WA WORLD ANAESTHESIA

UPDATE IN ANAESTHESIA

A journal for anaesthetists in developing countries

ISSN 1353-4882

EDITORIAL **No 4 1994**

Welcome to Update in Anaesthesia No 4. We have been delighted to receive so many comments from anaesthetists and surgeons who read the journal. At present we are distributing around 4000 copies to a total of 80 countries and receive regular requests from people wishing to be added to the mailing lists. In this regard our funds are best used if we can send copies of Update to a national society or missionary society who can then distribute copies locally. Overseas postage is expensive for individual copies. Readers wishing to subscribe or willing to help with distribution in developing countries please contact Dr Sarah-Jane Fearnley, Department of Anaesthetics, Torbay Hospital, Lawes Bridge, Torquay TQ2 7AA, UK.

One correspondent recently asked whether articles could be copied for teaching purposes. We are delighted if this is done and merely request that the source of the material is acknowledged.

Contents: No 4

Editorial
Anaesthesia for the Patient with a Full Stomach
Resuscitation of the Newborn
Sickle Cell Disease and Anaesthesia
Ketamine
Local Anaesthesia for Hernia Repair
The Pharmacology of Local Anaesthetic Agents
The Physiology of Neuromuscular Transmission
Maintenance of Laryngoscopes
Correspondence

If there are specific requests for topics to be covered, please write to us. If you have any questions on which you would like an expert's opinion please send them to us and we shall contact a suitably qualified person and publish the reply.

Dr Iain Wilson
Dr Roger Eltringham

ANAESTHESIA FOR THE PATIENT WITH A FULL STOMACH

Dr Mijumbi Cephas, Mulago Hospital, Uganda

One of the major risks posed by patients who have not been prepared for theatre is that they may not have an empty stomach. When consciousness is lost (as during induction of general anaesthesia) the patient with stomach contents may regurgitate gastric material via the oesophagus which may be aspirated into the lungs causing a severe pneumonitis (inflammation of the lungs) usually called "aspiration pneumonitis". This is especially severe, and often fatal, if the gastric contents are markedly acidic (pH < 2.5). As little as 30mls will cause a severe reaction. When solid foodstuffs are aspirated complete obstruction of the airway may occur.

Why do people regurgitate? Normally the specialised junction between the oesophagus and the stomach, the **oesphagogastric junction** (which may also be called the **cardia**) acts as a sphincter to prevent material returning to the oesophagus after entering the stomach. When the conscious level is

depressed this junction works less efficiently and if the pressure within the stomach (the intragastric pressure) is greater than the closing pressure of the sphincter then regurgitation will occur. Note that regurgitation is different from vomiting. Vomiting is an **active** process and involves contraction of the abdominal muscles; regurgitation is **passive** involving smooth muscles only.

Normally patients are fasted for 2 hours after clear fluids and 6 hours following a meal before they are anaesthetised. This is to reduce the chance of any residual food remaining within the stomach. However these periods of fasting may not always guarantee an empty stomach. Patients who have been traumatised, or are suffering from intra-abdominal pathology, or who have had opioid drugs or are in labour do not empty their stomachs efficiently and should always be treated as if they have a full stomach.

The risk of regurgitation is greater if the intragastric pressure is increased by the presence of food or liquid within the stomach, the lithotomy position (legs up with patient on their back), obesity or an intra-abdominal swelling such as pregnancy after 24 weeks or ovarian masses.

Pregnancy further increases the risk of regurgitation as hormonal changes decrease the efficiency of the oesophagogastric junction. A hiatus hernia may render the oesophagogastric junction ineffective; patients with this condition will usually give a history of 'heartburn' or indigestion when they lie down.

The Anaesthetic Approach to the Patient with a Full Stomach.

Identify the patient at risk. Any patient who falls into any of the categories above should be treated as having a 'full stomach'.

Consider the operation planned and its urgency. If the operation can be delayed to allow the stomach to empty then this approach should be adopted. However the patient's life should not be put at risk by delaying urgent procedures. It should be remembered that some ill patients may be unable to empty their stomachs.

If possible reduce the volume, pressure and acidity of the stomach contents. Patients with a stomach full of liquid, such as those with bowel obstruction or who are drunk should have a large nasogastric tube passed prior to general anaesthesia. Often the patients will vomit during attempts at passing of a nasogatric tube. Remember that even after passing the tube the stomach is unlikely to be completely empty as nasogastric tubes are inefficient for removing liquids and useless for solids.

As discussed earlier certain elective patients, such as pregnant females in the third trimester, are at risk of acid aspiration despite being adequately fasted. This group of patients is best treated by decreasing the acidity and volume of gastric fluids by the use of ranitidine or cimetidine given 1 to 2 hours preoperatively. Unfortunately this is not adequate for emergencies who should also be given 30mls of sodium citrate immediately before induction of anaesthesia. Such techniques will raise the pH of the gastric fluid and make the consequences of aspiration less serious. Unfortunately not all anaesthetists have access to these drugs but most pharmacies can make up sodium citrate.

Consider the Best Form of Anaesthesia

Due to the risks associated with general anaesthesia the use of a local anaesthetic technique should be considered. This will avoid depressing the conscious level. Beware however of using deep sedation in combination with local anaesthesia. Some anaesthetists believe that ketamine protects the airway by preserving laryngeal reflexes - this is not true.

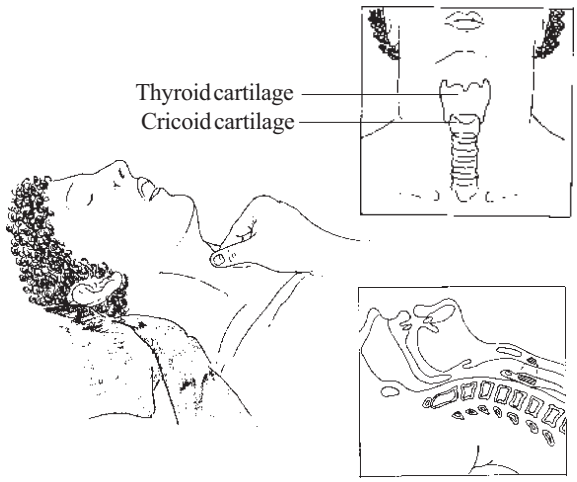
If general anaesthesia is required in a patient at risk of having a full stomach the airway should be protected by a cuffed endotracheal tube. (Under the age of 10 an uncuffed endotracheal tube should be used.) The safest technique for introducing an endotracheal tube in this situation is called a rapid sequence induction (RSI or crash induction) using **preoxygenation** and **cricoid pressure**.

Preoxygenation. Under normal circumstances the lungs contain a mixture of oxygen, nitrogen and carbon dioxide. At the end of expiration the volume of gas left in the lung (about 2 litres) is called the Functional Residual Capacity (FRC). This contains the oxygen reserve on which the patient depends when they are not breathing. Most of the gas in the lung is nitrogen which can be replaced with oxygen thereby increasing the oxygen reserve. The technique of replacing the nitrogen contained in the FRC with oxygen is called **preoxygenation** or **de-nitrogenation.** After 3 minutes of breathing 100%

oxygen most of the nitrogen has been replaced by oxygen.

Cricoid pressure. The cricoid is a ring shaped cartilage situated between the first tracheal ring and the thyroid cartilage. When firm backward pressure is applied to it, as shown in figure 1, the oesophagus is occluded preventing any regurgitated gastric fluid from entering the pharynx. It is completely reliable provided the pressure is put on the correct area. The backward pressure should be firm; if the equivalent pressure is applied to the bridge of the nose it feels uncomfortable.

Figure 1. Applying cricoid pressure



Technique of Rapid Sequence Induction

1. Prepare your equipment and drugs - where possible this should include all the apparatus listed in table 1. Check all the equipment carefully before starting and ensure that everything is to hand.

Table 1 - Equipment required for a crash induction.

Tilting trolley or operating table
Suction apparatus and tubing
Anaesthetic machine, source of oxygen, anaesthetic circuit and facemask
2 appropriately sized laryngoscopes
Correct size of endotracheal tube and one a size smaller
Endotracheal tube introducer, cuff syringe and connections to circuit
Range of oral airways
Anaesthesia drugs - induction agent, atropine and suxamethonium
A trained assistant

2. Consider whether a nasogastric tube should be passed.

3. Assess how difficult endotracheal intubation is likely to be. If you expect difficulties think again whether local anaesthetic could be used or consider an awake intubation.

4. Insert an intravenous cannula and demonstrate the position for cricoid pressure to your assistant.

5. **Preoxygenate** the patient. Using a Magill or other anaesthetic breathing circuit, turn the oxygen to 6 to 8 litres/minute and apply the facemask to the patient. Ensure that there is a good seal between the mask and the patient's face. Ask them to breathe oxygen for three minutes. Do not allow the patient to breathe even a single breath of air during this phase or else the preoxygenation will have to be repeated. This is due to the volume of nitrogen that is contained in a single breath of air.

6. Estimate the dose of induction agent which the patient will need (eg thiopentone 5mg/kg) and give this intravenously, immediately followed by suxamethonium 1.5mg/kg. As soon as consciousness is lost ask your assistant to apply cricoid pressure.

7. Keep the facemask in place but do not ventilate the patient manually as some of the oxygen may enter the stomach increasing the intragastric pressure. As soon as the suxamethonium is effective intubate the patient, inflate the endotracheal tube cuff and check the position of the tube by listening to the lungs with a stethoscope.

Note: if intubation is delayed for any reason, or the patient's colour deteriorates, manual inflation should be immediately carried out with cricoid pressure in place.

8. When you are satisfied that the tube is placed correctly, fix it and then instruct your assistant to release the cricoid pressure.

9. Proceed with the anaesthetic and surgery as planned. At the end of the surgery turn the patient on to their side and do not remove the endotracheal tube until the patient is fully awake and capable of protecting their own airway.

Difficulties with the Technique

1. Intubation is unexpectedly difficult. Ensure that the cricoid pressure is not pushing the larynx to one side. If it is, move the larynx and cricoid cartilage by moving your assistant's hand to the correct position. Do not release cricoid pressure. If the suxamethonium needs to be repeated remember to give atropine before the second dose to avoid bradycardia, and ventilate the patient gently to prevent hypoxia. Maintain cricoid pressure at all times. If intubation proves impossible then carry on as described under failed intubation.

2. No oxygen. Obviously no preoxygenation can take place but it is still possible to use cricoid pressure as discussed above. In this situation the patient will need to be gently ventilated with air to prevent hypoxia after apnoea develops.

3. No suxamethonium. The best option here is to induce the patient in a head down position on the left side using a inhalation (gas) induction with halothane or ether in oxygen or oxygen enriched air. Once the patient is deeply anaesthetised they may be intubated whilst still in the lateral position. Cricoid pressure is not necessary in this situation as any regurgitated material will automatically run out of the mouth.

4. Failed intubation. If intubation proves impossible then it is best to accept the situation and adopt an alternative anaesthetic technique instead of wasting time with repeated intubation attempts. The possible options are to continue with a mask anaesthetic (provided the airway is easy to maintain while keeping an cricoid pressure) or to wake the patient up after turning them on their side and head down and attempt the procedure under local anaesthetic. Alternatively the patient may be allowed to wake up and an awake tracheostomy or intubation performed. The best course will depend on the condition of the patient and their degree of fasting, the operation planned, the facilities and level of expertise available.

5. The cricoid cartilage is difficult to identify. Using firm pressure with your index finger follow a line down the front of the neck from the front of the mandible. The first 'solid' structure you meet is the hyoid bone, followed by the thyroid cartilage (Adam's apple) which is much more prominent in males. Immediately below this you will feel a gap between the cricoid and thyroid cartilages (the cricothyroid ligament) and then the cricoid cartilage. Encourage your assistants to practice finding the cricoid cartilage on other colleagues until they are confident. Non-skilled assistants can provide cricoid pressure if they receive adequate instruction, and the position of the cricoid ring is marked on the skin in ink before starting.

6. The patient regurgitates despite the application of cricoid pressure. If there is only a small quantity of fluid suck it out of the pharynx and intubate the patient. Use a suction catheter to aspirate the trachea after intubation. If there is copious fluid then the patient should be turned on to the side and placed head down to protect the airway. Suction the pharynx and then intubate the patient.

Note: When using small oxygen concentrators in association with drawover apparatus preoxygenation may be difficult as the machines can only provide 4 litres per minute of around 85 - 90% oxygen. When this mixture is used the patient will always entrain air into the drawover circuit making preoxygenation less efficient. One way round this is to fill a large plastic bag with 'oxygen' from the concentrator and use this as an oxygen reservoir during preoygenation. When used it should be attached to the inlet of the circuit. Remember to remove it before it empties completely.

Anticipated Difficult Intubation

Awake intubation. This technique can be used to place an endotracheal tube before inducing anaesthesia. It is useful for patients in whom you expect intubation may be difficult and in whom maintaining an airway under anaesthesia may become a problem.

The best technique uses a fibreoptic bronchoscope but these are rarely available. A simpler technique is to give the patient a drying premedication with intramuscular atropine and then

using some plain 2% lignocaine spray inside the mouth and then ask them to move the solution around the mouth. After a short time gently insert the laryngoscope as far as the patient will let you and spray some more lignocaine into the airway further down, then remove the scope. By repeating this manouvre you will soon see the epiglottis and cords and after spraying them well you be able to intubate the patient. Induce anaesthesia as soon as you have accomplished this. At all times be gentle and consider using sedation such as low dose diazepam and/or morphine to help you. Be careful however, not to depress respiration.

RESUSCITATION OF THE NEWBORN

Dr A E R Young, Registrar in Anaesthesia
Frenchay Hospital, Bristol

Professor D J Hatch, Portex Professor of Anaesthesia
Institute of Child Health, Great Ormond Street Hospital, London

For most newborn babies a clear airway and a warm environment are all that is required. However, 25% of all deliveries are at increased risk of requiring resuscitation, and a further number of babies require resuscitation after a normal birth with no apparent risk factors. For these babies effective and efficient basic and advanced life support must be readily available.

High Risk Deliveries

These include:

Delivery difficulties

Fetal distress, meconium staining, abnormal presentation, prolapsed cord, antepartum haemorrhage, forceps or Ventouse deliveries and Caesarean births.

Maternal factors

Diabetes, drug addiction, fever, heavy sedation, pre-eclampsia or other illness.

Fetal factors

Multiple pregnancy, preterm labour (<37 weeks), postmaturity, small-for-dates, rhesus disease or fetal abnormality.

Obstetrician or midwife expressing concern

Equipment for Resuscitation

* Resuscitation surface with tilting mechanism plus dry, warmed towel.
* Overhead radiant heater and light
* Oxygen source
* Clock or watch with minutes and seconds
* Equipment to provide intermittent positive pressure ventilation including - a face mask (preferably made of clear material so that the baby's colour can be observed), a paediatric self- inflating bag (500ml) with a blow - off valve set at 40cmH_2O and an oxygen reservoir.
* A supply of appropriately sized Guedel airways
* A straight bladed laryngoscope and tracheal tubes of sizes 2.5, 3.0 and 3.5
* Suction apparatus (set at 50-100cmH_2O) with suction catheters sizes 8 and 10.
* Drugs - naloxone (400mcg/ml or 20mcg/ml), vitamin K_1 and adrenaline.

Basic Life Support

Immediately after delivery, any excess fluid should be wiped off the baby. The baby should then be wrapped in a warm, dry towel. When pink and breathing regularly the baby should be handed back to the mother.

Those babies not responding to being touched, for whatever reason, should be transferred immediately to the resuscitation area, under a radiant heater

and the clock started. The baby should be placed flat or slightly head-down, with the head in the neutral position.

Administration of Oxygen and Assisted Ventilation

If the baby is breathing inadequately, oxygen should be given immediately, initially just blown over the face. If the breathing is persistently shallow or is absent or the heart rate is less than 100, bag and mask ventilation should be performed.

A transparent, soft face mask covering the baby's face from the bridge of the nose to the cleft of the chin should be used (figure 1). A rate of about 30-40 breaths/minute with pressures of up to 30cm H_2O is usually sufficient; however, a higher pressure may be required for the first few breaths to expand the lungs. This can be achieved by obstructing the pressure relief valve. An oxygen concentration of 100% should be used.

Figure 1. Transparent, soft face mask.

If there is not adequate expansion of the chest, the head should be repositioned and/or a Guedel airway inserted. Babies requiring mask ventilation for more than two minutes should have an orogastric tube inserted, aspirated and then removed to minimise gastric inflation.

For the healthy baby suctioning of the mouth and nasopharynx is not required. However, if there are copious secretions or meconium this may be important. (Meconium aspiration - see later). Vigorous suctioning in the healthy baby may lead to bradycardia and laryngospasm, and delay the onset of respiration.

Advanced Life Support

Indications for Tracheal Intubation

1. Difficulty in ventilation by mask
2. Prolonged bag/mask ventilation
3. Continued bradycardia during bag/mask ventilation
4. During external chest compression (ECC)
5. Meconium aspiration (see later)

Prolonged attempts at intubation should be avoided. Most newborn babies can be successfully oxygenated by bag and mask, while experienced help is requested.

All equipment should be available on the resuscitation trolley including tracheal tubes :

- size 2.5 for < 750g (<26 weeks)
- size 3.0 for 750-2000g (26-34 weeks)
- size 3.5 for >2000g (34 weeks +)

Use a Cole's tube or a straight sided tube (uncut or cut to 9 or 10cm). If a Cole's tube is used, it should be exchanged for a straight sided tracheal tube if ventilation is to be continued after initial resuscitation to avoid possible airway damage.

During intubation the baby's head should be in the neutral position, the laryngoscope blade should be positioned either in the vallecula or posterior to the epiglottis and the tracheal tube should be inserted with 2.0cm beyond the cords (figures 2 and 3).

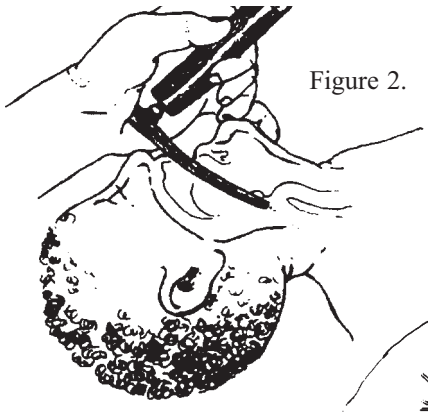


Figure 2.

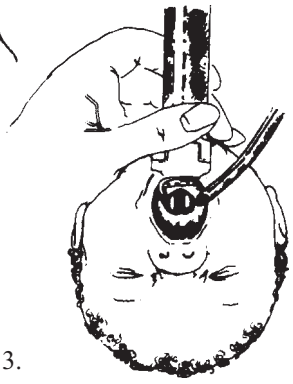


Figure 3.

The chest should be examined for bilateral movement and this confirmed by listening with a stethoscope. If there is air entry on one side only, the tube is probably in the right main bronchus and should be withdrawn slowly until bilateral air entry is heard. If no air entry is heard and the baby remains blue and bradycardic the intubation is likely to have been oesophageal. The baby should be extubated immediately and 100% oxygen administered by bag and mask before re-intubation. If there is any doubt about the position of the tracheal tube it should be removed. Attempts at intubation should only take 20-30 seconds.

The first few inflations may require higher pressures, sustained for at least one second. The pressure relief valve may need to be obstructed (with care!) for a few inflations. When the tracheal tube is in the correct position it should be firmly secured. If the tube is to remain in place following resuscitation, its position should be confirmed, preferably by chest X-Ray.

As soon as the baby's colour improves, the oxygen concentration should be reduced to minimise the risk of retinopathy.

External Chest Compression (ECC)

If the baby has a bradycardia of less than 60-80 beats per minute, it must be oxygenated by bag and mask, or intubated and external cardiac massage started (figure 4).

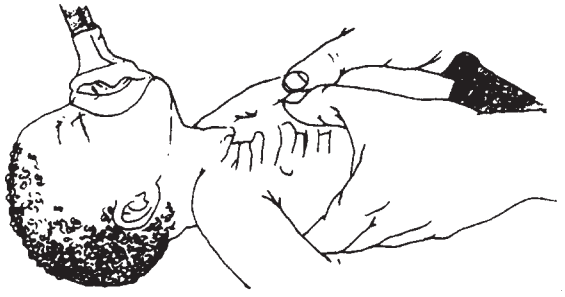


Figure 4. Oxygenation by bag & mask and external cardiac massage.

This can either be with the tips of two fingers over the junction of the middle and lower thirds of the sternum, or with the hands around the chest, compressing the sternum with two thumbs at a rate of 100-120 bpm, to a depth of 2cm. If the ventilation is by bag and mask, the ECC and ventilation should be coordinated in a 3:1 ratio (figure 5).

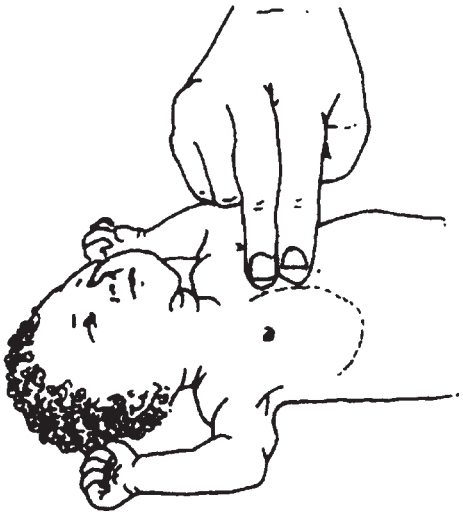


Figure 5. Two finger external cardiac massage

This should be continued until a heart rate of > 100 and spontaneous respirations have occurred. Resuscitation efforts should not be continued beyond half an hour unless the baby is making at least occasional respiratory efforts.

Drugs

Adrenaline: if after 10-15 seconds of ECC there is no response the baby should be given 10mcg/kg (0.1ml/kg of 1:10000) adrenaline. This can be given via an umbilical venous catheter, via a peripheral vein, or, less efficiently down the tracheal tube.

Sodium bicarbonate: 3 mMol/kg over 2-3 minutes through an umbilical catheter if there is no response to ECC and effective ventilation. A 4.8% solution (8.4% mixed with an equal volume of 10% dextrose) should be used (0.5 mMol/ml), given at a rate not exceeding 2.0ml/min.

Naloxone: 10 mcg/kg intravenously or 60 mcg/kg intramuscularly should be given to all babies whose respiration is depressed by the use of opiates in the mother pre delivery.

Glucose: administration should be considered after prolonged resuscitation, as glycogen stores may be depleted. Give 5ml/kg of 10% dextrose.
Drugs are very much a last line measure in resuscitation of the newborn, and should only be used after ensuring that the baby is well oxygenated.

Failure to Respond to Resuscitation

If resuscitation is not succeeding, the tracheal tube position should be rechecked, and airway pressure increased. The possibility of pneumothorax, intrauterine infection or unrecognised hypovolaemia should be considered. Rarer causes of poor response include the presence of congenital anomalies such as choanal atresia, diaphragmatic hernia or pulmonary hypoplasia.

Meconium Aspiration Syndrome

The cords should be visualised in all babies where meconium has been seen in the amniotic fluid before delivery. The nose and mouth should be suctioned on the perineum as soon as the head has been delivered, and after transfer to the resuscitation surface, the pharynx should be suctioned under direct vision. If there is thick meconium in the larynx, the trachea should be intubated and suctioned via a catheter, or the endotracheal tube removed while being suctioned directly. This can be continued, unless the heart rate falls below 60 bpm, until there is no more present. In this situation only, the baby should not undergo bag/mask ventilation as a first line procedure.

Preterm Resuscitation

Babies less than 32 weeks may do better if a more active resuscitation policy is adopted. However there is no proof that routine intubation of all very preterm babies leads to decreased morbidity. In fact unskilled intervention in a lively preterm baby may predispose to interventricular haemorrhage.

Apgar Scoring

Apgar Scoring is a means of scoring the severity of the asphyxia. A score of 0, 1, or 2 is given to five parameters; heart rate, respiratory effort, muscle tone, response to stimulation and colour. It is usually determined at one and five minutes and at eight minutes if the scores are low. It should be repeated at 5 minute intervals until a score of 7 is reached. Particular emphasis should be placed on the heart rate and respiratory effort.

Resuscitation should never be postponed to calculate the Apgar score.

Table 1. Apgar scoring

	0	1	2
Heart Rate	Absent	⩽100	>100
Respiratory Effort	Absent	Weak, cry or shallow	Good strong cry
Muscle Tone	Limp	Some flexion	Active well flexed
Reflex/ Irritability	None	Grimace	Cry
Colour	Pale/ blue	Body pink Extremities blue	Pink

In conclusion, most babies require no resuscitation at birth and can be handed directly to the mother. Those that do require resuscitation, almost always require only drying and being kept warm, suction, and facial oxygen, or possibly bag and mask ventilation for a few breaths. Intubation and the administration of drugs are very rarely required, but should be readily available, especially for high risk births.

SICKLE CELL DISEASE AND ANAESTHESIA

Dr K Henderson, Senior Registrar in Anaesthesia, Birmingham. (formerly: Lecturer in Anaesthesia, Accra, Ghana)

Introduction

Haemoglobin (Hb) is contained in red blood cells and is capable of combining with oxygen in the lungs, transporting it to the tissues and releasing it there. Normally it is composed of 4 polypeptide chains combined with 4 haem radicals. When the haemoglobin is combined with oxygen it is said to be **oxygenated**. When it is not combined with oxygen it is **deoxygenated** (sometimes also called "reduced").

Table 1

Types of Haemoglobin	Polypeptide chains	Notes
Haemoglobin A (HbA)	2 alpha, 2 beta	97% of normal adult haemoglobin
Haemoglobin A2 (HbA2)	2 alpha, 2 delta	2.5% of normal adult haemoglobin
Haemoglobin F (HbF)	2 alpha, 2 gamma	Normal fetal haemoglobin

Sickle cell disease is a genetically inherited abnormality of haemoglobin in which valine (an amino acid) replaces glutamine at the sixth position on the beta chains of the haemoglobin molecule. This haemoglobin is termed Haemoglobin S (usually written HbS). Unfortunately when HbS becomes deoxygenated it comes out of solution forming long crystals called "tactoids" which distort the red cell.

Two types of sickle cell illness are described depending on the genetic make-up. Everyone has 2 genes responsible for haemoglobin synthesis. When a person has one normal (HbA) gene and one sickle (HbS) gene they make a mixture of HbA and HbS. They are called heterozygous patients (meaning that they have both genes present) and are said to have **Sickle cell trait**. The mixed haemoglobin is described as HbAS; their blood contains around 20-45% HbS, the rest being HbA. Because their HbS is mixed with normal HbA, they are much less susceptible to the problems of sickle cell disorders described below.

Patients who have 2 sickle genes can only produce sickle haemoglobin which is called HbSS. They are said to be homozygous, meaning that both of their genes are abnormal. Their haemoglobin is 85-95% HbS, the remainder being made of HbF, a small amount of which is still produced in these patients. They are described as suffering from **Sickle cell disease** or **Sickle cell anaemia**.

Pathophysiology

Deoxygenated HbS is 50 times less soluble in blood than deoxygenated HbA. When HbS becomes deoxygenated it comes out of solution forming long crystals called "tactoids" which distort the red cell and cause it to become crescent shaped. Initially this is reversible with oxygenation but repeated sickling in the low oxygen tension of the microcirculation causes membrane damage. The cell wall becomes brittle and permanently deformed or "sickled". These cells are then susceptible to premature destruction resulting in a lifespan of only 10-20 days as opposed to a normal 120 days. This causes a chronic haemolytic anaemia with a haemoglobin of around 5-8g/dl.

The structural change and associated increase in blood viscosity promotes venous stasis. A vicious cycle is initiated with local blood vessel obstruction leading to tissue hypoxia producing further deoxygenation which promotes further sickling. This leads to cell death and tissue infarction at the site of obstruction. This is termed a sickle cell crisis.

These vaso-occlusive episodes commence from about 6 months of age after the reduction in fetal haemoglobin (HbF) which initially acts as a protective mechanism. Some sufferers are fortunate to maintain a higher that normal HbF production throughout their lives which improves their condition.

Many episodes of sickling occur spontaneously although certain factors may increase the risk. Apart from hypoxia, acidosis (irrespective of the prevailing oxygen tension) is important and is the principle reason for most sickling occurring in the venous circulation.

Infections (bacterial or viral) are potent inducers. Hypothermia and dehydration are also important causing venous stasis and hypoxia via vasoconstriction.

Clinical Features

Patients with sickle trait are usually fit and healthy. However patients with sickle cell disease will usually demonstrate multiple organ damage through repeated veno-occlusive episodes superimposed on a history of poor development and failure to thrive.

Haematological. An acute fall in Hb is usually secondary to infection induced haemolysis or an acute sequestration syndrome in the spleen (infants and children) or liver (children and adults). Blood transfusion is often essential. Bone marrow failure (aplastic crisis) also occurs with a high mortality. Damage to the spleen with increased susceptibility to infections occurs with age.

Respiratory. Dyspnoea, cough , haemoptysis and pleuritic chest pain are classical features of the "acute chest syndrome". Repeated episodes can lead to compromised lung function, pulmonary hypertension and respiratory failure.

Genitourinary. The relative hypoxia and hyperosmolarity of the renal medulla creates an environment for sickling in the vasa recta. The long Loops of Henle are destroyed causing renal failure as the kidney loses its ability to concentrate urine. Haematuria is also a complicating feature. Priapism (prolonged painful penile erection due to venous occlusion) is common, often requiring surgical decompression.

Liver. Jaundice and gallstone formation are a consequence of chronic haemolysis. Liver failure may supervene as a result of multiple infarcts or haemosiderosis from frequent blood transfusions.

Skeletal. Sickling and microvascular occlusion within bones and epiphyseal plates often leads to shortening of the limbs and gross deformity of joints. Osteomyelitis may occur.

Skin. Leg ulcers following skin infarcts are common and often complicated by trauma and poor hygiene.

Neurological. "Acute brain syndrome" is rare but serious. It is characterised by confusion with variable neurological defects. Whilst most resolve spontaneously permanent damage can occur. There is an increased incidence of subarachnoid haemorrhage, blindness and deafness.

Many patients with sickle cell anaemia have frequent hospital admissions for exacerbations of the disease. This can alter their mental health to an extent that considerable psychological, as well as physical, support is essential to their well being.

Preoperative Assessment

1. A careful medical history and examination should be performed in susceptible patients as not all patients have obvious symptoms or signs of the disease.

2. If improvements can be made to the function of the cardiovascular or respiratory systems then the operation should be deferred if possible until this has been achieved.

3. In patients with haemoglobinopathy the need for an operation should be considered very carefully, as sickle cell crises can mimic acute surgical events eg an acute abdomen.

Investigations

The following tests are useful to complement the history and examination.

1. **Full blood count**. If the haemoglobin level is normal then sickle cell disease can effectively be excluded. The presence of anaemia however does not always imply sickle cell disease. Further investigations should include blood microscopy to check for sickle cells, Howell Jolly bodies and sideroblasts, all features of the disease.

2. **Sickling** ("Sickledex") **test**. Mixing blood with the reducing agent, sodium metabisulphite, will induce sickling in susceptible cells. The test is simple and quick and the results can be viewed under a microscope after 20 minutes. Haemoglobin electrophoresis will differentiate between homozygous and heterozygous conditions. In the absence of electrophoresis, a positive sickling test associated with a normal haemoglobin is likely to indicate a patient with sickle cell trait.

3. **Urea and electrolyte estimations** will help to assess renal function.

4. **Liver function tests**. A raised alkaline phosphatase (Alk Phos) reflects obstructive liver disease and and elevated aspartate transferase (AST) indicates intrinsic damage. Unconjugated bilirubin may be raised in the blood as a result of the haemolytic anaemia.

5. **ECG** to look for evidence of cardiac damage.

6. **Chest X-ray** to assess lung fields and cardiac size.

Unfortunately many hospitals do not have access to such investigations and in this situation patients thought to be at risk should be treated as if they were susceptible.

General Anaesthesia for Elective Surgery

The aim is to prevent a sickle cell crisis whilst providing anaesthesia. Attempts should be made to improve the patient's condition preoperatively and to avoid hypoxia, acidosis, hypotension, dehydration and hypothermia perioperatively.

Anaesthesia Planning

1. Optimise haemoglobin. Blood transfusion should be considered preoperatively with a haemoglobin of less than 7g/dl particularly when major surgery or considerable blood loss is anticipated. Transfuse slowly to avoid an increase in blood viscosity. Aim to achieve HbA levels of more than 70% to limit sickling crises. Exchange transfusions have been used in emergencies but are not often practical. In the long term it is better to limit blood transfusions to avoid the problems of chronic iron deposition and formation of irregular antibodies.

2. Preoperative physiotherapy and breathing exercises decrease the incidence of postoperative atelectasis and lung collapse.

3. Premedication. If premedication is planned anxiolytics are preferable to opiates which may depress the respiration.

4. Avoid dehydration by instituting an intravenous infusion if the patient cannot take adequate fluids orally.

5. Preoxygenate for 2-3 minutes. Hypotension on induction should be avoided by careful titration of induction agents.

6. If there is any doubt about the airway a rapid sequence induction with intubation should be performed. Except for the shortest procedures ventilation should be controlled to ensure oxygenation and normocarbia (normal CO_2 level). A 30-50% inspired oxygen level is advisable.

7. Close monitoring of anaesthesia should prevent hypoxia, cardiovascular depression or acidosis developing. Clinical observation can be usefully supplemented by pulse oximetry, blood pressure measurement, ECG and end tidal CO_2 monitoring when available.

8. Replace fluid loss promptly. A central venous pressure line may help monitor fluid replacement. Monitor urinary output.

9. Temperature loss should be measured and minimised. Creating a warm ambient temperature is important. Cover all exposed parts of the body. Pre-warm bags of fluids using a bowl of water warmed to body temperature if a blood warmer is not available. Inspired gases can be partly warmed using a condenser humidifier such as a "Humidivent".

10. Venous stasis should be minimised. This may be a particular problem in the prone (face down) position when compression of the inferior vena cava may occur. Pay attention to the placement of supports. The use of tourniquets is controversial. Previously contraindicated, an increasing number of reports have shown no evidence of sickling with the use of tourniquets when there is an absolute need for a bloodless field. It is of no importance in sickle cell trait, contrary to popular opinion.

Regional Anaesthesia and Sickle Cell Anaemia

Certain regional techniques may have advantages over general anaesthesia and should be considered whenever possible. Benefits include:

1. Peripheral vasodilation secondary to sympathetic block. This improves blood flow to the extremities thereby limiting the possible devastating consequences of vasoconstriction.

2. Analgesia is improved in the early postoperative phase and may help to prevent the increased oxygen demand imposed by pain and shivering.

3. Skeletal abnormalities arising from the consequences of sickle cell disease may make intubation difficult. This potential problem is avoided with local anaesthesia.

There are disadvantages, however. Regional blocks may cause hypotension and hypoperfusion. Prevent these with adequate fluid loading and a careful technique. Use small doses of vasoconstrictors only if absolutely necessary. Do not mix adrenaline with the local anaesthetic as it may exacerbate a crisis.

Emergency Anaesthesia

The same guidelines should be followed as detailed above. Although less time for preoperative assessment will be available, prepare the patient as thoroughly as possible in the time available.

Postoperative Period

The immediate postoperative period is a critical time for patients with sickle cell disease. Hypoventilation resulting from general anaesthesia can easily result in a sickling crisis. The risk can be reduced by:

1. Careful monitoring of vital signs and conscious level. Ensure a clear airway, and where possible, postoperative oxygen therapy.

2. Neuromuscular function must be fully returned to normal before extubation is contemplated. Unless a nerve stimulator is available test this by checking if the patient can perform a sustained head lift (5 seconds) or a strong hand grip on command. Ventilation should be continued until these are apparent.

3. Extubation should be preceded by 2 - 3 minutes of breathing 100% oxygen and supplementary oxygen should be continued postoperatively. This will help to overcome the effects of any residual depressant effects of general anaesthesia, any shunt present in the lungs and will compensate for the increased oxygen demand resulting from pain or shivering. Regular chest physiotherapy should be available with the aim of preventing a chest infection.

4. Adequate analgesia is essential but must be balanced against the problems of hypoventilation with the use of opiates. Titrate the dose carefully. Regional and local blocks are useful. Non-steroidal anti-inflammatory drugs can be used unless renal function is impaired.

5. Maintenance of intravenous fluids is essential until the patient is able to eat and drink.

Summary

Sickle cell disease is an important cause of morbidity and mortality worldwide. When a patient presents for surgery an understanding of the implications of the illness will, when combined with careful preoperative preparation and anaesthesia lead to a successful outcome in most circumstances.

Note

Over the years there have been various treatments attempted in the management of Sickle cell disease. These have all failed to make an impact. Some are listed below.

1. Alkalisation using magnesium glutamate or sodium bicarbonate in an attempt to increase oxygen affinity to haemoglobin in the red blood cell.

2. Antiplatelet and anticoagulants to reduce infarction.

3. Hyperbaric oxygen, high concentration oxygen therapy.

KETAMINE

Dr Andy Tomlinson, City General Hospital, Newcastle Rd, Stoke on Trent, Staffs ST4 6QG.

Ketamine is frequently described as a "unique drug" because it has hypnotic (sleep producing), analgesic (pain relieving) and amnesic (short term memory loss) effects - no other drug used in clinical practice combines these three important features. It was first used clinically in 1970, and because of these combined effects it was thought that it might be the perfect anaesthetic agent. This is not quite the case, but its continued use in all parts of the world demonstrates that for certain situations, when used appropriately, it is a very valuable drug.

Ketamine is available in three different concentrations - 10mg/ml, 50 mg/ml and 100 mg/ml. The 10 mg/ml is for intravenous use; the 50 mg/ml and 100 mg/ml preparations are for intramuscular use. If only one strength is to be kept in a hospital, the 50 mg/ml ampoule is the best compromise as this may be diluted down to 10 mg/ml for intravenous injections.

Actions of Ketamine on the Body

Central nervous system (CNS).

After an intravenous (iv) injection the effects of ketamine on the CNS begin more slowly than after an iv injection of other anaesthetic induction agents (1-5 minutes for ketamine compared with 30-60 seconds for thiopentone). However, as already stated, it has quite different anaesthetic properties compared with these other drugs. The anaesthetic state produced is frequently called "dissociative anaesthesia" which implies that the patient is detached from their surroundings. Unlike other forms of general anaesthesia (ie. inhalational anaesthesia with ether or nitrous oxide, oxygen and halothane) the patient's eyes often remain open and constantly move from side to side (this is termed nystagmus).

The duration of action depends on the route of administration (see later), and in contrast to the smooth induction of anaesthesia, the patient may be agitated on recovery from ketamine. This is often called "emergence delirium", during which the patient may be disorientated, restless, and crying. Patients may continue to experience unpleasant dreams up to 24 hours after the drug has been given. The use of benzodiazepines (ie. diazepam) as premedication, as well as allowing the patient an undisturbed recovery helps to reduce these unpleasant side effects.

Ketamine causes a rise in intracranial pressure and should not be used in patients who have sustained a recent head injury.

Cardiovascular system (CVS).

Ketamine causes mild stimulation of the CVS. The blood pressure rises by about 25% (on average the systolic pressure rises by 20- 30 mmHg) and the heart rate is increased by about 20% - the overall effect is therefore to increase the workload of the heart.

In the majority of patients the blood pressure rises steadily over 3-5 minutes and then returns to normal 10-20 minutes after injection. There is wide individual variation in cardiovascular responses, and occasionally alarming increases in blood pressure can occur. These increases do not seem to be dose-related when more than 1 mg/kg is given and larger doses do not necessarily cause a greater increase in pressure. There is no evidence to suggest that patients with a high preoperative blood pressure are at greater risk of developing a rise in blood pressure following ketamine administration when compared with normotensive patients.

Premedication with diazepam reduces this rise in blood pressure. If the blood pressure rises excessively after induction, a further small intravenous dose of diazepam (2mg to the average 60-70 kg adult) may help to decrease the pressure. As the cardiovascular stimulation following ketamine is mediated through the sympathetic nervous system it would seem appropriate to give alpha or beta blockers to patients who develop excessively high blood pressures. However, the effects of these drugs are unpredictable, and they are probably best avoided in otherwise normal patients as there is no evidence of damage occurring from these short episodes of elevated blood pressure.

Respiratory system.

If ketamine is administered rapidly by intravenous injection it often causes the patient to stop breathing for a short time (up to one minute). After a slow intravenous induction, breathing is well maintained and may even increase slightly. The airway is usually well maintained during ketamine anaesthesia and

there is some preservation of pharyngeal and laryngeal reflexes in comparison with other intravenous agents. However this cannot be guaranteed, and normal airway care must be maintained to prevent obstruction or aspiration.

Recent research in Kenya using a pulse oximeter has shown that following an intravenous induction with ketamine (2 mg/kg) the oxygen saturation falls in a significant number of people (eight out of twenty three patients studied). Nevertheless, there were no untoward events even though this study took place at an elevation of 5000 feet where an increased incidence of hypoxia would be anticipated. The overall message is to observe the patient closely, and if oxygen is available give some during anaesthesia. A simple ward oxygen mask or nasal cannulae may be used.

Ketamine produces some bronchodilation making it a useful anaesthetic drug for patients with asthma.

Gastrointestinal tract.
Salivation is increased.

Skeletal muscle.
Muscle tone is often increased. Spontaneous movements may occur during anaesthesia but reflex response to surgery is uncommon if the patient is adequately anaesthetised.

Uterus and Placenta.
Ketamine crosses the placenta easily and concentrations in the fetus are approximately the same as those in the mother.

The eyes.
The pressure within the eyeball (intra-ocular pressure) rises for a short time following administration. Eye movements may continue throughout surgery. It is not suitable for use in patients with a perforated eye injury or for ophthalmic surgery where a still eye is required.

Routes of Administration

Ketamine can be given by either the intravenous or intramuscular routes to provide surgical anaesthesia. Excellent analgesia and sedation can be obtained with smaller intravenous doses. (It has also been used orally or rectally as a form of premedication. However, this only produces sedation, not surgical anaesthesia and is unpredictable in its effect).

Indications for Use

Ketamine may be used as the sole anaesthetic agent for a large number of superficial operations and procedures in both adults and children. Common procedures undertaken with ketamine anaesthesia include minor to intermediate orthopaedic surgery (especially distal arm or lower leg surgery including manipulation of fractures), gynaecological surgery (eg. dilatation and curettage and other minor surgical procedures), drainage of abscesses, debridement of burns, change of dressings and minor dental procedures, as well as a variety of examinations under anaesthesia.

Administering a Ketamine Anaesthetic (See Table)

Premedication.
As ketamine increases salivation it is best to give atropine at a dose of 10-20 mcg/kg (to a maximum dose of 600mcg) intramuscularly 30 minutes before the ketamine (or alternatively it can be given intravenously at the time of ketamine administration). Some workers now suggest that atropine is not necessary in adults as salivation is not a major problem. However, it has been this author's practice to administer atropine routinely before ketamine anaesthesia. Diazepam 0.15mg/kg orally in adults, or promethazine 0.5mg/kg orally in children may also be given one hour prior to administration of ketamine. Alternatively, diazepam 0.1mg/kg may be given intravenously on induction. Both these drugs will reduce the amount of ketamine required for superficial surgery.

Intramuscular Ketamine.
The traditional dose quoted to produce surgical anaesthesia is 8- 10 mg/kg. Surgery can start approximately 5 minutes after the injection and anaesthesia will last for 20-30 minutes. If the surgery is to last longer, a further intramuscular dose may be given, as half the original intramuscular dose used to produce anaesthesia. Further intramuscular increments may be given as required if surgery is prolonged. Alternatively, following the initial intramuscular dose a smaller intravenous top up dose may be given to maintain anaesthesia - this is especially useful in children (see case history).

In practice the author has found that for many minor surgical procedures (eg. change of burns dressing, setting of minor fractures) an initial dose of 5-7 mg/kg provides adequate anaesthesia especially when combined with diazepam (for adults) or promethazine (for children) premedication. In these instances, if further doses are required, half the original intramuscular dose should again be used. Care must be taken when ketamine is used in children especially if they are malnourished, and in these instances it is always better to start with a smaller dose (ie. 5-7 mg/kg).

Intravenous Ketamine.

If intravenous access is available this route is often preferred. A dose of 1-2 mg/kg is required for induction of anaesthesia and as noted earlier should be given slowly. Surgery can start about 2 minutes after injection with anaesthesia lasting 10-15 minutes. If the duration of anaesthetia needs to be lengthened further doses of 0.5 mg/kg may be given when the depth of anaesthesia lightens. During longer procedures, the anaesthetist should note the time interval between induction and the first top up. He is then able to slowly inject further increments at the appropriate time to reduce patient movement.

Alternatively, a continuous infusion of ketamine may be administered once anaesthesia has been induced. Ketamine is added to a bag of saline or dextrose to make a dilution of 1mg/ml and the infusion administered at a rate of 1-2mls per minute (that is 1-2 mg of ketamine per minute). This is an average adult dose and the rate of infusion should be adjusted as necessary. Some patients may need as much as 4mg/min - this is judged according to the depth of anaesthesia and the size of the patient, care being taken to avoid an overdose.

The amount of ketamine required is again determined by the nature of the surgery (minor procedures requiring a smaller dose) and whether or not the patient has received a premedication. A smaller dose of intravenous ketamine (ie. 1mg/kg) may be used in conjunction with intravenous diazepam (0.1mg/kg) or, in combination with intravenous thiopentone (1-2mg/kg), and both these drugs will help reduce the hypertensive responses occasionally seen when ketamine alone is administered. Care has to be taken when using combinations of drugs and close monitoring of the respiratory system is required to ensure that respiratory obstruction does not occur.

Ketamine administered intramuscularly or intravenously as described will provide adequate operating conditions for a wide variety of superficial minor or intermediate surgical procedures which do not require muscle relaxation. It is not unusual for patients to move spontaneously during this type of anaesthesia and this may be disconcerting to both surgeon and anaesthetist. However, given ketamine's unique properties, excellent anaesthesia for the patient is provided and these movements should not deter surgery. Experience with the drug will ensure that the surgeon and anaesthetist are able to differentiate between spontaneous ketamine movements occurring during full ketamine anaesthesia and spontaneous movements as a result of "lightening" of anaesthesia.

Intravenous infusions of ketamine may be used in conjunction with muscle relaxants and intermittent positive pressure ventilation to produce good conditions for intra-abdominal surgery. In this instance a non-depolarising muscle relaxant such as tubocurarine or alcuronium is used. It is best to avoid pancuronium as the combination of pancuronium and ketamine has been shown to produce marked increases in blood pressure. The patient should be intubated and ventilated with air or oxygen enriched air.

Although general anaesthesia can be provided for abdominal surgery using the techniques described above it is this author's opinion that the EMO system provides cheaper and easier general anesthesia for this type of surgery.

Finally, a case history is included to demonstrate how ketamine can provide adequate surgical conditions for a complex case in less than ideal circumstances.

Case History

A three month old child weighing approximately 7 kg was admitted to a district hospital for possible removal of a large tumour of the buttock which weighed a further 3 kg. An initial biopsy was performed under intramuscular ketamine, surgery lasting approximately 15 minutes.

Histology reported a benign tumour and the surgeons decided a full excision was required. Two choices of anaesthesia were available, either ketamine anaesthesia or ether via a Schimmelbusch mask - the author opted for the use of ketamine. Following an atropine premedication intramuscular ketamine at a dose of 50 mg (7 mg/kg) was used to obtain anaesthesia

to allow intravenous access to be gained. This proved to be difficult and eventually two further doses of 3 mg/kg intramuscular ketamine were used to maintain anaesthesia). Once intravenous access was obtained the operation for removal of the tumour took two hours. This was performed using intermittent intravenous ketamine with iv top up doses of 3 mg (approximately 0.5 mg/kg), resulting in doses of 15 mg of ketamine being given in both the first and second hour. The tumour was successfully removed with minimal blood loss and although spontaneous movement of the legs occurred throughout surgery, the surgeon stated that the conditions were satisfactory. There were no cardiovascular or respiratory complications and a full recovery was made. This demonstrates the value of ketamine anaesthesia when used in less than ideal circumstances.

Table 1. Simple guide to ketamine anaesthesia

1. Intramuscular Administration

Premedication	Atropine 20 mcg/kg IM 30 mins pre-op
	Diazepam 0.15 mg/kg orally 1 hr pre-op in adults
	Promethazine 0.5 mg/kg orally 1 hr pre-op in children
Induction	5-10 mg/kg
Maintenance	3-5 mg/kg IM or 0.5 mg/kg IV as bolus dose

2. Intravenous Administration

Premedication either as for intramuscular administration or, no premedication and administer atropine 10-20mcg/kg iv prior to ketamine

Induction	1-2 mg/kg
Maintenance	IV boluses 0.5 mg/kg

The addition of IV diazepam (0.1 mg/kg) or IV thiopentone (1-2 mg/kg) on induction allows a reduction in the initial dose of ketamine (to 1 mg/kg). These combinations should only be used if no oral diazepam premedication has been given.

3. Infusion techniques

(See text for full description)

LOCAL ANAESTHESIA FOR INGUINAL AND FEMORAL HERNIA REPAIR

Ms J Dunn, Department of Surgery, Bristol Royal Infirmary

Dr C J E Day, Department of Anaesthetics, Bristol Royal Infirmary

Introduction

Local anaesthesia may be employed in hernia operations, either on its own or combined with general anaesthesia. The choice of technique will be influenced not only by local resources and skills, but also by patient preference.

Advantages of Local Anaesthesia for Hernia Repair

With a careful technique, local anaesthesia causes minimal physiological disturbance. This may be particularly useful for patients with cardiovascular or respiratory disease for whom there may be advantages in avoiding a general anaesthetic. The absence of postoperative sedation or drowsiness allows early ambulation and diminishes the requirement for recovery facilities. Local anaesthesia provides postoperative analgesia for up to four hours and may be administered by the surgeon. When adrenaline is mixed with the local anaesthetic (normally in a dilution of 1:200,000) useful vasoconstriction is produced resulting in a relatively bloodless field.

Disadvantages of Local Anaesthesia for Hernia Repair.

Surgery on the awake patient under local anaesthesia must be carried out gently. Although pain sensation is usually blocked by the anaesthetic, traction on certain tissues, particularly the peritoneum, is uncomfortable. The patient should be warned that some sensation may be experienced during the operation but that it will not be painful. Larger hernias, particularly those with incarcerated bowel may prove unsuitable for local anaesthesia.

Some sedation during the operation may be required for anxious patients which loses some of the benefits of avoiding general anaesthesia. Patients who are excessively nervous may be unsuitable for surgery under local anaesthesia.

Local Anaesthetic Agents

Several anaesthetic agents may be used including lignocaine, bupivacaine, procaine and prilocaine. Lignocaine acts more quickly than bupivacaine but wears off more rapidly. Careful attention should be paid to the maximum doses of the local anaesthetic agent that can be used (see Pharmacology of Local Anaesthetics in this issue of Update). Plain lignocaine 0.5% or 1% lignocaine with adrenaline 1:200,000 or plain 0.25% bupivacaine are satisfactory and the maximum amounts that may be used are shown in table 1.

Table 1. Volumes and doses of local anaesthetic which should not be exceeded with different sized patients.

	Small adult 50 - 60 kg	**Medium adult 60 - 70 kg**	**Large adult 70 - 100 kg**
0.5% lignocaine plain	30mls (150mg)	36mls (180mg)	42mls (210mg)
0.5% lignocaine + adrenaline	70mls (350mg)	84mls (420mg)	98mls (490mg)
1% lignocaine + adrenaline	35mls (350mg)	42mls (420mg)	49mls (490mg)
0.25% bupivacaine	40mls (100mg)	48mls (120mg)	56mls (140mg)

These may be calculated as shown on p21 of this issue.

Technique

The patient should be weighed preoperatively and the maximum permissable volume of local anaesthetic calculated. Resuscitation equipment must be available in case the patient develops a reaction to the local anaesthetic and a cannula inserted into a vein.

Explain to the patient that since the operation will be carried out under a local anaesthetic they will not feel pain but that some sensation of touch and perhaps pulling will remain. Reassure the patient that if they experience any discomfort it can easily be remedied by the surgeon injecting some more local anaesthetic.

As the skin is being prepared for surgery explain to the patient what is happening as he may be aware of the sensation. If possible place a surgical towel so that the patient cannot see the operation site.

The patient must be observed throughout the procedure by a trained attendant. The pulse should be monitored and the blood pressure checked regularly. Nervous patients may enjoy talking quietly to a nurse who will be able to inform the surgeon if the patient is in any discomfort. The surgeon should avoid asking the patient if he can feel anything, but rather ask if he is comfortable.

Anatomy

The nerve supply to inguinal and femoral herniae comes from the anterior branches of the six lower intercostal nerves which continue forward on to the anterior abdominal wall accompanied by the last thoracic (subcostal) nerve. The iliohypogastric and ilioinguinal nerves (T12 and L1) supply the lower abdomen. They are blocked by an injection of local anaesthetic between internal and external oblique muscles just medial to the anterior superior iliac spine. The genitofemoral nerve (L1,2) supplies inguinal cord structures and the anterior scrotum via its genital branch and supplies the skin and subcutaneous tissues of the femoral triangle via the femoral branch.

The local anaesthesia should:

1. Produce skin anaesthesia in the line of the incision. This is best achieved by injecting local anaesthetic subcutaneously in the line of the incision.

2. Block the nerve supply to the deeper tissues which are to be dissected and manipulated.

3. Produce anaesthesia of the parietal peritoneum of the hernia and especially the neck of the sac which is very sensitive.

Method

Identify the anterior superior iliac spine and the pubic tubercle. From a point 2cm above and medial to the anterior superior iliac spine inject 5 - 10mls of local anaesthetic under the external oblique aponeurosis in a fanwise fashion. You may feel a 'click' as the needle pierces the aponeurosis. Now inject under the aponeurosis from just lateral to the pubic tubercle, 5mls towards the umbilicus and 5 mls laterally. Wait a short time and then infiltrate subcutaneously in the line of the incision. Allow time for the anaesthetic to take effect before starting the operation. Keep some local anaesthetic ready to inject into the sac when it is exposed, and to supplement any parts which are not adequately anaesthetised (figures 1, 2 & 3).

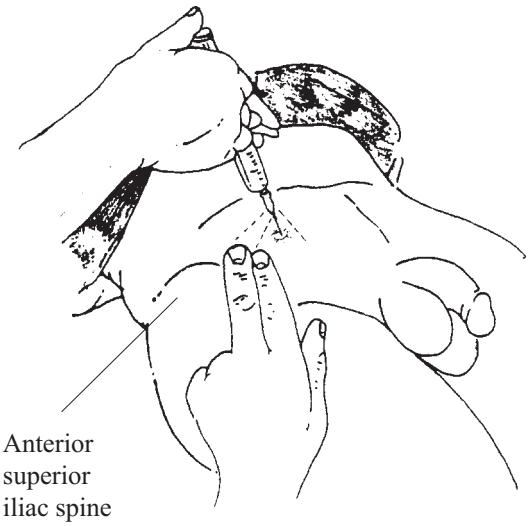
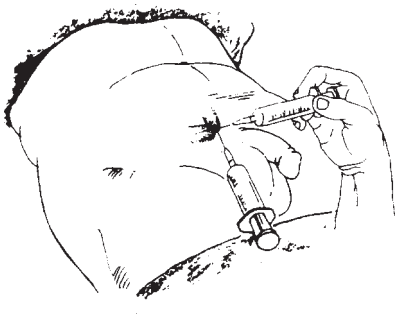
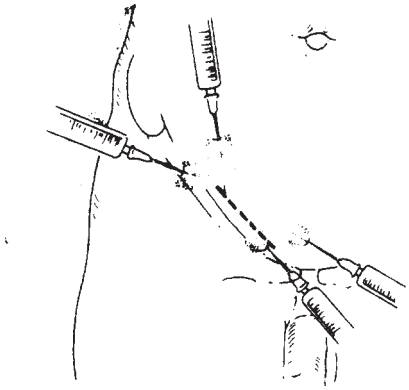


Figure 1.





Figures 2 & 3.

Complications

The side effects which may be produced by local anaesthetics and their management are described on page 23. These are much more likely to occur if local anaesthetic is injected whilst the tip of the needle is in a vein. Always, therefore keep the needle tip moving when infiltrating large volumes of local anaesthetic.

If the block proves inadequate for surgery consider converting to a general anaesthetic. Sometimes sedation with small doses of an intravenous opiate will help.

If traction is applied to the hernial sac without adequate anaesthesia the patient may feel faint and become bradycardic. This is best treated by interrupting the surgery, infiltrating more local anaesthetic and giving some intravenous atropine if required.

If bupivacaine is being used the block will take longer to develop compared with lignocaine. Some surgeons prefer to inject bupivacaine before putting on their gown and gloves and draping the patient. This gives extra time for the block to develop.

THE PHARMACOLOGY OF LOCAL ANAESTHETIC AGENTS

Dr J M Tuckley, Department of Anaesthetics,
Royal Devon and Exeter NHS Trust

Classification

Local anaesthetic agents can be defined as drugs which are used clinically to produce reversible loss of sensation in a circumscribed area of the body. At high concentrations, many drugs that are used for other purposes possess local anaesthetic or membrane stabilising properties. These include Beta-adrenoceptor antagonists, opioid analgesics, anticonvulsants and antihistamines. Most of the clinically useful local anaesthetic agents consist of an aromatic ring linked by a carbonyl containing moiety through a carbon chain to a substituted amino group.

There are 2 classes of local anaesthetic drugs defined by the nature of the carbonyl-containing linkage group. The ester agents include cocaine, procaine, amethocaine and chloroprocaine, whilst the amides include lignocaine, prilocaine, mepivacaine and bupivacaine. There are important practical differences between these two groups of local anaesthetic agents. Esters are relatively unstable in solution and are rapidly hydrolysed in the body by plasma cholinesterase (and other esterases). One of the main breakdown products is para-amino benzoate (PABA) which is associated with allergic phenomena and hypersensitivity reactions. In contrast, amides are relatively stable in solution, are slowly metabolised by hepatic amidases and hypersensitivity reactions to amide local anaesthetics are extremely rare. In current clinical practice esters have largely been superseded by the amides.

Mode of Action

Local anaesthetics cause reversible interruption of the conduction of impulses in peripheral nerves. The primary electrophysiological effect of these compounds is to cause a local decrease in the rate and degree of depolarisation of the nerve membrane

such that the threshold potential for transmission is not reached and the electrical impulse is not propagated down the nerve. There is no effect on the resting or threshold potential, although the refractory period and repolarisation may be prolonged. These effects are due to blockade of sodium channels, thereby impairing sodium ion flux, across the membrane.

Most local anaesthetic agents are tertiary amine bases (B) that are administered as water soluble hydrochlorides (B.HCl). After injection, the tertiary amine base is liberated by the relatively alkaline pH of tissue fluids:

$$B.HCl + HCO_3 \rightleftharpoons B + H_2CO_3 + Cl^-$$

In tissue fluid the local anaesthetic will be present in both an ionised (BH^+) and non-ionised form (B); their relative proportions will depend on the pH of the solution and the pKa of the individual drug. The non-ionised base (B) then diffuses through the nerve sheath, perineuronal tissues and the neuronal membrane, to reach the axoplasm where it partially ionises again:

$$B + H^+ \rightleftharpoons BH^+$$

In the ionised form BH^+, the local anaesthetic enters the sodium channel (from the interior of the nerve fibre) and either occludes the channel or combines with a specific receptor within the channel that results in channel blockade.

In clinical practice, local anaesthesia may be influenced by the local availability of free base (B), as only the unionised portion can diffuse through the neuronal membrane. Thus, local anaesthetics are relatively inactive when injected into tissues with an acid pH (e.g. pyogenic abscess) which is presumably due to reduced release of free base.

Preparations of Local Anaesthetics

Most local anaesthetics are bases that are almost insoluble in water. Solubility is greatly increased by preparation of their hydrochloride salts which are usually dissolved in modified isotonic Ringer solutions. Dilute preparations of local anaesthetics are usually acid (pH range 4.0-5.5), and contain a reducing agent (e.g. sodium metabisulphite) to enhance the stability of added vasoconstrictors. They also contain a preservative and a fungicide. The dilute preparations are presented as percentage solutions of local anaesthetic. For example lignocaine is available in 0.5, 1.0, 1.5 and 2% solutions for injection (with or without adrenaline). A solution expressed as 1% contains 1g of substance in each 100mls. The number of mg/ml can easily be calculated by multiplying the percentage strength by 10. Therefore a 1% solution of lignocaine contains 10mg/ml of solution. A 0.25% solution of bupivacaine has 2.5mg/ml.

Most local anaesthetics produce some degree of vasodilation, and they may be rapidly absorbed after local injection. Consequently, vasoconstrictors are frequently added, to enhance their potency and prolong their duration of action by localising them in tissues. In addition, vasoconstrictors decrease the systemic toxicity and increase the safety margin of local anaesthetics by reducing their rate of absorption (which is mainly dependent on local blood flow). Adrenaline is the most commonly used vasoconstrictor, it is added to local anaesthetic solutions in concentrations ranging from 1 in 80,000 to 1 in 300,000, although most are usually prepared to contain a 1 in 200,000 (5 microgram/ml) concentration of adrenaline.

Practical Point

Adrenaline 1:1000 contains 1 gram of adrenaline per 1000mls solution i.e. 1mg/ml.

To prepare a 1 in 200,000 solution the 1:1000 must be diluted 200 times. This is achieved by taking 0.1ml (= 0.1mg) and adding 19.9 mls of local anaesthetic solution.

The vasoconstrictor felypressin is added to some local anaesthetics (i.e. prilocaine) in a concentration of 0.003 i.u./ml. Felypressin is a non catecholamine vasoconstrictor that is chemically related to vasopressin, the posterior pituitary hormone.

The effect of vasoconstrictors on prolonging the duration of anaesthesia varies according to the local anaesthetic employed and the site of the injection. For example the duration of action of all agents is prolonged by the addition of adrenaline when used for infiltration anaesthesia and peripheral nerve blocks. Adrenaline also increases the duration of extradural anaesthesia when added to procaine,

mepivacaine and lignocaine but does not alter markedly the duration of action of extradural prilocaine, bupivacaine or etidocaine.

Adrenaline containing solutions should **never** be used for infiltration around end-arteries i.e. penis, ring block of fingers or other areas with a terminal vascular supply as the intense vasoconstriction may lead to severe ischaemia and necrosis. Maximum safe dosages are often quoted for local anaesthetics with and without vasoconstrictors (table 1), but such recommendations should be treated with caution as they ignore variations caused by factors such as the site of injection, the patient's general condition and the concomitant use of a general anaesthetic. For example if one assumes that a plasma concentration of lignocaine of 5 microgram/ml is required for the development of toxic symptoms, this would be achieved by the administration of approximately 300mg in the intercostal area, 500mg extradurally, 600mg in the region of the brachial plexus and 1000mg subcutaneously. Thus recommendation of a single maximum dose without regard to the site of injection is meaningless.

Table 1. Upper dose limits for commonly used local anaesthetic agents

	Plain solution mg/kg	**With adrenaline mg/kg**
Prilocaine	6	9
Lignocaine	3	7
Bupivacaine	2	2

The addition of adrenaline reduces the peak concentration in blood, but the degree of this reduction again depends on the site of injection and the specific local anaesthetic agent.

Clinical Uses of Local Anaesthetics

Local anaesthetic requirements and activity vary considerably. Selection of an appropriate agent in a specific situation requires knowledge of the clinical needs and pharmacological properties of the various anaesthetic drugs currently available (table 2).

Topical Anaesthesia

Local anaesthetics may be applied to the skin, the eye, the ear, the nose and the mouth as well as other mucous membranes. In general, cocaine, amethocaine, lignocaine and prilocaine are the most useful and effective local anaesthetics for this purpose. When used to produce topical anaesthesia, they usually have a rapid onset of action (5-10mins) and a moderate duration of action (30-60 mins). Cocaine is a potent vasoconstrictor and is useful in the reduction of bleeding as well as topical anaesthesia. Other local anaesthetic agents may be absorbed in significant amounts particularly after topical application to the more vascular areas, and fatalities have occurred after application of these agents to mucosal surfaces.

Absorption of local anaesthetics through intact skin is usually slow and unreliable and high concentrations (e.g. 20% benzocaine or 40% lignocaine) are required.

EMLA cream is a eutectic mixture of local anaesthetics which may be used to provide surface anaesthesia of the skin (particularly in paediatric practice). It is a mixture of the base forms of lignocaine and prilocaine in equal proportions in an emulsion. Cutaneous contact (usually under an occlusive dressing) should be maintained for at least 60 minutes prior to venepuncture.

Infiltration Anaesthesia

Infiltration techniques are used to provide anaesthesia for minor surgical procedures. Amide anaesthetics with a moderate duration of action are commonly used (lignocaine, prilocaine and mepivacaine). The site of action is at unmyelinated nerve endings and onset is almost immediate. The duration of local anaesthesia is variable. Procaine has a short duration of action (15-30 min), while lignocaine, mepivacaine and prilocaine have a moderate duration of action (70-140 min). Bupivacaine has the longest duration of action (approximately 200 min). The addition of adrenaline (1 in 200,000) will increase the quality and prolong the duration of anaesthesia.

Table 2. Pharmacological effects and clinical uses of local anaesthetics

	Ester or Amide	Onset of Action	Duration of Action	Clinical Use	Properties
Procaine	Ester	Slow	Short	Limited Vascular Spasm Diagnostic procedures	Vasodilatation Allergenic
Amethocaine	Ester	Slow	Long	Topical anaesthesia Spinal anaesthesia	High systemic toxicity
Chloroprocaine	Ester	Fast	Short	Peripheral anaesthesia Obstetric extradural blocks	Low systemic toxicity
Mepivacaine	Amide	Fast	Moderate	Infiltration Peripheral nerve blocks	Versatile Moderate vasodilatation
Prilocaine	Amide	Fast	Moderate	Infiltration IVRA Peripheral nerve blocks	Methaemoglobinaemia at high doses Least systemic toxicity of amides
Bupivacaine	Amide	Moderate	Long	Infiltration Peripheral nerve blocks Extradural & spinal blocks	Separation of sensory and motor blockades
Etidocaine	Amide	Fast	Long	Infiltration Peripheral nerve blocks Extradural blocks	Profound motor blockade
Lignocaine	Amide	Fast	Moderate	Infiltration/Topical IVRA Peripheral nerve blocks Extradural and spinal blocks	Most versatile agent Moderate vasodilatation

IVRA : Intravenous regional anaesthesia

Conduction Anaesthesia

Conduction anaesthesia can be divided into minor nerve blockade (e.g. ulnar, radial or intercostal), and major blockade of deeper nerves or trunks with a wide dermatomal distribution (e.g. brachial plexus blockade). For each individual agent the duration of anaesthesia will be determined more by the total dose of the drug rather than the volume or concentration of drug used.

When amide local anaesthetics are used to produce minor nerve blockade, they have a relatively rapid onset of action (5-10min). Lignocaine, mepivacaine and prilocaine have a moderate duration of action (1-2 hr), while bupivacaine and etidocaine produce local anaesthesia for 2-6 hrs.

In major nerve blockade the onset is more variable, mainly due to anatomical factors which can delay or restrict the access of the local anaesthetic to its site of action. In general lignocaine, mepivacaine and prilocaine have a faster onset of action (10- 15 min) than bupivacaine (15-30 min). Analgesia persists for 3- 4 hr with lignocaine, prilocaine and mepivacaine, but up to 10 hrs with bupivacaine.

Extradural Anaesthesia

Local anaesthetic solutions are deposited in the epidural space between the dura mater and the periosteum lining the vertebral canal. The epidural space contains adipose tissue, lymphatics and blood vessels. The injected local anaesthetic solution produces analgesia by blocking conduction at the intradural spinal nerve roots.

The quality and extent of the blockade produced by each agent is determined by the volume as well as the total dose of the drug. The spread of local anaesthetic solutions may be more extensive in pregnant women as the volume of the potential space is reduced by venous engorgement in the epidural space. Enhanced effects may also be seen in the elderly and in patients with arteriosclerosis due to impairment of vascular absorption from the epidural space.

Bupivacaine (0.5%) or lignocaine (1.5-2.0%) are usually used to produce extradural anaesthesia. Repeated administration of lignocaine or mepivacaine into the epidural space may result in a diminished response with each subsequent dose (tachyphylaxis). This may be due to local changes in pH due to the relative acidity of these solutions. The reduction in pH may reduce the amount of free base available for diffusion across the neuronal membrane.

Spinal Anaesthesia

The introduction of local anaesthetic solutions directly into the cerebrospinal fluid (CSF) produces spinal anaesthesia. The local anaesthetics do not have to cross tissue or diffusion barriers and also the central attachments of the ventral and dorsal nerve roots are unmyelinated, which allows rapid uptake of the free base. There is a faster onset of action and a smaller dose is required. Spinal anaesthesia produces a similar clinical effect with a dose approximately ten times smaller than that needed for extradural anaesthesia.

Solutions of amethocaine (0.2%), lignocaine (5%), prilocaine (5%) bupivacaine (0.5%) and mepivacaine (4%) are commonly used to produce spinal anaesthesia. Prilocaine and mepivacaine have a slightly longer duration of action than lignocaine; bupivacaine has the longest duration of action.

In pregnancy, compression of the inferior vena cava by the pregnant uterus leads to distension of the vertebral venous plexus and reduces the volume of the subarachnoid space. Consequently the degree of blockade is enhanced and reduced doses are required.

Intravenous Local Anaesthesia

Intravenous regional anaesthesia (IVRA) is a useful method of providing analgesia for minor surgical procedures. The local anaesthetic agent is injected into a vein of a limb that has been previously exsanguinated and occluded by a tourniquet. The site of action is probably the unmyelinated nerve fibres, reached by retrograde spread in the vascular bed. The onset of action is almost immediate.

Lignocaine or prilocaine are commonly used and systemic blood levels of these agents are unlikely to be significant if the tourniquet is released more than 15 min after injection.

Bupivacaine and etidocaine **should never be used for IVRA!** They are significantly protein bound and once the tourniquet is released there is a risk of cardiotoxicity. Several deaths have been reported during IVRA with bupivacaine.

Toxicity of Local Anaesthetic Agents

Local anaesthetic agents are relatively free from side effects if they are administered in an appropriate dosage and in the correct anatomical location. However, systemic and localised toxic reactions may occur, usually from the accidental intravascular or intrathecal injection, or the administration of an excessive dose of the local anaesthetic agent. Systemic reactions to local anaesthetics involve primarily the central nervous system (CNS) and the cardiovascular system.

The initial symptoms of CNS toxicity involve feelings of light- headedness, dizziness and circumoral paraesthesia which may precede visual and/or auditory disturbances such as difficulty focusing and tinnitus (ringing in the ears). Other subjective CNS symptoms include disorientation and feelings of drowsiness. Objective signs of CNS toxicity are usually excitatory in nature and include shivering, muscular twitching and tremors initially involving muscles of the face and distal parts of the extremities. Ultimately, generalised convulsions of a tonic-clonic nature occur. If a sufficiently large dose, or rapid intravenous injection of local anaesthetic is given, the initial signs of excitation may progress very rapidly to generalised CNS depression and coma. Respiratory depression may result in respiratory arrest. CNS toxicity is exacerbated by hypercarbia and acidosis.

Cardiovascular toxicity usually occurs at doses and blood concentrations which are higher than those required to produce CNS toxicity. Local anaesthetics can exert a direct effect both on the heart and the peripheral blood vessels.

Extremely high concentrations of local

anaesthetics depress spontaneous pacemaker activity in the sinus node resulting in sinus bradycardia and sinus arrest. They also exert a dose - dependent negative inotropic action on isolated cardiac tissue. The more potent local anaesthetics depress cardiac contractility at lower concentrations than the less potent drugs.

Local anaesthetic agents appear to exert a biphasic effect on peripheral vascular smooth muscle. In lower doses they may increase peripheral vascular resistance, and in higher doses, reduce it. Cocaine is the only anaesthetic that causes vasoconstriction consistently because of its ability to inhibit the re-uptake of noradrenaline by storage granules at the synapse. The excess concentration of free circulating noradrenaline is responsible for the vasoconstriction associated with the use of cocaine. In general, a direct relationship exists between the anaesthetic potency and cardiovascular depressant potential of the various agents. The more potent drugs e.g. bupivacaine and etidocaine, have been reported to cause rapid and profound cardiovascular depression in some patients following accidental intravascular injection. Severe cardiac arrhythmias such as resistant ventricular fibrillation may occur.

Management of Acute Toxicity

The airway is maintained and oxygen administered by facemask, using artificial ventilation if apnoea occurs. Convulsions should be treated with anticonvulsant drugs such as thiopentone (150-250mg I.V.) or diazepam (10-20 mg I.V.) repeated as necessary. Profound hypotension and brady-arrhythmias should be treated with intravenous atropine (0.5 - 1.5mg) and colloid or crystalloid infusions as plasma expanders may be necessary. Occasionally adrenaline may be required for severe hypotension or bradycardia.

In patients with ventricular fibrillation due to bupivacaine toxicity, cardiopulmonary resuscitation should be continued for at least 60mins. Bretyllium may facilitate cardioversion.

Practical Use of Local Anaesthetic Agents

Example 1

A 70 kg male is scheduled for axillary block. The anaesthetist decides to use 30 mls of solution. He only has 2% plain lignocaine available. What should he do?

A 2% solution contains 20mg/ml lignocaine. The toxic dose of lignocaine is 3mg/kg without adrenaline added and 7mg/kg with adrenaline.

The maximum safe dose of lignocaine for this patient is 210mg without and 490 mg with adrenaline.

30mls of 2% plain lignocaine gives 600mg. The anaesthetist must therefore dilute the lignocaine and add adrenaline to it.

20 mls of 2% plain lignocaine contains 400 mg lignocaine which can be made up to a 30 ml solution with 10 mls N. Saline.

The adrenaline is 1:1000 i.e. 1mg/ml and he requires 1:200,000 i.e. 5 microgram/ml. Therefore for every 20mls of local anaesthetic solution he should add 0.1ml of 1:1000 solution; a total of 0.15mls for his 30ml mixture.

Example 2

A 6 year old child weighing 20kg is scheduled for hernia repair. The anaesthetist wishes to supplement general anaesthesia with an ilioinguinal block. He only has 0.5% plain bupivacaine. What should he do? Ideally he would wish to use at least 10mls of solution. The maximum dose of bupivacaine which can be given is 2mg/kg ie. 40mg.

10 ml of 0.5% solution should be diluted with 10ml normal saline to give 20ml 0.25% solution. 10 ml of this solution should be used to produce an ilioinguinal block.

THE PHYSIOLOGY OF NEUROMUSCULAR TRANSMISSION

Dr Shalini V Dhir, Dr Achal K Dhir and Dr LKG Vimal, All India Institute of Medical Sciences, New Dehli, India and Ahmadu Bello University Teaching Hospitals, Zaria, Nigeria

A medullated motor nerve fibre loses its myelin sheath when it reaches a striated muscle fibre. Each terminal branch lies in a groove of the muscle fibre junctional cleft, forming the neuromuscular junction. Thus the nerve terminates at the 'pre-synaptic membrane', which is separated by a 'junctional/ synaptic cleft' from the 'post synaptic membrane' of the muscle (figure 1).

The mechanism of neuromuscular transmission is the liberation of acetyl choline which is synthesised in the terminal axoplasm from choline and acetyl coenzyme A under the influence of choline-O- acetyl transferase. It is loaded into vesicles by a specific carrier mediated system. Eighty percent of acetyl choline is in these vesicles and 20% is dissolved in the axoplasm. These vesicles are synthesised in the cell bodies of lower motor neurones of the spinal cord or brain stem and transported to the nerve terminals with the help of micro-tubules. In the nerve endings they are repeatedly refilled and re-used. About half a million vesicles are present in the axoplasm of each nerve ending and are concentrated near areas of thickened terminal axonal membrane ie. active zones.

There are four ways in which acetyl choline can be released:

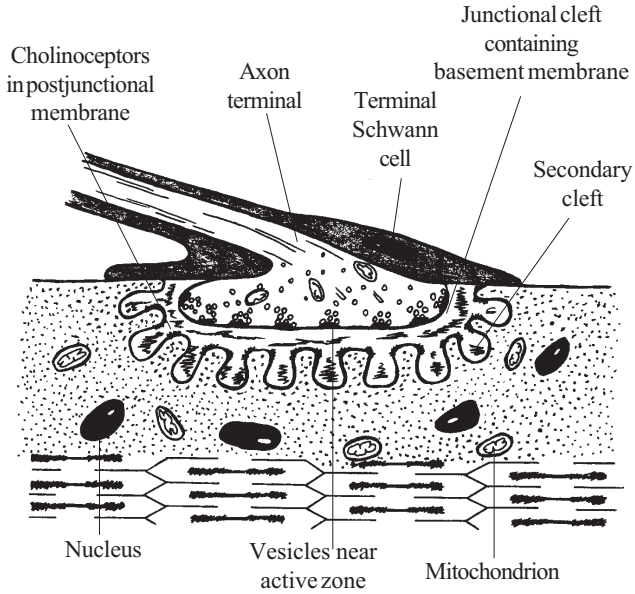


Figure 1. Neuromuscular junction

1. Constant leak or molecular sieve

2. Spontaneous quantal release leading to small transient depolarisations of 0.5mV giving rise to miniature end plate potential (mepp) at a frequency of about 2Hz. This is too small to cause muscle contraction. The function of mepp is not yet known.

3. Additional type of release that is quantal but unrelated to nerve impulse and occurs only when normal ion dependant quantal release is impaired eg botulinum toxin.

4. Nerve impulse evokes huge quantal release (=300 quanta) of acetyl choline and leads to the depolarisation of the post junctional membrane. This constitutes full size end plate potential (epp) and triggers excitation-contraction coupling followed by muscular activity.

Release of Acetyl Choline

Sodium channels are present at pre-terminal parts of axons, ie the region just after the end of myelination but absent from the terminal proper. Potassium and sodium channels are present at the terminal part of the ending ie. from where the transmitter release occurs. The nerve action potential causes an inward sodium current at the pre-terminal membrane. This promotes a local circuit current that depolarises the terminal part by electronic spread. Subsequently, K^+ current flows outwards through the terminal membrane to repolarise terminals. The depolarisation of terminal membranes causes opening of voltage dependant calcium (Ca^{2+}) channels and inward flow of Ca^{2+} begins. Outward K^+ far exceeds inward Ca^{2+} normally, so net current is outwards and repolarises the membrane thereby closing the Ca^{2+} channels. Ca^{2+} that flows into the terminal axoplasm is essential for acetyl choline release. By a process largely unknown but involving calcium calmodulin, there is a synchronous release of many quanta of acetyl choline into the gap.

Action of Released Acetyl Choline

Acetyl choline receptors or cholinoceptors are present in the post-junctional membrane of the motor end plate and are nicotinic in nature. These cholinoceptors are bound by cytoskeleton onto the shoulders of the

junctional fold in clusters so that each end plate has millions of receptors. The receptor has a central pore that functions as an ion channel when in open state. Acetyl choline molecules released in response to nerve impulses bind (about once each) with the recognition site of the receptors inducing a conformational change. This results in opening of receptor operated ion channels, allowing pulses of inward ionic current (mainly Na^+) to flow. Many elementary current pulses summate to produce end plate current (epc). The epc depolarises the end plate membrane (epm) to produce an end plate potential (epp). When the epp reaches a critical threshold, it triggers off an all-or-none propogating action potential that passes around the sarcolemma to activate the contractile mechanism via the T-tubules, sarcoplasmic reticulum and contractile proteins. So, release of acetyl choline constitutes an amplification process that allows minute electric current of nerve endings to excite enormously greater membranes of muscle fibres.

Fate of Acetyl Choline

Released acetyl choline is rapidly hydrolysed to inactive choline and acetate, catalysed by the enzyme acetyl cholinesterase.

MAINTAINING YOUR LARYNGOSCOPE

Mike Yeats, Derriford Hospital, Plymouth

If laryngoscopes are looked after they will last for many years. Most are simply specialised torches with a handle containing the batteries and a blade which holds the bulb (figure 1). The electrical current flows from the battery to the bulb through an insulated contact at the top of the handle. When the blade is opened for use the bulb lights because the contact in the handle makes a circuit with another contact in the blade which is connected to the bulb with a wire. The current flows back from the bulb to the battery via the metal of the blade and handle.

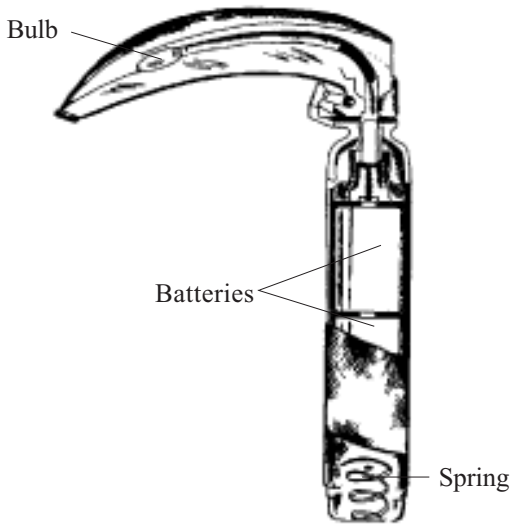


Figure 1. Laryngoscope

General care. Always clean your laryngoscope blade as soon as possible after use. Ideally use hot soapy water to remove secretions and then soak the blade in an antiseptic solution to disinfect it. A nailbrush will help you clean the blade.

Remove the batteries if the laryngoscope is not to be used for a few days as they may corrode the inside of the handle and cause severe damage. The only exception to this is if you use rechargeable (Nickel Cadmium or Nicad) batteries. Rechargeable batteries last for a long time and are a good investment provided you have a suitable charger and supply of electricity.

At regular intervals clean the handle and blade including the electrical contacts at the base of the blade and handle.

Problems with Laryngoscopes

No light. This may be caused by a loose bulb, a faulty bulb, flat batteries or a break somewhere in the electrical pathway between the batteries and the bulb. The instructions below will help you locate the problem. Follow them in order until your laryngoscope is working again.

1. Check with your fingers that the bulb is firmly screwed in.

2. Insert fresh batteries.

3. Replace the bulb with a new one.

4. If the laryngoscope is still not working the electrical pathway should be checked. An electrical meter is the best way to do this but if you don't have access to one you can make a suitable testing device as follows. Get a piece of wire about 6 inches long and strip back the ends of the insulation. Wrap one of the bare ends round a torch bulb and tape it in place as shown in figure 2. With this device you will be able to isolate and bypass each part of the electrical circuit. The bulb will light if current passes through it.

a) First demonstrate that the device works by holding the base of the bulb against the top (positive terminal) of the batteries whilst pressing the bare wire against the bottom (negative terminal) of the batteries. Hold the batteries firmly together while doing this. If the batteries are fresh and the torch bulb is working, it will light (figure 1).

Figure 1.

Figure 2.

b) Take a second piece of wire and confirm the laryngoscope bulb is working by wrapping one end of the wire around the bulb and checking it as described above (figure 2). After these two tests the batteries and bulb have each been demonstrated to be working and you should reassemble the laryngoscope and check whether it now works. If it does not, proceed with the rest of the checking procedure.

c) Replace the batteries and screw on the base of the scope. Hold the end of the bulb against the contact at the top of the handle and the other end of the wire against the body of the scope. If the bulb lights the contacts in the handle are working but if the scope does not work with the blade in place there is a fault in the blade. Test this as described in (d). If, however, the bulb does not work there is a fault in the contacts with the batteries in the handle. Remove the batteries and inspect the base of the handle. Remove and clean the spring and check where it contacts the body of the laryngoscope. Clean off any corrosion with fine sandpaper. Clean both the outside and inside ends of the contact at the top of the handle (use sandpaper wrapped around a screwdriver) and then replace the batteries and recheck as explained at the start of this section. If the bulb still does not light then you should remove the contact at the top of the handle using a correctly sized screwdriver. Inspect this and check that the insulation is intact and clean off any corrosion and then replace the contact. Note that this contact is sprung to help keep a good contact with the batteries.

The bulb should now light and you should try it with the laryngoscope blade in place. If the scope still does not work there is a fault in the electric pathway in the blade.

d). The contact on the blade is an insulated pin soldered to the wire going to the bulb. The contact point is formed by a blob of solder on top of the pin. The wire contacts the bulb via an insulated pin with a spring to help hold the bulb.

First clean the contact on the blade and check that it makes good contact with the handle. If the contact is worn it can be reformed using a soldering iron.

Using your testing equipment hold the positive end of the batteries to the blade contact whilst connecting the other end of the batteries to the wire which should be attached to the body of the blade. If the bulb does not light it indicates that the wire within the blade or its final contact with the bulb has failed. (You must be certain that the bulb is a good one and has been screwed in properly).

If the bulb contact or wire has failed it should be replaced by soldering a piece of telephone wire in its place. The wire is firstly detached from the blade contact and removed from the bulb end of the blade. It can then be replaced. Ensure that you relace the insulating sleeve accurately.

Peeling chrome. If this happens to the blade on your laryngoscope it may cut a patient. Ease off loose chrome with a scalpel blade and than rub over the edge of the remaining chrome with some fine sandpaper. Use some water on the blade to do this. When you have finished check the chrome edge is smooth by running cotton wool over it (it will catch on any rough edges).

Corroded batteries. If the batteries are stuck in the handle try boiling the handle in a pan for a while. If this does not loosen them the batteries must be removed using a large drill. However do not go too deeply or you will damage the contact at the top of the handle. Clean the inside up as well as you can after this problem.

CORRESPONDENCE

Sir,

I am very grateful to receive Update in Anaesthesia which is proving a valuable journal especially to nurse anaesthetists like myself working in Africa. In Dr Dobson's article on drawover anaesthesia (Issue 3) a diagram is included of a suggested plan for general anaesthesia. Dr Dobson suggests that if one is not trained in endotracheal intubation then spinal or ketamine anaesthesia is a good option.

I would like to question this advice. It is well reported, and I have personally seen spinal anaesthesia resulting in apnoea and severe hypotension. Without the knowledge of how to perform endotracheal intubation this situation could result in a serious outcome. What are your comments?

Mr Emmanuel Ladislaus
Department of Anaesthesia
Bugando Medical Centre
PO Box 1370
Mwanza Tanzania

Reply

Sir, Thank you for the opportunity to reply to Mr Ladislaus. I entirely agree with his statement that severe complications such as hypotension can result from the use of spinal anaesthesia. It is of course essential to be ready to treat complications by having the means of resuscitation ready whenever anaesthesia of any sort is administered, and I should probably have made this clearer - I was trying to make the diagram straightforward.

We do however have to recognise that in many hospitals there is no specialist anaesthetist, and the nurses and doctors providing anaesthesia may not be confident of their ability to intubate. In this situation it is important to get someone trained to perform this simple and lifesaving manoeuvre, but in the meantime, when a patient needs surgery they will probably be safest if a spinal or ketamine technique is used.

The most likely complication of spinals is hypotension, and the first line treatment in such a case is to give a rapid intravenous infusion of fluid and a vasoconstrictor such as ephedrine. Very severe hypotension, or a very high spinal can result in a cardiorespiratory arrest, but even then the patient's breathing could be supported with a facemask if no-one present had intubation skills.

It is of course of the greatest importance that those who practice anaesthesia should be fully proficient in airway skills, and especially intubation.

Dr M Dobson
Nuffield Department of Anaesthetics
John Radcliffe Hospital
Oxford OX3 9DU
UK

Sponsored by: Overseas Development Administration
Typeset by : Graphics Department, Royal Devon & Exeter Healthcare NHS Trust, Exeter UK.
Printed in Great Britain by : BPC Wheatons Ltd, Exeter UK.
Correspondence to: Dr I H Wilson, Anaesthetics Dept., Royal Devon & Exeter Healthcare NHS Trust, Barrack Road Exeter, EX2 5DW, UK