, and to consider intubation or LMA insertion if the period of decreased respiratory effort is noted to last for a longer time period. The infant should be ventilated until the effects are no longer evident.

There are possible complications in the neonatal population that can be linked to the administration of naloxone (these include seizures, pulmonary oedema and cardiac arrest) (Weiner, G. 2016)

**What about Sodium Bicarbonate?**

The following article sums up the use of bicarbonate in neonatal acidosis:


  **Bottom Line:** Sodium Bicarbonate is not the magic medication for acidosis we thought it was, it should NOT be used routinely in neonatal resuscitation and if used at all should be used very carefully with consultation.

**Fluid Administration**

VOLUME replacement is NOT for routine use in resuscitation of the neonate UNLESS there is a clear indication.

If there has been no response to the resuscitation of the patient after escalation through the various levels of care, or if the patient has a history of blood loss (placenta previa or blood loss from the umbilical cord prior to care) or fluid loss (dehydration, poor feeding,
vomiting, loose stools or signs of neglect), the patient should be considered for volume replacement.

If the patient has lost blood, blood should be considered as replacement fluid, if the patient has lost volume due to non-hemorrhagic shock, then fluid should be started.

Fluid bolus’ in small aliquots of 10ml/kg should be started if there is a clear indication and can be repeated up to 3 times if there is no response to the fluid and the indication still persists.

Packed red blood cells can be replaced if there is severe anaemia or blood loss present, up to volumes of 20ml/kg in small aliquots of 5-10ml/kg.

**FLUID SHOULD BE ADMINISTERED AT A RATE OF 10ML/KG OVER 5-10 MINUTES**

Fluid given too rapidly can result in damage to the fragile vessels of the brain and leakage of fluid into the interstitial space leading to increased intracranial pressure due to intraventricular haemorrhage (a greater risk in premature infants than in term infants).

**What fluid should be used?**

- Normal saline (0.9%)
- Ringers Lactate
- Packed RBC (emergency O RH Negative or cross-matched) for patients with blood loss or a history consistent with blood loss (Katwinkel, 2015)
References:
Bengton-Bok, E. 2013. Central Lines in Pre-term new-born infants. [available online] https://www.duo.uio.no/bitstream/handle/10852/41170/Till-DUO-Central-Lines-in-Preterm-Newborn-Infants.pdf?sequence=1


The infant with cyanosis/respiratory distress

The infant who presents with cyanosis needs to be assessed for life threats immediately and treated for those life threats as soon as possible.

This chapter will explore the possible causes of cyanosis in the infant and provide a plan for the assessment and differential diagnosis of the most common possible causes. The focus in this chapter will not be on ALL the possible causes of cyanosis, rather on those that may be related to severe illness/injury. The patient must be assessed rapidly, and supportive management started early to maintain oxygenation and perfusion for the cyanosed infant.

Some important concepts

Read over the chapter on the changes that occur at delivery of the infant (life on the outside), especially with regard to the change in circulatory and respiratory systems. These changes occur unhindered and without much assistance in the “normal” infant.

Cyanosis

This is defined as a blue discolouration of the skin as a result of increased percentage of deoxygenated haemoglobin present in the circulatory system.

Cyanosis can be found in central and peripheral tissues, if the cyanosis is only found in the peripheries, it is referred to as “acrocyanosis” this is a common finding in many infants in the first few hours post-delivery, and does not usually represent a life threat.
Notice in the image above, the infant has a pink central areas (lips and mucous membranes), with blue peripheries. This is a “normal” finding and does not mean there is a systemic problem, unless found with other signs of respiratory distress or cardiovascular failure.

Central cyanosis present on the mucous membranes and tongue of the infant is a far more ominous sign and indicates a systemic issue. Central cyanosis should be investigated and taken seriously as it represents a possible life threat.

There are 4 systems that can be responsible for cyanosis in an infant, these can be best described as:

- **The respiratory system**
  - The upper airway
  - The lower airway

- **The cardiovascular system**
  - The blood itself
  - The pump
The infant with cyanosis/respiratory distress

- The nervous system
  - Control of the respiratory system
- Other things that can result in cyanosis

The infant presenting with respiratory distress is very similar to the infant presenting with cyanosis, except that the differential diagnosis for distress alone might point the treating practitioner in different directions to the infant with cyanosis.

The diagram below shows the **signs of respiratory distress**

[Diagram of respiratory distress signs including Head bobbing, Increased rate (RR and HR), Grunting, Abdominal muscle use, Nasal Flaring, Cyanosis, Chest wall retraction, Sweating]
There are a number of possible causes of respiratory distress in the neonate, the diagram below leads the practitioner to one of several possible differential diagnoses.

1. Upper Airway problems

Infants who present with upper airway problems usually present early in life and can usually be recognised by external physical findings.

**Choanal Atresia**

This is a condition where the back of the nasal bone does not separate, the nasal bone is intact and there is no pathway from nose to the mouth. It is fairly common occurring 1:5000 infants. It usually happens in only one nostril. It can be diagnosed by the following clues: the
The infant with cyanosis/respiratory distress |

infant does better when crying than when quiet (as when the infant cries it breathes through its mouth), there is no way to pass a suction catheter through the nose, the infant’s nose runs as the mucous can only run out.

Reference:
https://www.aboutkidshealth.ca/Article?contentid=1029&language=English

Management?

Placement of an oropharyngeal airway often works to relieve cyanosis, as it opens up the airway and relieves some of the respiratory distress, long-term the infant will need surgery to correct the blockage.

Other anomalies should be hunted as the child with choanal atresia is high risk for other congenital malformations of the heart, ear/hearing, genitourinary system, stunted growth and development and eye)
**Pierre-Robin Syndrome**

This is a set of anomalies including micrognathia (small lower jaw), retrognathia (a lower jaw that is set further back than usual), glossoptosis (a tongue that is much further back than normal), and obstruction of the airway. Cleft palate is a common finding with this syndrome.


This syndrome is usually obvious on initial presentation of the infant as the physical signs are clear, it is normally noted immediately following birth. The patient has trouble breathing in the supine position as the posterior tongue and small mandible allow for occlusion of the upper airway, when the patient is placed prone, the respiratory distress/cyanosis often improves.

Reference: https://www.rch.org.au/kidsinfo/fact_sheets/Pierre_Robin_Sequence_PRS/
Management?

Placing the patient prone can relieve a lot of distress in the short-term, placement of an OPA may assist. These patients may need to have a tracheostomy for an extended period of time to maintain a patent airway until the mandible can grow to accommodate space for the airway.

Laryngomalacia

This condition is defined as collapse of supraglottic structures during inspiration. This is a congenital abnormality in the formation of the larynx and is a common cause of stridor in the infant. This is often recognised at birth, but it is possible that it is only noted a few weeks after birth. These patients present with difficulty breathing especially during feeding or crying, and if they have respiratory infections. Reflux is a common finding with these patients. The tissues are soft and floppy and are not properly formed, leading to difficulty in breathing, and often noisy breathing.

Management?

Most infants (reportedly up to 90% of infants) tolerate this well and will eventually outgrow the problem and noisy breathing by the age of 2. There are some infants who present with severe respiratory distress, increased work of breathing and stridor. For these children the management in the emergency setting is supportive and may include intubation.
**Vocal cord paralysis**

This condition can be as a result of birth trauma or surgical trauma. It is a common reason for cyanosis in the new-born. Usually only one side of the cords is paralysed, but this could affect both sides.

In the case of bilateral paralysis, the infant may need insertion of a tracheostomy, and other possible causes (such as congenital diseases) should be considered. The paralysed vocal cord can be seen in the first few minutes of the video below:

Reference: https://www.youtube.com/watch?v=pcTPOmNSIPI
**Haemangiomas**

A haemangioma is a vascular lesion that is fairly common (present in around 10% of the population). It is a malformation of capillaries and smaller vessels. They usually present initially as a small area of reddening, and then enter a growth phase, usually growing the most in the first few months.

These growths usually don’t cause too much threat to life and are only a threat to the child’s looks. However, if the growth occurs in the airway, this can threaten life.

**Management?**

Initial management of the infant may include supportive management of the airway and ventilation. Investigation is important to determine the cause of the airway obstruction. Surgery may be required to correct the problem, but in the interim, airway management and protection can be considered depending on where the haemangioma is. Propranolol is fast becoming the agent of choice for the management of these malformations chronically (until the growth shrinks or disappears).

*Large airway haemangioma (subglottic)*

This image shows a severe formation in the airway, this has occluded airflow and thus oxygenation of the infant in the image.
Haemangioma visible in the airway (occluding airflow)

Less severe presentations are more common, the degree of risk increases with size and area of growth.
Breathing Problems

These can be grouped into the following:

Lung Tissue Problems

Congenital Pneumonia

Usually acquired at the time of the delivery, and due to the physiology of the disease, presents with similar findings to RDS (Respiratory Distress Syndrome). Congenital pneumonia typically presents with diffuse infiltrates as opposed to lobar infiltrates (this means that on x-ray and in clinical assessment it may be difficult to diagnose).

Bacterial infection is the most common cause of congenital pneumonia, with the following history findings increasing the likelihood that the condition of the infant may be due to pneumonia: prolonged time from membrane rupture to delivery (more than 18 hours), maternal infection, maternal chlamydial infection.

Infection may present within a few hours of delivery, or up to 1-week post-partum. Depending on the organism causing the infection, the management will change.

Later in this chapter, there is an x-ray comparison of a number of respiratory diseases in the neonate.
**Hyaline Membrane Disease**

This is older term for Infant Respiratory Distress Syndrome (IRDS) or Respiratory Distress Syndrome. This syndrome is caused by insufficient surfactant produced by the infant lungs, and is a common finding when a premature neonate is treated. The infant without surfactant cannot keep the terminal air sacs (alveoli) open, this means they collapse and then with each breath are torn open again, resulting in inflammation, leaky membranes and accumulation of protein rich fluid in the space between the alveoli and the capillary as well as in the alveolar space. This makes gaseous exchange even more challenging and results in poor oxygen delivery and CO2 release.

Management:
Supportive management of the infant’s airway, breathing and circulation are important. Avoiding hypoxia will decrease the effect of pulmonary vasoconstriction. Decreasing work of breathing (WOB) as far as possible using CPAP/assisted ventilation through either non-invasive or invasive methods is vital. Oxygen administration is important but should be considered in conjunction with pressure assisted ventilation. Replacement of surfactant can be considered where it is resumed to be the problem.
Meconium Aspiration Syndrome (MAS)

Meconium aspiration syndrome is a common respiratory problem in neonates, this substance is the first intestinal discharge from the infant, it is a thick greenish/brown substance made of digested amniotic fluid, skin cells, lanugo and foetal urine while in utero. Often the passage of meconium prior to or during the delivery indicates possible distress of the infant. Post-dates infants are also at risk for the passing of meconium. The
mechanical obstruction to the airway is less dangerous in these patients than the longer-term chemical pneumonitis that develops with time.

Below is a picture of what meconium may look like:

IMPORTANT 2015 GUIDELINE UPDATE

Routine suctioning of the infant with meconium aspiration (or suspected aspiration) is no longer recommended, the focus should be on achieving oxygenation and ventilation as soon as possible. Even if the infant is NOT vigorous, the recommendation is to perform the assessment and initial management of the neonate in the same way as for any other infant.
The only time the infant with meconium exposure will be suctioned is if there is an obstruction to air movement on ventilation. There is no evidence that routine suctioning improves outcome, and the delay to first ventilation may be more dangerous than the risk of ventilating an infant who has meconium in the airway.

“The most important step is to achieve effective ventilation to inflate the lung and start the child breathing, because that is what will bring up the heart rate and stabilize the baby. In the 2015 guidelines, the steps for achieving effective ventilation and adequate breathing have not changed” (Wykoff. 2015)

Pathophysiology of harm with meconium aspiration syndrome:

**Pulmonary Oedema**

Pulmonary oedema can occur in the neonate as a result of many different insults to the neonatal physiology. The causes are multiple and complex, and as the treatment of each differs, the cause must be found to fix the problem. Pulmonary oedema is a symptom of another problem and should lead the practitioner on a hunt to determine the possible causes rather than simple symptomatic management.
Below is a short list of common causes of pulmonary oedema, this list is by no means exhaustive:

- perinatal asphyxia
- heart failure
- hyaline membrane disease
- persistent patency of the ductus arteriosus
- pneumonitis
- chronic lung disease (bronchopulmonary dysplasia)
- cardiac anomalies/congenital malformations

Pathophysiology of Pulmonary Oedema
Lower Airway Problems

Congenital Malformations

These are rare causes of respiratory distress in an infant, though possible.

Most dangerous and common is the congenital diaphragmatic hernia, due to the fact that there are a number of dangerous findings that occur with this condition, it is usually picked up as a problem immediately after birth.
Diaphragmatic Hernia

The diaphragm fails to close properly, and the contents of the abdomen can move up into the chest space. This puts undue pressure on the lung on the affected side (which is usually the left and the liver is in the way on the right). This abnormality can result in hypoplasia of the left lung, and after delivery failure to oxygenate and ventilate effectively, as well as pulmonary hypertension as a result of the failure of the lung to expand and perfuse appropriately.
Vital assessment clues:

- Infant gets worse with PPV (positive pressure ventilation), as pressure into the stomach increases distention of the abdominal organs in the chest and thus decreases the expansion of the lungs
- Bowel sounds heard in the chest cavity
- Scaphoid abdomen (or an abdomen that appears “empty”)
- Fuller looking chest
- Respiratory distress, with shallow rapid breathing
- Loops of bowel visible in the chest cavity on xray/ultrasound
Management?

- Early recognition will assist in early management
- Intubation asap and ensure the tube is in the trachea
- Positive pressure ventilation with ETT (avoid BVM ventilation if possible)
- Orogastric tube insertion asap to decompress the stomach and decrease pressure on the thoracic organs by expanding abdominal organs
- Surgical intervention will be required, infant must be taken to the appropriate facility for this to occur asap.

Air where it shouldn’t be

As air in other places is a rare finding (pneumomediastinum, pneumoperitoneum, pneumopericardium) and these will not be discussed at length, although images of these can be found below:

Pneumoperitoneum can be found with necrotising enterocolitis with gastric perforation, or from trauma.
The most common problem when air goes where it shouldn’t is when air ends up in the pleural space. There are a number of reasons this could happen.

**Pneumothorax**

Neonatal lungs are particularly delicate, when large pressures are applied to normal lungs, or even smaller pressures are applied to malformed or injured lungs, small air leaks can easily develop as alveoli burst.

Air that leaks into the pleural space is called a pneumothorax, and if this collection of air gets large enough (or is placed under enough pressure) then the infant will present with severe respiratory distress as the lung can no longer expand, and shock as the vasculature is no longer able to conduct blood through the pressure filled space.
The following findings assist in the diagnosis:

- Decreased or absent air entry on the affected side (this can be challenging in infants as their chests are so small that often sound is transmitted even when there is no air entry on a side)
- Respiratory distress (increased RR, decreased tidal volumes, increased WOB, cyanosis and decreasing SPO2 readings)
- Signs of shock
- Transillumination with a light can show increased light transmission on the affected side (this is not a good sign on its own though if used in conjunction with other clinical signs can assist with the diagnosis)

**Management**

A tension pneumothorax should be managed by relieving pressure of the air in the space by inserting an IV cannula into the space and removing the air that is causing the problem, this can be found in video format on the NERS pressbooks manual.

**Persistent Pulmonary Hypertension**

In short, this condition occurs when the foetal circulation fails to convert to infant circulation for a number of reasons described below. This results in persistent increased pressures on the pulmonary circuit/right side of the heart, and poor perfusion of the alveoli, resulting in a ventilation/perfusion mismatch and shunt. The infant will become acidotic and severely hypoxic as a result. This is another example of a condition that is a symptom of something else.

- RDS: respiratory distress syndrome
- MAS: meconium aspiration syndrome
- TTN: Transient tachypnoea of the new-born
- CDH: Congenital diaphragmatic hernia
- ACDMPV: Alveolar Capillary Dysplasia with misalignment of pulmonary veins
References


The Vomiting Infant

Before we discuss the approach to the infant who is vomiting, we need to understand the possible causes (pathophysiology) of vomiting in the neonate and infant.

Infants are at high risk for fluid loss and dehydration even with seemingly insignificant episodes of vomiting. The job of the clinician is to rule out the possible life threats and rule in possible differential diagnoses for the management of the patient.

Pathophysiology of vomiting

Vomiting is the forceful expulsion of the stomach contents through the mouth after the muscles of the abdomen and chest wall spasm.

Vomiting can be as a result of a number of different things:

- Irritation of the gastric mucosa (toxins, medications, distention)
- Stimulation of the brain through fear or pain
- Stimulation of the chemo-emetic trigger zone (toxins, electrolyte imbalance, other metabolic disturbances or increased intracranial pressure)

Gastrointestinal Tract causes should always come to mind first, but there are many other causes that must be investigated.
Mostly vomiting in the neonate is self-limiting and benign, but ALL vomiting requires a thorough assessment approach to ensure nothing life threatening is missed.
Approach to the assessment of the infant with vomiting

### Primary Assessment

<table>
<thead>
<tr>
<th>Airway</th>
<th>Breathing</th>
<th>Circulation</th>
<th>Disability</th>
<th>Exposure</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ensure airway open, maintained</td>
<td>Rate: normal for infant 40-60b/min</td>
<td>Rate: 110-180 is around normal</td>
<td>HGT</td>
<td>Temperature (hot/cold both</td>
</tr>
<tr>
<td>and protected (use basic to</td>
<td>A/E should be clear on both</td>
<td>AVPU</td>
<td>Pupils</td>
<td>concerning)</td>
</tr>
<tr>
<td>advanced if needed)</td>
<td>sides</td>
<td>Colour: mottling, pale, pallor</td>
<td></td>
<td>Rashes, trauma, toxins</td>
</tr>
<tr>
<td></td>
<td>Effort: Nares, accessory muscle</td>
<td>End organ perfusion: CRT&lt;2sec, wet</td>
<td></td>
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<tr>
<td></td>
<td>use, retractions, tugs, abdominal</td>
<td>nappies in the last 24 hours</td>
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<tr>
<td></td>
<td>muscle, head bobbing.</td>
<td>Systolic BP: reference norms for</td>
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<tr>
<td></td>
<td>SPO2: pre and post ductal sats</td>
<td>age</td>
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<td>NB</td>
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### Secondary Assessment

<table>
<thead>
<tr>
<th>History</th>
<th>Important clinical assessments</th>
<th>Nervous system</th>
<th>Infection</th>
<th>Abdominal</th>
<th>Labs and other assessments</th>
</tr>
</thead>
<tbody>
<tr>
<td>SAMPLE</td>
<td></td>
<td>Focal neurological signs</td>
<td>Typanic membrane</td>
<td>Distention/pain</td>
<td>FBC, U+E (only if indicated)</td>
</tr>
<tr>
<td>More detailed for vomiting:</td>
<td></td>
<td>Cranial nerve assessment</td>
<td>Pharynx</td>
<td>Bruising/rashes</td>
<td>Glucose test</td>
</tr>
<tr>
<td>Amount of vomit</td>
<td></td>
<td></td>
<td>Chest</td>
<td>Auscultation</td>
<td>Urine analysis</td>
</tr>
<tr>
<td>Frequency</td>
<td></td>
<td></td>
<td>Meningism</td>
<td>Liver distention</td>
<td>Stool MC+S if indicated</td>
</tr>
<tr>
<td>Colour (bile or not, bloody?)</td>
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<td></td>
<td>Masses or organ enlargement</td>
<td>Blood cultures</td>
</tr>
<tr>
<td>Consistency</td>
<td></td>
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<td>Abdominal imaging</td>
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<td>Projectile?</td>
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<td>Febile/afebrile current and in</td>
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<td>last few days</td>
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<tr>
<td>Other GI symptoms</td>
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<tr>
<td>(diarrhoea, distention, pain)</td>
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<tr>
<td>Other symptoms</td>
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<td>(thirst/hunger, cough,</td>
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<td>respiratory distress)</td>
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</table>

### Hydration status

- Mucous membranes (moist/dry)
- Fontanelles (sunken/bulging)
- Skin turgor (tenting or normal)
- Oral intake
- Tear production
- Urine production/nappy changes
- Weight changes
Ethics and Difficult Decisions

Ethical dilemmas that present with the neonatal patient provide really challenging environments. Watch the video in this chapter in the electronic book format.

This video and information presented in this video will be discussed in depth in the course you attend.

What principles apply with regard to ethics in medicine?

- **Autonomy**

- **Nonmaleficence**

- **Beneficence**

- **Justice**

These principles above are not in any order, it is important to note that they are all as important as the other, there is no one that “trumps” the other.

Each case will present with its own set of problems that will need to be solved through careful balancing of the principles of medical law. These principles are “empty of fact” meaning that only in the context of the case will the principles begin to carry weight.

**Autonomy:**

The patient has capacity to act intentionally, with understanding, without controlling influences that would mitigate a free and voluntary act. This principle forms the basis of “informed consent” (McCormick, T. et al. 2013).
Having respect for autonomy doesn’t just mean allowing the patient a voice, it means having an approach to the patient that creates the opportunity for the patient to have their voice heard. It means creating the space for the autonomous actions of the patient to be promoted.

Even when the spiritual, religious, loyal, values of the patient are in conflict with the “accepted” path of action, they must be respected. For example, a person who is a Jehovah’s witness will refuse blood even if it means they may die. The patient must be given the information needed to help them make the informed decision for their healthcare, but then the patient’s wishes should be respected. In the case of parents of a child who is a Jehovah’s witness, this becomes a bit trickier as the principle of avoiding death outweighs the parental beliefs.

**Nonmaleficence:**

This principle centres around not harming the patient, whether through acts of commission (something was done), or omission (something was not done). This principle makes the point that practitioners need to be competent and should do whatever is reasonable to avoid harm.
It is accepted that with all medical intervention there will be an element of harm to the patient, we are morally and ethically bound to choose the course of action which will cause the least harm with the most benefit.

The patient is the point of reference for this principle as they patient is the one who will be experiencing the harm through intervention. If the patient is unable to make that decision for themselves, it falls to the surrogate decision makers.

There are four conditions that apply to this principle, that can be used to help make better decisions regarding AVOIDING HARM.

- The nature of the act: the intended action can’t be intrinsically wrong, it must be at the least a neutral act, and at best, a morally correct act.
- The intention of the agent: the intention of the agent must be good, even if the outcome has some harm, the intention should be good.
- Means and effect: the bad effect should not be the way that the good effect occurs
- Proportionality between good and bad effect: The good effect must outweigh the evil that is permitted (the benefit must outweigh the risk)

**Beneficence:**

This principle is the basis for implied consent and takes into account the fact that where possible, the medical provider should take all steps possible to prevent harm to a patient to provide the patient with benefit. Sometimes the actions for the care of the patient are taken without any consent as they are considered to be in the best interests of the patient at the time. Resuscitation fits this principle well, as the patient who needs resuscitation is usually unable to consent to it, but any reasonable person with a possibly reversible pathology would request to be resuscitated.

As with all ethical principles, this requires that it be applied to the situation particular to the patient at the time.
**Justice:**

The best way to understand this principle is to understand the idea of fairness in medical care. There are limited resources available, and these must be as evenly distributed to patients as possible. Scarce resources should be allocated to allow as many patient’s access as possible, and access for those who need it most urgently, first. The organ recipient list is a good example of justice in action.

**Withholding and withdrawing care**

Please refer to the following HPCSA document with regard to the topic above:  

**When should we resuscitate, and when should we not?**

The decision to withhold or withdraw care is difficult no matter who the patient is, it is particularly difficult when faced with an infant either who is unlikely to survive due to numerous factors, or the premature neonate who is at a disadvantage purely from development.

It has been suggested that the following are questions that can assist in better guiding decisions for the infant in care.
There are some gestational age guidelines to help with decision making in neonatal resuscitation. The following information has been taken from the following reference: 

Most extremely premature infants die, regardless of what is done for them in terms of treatment. Those who do survive are usually severely disabled. Extreme prematurity is any delivery below 25 weeks and 6 days gestational age.

The definition “born alive” means that the infant can breathe independently or with the help of a ventilator.
<table>
<thead>
<tr>
<th>Gestational age</th>
<th>Suggested Plan of action</th>
<th>Notes</th>
</tr>
</thead>
<tbody>
<tr>
<td>&gt; 25 weeks and 6 days</td>
<td>It is reasonable to resuscitate all infants of this gestational age provided there is no condition that would make this decision the wrong one</td>
<td>Infants born at this gestational age have good survival rates, and low risks of severe disability. Stats: Of 424 infants born at 25 weeks, 57% had died by age 6, 8% survived with no disability, 12% with mild impairment</td>
</tr>
<tr>
<td>24 weeks – 24 weeks 6 days</td>
<td>Offer full invasive ICU care if possible, UNLESS parents are not willing to consent to resuscitation</td>
<td>Infants born at this gestational age are tricky, they tend to do worse than those born later. Stats: Of 382 infants born at 24 weeks 74% had died at 6 year follow up, and 3% survived with no impairment, while 7% survived with mild impairment</td>
</tr>
<tr>
<td>23 weeks – 23 weeks 5 days</td>
<td>Parents should be given all the information and their decision regarding resuscitation taken into account, providers are not obligated to resuscitate</td>
<td>Survival with good outcome is not likely Stats: Of 241 infants born at 23 weeks 90% had died by age 6. Only 1% of those who survived had no impairment, 2% survived</td>
</tr>
</tbody>
</table>


Some important things to think about

- Intolerability: refers to situations which would not be in the best interests of the infant, where the life-sustaining care would make life intolerable for the infant. This must be considered for EACH intervention, and decision.
- Best interests of the infant in front of you: this is a vital point, the best interests of the infant are your concern, the parents/ surrogate caregivers wishes come second to the infant.
  - Quality of life is important
  - Life/death must be taken into account
- Withdrawal or withholding care are both acceptable, as long as the best interests of the infant are taken into account. Ending of life (active euthanasia) is not acceptable.
potentially life-shortening medications may be given to relieve pain and distress if needed.

### Some tools for the end

The following article provides a good summary of the care of the family and child at the end of life in the emergency setting:

http://pediatrics.aappublications.org/content/134/1/e313

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<table>
<thead>
<tr>
<th>Essential components of care in the ED when a child dies</th>
</tr>
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<tbody>
<tr>
<td><strong>Clinical</strong></td>
</tr>
<tr>
<td>- Best practice resuscitation</td>
</tr>
<tr>
<td>- Termination of resuscitation</td>
</tr>
<tr>
<td>- Advanced care directive?</td>
</tr>
<tr>
<td>- Death pause</td>
</tr>
<tr>
<td><strong>Operational</strong></td>
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<tr>
<td>- Staff training in communication</td>
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<tr>
<td>- Team work and response (include security, support staff, social work, administration staff)</td>
</tr>
<tr>
<td>- Family presence during resus?</td>
</tr>
<tr>
<td>- Media (if required), and media policy</td>
</tr>
<tr>
<td>- Team clinical and emotional brief</td>
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<tr>
<td>- Room for family (comfort room)</td>
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<tr>
<td><strong>Ethical</strong></td>
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<tr>
<td>- Resuscitation length?</td>
</tr>
<tr>
<td>- Prolonged resuscitation criteria? (Organ donation, family presence)</td>
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<tr>
<td>- Start resuscitation at all (extreme prematurity?)</td>
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<td>Adaptable from: O’Malley, P. 2014. Death of a child in the ED. Ann Emerg Med. 64; E1-e17</td>
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I think I need to stop resuscitation…how??

Read the attached article for some information on how to go about terminating resuscitation

**CEASE**

Deciding when to stop resuscitation can be really difficult, whilst there are no definitive methods for deciding which patients are more likely to survive resuscitation than others, there are a number of things to consider which create a bigger clinical picture and make the decision to stop resuscitation more objective.
Think about the Pause

https://consultqd.clevelandclinic.org/why-the-pause-is-important-for-patients-at-the-end-of-life/

Refer to the video on the pressbooks platform to understand this a bit better
References:
Team-Work and the Human Factor

The information presented here will be discussed throughout the NERS course, please read through the practical application of Human Factors awareness in medicine in the PDF linked below and spend some time watching the videos that follow:

Refer to the videos on the online pressbooks platform.

A YouTube element has been excluded from this version of the text. You can view it online here: https://neonates.pressbooks.com/?p=70
Teamwork and the Human Factor

Communication is important

Poor communication in the care of critically ill patients has been shown to increase mortality and suffering for the patient. Poor communication can be a cause of adverse events in the high-pressure ICU and ED environments. Communication is a skill that we can work on to reduce the risk of adverse events. To read more on this important topic see the links below.

- http://eprints.lse.ac.uk/29086/1/Communication_skills_and_error_in_the_Intensive_Care_Unit_(LSERO).pdf
- https://www.ncbi.nlm.nih.gov/pmc/articles/PMC1765783/pdf/v013p00i85.pdf
The topic of human factors and communication will be dealt with throughout the course, as it is an applied learning topic.
Part 4: SKILLS

The skills that will be assessed in the NERS practical day are linked below. Please familiarise yourself with the skills and expectations to ensure a smooth approach to them during the practical sessions. These skills can all be found in the online pressbooks platform.