FOREWORD AND INTRODUCTION

It is well known that obstetric anaesthesia can be the most clinically and technically challenging type of anaesthesia to perform. In some countries, obstetric anaesthesia is a specific subspecialty of the discipline, indicating the high degree of perceived risk and the priority placed on correct management of this patient group.

South Africa, despite high standards of specialist training in internationally recognized schools of excellence, has but a handful of dedicated subspecialty obstetric anaesthesiologists, solely in tertiary training institutions. Their passion for obstetric anaesthesia is, however, often passed onto their trainees who incorporate this into their everyday generalist specialist practice, mostly in the private sector.

The reality in the public sector, however, is that anaesthesia and surgery for the vast majority of procedures in pregnant women and parturients, is performed by inexperienced junior doctors, often with little or no exposure to obstetric anaesthesia. Many of these procedures are acute emergencies, performed at level 1 hospitals and often under conditions that would test the skills of many a specialist.

This reality is reflected in the high maternal mortality rate in our country and the direct contribution of anaesthesia to this mortality rate. The third triennial report of the Confidential Enquiry into Maternal Deaths in South Africa (2002-2004) indicated that anaesthesia was the third leading direct cause of maternal deaths in level 1 hospitals in this period, causing 67 deaths!

The majority of these anaesthetic-related deaths were completely avoidable. The principal causes of these deaths were acute cardiovascular collapse under spinal anaesthesia, difficult or failed intubation under general anaesthesia, with both causes compounded by poor resuscitation skills. These problems can, to a great degree, be addressed by proper training.

The abovementioned report, known nationally as the Saving Mothers Report, made 10 key recommendations to the Minister of Health, one of which was “Skills in anaesthesia should be improved at all levels of health care particularly at level 1 hospitals”.

This ‘book’ or guide is an attempt to help meet this recommendation made by the Saving Mothers Report, at least in the Northern Cape province, where I work.

It is not intended to be a definitive text on obstetric anaesthesia, nor a rigid ‘How to...’ guide. It is, however, a framework with varying layers of academic information, relevant trials, practical points, tips, hints and caveats – many of which have been of practical use to myself and others. In writing this, I have borrowed widely from standard textbooks, journal articles, topic reviews, internet-based summaries and combined these with the practical experience of a number of my colleagues, all specialists in anaesthesia.
Inevitably, as with all texts of this nature, there is a bias towards a personal style of approach to clinical problems and scenarios. This might not be the preference of many specialist colleagues, thus there will undoubtedly be some completely acceptable criticism to this book.

However, this book is aimed at the junior doctor or junior anaesthetist, with emphasis placed on attaining a safe acceptable standard of obstetric anaesthesia.

Thus, the result which I hope I have achieved is a readable, interesting, informative and easy to understand practical guide aimed at the junior doctors who have to perform unsupervised obstetric anaesthesia.

The ideal scenario would be to combine the reading of this book with 1-2 months’ consultant supervised experience in anaesthesia, with emphasis on obstetrics, to put theory into practice.

The desired end result is a thinking junior doctor who can confidently perform safe obstetric anaesthesia in any level facility in South Africa. Achieving this would be of great benefit to our country’s mothers, who deserve optimal healthcare before, during and after labour.

The statistical measured parameter, the maternal mortality rate and more specifically, the anaesthesia-related maternal death rate, would hopefully decrease as a matter of course, once this objective is achieved. Getting this right is the ongoing challenge.

I trust this guide will be of benefit to those who face the challenge of obstetric anaesthesia in rural South Africa.

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(REVISED DECEMBER 2010)
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PHYSIOLOGIC CHANGES IN PREGNANCY

(AND THEIR CLINICAL SIGNIFICANCE)

The pregnant patient is not just a physiologically normal patient who also happens to be pregnant, she is a patient whose physiology started changing at the moment of conception and continues to change until full involution of the uterus at 6 weeks after birth.

These changes have a great impact on her wellbeing as well as on our approach to her anaesthetic management at any stage of the pregnancy. To be able to provide optimal care, it is core knowledge to know what these changes in physiology are and how they affect anaesthetic care.

Respiratory Changes

Respiratory rate increases by 15% and tidal volume by 50% resulting in an increased minute ventilation of 50%. This results in a mild respiratory alkalosis (↓PCO$_2$), which is compensated for by a mild drop in bicarbonate.

The main changes in lung volumes and capacities are a decreased functional residual capacity (15 - 20%), a 20% decreased residual volume and a 20% decrease in expiratory reserve volume due to the elevation of the diaphragm from the pregnant uterus. As pregnancy progresses, abdominal breathing decreases in favor of thoracic breathing. In the supine position, one-third of parturients have airway closure during normal tidal ventilation and are hypoxic. This also makes them prone to atelectasis.

Oxygen uptake increases 20% percent during pregnancy, due to increased maternal metabolism, greater work of breathing and fetal metabolism. The oxyhemoglobin dissociation curve shifts to the right during normal pregnancy (increased P50), allowing a greater volume of oxygen to be unloaded to the tissues (fetus) at a given arterial oxygen pressure.

All of the above factors combine to give the frequently seen clinical scenario of rapid O$_2$ desaturation during induction of general anaesthesia. Effective pre-oxygenation prior to induction is obviously of paramount importance.

**Respiratory changes cause rapid hypoxia therefore effective pre-oxygenation is mandatory prior to induction of anaesthesia!**

The Airway

During pregnancy, capillary engorgement of the mucosa occurs throughout the respiratory tract, potentially causing edema in the nasopharynx, oropharynx, larynx, and trachea, therefore any manipulation of the upper
airway requires extreme care. Suctioning of the oropharynx, insertion of airways, and laryngoscopy may cause further edema and bleeding.

Because the area of false cords may be swollen, a small, cuffed endotracheal tube (6.5–7.0 mm) is recommended for use when the trachea is intubated. Repeated attempts at laryngoscopy during management of a difficult airway must be minimized to prevent obstructing airway edema.

- The airway may become more difficult to manage and a smaller ET tube size is needed.

Cardiovascular Changes

The cardiovascular system undergoes great changes during pregnancy and is particularly stressed during childbirth.

These changes already appear during the first trimester of pregnancy, eg there is an increase in cardiac output of 22% and decrease in systemic vascular resistance [SVR] by 30% at 8 weeks' gestation. These progress into the second and early third trimester of pregnancy, when cardiac output increases to approximately 30 to 40 percent above nonpregnant values.

Increased cardiac output is mainly due to increased stroke volume (30%) and an increase in heart rate of 10–15 bpm. (Remember HR x SV = Cardiac output)

The increased blood volume and cardiac output may produce changes in the cardiac examination of the pregnant patient. A wide, loud split first sound and a soft (Gr I/II) systolic ejection murmur are commonly heard. The apex of the heart is displaced to the left by the elevated diaphragm at term. This changes the axis of the heart on the electrocardiogram to the left and the ECG may show minor, nonspecific ST-, T-, and Q-wave changes and benign arrhythmias.

NB!! These normal findings should be differentiated from those indicating heart disease. Signs of significant cardiac pathology in the pregnant woman include true cardiac enlargement, severe arrhythmias, systolic murmur greater than grade 3, or any diastolic murmur.

Ironically, the term mother is susceptible to hypotension when supine. Up to 10% of pregnant patients near term develop signs of shock (hypotension, pallor, sweating, nausea, vomiting, changes in cerebration) when they assume this position. This is due to the aortacaval syndrome, where the large uterus partially or completely compresses the aorta and inferior vena cava, decreasing venous return to the heart, causing a decreased cardiac output, hypotension, and reduced uterine blood flow.
The anesthetist must know of and recognize the aortocaval compression syndrome because of the acute collapse that can be precipitated by spinal and general anaesthesia in the supine position. Aortocaval compression is prevented by manually displacing the uterus to the left, moving it off the great vessels. During labour, the patient should be positioned either on her side or supine with a left lateral tilt.

During delivery, the operating or delivery table must be tilted laterally to the left by about 15°. This can be achieved effectively by placing a small pillow or foam rubber wedge under the patient's right buttock.

During labour, pain and apprehension adds to the cardiac work of pregnancy and increases stroke volume and cardiac output by 45% over prelabour values occur. Additional stresses occur with each uterine contractions, which cause, in effect, an autotransfusion. Blood from the body of the uterus is pushed into the central circulation, and blood volume and cardiac output increase acutely by 10 to 25 percent. The same autotransfusion occurs directly after delivery. Added to this increase in central blood volume, obstruction of the vena cava by the uterus is relieved, resulting in a massive increase in stroke volume and cardiac output immediately postpartum (up to 80% of prelabour values). Patients with limited cardiac reserve may experience cardiac failure at this time.

Cardiovascular changes return to normal by 4 weeks postpartum.

- **Aortacaval compression requires of you to left-tilt or wedge (under right hip) all patients for Caesarean section**
- **Pregnant cardiac patients must be referred early to a main centre and early to an Anaesthesia Consultant.**

**Haematological**

Maternal blood volume increases progressively during pregnancy and is 35-40% more at term (> 1,000 ml) than pre-pregnancy values.

Plasma volume increases from 40 ml/kg before pregnancy to 70 ml/kg during late pregnancy, and RBC volume increases from 25 to 30 ml/kg.

RBC volume increases at a slower rate than the plasma volume, causing the relative anemia of pregnancy.

This dilutional anemia of pregnancy decreases the O₂-carrying capacity of blood, but there is compensation for this by an increase in PO₂, a decrease in blood viscosity, the increase in cardiac output, and the rightward shift in the maternal oxyhemoglobin dissociation curve.
The pregnant woman at term is in a hypercoagulable state owing to increases in factors VII, VIII, X, and plasma fibrinogen. Estimates of blood loss at delivery vary but average around 500 ml for an uncomplicated vaginal delivery and 500-1400ml during cesarean section.

- Term pregnant patients are anemic and hypercoagulable, ∴ mobilise early after any operation!

**Hepatic Changes**

Liver enzymes ALP, serum transaminases, LD and cholesterol increase slightly during pregnancy, but serum bilirubin levels and hepatic blood flow are unchanged. Thickened bile in the gallbladder combined with decreased emptying make pregnant women prone to gallstones and cholecystitis in pregnancy.

Total protein level and the albumin-to-globulin ratio decrease. Although plasma cholinesterase activity is reduced during pregnancy, moderate doses of succinylcholine are metabolized easily and no clinical effects are usually seen.

**Gastrointestinal Changes**

In pregnancy, the secretion of gastric acid increases. During late pregnancy, gastric emptying is delayed by displacement of the pylorus by the enlarged uterus. Pain, anxiety, and use of opioids during labour also contribute to delayed gastric emptying.

In a clinical trial of pregnant patients in labour, solid food was found in >40% of those who had not eaten for 8 to 24 hours before examination, compared to none in non-pregnant women or pregnant women not in labour. Intragastric pressure is increased during pregnancy, while lower esophageal sphincter pressure is decreased.

All these changes increase the risk of regurgitation and aspiration during either general anesthesia or impaired consciousness from any other cause. Pregnant women have at least a tenfold increased risk of gastric aspiration compared to the general surgical population.

It remains controversial, but is presently accepted that this increased risk is present from 12weeks of pregnancy to 14days postpartum. These patients should thus receive antacid prophylaxis and rapid sequence endotracheal intubation for general anaesthesia during this period. (See Protocol)
 Pregnant patients are at increased risk for regurgitation and aspiration of stomach contents from 12 weeks gestation to 14 days postpartum. Routine antacid prophylaxis and rapid sequence intubation!

Central Nervous System Changes

Pregnancy reduces anesthetic requirements both during regional and general anesthesia. For spinal or epidural anesthesia, less local anesthetic is required to produce a given level of anesthesia.

The MAC requirement for inhaled anesthetics is decreased, probably due to a pregnancy-induced activation of the endorphin system.

- The parturient requires a much smaller dose of local anaesthetic for her spinal anaesthesia! (the ‘standard dose’ is 1.8ml of 0.5% Bupivacaine with dextrose plus 10μg Fentanyl – 2.0ml total volume)

Renal Changes

Renal plasma flow and glomerular filtration rate increase rapidly during pregnancy, reflecting changes in cardiac output. During the third trimester, they slowly return toward normal. Creatinine clearance usually increases, and therefore the upper limits of normal for serum ureum and serum creatinine are lower in the pregnant woman.

Uterine Blood Flow

Uterine blood flow in the parturient at term is approximately 700 mL/min and is determined by the following relationship:

\[
\text{Uterine blood flow} = \frac{\text{uterine arterial pressure} - \text{uterine venous pressure}}{\text{uterine vascular resistance}}
\]

There is no autoregulation of uterine blood flow and the vessels are maximally dilated during pregnancy. Therefore uterine bloodflow is a passive function directly dependent on maternal blood pressure and cardiac output.

Uterine blood flow (thus placental bloodflow) decreases during maternal hypotension (sympathetic block, hypovolemia, hemorrhage, compression of the inferior vena cava), in circumstances in which uterine venous pressure is increased (compression of the inferior vena cava, abruptio placentae),
and with increases in uterine vascular resistance (maternal hypertension, α-agonists, uterine hypercontractility).

Obstetric anesthesia may affect uterine blood flow by changing the perfusion pressure (i.e., by altering the uterine arterial or venous pressure) or by changing uterine vascular resistance, either directly through changes in vascular tone or indirectly by altering uterine contractions or uterine muscle tone.

**Ephedrine is the vasopressor of choice** for the treatment of hypotension during obstetric anesthesia. Thus, when maternal blood pressure and cardiac output is restored with ephedrine therapy, uterine blood flow increases.

**Phenylephrine** (50- to 100-µg boluses) has also been used successfully in normovolemic, healthy cesarean section patients in correcting maternal blood pressure during regional anesthesia without adverse effects on neonatal outcome.

❖ **Uteroplacental (and fetal) perfusion is not autoregulated, relies on maternal perfusion pressure, thus hypotension must be treated immediately!**

*(Many obstetric anaesthetists feel that Phenylephrine has now superceded Ephedrine in this regard. However, Phenylephrine is frequently unavailable in smaller institutions and therefore Ephedrine remains drug of choice generally. If available, use it. NB! See later text for discussion regarding choice of vasopressors in the clinical context)*
AIRWAY EVALUATION IN THE OBSTETRIC PATIENT

Airway evaluation of any and every patient who is to undergo an anaesthetic is an absolute necessity. Nowhere is this more important than in the obstetric patient.

Note that in the above introduction, the sentence does not read “….to undergo a general anaesthetic” –

AIRWAY EVALUATION MUST BE DONE FOR ALL PATIENTS!

Conversion of a failed spinal anaesthesia to general anaesthesia is an independent risk factor for maternal airway problems (and thus mortality!) and this risk is mainly due to lack of prior airway examination.

Obstetric patients have notoriously difficult airways. A recent study of maternal airways, evaluated by MRI scanning, found that the average maternal airway changed from a Mallampati class I during the course of pregnancy. A significant number of these changed to a Mallampati class III by the time the mother reached term pregnancy! (See below for the Mallampati classification)

Statistically, when compared to the general surgical population, they been proven to have an 18 times greater difficult intubation rate and also a 10 times greater failed intubation rate!

Anaesthetic-related cause of maternal death is due to intubation problems in >50% of cases and the maternal airway is thus not to be underestimated.

GENERAL RISK FACTORS FOR A DIFFICULT AIRWAY

- Obesity *
- Thick neck *
- Short neck *
- Large tongue in relation to oral cavity *
- Poor neck extension *
- Large breasts *
- Absent or prominent upper incisors *
- Poor mouth opening
- Mandibular, maxillary or palatal anomalies
- (Beard)

The above factors marked with an asterisk are those commonly found in pregnant patients.

The maternal airway at term is further complicated by the fact that capillary engorgement of the mucosa occurs throughout the respiratory tract, causing edema in the nasopharynx, oropharynx, larynx, and trachea. Any manipulation, even suctioning, may cause the mucosa to bleed, further worsening visualization of airway structures.
EXAMINATION AND EVALUATION OF AN AIRWAY

The method of examination of an airway can easily be remembered by the 3 M’s, namely Mouth, Movements and Measurements.

A. MOUTH
The mouth examination consists of 3M’s, mouth opening, mouth contents and Mallampati score.

**Mouth opening** should be at least 2 patient fingerbreadths, preferably 3. Ask the patient to place at least 2 fingers vertically between her teeth. Measured, the interincisor gap should be >4cm.

**Mouth contents** are teeth, tongue and soft tissue (3T’s). Prominent or absent upper incisors, loose front teeth and abnormal occlusion, eg lack of normal maxillary overbite, may indicate potential problems.

Examination of the tongue indicates whether the tongue is mobile, is normal in structure and size in relation to the oral cavity.

Soft tissue includes the palate – a high narrow palate often causes problems with laryngoscopy – and the mouth floor, which should be soft and appear normal.

**Mallampati**
The Mallampati classification is divided into 4 grades, according to the visualization of the palate, pillars, uvula and base of tongue. There is increased difficulty with intubation with grades 3 and 4 (up to10% failed intubation), whereas there is usually good correlation with ease of intubation with Mallampati grades 1 and 2 (93% easy intubation, <1% failed).

Combining Mallampati score with other parameters will decrease the likelihood of having an unrecognised difficult airway.

- **Class I** = visualization of the soft palate, fauces, uvula, anterior and posterior pillars.
- **Class II** = visualization of the soft palate, fauces and uvula.
- **Class III** = visualization of the soft palate and the base of the uvula.
- **Class IV** = soft palate is not visible at all.
B: MOVEMENTS

Evaluation of movements of the neck, head and jaw can indicate the potential for problems with intubation or airway management.

Full neck flexion (place chin on chest) and neck extension (occiput on back) indicate a decreased potential for airway problems.
A neck that cannot be extended beyond the neutral position indicates a high probability for airway problems or a difficult intubation, while limited extension indicates a risk.

The jaw movement most indicative of good TMJ function and anterior jaw movement during laryngoscopy, is obtained by asking the patient to protrude her mandibular teeth anterior to the maxillary teeth (like a bulldog). Inability to do this indicates a possible airway problem.

C: MEASUREMENTS

There are many measurements that are used in the head and neck, but the ones most practical to use that give the most information are the following three:
- Inter-incisor distance > 4cm (see mouth opening above)
- Thyromental distance > 5cm (should be able to insert 3 or more fingerbreadths from the top of the thyroid cartilage to the tip of the chin)
- Sternomental distance (suprasternal notch to tip of chin) > 12cm – easily tested by inserting a pen into this space
Added to all of these is your physical clinical impression of the patient’s head and neck from the front and in profile. Any abnormal appearance, eg micrognathia, macrognathia, midfacial hypoplasia or obvious disproportion should make you sit up and take notice.

A recognised stepwise approach used by Mallampati himself is:
1. Mallampati classification
2. Thyromental distance
3. Movements

With a small amount of practice, a complete airway assessment can be done in a matter of minutes and becomes second nature.

NB! It must be understood that although this evaluation will identify most potential airway management problems, it does not completely rule out the possibility of experiencing an airway problem.

❖ THE ANAESTHETIST SHOULD ALWAYS BE READY TO MANAGE AN UNRECOGNISED DIFFICULT AIRWAY.

❖ There are other mnemonics available to help assess airways, eg the MELON/LEMON and LM-MAP approaches – these are basic and easy to remember – see on the Internet.

INDICATORS OF A DIFFICULT OBSTETRIC AIRWAY:

- history of previous airway difficulties
- obesity or rapid heavy weight gain during pregnancy
- large breasts
- absent upper incisors
- short thick neck
- PET/eclampsia, especially with associated oedema
- Facial oedema in PET – indicates possible laryngeal oedema
- Hoarse/”Donald Duck” voice or recent voice change in PET – indicates swelling of the glottis
- Comorbid disease eg diabetes, rheumatoid arthritis
PRACTICAL TIPS TO IMPROVE FIRST-TIME INTUBATION RATE

A. Basic Manouevres

- Place patient in the sniffing position (the position of smelling coffee in the morning – this optimally aligns the trachea with pharynx-larynx) NB! Obese and pregnant patients usually require an exaggerated sniff position
- Use the right size laryngoscope blade (most take a size 4) and ensure that it is properly placed – too shallow or too deep results in a poor laryngoscopic view
- Do not lever your wrist in an attempt to improve your laryngoscopic view, rather push your bent arm rigidly forward (brachioradialis muscle) from your shoulder
- Get your assistant to insert his/her pinky into the right mouth corner and retract laterally, this improves your view of the larynx.
- After insertion of the laryngoscope blade, perform the OELM (optimal external laryngeal manipulation) manoeuvre if the laryngoscopy view is not grade I. This manoeuvre should be automatic and can still be performed even with cricoid pressure applied. It consists of the anaesthetist placing the right hand over the external larynx (over the assistant’s hand) and displacing the larynx laterally to the right (the assistant must not remove cricoid pressure) – this either brings the glottis into view or improves the laryngoscopy grade significantly.
- Optimise your eye’s alignment with the trachea, ie intubate from behind, not above. This might mean that you have to sit or get down on your knees, or raise the head of the table
- On laryngoscopy, if your view, even after OELM and other manoeuvres, is still not good, quickly look and see if you can identify the protuberances of the arytenoid cartilages (the corniculate tubercles) – see below. An imaginary transverse line between the two protuberances is the dividing line between trachea and oesophagus. Anterior to the line is trachea. A malleable introducer can be used to provide angulation of the ET tube tip if necessary.
B. Advanced Manoeuvres

1. The Obese Patient:

   Place a pillow between the scapulae, exaggerate the sniffing position; raise the head of the bed. If she is very obese, consider the HELP position (Head Elevated Laryngoscopy Position) prior to induction.

   Positioning of Patient on Irrigation Bags

   The patient's head is raised high enough for the anterior neck to be in the same horizontal plane as the sternal angle (angle of Louis)

2. The Patient with Large Breasts:

   Get a person to pull the breasts downwards using a flat hand just after the patient is induced. This opens the neck anteriorly and improves the effect of the technical/manual manoeuvres. If the patient is also obese, apply the additional above manoeuvres mentioned in 1.

3. The Patient with Absent Upper Incisors:

   The laryngoscope blade tends to slip into the gap and distorts your view or confounds your laryngoscopy and intubation attempt. Place one of the following in the gap to prevent this: rubber dental bite-block, tip of oral (Guedel) airway upside down, 10ml or 20ml syringe, 4-5 wooden tongue spatulas.

4. The Patient with a Small Mouth/large tongue:

   These patients are often difficult to intubate due to the fact that once inserted, the laryngoscope blade obstructs your line of sight and makes you hesitant to insert the ET tube. Have your assistant retract the right mouth corner as described earlier under basic manoeuvres. You can also insert your ET tube from laterally, so that the shaft of the tube points away from you instead directly in your line of vision.
5. Oesophageal Intubation:
   If you immediately recognise that you have intubated the oesophagus and if the mouth space permits, DO NOT REMOVE THE ET TUBE! Deflate the cuff, take another tube and intubate anteriorly to the ET tube already there – it should enter the trachea. Alternatively, use an intubating bougie (see in the next chapter) to enter the trachea and railroad your ET tube over the bougie.
ADVANCED AIRWAY MANAGEMENT

The implementation of a protocol for managing a difficult airway has resulted in a great reduction in the morbidity and mortality associated with airway problems.

The American Society of Anaesthetists brought out a national protocol which has been implemented in many parts of the world and this will be used as a basis for advanced airway management.

The most important factor in airway management is to try and avoid airway problems by recognizing the difficult airway prior to surgery, thus having plans in place at the onset of induction. It is important to have a backup plan(s) in case your original plan fails.

Obviously each hospital has different equipment and anybody planning an anaesthetic must know what equipment is available to him/her in that hospital. The management plan is thus dependant on the available equipment. However, most airways can be managed safely with the minimum of equipment, a plan of action and a cool head.

EQUIPMENT

It is important to have a fully stocked trolley with equipment on that does not get used as a routine. This should be your Difficult Intubation Trolley and must be separate to your normal tray. On this special trolley should be all the additional equipment you might need in your setting to manage the airway. In the obstetric theatre you should have:

1. LARYNGOSCOPES
   Working laryngoscope handles with at least 2 different sized blades, namely size 3+4 (with functioning bulbs!) should be available. NB! These should be checked as part of your machine check. If your hospital has the funds for it, a lever-tipped McCoy laryngoscope is a good addition to the standard laryngoscopes.

2. SUPRAGLOTTIC AIRWAYS
   The best known supraglottic airway is the LMA, of which there are a number of variations and more recently, disposable models. The basis of all the supraglottic airways is the introduction of a tube with a soft inflatable seal into the hypopharynx. The tip of the seal usually lies at the oesophageal opening, with the tube opening just above the glottis. Once sealed the patient has a well established airway without intubating the trachea. Manual and even mechanical ventilation can be performed through this airway.
   It is important to remember that this is still only a supraglottic airway and does not protect against aspiration. At least sizes 3 and 4 should be readily available in the obstetric theatre.
Two variations of good use in the difficult airway are the Combitube® and the Fastrach® (or ILMA – intubating LMA). Either of these would be excellent additions to a difficult airway trolley. The Combitube® requires little to no extra expertise to use effectively, whereas the Fastrach® requires some experience with an LMA plus a modified insertion technique.

3. **STYLETs**

The most effective and commonly used stylet for difficult airways is the gum elastic bougie. This is a reusable and malleable stylet with an anteriorly angled tip which allows passage through the vocal cords in a high and angled glottis. An endotracheal tube is then railroaded over the bougie.

A similar stylet is available with a battery-operated light at the tip, known as the LightWand®, which transilluminates the anterior larynx when the tip is in the trachea.

4. **SURGICAL AIRWAY EQUIPMENT**

In an emergency situation where a surgical airway is the *only option*, a needle cricothyrotomy or surgical cricothyrotomy are the only feasible procedures to perform. The needle cricothyrotomy combined with manual or jet ventilation is lifesaving and buys time for a surgical cricothyrotomy, tracheostomy or calling for help from an experienced airway manager.

However, the decision to undertake the surgical airway option should not be taken lightly, as all surgical airways are highly invasive and can have high complication rates. This decision is usually taken only in the case of a CICV (“can’t intubate-can’t ventilate”) scenario. (see later)

A **needle cricothyrotomy** can be performed with a 16G or 14G cannula inserted through the cricothyroid membrane. A 2ml syringe with its plunger removed is pre-fitted with the connector of an ET tube (usually 7.5) and the syringe is connected to the cannula. An AMBU bag or ventilator can be attached to the connector and the patient ventilated. It may be a good idea to have one already set up on your trolley (keep the sterile needle separate).

There are a number of surgical airway kits available to perform a **surgical cricothyrotomy**. Basically the idea is to incise through the skin over the cricothyroid membrane, puncture the membrane with a blade and insert a 5.0 ET tube into the trachea. This can be done without a kit, but prepacked kits have everything required, with a tracheostomy type tube instead of an ET tube.

This is not the place to discuss these techniques in detail and you are referred to standard texts or emergency medical manuals. However, it is strongly suggested that you attend an ATLS course, which manages these procedures in detail and practise.
Retrograde Intubation

- A Tuohy epidural needle is inserted through the cricothyroid membrane
- Position is confirmed by aspiration of air through a saline filled syringe
- A guidewire (CVP guidewire is ideal) is inserted through the needle and emerges through the mouth or nose
- An appropriately sized ETT is then guided along the catheter until it rests against the exit of the catheter from the larynx
- With continued application of gentle pressure in a rotating or corkscrew fashion, the ETT is advanced into the larynx

There are various forms of this technique, generally known as guided "blind" retrograde oral or nasal intubation; the major difficulty with the technique is passage of the ETT past the epiglottis, tending to be more difficult from the oral route

Basic Airway Management

The basic airway management techniques will only be mentioned here, because it is accepted that these are performed routinely.

These are:
- Supplemental 100% O₂ with tight-fitting mask and mask ventilation
- Chin lift
- Jaw thrust
- Bimanual jaw thrust with assistant doing bag compression
- Oral/nasal airways
- Sniffing position and use of pillows

Advanced Airway Management (General)

The basic progression of invasiveness for difficult airway management is:
- LMA or supraglottic airway
- Laryngoscopy (2 attempts with blade change)
- Laryngoscopy with McCoy laryngoscope (if available)
- Laryngoscopy and gum elastic bougie
- Lightwand (if available)
- Blind nasotracheal/orotracheal technique
- Retrograde technique
- Fibreoptic intubation
- Surgical airway

The above progression is a guideline and is dependent on the skill and experience of the airway operator.
The Recognized Difficult Airway

As previously stated, the prior recognition of the possible difficult airway is the best management of a difficult airway, because proper preparations can then be made. In the obstetric patient this can even mean elective referral to a higher level of care.

There are some questions to be asked when a possibly difficult airway is recognized and in obstetric patients these questions should probably be routine.

1. **Is this an emergency procedure?** If so, only a truly dire emergency should prevent you from calling for extra assistance from colleagues or organizing which plans you want to implement. These are also often the cases that aren’t amenable to spinal anaesthesia and are therefore all the more reason why a rapid but thorough airway assessment should be done.

2. **Is airway management required?** The tendency to do regional techniques for obstetric patients means that airway management can often be avoided, but this does not obviate the need for airway assessment. Sometimes an unexpected failure of the regional technique occurs, requiring rapid conversion to GA. This in itself is an independent risk factor for airway problems.

3. **Is laryngoscopy expected to be difficult?** The chapter on airway assessment gave all of the risk factors and clinical indices for difficult laryngoscopy. However, these are not foolproof and the anaesthetist must not be lulled into a false sense of security – always be prepared to manage a difficult airway.

4. **Can the patient be ventilated with a supraglottic airway?** This is not usually an option for a patient with an aspiration risk. However, the use of the LMA as a rescue device for a failed intubation in the obstetric patient is well established and is indeed part of the failed intubation protocol in these patients. The proviso is that cricoid pressure must be maintained throughout the duration of the procedure and high pressure manual ventilation be avoided.

**WHAT DO YOU DO ONCE A POSSIBLE DIFFICULT AIRWAY IS RECOGNIZED?**

1. Communicate your problem to the patient and advise them on the possible and probable scenarios that may ensue, eg wake-up without operation; delay to wait for experienced hands; referral; surgical airway.

2. Ensure that your patient has had optimal antacid prophylaxis (Protocol).
3. Contact your most senior anaesthetist available for help and/or advice. This may require a call to your regional hospital (Kimberley Hospital) to speak to the Consultant if you have no experienced senior locally.

4. Inform your team of the possibility of a problematic airway and advise them on what interactions you may require from them, eg the surgeon should not scrub up, but be available to assist you; the anaesthetic nurse should double check all the equipment you have identified as possibly needed.

5. Have your plans well formulated in your mind and all your equipment at hand. It might be helpful to share your plans with the team, so they know how you think. It will then be a logical extension of the thought process for them to assist you.

6. If the airway pathology is severe enough for you to suspect that your plans might fail, contact the Consultant Anaesthetist at your regional hospital for advice and possible timeous referral of the case for expert management. It is important to recognize that correct referral is also proper management rather than a ‘failure’ of your anaesthetic ability.

7. Institute your plans with care and confidence, but always be ready to institute your Failed Intubation Protocol.

PRACTICAL ISSUES (Not specific to the Obstetric patient)

- When faced with a difficult/failed intubation, do not persist in trying to intubate at all costs, because you will intubate your patient to death! Get control of the airway with mask ventilation, then decide on further steps. Insert an LMA if you cannot ventilate effectively with a mask.

- In the elective case, if feasible (usually not in the obstetric setting), do a gas induction maintaining spontaneous respiration. When the patient is deep enough, carefully inspect the airway with the laryngoscope. If the airway looks easy, deepen the patient with an IV induction agent and paralyse with Sux to intubate. If the vocal cords cannot be seen on inspection, maintain spontaneous respiration with the volatile agent. Reattempt laryngoscopy when patient is deep; if the cords cannot be visualized again, decide if a supraglottic airway will be acceptable for surgery. If it is, insert the LMA and continue; if not, waken the patient.

- In the elective case requiring intubation, where airway pathology is of such a nature that it is obvious that intubation will be impossible, refer the patient to the regional hospital for expert attention.

- In the emergency case, a regional anaesthesia is the obvious choice, but if not feasible, consider an awake intubation. If an awake intubation is not feasible and surgery cannot be delayed, continue a cautious induction with all plans and equipment in place. Optimise patient position and all postural manoeuvres (pillows, sniffing position) prior to
induction. Use only short-acting drugs and maintain brain oxygenation at all costs. Attempt intubation, but have a low threshold for using a bougie; if this fails, insert a supraglottic airway and maintain full cricoid pressure for the duration of the procedure. Allow either spontaneous respiration or assist the ventilation manually with low pressure (<20cmH₂O).

- In the emergency Caesarean section where a spinal anaesthetic cannot be employed, where airway pathology is of such a nature that it is obvious that intubation will be impossible, and where surgery cannot be delayed, the only option left is to employ a sequential local anaesthetic infiltration combined with low-dose Ketamine (0.1-0.5mg/kg).

  The local anaesthetic should be lignocaine combined with adrenaline to allow for greater volume (max 7mg/kg). The surgeon must infiltrate layer for layer as the surgery progresses. Opiates and other sedatives should be avoided if at all possible.

Unfortunately, many of the difficult obstetric airways go unrecognised and the anaesthetist is faced with the unrecognised difficult airway and possible failed intubation, a scenario where there are usually no plans or preparations in place. This is usually compounded by the fact that the surgery is emergent. Unless the anaesthetist keeps his/her head and takes control of the situation, death or severe neurological damage may await both mother and baby.

**The Unrecognized Difficult Airway**

**PRACTICAL ISSUES**

- Have a general plan in place for a DIFFICULT AIRWAY/FAILED INTUBATION SCENARIO in YOUR theatre suited for your needs, taking into account your experience, the experience of your staff and the equipment you have available. If needs be, have your plan or protocol printed and laminated and stick it on the wall next to your anaesthetic machine.
- Ensure that your theatre staff and colleagues are aware of your protocol and what will be expected of them.
- Practice the failed intubation scenario as a drill with your staff to ensure they understand what to do.
- When faced with a difficult/failed intubation, do not persist in trying to intubate at all costs, because you will **intubate your patient to death**! Get control of the airway with mask ventilation, then decide on further steps.
- The failed intubation scenario is not the place to ‘try out’ newly read or heard-of techniques unless as a last life-saving resort.
WHAT DO YOU DO WHEN AN UNRECOGNISED DIFFICULT AIRWAY/FAILED INTUBATION SCENARIO OCCURS?

- Immediately declare a failed intubation scenario so that all present in theatre know that there is a life-threatening situation on hand.
- Get your failed intubation trolley and institute your failed intubation protocol.
- Immediately decide whether you can mask ventilate effectively – if you can, then you have airway control and the same approach can be taken as discussed above. If mask ventilation is difficult or impossible and none of the normal airway manoeuvres (including LMA) are helpful, perform a needle crico-thyrotomy and ventilate until control is obtained.
- Once ventilation control is obtained, a decision must be made by the team as to whether the surgery must continue. If surgery can be delayed, wake the patient up. If surgery is absolutely necessary, the airway must be definitively controlled first, either by a surgical cricothyrotomy/tracheostomy or retrograde intubation. Keep the patient asleep with small boluses of an induction agent.
- After the procedure it is important that the patient get a MedicAlert bracelet to prevent a repeat of a disastrous scenario! This is the anaesthetist’s job.

Below is a summary of management of the unrecognised difficult airway:

<table>
<thead>
<tr>
<th>Can’t Intubate – Can Ventilate!</th>
<th>Can’t Intubate – Can’t Ventilate!!</th>
</tr>
</thead>
<tbody>
<tr>
<td>Intubating your patient to death!</td>
<td>Intubating your patient to death!</td>
</tr>
<tr>
<td>Assess need for intubation.</td>
<td>CICV ≈ Surgical Airway!!</td>
</tr>
<tr>
<td>Call for equipment and assistance!</td>
<td>Call for equipment and assistance!</td>
</tr>
<tr>
<td>Keep your head!</td>
<td>Keep your head!</td>
</tr>
<tr>
<td>Remember to follow your protocol (ASA algorithm)</td>
<td>Remember your basic Surgical Airway Anatomy</td>
</tr>
<tr>
<td>Types of non-surgical interventions:</td>
<td>Types of Surgical Airways:</td>
</tr>
<tr>
<td>- LMA and supraglottic airways</td>
<td>- needle cricothyrotomy ± TTJV</td>
</tr>
<tr>
<td>- bougies</td>
<td>- surgical cricothyrotomy</td>
</tr>
<tr>
<td>- light wand</td>
<td>- percutaneous tracheotomies</td>
</tr>
<tr>
<td>- fibre-optics</td>
<td>- formal tracheostomy</td>
</tr>
<tr>
<td>Decision to continue based on urgency, paralysis status and postoperative factors</td>
<td>Decision to continue based on urgency, paralysis status and postoperative factors</td>
</tr>
</tbody>
</table>

**MEDICALERT  MEDICALERT**

The Saving Mothers Group has suggested to follow the protocol below.
FAILED INTUBATION

MAINTAIN CRICOID PRESSURE
VENTILATE WITH 100% O2

F0ETAL DISTRESS REQUIRING IMMEDIATE DELIVERY
VENTILATION EASY
VOLATILE WITH 100% O2
CONTINUE VENTILATION WITH CRICOID PRESSURE
DELIVER BABY

MINIMAL OR NO FOETAL DISTRESS
VENTILATION DIFFICULT
CRICOTHYROTOMY
VENTILATION DIFFICULT
VENTILATION EASY
WAKE PATIENT

INSERT LMA
VENTILATION EASY
VENTILATION DIFFICULT
ANAESTHESIA FOR THE PREGNANT PATIENT
(for non-obstetric procedures)

Approximately 2% of all pregnant patients will present for a non-obstetric surgical procedure requiring an anaesthetic during their pregnancy.

These are mostly trauma-related, but abdominal procedures for appendicitis, ovarian cyst torsion/rupture and cholecystectomy are not uncommon. Placement of McDonald’s and Shirodkar sutures and breast lump surgery is also frequent.

GENERAL GUIDELINES FOR SURGERY DURING PREGNANCY

- Only emergency surgery should be performed during pregnancy, particularly in the first trimester.
- Urgent surgery should be deferred to 2nd or 3rd trimesters
- Elective surgery should wait until 6 weeks postpartum.

The possibility of pregnancy should be considered in all female surgical patients of reproductive age (ie 12-50yrs). Menstrual history should be obtained and β-HCG tested for routinely if the patient has unsure dates or >3 weeks after last menstruation.

RISKS

Pregnant patients are naturally apprehensive for the fetus’s wellbeing when faced with the prospect of surgery and anaesthesia. It is therefore important to be able to confidently spell out the possible risks of fetal loss or premature labour to the mother.

- The risk of a 1st trimester miscarriage increases from 5% to 8% if a patient undergoes surgery.
- The risk of premature labour increases from 5% to 7.5% if a patient undergoes surgery.

The important thing to recognize is that the figures given are the combined risk of anaesthesia, surgery and the pathology for which the surgery is being performed. Anaesthesia is probably the least contributory, according to all available evidence.

Teratogenic effects of anaesthetic drugs (including volatiles, narcotics, induction agents, muscle relaxants and local anaesthetics) are minimal to non-existent and have a long history of safety. Despite this, it is generally accepted that exposure to anaesthetic drugs during organogenesis should be avoided.

PHYSIOLOGICAL EFFECTS OF PREGNANCY:

There are a host of changes on maternal physiology, the most important being:

CVS: Increased cardiac output (22%) and decreased SVR (30%) by 8weeks
      Increased blood volume (30-40% at term)
      Dilutional anaemia
      Aortacaval compression (10% have supine hypotension from start of 2nd trimester)
      Decreased vascular responsiveness, but increased baroreceptor responsiveness
RESP: 20% higher O\textsubscript{2} consumption
Decreased FRC (with closing volume falling within normal tidal volume respiration in a third of supine patients at term)
Lower PCO\textsubscript{2} due to increased minute ventilation

The above 3 factors give rise to the rapid desaturation of pregnant patients on induction, making effective preoxygenation mandatory.

There is an 18-fold increase in difficult intubations.

GIT: Delayed stomach emptying occurs from as early as 12 weeks
Stomach contents are more acid due to increased gastrin secretion
Lower gastro-oesophageal tone is decreased.

This together with the increased likelihood of difficult intubation increases the risk of aspiration.

CNS: MAC for volatiles is decreased
Local anaesthetic dose requirement is decreased.

Based on the above discussion, the following approach is suggested for anesthesia in the pregnant patient:

Pre-op:
- A reassuring pre-anaesthetic visit must be done to allay patients fears and counsel on risks.
- Decide on GA/regional technique in conjunction with patient and surgeon. Advantages of regional technique are minimal surgical stress, avoidance of drugs and intubation, and minimal changes in FHR variability.
- Administration of sedative and analgesia as required.
- Antacid prophylaxis should be given. Ranitidine and metoclopramide may be useful and administration of a non-particulate antacid, 15–30 ml, within half an hour of induction of anaesthesia.
- Discuss tocolysis with the obstetrician (Magnesium sulphate or Indomethacin supp)

Induction:
- Beginning in the second trimester, uterine displacement must be maintained at all times (wedging or 15° left tilt).
- Hypotension related to spinal or epidural anesthesia must be prevented as much as possible by rapid IV crystalloid infusion before induction (co-loading). Maternal hypotension should be promptly treated with IV ephedrine, etilephrine or phenylephrine.
- General anesthesia should be preceded by effective denitrogenation (100% O\textsubscript{2} @ 8 l/min x 3min with at least 8 deep breaths per min)
- The risk of aspiration should be minimized by application of cricoid pressure and rapid tracheal intubation with a cuffed tube.
- Particularly during the first trimester, it is preferable to choose drugs with a long history of safety; these drugs include thiopentone, propofol, morphine, pethidine and any of the muscle relaxants.
Maintenance:
- Avoid maternal hyperventilation - monitor ET CO2.
- Ideally, fetal heart rate should be monitored continuously throughout surgery and anesthesia after 20-24 weeks gestation (if practical). Loss of FHR variability or decelerations may indicate intra-uterine stress and the need for intra-uterine resuscitation (increase oxygenation, raise BP, increase uterine displacement, tocolysis or change of site of surgical traction)
- Maintain normotension, low normocarbia, normothermia.
- Use high FIO₂ (at least 50%)
- Use low MAC of volatile agents, preferably 1MAC or less
- Low concentration of nitrous oxide may be used
- Provide effective pain relief intra-op. Catecholamine surges increase uterine tone and decrease placental bloodflow.
- The general approach would be to use a combination GA using short-acting drugs at low doses.
- Monitor the blood glucose if the procedure is long.

Emergence:
- Effective pain relief
- Reversal with Neostigmine and Atropine
- Awake extubation in lateral head-down position.
- Maintain oxygenation and uterine displacement.

Postop:
- Maintain oxygenation and uterine displacement
- Monitor FHR and uterine activity
- Effective pain relief! NB! Systemic analgesics may decrease FHR variability.
- Early mobilization, because pregnant patients are hypercoagulable.
TRAUMA IN PREGNANCY

In many countries, trauma is the leading non-obstetric cause of maternal morbidity and mortality. There is an increased incidence of falls, assaults and iatrogenic injury in pregnancy.

Major trauma in pregnancy has a mortality rate of 7-24%.

Physiological changes

<table>
<thead>
<tr>
<th>CHANGE</th>
<th>IMPLICATION</th>
</tr>
</thead>
<tbody>
<tr>
<td>↑ maternal blood volume</td>
<td>Attenuated initial response to haemorrhage. <strong>Up to 2L blood loss may not be easily detected and 35% of blood volume may be lost before hypotension occurs</strong></td>
</tr>
<tr>
<td></td>
<td>Anaemia of pregnancy</td>
</tr>
<tr>
<td>↑ Uterine enlargement</td>
<td>Risk of aortocaval compression ⇒ 25% reduction in cardiac output</td>
</tr>
<tr>
<td>↓ FRC</td>
<td>Hypoxia from atelectasis more likely</td>
</tr>
<tr>
<td>↑ Minute ventilation</td>
<td>Compensated respiratory alkalosis, ↓ buffering capacity</td>
</tr>
<tr>
<td>↓ GI motility</td>
<td>↑ risk of aspiration</td>
</tr>
<tr>
<td>Chronic stretching of peritoneum</td>
<td>↓ signs of peritonism</td>
</tr>
<tr>
<td>↑↑↑ fibrinogen</td>
<td>Low-normal value may indicate abnormal consumption</td>
</tr>
</tbody>
</table>

Types of injury

- Before 12 weeks the small size and pelvic location of the uterus make it relatively resistant to injury. However after it becomes abdominal it is prone to injury from blunt or penetrating abdominal trauma. Perhaps more importantly women in the second and third trimesters are at increased risk of significant haemorrhage associated with uterine or pelvic trauma as a result of markedly increased uteropelvic blood flow
- 2nd & 3rd trimester bladder injuries occur frequently, because from 12weeks gestation, the bladder is an intra-abdominal organ
- It is important to recognize that, in pregnant women, assaults tend to be aimed at the uterus, increasing the risk of injury to the fetus, uterus and bladder
- Splenic injury, retroperitoneal injury and haematomas and hepatic injury are more frequent. Up to 25% of pregnant women with severe blunt abdominal trauma have haemodynamically significant hepatic or splenic injuries.
- Bowel injuries are less common
- Direct fetal injuries and fractures complicate less than 1% of cases of severe blunt abdominal trauma in pregnant women. Most of these cases occur in late pregnancy and in women with significant other injuries. Fetal head injury may result from incorrect positioning of lap belt over uterus rather than over anterior iliac spines
Uterine rupture

- uncommon
- associated with previous cesarean section and pelvic fractures
- risk factors
  - increased gestational age
  - increased force of trauma
- clinical features are variable
- rupture is frequently life threatening
  - fetal mortality nearly 100%
  - maternal mortality ~10%
- repair and management of rupture per se similar to uterine rupture from other causes

Placental abruption

- forces placed on the placental-uterine interface during blunt injury, combined with the relative inelasticity of that interface, frequently result in some degree of abruption
- occurs in up to 40% of cases of severe blunt trauma and in 2-3% of cases of otherwise minor trauma, provided it is associated with deceleration and/or uterine-directed force
- the classic clinical triad is only present in approximately 40% of cases
  - vaginal bleeding
  - abdominal pain
  - uterine irritability
- complications:
  - fetal: premature delivery, death
  - maternal: haemorrhage, DIC, death

Penetrating abdominal trauma

- the pregnant uterus may protect the other abdominal organs. Although fetal mortality is high, maternal mortality is significantly lower than in non-pregnant women
- the pattern of injury is modified, with small bowel injury being more common following upper abdominal penetrating injury than it is in non-pregnant women

Investigations

- Necessary radiological investigations as indicated
  - Radiation hazard to fetus is unlikely but appropriate shielding should be used
- Consider possibility of uterine injury in association with pelvic fractures
- Diagnostic peritoneal lavage
  - perform through a surgical incision above the fundus
- CT may miss injuries due to abdominal crowding
• Ultrasound
  o useful for assessment of abdominal injury in pregnant women
  o can accurately detect free intra-abdominal fluid, confirm gestation
    and fetal well-being and identify placental abnormalities, but its
    sensitivity for placental abruption is only 50%
  o important to exclude herniation of abdominal contents through a
    ruptured diaphragm
  o pregnancy does not appear to alter sensitivity and specificity of
    ultrasound assessment of intra-peritoneal fluid but data very
    limited
• Cardiotocography
  o indicated in all patients >24 weeks gestation
  o <8 contractions/hr associated with low risk of fetal or uterine injury
  o loss of beat to beat variability in fetal heart rate and (particularly
    late) decelerations in fetal heart rate are indicative of fetal distress
    and may indicate fetal or maternal injury
  o normal fetal heart rate: 120-160/min

Management

• The first priority is evaluation, resuscitation and stabilization of the
  mother. Most circumstances that produce maternal instability are also
  deleterious to the unborn fetus. Therefore, with few exceptions,
  treatment priorities are similar in pregnant and non-pregnant trauma
  victims. On presentation attention to potentially life-threatening maternal
  injuries should not be distracted by extensive fetal evaluation.

• Primary survey and initial resuscitation along usual lines except use
  lateral positioning or manual uterine displacement to avoid aortocaval
  compression if obviously pregnant
  o uterine blood flow is not autoregulated and may be decreased
    despite normal maternal haemodynamics, so slight
    hypervolaemia is preferable to any hypovolaemia.
  o Maternal shock is associated with a fetal mortality of 80%

After the primary survey and initial resuscitation has been completed quickly
assess the size of the uterus. At 24 weeks the uterus is usually at the level of
the umbilicus

If the uterine size does not exceed 24 weeks initial treatment should be as for a
non-pregnant patient. The pregnancy is a secondary consideration.

On the other hand, if the uterine size does exceed 24 weeks a few simple
modifications have to be made to standard initial care:

  ▪ treatment of hypotension includes lateral positioning or manual
    uterine displacement to avoid aortocaval compression
  ▪ The presence of a fetal heartbeat should be confirmed briefly
  ▪ More complete fetal monitoring and evaluation should be carried
    out after maternal resuscitation and stabilization
- Fetal assessment should be carried out as part of the secondary survey. As there is a significant risk of placental abruption this should be actively looked for. Ultrasound does not reliably detect placental abruption and cardiotocography is probably a better tool for risk assessment. Contractions occurring more frequently than once in every 10 minutes are associated with a 20% risk of placental abruption. Cardiotocography should be continued for a minimum of 4-6 hours and should probably be continued for 24h in patients with frequent uterine contractions, a non-reassuring fetal heart rate pattern, vaginal bleeding, uterine tenderness and in cases of severe maternal trauma. Until cardiotocography is available monitor fetal heart rate (normal 120-160/min)

- Most authorities do not recommend the routine use of the Kleihauer-Betke test to detect the presence of fetal red blood cells in maternal blood as CTG is a more reliable tool for acute fetal evaluation. However it is useful in Rh-negative mothers in whom it is used to identify those infrequent cases in which fetomaternal haemorrhage exceeds the 30 ml for which the standard dose of anti-D immunoglobulin prevents sensitization (NB prior to 16 weeks fetus has <30 ml of blood). 10 mcg anti-D immunoglobulin required for each ml of fetal blood.
- Anti-D immune globulin 300 mcg within 72 h of injury should be considered for all Rh D negative women even if Kleihauer-Betke test negative (routinely negative if fetomaternal haemorrhage <1 ml but even these small volumes of blood sufficient to result in sensitization)
- If vasopressors are required, ephedrine should be first choice as it preserves uterine blood flow but there should be no hesitation in using other vasopressors if and when necessary
- Place chest drains slightly higher than normal (3rd or 4th interspace)
- Tetanus prophylaxis safe in pregnancy

It is importance to realize that any surgical intervention for trauma-related injury in pregnancy is complicated by the size of the intra-abdominal uterus, the engorgement of blood vessels and the small space for the surgeon to work. Technically, it is thus more difficult to operate and control active haemorrhage.

The fetal blood supply may be easily compromised by manipulation of the uterus and uterine muscle contraction is easily initiated. Thus, surgery may induce premature labour, premature rupture of membranes and fetal loss.
CPR IN THE PREGNANT PATIENT

CPR in the pregnant patient, especially after 28 weeks gestation is different to CPR in any other type of patient, because of the effects of physiological changes of pregnancy and the presence of the fetus.

The following aspects of Basic Life Support are accepted internationally:

1. **Turn the patient onto her back with left lateral tilt**
   In a noticeably pregnant woman i.e. one with a significant intra-abdominal mass (usually by 20 weeks) it is important to obtain a left lateral tilt of the pelvis at the earliest opportunity to minimise the risk of aortocaval compression.

   ![Left Lateral Tilt](image)

2. **Open the airway**
   Check in the mouth for foreign body or material. Use suction if required or remove foreign body with careful use of forceps.
   Open the airway by gentle neck extension, chin lift and if needed, jaw thrust to displace tongue from the pharynx.
   If injury to the neck is suspected, use manual in-line stabilisation, avoid head tilt and use mainly jaw thrust to open the airway.

3. **Assess breathing (and circulation)**
   Assess breathing for no more than ten seconds by looking for chest movements, listening for breath sounds and feeling for the movement of air. Absence of breathing in the presence of a clear airway is now used as a marker of absence of circulation. Experienced staff may want to check the carotid pulse for no more than 10 seconds at the same time as assessing breathing.

   Gaspng or agonal breathing may be seen in the immediate time after cardiac arrest and should not be taken as a sign of life – it is a sign of dying and CPR should commence immediately.

4. **Start CPR**
   If no circulation (or if unsure) → give 30 chest compressions followed by 2 ventilations
   a. The position for chest compressions should be the middle of the lower half of the sternum.
   Place the heel of one hand there, with the other hand on top of the first. Interlock the fingers of both hands and lift the fingers to ensure that pressure is not applied over the patient's ribs.
   Keep in the midline at all times. Do not apply any pressure over the top of the abdomen or bottom tip of the sternum.
Position yourself above the patient’s chest and with your arms straight, press down on the sternum to depress it 4–5 cm at a rate of 100 beats per minute. Change the person doing chest compressions every 2 minutes to maintain efficiency but avoid delays in changeovers.

b. Ventilation breaths. Keep an open airway and provide ventilation with appropriate adjuncts. This might be a pocket mask, oral airway or self inflating bag with mask. Oxygen at high flow should be added as soon as possible.

c. Each ventilatory breath should last about 1 second and should make the chest rise. Tracheal intubation is the most effective way of providing adequate ventilation and should be performed as soon as a trained member of staff is available.

d. Once the patient is intubated, ventilation should continue at 10 breaths per minute but does not need to be synchronised with chest compressions. These should then be uninterrupted.

e. Mouth to mouth breathing (not usually required). Ensuring head tilt and chin lift. Close the soft part of the patient’s nose with your thumb and index finger. Open her mouth a little but maintain chin lift. Take a breath and place your lips around her mouth, making sure that you have a good seal. Blow steadily into her mouth over 1 second, watching for her chest to rise. Maintaining head tilt and chin lift, take your mouth away from the patient and watch for her chest to fall as the air comes out. Repeat the sequence to give another effective breath. Return to chest compressions quickly.

If circulation present but no breathing (respiratory arrest) → continue rescue breathing at a rate of 10 breaths/minute
Recheck the circulation every ten breaths, taking no more than ten seconds each time. If the patient starts to breathe on her own but remains unconscious, turn her into the recovery position and apply oxygen 15 litres/minute. Check her condition and be ready to turn her back to start rescue breathing if she stops breathing.

5. Use defibrillator
As soon as possible attach the defibrillator and pause briefly to assess the rhythm. The use of adhesive pads or using the paddles held over the gel pads may be quicker than attaching the ECG stickers. Follow the automated external defibrillator (AED) voice prompts or use manual defibrillation as appropriate.

Automated external defibrillator
If automated external defibrillator (AED) is available, attach, analyse rhythm and defibrillate as indicated in the applicable algorithm.

The most frequent initial rhythm in the context of sudden collapse (i.e. not preceded by gradual deterioration or illness) is ventricular fibrillation (VF). The chances of successful defibrillation diminishes with time. The AED allows for early defibrillation by lesser-trained personnel, as it performs rhythm analysis, gives information by voice or visual display and the delivery of the shock is then delivered automatically.

Turn immediately to advanced life support algorithm
When advanced life support arrives, the rhythm is assessed as a shockable rhythm or nonshockable rhythm and defibrillation is instituted if required. An airway is secured and intravenous access obtained.
Defibrillation sequence and use of drugs can be followed on the algorithms.

Shockable rhythms:
Shockable rhythms are treated with a single shock followed by immediate continuation of CPR without stopping for a rhythm or pulse check
Every 2 minutes the rhythm should be assessed and if necessary a further shock delivered.
The pulse is not checked unless there is organised electrical activity i.e. something which looks as though it might produce an output.
The energy used for defibrillation depends on whether it is a monophasic or biphasic defibrillator. Most modern defibrillators are biphasic as this is the most efficient way of delivering energy. The charge needed is therefore lower than on the older monophasic machines.

The initial and subsequent shocks should be 150 - 200J from a biphasic machine or 360J from a monophasic machine.

On the shockable side of the algorithm, adrenaline (epinephrine) 1mg IV is given immediately before the 3rd and every subsequent alternate shock i.e. approximately every 4 minutes. Amiodarone 300mg IV is given before the 4th shock.

**Non shockable rhythms:**

On the non-shockable side of the algorithm i.e. pulseless electrical activity or asystole, adrenaline (epinephrine) 1mg should be given as soon as intravenous access is available. Atropine 3mg IV may be given once for asystole or slow rate i.e. <60 bpm. This will minimise any vagal tone if present.

Reversible causes of cardiac arrest are considered and treated as necessary. Those highlighted are most common causes of cardiac arrest/collapse in pregnancy.

**Four H’s:**
- hypoxia
- hypovolaemia (haemorrhage or sepsis)
- hyperkalaemia and other metabolic disorders
- hypothermia

**Four T’s:**
- thromboembolism
- toxicity (drugs associated with regional or general anaesthesia)
- tension pneumothorax
- cardiac tamponade

**Doubt about the rhythm**

If there is doubt about whether the rhythm is asystole or fine VF, CPR should be maintained and treat as for asystole.

**Other drugs**

*Sodium bicarbonate: 50mmol IV* should only be given to patients if the arrest is associated with tricyclic antidepressant overdose or hyperkalaemia. Otherwise it is given in response to the clinical condition of the patient e.g. severe acidosis pH < 7.1 or base excess > -10.

*Magnesium sulphate: 8mmol* (4 mls of 50% solution) may be used for refractory VF. Other use may be in possible hypomagnesaemia, torsade de pointes (a persistent VF) or digoxin toxicity. These are unlikely in pregnancy.

*Calcium: 10ml 10% calcium chloride* (6.8 mmol Ca²⁺) IV can be used if it is thought that PEA is caused by hyperkalemia, hypocalcemia, overdose of calcium channel blocking drugs or overdose of magnesium (for treatment of pre-eclampsia). Calcium can be given as a bolus if the patient has no output, but not in the same line as sodium bicarbonate as this will precipitate.

**PHYSIOLOGICAL CHANGES IN PREGNANCY AFFECTING RESUSCITATION**

There are a number of reasons why the processes of cardiopulmonary resuscitation are more difficult to perform and may be less effective in the pregnant than in the non-pregnant population. When these changes occur is not precise, but gradually the presence of increasing mass in the abdomen compromises resuscitative efforts. This may be the case from 20 weeks but will be more marked as the mother approaches term.
**Vena caval occlusion**
At term, in a well woman, the vena cava is completely occluded in 90% of supine pregnant patients and the stroke volume may be only 30% of that of a non-pregnant woman. As soon as the infant is delivered, the vena cava returns towards normal and adequate venous return and consequently cardiac output is restored.
During cardiac arrest, in order to minimise the effects of the gravid uterus on venous return and cardiac output, a maternal pelvic tilt to the left of greater than 15 degrees is recommended. The tilt needs to be less than 30 degrees for effective closed chest compression to take place.
An alternative, manual displacement of uterus to the left should be effective.
Delivery of the fetus during cardiac arrest will reduce the oxygen demands on the mother and also increase the venous return to the heart making it more possible that resuscitation will be successful (see later).

![Manual displacement of the uterus to the left](image)

**Changes in lung function**
Mothers become hypoxic more readily because of a 20% decrease in their functional residual capacity due to the pressure from the gravid uterus on the diaphragm and the lungs i.e. there is less of a reservoir of oxygen in the lungs so they become hypoxic much more quickly. This is exacerbated by a 20% increase in their resting oxygen demand due to servicing the needs of the fetus and uterus.
These changes make it difficult to provide enough oxygen delivery using CPR to resuscitate a near term pregnant mother.

**Effectiveness of ventilation**
In the later part of pregnancy it becomes increasingly difficult to provide effective ventilation breaths during CPR due to the increased weight of the abdominal contents and the breasts.
In addition the oesophageal sphincter is more relaxed so the ease of introducing air into the stomach is increased.
Passive regurgitation of stomach contents is a very real concern as these are greater in volume and more acidic in pregnancy so more likely to lead to damaging acid aspiration into the lungs.
It is imperative that experienced staff provide a protected airway and adequate ventilation via an ET tube as quickly as possible following cardiac arrest.
PERIARREST / PERIMORTEM CAESAREAN SECTION TO IMPROVE
CHANCES OF MATERNAL SURVIVAL

The Resuscitation Council for special situations has recommended that prompt caesarean
delivery should be considered as a resuscitative procedure for cardiac arrest in near-term
pregnancy.

Delivery of the fetus will obviate the effects of aortocaval compression and significantly improve
the chance for maternal resuscitation. This will reduce maternal oxygen consumption, increase
venous return, make ventilation easier and allow CPR in the supine position.

When to do it
Evidence from literature and review of maternal and fetal physiology suggests that a caesarean
delivery should begin within four minutes of cardiac arrest and delivery be accomplished
by five minutes.

Pregnant women develop anoxia faster than non-pregnant women and can suffer irreversible
brain damage within four to six minutes after cardiac arrest.

When a mother in the second half of her pregnancy suffers a cardiac arrest, immediate
resuscitation should commence. Should immediate resuscitation fail, every attempt should be
made to start the caesarean section by four minutes and deliver the infant by five minutes.

CPR must be continued throughout the caesarean section and afterwards, as this increases the
chances of a successful neonatal and maternal outcome.

Where to do it
Moving the mother to an operating theatre (e.g. from a labour room or accident and emergency
department) is unnecessary. Diathermy is not be needed initially, as there is little blood loss if
there is no cardiac output. If the mother is successfully resuscitated, she can be moved to a
theatre to complete the operation.

How to do it
A limited amount of equipment is required and sterile preparation and drapes are unlikely to
improve survival. A surgical knife and forceps should be sufficient to effect delivery of the baby.

There are no recommendations regarding the surgical approach for caesarean section but
there is no doubt that the classical approach is aided by the natural diastasis of recti abdomini
that occurs in late pregnancy and a bloodless field in this clinical situation.

It is accepted, that operators should use the technique with which they are most
comfortable, and most obstetricians can deliver a baby via a routine approach in less than a
minute.

Consider open cardiac massage during Caesarean section when the abdomen is already open
and the heart can be reached relatively easily through the diaphragm.

It is important that an anaesthetist is in attendance at the earliest opportunity. The anaesthetist
should provide a protected airway, ensure continuity of effective CPR and help determine and
treat any underlying cause (see 4 H's and 4 T's).

Should the resuscitation be successful and the mother regains a cardiac output, appropriate
sedation/general anaesthetic needs to be administered to provide amnesia and pain relief and
the mother should be moved to a theatre to complete the operation.

Fetal outcome
Timing of delivery is also important for the survival of the infant and its normal neurological
development.

There is no doubt that uterine evacuation is an important step during maternal resuscitation.
However, there seems to be reluctance among obstetricians to perform peri-arrest caesarean
sections. Concerns include worries about neurological damage to the delivered infant.

In a comprehensive review of postmortem caesarean deliveries between 1900 and 1985 by
Katz et al., 70% (42/61) of infants delivered within five minutes survived and all developed
normally.

However, only 13% (8/61) of those delivered at 10 minutes and 12% (7/61) of infants
delivered at 15 minutes survived.

One infant in both of these groups of later survivors had neurological sequelae.
While the optimal interval from arrest to delivery is five minutes, there are case reports of intact infant survival after more than 20 minutes of maternal cardiac arrest. Review of postmortem caesarean section, as reported in Confidential Enquiries over the past 25 years, shows that there was no reported case where survival beyond the early neonatal period was accompanied by neurological disability.

Evidence suggests that if the fetus survives the neonatal period then the chances of normal neurological development are good.

**Make decision to abandon CPR if unsuccessful**
Do not abandon CPR if rhythm continues as VF/VT.
A decision to abandon CPR should only be made after discussion with the consultant obstetrician and senior clinicians.

**Medico-legal issues**
No doctor has been found liable for performing a postmortem caesarean section. Theoretically, liability may concern either criminal or civil wrongdoing. Operating without consent may be argued as assault or battery if the mother is successfully resuscitated. However, the doctrine of emergency exception would be applied because a delay in treatment could cause harm. The second criminal offence could be 'mutilation of corpse'. An operation performed to save the infant would not be wrongful, because there would be no criminal intent. The unanimous consensus of the literature is that a civil suit for performing perimortem caesarean is very unlikely to succeed.

**Communication and teamwork**
Wherever possible, have senior input from the obstetric, anaesthetic and midwifery professions from early on as possible.
Ensure that the family is looked after and kept informed.
Document timings and interventions accurately.
If the mother dies, you will need to report the death as a maternal death and possibly inform the SAPS and Forensics unit.

- Where the cause of death is obviously of natural reasons (eg eclampsia with cerebrovascular bleed), SAPS and forensics will not be involved, but an autopsy should still be performed if at all possible. (The utmost should be done to see if this can be arranged, because the information is important for the triennial Saving Mothers Report)
- Where there is any possibility of an unnatural cause, eg assault, MVA, suicide or where death occurs perioperatively, SAPS and Forensics must be involved.
ASPIRATION AND ASPIRATION PNEUMONITIS

The incidence of aspiration in a large multicentre study of elective general surgical patients in France was found to be 1.4:10 000 anaesthetics and 50% of these occurred post-operatively. Other similar studies in other countries have found an incidence ranging from 1.6-6 per 10 000 anaesthetics.

This apparently low incidence, which translates roughly to 1 case of aspiration every 2131-6250 anaesthetics, changes dramatically in emergency surgery (1:895) and in obstetric patients (15:10 000), ie 1 per 667 cases.

In one study, 36% of those who aspirated developed symptoms (decreased O$_2$ saturation, cough, wheeze or CxR infiltrates) within 2hrs. Half of these required mechanical ventilation, mostly <24hrs. The overall mortality of those who aspirated was 5%.

Obstetric patients have an obviously greater risk for aspiration and this requires greater caution and meticulous attention to detail when a general anaesthetic is required.

GENERAL RISK FACTORS FOR ASPIRATION

- ASA level >2
- Depressed level of consciousness – hypotension, hypoxia, hypercapnia
  - anaesthesia, ethanol, drug overdose
  - trauma, epilepsy, cardiac arrest, CVA
- Impaired airway reflexes
  - drugs (CNS, NMJ blockers, airway blocks)
  - intubation/extubation
  - elderly
- Increased regurgitation
  - pregnancy
  - hiatus hernia
  - LOS(lower oesophageal sphincter) dysfunction
  - obesity
  - NG tube
  - bowel obstruction
  - delayed gastric emptying
- Systemic illnesses
  - diabetes
  - chronic renal failure
  - uremia
  - hypothyroidism

In many books, an at risk patient has been defined as having a gastric volume of more than 0.4 ml/kg ~ 25 ml and a gastric pH < 2.5. These limits were based on a paper by Roberts and Shirley (1974), who used unpublished data from Rhesus monkeys and extrapolated this to humans.

More recent work found that a much greater volume (~ 0.8 ml/kg) was required to produce classic Mendelson's syndrome, but these animal models involved the direct installation of acid into the lungs.

Other studies of non-lethal aspiration support that pH and not volume is the more important factor.
PHYSIOLOGY
The physiology of swallowing, regurgitation and vomiting is very complex and controlled by multiple brain centers. Normally, if all reflexes are intact, aspiration does not occur.

Aspiration is dependent on there being a considerable gastric volume of low pH and decreased rate of gastric emptying.

Factors that delay gastric emptying:
- hyper/hypo-osmolar contents
- high calorie solids > low calorie solids > liquids
- acid within the duodenum & cholecystokinin
- gastrin, motilin and parasympathetic agents which increase emptying
- pain, anxiety, opioids and labour
- disease states, ie. inflammatory bowel disease, diabetes, hypothyroidism, peptic ulcer disease, electrolyte disorders.

Generally, gastric secretions continue at 50ml/hr and saliva is swallowed at 1ml/kg/hr. Gastric emptying of fluids is rapid (1-2hrs) and solids slower at 4-6hrs under normal conditions. Gastric emptying in pregnancy is significantly delayed and >50% of patients in labour have a significant volume of particulate matter 8-24hrs after a meal. The residual gastric volume for particulate contents in general surgical patients and obstetric patients not in labour do not seem to be significantly different at 6hrs after a solid meal.

In pregnancy, the normal physiology is deranged and the risk for aspiration is increased by multiple factors:
- Delayed gastric emptying - displacement of the pylorus
  - increased progesterone (antagonizes motilin)
  - labour pain, anxiety, narcotic analgesics

- Increased gastric acidity - placental gastrin secretion

- Decreased LOS tone - loss of gastro-oesophageal angle
  - narcotic analgesics
  - anticholinergics

- Increased intragastric pressure - mechanical effect of the uterus
  - lithotomy

It is controversial as to when these changes start in pregnancy and when they resolve postpartum, but it is generally accepted that a pregnant patient must be considered at high risk from 12 weeks gestation until 2 weeks postpartum.
Thus any general anaesthetic given during this period should be done with full precautionary measures in place, ie chemoprophylaxis and rapid sequence induction technique (see later).

PREVENTION OF ASPIRATION
1. **Nil per os rule** - it is prudent for elective obstetric procedures to be starved for solids for at least 6hrs and for clear fluids for at least 2hrs.
Patients in labour with the possibility of a Caesarean section (eg trial of labour or trial of scar) should be kept off solids, but be allowed clear fluids.
2. **Antacids** - it should be pointed out that antacids only raise the pH of the gastric contents, but do nothing to protect against particulate matter aspiration. Particulate antacids (Mg-trisilicate) are to be avoided where at all possible, because of their propensity to cause granulomatous infiltrations in the lung if aspirated. Non-particulate antacids(sodium citrate) rapidly raise the pH, last for 1-3hrs and have no effect on the lung if aspirated.

3. **H₂-receptor blockers** - all of the drugs in this group (eg cimetidine, ranitidine) effectively reduce gastric acidity and volume of secretions, but need 45-60min to act if given IV/IM and 1-2 hrs if given orally. They have no effect on contents already present in the stomach. Routine use in obstetric patients is not indicated, except in patients who have a history of severe reflux/peptic ulcer disease.

4. **Metoclopramide** - acts both centrally and peripherally to increase gastric emptying and increase LOS tone while decreasing pyloric tone. As an anti-emetic, it is a poor choice. It probably has a place in obstetric patients who are morbidly obese or who have recently eaten.

5. **Head-up position**

6. **Avoid general anaesthesia**

7. **Rapid sequence induction technique** - in obstetric patients for GA, this technique is mandatory. This should be meticulous and you should have a practiced, smooth technique. A suggested protocol for rapid sequence induction is given below.

8. **Suction** the stomach contents after intubation.

9. **Awake extubation**

10. **Alert, well-trained recovery staff**

**CLINICAL PRESENTATION OF ASPIRATION**

Classically, aspiration occurs in an at risk patient (often unrecognised) where cricoid pressure was not applied or incorrectly applied or an unprotected airway used. Some time into the procedure, usually 20-30min, airway pressures increase, the patient becomes hypoxic and difficult to ventilate, the lung sounds become noisy and there is wheeze or severe bronchospasm.

The event of aspiration more often occurs when the anaesthetic is poorly conducted, rather than as a result of risk factors.

Most cases of aspiration on the theatre table occur when the patient is not deep enough for airway manipulation and/or there is insufficient paralysis.

Remember, a paralysed patient cannot vomit (an active reflex), but may regurgitate passively.
MANAGEMENT OF ASPIRATION

- Once aspiration occurs or is suspected, secure the tube, but avoid ventilation which might force further contents into the trachea.
- Place patient head down and suction as much contents as possible away from the glottis.
- Make an effort to remove any food or particulate matter from the trachea.
- Identify the contents – are they fluid only, or particulate?
- Ensure oxygenation.
- Suction the stomach to remove all remaining contents.
- Do not flush the trachea with saline or alkaline fluids.
- If available, get somebody to perform a bronchoscopy to ensure all particulate matter is removed. If not available, do not worry.
- There is no proven place for the use of steroids and/or antibiotics in the acute phase.
- At the end of the procedure, if the patient is able to manage themselves on <40% O₂, do an awake extubation and monitor the patient in the recovery room for the next 2hrs. (Otherwise keep intubated, ventilate and refer to a high care/ICU)
- Get a mobile CxR in the interim.
- If, after 2hrs, the patient is asymptomatic and the CxR appears clear, discharge from the recovery room and give the patient supplemental O₂ if required in the ward for 6-12 hrs. Reassess and discharge if stable.
- If the CxR shows infiltrates, or the patient has mild symptoms, but is not hypoxic on room air or up to 40% O₂ by mask, send to the ward on supplemental O₂ and monitor with pulse oximetry for up to 48hrs. Reassess every 4hrs. Check vital signs, T° 4hly and do daily CxR and WCC. Discharge if condition remains improved after 48hrs.
- If the patient’s condition worsens, intubate, ventilate and refer to a high care/ICU.

THE RAPID SEQUENCE INDUCTION TECHNIQUE (RSI)

It is very important to know exactly what this technique means, what your endpoint is and thus by implication what it doesn’t mean.

The RSI is a technique applied to achieve a definitive airway in a patient at risk for aspiration of gastric contents, in the shortest time and safest way possible. This does not equate to induction agent – muscle relaxant – tube in the shortest period possible!! On the contrary, this shotgun approach might just result in aspiration.

Despite the pressure often exerted on the anaesthetist to “get the tube down” as soon as possible in an emergency, it is your job intubate the patient in a controlled manner, at the same time safeguarding the patient’s/mother’s airway and maintaining cerebral oxygenation. This is attained by:

- **“Pre-oxygenation”** - this is a misnomer, because most patients already have an almost fully saturated Hb on room air. What you achieve by the so-called pre-oxygenation is actually denitrogenation of air in the lungs. By replacing the air with 100% O₂, you wash out the nitrogen, thus filling up the FRC (functional residual capacity) of the lungs with 100% O₂, instead of only 21% O₂. This buys you precious time to use for intubation.
Effective denitrogenation is achieved by 100% $O_2$ at 8l/min flow applied with a tight-fitting face-mask with the patient breathing at least 8 breaths per min, for 3-5min. Alternatively, in the acute situation, the same can be achieved with the patient taking at least 4 full vital capacity (VC) breaths in 1min.

- **Induction** with a sleep-dose of an induction agent, not a titrated dose.

- **Cricoid pressure** is applied as the patient falls asleep. This is maintained until the ETT has been inserted and confirmed to be in the correct place (see below).

- **Effective muscle relaxation** must be rapidly obtained. The drug of choice is suxamethonium (Scoline) in a dose of 1-1.5mg/kg, given as a bolus at the same time as cricoid pressure is applied. If Sux is contra-indicated in this patient, rocuronium (Esmeron) must be given in a dose of 0.9-1.0mg/kg ideal body mass. Both of these drugs should result in effective muscle relaxation within 40-60s. The downside of using rocuronium in this dose is the extended time of relaxation (± 60min) needed before reversal can be considered.

  When using Sux, it is important to time your bolus as well as look for fasciculations, because 30% or more of patients will not have visible muscle fasciculations. Wait for the fasciculation to end before attempting intubation.

  Do not attempt to mask ventilate your patient during the time you wait for effective muscle relaxation to be established, otherwise the stomach may inflate with air and increase the risk of aspiration. Some experienced anaesthetists will consider some mask ventilation, but closely monitor their generated airway pressures.

- **Endotracheal intubation with a cuffed ETT** should be performed as smoothly and slickly as possible. Cricoid pressure must be maintained until confirmation of correct position of the ETT.

  There are only two foolproof ways of confirming tracheal intubation:

  1. the direct visualization of the cuff entering the vocal cords and
  2. a capnograph tracing for at least 6 ventilated breaths.

  Listening for equal breath sounds and looking for chest movement is mandatory, but is not fully reliable as confirmation of tracheal intubation. Note that pulse oximetry readings are a poor indicator, because changes occur late, especially if effective denitrogenation has been obtained.

- **Fasten the tube and release cricoid pressure**

- **Awake extubation**, if needs be in a head down position and with the patient lying on the left side.

  - Regard mothers to be at high risk for aspiration from 12 weeks gestation until 2weeks postpartum
  - Any general anaesthetic given during this period should must get antacid chemoprophylaxis and rapid sequence induction technique
PREGNANCY INDUCED HYPERTENSION AND ECLAMPSIA

Hypertensive conditions of pregnancy (Pregnancy induced hypertension and eclampsia) remain a severe disease and a main cause of maternal morbidity and mortality worldwide, South Africa being no exception.

In the 2002-2004 three year period in South Africa, hypertensive disorders of pregnancy were the second most frequent cause of maternal death, with 628 recorded maternal deaths - most of these presented as emergencies!!

You will undoubtedly be faced with these hypertensive conditions, as elective, semi-urgent and emergency cases. Correct management of these cases is vital to reduce maternal morbidity and mortality.

DEFINITIONS:

Pre-eclampsia is regarded as the development of a raised blood pressure of >140mmHg systolic and/or 90mmHg diastolic plus proteinuria (>300mg/24hrs) after 20 weeks gestation.

Severe pre-eclampsia is characterized by systolic blood pressure exceeding 160mmHg and/or diastolic blood pressure exceeding 110mmHg, together with proteinuria (>5 g/24 h).

It can be accompanied by symptoms or signs of imminent eclampsia:
- Headache
- Visual disturbances (flickers)
- Epigastric pain
- Hyperreflexia

or may be complicated by pulmonary oedema, oliguric renal dysfunction/failure or HELLP syndrome (haemolysis, elevated liver enzymes, and low platelets).

Anaesthetists not only have to provide safe labour analgesia and anaesthesia for caesarean section in these patients, but play pivotal roles in their resuscitation and intensive care management. In larger centres, anaesthetists also provide invasive monitoring and clinically manage these patients to limit the cardio-respiratory, cerebral and renal complications.

To perform this function, an up-to-date understanding of the pathophysiology of the disease is vital, as well as the clinical application of internationally accepted guidelines based on recent studies.

A summary of up-to-date understanding of the pathophysiology:
The key pathophysiological feature in pre-eclampsia is vascular endothelial dysfunction, which results in damage to the placenta, brain and kidney. Incomplete trophoblastic invasion of the spiral arteries during the first trimester causes placental ischaemia in the 2nd trimester, due to high pressure perfusion of the placenta. This ischemia results in the release of inflammatory mediators, causing leukocyte activation and resulting in the endothelial dysfunction.

Through this systemic inflammatory response the normal vasodilatory condition of pregnancy is disrupted and a constant state of vasoconstriction is brought about by the combined effect of deficient prostacyclin (vasodilator), and an excessive production of
thromboxane (potent vasoconstrictor). This leads to the concept that these patients are intravascularly dehydrated and thus require volume expansion.

Dyslipidaemia is common in pre-eclampsia and this has been associated with oxidative stress and possibly genetic susceptibility. Immune mechanisms may also play a role in the development of PET, but this has not yet been proven.

**RISK FACTORS FOR DEVELOPING PRE-ECLAMPSIA**

- Primigravidas, both young (<20yrs) and old (>35yrs)
- Multiple pregnancies (bigger placenta)
- Diabetes (type1, type2 and gestational diabetes)
- Obesity
- Pre-existing hypertension
- Renal disease
- Hydatid mole
- Connective tissue diseases, esp SLE

**PREVENTION OF PRE-ECLAMPSIA**

A wide variety of agents have been studied as preventative therapy, with little success. The only agent having convincing evidence of reduction of the incidence of PET is the use of low-dose aspirin, preferably started between 12 and 20 weeks gestation. Aspirin inhibits the excessive production of thromboxane and prevents platelet aggregation. It should be stopped at 36wks gestation.

Only magnesium sulphate has been shown to be advantageous in preventing the progression of severe pre-eclampsia to eclampsia.

**MANAGEMENT OF PRE-ECLAMPSIA**

The cornerstones of management of PET are:
- Controlling the blood pressure
- Preventing and treating convulsions
- Fluid therapy
- Expediting delivery

**CONTROLLING THE BLOOD PRESSURE**

The aim should be to maintain the mean arterial pressure between 100 and 140mmHg, which in practice means to keep the BP between 130/90mmHg and 170/110mmHg. It is usually not appropriate to reduce blood pressure acutely by > 30/15mmHg in severe cases or to < 90mmHg diastolic generally, because of the reduction in placental perfusion that occurs. An acute reduction could thus precipitate fetal distress.

Initial treatment is strict bedrest, left lateral position and institution of oral anti-hypertensives – usually methyldopa (Aldomet/Hypotone) up to 1g tds. The aim is to stabilize the BP to protect the mother from complications of severe hypertension and allow the fetus to mature.

Acutely high blood pressures may be reduced by the use of oral fast-acting Nifedipine (Adalat®) 5mg, but as a bite-and-swallow approach, not as sublingual administration. This may be the drug of choice in smaller facilities where IV drugs/infusions cannot be given or effectively monitored.
Blood pressures greater than 170/110 or those that fail to settle require IV antihypertensives. The aim is to achieve a rapid but smooth reduction in blood pressure to acceptably high levels, as suggested above. Especially IV hypertensives must be preceded and accompanied by volume expansion, to reduce acute BP drops or wide swings.

**IV drugs to use are:**

Labetolol (Trandate®, an \(\alpha\)– and \(\beta\)-blocker) in doses of a slow bolus of 20mg over 10min repeated every 10min; some advocate incremental doses of 20, 40, 60, 80mg at 10 minute intervals until control is achieved (until a maximum cumulative dose of 200mg) followed by a maintenance infusion.

Dihydrallazine (Nepresol®, a direct acting vasodilator) 5mg slow bolus over 5min every 20min up to a maximum of 20mg, followed by an infusion of 5-20mg/hr. NB! The onset time of dihydrallazine is slow, so care must be taken not to be too hasty between doses. Volume expansion prior to initiation of the drug is important to prevent acute severe hypotension.

➢ **PREVENTING AND TREATING CONVULSIONS**

Magnesium sulphate is unequivocally the drug of choice for both preventing and treating eclamptic convulsions. This is supported by the results of the large multicentre randomized trial called the MAGPIE trial (Magnesium Sulphate for Prevention of Eclampsia).

Magnesium sulphate reduces the relative risk of developing eclampsia by 50% as well as reducing maternal death. Magnesium sulphate is a potent catecholamine antagonist, is a physiological calcium antagonist, stabilizes cell membranes and is a cerebral vasodilator. These actions of magnesium reduce the cerebral vasospasm, hypoxia and vasogenic oedema presently accepted to be the underlying pathophysiology of this condition.

There are 2 commonly accepted intramuscular and intravenous dosage regimes:

The first is the **intramuscular regime** – it is used where nursing expertise or facilities are lacking for managing IV infusions. It consists of an initial 4g slow IV bolus (4g in 100ml saline over 20min) combined with 10g deep IMI, then followed by 5g deep IMI every 4hrs. Remember that IM magnesium injection is painful and must be deep. The formulation is 1g/2ml, so volumes are large and should be divided. Sites of injection should be intragluteal or the lateral thigh only. A personal preference is to draw up the magnesium with 1-2ml of lignocaine before administering IM if this regime is used.

The **intravenous regime** is the preferable regime, but nursing expertise and facilities for managing and monitoring IV infusions must be available. It consists of the same 4g slow IV bolus as in the IMI regime, but is then followed by a constant infusion of 1-3g/hr through an IVAC. This is obtained by adding 20g (ie 40ml) magnesium sulphate to 200ml saline and infusing at 12-36ml/hr (12ml=1g).

Although magnesium sulphate is a very safe drug, it may have severe side-effects with overdose or if plasma levels reach toxic levels due to renal dysfunction, as may occur in PET patients. Common side-effects are flushing and nausea and vomiting; in volume-depleted patients, hypotension, tachycardia and dysrhythmias may occur.
Normal plasma levels are 0.75-1mmol/l and target plasma levels for therapy are 2-4mmol/l. Magnesium presynaptically inhibits neurotransmitter release at nerve endings, including the neuromuscular junction and it exerts its own neuromuscular blocking effect at plasma levels above 5mmol/l. This can be seen clinically by the depression of the patellar tendon reflex, so this tendon reflex remains the clinical test for early onset of toxicity and should be tested for hourly. At plasma levels above this range, respiratory depression, myocardial depression and CNS depression develop in that order.

Other effects of magnesium of importance to the anaesthetist are:
- It is highly effective at suppressing the intubation reponse, especially when combined with an effective dose of opiates. This is very important in PET patients – see later.
- It has a depressive effect on the neuromuscular junction, thus making patients sensitive to and prolonging the effect of nondepolarizing neuromuscular blockers. Decreased dosing of muscle relaxants and careful monitoring of neuromuscular function is essential. There is no prolonged effect of scoline, but fasciculations are frequently not seen when it is used during magnesium therapy (see later).

**FLUID THERAPY**

Fluid management of these patients is both controversial and complicated. We know that these patients are intravascularly volume depleted and that volume expansion appears to benefit them by promoting maternal and uteroplacental circulation and reducing hypotension with vasodilator therapy, without increasing the blood pressure.

However, these patients are hypoalbuminemic, have endothelial dysfunction and severe cases also have left ventricular dysfunction, thus they are prone to pulmonary oedema and cerebral oedema. Additionally, the effect of plasma expansion is transient, especially when crystalloids are used.

The use of a CVP as guide to fluid therapy is also debatable, because of the poor correlation between CVP values and pulmonary capillary wedge pressure. Some clinicians will use the trend values of the CVP by measuring the response of the CVP value to a known fluid load. This allows the clinician to determine the ability of the ventricle to manage the fluid load in an attempt to reduce the risk of pulmonary oedema.

In larger centres, Swan-Ganz catheters (pulmonary artery catheters) are used in severe cases to monitor pulmonary capillary wedge pressures directly.

**So what does all this mean for our clinical management?**

It is acceptable and even prudent to institute some degree of volume expansion in these patients, with full awareness of the risks of fluid overload. The debate over the most suitable fluid ie crystalloid versus colloid, is never-ending and fluid administration guidelines are virtually non-existent. The fluid administration guidelines below are not based on any literature studies and are not rigid. Fluid therapy must be tailored for each patient according to your clinical assessment.
On a practical note, it is probably wise to start with a crystalloid like Ringers lactate and give 500ml-1000ml over the course of an hour. Where spinal anaesthesia is to be performed, this volume is given over 10-20min. Thereafter give a colloid like Voluven or Gelofusine at 100ml/hr. 500ml of a colloid can then be alternated with 1000ml of a crystalloid at the same maintenance rate. This would give a maximum administered volume of 2900-3400ml in the first 24hrs, including the initial bolus.

If further fluid is required, 200ml intermittent boluses of the alternate fluid may be given over 20min additional to the maintenance.

The tendency, however, is to apply a more restrictive fluid policy than would be the case in other conditions. Frequent clinical assessment of the lungs for basal crepitations is mandatory. Reduced fluid volumes should be used in patients with renal dysfunction or severe hypoalbuminemia.

The times when volume expansion is most beneficial would be with the institution of vasodilator therapy, immediately prior to delivery and as coloading with insertion of spinal, epidural or combined spinal-epidural anaesthesia.

- **EXPEDITING DELIVERY**

The decision to deliver is usually made between the obstetricians and paediatricians, but in severe cases, the anaesthetist may also have to give input, because the patient is often at her most critical at this time. In small hospitals, where manpower is usually at critical levels and these patients present as emergencies, the medical officers involved are often “all-rounders” and inevitably take these decisions together.

The decision to deliver is usually an attempt to balance fetal viability and maternal morbidity. Although this is true, the anaesthetist’s primary concern is the welfare of the mother.

Care must be taken, especially in emergency cases, to properly assess the mother and get her condition as optimal as possible in the short time available.

In mild and moderate PET, normal labour may continue.

In severe PET, the quickest method of delivery is the chosen one. This is usually a Caesarean section.

**SPINAL ANAESTHESIA OR GENERAL ANAESTHESIA??**

The choice between these techniques depends on a number of factors, one of the most important being the experience and expertise of the anaesthetist. The ideal is to be sufficiently experienced in both techniques, but this is often not the case, especially in junior doctors.

Worldwide there is a loss of experience in general anaesthesia for obstetrics due to the widespread use of neuraxial techniques.

On the other hand, in South Africa’s rural areas, many of the GP’s have years of experience in general anaesthesia and do not perform neuraxial techniques.

Often, general anaesthesia is the only approach to take in critically ill obstetric patients, including patients with severe PET, imminent eclampsia, post-ictal patients and those with coagulation abnormalities (DIC, thrombocytopenia).
There are advantages/ disadvantages for both approaches in patients with PET:

<table>
<thead>
<tr>
<th>FACTOR</th>
<th>REGIONAL ANAESTHESIA</th>
<th>GENERAL ANAESTHESIA</th>
</tr>
</thead>
<tbody>
<tr>
<td>AIRWAY</td>
<td>No intubation response or risk of failed intubation</td>
<td>No control of the airway</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Control of the airway</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Exaggerated intubation response and increased risk of failed intubation</td>
</tr>
<tr>
<td>CONVULSIONS</td>
<td>None</td>
<td>Risk of convulsions</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Active control</td>
</tr>
<tr>
<td>DRUGS AND TECHNIQUE</td>
<td>No sedative drugs</td>
<td>Risk of convulsions</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Risk of high block</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Maternal awareness and fetal depression</td>
</tr>
<tr>
<td>SPEED</td>
<td>Spinals quick, 5-10min</td>
<td>Epidural slow, 20-30min</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Fast, &lt;5min</td>
</tr>
<tr>
<td>BP CONTROL</td>
<td>Less instability, Lower catecholamines</td>
<td>Risk of hypotension</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Less hypotension</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Increases in wedge pressure, CVP and arterial BP on intubation; increase in catecholamines</td>
</tr>
<tr>
<td>COAGULATION</td>
<td>No airway instrumentation</td>
<td>Risk of spinal hematoma</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Avoids spinal hematoma</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Risk of airway haemorrhage</td>
</tr>
</tbody>
</table>

**SPINAL ANAESTHESIA CONCERNS IN PET**

The same concerns for spinal anaesthesia as discussed in the chapter Anaesthesia for Caesarean section, are applicable here.

**Additional important concerns are:**

**SEVERE HYPOTENSION**

There has long been resistance to performing spinal anaesthesia for PET patients, on grounds of causing severe hypotension. Although hypotension occurs in these patients, recent trials indicate that spinal anaesthesia does not decrease placental perfusion. Cardiac output appears to remain stable and may even improve due to the decrease in systemic vascular resistance.

Rapid co-loading of crystalloid fluid (Ringers) in the order of 20ml/kg offsets hypotension. Aggressive early management of hypotension with bolus doses of ephedrine or phenylephrine also reduce these concerns. Patients who are stable on vasodilator anti-hypertensives also appear to be more stable during spinal anaesthesia than those who are untreated. There does not appear to be more hypotension in patients on magnesium therapy.

**THROMBOCYTOPENIA**

There is no fixed platelet count at which spinal anaesthesia is specifically contra-indicated, but there are internationally accepted guidelines.

In patients with PET, thrombocytopenia may occur alone, as part of the HELLP syndrome or secondary to HIV. Platelet quality, ie platelet function, is definitely impaired in these conditions, which further complicates this issue.
Platelet counts below 100 are of some concern, but are completely acceptable, both for the purposes of surgery and spinal/epidural blocks. Despite the lack of literature, present guidelines, however, do not support spinal anaesthesia in patients with platelet counts below 70. As with all arbitrary numbers, there may be exceptions to the ‘rule’. In this case, where faced with the choice between an obviously high-risk airway and the small risk of a spinal hematoma from a single-shot spinal due to thrombocytopenia, the calculated risk is to do the spinal. If ever in doubt, contact your nearest specialist anaesthetist for advice.

GENERAL ANAESTHESIA CONCERNS IN PET

It is obvious that all of the concerns for general anaesthesia in obstetric patients already discussed in the chapter Anaesthesia for Caesarean section, eg difficult airway, preoxygenation and rapid sequence induction, are just as relevant in these patients. However, there are some important additional concerns for patients with PET.

AIRWAY
The airway may be swollen and oedematous and a difficult intubation risk is high, although in practiced hands, failed intubation is infrequent. Predicting airway oedema is difficult, but suspicion must be very high when there is facial swelling and especially if there are phonation changes. Patients may sound hoarse or Donald Duck-like and it is a good principle to question the patient specifically about any recent voice change.

Other indictors of difficult intubation are post-ictal patients with mucosal bleeds or tongue lacerations and (morbid) obesity.

In PET patients, then, it is mandatory to have all of the facilities at your disposal for difficult airway management, ready for every case.

LARYNGOSCOPY AND THE INTUBATION RESPONSE
The brain is one of the principal target organs in PET and it is in a state of vasospasm, hypoxia and possibly oedema at the time of surgery. There is a high risk of cerebral bleed and/or hypoxic infarct due to the hypertensive response to laryngoscopy and intubation and this response must be blunted at all costs.

One trial conducted to compare two drugs to placebo for reducing intubation response, found the average increase in mean arterial pressure on intubation in the placebo group to be in the order of 56mmHg!! A similar response in a patient with severe PET could obviously have disastrous effects on the patient’s brain.

Methods to blunt the intubation response:
- **Lignocaine** 1mg/kg on induction. Not very effective and may have negative effects on the baby. Rather avoid, unless no other drug is available.
- **β-blockers.** Esmolol has been used with success, but with negative effects on the baby. There is also a school of thought that suggests β-blockade is totally contra-indicated because it allows unopposed α-effects, ie vasoconstriction. Rather avoid.
- **α-β blockes** ie Labetolol. Although an effective anti-hypertensive for titrated parenteral control, acute bolus use in this setting has not been studied, may cause prolonged hypotension and is therefore best avoided.
Short-acting opiates. Alfentanil (Rapifen®) is the drug of choice and is very effective in a dose of 10μg/kg, just before suxamethonium is given. The respiratory depressive effects on the baby are usually minimal because of its short duration of action, but the paediatrician must still be warned of its use beforehand. When used in combination with magnesium sulphate, doses of both drugs may be reduced (Alfentanil 7.5μg/kg + Magnesium sulphate 30mg/kg).

[remifentanil is also highly effective in a dose of 1μg/kg just before Sux; it is shorter acting than alfentanil, but is very expensive and not readily available in the public sector; it may give rise to fetal bradycardia]

NB! If you have no Alfentanil available, Fentanyl may be used in a dose of 2μg/kg, but must be given at least 3min before induction. The pediatrician must be warned, because neonatal respiratory depression can be profound and prolonged and will require naloxone to reverse!

Magnesium sulphate. As discussed earlier, magnesium is a highly effective anti-catecholamine with vasodilatory action. It effectively obtunds the intubation response without a hypotensive effect, when used in a bolus dose of 40mg/kg just after induction. It may be combined with a short-acting opiate (Alfentanil) in severe cases (see above). Where maternal risk is high, much higher doses may be considered – Alfentanil 30μg/kg + Magnesium sulphate 60mg/kg. When these doses are used, you must be prepared to manage hypotension after intubation.

ENDOTRACHEAL TUBE SIZE
Use one half tube size smaller than normal and have another half size still smaller readily available as preparation for possible laryngeal oedema.

MUSCLE RELAXANTS
As previously mentioned, magnesium sulphate has effects on muscle relaxants of importance to the anaesthetist.
Firstly, the muscle fasciculations usually seen with suxamethonium frequently do not occur, so a timed intubation of 45-60s after Sux administration is advised (a good habit to develop in all rapid sequence intubations).
Secondly, precurarization is contra-indicated, because profound muscle relaxation can be achieved before induction.
Thirdly, the effect of non-depolarizing muscle relaxants is greatly enhanced, but highly unpredictable. Much smaller doses must be used and monitoring with a nerve stimulator is advisable. Some centres avoid non-depolarizing blockers altogether, especially if the surgeon is quite fast.

EXTUBATION
This is a commonly forgotten aspect of anaesthesia, but the extubation response may be just as profound as intubation, with similar disastrous results in PET patients.
However, it is counter-intuitive to use a magnesium sulphate bolus in a patient who has just had reversal of a non-depolarizing neuromuscular blocker, or to repeat a dose of Alfentanil in a patient just waking from anaesthesia.
In this scenario, Lignocaine 1mg/kg bolus or Esmolol 0.2-0.5mg/kg may be used.

PAIN RELIEF
Effective pain relief is required in these patients to prevent a rapid severe rise in blood pressure on awakening and in the early postoperative phase.
The use of NSAIDs in these patients, although effective, are contra-indicated in the face of the endothelial dysfunction, labile cardiovascular condition, renal dysfunction and platelet dysfunction.

Morphine is the drug of choice and this can be titrated IV to effect directly postoperatively, with frequent and sufficient dosing afterwards (see the chapter on pain control).

In the private sector, a very effective drug to use in combination with Morphine is Perfalgan®, an intravenous formulation of paracetamol, which effectively reduces opiate requirements.

**POSTOPERATIVE CARE**

Patients with severe PET often require admission to a High-Care post-delivery. In small hospitals this might require extra staff to ‘special’ the patient. It is obviously preferable to have these patients identified early and referred early to a bigger centre, rather than managed in a small hospital.

However, the change from a mild to moderate pre-eclampsia to fulminating pre-eclampsia, convulsions or worse, may occur rapidly. Also, as shown by maternal morbidity and mortality statistics, many patients present as emergencies, unbooked and untreated.

Mild and moderate PET patients are generally stable post-delivery and are often easily managed in a post-delivery ward, without much trouble. Immediate postoperative concerns are laryngeal oedema and respiratory distress after extubation and the development of pulmonary oedema due to aggressive fluid management.

It must be noted that contrary to popular belief, the risk of eclampsia does not resolve with the delivery of the baby and >40% of eclamptic convulsions present in the postpartum period. Pulmonary complications in the same period are probably just as high.

It is thus necessary for continued monitoring of vital signs and urine output well into the postpartum period; this may mean up to 7-14 days. Fluid balance should receive high priority, especially in patients with oliguria.

Magnesium should be continued for at least as long the patient has residual symptoms. Anti-hypertensive treatment must be continued for as long as clinically required; patients should be stable on oral medications for at least 48hrs after the last dose change and frequent follow-up after discharge is required.
THE PREGNANT CARDIAC PATIENT

Anaesthesia for pregnant patients with cardiac disease is fraught with danger for the mother, the fetus and the anaesthetist. The pregnant patient population is generally healthy, but in this country, cardiac disease occurs not infrequently in pregnancy and usually comprises of valvular disease or cardiomyopathy.

It is not expected of the junior or rural anaesthetist to attempt anaesthesia for Caesarean section in these patients. The most important principles to apply in these cases, are those of early recognition and early referral to a centre where these cases may be safely managed. In the Northern Cape, this centre would be Kimberley Hospital. Further upline referral to Bloemfontein will then be decided on.

However, it may occur that a pregnant cardiac patient may present in labour to a smaller hospital and some knowledge is required. This section thus contains two article extracts which concentrate on the practical issues of identifying pregnant cardiac patients, assessing their risk and providing basic management.

The first extract supplied below is from an excellent recent review article on valvular disease in pregnancy and gives us principles on assessment. The specific valve lesions and their management will not be discussed here, but these are available in the full article. (The full review article can be downloaded free online in pdf format at www.nda.ox.ac.uk, go to Update in Anaesthesia, issue 19).

The second extract below gives the complete obstetric guidelines for cardiac patients in labour, as suggested by the Saving Mothers Task Force. It does not have much information for anaesthetists, but the general principles of management can be clearly seen.

1. Anaesthesia For The Pregnant Patient With Acquired Valvular Heart Disease

Joubert IA, Dyer RA. Department of Anaesthesia, University of Cape Town and Groote Schuur Hospital, Cape Town, South Africa.

Cardiovascular physiology in pregnancy

Pregnancy stresses the cardiovascular system. Wide fluctuations in haemodynamic stress can be anticipated during labour and delivery. Patients with stenotic valvular lesions are particularly prone to complications at delivery, and the anaesthesiologist should be familiar with anticipated difficulties and their management. Patients may require invasive cardiac monitoring during labour, particularly where an operative delivery is anticipated.

Although patients may present with previously diagnosed valvular disease, cardiac compromise frequently only becomes apparent during pregnancy. This is largely due to the fact that normal pregnancy is associated with a 30 to 50 percent increase in blood volume and corresponding increases in cardiac output.

Stroke volume normally increases by 25 to 30 percent, with the remaining increase in cardiac output being accounted for by changes in heart rate. Not surprisingly, where valvular heart disease limits these changes, cardiac compromise with pulmonary oedema or biventricular failure may present early in pregnancy.

Early signs of cardiac compromise may become apparent in the first trimester and peak at 20 to 24 weeks of pregnancy when cardiac output reaches a maximum. From 24 weeks onward cardiac output is maintained at high levels. Cardiac output only begins to decline in the post-partum period.

During labour, the sympathetic response to pain, as well as uterine contractions, induce profound fluctuations in the patient’s haemodynamic status. Between 300 and 500 ml of blood is injected into the general circulation with each contraction. Stroke volume rises by an
estimated additional 50 percent. At the same time, systemic vascular resistance is increased, exacerbating the additional stress placed on the cardiovascular system. At delivery a predicted blood loss of between 400 and 800 ml does little to maintain stability in an already compromised circulatory system.

Many normal women manifest subtle signs of cardiac failure during uncomplicated pregnancy and delivery. Dyspnoea and fatigue are common, together with a reduced exercise capacity. A large proportion of pregnant patients have peripheral oedema with distension of the central veins; many have audible flow murmurs and a 3rd heart sound indicating volume overload.

Where underlying valvular disease is present it is hardly surprising that symptoms and signs of cardiac failure may occur during pregnancy or at the onset of labour. Following delivery the cardiovascular status of the patient will normalise at 6 to 8 weeks post delivery.

Identifying cardiac disease in pregnancy
The anaesthesiologist should be able to identify cardiac disease in pregnancy and labour if appropriate management decisions are to be made. The presence of the following physical signs should always be regarded as abnormal in pregnancy and alert attending physicians to the potential presence of underlying cardiac disease:

- A loud fourth heart sound
- Any diastolic murmur
- A grade 3/6 or more systolic murmur
- Fixed splitting of the second heart sound
- An opening snap

The presence of one or more of these signs indicates the need for echocardiographic evaluation of the heart. Echocardiography has the ability not only to diagnose specific cardiac disease, but also to quantify the severity of cardiac lesions observed. This information is invaluable in both planning anaesthesia and anticipating complications.

Assessing risk in pregnant patients with cardiac disease
The New York Heart Association functional class has been used to identify patients at high risk of complication in pregnancy. A New York Heart Association functional class of III or IV has been estimated to carry a greater than 7 percent risk of mortality and a 30 percent risk of morbidity. Although women in these functional classes should be counselled against child-bearing, it is not infrequent that they are encountered in the prenatal clinic (or the labour ward, or even at the theatre door!).

The American Heart Association has classified cardiac lesions according to their associated risk. This is shown in the table below.

**New York Heart Association (NYHA) Classification** (A functional classification of physical activity for cardiac patients).

- **Class I:** patients with no limitations of activities; they suffer no symptoms from ordinary activities
- **Class II:** patients with slight, mild limitation of activity; they are comfortable with rest or mild exertion
- **Class III:** patients with marked limitation of activity; they are comfortable only at rest
- **Class IV:** patients who should be at complete rest, confined to bed or chair; any physical activity brings on discomfort and symptoms occur at rest

Following a study of 252 completed pregnancies in patients with cardiac disease, five risk factors were identified as being predictive of poor maternal and or neonatal outcome:

1. Prior cardiac events (heart failure, transient ischaemic attack or stroke)
2. Prior arrhythmias (symptomatic brady- or tachy-arrhythmia requiring therapy)
3. New York functional > class II or the presence of cyanosis.
4. Valvular or outflow tract obstruction (aortic valve area of less than 1.5 cm$^2$ or mitral valve area of less than 2 cm$^2$; left ventricular outflow tract pressure gradient of more than 30mmHg)
5. Myocardial dysfunction (left ventricular ejection fraction of less than 40 percent, or a restrictive or hypertrophic cardiomyopathy)

A subsequently revised risk index identified four factors as being predictive of poor maternal and fetal outcome:
1. Prior cardiac event
2. Poor NYHA functional class or cyanosis
3. Left heart obstruction
4. Systemic ventricular dysfunction

Valvular heart lesions and risk during pregnancy
During pregnancy, valvular heart lesions may carry risk for both mother and fetus. Complications ascribed to valvular heart disease include:

- increased incidence of maternal cardiac failure and mortality
- increased risk of premature delivery
- lower APGAR scores and lower birth weight
- a higher incidence of interventional and assisted deliveries.

It is important for the anaesthesiologist to be aware of the attendant risk that a patient suffers as a result of valvular heart disease. Those patients carrying the highest risk warrant additional care, invasive haemodynamic monitoring and appropriate modification of anaesthetic technique. In the absence of echocardiography, where more than one valvular lesion co-exists, the anaesthesiologist must attempt to identify the most clinically significant problem.

2. Guidelines for management of cardiac patients in labour

NOTE: Do not hesitate to consult if you are unsure of what to do.

General principles
1. The patient needs to have been assessed by a team consisting of an obstetrician and cardiologist/internal medicine specialist in the current pregnancy
2. A plan of management including the mode of delivery must be made as soon as possible in the antenatal period. As far as possible vaginal delivery should be the aim.
3. Patients must deliver at a level 2 hospital and above.
4. It is best to await spontaneous onset of labour.

At onset of labour;

First Stage:
- Insert a intravenous line containing a 200mls bag of normal saline or ringers lactate using a 60 dropper administration set just to keep the vein open (run at 10-20mls per hour) and use a rate minder/ or infusion(IVAC) pump.
- Regular BP and pulse charting and intake/output chart
- Provide adequate analgesia.
- Patient to sit up in bed or semi Fowler’s position
- Avoid routine rupturing of membranes.
• Once patient in active phase of labour, she should progress at least 1cm cervical dilatation per hour. (labour must follow the alert line of the graph)

• If there is need for augmentation of labour:
  ▪ Use 2 unit oxytocin in 200mls ringers lactate/normal saline starting at 6mls, 12mls, 18mls, 24mls, 30mls per hour.
  ▪ No progress or poor progress of labour 2 hours after start of oxytocin is an indication for caesarian section.

• Antibiotic prophylaxis for infective endocarditis:
  ▪ Ampicillin 500mg IVI 6 hourly up to 24 hours post delivery
  ▪ Gentamycin 3–5mg/kg body weight IVI daily up to 24 hours post delivery.

• Always keep the emergency / resuscitation trolley easily accessible in order to be able to treat acute emergencies e.g. acute pulmonary oedema.

**Second Stage:**
• Avoid the lithotomy position. (the increase venous return may cause cardiac failure and acute pulmonary oedema)
• Sit patient in a semi fowlers position, bring her at the edge of the bed. Get her to rest her feet on benches or chairs so that her legs and feet are lower than the rest of her body
• Avoid prolonged second stage.
• Avoid routine episiotomy (an episiotomy only if it is necessary)
• Minimise maternal bearing down efforts or prolonged second stage.
• A forceps assisted delivery is recommended over a vacuum assisted delivery.

**Third Stage:**
• Give syntocinon IMI with delivery of anterior shoulder (Avoid Ergometrine or any ergot-containing oxytocic)
• Administer furosemide 80mg IVI immediately after delivery.
• Repair perineal tears/episiotomy if applicable.
• Prevent and monitor for PPH.
• Keep patient for 24hrs in high care section of maternity for observations
• Re-discuss contraception needs before she is discharged.

It is thus clear from the above 2 articles, that cardiac disease in pregnancy carries a high risk of morbidity and mortality. It requires tertiary level investigation, facilities and multidisciplinary evaluation and expertise (including Anaesthesia) to provide an acceptable outcome.

❖ Elective referral to a major centre early in pregnancy is mandatory.

❖ Patients presenting late in pregnancy must be referred urgently.

❖ If faced with a cardiac patient in labour and referral is not feasible, immediate contact must be made with experts at a major centre.
INTRAUTERINE RESUSCITATION OF THE STRESSED FETUS

Many obstetric patients are referred to you as an anaesthetist for emergency Caesarean section with a diagnosis of fetal distress.

**Fetal distress** has been defined as “**progressive fetal asphyxia** that if left uncorrected, will result in decompensation in physiological responses that may result in permanent CNS damage or death”. Worldwide there is a move away from this term, because it is non-specific and poorly defined. The parameters often given as proof of fetal distress are also poorly defined. Thus it is also used where suspicious fetal heart rate traces are present without proven acidosis.

However, the syndrome of progressive fetal asphyxia with hypoxia and acidosis, which is diagnosed by the presence of characteristic features in fetal heart rate patterns, with or without fetal pH measurements, gives rise to clinical decisions that often result in emergency procedures.

There are a number of interim measures that can be instituted to attempt to improve placental blood flow and oxygen delivery, which helps reverse the hypoxia and acidosis. In the rush to prepare the patient for theatre, these measures are often neglected, worsening the fetus’s already compromised condition.

These measures may have a dramatic effect on the parameters used to make the original diagnosis, buy time for the emergent delivery and improve the fetus’s condition at birth. In smaller hospitals, anaesthetists are often requested to resuscitate the neonate, thus instituting these measures early may make life easier for the anaesthetist at the birth.

PROBLEMS WITH THE DIAGNOSIS OF FETAL DISTRESS

The diagnosis of ‘fetal distress’ is freely used by obstetricians to denote a wide range of stages on the continuum of fetal compromise and is sometimes abused to confer a status of emergency to get the patient to theatre quicker. This attitude is unacceptable because it adds an enormous stress factor to a situation that may not need to be so, as well as increasing maternal risks. Unfortunately, the subjective diagnosis of fetal distress and the lack of use of specific criteria to diagnose it has relegated it to a dustbin diagnosis, where it has lost the sense of urgency for many anaesthetists.

This situation should be avoided at all costs, since it leads to delayed action, to the detriment of mother and baby. The ideal is that your hospital has specific criteria in the form of a written protocol (evidence based) that determines how urgent the intervention should be. In the very small hospitals where MO’s often cover all departments, it is easy to confer and agree on the degree of emergency.

It should be noted that it is **not the place of the anaesthetist to refuse the operation** (unless gross abuse has occurred and the patient is placed in
danger), but rather to agree on the degree of urgency and to allow time for intrauterine fetal resuscitation.

**URGENCY FOR CAESAREAN SECTION**
A new easily remembered classification has been proposed for this:

<table>
<thead>
<tr>
<th>GRADE</th>
<th>DEFINITION</th>
</tr>
</thead>
<tbody>
<tr>
<td>1 – EMERGENCY</td>
<td>Immediate threat to life of mother or baby</td>
</tr>
<tr>
<td>2 – URGENT</td>
<td>Distress not immediately life-threatening</td>
</tr>
<tr>
<td>3 – SCHEDULED</td>
<td>Needing early delivery but no distress</td>
</tr>
<tr>
<td>4 – ELECTIVE</td>
<td>At a time to suit the patient and maternity team</td>
</tr>
</tbody>
</table>

**INTERIM MEASURES TO BE TAKEN**

These measures should be instituted by the obstetrician, but it is important that the anaesthetist ascertains whether these measures have been instituted and what the result has been on the fetal heart rate (FHR) pattern.

Again, this is not to criticise or undermine the obstetric team or the obstetric management, but unless the situation is a Grade 1 emergency as above, the instituted measures should be given time to work to buy time for fetal wellbeing. If you are going to have to resuscitate the baby as well, this may mean the difference between a neonate with a good Apgar score and having to abandon a low Apgar neonate to concentrate on the maternal condition, which is your absolute priority.

- **Alteration of maternal position (Reduce aortacaval compression)**
  Only a minority of women in labour have clinical hypotension in the supine position, but the majority of women have reduced venous return due to pressure of the gravid uterus on the inferior vena cava. The reduced venous return can cause a drop in the cardiac output, which in turn decreases uterine bloodflow.
  Instituting a left lateral position of the mother removes compression and improves venous return. This is especially relevant when spinal or epidural anaesthesia is performed where the venous return is further diminished. Practically, this position should be maintained on the theatre table by inserting a wedge under the mother’s right buttock or tilting the table by 15º.

  If the left lateral position makes no improvement, the right lateral position and even the elbow-knee position may be used.

**NB!** It is also important to consider and aggressively treat other causes of maternal hypotension with fluids and vasoconstrictors if manoeuvres to reduce aortacaval compression are not rapidly effective.

- **Hydration**
  Hydration is an integral part of intrauterine resuscitation unless there is a contra-indication to fluid loading (PET, cardiac disease) with Ringers.
Infusing 1l Ringers increases intravascular volume, improves venous return and placental flow and has a tocolytic effect on the uterus. Slower hydration at 180 - 200 ml of Ringers per hour also has beneficial effects. In cases where hypotension is expected e.g. epidural analgesia, maternal bleeding, etc it is important to ensure adequate hydration to prevent FHR changes.

- **Supplemental Oxygen**
  It is important to understand that oxygen transfer at the placental interface is a function of adequate perfusion rather than a high inspired fraction of $O_2$ (FIO$_2$). The only efficient way to increase FIO$_2$ adequately is to use an anaesthetic circuit.
  A moderate increase can be obtained by using a Hudson face mask with a reservoir bag. A normal face mask is inefficient and will probably only increase the inspired fraction to 35% with high flowrates. Despite the lack of definite scientific evidence to say whether supplemental O$_2$ makes a difference in outcome, it is still regarded as a beneficial part of intra-uterine resuscitation.

- **Intravenous hypertonic dextrose**
  Previously, bolus doses of hypertonic dextrose have been used for the management of fetal distress, but this has been shown to be of little value. There is some evidence that hyperglycemia may cause acidosis in the fetus with severe IUGR. There is thus no place for this intervention in intrauterine resuscitation.

- **Tocolytics**
  Physiologically, each uterine contraction causes the placental intervillous blood flow to stop, resulting in a short period of fetal hypoxia, which takes 60-90s to recover.
  In active labour, 4-5 contractions occur in 10 minutes, which may result in fetal hypoxia if contraction pressure or duration is excessive. Reducing contraction and frequency may thus improve placental perfusion.

  Inducing labour or augmenting contractions using Oxytocin can cause uterine hyperstimulation and fetal hypoxia and acidosis. Intravaginal prostaglandin therapy has the same effect. Thus, just stopping the oxytocin infusion or washing out the intravaginal prostaglandins is a first step in intrauterine resuscitation.
  However, it takes 15-45min to substantially reduce the effect on the uterus and tocolytics are indicated.

  As mentioned earlier, rapid infusion of 1l Ringers may have a rapid tocolytic effect, but uterine activity returns to baseline after 20min.

  $\beta$-agonists are often used as tocolytics for fetal distress. They are very effective in this regard, but have severe side-effects if used inappropriately. Tachycardia, dysrhythmias, tremors and nausea and vomiting may occur. Salbutamol (Ventolin), hexoprenaline (Ipradol) and others have been used. For rapid suppression, 5μg hexoprenaline IV over 2min is very effective.
**Calcium-channel blockers:** Nifedipine (Adalat®) and other calcium channel blockers are also employed to suppress uterine contractions effectively with less side-effects than some of the β-agonists. Many units now use these drugs as drugs of choice.

**Magnesium sulphate** has also been used in the acute stage to suppress active contractions, but is not as effective as β-agonists.

**Glyceryl trinitrate spray** sublingually (2puffs every minute to a maximum of 3doses) has been used effectively but unproven in proper trials as yet and is not yet licensed for this use).

**NB!** Tocolysis is to be avoided in cases of obvious abruption or antepartum haemorrhage.

**CHECK TABLE FOR THE INTRA-UTERINE RESUSCITATION OF A DISTRESSED FETUS**

<table>
<thead>
<tr>
<th>FACTOR</th>
<th>INTERVENTION</th>
</tr>
</thead>
<tbody>
<tr>
<td>1 Oxytocin/Prostaglandin</td>
<td>Turn off oxytocin infusion or wash out intravaginal PG</td>
</tr>
<tr>
<td>2 Position</td>
<td>Full left lateral position, continued during transfer and on theatre table; in cases of umbilical cord compression, try right lateral or knee-elbow position if fetal heart rate remains severely abnormal</td>
</tr>
<tr>
<td>3 Oxygen</td>
<td>Maximum flow via tight-fitting Hudson mask with reservoir (if available)</td>
</tr>
<tr>
<td>4 IV Fluids</td>
<td>1l rapid infusion of Ringers lactate unless fluid intake is restricted eg. PET</td>
</tr>
<tr>
<td>5 Hypotension</td>
<td>Consider IV Ephedrine 5-10mg, Etilephrine 1-2mg or Phenylephrine 50-100μg boluses</td>
</tr>
<tr>
<td>6 Tocolysis</td>
<td>IV Hexoprenaline 5μg over 2min or IV Magnesium sulphate 2g IV over 2min or IV Salbutamol 100-250μg slow injection over 5min; oral Nifedipine 30mg or GTN sublingual spray(not yet licensed for use)</td>
</tr>
</tbody>
</table>
ANAESTHESIA FOR CAESAREAN SECTION - SPINAL

Why should we be doing spinal anaesthesia for a Caesarean section?
The question is often asked by obstetric colleagues and even by anaesthetic trained colleagues, especially if they have done many C-sections under general anaesthesia.

What is the evidence for choosing either approach and what are the factors we should be taking into account when we make our choice?
Are the same concerns valid for emergency C-sections?
Let us look at the evidence we have for making our decisions.

ELECTIVE CAESAREAN SECTION
Maternal outcome in elective CS is better with regional than with general anaesthesia. A large study showed a case fatality rate of 32 per million for general anaesthesia compared to 2 per million for spinal anaesthesia.

The causes for the difference were mainly due to airway management problems (see the chapter on airway assessment). Neonatal outcome was also better in spinal anaesthesia compared to GA in elective CS.

Clinical parameters in neonates in a number of large studies all show the same result, namely improved Apgar scores and a decreased need for assisted ventilation in those delivered by spinal. Umbilical artery pH was found to be lower in those delivered by spinal, but these were predominantly (80%) respiratory acidosis, which does not have an increase in neonatal complications. More recent controlled studies looking specifically at this acidemia have found the acidemia to be less in the spinal group.

EMERGENCY CAESAREAN SECTION
As mentioned in the chapter on airway management, emergency CS is in itself a risk factor for a difficult airway, thus it is no surprise that maternal outcome under spinal anaesthesia is improved as compared to GA.

Neonatal outcome in emergency CS has not been well studied under randomized controlled trials, but all the retrospective studies show an improved outcome.

What about emergency CS for fetal distress?
This is a highly emotive topic between obstetricians and anaesthetists, especially since there is not much well-documented evidence in the literature.

A trial by Marx et al of 126 women for emergency CS for fetal distress (severe heart rate abnormalities and fetal scalp blood pH<7.20) found that there was no difference between GA, spinal or epidural regarding fetal biochemical data, but the group that underwent spinal anaesthesia had better Apgar scores at 1min. These however were not true 'stat' emergency CS procedures.

The American College of Obstetricians and Gynaecologists recognises this issue in their goals to promote use of regional anaesthesia and minimize the use of GA and have stated: "recognition that Caesarean delivery for a non-reassuring fetal status does not necessary preclude the use of regional anaesthesia".
The main concerns for the obstetrician are that the placement of a spinal ‘may take too long’ and that the hypotension from the spinal may worsen utero-placental bloodflow and neonatal outcome. These valid concerns must be weighed up against the urgency of the situation and the risks of a general anaesthetic for that specific patient.

It is also important to include the experience of the anaesthetist into this situation, because this is not a ‘one size fits all’ scenario.

**Factors influencing outcome of fetal distress**

It is important to realise that the type of anaesthesia is not the sole determinant of neonatal outcome in fetal distress – in fact, it is not even the most important factor.

Other more important factors are:

- Time from decision to do a stat CS to time of delivery – should be < 30min
- Time of induction (GA) to time of delivery – should be less than 10min
- Time from skin incision to time of uterine incision – should be <5min
- Time of uterine incision to time of delivery – should be <3min

The thinking behind this is that it doesn’t change the neonatal outcome by rushing the anaesthetist and dictating the anaesthetic approach, but then not improving the above intervention times, which have a greater impact on neonatal outcome.

This however does not mean that one can take your time and cause delay. One should make a rapid clinical preoperative assessment and choose the anaesthetic approach that will be the safest and quickest for optimal outcome for mother and baby.

The surgeon, the assistant and staff should be scrubbed, ready and waiting to start the procedure as soon as the mother is induced/spinal inserted.

**SPINAL ANAESTHESIA**

Spinal anaesthesia, because of its apparent safety and ease of technique, is often considered by many, both surgical colleagues and inadequately trained anaesthesia colleagues alike, to be an ‘autopilot anaesthesia’.

This ignorant view of spinal anaesthesia, plus the use of inappropriate doses of local anaesthetic, lack of proper monitoring and poor fluid management, have led (and still lead) to severe hypotension, high spinals and death.

Spinal anaesthesia should thus be considered to be an alternate form of anaesthesia, but no less invasive or less risky as GA.

**ADVANTAGES OF SPINAL ANAESTHESIA**

- Easy technique
- Small doses of drugs
- Low failure rate (3-5%)
- Rapid onset
- Awake mother
- Minimal neonatal depression
- Avoidance of risks associated with GA (especially airway problems)
DISADVANTAGES

- High incidence of hypotension
- Limited duration of effective anaesthesia (1.5-2hrs)
- Ignorance and/or poor acceptance by the public – this is often easily countered by explaining the technique and advantages to the mother.

WHAT MUST YOU TELL THE MOTHER?

- Mothers are always scared of having ‘the needle in the back’ - tell your patient that it is usually less painful than the insertion of the drip, which it usually is.
- Hypotension and nausea may often occur – these will be monitored for and immediately treated. Inform the patient beforehand of the frequent (every minute) inflation rate of the BP cuff for at least the first 10-15min.
- Proposed rectal administration of diclofenac (Voltaren suppositories)
- The possibility of block failure and conversion to GA (5%)
- Possibility of intraoperative sensation and its perception by the brain as pain (tell your patients that it is like having an injection at the dentist; they may still experience pressure sensation, but should feel no pain; however, sometimes in the abdomen, pressure or stretching on the inside of the abdomen (swabbing, insertion of swabs or a rough surgeon) may register as discomfort in the brain. The normal pathway to discriminate between pain and discomfort has been blocked by the spinal, thus the brain may tell the body this sensation is pain).
- That they may shiver or feel cold.
- There is a small chance (<5%) of developing a bad headache after the spinal, but this will be treated if occurs (see Postdural puncture headache later)
- Tachycardia and flushing may occur with oxytocin administration.

WHAT ARE THE LANDMARKS FOR DOING THE BLOCK?

Before attempting the subarachnoid (spinal) block, it is important to know the surface anatomy and level of the spinal cord.

In adults, the spinal cord ends at the level of L1, so generally a spinal anaesthesia attempted below this level is usually safe. The spinal cord ends in the cauda equina, but this floats freely in the CSF and therefore is unlikely to be involved in injection problems.

An imaginary line drawn between the highest points of the iliac crests (the intercristal or Tuffier’s line) is at the level of L4 or the L4/5 interspace. This is used as the starting point to count the vertebrae.

The T formed by the midline of the vertebrae and the space at the level or just above the level of Tuffier’s line indicates the puncture site. Remember that the lumbar spinous processes are pointed downwards from the vertebrae to the skin, so the direction of the needle should be moderately upwards (cephalad).

The ‘spinal’ is basically a lumbar puncture (LP), but the best position for pregnant patients is usually in the sitting position and not the lateral decubitus position, although this can be used.
It is important to appreciate and visualize the layers that are traversed by the needle when doing a spinal, as the ‘feel’ for this increases your first attempt ‘hit rate’:

- Skin
- Subcutaneous tissue
- Supraspinous ligament
- Intraspinal ligament/muscle
- Ligamentum flavum
- Epidural space
- Dura
- Subarachnoid (spinal) space

When traversing the ligamentous layers, increased resistance will be felt. Knowing this, it is easier to ‘feel’ that you are in the subarachnoid space.
CONTRA-INDICATIONS TO NEURAXIAL BLOCKADE

**Absolute**
- Infection at the injection site
- Patient refusal!!
- Severe hypovolemia
- Fixed cardiac output conditions (cardiac failure, mitral or aortic stenosis)
- Coagulopathy or bleeding diathesis
- Increased intracranial pressure

**Relative**
- Uncooperative patient (or cerebrally depressed, eg eclamptic)
- Pre-existing neurological defects
- Severe spinal deformity
- Stenotic valve lesions

**Controversial**
- Where prolonged surgery is anticipated (>90min)
- Where major blood loss is anticipated
- Prior back surgery at the site of injection
- Inability to communicate with the patient

COMPLICATIONS AND MANAGEMENT

Various texts have various headings under which they classify or list the possible complications of neuraxial blockade. However, I consider the following time-based approach to be the most practical:

**IMMEDIATE** (often life-threatening) COMPLICATIONS
- Hypotension (regarded by many to be a normal result of a spinal)
- High block
- Total spinal block
- Cardiac arrest
- Systemic toxicity to local anaesthetics
- Failed/inadequate block

**EARLY COMPLICATIONS** (within 24hrs)
- Nerve root/spinal cord damage
- Intraspinal/epidural hematoma
- Transient neurological symptoms
- Urinary retention

**LATE COMPLICATIONS** (after 24hrs)
- Postdural puncture headache
- Backache
- Infection – epidural abscess or meningitis
MANAGEMENT OF THE COMPLICATIONS

The management of many of these complications is avoidance, based on pro-active monitoring, meticulous and careful technique, early recognition and aggressive early therapy once a problem is recognized.

Immediate Complications

Hypotension, high block, total spinal block and cardiac arrest may be considered to be points on the same continuum. Progression from hypotension to cardiac arrest usually occurs due to lack of close monitoring and delayed reaction and may occur in the space of a few minutes.

For this reason, frequent monitoring is absolutely mandatory – by frequent is meant every minute, starting immediately after the patient lies down. Remember that wedging under the right buttock or 15° left lateral tilt of the theatre table must be ensured in each patient to prevent aortacaval pressure syndrome.

Using the initial BP readings prior to injection as the baseline, use vasopressors and inotropic drugs to maintain the pressures close to the original readings (see below).

Fluid loading reduces the severity of hypotension to a degree, but only if given during the 15-30minutes directly prior to the block. This is called co-loading, in contrast to the previously suggested pre-loading of fluid >1hr prior to a spinal block. A volume of at least 500-1000ml of crystalloid is needed for it to have any effect.

Obviously the higher the spinal block progresses, the more segments of the sympathetic chain are blocked and the greater the vasodilatation due to the chemical sympathectomy and the greater the degree of hypotension. Aggressive management with vasopressors and/or inotropes is needed.

Several inotropes and vasoconstrictors are commonly available. Mechanism of action, formulation, dose and suggested use may be considered according to the table below:

<table>
<thead>
<tr>
<th>DRUG</th>
<th>Ephedrine</th>
<th>Etilephrine (Effortil®)</th>
<th>Phenylephrine</th>
<th>Adrenaline</th>
</tr>
</thead>
<tbody>
<tr>
<td>RECEPTORS</td>
<td>α1, β1+2</td>
<td>α1, β1+2</td>
<td>Pure α1</td>
<td>α1, β1+2</td>
</tr>
<tr>
<td>EFFECT</td>
<td>Vasoconstriction, ↑BP, ↑HR, ↑CO</td>
<td>Vasoconstriction, ↑BP, ↑HR, ↑CO</td>
<td>Vasoconstriction, ↑↑BP, ↑↑HR, ↑↑CO</td>
<td>Vasoconstriction, ↑↑BP, ↑↑HR, ↑↑CO</td>
</tr>
<tr>
<td>POTENCY</td>
<td>2+</td>
<td>2+</td>
<td>4+</td>
<td>5+</td>
</tr>
<tr>
<td>FORMULATION</td>
<td>50mg/ml</td>
<td>10mg/ml</td>
<td>10mg/ml</td>
<td>1mg/ml(1:1000)</td>
</tr>
<tr>
<td>DILUTION</td>
<td>50mg in 10ml, ie 5mg/ml</td>
<td>10mg in 10ml, ie 1mg/ml</td>
<td>10mg in 200ml saline, ie 50μg/ml</td>
<td>1mg in 20ml, ie 50μg/ml</td>
</tr>
<tr>
<td>DOSE</td>
<td>5-10mg boluses every 1-2min</td>
<td>1-2mg boluses every 1-2min</td>
<td>50-100μg bolus every 1-2min</td>
<td>10-50μg bolus every min</td>
</tr>
</tbody>
</table>

The suggested doses are guidelines for the treatment of hypotension with spinal anaesthesia. Obviously, if the hypotension is severe, both the dosage and frequency of the inotrope/vasoconstrictor being used, must be increased!

A high spinal above the level of T4 may start to produce symptoms not only due to the hypotension (compensatory tachycardia, dizziness, nausea), but also due to respiratory impairment (loss of intercostal muscles) and weakness in the arms and shoulders. If a patient exhibits respiratory distress and upper limb weakness, immediate preparations should be made to convert to a GA. If unsure, check the sensory level before inducing the patient for a GA.
At this stage, the patient may also develop a bradycardia due to the blockade of the sympathetic ‘cardio-acceleratory’ fibres to the SA and AV nodes (from levels T1-T4), resulting in vagal (parasympathetic) dominance.

This combination of a severe hypotension and a bradycardia spells disaster, because the compensatory physiological response to hypovolemia and decreased cardiac output, namely an increase in heart rate, is blocked. If this situation has not been recognized until this point, or remains unchecked, acute collapse occurs and the patient is often resistant to resuscitation efforts. Adrenaline is the only drug to use in this scenario.

Total spinal block is a pre-arrest scenario, with the patient in a low cardiac output, moribund state and with miotic pupils. It usually occurs with epidural rather than spinal anaesthesia. As in the arrest scenario, immediate intubation, ventilation, CPR and restoration of sinus rhythm with cardiac output is needed.

**Systemic toxicity to local anaesthetics** also usually occurs in epidural rather than spinal anaesthesia, due to the large volumes of local anaesthetics used. There is usually inadvertent injection into a vein. The resultant toxic symptoms depend on the local anaesthetic used.

**Lignocaine** gives rise to predominantly neurological symptomatology – perioral paraesthesia is usually the initial symptom, followed by tinnitus, dizziness, agitation described by patients as a feeling of impending doom, twitching, loss of consciousness, convulsions and cardiorespiratory arrest. The progression from initial symptoms to convulsions and arrest may occur in the span of a few minutes. Management depends on early recognition and prevention of progression to convulsions and arrest.

Hyperventilation with 100% O₂ increases the convulsion threshold; convulsions should be stopped by intravenous thiopentone 50-100mg, midazolam 2-5mg or propofol 50-100mg.

The airway should be controlled and oxygenation maintained. If needed, intubation should be performed using a rapid sequence technique with suxamethonium.

**Bupivacaine toxicity** usually presents with massive cardiovascular collapse, severe and bizarre dysrhythmias and seizure activity. Resuscitation is renowned to be complex, prolonged and aggravated by severe acidosis and hypoxia.

Management is intubation, ventilation, supportive measures to maintain cardiac output and treatment of dysrhythmias. Amiodarone has been used with some success in treating dysrhythmias. If available, Intralipid (TPN fatty emulsion) may be beneficial if used, according to anecdotal reports.

**POSTDURAL PUNCTURE HEADACHE (‘Post-spinal headache’)**

Postdural puncture headache (PDPH) is a persistent problem in anaesthesia, especially obstetric anaesthesia and is a major source of legal claims against anaesthetists in the USA. Many of these are secondary to insertion of epidurals with inadvertent dural puncture (1.5% of epidurals)

PDPH may be mild, with a brief duration and no sequelae. Although the tendency is for PDPH to resolve spontaneously, it may be severe and debilitating, leaving patients bedridden and unable to care for their newborns and delaying their discharge home. Other symptoms commonly seen are nausea, vomiting, visual disturbances and alteration in hearing.
**Definition**
The International Headache Society defines PDPH as:
A bilateral occipito-frontal headache which develops within 7 days and disappears within 14 days of a lumbar puncture. The headache worsens within 15 minutes of assuming the upright position and disappears or worsens within 30 minutes of lying flat.

**Risk factors**
There are a number of risk factors for the PDPH, an important one of which is pregnancy, especially if they have been in active labour and have been bearing down. The two most important risk factors that we can modify are the shape and size of spinal needles.
The Quincke needle (which is the one we use here in the Province) has a higher incidence of PDPH than the blunter pencil point type needles. The smaller the diameter (higher gauge) of the needle, the less the incidence of PDPH. Thus the incidence of junior anaesthetists using a 22G Quincke needle (the general situation) has an incidence of PDPH of 5-10%, while a senior anaesthetist using a 25G pencil point is <1%.

**Etiology**
The brain is cushioned by the CSF; the slow leakage of CSF due to dural puncture causes traction on pain-sensitive structures in the skull due to loss of this cushioning effect. This generally accepted explanation has been contended by an alternative hypothesis of cerebral venous dilatation due to the loss of CSF.
The use of air instead of saline for the loss of resistance techniques when inserting an epidural in itself may give rise to a PDPH.

**Prevention** (other than type and gauge of needle)
**Bedrest** – although many advocate this as preventative, no studies have shown any difference in outcome.

**Bevel alignment** – the cutting edge bevel of the Quincke needle has been associated with an increased incidence of PDPH. Aligning the bevel vertically for insertion (90° to the usual insertion plane), ie parallel with the fibres of the dura, has been shown to decrease the incidence of PDPH. This has recently been questioned and remains a controversial issue.
Patient positioning and number of attempts at insertion are indicative of the experience of the doctor, rather than being true modifiable factors for prevention. The experience of the doctor is a definite factor in the incidence of PDPH.

**Diagnosis**
The diagnosis of PDPH is clinical and is based on the abovementioned definition, but especially on the fact that the headache is positional (initiated or worsened by assuming the upright position and improved on recumbency).

Remember that postpartum patients may be prone to headache, sometimes severe. Not every headache in the patient who has had a spinal anaesthesia is a PDPH.

Evaluate the patient’s headache and ask pertinent questions without leading, eg rather ask “what happens to your headache when you change your position?” or what makes your headache better or worse?” rather than “does your headache get worse when you sit up?”

Patients with a PDPH will usually offer the answers without any prompting and one should question the diagnosis of PDPH if the positional quality of the headache is vague or in doubt.

You do have some time on your side regarding the management of the headache, so you do not have to rush the diagnosis of a PDPH. Reassessment of the headache after 12hrs is acceptable practice if you are unsure (see **Treatment**)

**Treatment**
The treatment ranges from conservative to invasive.
Conservative measures include:

- bedrest
- analgesics, esp NSAIDs
- intravenous fluid loading
- cerebral vasoconstrictors (caffeine, sumatriptan)

Invasive measures include:

- epidural saline 30ml (~60% effective)
- epidural blood patch 15-20ml (75%-93% effective in various trials) of the patient’s own blood obtained under sterile conditions immediately prior to injection
- more recently, epidural injection of dextran has also been used but this is still being investigated

A summary of the approach to treatment of a patient you consider to have a definite PDPH, may be considered to be the following:

- **<24hrs after dural puncture:**
  start with conservative measures such as bedrest; simple analgesics, especially NSAIDs; high fluid intake(oral and IV) as well as strong coffee – if IV caffeine is available(highly unlikely), give 500mg IV. Oral caffeine, although recommended, does not appear to improve outcome or prevent the need for epidural blood patch. (Some obstetricians have used Red Bull in the place of coffee!)

- **>24hrs after dural puncture** (including failed conservative management):
  Epidural blood patch (EBP) is the treatment of choice.
Conservative treatment may be continued if there has been a major improvement on conservative therapy, or if the patient refuses an epidural blood patch – but only continue for 24hrs.
The best time to perform an EBP is between 24 and 72hrs after dural puncture in the symptomatic patient, with best results around 48hrs. 5-8% of patients may require a repeat EBP, so patients must be informed of this possibility.

A SUGGESTED TECHNIQUE FOR SPINAL ANAESTHESIA

- Check your anaesthesia machine and prepare as if you are going to have to perform a GA (airways, tubes, laryngoscope, suction etc).
- Transport the patient to the theatre in the left lateral position.
- If not already done, administer a non-particulate oral antacid (30ml 0.3MOL sodium citrate).
- Establish that there is a working large-gauge venous access present and start rapid infusion of 500ml – 1000ml dextrose-free balanced salt solution (so-called ‘co-loading’)
- Attach monitors and obtain baseline readings. Make sure the correct size BP cuff is used.
- Have GA drugs readily available, but not drawn up; make sure you have Ephedrine/Phenylephrine drawn up for treating hypotension.
- Seat the patient with her legs hanging over the side of the table, feet resting on a stool

- Make sure you are wearing your mask, then scrub up.
- Draw up your block drugs, sterile drugs first, then your local anaesthetic for infiltrating the skin; the drugs and doses used most widely are 1.8ml (9mg) hyperbaric 0.5% Bupivacaine (Macaine 0.5% Spinal with dextrose), plus 10μg (0.2ml) Fentanyl – total volume 2.0ml – in most cases this is sufficient for a T4 level block, effective for 90-120min
- Use the smallest gauge needle available (unfortunately, the only one readily available at present in the state sector of the Northern Cape is the 22G Quincke type needle)
- Administer the block
- Immediately after inserting the block, cover the puncture site with a small occlusive dressing and lie the patient down. Make sure the right hip is wedged or the table tilted to the left by 15degrees and the head slightly raised.
- Immediately take the BP – while waiting for the reading, administer oxygen at 3 l/min flow via mask or nasal prongs.
- Take the BP at 1min intervals for at least the first 10-15min and treat any hypotension aggressively
- Make sure the patient is comfortable and do not be hesitant to converse with your patient throughout the procedure
- Before the surgeon makes the incision, let him/her pinch the skin a few times with a toothed forceps and check for a pain response from your patient, or check for a sensory level with an ice-pack. Chart the level.
- Allow no more than 10-20% drop in BP before reacting with vaso-pressors. The idea is to keep maternal BP close to the baseline measurements.
- Practical tips for vasopressor use are to have both Ephedrine and Phenylephrine drawn up ready. If the patient is tachycardic with a low BP, then it is preferable to raise the BP using the Phenylephrine, which should slow the heart rate somewhat, allowing improved diastolic filling of the heart. If the heart rate is normal to low with the hypotension, then it is preferable to use the Ephedrine to raise both the blood pressure and the heart rate.
- Warn the patient well beforehand (and just prior to it happening) of the upper abdominal discomfort which will be experienced when fundal pressure is applied to deliver the baby.
- At delivery, decide on your choice of oxytocic drug. Be wary of using oxytocin (Syntocin®) in patients with hypotension, hypovolemia or severe tachycardia. Oxytocin in unstable patients may precipitate acute collapse, especially when given in overdose (>5U bolus). It is important to know that the maximum standard bolus dose is 5IU (half an ampoule). Rather opt for the use of ergometrin (Synthometrin®) in these patients.
- The surgeon may request you to give a repeat dose of the oxytocic drug. Make sure if this repeat dose is really needed and rather urge the surgeon and/or assistant to perform manual rubbing-up of the uterus. Again, be wary of oxytocin – if it must be given, dilute the remaining 5IU to 5ml in a syringe and give 1IU every 1-2min rather than a repeat 5IU bolus.
- The surgeon also often asks for oxytocin in the drip. This request varies between surgeons and may be anything from 10IU to 50IU. The standard request is usually 20IU in a litre. This practice may be dangerous, unless you have a written protocol in place. There are a few reasons why this may be so:
  - There is no control over the flow rate – nursing protocol traditionally includes stopping all infusions prior to transfer to the ward, which may mean the infusion is stopped for anything up to an hour, during which time the uterus may relax and bleed
  - The first reaction to a bleed or hypotension is to infuse 500ml –1l of fluid, which means the patient receives the full dose of oxytocin meant for over 6-12hrs in 30min or less. In a shocked patient this may be disastrous.
  - The medicolegal responsibility for this infusion is yours
- The sensible approach to a request for an oxytocin infusion is to discuss the need for it with the surgeon (if not standard practice).
- Is the patient at risk for PPH or is the oxytocin being used because of tradition? If it is standard obstetric practice at your institution, for the prevention of PPH, this is totally acceptable.
- Limit the maximum dose of oxytocin in the infusion to 20IU. (If the surgeon wants more than that dose, he/she can write up the prescription or prepare the infusion on his/her own responsibility).
- Rather prepare the infusion as a “piggy-back drip” in 200ml saline and let it be infused either through an IVAC (if available) or through a 60-dropper line with a Dosi-Flow to determine an approximate rate. As always, these infusions should be well marked on a sticker on the saline bag.

- Get the surgeon/sister to insert a Diclofenac suppository during the cleaning of the patient or expression of clots from the uterus and vagina (NB! - not for PET/Eclampsia and renal dysfunction patients!).
- Ensure that your orders for recovery and ward management of the patient are written up. These should include adequate pain management and anti-emetics.
- Do not leave the theatre complex until the patient has been recovered properly and ready for discharge to the ward. See the chapter on Recovery Room issues for determining discharge suitability. (If the patient has to go to high-care or ICU, you must accompany the patient and do a proper hand-over to the receiving staff).

- Last, but not least: before discharging your patient from the recovery room, ensure that she is comfortable and that her vitals are stable. Check her sensory level with an ice-block and chart the level in your notes: there should preferably be a 2-level regression of her block from your initial level. Finally, pull back her covers to check there is no active bleeding from vagina or wound.
ANAESTHESIA FOR CAESAREAN SECTION – GENERAL ANAESTHESIA

In the previous section, the advantages of spinal anaesthesia over general anaesthesia in obstetric patients were discussed. However, one should not lose sight of the fact that general anaesthesia for Caesarean section is sometimes preferable and spinal anaesthesia may be a poor choice, even a dangerous choice at times.

In the elective situation, the advantages of a regional technique should be explained to your patient. Without trying to force a decision for regional on a woman who chooses general anaesthesia, the patient should nevertheless be warned about the consequences of her decision upon neonatal sedation, blood loss and postoperative pain (all increased).

Awareness, failed intubation and aspiration are not normally mentioned since they are uncommon in trained hands. However, these factors should be mentioned if the anaesthetist is junior or not very experienced in general anaesthesia for obstetric patients.

In the emergency situation, it is appropriate to warn the mother of the overall increased maternal risk of GA compared to spinal anaesthesia. This should not be abused or overemphasized in an attempt to persuade the mother to opt for regional anaesthesia. If for some reason your block fails, you will have your hands full reassuring the patient as to the safety of the GA you now want to do!

A suitable way to explain the risks would be to emphasize the positive aspects of regional techniques; then reassure the patient that although both regional and GA techniques are safe, regional techniques are definitely safer.

When faced with a parturient who refuses spinal anaesthesia, but who has a definite high-risk airway on assessment, obviously failed intubation and gastric aspiration and their consequences must be explained. Explain that as her anaesthetist, you would prefer her to opt for a regional technique, and that a GA would only be your choice out of necessity.

WHEN TO DO A GENERAL ANAESTHETIC

- When the patient refuses regional techniques
- When regional techniques are absolutely contra-indicated (see list in previous chapter)
- When the patient is unstable, dehydrated, hypovolemic or clinically very ill and the sympathectomy caused by the spinal anaesthesia will probably worsen the clinical picture
- When relative contra-indications exist and there are any other possible factors which may complicate a regional technique
- Where the anaesthetist’s training/experience regarding regional techniques is poor, but is well-experienced in general anaesthesia.
As with regional techniques, all of the same precautions regarding physiological changes, full stomach, aspiration risk and patient positioning hold a prominent place in general anaesthesia.

An additional factor that has to be taken into account in general anaesthesia, is the effect of anaesthetic drugs on the mother and fetus. Generally speaking, the mother is more sensitive to many of the anaesthetic drugs, as can be seen by a reduced MAC (minimum inhibitory concentration) of volatile agents, reduced doses of local anaesthetics required for effect.

**PLACENTAL TRANSFER OF ANAESTHETIC DRUGS**

Drugs administered to the mother cross the placenta by passive diffusion, which is influenced by the concentration gradient across the placental bed (drug dose and concentration), rate of drug supply (uterine and umbilical bloodflow) and the diffusion constant for the drug across the placental bed.

Factors that increase maternal drug concentration are:
- High total doses administered
- Use of drugs with slow metabolism (higher concentration of free drug)
- Injection of drugs into highly vascular areas (rapid uptake into the bloodstream and thus higher concentration of free drug)
- Low protein binding of a drug (higher concentration of free drug)

Factors effecting a rapid diffusion of drugs are:
- Low molecular weight (crosses cell membranes easier)
- High lipid solubility (crosses cell membranes easier)
- Low protein binding (higher concentration of free drug)
- Low degree of ionization (higher concentration of free drug)

These general principles of pharmacokinetics are all characteristics found in nearly all anaesthetic, analgesic and sedative drugs. These drugs thus cross the placenta with ease.

Neuromuscular blocking agents are poorly lipid soluble and are highly ionized and thus do not readily cross the placenta.

Once across the placenta, the drug is subject to fetal factors that effect drug distribution and metabolism. Without going into too much detail, the fetal circulation delivers ± 50% of the drug to the liver via the umbilical vein. Although the fetal liver is immature, its cyt-P450 and other enzyme systems are capable of metabolic functions. The other 50% bypasses the liver and is diluted by atrial blood and some is shunted across the foramen ovale and ductus arteriosus.

All of these factors are protective for the fetus, reducing high concentrations of drugs in the heart and CNS and thus diminishing possible effects.

It is NB! to note that fetal pH and protein binding also affect drug levels. This is relevant in the stressed fetus, where acidosis may ‘trap’ ionized drugs (eg local anaesthetics) in the fetal circulation.
ADVANTAGES OF GENERAL ANAESTHESIA
- Rapid induction
- Less hypotension
- Less cardiovascular instability
- Definitive airway control and management

DISADVANTAGES OF GENERAL ANAESTHESIA
- Airway problems (failed intubation)
- Increased aspiration risk
- Maternal hyperventilation
- Neonatal depression
- More uterine atony

A SUGGESTED TECHNIQUE FOR GENERAL ANAESTHESIA
- ALWAYS examine your patient – including airway assessment
- Check your anaesthesia machine and prepare for administration of a GA (airways, tubes, laryngoscope, suction etc).
- Transport the patient to the theatre in the left lateral position.
- If not already done, administer a non-particulate oral antacid (30ml 0.3MOL sodium citrate).
- Establish that there is a working large-gauge venous access present and start rapid infusion of dextrose-free balanced salt solution.
- Attach monitors and obtain baseline readings. Make sure the correct size BP cuff is used.
- Start pre-oxygenation with high flow rates of oxygen (see rapid-sequence induction protocol).
- Warn your patient prior to induction of the application of cricoid pressure by your assistant.
- Inject your preferred induction agent; as loss of eyelid response occurs, inject suxamethonium (1.5mg/kg) and note the time.
- Do not bag the patient at this stage. Wait for the fasciculations to stop before attempting intubation. If no fasciculations occur, wait for 45-60s from the time you noted at injection of the suxamethonium, then intubate.
- Intubate the patient AND MAINTAIN CRICOID PRESSURE UNTIL THE TUBE POSITION HAS BEEN VERIFIED AND THE CUFF IS INFLATED.
- Ventilate the patient and use 3-5cmH₂O PEEP, but do not hyperventilate.
- Open the volatile agent of your choice to 0.8-1MAC, with or without N₂O. If N₂O is used, maintain it at 50% combined with 0.8MAC volatile.
- The use of muscle relaxant is often not needed, therefore do not use routinely. If muscle relaxant is needed, use only a small maintenance dose, eg Vecuronium 0.02mg/kg, Rocuronium 0.15mg/kg or cis-Atracurium 0.15mg/kg.
- As soon as the cord is clamped, deepen anaesthesia by adding opioid; continue N₂O if used; maintain volatile agent, but try to use no more than 1MAC to prevent uterine relaxation
The same considerations given to the use of oxytocics and vasopressors/inotropes as discussed in the previous chapter on spinal anaesthesia, are applicable to patients having a GA.

At the end of the procedure, reverse any muscular paralysis and **EXTUBATE THE PATIENT AWAKE**, preferably on her left side.

Get the surgeon/sister to insert a Diclofenac suppository during the cleaning of the patient or expression of clots from the uterus and vagina.

Do not leave the theatre complex until the patient has been recovered properly and ready for discharge to the ward. (If the patient has to go to high-care or ICU, you must accompany the patient and do a proper hand-over to the receiving staff).

Last, but not least: before discharging your patient from the recovery room, ensure she is comfortable, her vitals are stable and pull back her covers to check there is no active bleeding from vagina or wound.
NEONATAL RESUSCITATION

The anaesthetist, by nature of the job, is usually more adept at airway management and resuscitation than many other disciplines in medicine. For this reason, the anaesthetist is often called upon to assist a neonate in distress in the labour theatre/ward.

This frequently happens worldwide, despite the fact that internationally accepted guidelines clearly state that the primary responsibility of the anaesthetist is to care for the mother and that “qualified personnel other than the anaesthetist, must be immediately available to assume responsibility for resuscitation of the newborn”.

In South Africa, especially in the peripheral hospitals, it is often more the rule than the exception that the anaesthetist must care for the neonate. Often, the primary attendant is a pediatric or labour ward nurse and the anaesthetist is called to assist when problems arise.

The anaesthetist must thus not only be capable of acute neonatal care, but should anticipate that he/she must make provision to be actively involved in neonatal resuscitation at very short notice.

PREPARATION
- The anaesthetist must be aware of the resuscitation equipment available in the labour ward and know how to use it.
- A specific emergency drug tray and equipment trolley must be kept ready in the labour theatre, with frequent checking of equipment and drugs. Frequent checking means prior to and just after each delivery. Drugs should be replaced on use.
- At least one person trained in neonatal resuscitation should attend each delivery; more staff should be available for high-risk deliveries.
- There should be a fixed protocol for the resuscitation – this should be followed by ALL staff, doctors and sisters alike – and should be visible at the area where neonatal resuscitation is usually performed.

NEONATAL ADAPTATIONS TO EXTRAUTERINE LIFE

The transition from intra-uterine to extra-uterine life involves great physiological changes and adaptations for the fetus. Knowledge of these transitional changes gives one greater insight into the objectives that have to be achieved to successfully resuscitate the newborn. These changes can be shortly summarized as follows:

Intrauterine condition:
- The fetus depends on placental bloodflow for gaseous exchange. The placenta receives almost 50% of fetal cardiac output.
- Oxygenated blood returning from the placenta mixes with deoxygenated blood from the lower body and reaches the right atrium. Here it mixes with deoxygenated blood from the upper body, but ⅔ of the blood from the IVC flows preferentially through the patent foramen ovale into the left atrium.
- From the left atrium, blood flows to the left ventricle which then pumps it to the upper body (brain and heart tissue) via the aorta.
- The blood entering the right ventricle is pumped into the pulmonary artery. However, the fetal lungs are unexpanded and therefore resistance to bloodflow (pulmonary vascular resistance) is high. The bulk of the flow (90%) is shunted
through the patent ductus arteriosus into the descending aorta, since resistance to bloodflow (systemic vascular resistance) is low. The remaining 10% is sufficient to perfuse lung tissue.

**Birth:**
- The process of birth expels amniotic fluid from the mouth and upper airways and at birth, the fetus gulps and cries, releasing surfactant, filling the lungs with air, increasing oxygenation. The lungs suddenly change from a high resistance vascular bed to one of low resistance, promoting pulmonary bloodflow.
- Clamping of the umbilical cord removes the low resistance placental bed and thus greatly increases the systemic vascular resistance.
- These pressure changes result in redirection of bloodflow – in the right atrium towards the low pressure right ventricle, pulmonary artery and lungs – this massively reduces the shunting across the foramen ovale and ductus arteriosus and functionally closes these shunts.
- Transient hypoxemia is usually well-tolerated by the neonate, but prolonged hypoxemia, hypercarbia and acidosis significantly retard the transition to neonatal circulation. This keeps the shunts open and maintains a state of high pulmonary vascular resistance, poor oxygenation combined with blood-mixing.

Thus it can be seen that prompt active intervention is required in the newborn, to increase oxygenation, reduce hypercarbia and perfuse vital tissues and reduce the progressive acidosis, all of which tend to maintain the fetal circulation.

**PREDICTING THE NEED FOR FETAL RESUSCITATION**
The need for fetal resuscitation can be predicted in about 80% of cases providing adequate antepartum and intrapartum assessments are done.

Antepartum assessments include the screening for major fetal anomalies and identifying maternal factors that influence fetal viability. It is safe to comment that one should always be prepared to resuscitate the newborn, but specific identifiable factors increase this preparation because they indicate high risk.

| MATERNAL CONDITION | Diabetes or gestational diabetes  
| Chronic hypertension  
| Pregnancy-induced hypertension (PET)  
| Maternal infection  
| Maternal drug therapy  
| Maternal substance abuse (alcohol!!) |
|---|---|
| PREGNANCY FACTORS | Previous stillbirth  
| Previous Rh sensitization  
| Lack of prenatal care/late booking  
| Antepartum haemorrhage (2nd/3rd trimester)  
| Preterm or post-term gestation  
| Size-dates discrepancy |
| FETAL FACTORS | Multiple gestation  
| Known fetal anomalies  
| Oligohydramnios  
| Polyhydramnios |
Intrapartum factors associated with a need for neonatal resuscitation constitutes a long list, but can be basically summarized to ANY INTRAPARTUM COMPLICATION AND ANY INDICATION FOR A CAESAREAN SECTION.

The most common predictors for the need to intubate the neonate are low infant weight and administration of general anaesthesia to the mother.

NEONATAL RESUSCITATION PROTOCOL

The following protocol is that which is suggested by the American Heart Association/American Academy of Pediatrics.

**0sec**

BIRTH

- Clear of meconium?
- Breathing or crying?
- Good muscle tone?
- Colour pink?
- Term gestation?

**YES**

Routine care:
- Provide warmth
- Clear airway
- Dry

**NO**

Provide warmth
- Position, clear airway
- Dry, stimulate, reposition
- Give O₂ as necessary

**30sec**

Evaluate respirations, heart rate and colour

- Breathing, pink HR > 100
- Apnoea or HR < 100
- Ventilating, pink HR > 100

**Supportive care**

**60sec**

Provide positive pressure ventilation

- HR < 60
- HR > 60

**Supportive care**

**90sec**

Administer adrenaline

- HR < 60
- HR > 60
A summary of this protocol with applicable timelines is:

- The initial steps include assessment of the overall condition of the neonate and minimizing heat loss
- Within the first 20 sec after birth, the neonate must be dried, placed under a warmer and suctioned (mouth and nose)
- Within the first 30 sec after birth, respiration is assessed and any problem treated
- Within the first minute of birth, the neonatal heart rate is assessed

Let us have a short look at these points in turn.

- The emphasis on minimizing heat loss is because cold stress in the neonate results in hypoxemia, hypercarbia and metabolic acidosis, all of which hinder resuscitation by promoting the persistence of the fetal circulation. This is compounded in the depressed, asphyxiated neonate due to their unstable thermoregulatory systems.
- On respiratory assessment, the response to a gasping or apneic neonate should be positive pressure mask ventilation (PPMV) at 40-60 breaths per minute with 100% oxygen. Most neonates will respond to these first 2 steps. Be aware that it is often necessary to decompress the stomach after PPMV by inserting an oro/nasogastric tube. Failure to do this may result in splinting of the diaphragm, hypoventilation and hypoxemia.
- Indications for endotracheal intubation are ineffective bag-mask ventilation, anticipating the need for prolonged mechanical ventilation and as a route for administering emergency medications!
- It is interesting to note that the LMA has been successfully used in a number of clinical trials involving neonatal resuscitation. Time for insertion was generally less than 10 sec, audible leaks occurred at ± 22 cmH2O. Positive pressure ventilation was easily managed without gastric distension. This might be a good accessory airway to keep in reserve, especially for a difficult neonatal airway, eg Pierre-Robin syndrome.
- It is important to note that it is no longer recommended that the presence of meconium requires routine intubation and suction. Tracheal suctioning is now only recommended if there is meconium AND the baby is not vigorous.
- Chest compressions are only done AFTER at least 30 sec of PPMV and the heart rate remains below 60. Chest compressions are done at a rate of 90 per min and should be stopped when the heart rate is above 60. The compression-ventilation ratio should be 3:1 (ie 90:30 per min), applied to the lower ⅓ of the sternum and producing a compression depth of about ⅓ of the AP diameter.
- NB! The resuscitation should not be stopped to do an Apgar evaluation.
- Medications must be considered if the heart rate remains below 60 after 30 sec of effective compressions and adequate ventilation with 100% O₂

**MEDICATIONS**

**OXYGEN** must be regarded as a drug, with its own side-effects. In the scenario of resuscitation of the newborn, it is acceptable to use 100% O₂ for a short period, but this must not be continued as a maintenance drug. Even short exposures to 100% O₂
may result in the formation of highly toxic free radicals, resulting in pulmonary and retinal damage.

NALOXONE is indicated specifically for respiratory depression due to maternal opioids, but not in the case of a narcotic-addicted mother.

ATROPINE is generally not indicated in neonatal resuscitation, because bradycardia in the newborn is almost always related to hypoxia and not vagal stimulation.

SODIUM BICARBONATE should only be given if the neonate is being adequately ventilated AND documented or presumed metabolic acidosis does not respond to other measures, otherwise respiratory acidosis will replace metabolic acidosis.

VOLUME EXPANDERS are rarely indicated in neonatal resuscitation. They are specifically used where there is acute blood loss with signs of shock. They may be detrimental in other scenarios.

DEXTROSE should be remembered as a drug if the neonate is lethargic at birth without an obvious cause. Approximately 10% of healthy term neonates may experience transient hypoglycaemia, more especially in the diabetic mother, the mother who received dextrose infusions in labour, macrosomic babies and neonates who are pre- or post-term.

Medications can be given via peripheral veins, umbilical veins or via the ET tube.

**ENDOTRACHEAL MEDICATIONS**

The following drugs may be given endotracheally and can easily be remembered with the mnemonic LANE:
- Lignocaine
- Atropine
- Naloxone
- Epinephrine (Adrenaline)

**NB!** Effects of endotracheal adrenaline are delayed by about a minute as compared to intravenous administration. Unlike the recommendations in pediatric resuscitation, where up to 3x the intravenous dose is administered endotracheally, in the neonate THE SAME DOSE IS GIVEN INTRAVENOUSLY OR ENDOTRACHEALLY (0.01mg/kg). This is to prevent the devastating complication of an intracranial haemorrhage associated with adrenaline-induced hypertension.

<table>
<thead>
<tr>
<th>MEDICATION</th>
<th>CONCENTRATION</th>
<th>DOSAGE/ROUTE</th>
<th>RATE</th>
</tr>
</thead>
<tbody>
<tr>
<td>Adrenaline</td>
<td>1:10 000 (1 ampoule diluted to 10ml)</td>
<td>0.1-0.3ml of the diluted solution per kg, IV or via ET tube</td>
<td>Give rapidly and flush with saline</td>
</tr>
<tr>
<td>Naloxone</td>
<td>0.4mg/ml</td>
<td>0.1mg/kg IV or via ET tube</td>
<td>Give rapidly</td>
</tr>
<tr>
<td>Sodium bicarbonate</td>
<td>4.2% solution (0.5mEq/ml)</td>
<td>2mEq/kg (4ml/kg) IV and ensure effective ventilation</td>
<td>Give slowly, at least over 2min</td>
</tr>
<tr>
<td>Dopamine</td>
<td>200mg in 200ml N/S =1mg/ml =1000µg/ml</td>
<td>2-20µg/kg/min IV</td>
<td>Give as a titrated infusion</td>
</tr>
<tr>
<td>Volume expanders</td>
<td>O-negative blood or normal saline</td>
<td>10ml/kg IV</td>
<td></td>
</tr>
</tbody>
</table>
Identifying neonates at risk, being prepared for resuscitation every time you enter labour ward, following the protocol and knowledge of the drugs and fluids you use will make you adept at neonatal resuscitation and give the neonate the best chance to survive the transition without morbidity.

Lastly, but most importantly, the mother is not to be forgotten in the chaos of a difficult resuscitation. As an anaesthetist, your first priority remains the mother. In the event of a neonatal resuscitation and an unstable or critically ill mother, YOU MUST attend to the mother first and foremost. Pre-empting this situation by organising a paediatrician is preferable, otherwise the attendant staff must continue with the RESUSCITATION PROTOCOL as best as they are able.
MANAGING THE PREGNANT HIV+ PATIENT

South Africa has one of the highest, if not the highest, rates of HIV/AIDS infection in the world, with women of reproductive age being the fastest growing population with HIV. Women comprise 58% of infected adults.

Pregnant patients are thus in the high-risk patient populations due to their age and sexual activity. As an anaesthetist, you will undoubtedly face these patients frequently, both as emergencies and non-emergencies.

HIV infection must be seen as a multi-system disease. The degree to which each system is involved in the disease process is highly variable and dependent on:
- Duration of HIV infection
- CD4 count and viral load
- Underlying comorbid disease
- Nutritional state
- Whether the patient is on the ARV programme and compliant with therapy

Thus you may experience pregnant HIV+ patients who are clinically well, with no underlying disease, good nutritional state and no added risks; similarly you may come across an extremely ill HIV+ patient, with advanced AIDS-related disease, wasted, with severe infection and requiring an urgent Caesarean section.

It is thus mandatory to have a working knowledge of how HIV disease affects organ systems and what this implies for the safe conduct of anaesthesia.

EFFECT OF HIV ON ORGAN SYSTEMS

It is important to realise that patients on ARV’s may still have many of the clinical manifestations of HIV disease, but these may be subclinical or moderated. ARV therapy itself may cause problems and this should also be taken into account. In South Africa, the ARV rollout is picking up momentum, thus we can expect the present gross pathology seen with untreated HIV disease to change into more subtle presentations of the disease.

CARDIOVASCULAR

Up to 50% of HIV-infected patients may have subclinical cardiac involvement, thus cardiac evaluation is important.
Dilated cardiomyopathy has been found in 30-40% of HIV+ patients on routine echocardiography and pulmonary hypertension in 0.5%. HIV-related cardiomyopathy has not been shown to respond to ARV therapy.
Cardiac autonomic neuropathy may occur in up to 15% of patients.
Accelerated coronary arteriosclerosis occurs, which may result in early myocardial infarction or angina in young patients.
Other prominent manifestations are pericardial effusion, myocarditis due to opportunistic infections and endocarditis in IV drug abusers.
KEY POINTS:
The cardiovascular system may be severely impaired and because these patients are mainly young, it is unexpected and not looked for. Specifically look for CVS symptoms and signs, especially of cardiac failure; specifically ask about effort intolerance, dysrhythmias and chest pain (angina). Spinal anaesthesia should be avoided in HIV+ pregnant patients with CVS symptoms/signs and these patients should preferably be referred to a main centre for proper cardiac evaluation. Obtain specialist advice in the emergency situation before proceeding with anaesthesia, because spinal anaesthesia may be contra-indicated.

PULMONARY
Pulmonary problems are caused mainly by opportunistic infections. In South Africa this is mainly bacterial pneumonia and TB, with PCP (pneumocystis carinii pneumonia) having a much lower incidence. This relationship is the reverse overseas, with PCP being the most common opportunistic lung infection. PCP incidence is now also much lower due to the use of ARV’s and prophylactic cotrimoxazole.

The incidence of HIV-associated TB is increasing, especially among women of childbearing age. Unfortunately, the presentation of many of these TB infections are atypical, leading to late diagnoses and advanced pathology.

KEY POINTS:
Active TB and bacterial pneumonias are frequent and should be looked for, since typical symptoms are often absent.
Patients may present acutely in respiratory failure.
Remember that dyspnoea and/or lung signs totally out of keeping with the XR picture may be PCP (CXR may be normal in up to 40% of PCP infections)
Clinical assessment of pulmonary function is needed before embarking on anaesthesia – postoperatively the patient may require respiratory support.

NEUROLOGICAL
HIV is a neurotropic virus, affecting the CNS early in the disease, producing headaches, photophobia, depression, meningo-encephalitis and mild cognitive dysfunction.
CSF is infected early. (spinals!)
Cranial and peripheral neuropathies are common (35%), myopathy is frequent and an HIV-associated Guillain-Barre-like syndrome may occur.
Late CNS disease may include cryptococcal or TB-meningitis, dementia, myopathy, encephalopathy and CNS neoplasms, resulting in raised ICP and cerebral oedema. ARV treatment may also induce a myopathy.

KEY POINTS:
The CNS is affected early and neuropathies and myopathies are frequently seen, thus use of muscle relaxants (Sux and NDP’s) must be used judiciously. There is no contra-indication to either regional techniques or epidural blood patches on grounds of HIV infection alone, but these techniques should be avoided in cerebral disease or where raised ICP/brain oedema is suspected. Patients may be more predisposed to postoperative confusion.
GASTROINTESTINAL
Dysphagia is common due to candida oesophagitis.
Other common pathogens may affect the oropharynx or rest of the GI tract eg. herpes, CMV, histoplasmosis.
Kaposi’s sarcoma and squamous cell carcinoma are common in the oropharynx.
Patients are prone to gastro-oesophageal reflux, especially in advanced disease.
Upper GI pain due to candidiasis, oral lesions and odynophagia reduce oral intake and patients are often dehydrated.
Frequent diarrhoea also predisposes patients to electrolyte abnormalities.
Liver function tests are often raised, due to infection or as an indication of decreased functional ability in advanced disease.

KEY POINTS:
Patients are usually malnourished and dehydrated due to nausea, poor intake and loss of appetite. Electrolyte levels should thus be checked in any patient with advanced disease, dehydration, wasting or recent poor oral intake.
The risk of reflux and aspiration on induction should be considered in these patients.
Hypotension or hypovolemia may worsen liver perfusion and result in ischemic hepatitis of an already poorly functioning liver.

RENAL
HIV patients are at risk for various renal diseases due to the disease itself, associated hepatitis, ARV drugs, dehydration and drug abuse. A nephrotic syndrome-like condition called HIV-associated nephropathy exists which rapidly progresses to end-stage renal failure.

KEY POINTS:
Renal impairment is common; hypotension and hypovolemia should be avoided to prevent compounding renal dysfunction.
Urea, electrolytes and creatinine values should be obtained routinely.

ENDOCRINE AND METABOLIC
Primary or secondary adrenal insufficiency is the most serious endocrine problem in these patients.
Abnormal thyroid function tests are frequent, but clinical hypothyroidism is rare.
Hypoglycaemia may occur due to protease inhibitors, pentamidine treatment or hypopituitarism.
Some of the protease inhibitors may cause a metabolic syndrome with raised triglycerides.

KEY POINTS:
Hypotension not responsive to volume loading and vasoconstrictors should be considered to be secondary to adrenal insufficiency and the patient should be given IV hydrocortisone.
Considering the frequency of hypoglycaemia, it would be prudent to monitor glucose levels, especially in prolonged procedures.
HEMATOLOGIC
A wide range of hematological abnormalities may occur at any stage of the disease and may be due to multiple factors. Anaemia, leukopenia, lymphopenia and thrombocytopenia occur commonly. Bone-marrow suppression may be due to infections, ARV and other drugs or nutritional factors.
Bone-marrow infiltration occurs with some neoplasms. Patients are at risk for hypercoagulability with thrombo-embolic events and these are predisposed to by co-existing malignancies, auto-immune disease and ARV therapy.
A specific coagulation abnormality seen is ITP (idiopathic thrombocytopenic purpura).

KEY POINTS:
It is preferable to have a FBC, platelets and clotting profile on patients with advanced disease, especially if regional techniques are to be used. At least an Hb and platelet count should be obtained in all HIV+ cases to undergo regional anaesthesia. If oral petechiae are seen, remember that the laryngeotracheal mucosa is probably in the same condition; thus all instrumentation or manoeuvres of the airway should be careful and gentle to prevent bleeding and the development of an iatrogenically difficult airway. ARV treatment may also induce a myopathy. Remember the higher risk for thromboembolism; anticoagulation should be considered in moderate to high-risk surgery.

THE AIRWAY IN HIV+ PATIENTS
The airway must be considered as an affected ‘organ’ in HIV/AIDS patients.
As discussed above under the GI tract, HIV may have a number of effects on the oropharynx – obviously then these conditions may also impact on the airway management of HIV/AIDS patients. At some stage in the course of their disease, 75-100% of HIV/AIDS patients will have a head or neck manifestation that may impact on anaesthetic or airway management.
Thus, proper and thorough airway evaluation of any and every HIV-infected patient is mandatory – in pregnant patients, this is doubly so. Using the WHO staging of HIV/AIDS and correlating disease as a guide, one may expect exposure to the following conditions in the airway:

STAGE 1: Persistent generalized lymphadenopathy
Bilateral parotid hypertrophy

STAGE 2: Cutaneous manifestations
- abscesses
- varicella zoster (shingles)
- dermatitis
Mucocutaneous manifestations
- gingivitis with/without petechial bleeding
- oropharyngeal ulceration (herpetic, aphthous)
- advanced dental caries and dental abscesses

Infections
- upper airway
- sinusitis

STAGE 3: Severe bacterial infections
- Dental abscesses
- Ludwig’s angina
- Deep neck space infections which may progress to necrotizing fasciitis with high mortality

Oropharyngeal candidiasis (may cause candidial epiglottitis)
Oral hairy leucoplakia
Pulmonary TB

STAGE 4: Neoplastic conditions
- Kaposi’s sarcoma (may be multiple oral or orotracheal lesions which bleed easily)
- Burkitt’s (massive tumours) and non-Hodgkins lymphomas

Viral conditions
- persistent herpetic ulceration (often large ulcers)
- molluscum contagiosum
- CMV

Bacterial conditions
- fulminating pneumonias (± lung abscesses)
- TB, incl. Multi-drug resistant TB, miliary/disseminated TB

Fungal conditions
- florid oesophageal or orotracheal candidiasis which may result in stricture formation

It is important to note that many of these conditions are accompanied by lymphadenopathy, which may be severe. Pulmonary TB may cause mediastinal lymphadenopathy with compression of the tracheobronchial tree; large cervical TB nodes (scrofula) may also occur.

As is seen from the above extensive list of conditions, airway management may be problematic from early on in HIV disease. It is also very important to realise that you as anaesthetist are exposed to all of these conditions during the course of your normal duties – thus you must exercise as many protective barrier methods as feasible – gloves, masks, goggles, gowns etc.
EFFECTS OF HAART

The list of drugs being used for the treatment of HIV/AIDS is ever expanding, including the group of highly active anti-retroviral treatment (HAART) group. These drugs are potent and have side-effects that may also influence your anaesthetic management, but have reduced opportunistic infections, increased lifespan and greatly decreased mother to child transmission.

Some side-effects may mimic signs and symptoms of the disease itself, eg myopathy, neuropathy, pancytopenia and GI disturbances. Others result in hyperglycemia, changes in fat distribution (buffalo hump or facial wasting) and changes in enzyme systems, esp cytP450. The table below indicates the main side-effects.

HAART DRUGS AND SIDE-EFFECTS

<table>
<thead>
<tr>
<th>DRUG</th>
<th>SIDE-EFFECT</th>
</tr>
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<tbody>
<tr>
<td><strong>NUCLEOSIDE ANALOGUES</strong></td>
<td></td>
</tr>
<tr>
<td>Zidovudine</td>
<td>Headache, nausea and vomiting, myalgias, bone-marrow suppression, myopathy, cytochrome p450 inhibition</td>
</tr>
<tr>
<td>Lamivudine</td>
<td>Generally well-tolerated; headache, diarrhoea, peripheral neuropathy</td>
</tr>
<tr>
<td>Stavudine</td>
<td>Peripheral neuropathy, pancreatitis</td>
</tr>
<tr>
<td>Tenofovir</td>
<td>Renal toxicity</td>
</tr>
<tr>
<td>Didanosine</td>
<td>Diarrhoea, peripheral neuropathy, pancreatitis</td>
</tr>
<tr>
<td><strong>PROTEASE INHIBITORS</strong></td>
<td></td>
</tr>
<tr>
<td>Indinavir</td>
<td>Kidney stones, cytochrome p450 inhibition, hyperglycemia, changes in fat distribution</td>
</tr>
<tr>
<td>Saquinavir</td>
<td>Diarrhoea, headache, cytochrome p450 inhibition, hyperglycemia, lipodystrophy</td>
</tr>
<tr>
<td>Ritonavir</td>
<td>Cytochrome p450 inhibition, elevated triglycerides, hyperglycemia, raised liver enzymes</td>
</tr>
<tr>
<td>Atazanavir</td>
<td>Diarrhoea, jaundice</td>
</tr>
<tr>
<td><strong>NON-NUCLEOSIDES</strong></td>
<td></td>
</tr>
<tr>
<td>Efavirenz</td>
<td>Skin rash, raised liver enzymes, GI disturbances, potent teratogenic</td>
</tr>
<tr>
<td>Nevirapine</td>
<td>Skin rash, raised liver enzymes, GI disturbances, <em>induces</em> cytochrome p450</td>
</tr>
</tbody>
</table>

The effects of other commonly used drugs should also not be forgotten, eg:
- Cotrimoxazole → skin rash, raised liver enzymes
- Antifungals → lengthened QT time,
- Anti-TB drugs → raised hepatic enzymes, thrombocytopenia
HAART DRUG INTERACTION WITH ANAESTHESIA

Inhibitors of cytochrome p450 enzymes impair the metabolism of many anaesthetic drugs, including midazolam, fentanyl and amiodarone. This increases free drug levels and delays breakdown, so toxic levels or increased effect and duration of effect are seen.

Inducers of cytochrome p450 (only Nevirapine) have the opposite effect to the above and thus decrease the levels and clinical effect of the drugs mentioned.

Etomidate, desflurane, atracurium, cis-atracurium and remifentanil are all drugs that are metabolized independently of the cytochrome p450 enzymes and thus are useful in these patients.

Patients with raised hepatic enzymes, renal or hepatic impairment should be managed with drugs that do not have further effect on these organs and care should be taken to maintain adequate organ perfusion by normovolemia and normotension intraoperatively. Avoid drugs whose main means of metabolism is by the affected organ.

ANAESTHETIC MANAGEMENT

- **Adequately assess the patient** - specifically look for signs of opportunistic infections, the most common comorbidities (esp CVS) and obvious neoplasms.

- **Is the patient on HAART?** Which drugs are being taken and what is the most recent CD4 count? (A patient with a CD4 count <50 has a postoperative mortality rate of 13% at 6months, irrespective of the procedure, compared to 0.8% where the count is >200).

- **Assess the airway!**

- **Investigations:** if possible, always get a FBC (alternatively a lab Hb), with platelet count, and u+e’s. A CxR and ECG is indicated if there are any respiratory problems. An Echo is indicated if the patient has symptoms or signs of cardiac disease. It is important to evaluate properly, because the normal physiologic cardiac changes of pregnancy are not included here. It is impossible to refer every patient with a murmur for an Echo.

- **Decide on your anaesthetic technique.** Generally this will be a regional technique for Caesarean section, BUT this is not written in stone. The usual contra-indications count, but others also exist in these patients, eg any CNS infection, encephalopathy, myelopathy or suspicion of raised ICP. Regional anaesthesia is probably preferable in patients with respiratory disease, but care should be taken to avoid a high motor block, because knocking out intercostals muscle function will not be tolerated well.
Patients with neuropathy or myopathy are probably better candidates for general anaesthesia. Patients with dementia or neuropsychiatric symptoms should undergo GA, but it should be remembered that they will be more sensitive to sedatives and psychoactive drugs and more prone to postoperative confusion.

- It is important to note that although the use of Sux is generally avoided in patients with muscle wasting or progressive neuropathy/myopathy, there is no literature to support this in HIV-related disease, thus Sux use is not contra-indicated in these patients.

- Ensure your patient is normovolemic and manage intraoperative fluid losses and deficits aggressively. Intraoperative hypotension or hypotension secondary to spinal anaesthesia should likewise be aggressively managed.

- In the emergency Caesarean section, many of the components of a planned procedure fall away and there is little time to wait for special investigations. Do an adequate assessment and if possible, contact a senior or specialist if indicated. Manage the patient as well as you possibly can with a well-conducted anaesthesia, sticking to basic principles.

- Protect yourself: Anaesthetists are renowned for their poor measures to protect themselves. Remember that HIV is only one of a number of transmissible diseases against which you should safeguard yourself. (See the list of Do’s and Don’ts below)

<table>
<thead>
<tr>
<th>SHARPS</th>
<th>DO</th>
<th>DO NOT</th>
</tr>
</thead>
<tbody>
<tr>
<td>Have a sharp bin or kidney dish at hand for direct disposal after use (incl spinals, CVP’s, etc) Train your anaesthetic nurse to follow the same precautions and to have means to dispose sharps at the ready</td>
<td>…reseath a used needle. …stick the used needle into the bed’s mattress as protection. …walk around with a sharp. …hand a sharp to somebody else to discard.</td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>FOOTWEAR</th>
<th>DO</th>
<th>DO NOT</th>
</tr>
</thead>
<tbody>
<tr>
<td>Wear waterproof shoes</td>
<td>…wear open shoes without socks</td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>MASKS</th>
<th>DO</th>
<th>DO NOT</th>
</tr>
</thead>
<tbody>
<tr>
<td>Wear masks during airway management and every procedure</td>
<td>…touch your mask with wet or bloodsoiled hands/gloves – change masks if soiled</td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>EYEWEAR</th>
<th>DO</th>
<th>DO NOT</th>
</tr>
</thead>
<tbody>
<tr>
<td>Wear eyeshields, glasses or goggles during all procedures, including airway management</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>DO</td>
<td>DO NOT</td>
</tr>
<tr>
<td>----------------------</td>
<td>-----------------------------------------</td>
<td>------------------------------------------------------------------------</td>
</tr>
<tr>
<td><strong>GOWNS</strong></td>
<td>Wear gowns or other protective clothing</td>
<td>…continue to wear clothing soiled with blood/body fluids – change clothing immediately</td>
</tr>
</tbody>
</table>
| **SHARP BINS**       | Always have one ready, not more than 75% full | …attempt to force sharps into a full sharp bin  
…place normal used drug syringes in a sharp bin |
| **STAFF AND COLLEAGUES** | Be strict and consistent with all theatre staff’s sharps management | …allow a colleague to be lax with sharps, even if they are senior. The rules are for everybody’s safety. |
FLUID AND BLOOD THERAPY

GENERAL PRINCIPLES
It is good to have a general approach to fluid therapy as well fixed guidelines when it comes to fluid resuscitation. However, these remain general guidelines and must be modified to suit the patient in front of you.

FLUID RESUSCITATION
In the dehydrated, hypovolemic or shocked patient, aggressive fluid resuscitation is the norm and may mean the difference between life and death.

The ATLS guidelines are a good basis for fluid resuscitation. Its basis is the administration of fluid volume to decrease the morbidity and mortality of shocked patients by increasing their organ perfusion so as to maintain vital organ function. This is not definitive, because the cause for the shock has not yet been removed, ie fluid resuscitation buys you time to organise definitive treatment while preventing vital organ failure. A good example of this is the fluid resuscitation of a ruptured ectopic pregnancy until surgical intervention.

BASIC PRINCIPLES
- ABC’s – ensure the airway is patent, breathing is not impaired and supplement with oxygen
- Monitoring of vitals – pulse, BP, respiratory rate, temperature, level of consciousness (± pain score and urine output)
- Establish GOOD venous access – 2 large bore IV lines (absolute minimum bore is the green 18G, but preferably grey 16G or larger) in the antecubital fossae
- Rapid initial administration of resuscitation fluids (20ml/kg of crystalloids ie saline or Ringers = 1-2l in an adult; or 10ml/kg of a colloid fluid = 500ml-1l in an adult)
- Monitor for an effective response to fluid resuscitation (decrease in pulse rate, widening of pulse pressure, increase in BP, increase in urine output and increased level of consciousness) and tailor further fluid administration accordingly.
- Make an early decision regarding the need to transfuse blood.

EVALUATING SHOCK
Rapid clinical evaluation of shock gives us a basis on which to apply fluid resuscitation. Most obstetric causes of shock are haemorrhagic in nature, thus the emphasis in this chapter will be for this type of shock.

There are a number of guidelines to use but a modified version of that used in ATLS will be used here.

The volume of blood per kilogram body mass is ± 70ml/kg. This is preferably calculated on expected lean body mass rather than true mass in obese individuals. Using this information allows us to roughly calculate the volume and % of blood lost based on clinical signs.

Thus in an average adult of 70kg, the following blood volumes apply. These can easily be recalculated for other masses using multiples of 10kg to simplify things.

<table>
<thead>
<tr>
<th>% BLOOD LOSS / APPROXIMATE VOLUME (ADULT)</th>
<th>&lt;15% (&lt;750ml)</th>
<th>15-30% (750ml-1.5l)</th>
<th>30-40% (1.5-2.0l)</th>
<th>&gt;40% (&gt;2.0l)</th>
</tr>
</thead>
<tbody>
<tr>
<td>VITAL SIGN</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Heart rate</td>
<td>&lt;100</td>
<td>100-120</td>
<td>&gt;120</td>
<td>&gt;140</td>
</tr>
<tr>
<td>Systolic BP</td>
<td>Normal</td>
<td>Normal</td>
<td>Decreased</td>
<td>Decreased</td>
</tr>
<tr>
<td>Pulse pressure</td>
<td>Normal to</td>
<td>Decreased</td>
<td>Decreased</td>
<td>Decreased</td>
</tr>
<tr>
<td>Capillary refill</td>
<td>Decreased</td>
<td>Decreased to absent</td>
<td>Absent</td>
<td></td>
</tr>
<tr>
<td>Respiratory rate</td>
<td>14-20</td>
<td>20-30</td>
<td>30-40</td>
<td>&gt;35</td>
</tr>
<tr>
<td>CNS status</td>
<td>Normal to</td>
<td>Anxious</td>
<td>Anxious +</td>
<td>Confused +</td>
</tr>
<tr>
<td></td>
<td>Anxious</td>
<td>confused</td>
<td>Lethargic</td>
<td></td>
</tr>
</tbody>
</table>
Unfortunately, this is a very simplistic approach and starts with the assumption that the initial physiology base is normal. This might hold true for early pregnancy eg in ruptured ectopic pregnancies or incomplete abortions, but is difficult to apply in late pregnancy, comorbid conditions or in complicated pregnancies. However, the principles behind the guidelines remain true and knowing these makes your management of fluid resuscitation a lot more effective.

CHOICE OF FLUIDS

The choice of fluids is an area of controversy when it comes to arguing whether crystalloids or colloids are more effective in fluid resuscitation. However, we know that the basis of fluid resuscitation is expansion of intravascular volume and both types of fluids achieve this.

A suggested approach acceptable to many clinicians is to start with rapid crystalloid infusion, monitor the response and then to use a combination of crystalloids and colloids for further fluid administration.

Patients in severe haemorrhagic shock, but who respond to fluid resuscitation, are often not transfused or “pushed” with fluid in an attempt to normalise vital signs. Acceptably low systolic pressures are maintained to allow vital organ perfusion until surgical intervention gains control of the cause for the haemorrhage – so-called “hypotensive resuscitation”. This does not mean that blood is not ordered or withheld, only that it is utilized to the best advantage of the patient. A perfect example of the use of this technique is in ruptured ectopic pregnancies, where the blood is preferably given when the bleeding salpinx is clamped.

NB! THIS TECHNIQUE OF RESUSCITATION MAY BE CRITICAL IN A SMALL HOSPITAL WHERE THE ONLY BLOOD AVAILABLE IS THE STANDARD 2UNITS OF EMERGENCY O-NEGATIVE PACKED CELLS.

Crystalloids – the crystalloids used generally for resuscitation are the replacement-type balanced salt solutions like 0.9% saline or Ringer's lactate; these are given in a 3:1 ratio of fluid to the approximated blood volume lost. Ringer's lactate is by far the more physiological fluid, especially when given in large volumes. The lactate in it is readily converted into bicarbonate by the liver. 0.9% saline solution has high concentrations of sodium and chloride and when given in large volumes produces a hypernatremic hyperchloremic metabolic acidosis.

Transit time of crystalloids in the intravascular space is generally very short (intravascular half-life of 20-30min). Once large volumes of crystalloid fluids have been given, the plasma proteins are diluted and their oncotic effect in retaining fluid intravascularly decreases. This situation is worse in patients with acute depletion of intravascular volume (as in haemorrhage), since crystalloid fluid resuscitation does not promote increased oncotic pressure – thus the tendency is for this fluid to move interstitially and cause tissue oedema. This is self-perpetuating because further crystalloid fluid administration will further decrease plasma protein concentration and hasten fluid movement into the tissues. This is the reason why many clinicians will switch to colloid fluids after initial resuscitation with crystalloids.

In many medical texts, a dogmatic 3-4l of crystalloid fluid resuscitation is suggested before switching to colloids. However, this does not take into account the formation of tissue oedema and the resulting tissue hypoxia. This is acceptable if no colloid fluid is available and resuscitation is ongoing. However, where colloids are available, the switch should be relatively early. Newer trials indicate that a 20ml/kg initial crystalloid fluid administration should then rather be followed by a combined crystalloid-colloid fluid administration, than either only crystalloids or only colloids.

Colloids – these fluids may be synthetic or natural and contain high molecular weight substances. These substances both raise the oncotic pressure and have osmotic effects, which helps retain free fluid within the intravascular space. Compared to the crystalloids,
Colloid fluid transit time is much longer, with an intravascular half-life varying between 3 and 6 hrs, depending on the type of fluid used. However, they are expensive and have complications associated with their use. Many of the synthetic colloids are prepared in 0.9% saline and thus share the problem of hyperchloremic acidosis when large volumes are administered. Synthetic colloids are dextrans, gelatins or starches, while the ‘natural’ colloids are blood-derived and include human blood, FFP’s, cryoprecipitate, freeze-dried plasma and albumin.

They are given in a volume ratio of 1:1 of colloid fluid to the approximate blood volume they are required to replace.

**Dextrans:** in South Africa, dextrans are not popular for use in fluid resuscitation. They have anti-platelet effects, prolong bleeding time, interfere with blood grouping (>20ml/kg/day), are associated with renal dysfunction and are antigenic, associated with anaphylactic reactions.

**Gelatins:** the popular available gelatin in South Africa is Gelofusine®. Gelatins are made from bovine collagen extracts and have been associated with histamine-mediated allergic reactions, but have little to no effect on platelets, bleeding time or blood grouping. They are effective plasma expanders especially in haemorrhagic shock.

**Starches:** these are hydroxyethylated polysaccharide compounds made from plant starches. They are non-antigenic and anaphylactoid reactions are rare. Administration of large volumes have effects on coagulation and platelet function, but not on blood grouping. The popularly used starches in South Africa are Haesteril® (being phased out), Voluven® and a brand new product called Venofundin®. All are effective plasma expanders.

**Blood-derived colloids:** these are all expensive products and are all associated with allergic reactions due to the high antigenic nature of human proteins. Infective risks are low with albumin and freeze-dried plasma, due to heat treatment, but all other blood products have an associated risk of infection. Freeze-dried plasma (FDP) warrants special mention for its long shelf-life, easy reconstitution and it should be available in all small hospitals as a plasma-protein containing plasma expander. This may be life-saving in obstetric emergencies in rural hospitals especially where the only blood available may be the standard 2 units of O-negative packed cells.

It should also be noted that the routine use of platelets and FFP’s in patients receiving more than one unit of packed cell blood transfusion, cannot be justified. It is often required when a massive blood transfusion or more than one patient blood volume is transfused and there are signs of coagulopathy. This should be done in conjunction with lab results of the clotting profile (INR and/or PTT >1.6x normal values).

**BLOOD TRANSFUSION**

The normal adult blood volume is ±65ml/kg in females and up to 75ml/kg in males. Pediatric blood volumes are ±85ml/kg and neonates 100-120ml/kg.

When should blood be transfused? Blood and blood products are not innocuous and may cause major problems for the recipient. Blood products should be regarded as a scarce resource and therefore not be used irresponsibly.

Previously it was felt that blood should be given when a "transfusion trigger" was reached. This was usually in the form of a measurable parameter, eg Hb or Hct and was usually used empirically (the 10 & 30 rule, indicating Hb and Hct levels).

This is no longer acceptable, considering the risks of blood transfusion. The decision to transfuse or not is based on clinical judgement and depends on the risk-benefit ratio to the patient as decided on by the anaesthetist. Kimberley Hospital now has a Blood Component Transfusion Protocol, which will probably be standardized throughout the Northern Cape.
will help to optimize the use of blood and blood components and prevent wastage of a valuable but scarce resource.

Factors to take into account are:
1. duration of anemia (acute/chronic)
2. intravascular volume status
3. type of injury, if any
4. extent of proposed procedure
5. probability of massive blood loss
6. comorbid conditions eg COPD, IHD, peripheral vascular disease, chronic renal failure

With blood loss, effective compensatory mechanisms are initiated as long as intravascular volume is maintained (acute isovolemic hemodilution). These are the following:

(1) an increase in cardiac output (↑SV due to ↓SVR)
(2) redistribution of blood flow to organs with high oxygen extraction ratios, eg heart, kidneys, brain
(3) an increase in oxygen extraction ratios of some vascular beds, and
(4) alteration of oxygen binding to Hb to allow the Hb to deliver oxygen at lower oxygen tensions.

This fits in with the oxygen delivery formula: \( \text{DO}_2 = \text{CO} \times \text{Hb} \times \text{SaO}_2 \times 1.34 \), where cardiac output can increase in face of decreased Hb to maintain \( \text{DO}_2 \).

At an Hb of 10g%, \( \text{O}_2 \) transport, cardiac output and blood viscosity are optimal, thus transfusing blood above an Hb of 10g% makes poor physiological sense.

Cardiac output increases as compensation for acute isovolemic hemodilution and reaches a peak at ± 180% of resting output as the Hct approaches 20%. This is obviously variable and dependent on age, comorbid conditions and the rate of onset of anemia. Chronic anemias often only start with compensatory mechanisms at Hb 7-8g%.

Tissues with already high \( \text{O}_2 \) extraction ratios (heart, brain) are usually at maximal extraction and thus only benefit by redistribution of blood flow (coronary flow increases 400-600%). Once this limit is reached, the myocardium is prone to injury (even in young healthy patients) by any further decrease in \( \text{O}_2 \) delivery, or increase in \( \text{O}_2 \) demand.

Blood is usually administered as units of packed red cell concentrates, with an Hb of 70% in a 300-350ml volume and containing the anticoagulant CPDA (citrate, phosphate, dextrose-adenine) or ADSOL. Storage life is 35 days with CPDA and up to 45 days with ADSOL. Administration is best through a standard blood administration set with 170µm filter, with normal saline. Saline facilitates infusion, minimizes hemolysis and avoids clumping. Ringers contains calcium which may cause clots.

It is preferable to warm blood during or prior to administration, but if 1 or 2 units are to be infused slowly, warming is not necessary. Warming of blood is mandatory in massive transfusions, pediatric patients and patients with cold agglutinins.

A unit of packed cells will increase Hct by 3-4% and Hb by 1-1.5% in the average adult.

**BLOOD STORAGE**

Blood is stored at 1-6°C, which decreases red cell metabolism. CPDA is added after 250ml plasma is removed from the donated whole blood. CPDA has the following effects on the blood:
- Citrate chelates calcium to prevent coagulation
- Phosphate increases 2,3 DPG and ATP levels
- Dextrose supplies the substrate for glycolysis
- Adenine improves maintenance of ATP levels and the viability of stored cells

Mannitol is added to optimize rheology and osmotic balance.
What happens to blood when it is stored?

Red cell metabolic processes continue, using up all the dissolved O\textsubscript{2}, then cells convert to anaerobic glycolysis. End products are thus CO\textsubscript{2} and lactate. Lysis of some of the cells raises the K\textsuperscript{+} level by ±1mmol/l/day of storage. Simultaneously the pH decreases, with plasma bicarbonate decreasing to buffer the CO\textsubscript{2} increase (Henderson-Hasselbach).

These metabolic changes are called the **storage defect/lesion**.

<table>
<thead>
<tr>
<th>Test</th>
<th>Day1</th>
<th>Day7</th>
<th>Day14</th>
<th>Day21</th>
</tr>
</thead>
<tbody>
<tr>
<td>Blood PCO\textsubscript{2}</td>
<td>48</td>
<td>80</td>
<td>110</td>
<td>140</td>
</tr>
<tr>
<td>Blood pH</td>
<td>7.1</td>
<td>7.0</td>
<td>7.0</td>
<td>6.9</td>
</tr>
<tr>
<td>Blood lactate</td>
<td>41</td>
<td>101</td>
<td>145</td>
<td>179</td>
</tr>
<tr>
<td>Plasma bicarbonate (meq/ml)</td>
<td>18</td>
<td>15</td>
<td>12</td>
<td>11</td>
</tr>
<tr>
<td>Plasma K\textsuperscript{+} (meq/ml)</td>
<td>3.9</td>
<td>12</td>
<td>17</td>
<td>21</td>
</tr>
<tr>
<td>Plasma dextrose (mg/dl)</td>
<td>345</td>
<td>312</td>
<td>181</td>
<td>131</td>
</tr>
<tr>
<td>Plasma Hb (mg/dl)</td>
<td>1.7</td>
<td>7.8</td>
<td>13</td>
<td>19</td>
</tr>
<tr>
<td>2,3 DPG (nM/ml)</td>
<td>4.8</td>
<td>1.2</td>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td>Platelets (%)</td>
<td>10</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Factor V and VIII (%)</td>
<td>70</td>
<td>50</td>
<td>40</td>
<td>20</td>
</tr>
</tbody>
</table>

These changes are important to consider when massive blood transfusions are involved in patient management (see later).

**MASSIVE BLOOD TRANSFUSIONS:**

Definitions: a large and rapid transfusion in relation to the patient’s blood volume, ie

- > 35ml/kg/hr
- > 0.5 x the patient’s blood volume in 1hr
- > 1 x the patient’s blood volume in 24hrs

Problems related to massive blood transfusion can be classified as follows:

1. Metabolic changes related to the storage defect.
2. Coagulopathy
3. Infective risks

1. **METABOLIC CHANGES:**

- **Hypothermia** is a real risk if transfusion is rapid (>150ml/min), especially in shocked and already hypothermic patients. Cold blood has a direct effect on cardiac conduction (CVP!), causing VF by SA and AV nodal block. This can be prevented by prewarming or warming during transfusion. Hypothermia also worsens other metabolic effects eg decreased tissue O\textsubscript{2} delivery (L-shift of Hb-O\textsubscript{2} curve), decreased citrate metabolism, pH and electrolyte changes, decreased cardiac output and platelet dysfunction.

- **Metabolic acidosis** occurs mainly due to patient shock and hypoperfusion, rather than transfused blood. The low pH of transfused blood (CO\textsubscript{2}, lactate and citric acid) is offset by optimal patient ventilation and rapid hepatic metabolism of lactate and citrate to bicarbonate, causing metabolic alkalosis.

- **Hyperkalemia** may occur in massive blood transfusions. A unit of packed cells has a K\textsuperscript{+} load of ±7mmol. However, this is offset by warming, which causes rapid intracellular shift of K\textsuperscript{+}. Low potassium levels often occur secondary to metabolic alkalosis.

- **Citrate toxicity** occurs due to citrate in transfused blood causing hypocalcemia and myocardial depression. It is not common because of rapid hepatic metabolism of citrate and the rapid response of calcium homeostasis through PTH. Thus routine calcium administration is not recommended, unless acute Q or T wave changes indicative of hypocalcemia occur on the ECG.
At risk patients are:
- rapid transfusion > 150ml/min
- pediatric recipients
- hypothermia
- pre-existing liver disease

- Hb dysfunction occurs due to the decreased 2,3 DPG levels, the low pH and high \( \text{CO}_2 \) in stored blood. The nett effect is that of decreased \( \text{O}_2 \) delivery to the tissues, especially if the blood is administered cold. This may compromise \( \text{O}_2 \) delivery in patients with limited cardiac reserve. This effect can be countered by warming blood, avoiding bicarbonate administration and using blood < 7 days old in patients with limited cardiac reserve.

2. COAGULOPATHY
Transfusion related haemorrhage is usually a generalized oozing from the surgical field, hematuria, mucocutaneous tissues and puncture sites.
**Thrombocytopenia** is common, but usually the cause of haemorrhage only after an acute replacement of a full blood volume.
**Dilutional coagulopathy** is also usually late in onset.
Earlier haemorrhage indicates a consumption coagulopathy rather than the above two causes.
**Acute hemolytic transfusion reactions** may occur, most commonly due to the administration of incompatible blood from clerical error.
**DIC** may occur due to critical injuries or transfusion reactions.
**Pulmonary insufficiency** may occur due to a load of micro-aggregates of fibrin, platelets and degenerated white cells being deposited in alveolar capillaries.

3. INFECTIVE RISKS
Infective agents may be passed through transfusion and include bacteria, viruses, spirochetes and parasites.
The most common infective risks are:
- Hepatitis C – up to 1:100 per unit. This accounts for 90% of post-transfusion hepatitis. 50% become chronic carriers of which 20% develop cirrhosis.
- HIV – currently estimated as 1:250 000 to 1:500 000 per unit
- Hepatitis B – low incidence due to screening. 9% become chronic carriers, 1% develop cirrhosis and 1% hepatocellular Ca.
- Hepatitis A is of little concern.

✔ As a practical point, if you have to transfuse emergency O-negative blood to a patient, draw a tube of blood for blood grouping prior to the transfusion, otherwise the Blood Bank may have difficulty determining the patient’s correct group.
Caesarean sections are equal to laparotomies in many aspects, pain relief not an exception.

The widespread use of spinal anaesthesia often lends to the mistaken belief that these procedures are not that painful. This is reinforced by the fact that mothers are also often more concerned with their baby’s needs than their own. They have multiple tasks to fulfil early on eg initiating breastfeeding, emotional bonding and are on an emotional and hormonal ‘high’ with the baby’s delivery. Thus pain is often endured or cortically suppressed for the benefit of the baby.

Doctors are also loathe to dose the mother with major analgesics out of fear of making the mother unable to perform these functions and/or suppressing the baby via breast milk.

**Caesarean section Pain**
The pain after Caesarean section is due to 2 main factors, namely wound pain and uterine contraction pain.

Wound pain is usually mild when the mother is at rest, but becomes moderate to severe on movement. Uterine contraction pain is intermittent and can be severe.

It is well known that uterine contraction pain, similar to menstrual pain, responds well to NSAIDs, while wound pain is reduced by opioids.

Both these types of pain are initially severe, but are greatly reduced by 48hrs postoperatively. It seems that early mobilization, although causing severe pain initially, helps reduce pain intensity rapidly. Thus aggressive pain management should be concentrated on in the first 48hrs post delivery.

**Morphine in breast milk**
There are conflicting data on morphine concentrations in breast milk. These have varied from trace amounts up to 12% of the maternal dose. However, the present consensus is that the use of morphine in postoperative lactating mothers is safe and that the transferred dose to the neonate is minimal.

Using a multimodal analgesic approach will further reduce possible neonatal exposure to opioids in breast milk.

**GENERAL PRINCIPLES OF PAIN MANAGEMENT**

- **Speak to your patient preoperatively** regarding the pain they can expect and how you are going to manage their pain. This reduces patient anxiety, instills patient confidence in you as doctor and lets the patient know you take their pain seriously.

- Where possible, **pre-empt pain** rather than to allow it to become established. It is easier to manage incomplete pain relief than to effectively reduce established pain.

- **Listen to the patient** – she is the one with the pain. Patients tend to underplay their pain for a variety of reasons and their visible expression does not correlate with the degree of pain they experience.

- **Each patient’s experience of pain is different**, so what may seem to be not so painful for one patient, may be the worst pain another has ever experienced. Similarly, treatment that might be highly effective for one, may not be as effective in another.

- **Be Aggressive!** Do not be afraid to use effective drugs and their dosage schedules.
• Patients in acute pain DO NOT develop addiction to opioids.

• It borders on negligence to allow a patient to leave your theatre and/or recovery room in severe pain.

• DO NOT write up medication for acute/postoperative pain as a prn administration. PRN stands for ‘PATIENT RECEIVES NOTHING’.

• Use a multimodal approach to pain. (see the protocol)

• The experience of pain is multifaceted and multifactorial. If there is a strong psychological component to the pain, treating this will improve the efficacy of your pain management.

• Leave clear orders for staff to contact you if your patient experiences breakthrough pain. Do not delegate this to the inexperienced junior on call unless he/she has been adequately trained in pain management (rotation through Anaesthesia).

**GENERAL TIPS AND SUGGESTIONS**

Irrespective of the route, opioids used for people who are not in pain, or in doses larger than necessary to control pain, can slow or even stop breathing. The key principle is to **titrate the dose against the desired effect** – pain relief – and minimise unwanted effects, like nausea, itching, headache and flushing.

**NB!** When using opioids, **morphine is the drug of first choice**. Repeated or prolonged dosing with pethidine can result in disorientation, hallucinations or seizures due to norpethidine toxicity. Consequently, pethidine is used preferably only when side effects or allergic reactions prohibit the use of morphine.

If the patient is still complaining of pain and you are sure that the drug has all been delivered and absorbed, then it is safe to give another, usually smaller, dose (five minutes after intravenous administration, 60 minutes after subcutaneous or intramuscular administration or 90 minutes after oral morphine). If the second dose is also ineffective, then repeat the process or change the administration route to achieve faster control.

**Always use the maximum dose and shortest dosage interval permitted, especially initially, otherwise your patient will get a see-saw or on-off type of pain control.**

**CHOICE OF DRUGS**

A known or possible allergy to a specific drug obviously precludes its use. There are lists of contra-indications or cautions for the use of many analgesics, but some of the more important ones are:

• paracetamol use in liver failure

• high-dose opioid use in patients with underlying severe respiratory impairment, cerebral depression or impaired airway reflexes

• pethidine is a neuro-excitatory drug and should be avoided in epileptics, eclamptics, patients with psychosis of any origin, brain injury, CVA’s and patients on MAO-inhibitor drugs or anti-psychotic drugs.

• the use of NSAIDs in aspirin-induced asthma, renal dysfunction/failure, the elderly, GI ulcers/bleeds, dehydrated patients, severe PET, ischaemic heart disease, uncontrolled hypertension and patients on Warfarin. Specifically for pregnant patients, NSAIDs may not be used after 32 weeks gestation, because of the possibility of it resulting in the closure of the ductus arteriosus in the fetal circulation.

• Avoid suppositories in patients with diarrhoea, proctitis or other anorectal inflammation.
THE ACUTE PAIN PROTOCOL

ORAL INTAKE

MILD PAIN
- Paracetamol or Paracetamol+Codeine
- Oral NSAID if needed

MODERATE PAIN
- as for mild pain, PLUS
- NSAID suppository/IMI
- IMI opioid if needed

SEVERE PAIN
- as for moderate pain, PLUS
- IMI opioids, PLUS consider adding IMI hydroxyzine (Aterax®)

VERY SEVERE PAIN
- initiate IV opioid protocol, then continue as for severe pain.
- if pain is not controlling on all of the above, contact the MO on call for the Acute Pain Service.

NO ORAL INTAKE

MILD PAIN
- NSAID suppository or
- NSAID IMI (diclophenac)

MODERATE PAIN
- as for mild pain, PLUS
- IMI opioid of choice

SEVERE PAIN
- initiate IV opioid protocol then continue as for moderate pain, PLUS consider adding IMI hydroxyzine (Aterax®)

VERY SEVERE PAIN
- if pain is not controlling on the above, contact the MO on call for the Acute Pain Service. (KHC)

THE IV OPIOID PROTOCOL

According to the above ACUTE PAIN PROTOCOL, patients in severe or very severe acute pain must be started on the IV OPIOID PROTOCOL. This is an interim measure to reduce the patient’s pain and obtain adequate opioid levels in the blood. Thereafter, the patient is managed on IM opioids.

<table>
<thead>
<tr>
<th>OPIOID</th>
<th>DOSE</th>
<th>DILUTION</th>
<th>CONCENTRATION</th>
<th>ADMINISTRATION</th>
<th>TOTAL</th>
</tr>
</thead>
<tbody>
<tr>
<td>MORPHINE</td>
<td>10mg/ml</td>
<td>To 10ml with N/Saline or water</td>
<td>1mg/ml</td>
<td>2.5ml (2.5mg)x2, then 1ml (1mg). Interval between doses 3-5min</td>
<td>10ml(10mg)</td>
</tr>
<tr>
<td>PETHIDINE</td>
<td>100mg/2ml</td>
<td>To 10ml with N/Saline or water</td>
<td>10mg/ml</td>
<td>2.5ml (25mg)x2, then 1ml (10mg). Interval between doses 3-5min</td>
<td>10ml(100mg)</td>
</tr>
</tbody>
</table>
**DRUGS, DOSES AND DOSAGE INTERVALS**

*Paracetamol / Paracetamol+codeine*

**Preparations:** Tablets 500mg:

**Dosage:**
- 20-45kg body mass - 500mg 6hly
- >45kg body mass - 1g 6hly

Syrup 120mg/5ml

**Dosage:**
- **Initial Loading dose:** Oral: 40mg/kg up to 500mg
- **Maintenance:** Oral: 15-20mg/kg/dose 6hrly

**NB!** Do not exceed 90-100mg/kg/24hrs for children <45kg, or 4gm/24hrs for adults (or if weight > 45kg)

*Ibuprofen:*

**Preparations:** Tablets 200mg and 400mg

Suspension 100mg/5ml (>6mths age or >7kg mass)

**Dosage:**
- 5-10mg/kg/dose 6 to 8 hourly (normal sized adult 400mg 8hly) – 48hrs maximum

*Diclophenac*

**General dose ~ 1mg/kg/dose lean body mass for 48hrs max**

**Preparations:** Tablets 25mg/50mg

**Dosage:**
- Adult >50kg body mass = 50mg 8hly - daily maximum 150mg
- 75mg IMI injection (only >50kg body mass)
- **Dosage:** 75mg deep IMI (intragluteal or lateral quadriceps)12hly

**Suppositories** (12,5mg; 25mg; 100mg)

**Dosage:**
- Children 15-25kg - 12,5mg 8-12hly pr
- Children 25-50kg - 25mg 8-12hly pr
- Adults >50kg - 100mg 12hly pr

**NB!** Maximum daily paediatric dose is 3mg/kg in 2-3 divided doses.

**NB!** Never cut higher dose suppositories in an attempt to reduce the administered dose, because the active ingredient is not equally distributed in a suppository.

*Indomethacin*

**NB!** This drug has a very high incidence of GI and CNS side-effects, therefore preferably used only if Diclofenac and Ibuprofen unavailable. No paediatric formulation.

Capsules (25mg) - 25-50mg tds [max 48hrs]

Suppositories (100mg) - 100mg 12hly pr [max 48hrs]

*Tilden (Valoron®) Drops* (paediatric use only)

1drop = 2.5mg

**Dosage:**
- 1mg/kg/dose 6hly, therefore 1drop per 2.5kg body mass every 6hrs sublingually

*Pethidine*

**Preparations:** injection 25mg/ml; 50mg/ml; 100mg/2ml

**Dosage:**
- **general dose is 1mg/kg/dose IMI 4hly, esp <50kg.**
- **Adults > 50kg, 75mg – 100mg IMI 4hly**

For severe pain, an IV dose of 0.5mg/kg over 5min may be given. This is very effective in renal colic and biliary colic pain.
**Morphine**

**Preparations:** Syrup 15mg/10ml (ie 1.5mg/ml)

**Dosage:** 0.2 - 0.5mg/kg/dose 4hly

**Injection 10mg/ml; 15mg/ml**

**Dosage:**
- IMI – 0.1mg/kg/dose 4hly up to 50kg
  - Above 50kg, 0.1 – 0.2 mg/kg/dose (7.5 – 15mg) 4hly depending on age, mass and severity of pain

- IVI - for acute severe pain in children, an IV bolus of **0.02 - 0.05mg/kg over 2-5min** (see also IV opioid protocol)

- For acute severe pain in adults, an IV bolus of **0.1mg/kg over 2-5min** (see also IV opioid protocol)
  - This is generally given as 5mg initially, then 1-2mg every 5min until pain relief is achieved. Caution advised when 10mg total dose is reached.

**NB!** It is mandatory that patients who have been given IV boluses of opioids should receive supplemental oxygen by mask or nasal prongs and should be closely monitored for at least 30min after the IV boluses have been completed, preferably with pulse oximetry.

**NB!** Any patient (especially trauma) in severe pain should be thoroughly assessed for hypovolemia and haemorrhagic shock prior to administering an IV bolus, to prevent acute collapse. If hypovolemia is suspected, fluid resuscitation should be instituted prior to the bolus, which should be given as smaller dose increments.

**A SUGGESTED PAIN CONTROL REGIME FOR CAESAREAN SECTIONS**

Caesarean section patients are often kept NPO for up to 48hrs postoperatively, thus the approach is mainly parenteral.

**SPINAL ANAESTHESIA**
- at the end of the procedure, insert 100mg Diclofenac or 100mg Indomethacin suppository
- write up repeat doses of the same suppository 12hly for 48hrs
- write up morphine 7.5-10mg 4hly IMI for 48hrs
- after 48hrs or as soon as the patient is eating or drinking, convert to oral medications ie paracetamol (± codeine) 1g 6hly + diclofenac 50mg po (or ibuprofen 400mg po) 8hly + morphine 5mg 4hly IMI prn (if pain still requires opioids)
- **NB!** If using pethidine, 100mg 4hly is required

**GENERAL ANAESTHESIA**
- during the procedure, after the baby has been delivered, load the patient with morphine, in 1-2mg boluses every 5min up to 6mg; the rest may be given in increments in recovery if needed
  - (if using pethidine, give 25mg initial bolus, followed by 10mg every 5min)
  - the further management is as suggested above under spinal anaesthesia
- **NB!** Always write up anti-emetic therapy with opioids.

**************************************************
RECOVERY ROOM ISSUES

There are certain standards of care expected in the recovery room, all of which are designed to protect the patient and prevent potentially catastrophic complications.

The following are internationally recognized standards for post-anaesthesia care in all locations:

- ALL patients who have received general anaesthesia, regional anaesthesia or monitored anaesthesia care shall receive appropriate post-anaesthesia care. All patients must be admitted to the recovery room unless expressly ordered by the anaesthetist, in which case the recovery of the patient will occur in the theatre until transfer to the ward. The care given is that governed by policies as decided on by the Department of Anaesthesiology.

- The patient must be escorted to the recovery area by the anaesthetist and formally handed over to the recovery room nurse. Continual monitoring and evaluation must occur during transport if the patient’s condition warrants it.

- On arrival in the recovery area, the patient must be re-evaluated by the anaesthetist, the status on arrival must be documented and the anaesthetist must remain in the recovery room until the nurse accepts responsibility for further care.

- The recovery nurse will further monitor, observe and record the patient’s oxygenation, ventilation, circulation and temperature. A standard recovery room document must be used to record this information and standardized scoring systems are encouraged.

- Medical supervision and co-ordination of patient care in the recovery room is the responsibility of an anaesthetist, who must be available to manage complications and provide CPR in the recovery area.

- The anaesthetist remains responsible for the discharge of the patient from the recovery area – his/her name is noted on the record with their signature.

COMPLICATIONS IN RECOVERY

Large surveys have been done to determine the rate and type of complications that occur in recovery rooms. One survey of more than 18000 general surgical patients found the incidence of complications to be as high as 24%, with the most common problems being:

<table>
<thead>
<tr>
<th>Condition</th>
<th>Incidence</th>
</tr>
</thead>
<tbody>
<tr>
<td>PONV (post-op nausea and vomiting)</td>
<td>9.8%</td>
</tr>
<tr>
<td>NEED FOR AIRWAY SUPPORT</td>
<td>6.9%</td>
</tr>
<tr>
<td>HYPOTENSION</td>
<td>2.7%</td>
</tr>
<tr>
<td>DYSRHYTHMIA</td>
<td>1.4%</td>
</tr>
<tr>
<td>HYPERTENSION</td>
<td>1.1%</td>
</tr>
<tr>
<td>ALTERED MENTAL STATUS</td>
<td>0.6%</td>
</tr>
<tr>
<td>MAJOR CARDIAC EVENTS</td>
<td>0.3%</td>
</tr>
<tr>
<td>RULE OUT ACUTE MI</td>
<td>0.3%</td>
</tr>
</tbody>
</table>

NB! This table does not include the most common problem of uncontrolled pain.

The factors that had the most influence on the incidence of complications were:

- ASA class II
- Emergency procedures
- Type of procedure (abdominal and orthopaedic highest)
- Anaesthetic duration between 2-4hrs
- Smokers
- Hypothermia
As can be seen from the above factors, obstetric patients frequently attract at least three of these factors and we can thus expect a high incidence of recovery room complications.

This chapter will not discuss all recovery room problems that anaesthetists face, eg airway obstruction. It is accepted that the anaesthetist performing obstetric anaesthesia already has the necessary experience to deal with these general problems.

**SPECIFIC PROBLEMS**

1. **UNCONTROLLED PAIN**
   This problem has been addressed under the chapter on postoperative pain management.

2. **PONV**
   This is a frequent and distressing complication for a patient. In the pregnant patient it also has importance because of the potential for gastric content aspiration, especially if full airway and swallowing reflexes are absent.

   Prevention is important, with antacid prophylaxis at the helm, followed by PONV prophylaxis. Sodium citrate (30ml of 0.3Mol solution) with metoclopramide with/without an H₂-antagonist as antacid prophylaxis (see protocol), is the norm. Metoclopramide will improve gastric emptying but has no effect on PONV. Prevention of PONV with 1.25mg Droperidol (¼ ampoule Inapsin®) is as effective as giving Ondansetron (Zofran®). The same dose can be given for treatment of nausea and vomiting.

3. **SHIVERING AND HYPOTHERMIA**
   Shivering is a common occurrence in the recovery room and may occur with both spinal and general anaesthesia cases. It is also distressing to the patient and is something they strongly remember.

   In the case of spinal anaesthesia it is due to the effect of the deafferentation of the area below the spinal level involved. In the case of general anaesthesia, it is often due to the use of volatile agents (the so-called “halothane shakes”) or due to hypothermia per se.

   Obviously, hypothermic patients should be actively warmed, especially if where hypothermia was caused by a prolonged procedure where there was major blood loss. Shivering, irrespective of the cause, increases metabolic rate and thus oxygen consumption by up to 400%! This necessitates the administration of supplemental oxygen to these recovering patients to prevent hypoxia.

   Shivering can be treated by giving Pethidine 25mg IV or MgSO₄ 30mg/kg slowly IV.

4. **AGITATION**
   Agitation in the recovery room may be a big problem in the semi-awake patient and must be attended to immediately.
   The most important cause of agitation in the recovery room is HYPOXIA; this must be immediately looked for and corrected.
   Other causes are hypercapnia (due to hypoventilation), respiratory distress (especially due to incomplete neuromuscular block reversal), pain, full bladder, gastric distension and drug effects (eg Ketamine).

   Treating the cause will treat the agitation. It is important to note that some patients do not become agitated, but are hypertensive and tachycardic and may get dysrhythmias. Again, this responds to treating the cause.

5. **HYPOTENSION**
   In the obstetric patient, hypotension is most commonly due to hypovolemia or the effects of the spinal anaesthesia.
   Assessment of the postoperative cutaneous sensation level is needed when spinal anaesthesia has been performed. Treatment with fluids and/or vasopressors is indicated.
Hypovolemia may be due to underestimated intraoperative blood loss, active bleeding intraabdominally, from the wound or uterine atony. Inspection of the wound and the pelvic area must be done to determine if there is haemorrhage or a poorly contracted uterus, ie LIFT THE COVERS AND LOOK!

UNDER NO CIRCUMSTANCE IS IT ACCEPTABLE THAT A PATIENT WHO IS BLEEDING IN THE RECOVERY AREA BE ALLOWED TO RETURN TO THE WARD UNTIL THE SITUATION HAS BEEN PROPERLY ASSESSED AND CONTROLLED.

NB! If the patient's hypotension does not respond to fluids and vasopressors and the Hb or hematocrit is much lower than anticipated, it must be accepted that there is ongoing blood loss that is not obvious, ie intra-abdominal; the patient will need to be returned to theatre.

It is very important that if you suspect blood loss as the cause of the hypotension, irrespective of the cause, that the surgeon be immediately informed. If he/she has already left the theatre complex, they must return urgently to assess the patient.

6. FAILURE TO REGAIN CONSCIOUSNESS
This is an uncommon, but important problem in obstetric patients.

The most common cause is drug-related. Preoperative alcohol or drug use may be a cause. Residual effects of sedative, premed or anaesthetic drugs is most likely and if this is due to opioids, small increments of naloxone (0.04mg – ie 1amp diluted to 10ml, giving 1ml boluses) every minute should reverse this effectively within a few minutes. Be careful not to reverse fully, otherwise the patient will have no pain relief.

Metabolic causes like hypoglycaemia, severe hyperglycemia or hypercarbia, metabolic acidosis or severe hypothermia (<33°C) must be ruled out and immediately treated if identified.

Last, but not least, structural neurological problems should be sought. This is especially relevant in patients with PET, fulminating PET or eclampsia. Cerebral haemorrhage is known to occur in these patients and this may occur intra-operatively, especially if there is a profound intubation response.

DISCHARGING PATIENTS FROM THE RECOVERY ROOM

Patients should be ‘ward ready’ before being discharged from recovery. The best way to determine this is to have standard discharge criteria, so that irrespective of the recovery nurse’s experience, a certain standard is obtained as long as the criteria are followed.

There are a number of published international discharge protocols, one of the more practical being the Aldrete Criteria (applicable to general anaesthesia):

<table>
<thead>
<tr>
<th>SCORE</th>
<th>2</th>
<th>1</th>
<th>0</th>
</tr>
</thead>
<tbody>
<tr>
<td>ACTIVITY</td>
<td>Moves all 4 limbs</td>
<td>Moves 2 limbs</td>
<td>No limb movement</td>
</tr>
<tr>
<td>RESPIRATION</td>
<td>Deep breathing + cough</td>
<td>Limited breathing</td>
<td>Apnoea</td>
</tr>
<tr>
<td>CIRCULATION</td>
<td>BP within 20% of normal</td>
<td>BP 20-50% of normal</td>
<td>BP &lt;50% of normal</td>
</tr>
<tr>
<td>CONSCIOUSNESS</td>
<td>Can answer questions</td>
<td>Aroused by calling name</td>
<td>No response to audible stimulus</td>
</tr>
<tr>
<td>COLOUR</td>
<td>Pink (O₂sat&gt; 90% after 5min on room air)</td>
<td>Neither pink nor cyanotic</td>
<td>Cyanotic (O₂sat&lt;85% on room air)</td>
</tr>
</tbody>
</table>

WARD READY: SCORE = 8 OR >8
NB!! A PATIENT WHO HAS HAD SPINAL ANAESTHESIA MUST BE KEPT IN THE RECOVERY ROOM FOR AT LEAST AN HOUR (PREFERABLY 90min) AFTER THE TIME OF INSERTION OF THE SPINAL AND MUST SPEND A MINIMUM OF 30min IN THE RECOVERY ROOM.
THE CUTANEOUS SENSATION LEVEL SHOULD BE CHECKED AND CHARTED ON THE ANAESTHETIC CHART PRIOR TO THE PATIENT’S DISCHARGE.

The reason for this is that the patient can still develop a high spinal or hypotension for up to 90min post-insertion. Most patients are stable after 1hr post-insertion, according to available literature.

**Charting the sensory level is a valuable clinical and medicolegal tool.** If a problem arises in the ward, eg hypotension, you will be called because your patient has 'developed a high spinal'. Having a fixed starting point allows you to objectively assess your patient and qualifies your response.

For instance, you charted a T6 sensory level on your chart just prior to your patient's discharge from recovery, 80min after the spinal was inserted. An hour later you are called to see the patient, whose BP in the ward has dropped from 110/60 to 90/40. The patient has mild lower abdominal pain and a tachycardia. You can now reassess the sensory level, which you find to be T11.

The hypotension is almost definitely not due to the 'high spinal', as evidenced by the rapid regression of the sensory level and the presence of mild lower abdominal pain. Other causes must be sought and an intra-abdominal bleed be excluded.

If the same patient's level was not checked and charted and your assessment in the ward reveals a sensory level of, say T8, you will have to actively prove by exclusion, that the problem is not due to your spinal anaesthesia. A diagnosis of an active intra-abdominal bleed may then be delayed and the patient endangered.

Similarly, if the same patient collapses in the ward and subsequently dies or suffers hypoxic brain injury, you may be blamed for the death or disability, which may be due to a totally different reason. However, having charted the sensory level at T6 at 80min, supplies the investigator the information to almost certainly exclude the spinal anaesthesia (and you) as the cause for the problem.

**HAVE A HIGH INDEX OF SUSPICION AND ANTICIPATION FOR PROBLEMS IN THE RECOVERY ROOM AND STICK TO THE RULES OF DISCHARGE CRITERIA AND BOTH YOU AND YOUR PATIENTS WILL SLEEP WELL AFTER OPERATION.**
POSTPARTUM HAEMORRHAGE (PPH)

Severe postpartum haemorrhage is a leading cause of maternal morbidity and mortality worldwide, even in highly developed countries. It occurs in 2-3% of deliveries and accounts for 25% of the global number of maternal deaths.

Postpartum haemorrhage is defined as the loss of >500ml from the genital tract or >10% fall in the Hb in the first 24hrs after delivery.

Severe postpartum haemorrhage is defined as a blood loss of >1000ml and associated with signs of haemorrhagic shock.

Cases of PPH often need to go back to theatre to remove the cause and/or control the haemorrhage, since some of the most common causes of PPH are uterine atony, retained placenta, placenta accreta and abruption placentae.

Antenatal follow-up should identify women with antepartum haemorrhage and those who have definite risk factors for PPH – multiple pregnancies, polyhydramnios, the grande multipara, those with a history of previous Caesarean section and previous PPH which required blood transfusion – should be considered for referral prior to labour.

Active management of the third stage of labour and routine monitoring of the mother after delivery to detect PPH early, have been proven to reduce PPH, especially massive PPH.

A standard protocol for managing PPH at your institution should be known by all the participating healthcare providers; this includes having sufficient IV fluids, oxytocic drugs and emergency(O-neg blood) available as well as staff with appropriate skill levels. Part of the protocol should be to involve the anaesthetist early on when the diagnosis is made and not post facto when the surgeon wants to rush the patient to theatre.

Freeze-dried plasma is a colloid blood product often overlooked for use in level 1 hospitals. It has a long shelf-life and is easily reconstituted - this product should be available at all level 1 hospitals. If it is not readily available, the anaesthetist should consider this part of his/her duties to speak to the relevant pharmacy/logistical staff to ensure it is ordered.

Resuscitation of the patient with PPH

Once the diagnosis of PPH has been made, aggressive management should be the order of the day. This should be instituted immediately and continued while the cause for the PPH is being ascertained.

Signs of shock (tachycardia, hypotension, delayed nailbed capillary refill, poor peripheral perfusion, change in level of consciousness and oliguria) should be looked for actively. External or visual haemorrhaging is not always indicative of the degree of blood loss and is often difficult to assess when absorbed into pads, green cloths, sheets etc. Clots and soiled sheets have often been removed by nursing staff before your arrival.

Immediate large gauge venous access should be obtained. This requires 2 intravenous infusions with large gauge needles - at least size 16 (Grey Jelco) and preferably size 14 (if available) - and rapid infusion of crystalloids (e.g. Plasmalyte B or Ringers lactate) or colloids (Voluven, Gelofusine, fresh frozen plasma). The volume given should be titrated against the clinical picture, ie increase in BP, improved capillary refill, decrease in degree of tachycardia, improved level of consciousness and improved urine output.

Guidelines for the administration of resuscitation fluids:

- Estimate the volume of blood lost (visually and according to the clinical signs mentioned above) – The correlation between blood loss and clinical picture is shown in the table below (this has been modified to accept a total blood volume of 4500ml), but
remember that in the pregnant patient this estimation is more difficult than in the non-pregnant patient because the physiological changes of pregnancy act as a buffer.

- **Remember that these mothers are postpartum** and have already lost 500ml or more of blood during the birth process.

<table>
<thead>
<tr>
<th>CLINICAL FEATURE</th>
<th>GROUP 1</th>
<th>GROUP 2</th>
<th>GROUPS 3+4</th>
</tr>
</thead>
<tbody>
<tr>
<td>% of volume lost</td>
<td>&lt; 15%</td>
<td>25-30%</td>
<td>&gt; 40%</td>
</tr>
<tr>
<td>Estimated volume lost</td>
<td>&lt; 750ml</td>
<td>1100ml - 1500ml</td>
<td>&gt; 1800ml</td>
</tr>
<tr>
<td>Pulse rate</td>
<td>normal</td>
<td>Mild tachycardia</td>
<td>Severe tachycardic</td>
</tr>
<tr>
<td>Pulse volume</td>
<td>Normal to mildly ↓</td>
<td>Decreased</td>
<td>Weak/thready</td>
</tr>
<tr>
<td>Rhythm</td>
<td>normal</td>
<td>Usually normal</td>
<td>Usually abnormal</td>
</tr>
<tr>
<td>Respiratory rate</td>
<td>normal</td>
<td>tachypnoic</td>
<td>Marked tachypnoea</td>
</tr>
<tr>
<td>Peripheries</td>
<td>normal</td>
<td>Cold and clammy, decreased capillary refill</td>
<td>Cold and pale, unable to assess capillary refill</td>
</tr>
<tr>
<td>Level of consciousness</td>
<td>normal</td>
<td>Anxious/mild confusion</td>
<td>Depressed consciousness/ unresponsive</td>
</tr>
<tr>
<td>Urine output</td>
<td>normal</td>
<td>Decreased output</td>
<td>Oliguric to anuric</td>
</tr>
</tbody>
</table>

- **Apply the 3:1 rule for blood loss up to 750ml (Group 1 shock)**, i.e., for every 1ml blood lost, replace with 3ml of crystalloid fluid (Ringers lactate or Plasmalyte B)

- **Replace further blood loss with colloids** on a ml for ml basis (Voluven, Haemaccel, Gelofusine; in cases where coagulopathy is present or developing, freeze-dried plasma or FFP’s) – up to 25% blood loss.

- **Any further blood loss must receive blood!** Where at all possible, in a level 1 hospital, where only emergency blood is available (and usually only 2 units at that), blood transfusion should be delayed until surgical control or improvement due to oxytocics has been achieved. Until then, colloid infusions must be used as far as possible, including plasma, UNLESS THE PATIENT IS EXSANGUINATED AND REQUIRES THE BLOOD TO MAINTAIN HEMODYNAMIC STATUS. The rationale behind this is that the actively bleeding patient will only bleed out the transfused blood and if you have a limited supply (2U) and no local bloodbank, this may in fact ‘waste’ the blood. This blood can be given after control is obtained over the haemorrhage, especially if the hemodynamics can be maintained with other colloids.

One can also partially resuscitate or ‘dry-resuscitate’ these patients with colloids sufficiently enough to maintain vital organ perfusion, but with lower than normal pressures. This is in an attempt to limit further bleeding prior to operation and before transfusing precious blood.
THE FOLLOWING ARE THE GUIDELINES SET OUT IN THE SAVING MOTHERS REPORT

MANAGEMENT OF APH

**Abruptio Placentae**
- Active resuscitation and prompt referral to a level 2 or level 3 hospital is mandatory when the diagnosis of abruptio placentae with an intrauterine death has been made. O-negative blood and freeze dried plasma can be administered at a level 1 hospital prior to transfer.
- The route of delivery in the event of abruptio placentae with an intra-uterine death should be vaginal, unless there are obstetric indications for caesarean section. If the fetus is confirmed to be alive, an emergency Caesarean section is indicated to save the fetus.

**Major placenta praevia with or without previous caesarean section**
If possible, these patients should be operated upon at level 2 or 3 facilities, with the most senior doctor available.

**Emergency management of PPH**

**PPH; Placenta delivered.**
The initial emergency management of all patients with postpartum haemorrhage must include:

- **Step 1:** The uterus must immediately be rubbed up. This will cause the uterus to contract and reduce the blood loss.
- **Step 2:** Call for help. One health worker alone will not be able to manage a postpartum haemorrhage.
- **Step 3:** A rapid intravenous infusion of 20 units oxytocin in a litre of intravenous fluids must be started. Once again make sure the uterus is well contracted.

➢ These three steps must always be carried out irrespective of the cause of the postpartum haemorrhage.

- **Step 4** The patient’s bladder must now be emptied. A full bladder may cause poor contraction of the uterus with resultant haemorrhage. If the uterus remains atonic, intravenous or intramuscular ergometrine, 0.5 mg, can also be given, provided the patient is not hypertensive or cardiac, or 600 micrograms misoprostol can be given rectally, *(The latter medication is not currently available in clinics, pending further research on its efficacy.)*
- **Step 5:** Suture any vaginal or perineal tears that are bleeding.
- **Step 6:** All patients managed at level 1 clinics or hospitals with no theatre facilities, where bleeding persists following these initial steps must be referred to the next level of care as an acute emergency.

An atonic uterus not responding to steps 1 to 4 must be bimanually compressed while the patient is being transferred to the next level of care. Paramedics need to be taught this manoeuvre.

**PPH; Retained placenta**
- Active resuscitation must be performed.
- Level one facilities, with no available blood and/or theatre facilities, must refer as an acute emergency to the next level of care.
- Patients with retained placenta must always have an intravenous infusion of 20 units oxytocin in a litre commenced.
- At referral hospitals with theatre facilities, manual removal of placenta under anaesthesia must be performed urgently. Delays can result in serious deterioration of
the patient. Level one hospitals with theatre facilities should be staffed with personnel able to perform this procedure, as well as level 2 and 3 facilities. Blood needs to be available for this procedure.

**Persistent bleeding/Further treatment modalities**

This section refers to further treatment of persistent bleeding at referral hospital: level 1 hospital with theatre facilities and appropriately skilled personnel, level 2, and level 3 hospitals.

- Review the initial diagnosis of the cause of bleeding.
- Consider the possibility of intractable uterine atony, retained products of conception, cervical tears, or uterine rupture. Suspect coagulopathy secondary to massive haemorrhage.
- Cross match extra blood and order fresh frozen plasma. Assign responsibility for resuscitation to one staff member, who will also document events.
- Further treatment modalities to arrest haemorrhage:

**Level 1 Hospital (theatre facilities)**

- Additional uterotonic drugs to aid uterine contraction: ergometrine, prostaglandin F2 alpha or rectal misoprostol
- Explore for cervical or high vaginal tears and suture them.
- Manual removal of placenta
- Examination under anaesthesia –
  - Suture Cervical tear
  - Removal of retained products of conception
- Laparotomy –
  - B lynch haemostatic suture
  - Uterine artery ligation/ stepwise devascularisation of the uterus.

*NB. These procedures are also useful for treating caesarean section associated haemorrhage and should be taught to all doctors learning to do caesarean section.*

In the event of doctors at level one hospital being unable to definitively arrest the bleeding at laparotomy, then clamping of major bleeding vessels plus tight intra-abdominal packing may temporize the situation so the abdomen can be closed and the patient referred. Telephonic communication between level one doctor and specialists in level 2 may be useful in this situation.

**Level 2 and Level 3 hospitals**

All of the above.

Hysterectomy. This procedure is a skilled surgical procedure that should be performed timeously. Maternal deaths have occurred from delays in performing a life saving hysterectomy due to persisting for too long with attempts to conserve the uterus, particularly in women of low parity.
WHEN TO REFER PATIENTS TO THE TERTIARY INSTITUTION

The Northern Cape does not have a fully-fledged tertiary institution, but Kimberley Hospital Complex (KHC) is the regional hospital. As such, it acts as a tertiary institution, is closely affiliated to the university hospital (Universitas) in Bloemfontein and indeed performs many procedures associated with tertiary institutions.

The Northern Cape, with the exception of Upington, does not have secondary institutions to speak of, therefore referrals are often direct to KHC. It is important to have a good idea when to timeously refer obstetric cases, in order to prevent maternal and fetal morbidity and mortality. This is often necessary on both obstetric and anaesthetic grounds, since the anaesthetic problems are inextricably interwoven with the maternal-obstetric condition.

This short discussion will refer mainly to the anaesthesiological side of the matter. Conditions will not be discussed in detail, these can be read up under the chapter on specific conditions.

Again, the smaller hospitals where MO’s are directly involved in other disciplines, have an advantage in this regard. High-risk patients can be identified early and thus be referred early.

GENERAL PRINCIPLES OF REFERRAL

Referrals must be made based on clinical and scientific information and not for convenience sake, but likewise, referrals should not be delayed or ignored – selection of patients is important.

Proper communication with the referral hospital is of vital importance. Do not refer without discussion with the relevant person at KHC, but at the same time, do not communicate only with an intern or Community Service MO. Try and reach the most senior person available/on call.

Do not try and explain to the Obstetric team why you want to refer a patient if the main problem is related to Anaesthesia – rather contact the most senior anaesthetist first and discuss the problem with him/her. They can also then discuss the problem with the Obstetric team after you have spoken to them. This is especially relevant if the main problem is anaesthetic in nature and the Obstetrician is reluctant to accept the patient.

Always write a complete referral letter addressed to the Obstetric doctor you spoke to, with the patient’s name also on the envelope. Mention in your letter that you also discussed the problem with the Anaesthetist.

Ensure that the patient is optimally resuscitated or treated before transfer in the ambulance and give clear instructions to the nursing sister about care in the ambulance, if you are not accompanying the patient yourself.

In urgent referrals, speak directly to the person in charge at the ambulance call centre to impress upon them the urgency of the referral. Repeat these instructions to the ambulance driver/paramedic when loading the patient, so that they are aware of the emergency. Due to the enormous distances that sometimes need to be travelled in this province, ambulance drivers sometimes make detours to pick up other non-urgent patients. This is not acceptable in the case of emergencies and this must be made clear to both the ambulance centre and the driver. Also make sure that the accompanying nursing sister is also aware of the urgency, so that the patient is not left unattended in the back of the ambulance.

NB! In the Northern Cape, the official policy is that obstetric patients are top priority transfers and there should be no excuse for delay due to transport.
REASONS FOR REFERRAL

This discussion is not going to supply an exhaustive list of conditions that require referral, but rather the thinking behind what requires referral.

PATIENT FACTORS

- Patients with severe comorbid conditions that are uncontrolled or poorly compensated and will undoubtedly worsen during term pregnancy, labour or post-delivery. Good examples of these are patients with cardiac problems like cardiomyopathy, congestive cardiac failure, valve lesions; asthma, active TB or old TB with lung destruction; AIDS; hepatic or renal failure
- Obvious airway problems (see the chapters on Airway Evaluation and Advanced Airway Management).
- Other surgical conditions in pregnancy
- Multitrauma in pregnancy
- Morbid obesity

OBSTETRIC FACTORS

- Multiple pregnancy
- Grand multipara
- High grade placenta praevia on sonar
- High risk for uterine rupture
- Fulminating PET or eclampsia where the cervix is unfavourable for early delivery
- HELLP syndrome
- Premature rupture of membranes (<34wks)

FETAL FACTORS

- Severe IUGR
- Fetal malpresentations
- Pre- and postmaturity

It is obvious from the above that the reasons for referral are where underlying patient conditions may rapidly outstrip both human and physical resources available locally and where there is time to refer the patient (and baby) to better care.

This should by no means be regarded as failing in your duty, but rather as knowing your limitations as primary healthcare professional. You may be able to manage the patient’s condition as a doctor, but your resources may not support your expertise. Knowing where to refer rather than to keep the patient until the problem has become critical is the sign of a good and cautious professional.

The resources differ from hospital to hospital and may be as simple as the presence of a single knowledgeable sister or as basic as the lack of adequate oxygen supply. The onus is on you and your obstetric MO colleague to confer and decide upon the correct management.

This scenario changes in an emergency or when delivery is imminent. Decision-making becomes more difficult and many factors must be weighed up regarding mother, baby, delivery, stabilizing the condition and whether transfer could possibly occur in time. It is much more preferable to have the patient in an under-resourced hospital than in transit, where minimal support would be available.
WHAT ABOUT AIR AMBULANCE?

There are many misconceptions regarding aeromedical transfer.
The air ambulance service is available to be used, but it should be realized this is not always
the optimal mode of transfer.
Globally, it is accepted that road ambulance transfer is nearly always indicated if the distance
from the referring hospital is less than 2-3 hours travelling time from the tertiary institution,
because this is the minimum time it usually takes to scramble the flight medical team, prepare
and warm-up the fixed-wing aircraft and fly the patient in.
Flying a patient in becomes a lot more realistic when the distance to be travelled is more than
250-300 km, but this still has to be weighed up against road transfer.

Often the aircraft is already on a trip and it may be many hours before your patient can be
fetched.
Other uncontrollable factors may influence departure of the aircraft to your hospital, eg weather,
pilot availability, flying staff availability, technical factors and costs.

It is thus extremely important to phone the air ambulance call centre/co-ordinator and discuss
all these issues, calculate the time factor and determine whether it is still feasible to utilize this
resource.

There are some clinical technical aspects regarding air transfer of patients that need to be
taken into account – these are issues that are often overlooked, because they are not relevant
for road transfer. They will be mentioned below, but if any doubt is present, communication
with somebody well-trained in aeromedical transfer of patients is necessary!

1. Monitoring
   Aeromedical transfers are renowned for their difficulty in monitoring patients, due to
   environmental noise, vibration etc. This may be a major problem in patients requiring
   close monitoring during transfer.

2. Intubated or ventilated patients
   Intubated patients, as a general rule, need to be sedated, paralysed and mechanically
   ventilated for air transfer. ET tubes have to be inflated with water/saline and not air.
   Ventilated patients have to be ‘packaged’ for air transfer, with patients in the lateral
   position. Ventilator settings must be measured and monitored during air transfer, to
   prevent hyper/hypoventilation and thus hypo/hypercarbia. This is of great importance
   in brain-injured patients or brain swollen (PET/eclampsia) patients, because of the
   effect on brain perfusion and intracranial pressure.

3. Air transfer patients should preferably be stabilized before transfer, because of the
   inability to monitor and treat problems in-flight.

Therefore use your common sense and clinical judgement when referring patients up the line
and deciding which mode of transfer to use. At all times consider obtaining telephonic advice
at the highest possible clinical level before embarking on transfer procedures.
WHAT TO DO IN THE CASE OF AN ADVERSE EVENT

It is very important to realise that adverse incidents occur even when good doctors practice good medicine and there is no negligence.

DEFINITIONS

- **Adverse event** – a problem arising during anaesthetic that causes or has the potential to cause an adverse outcome
- **Adverse outcome** – when the adverse event causes patient injury or death, escalation of care or operational inefficiencies
- **Critical incident** – when an adverse event does not result in an adverse outcome

Anaesthesia is always potentially dangerous and obstetric anaesthesia even more so. Anaesthetic-related maternal deaths, most of which are highly preventable, make up 6% of all maternal deaths. Roughly 50% of these are due to airway problems, most of the other 50% are related to problems with spinal anaesthesia.

Many of the underlying problems are self-compounding eg an obese unbooked primigravida at 34wks gestation with undiagnosed fulminating PET who presents with a tonic-clonic convulsion and requires urgent Caesarean section. With no history and a semi-comatose patient, you are forced to perform a GA on a probably problematic airway and a full stomach.

These are the ingredients of a disaster, none of which are your making. The most that can be expected of you is to act within your clinical and professional expertise to the best of your ability. It is here that the focus should be on preparation for all the possible scenarios and not rushing into the procedure blindly. There might be pressure on you from the surgeon to just ‘get on with it’, but taking 10 minutes longer to stabilize the patient, prepare your mind and have plans and equipment in place, may well be the difference between a live mother and baby and having to fill in the ‘white book’.

This may sound melodramatic, but most disasters, medical or not, begin with a single event and cascade, like a row of dominoes, with you at the end. Interventions may or may not halt this cascade. Most adverse outcomes are not due to negligence, but due to events beyond our control. However, there are adverse events that are due to negligence and it is our job to keep these events to an absolute minimum by means of training, practice of preparations for disasters (eg the failed intubation) and continued professional development.

STRATEGIES TO MINIMIZE ADVERSE OUTCOMES

- Keep up to date with current trends, standards and guidelines
- Have standardised policies and protocols in place that are optimally suited to your physical resources and environment
- Always examine and evaluate your patient preoperatively
- Believe your anaesthetic monitors eg do not accept that the poor pulse oximeter reading or ECG trace is due to faulty probes or ECG stickers – always go to the patient first and check peripheral pulse volume and rhythm.
- Maintain a high state of vigilance and a healthy degree of suspicion
- Have accurate charting of your monitoring
- Before starting a procedure, always ask yourself “what might go wrong?” and “am I prepared?”
- Maintain good communications and relations with your referral hospital and the department’s Consultants – don’t be afraid to consult with them
WHAT TO DO IF YOU EXPERIENCE AN ADVERSE EVENT/OUTCOME

- Immediately inform the surgeon and theatre staff of the problem and get as many hands to help as possible
- If the event might end in a possible fatality and there is no immediate surgical factor involved (eg a failed intubation), the surgeon should immediately stop surgery and assist you with the problem
- If there are enough hands in theatre, designate the most junior person to stand and chart all drugs administered and their times
- If the adverse event is not acutely life-threatening eg severe hypotension after spinal anaesthesia, inform the staff, treat aggressively and chart the duration accurately on your records. NB! It is not acceptable to alter your records after the fact; it is however acceptable to complete your chart after the rush and turmoil of the event and even correct an error on your chart as long as an explanatory note is written alongside the change – dated, timed and signed.
- Sit down and right a concise, accurate factual account of the events as they evolved, writing your opinion, but avoiding accusatory or unsubstantiated statements. The scrub sister and the surgeon should do the same.
- Take time to discuss the situation with the sister and surgeon involved before leaving theatre. This is not to collude or to accuse, but to get clarity on the events and have agreement on informing the family. This is very important in the event of a death, to discuss what occurred and to psychologically ‘debrief’ those involved – these events are always traumatic for the staff involved and a short discussion often clears questions up that are maybe being thought about but not voiced.
- Do not allow the surgeon to speak to the family alone, because surgical perceptions cannot be extrapolated to the anaesthetic experience. The family should be informed as a team, to prevent the family hearing two seemingly different versions of the event. If there has been a death, this should be done in a private room and with empathy. Do not try to give the distraught family too much detail, which won’t be remembered. However, let the family understand that they may approach you at any time for further details, if so needed. Assist them as far as possible with information regarding what must be done next, eg autopsy/burial.
- Make copies of all the records and keep them in a safe place — if a court appearance is necessary, this might only be 5yrs later.
- If the event occurs on an elective list, a decision must be made by the whole team as to whether the list should continue — it is generally advisable to cancel the rest of the list, especially in the event of a death. If this cannot be done, then the anaesthetist should ideally be replaced by a colleague.
- Especially in cases where there was no obvious cause for the adverse event, it is important to remain in contact with the family and inform them of results of tests or autopsy — if this is not done, the patient’s family tend to perceive this as a ‘cover-up’ or collusion.
- Contact your Medical Protection Society, inform them of the event and ask for advice.
- Do a literature search regarding the event to become better informed about the possibilities.

It is very important to recognize that adverse events result in a great deal of ongoing stress and anxiety for the anaesthetist. If this is protracted due to legal proceedings, the stress may result in the impairment of functioning of the anaesthetist. Early support from psychologists or a psychiatrist may be indicated. This aspect of care should not be neglected.

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