UPDATE IN ANAESTHESIA

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## WORLD ANAESTHESIA

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### Editorial

Welcome to Update in Anaesthesia issue 18!

About 4 months ago, we received a request to publish an article on explosions in the operating theatre, for colleagues using ether anaesthesia. Dr Busato, one of our members from Italy, kindly wrote the paper in this edition, and we hope that the information will prove practical. The editors of Update are always delighted to receive suggestions for articles, and will do our best to cover the subjects requested.

The World Congress in Anaesthesia is taking place in April 04 - please see the advert on the back page. We are looking forward to meeting colleagues from many different parts of the world. The WFSA will have a stand at the meeting, and this should be a useful meeting point.

The Association of Anaesthetists of Great Britain and Ireland (AAGBI) has recently sponsored the production of an anaesthesia resource CDROM in conjunction with World Anaesthesia. This will contain the text of all previous editions of Update, the Primary Trauma Care Manual and the latest UK Resuscitation Council Guidelines. The World Health Organisation publications - Anaesthesia at the District Hospital, WHO Drug Formulary and The Clinical Use of Blood are included. In addition we are delighted that the Editor of Anaesthesia (the journal of the AAGBI) has given us permission to put the last fours years of the journal on the CDROM. Altogether this library of anaesthesia related material will prove a fantastic resource to anyone with access to a computer. The CDROMs will be available for a small cost in Paris at the AAGBI stand, and readers of Update can contact michael.dobson@nda.ox.ac.uk to find out how to receive a copy.

The editors of Update in Anaesthesia would like to express their grateful thanks to Dr Kester Brown, WFSA President, and Dr Roger Eltringham Chairman of the WFSA Publications Committee, for their kind help and enthusiastic support over the last 4 years.

**Iain Wilson**
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# TRANSPORTATION OF THE CRITICALLY ILL AND INJURED PATIENT

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## Terminology

***Primary transport***: from the incident site to a medical facility.

***Secondary transport (Inter-hospital)***: patient moved between two hospitals, usually for an increased level of medical care not available locally.

***Intrahospital transport***: movement of patients within the hospital or its campus for investigations or treatment not available at the ward or intensive care location. (eg CT scan)

## History

The primary and secondary transport of critically ill patients are complementary to one another. Primary transport from the site of illness or incident to organised medical care has now moved on from the old 'scoop and run' philosophy. Developments often occurred following experiences in major conflict. The Knights of St John crusading in the 11th century received training from Arab and Greek physicians. They acted as attendants to soldiers at the point of injury and then transported them to treatment points. Baron Dominique Jean Larrey, Napoleon's surgeon-in-chief, is credited with the first organised vehicular ambulance service, taking medical attendants into the battlefield with the French army. Until recently, ambulances were still not designed with the patient's well being in mind. 1944 saw the first helicopter evacuation of combat casualties in Burma. In Vietnam 90% of hospitalised US battle casualties were evacuated by helicopter.

Physician escorts for secondary transports are a relatively recent phenomenon. Systems in North America were the first to formalise these arrangements in the 1950's. Since then transfer teams and 'retrieval' services have been introduced in many large hospitals and health systems world-wide including Africa and SE Asia.

## Primary transport

Currently most injured patients are transported from an accident site to the nearest hospital emergency department by land-ambulance with ambulance paramedics providing care at the incident. The presence of a doctor on board primary response units continues to be a source of controversy. Some data have suggested improved pre-hospital stabilisation and long term survival in victims of major trauma attended by a medical team containing a doctor, as opposed to a paramedic only response unit.

Most of the principles of trauma care covered in the primary trauma care course (PTC) are applicable in the pre-hospital setting. This will obviously depend on the level of training of the attendants and whether the facilities exist for more advanced levels of care. However, there are some simple cornerstones of treatment which form the basis of care and are available to the most basic of services:

- **Give oxygen** preferably by face mask and at high flows
- **Preserve blood volume**; compression of bleeding sites and limited fluid resuscitation
- **Splinting and packaging**; unnecessary movement of injured patients provokes bleeding. Basic splinting of fractures will provide good analgesia and contribute to preserving blood volume.
- **Analgesia**; this is humane and should be given when needed. Morphine is cheap and effective. Non-steroidal analgesia should be avoided initially in the trauma patient due to adverse effects on platelet aggregation and renal function.
- **Expeditious transport**; ultimately the patient needs to be in hospital and receiving a higher level of care than can be provided 'in the street'. Unless treatment pre-hospital is beneficial it is often better to accept the limitations of what is achievable and move them to hospital quickly but safely.

It has been argued in the past that the provision of more advanced pre-hospital care services in less affluent areas of the world is of little benefit unless sophisticated levels of care exist in receiving hospitals. This has recently been refuted in a study looking at pre-hospital care services in Cambodia and Iraq, showing a 40% reduction in mortality from major trauma after the introduction of an ambulance service with trained attendants.[1]

## Secondary transport

Guidelines for the secondary transport of patients have been produced by the Australian and New Zealand College of Anaesthetists in 2003 and the UK Intensive Care Society in 2001. These attempt to bring together advice from different sources and encourage an improvement in standards. The safe and successful transport of the critically ill should follow these principles:

- **Organisation**. Planning of transfers should reflect local facilities and the availability of appropriately trained staff. Clear guidelines and channels of communication must exist in each hospital. In the absence of a recognised transfer team, each hospital must provide adequate staff and facilities for outgoing patients, adhering as closely as possible to the standards of care provided in the hospital. Flexibility in staffing and rostering arrangements must exist to allow this to happen, where possible not organising transfers in the middle of the night when resources and staffing are at their most stretched. The planning phase is vital for a smooth transfer and a briefing format is worth considering. The military use these as a basis for most missions.
- **Transfer decisions**. These must be made jointly by senior medical staff in both the receiving and referring hospitals. The risk of transfer arising from the patient's condition must be set against the additional risk from the movement (tipping, vibration,

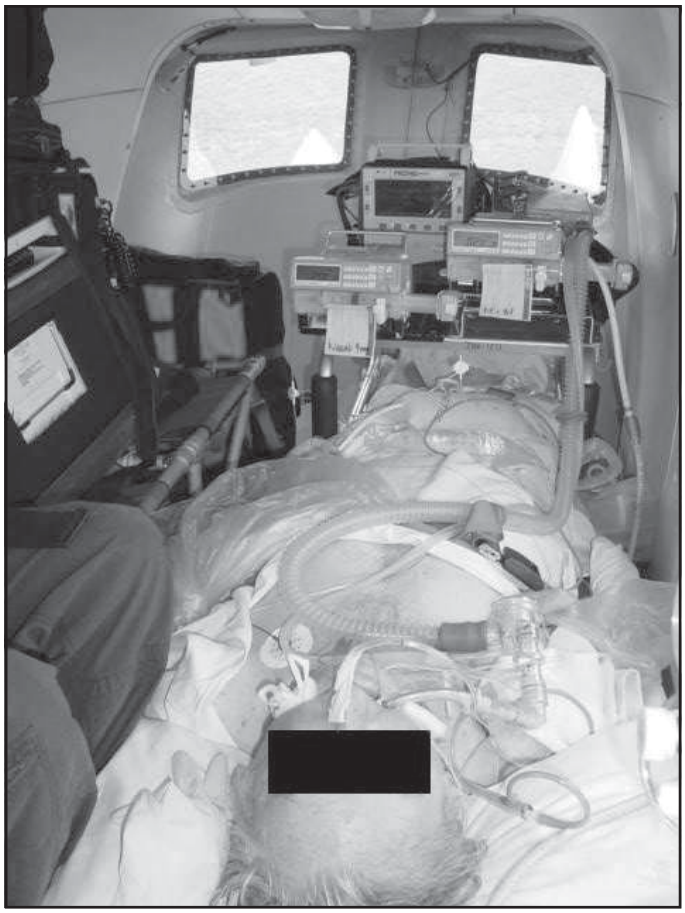
acceleration/ deceleration etc) as well as possible pressure and temperature changes which may adversely affect cardio-respiratory physiology. The risks to staff of injury or accident must not be overlooked especially in adverse weather and in unsecured areas at night. The decision to send a retrieval team or use staff from the referring hospital will depend on the availability of such resources and the clinical urgency of the case.

- **Transfer mode**. Keeping things simple has much to commend it. The choice of transport will obviously be influenced by what is available but other factors will come into play. The urgency of the transfer and prevailing road and weather conditions, as well as the range and speed of available vehicles. It may be necessary for a receiving hospital to provide the vehicle (and sometimes the personnel!) if this is considered appropriate. The use of aircraft and in particularly helicopters can appear attractive, however they usually do not reduce travel time unless the distances are large and the terrain rough.

- **Transfer vehicle requirements**. Well-maintained and adequately equipped vehicles should be used. Ease of access, proper heating control, lighting and good communications are all considerations. An oxygen supply and suction are also mandatory. Safety provision for staff is important and noise and vibration levels should be at acceptable levels.

- **Accompanying personnel**. In addition to the normal complement of crew on a given vehicle there should be two accompanying staff for the critically ill patient. An experienced doctor with skills in resuscitation and airway control should be responsible for the patient and preferably be experienced in undertaking transfers. Another doctor or experienced nurse, paramedic or technician with familiarity in transfer procedures, the vehicle and equipment should be present and acts as an assistant to the responsible doctor.

- **Equipment** must be suited to the environment i.e. be durable and lightweight and have sufficient battery life. A monitored oxygen supply with a safety margin of two hours on the transfer time is essential. There should be storage space for equipment and staff should be appropriately clothed. A portable ventilator with disconnection and high-pressure alarms and the ability to provide PEEP and variable $FiO_2$, I: E ratio, respiratory rate and tidal volume. Portable monitors giving $SaO_2$, ECG and non-invasive BP. The facility to monitor invasive pressures (arterial and CVP) are preferable depending on resources. A dedicated equipment bridge, containing ventilator, monitoring equipment and infusion devices is becoming the method of choice for providing theses requirements. (Figure 1) These can be manufactured locally but need to be robust and withstand the rigors of transport, especially over rough terrain. Alarms should be visible as well as audible. Suction and defibrillation should be immediately available. A warming blanket is also a consideration in cold climates. A reasonable range and supply of drugs should be carried with syringe pumps to administer them, ensuring that all such devices have charged and spare batteries (the Braun Perfusor FM compact syringe driver will run off standard AA battery power). **Non of these requirements should seriously affect the referring hospitals ability to deal with emergencies in terms of staff or equipment whilst the transfer team is away**. The vehicle should have communications able to contact the base hospital in emergencies and the receiving hospital to fore warn of any problems.



- **Preparation for transfer**. Stabilisation and meticulous preparation are the keys to a successful transfer. All personnel should familiarise themselves with the patient and the current treatment. As with the planning phase, it is useful to have a checklist to avoid omissions. This list is a useful starting point, when considering any treatment outside the hospital setting, whatever the circumstances. Full clinical examination with reference to on-going monitoring should be carried out. Chest drains should be fitted with flutter valves and be easily observed. A review of recent investigations: CXR, other X-rays, haematology and biochemistry results.

**Preparation for transfer checklist**

- Respiration
- Circulation
- Head
- Other injuries
- Monitoring
- Line placement and securing
- Investigations

A patient should not be transported until all possible sources of continuing blood loss and sepsis have been located and controlled. Satisfactory perfusion and optimum tissue oxygen delivery must be achieved. Respiratory support is fundamental. Intubation during transfer is difficult and hazardous; if any doubts exist about

respiratory function intubation and mechanical ventilation must be carried out pre-transport. For ventilated patients the pattern of ventilation should be established and a base-line end tidal CO2 achieved pre-transport.

Adequate venous access must be in place. A urinary catheter and a naso/ orogastric tube should be passed. All lines and tubes need to be securely fixed.

All documentation including referral letters should be gathered and the receiving hospital re-contacted prior to departure to confirm availability of the bed and also to confirm their understanding of what they are accepting.

- **Monitoring during transfer** should approach that expected within the hospital setting. Oxygen saturation and ECG and should be monitored continuously *whenever possible*. Invasive BP is advisable as non-invasive measurements are subject to movement artefact. Mechanically ventilated patients need end tidal $CO_2$ monitoring and a disconnection alarm should be used with mechanical ventilators. For long journeys and in cold weather, temperature monitoring should be instituted. The use of a stretcher bridge, with all monitoring self-contained is to be recommended.

**Ideal basic ambulance equipment requirements**

- Protective clothing and footwear
- Hard hats
- Robust gloves
- Safety glasses
- Simple tools and cutting equipment
- Communications
- Lighting and torches
- Restraints for staff and equipment
- Splints
- Oxygen
- Suction unit
- Secure stretcher
- Extrication (spinal) board
- Neck collars
- Defibrillators
- emperature control systems
- Dressings
- Oxygen masks

- **Inter-hospital management**. Despite good preparation interventions may need to be carried out en-route; this may involve stopping the vehicle if transport is by road. A slow smooth journey may be preferable to a fast bumpy one! Once patients are secured on transfer stretchers and monitoring attached it is difficult to gain good access for continued treatment.

- **Aeromedical considerations**. The use of aircraft is not without risk and this is especially true if the attendants are not familiar with the flight environment. Increasing altitude potentates hypoxia and the reduction in alveolar partial pressure of oxygen necessitates supplemental oxygen in all patients. Pressurised commercial aircraft have cabin altitudes of 6000-8000 feet; pneumothoraces will expand by 20% in these conditions, hence chest drainage is mandatory if they are even remotely suspected. The air in endotracheal cuffs will similarly expand at altitude; the risk of tracheal wall pressure leading to possible airway oedema and necrosis. The pressure in air-filled cuffs should be checked regularly. Alternatively saline can be used to inflate cuffs, which will not expand with changes in pressure. Temperature control, especially in helicopters, can be a problem. Most rotary wing and small fixed wing aircraft have excessive levels of noise and vibration. Communication, monitoring, the function of equipment and the administration of fluids can all be affected. The environment is unfamiliar to most. The available space, particularly in helicopters can be limited and they can be cramped and noisy. Ideally, dedicated aircraft should be used with adaptations making them suitable for aero medical use. Both staff and patients can be affected by motion sickness. Staff who recurrently suffer with this problem should not be selected, similarly those who are unable to equalise their middle ear pressure are unsuitable as escorts. Long distance flights from abroad have special considerations and specialist advice should be sought.

**Suggested briefing format for interhospital transfers**

**S** - Situation

**M** - Mission

**E** - Equipment

**A** - Administration

**C** - Communnications

- **Receiving hospital handover**. On arrival the responsible doctor must liase with the medical officer taking over the care. A written summary of events in transfer should be added to the clinical notes and ideally a copy kept for the records at the referring centre. This will enable information to be supplied for local audit and, if necessary, regional or national audit. Without good documentation it is difficult to measure meaningful outcomes and improve care in the future. The Australian Patient Safety Foundation introduced an anonymous self-reporting system for critical incidents during patient retrievals in July 1999. Any team member who felt that there had been a problem endangering patient or staff safety can report this on a standard form. These are collated centrally for the whole country in an attempt to identify recurring problems and improve the service. It would be a positive step if such systems could be adopted routinely everywhere. Whilst this is unrealistic for many, the utilisation of formal debriefing and mission analysis forms will enable problems to be highlighted and lessons learned at a local level.

- **Training**. Staff employed in such transfers should ideally be specifically trained and have had the opportunity to act as observers in previous cases. This is often overlooked as it takes time and often involves more staff than can be spared. Safety aspects of the vehicle employed, including safe approach and escape routes, should be highlighted. All staff should have had basic orientation and safety training whatever the mode of transport employed.

**Intrahospital transport**

It must be remembered that transporting critically ill patients within the hospital is in also potentially dangerous. These patients have reduced physiological reserves and adverse changes can occur during the transport process. Careful planning is required when moving patients between facilities (eg theatres, wards, X-ray etc). Many of the points described above under interhospital transport are applicable. In addition the transport team should be freed from other duties and the departure and destination times be agreed well in advance. All equipment to be used should be checked beforehand and in particular emergency equipment such as resuscitators and suction units. The route used within the hospital should be identified and lifts and corridors secured as necessary before the transfer begins. Any physiological changes occurring during transport should be acted on where appropriate and the patient transferred back to the Intensive Care Unit if necessary. Documentation of the transport process and any adverse events should be made in the clinical record. The overall process in any one hospital should be evaluated regularly, so that recurrent problems can be identified and appropriate changes made.

**Considerations in intra-hospital transport**

- Is it necessary ?
- What is the best route ?
- Who should act as escort ?
- What equipment is required ?
- Do they know we are coming ?
- Do the benefits outweight the risks ?
- When should it happen ?
- What preparations are necessary ?
- Has the equipment been checked ?
- Have we got notes and request forms ?

**Case study: Closed head injury**

A 25 year-old man is involved in a road traffic accident and is brought to the emergency room of a rural hospital. He is semi-conscious with a Glasgow Coma Score of 6. Following an 'ABCDE' assessment in line with Primary trauma care guidelines he is intubated, with appropriate neck control and a hard cervical collar applied. The nearest hospital with imaging facilities and an intensive care unit is 65 miles away. The doctor in charge of care at the rural hospital makes contact with the doctor in charge of the intensive care unit at the regional hospital. It is decided that the patient will be transferred by the rural hospital team in an ambulance sent by the regional hospital. They will send an oxygen supply for the trip. The rural hospital can supply an anaesthetic technical officer and nurse to escort the patient. They use the time whilst waiting for the ambulance to re-examine the patient and make sure all his venous access is secure and he is cardiovascularly stable. They ensure he has been given sedative drugs and muscle relaxants and these are reviewed regularly. The position of the endotracheal tube is checked on chest X-ray and a pelvic and C-spine films are performed. The pelvic and chest x-rays are normal.

His family arrive to find out what has happened to him and the doctor speaks to them about his current condition and need to go to the regional centre. The ambulance arrives and the escorting personnel check that it is suitable before moving the patient. He is attached to a portable blood pressure, ECG and saturation probe. He is hand ventilated with a self-inflating respirator attached. All notes and X-rays are collected to be passed onto the receiving medical team. Ideally, this man would be transported on a mechanical ventilator such as an Oxylog 2000 (Draeger Corporation), with end-tidal CO2 monitoring and with an arterial line in-situ for continuous blood pressure measurement and an anaesthetist to escort. The reality is that many hospitals do not have the resources to provide these for the transported patients. The balance of risks for this patient are that he is better cared for in the regional hospital with its better imaging and intensive care facilities despite the less than ideal transport conditions. On arrival, the anaesthetic technical officer and nurse give a verbal hand-over of the case and pass all notes and x-rays to the medical team.

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# PAIN RELIEF IN LABOUR - REVIEW ARTICLE

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## Introduction

Giving birth is a painful process. This applies to all social and ethnic groups and has probably been so since mankind walked upright. It is very difficult to measure pain which is recognised via the signals carried through the nervous system and the woman's intellectual response to the stimulus.

## Physiology of pain in labour

Labour pain is the result of many complex interactions, physiological and psychological, excitatory as well as inhibitory. Pain during the first stage of labour is due to distention of the lower uterine segment, mechanical dilatation of the cervix and lastly due to stretching of excitatory nociceptive afferents resulting from the contraction of the uterine muscles[1]. The severity of pain parallels with the duration and intensity of contraction[2].

In the second stage additional factors, such as traction and pressure on the parietal peritoneum, uterine ligaments, urethra, bladder, rectum, lumbosacral plexus, fascia and muscles of the pelvic floor increase the intensity of pain.

## Neural pathway of pain

The uterus and cervix are supplied by afferents accompanying sympathetic nerves in the uterine and cervical plexuses, the inferior, middle and superior hypogastric plexuses and the aortic plexus. The small unmyelinated 'C' visceral fibres[3] transmit nociception through lumbar and lower thoracic sympathetic chains to the posterior nerve roots of the 10th, 11th and 12th thoracic and also to 1st lumbar nerves to synapse in the dorsal horn[4]. The chemical mediators involved are bradykinin, leukotrienes, prostaglandins, serotonin, substance P and lactic acid[5]. As the labour progresses severe pain is referred to the dermatomes supplied by T10 and L1.

In the second stage, the direct pressure by the presenting part on the lumbosacral plexus causes neuropathic pain. Stretching of the vagina and perineum results in stimulation of the pudendal nerve (S2,3,4) via fine, myelinated, rapidly transmitting 'A delta' fibres[3]. From these areas, the impulses pass to dorsal horn cells and finally to the brain via the spino-thalamic tract.

## The stress response to pain in labour

Segmental and supra-segmental reflex-responses from the pain of labour may affect respiratory, cardiovascular, gastro-intestinal, urinary and neuro-endocrine functions.

**Respiratory** - Pain in labour initiates hyperventilation leading to maternal hypocarbia, respiratory alkalosis and subsequent compensatory metabolic acidosis. The oxygen dissociation curve is shifted to the left and thus reduces tissue oxygen transfer, which is already compromised by the increased oxygen consumption associated with labour[6].

**Cardiovascular** - Labour results in a progressive increase in maternal cardiac output, primarily due to an increase in stroke volume, and, to a lesser extent, maternal heart rate. The greatest increase in cardiac output occurs immediately after delivery, from the increased venous return associated with relief of venocaval compression and the autotransfusion resulting from uterine involution.

**Hormonal** - Stimulation of pain results in the release of beta-endorphine and ACTH from the anterior pituitary. Associated anxiety also initiates further pituitary response[7].

Pain also stimulates the increased release of both adrenaline and noradrenaline from the adrenal medulla which may lead to a progressive rise in peripheral resistance and cardiac output. Excessive, sympathetic activity may result in incoordinate uterine action, prolonged labour and abnormal fetal heart-rate patterns. Activation of the autonomic nervous system also delays gastric emptying and reduces intestinal peristalsis.

**Metabolic - Maternal**: During labour, glucagon, growth hormone, renin and ADH level increases while insulin and testosterone level decreases[7]. Circulating free fatty acids and lactate also increase with a peak level at the time of delivery. **Fetal** : Maternal catecholamines secreted as a result of labour pain may cause fetal acidosis due to low placental blood flow[8].

## Severity of labour pain

The severity of labour pain varies greatly among women in labour. If women are asked during or shortly after birth to score their labour pain most rate it as severe while few mention little or no pain[9,10]. Using the McGill pain questionnaire, Melzack et al in Montreal, Canada, found that labour pain usually rated a high score particularly among primiparae, those with a history of dysmenorrhoea and those belonging to low socio-economic status[9].

Principles of pain relief

The essentials of obstetric pain relief are:

- Simplicity
- Safety
- Preservation of fetal homeostasis

Women who are given any form of analgesia should be monitored closely. After spinal or epidural anaesthesia they should be monitored with frequent measurements of blood pressure, level of consciousness and maternal oxygen saturation by pulse oximetry.

## Role of the Anaesthesiologist

Anaesthesiologists do have a role antenatally. They should be ready to answer the mothers' questions about the methods of

analgesia. It is important that women with serious underlying chronic disease should be assessed antenatally by the anaesthesiologist to adopt a management plan before the onset of labour. Good communication between obstetrician, physician, haematologist and any other relevant specialist can help the anaesthesiologist in the management of high-risk pregnancy.

**History of pain relief**

Ancient methods of pain relief included various plant-derived sedatives, acupuncture and physical methods such as binding.

- In 1847 James Young Simpson administered the first obstetric general anaesthetic using ether.
- In 1853 John Snow delivered Queen Victoria's eighth child under chloroform.
- In 1881 Stanislav Klikovitch described the use of nitrous oxide for labour in Russia.
- In 1902 morphine and hyoscine was first used in labour. Pethidine was first used in 1940.
- In 1931 Eugen Bogdan Aburel, Romanian obstetrician, described continous caudal plus lumboaortic plexus blocks in labour.
- In 1945 Curtis Mendelson described the syndrome of acid aspiration under general anaesthesia for caesarean section.
- In 1949 Cleland described continuous lumbar epidural block in labour.
- In 1958 Ferdinand Lamaze published his book suggesting that pain was a conditioned reflex triggered by uterine contractions, and that psychoprophylaxis could reduce pain.
- In 1961 Brian Sellick described cricoid pressure as a means of preventing gastric aspiration.

**Psychological methods of pain relief**

Methods of psychological analgesia can be divided into three broad categories :

- Natural child birth - the Read method.
- Psychoprophylaxis - the Lamaze technique.
- Hypnosis

Each technique claims the elimination of pain without any harm to the mother, the baby or to the progress of labour and without the need for chemical analgesia. All require adequate antenatal preparation. Still most women experience severe labour pain[9]. Furthermore, psychological analgesia can place increased demand on the staff.

**Support during labour**

A friendly atmosphere in the labour room is preferable to help a woman to cope with pain. Homely surroundings help to allay anxiety and reduce the need for pharmacological analgesia.

- **Hypnosis**. Hypnosis (hypnos, sleep) can produce analgesia and amnesia during labour and delivery for some selected patients. Only about 25% of women however are suitable as deep trance hypnotic subjects. And the technique relies on extensive preparation.
- **Bio-feedback**. This is borderline between psychological and physical methods of analgesia. Relaxation is a major component of psychological preparation for child-birth and is claimed to relieve pain, reduce anxiety and speed labour.

**Physical methods of pain relief**

- **Transcutenous Electrical Nerve Stimulation (TENS)**. TENS was introduced to relieve pain in childbirth in the early 1980s. Since then the use of TENS in labour has become increasingly popular as it is simple to use and is non-invasive. The mode of action depends on the two principal theories. One that A-fibres are stimulated by the electrical stimulation preventing the transmission of afferent noxious stimulus originating from C-fibres, the other that the electrical stimulus increases endorphines and enkephalins within the system. TENS electrodes are applied over the dermatomes supplied by T10 to L1. The TENS machine then gives a low background stimulus which can be augmented at the time of each contraction. It has been observed in clinical practice that TENS may provide limited pain relief during the first stage of labour. Meta-analysis of randomised controlled trials of TENS in labour does not, however, confirm its efficacy.
- **Acupuncture**. Mentioned in the literature in 581 B. C. and widely practiced in China. Acupuncture is not used for childbirth in China, however, and there are no acupuncture points described for pain relief in labour.
- **Water (bath or shower)**. A bath or shower is relaxing and should be encouraged. There has been enthusiasm in some quarters to extend this to the delivery of the baby under water and many maternity units have the facility to offer water birth. However, while its use during the first stage of labour is not discouraged, very few units would encourage the use of the birthing pool for the delivery of the baby. At present there is little evidence to support the use of immersion in water during labour[11].

**Inhalational analgesia**

Several inhalational agents, both gaseous and volatile, have been used successfully in labour. The earliest to be used were ether, chloroform[12] and cyclopropane,[13] followed by trichloroethylene and methoxyflurane.[14] Enflurane, isoflurane and desflurane[15] are more recent additions.

Analgesia during labour can be provided by the inhalational anaesthetic agents in subanaesthetic concentrations thus relieving pain whilst maintaining maternal consciousness and avoiding regurgitation or aspiration of stomach contents. In fact, the competence of the upper oesophageal sphincter is well maintained under light general anaesthesia, although lost under mild sedation with barbiturate or diazepam[16]. Inhalational agents readily cross the placenta and the concentration in foetal blood soon approaches that of the mother but, since these agents are excreted almost entirely through the lungs, they are readily excreted from the newborn.

The efficacy of inhalational analgesia depends on the analgesic strength of the agent and on how quickly it reaches analgesic concentration after the start of inspiration. A rapid offset with complete elimination between contractions would prevent

accumulation completely. Nitrous oxide is the best match in current use.

Various portable machines exist for administration of nitrous oxide blended with oxygen through an on-demand valve. Nitrous oxide concentrations can be varied from 0 to 75% in oxygen. For self-administration, a concentration above 50% nitrous oxide should not be allowed. Entonox, which is a mixture of 50% nitrous oxide and 50% oxygen is most commonly used.

- **Ether** has several side effects including potent emetic effects with an unpleasant pungent odour, irritant to the respiratory tract and explosive. Chloroform has a pleasant odour, is non-irritant, more potent and faster acting than ether but has undesirable, dose-related side effects, namely arrhythmias and liver damage.

- **Methoxyflurane and trichloroethyline** have been used for analgesia in labour, but have been withdrawn for other, non-obstetric, reasons.

- **Enflurane and isoflurane** have been given via a draw-over vaporiser in subanaesthetic concentrations to relieve pain in labour. The usual concentrations, in oxygen, of enflurane and isoflurane for self administration are 0.3-1% and 0.2-0.7%. Such concentrations will not change uterine contractility or responsiveness to oxytocin. The neonate is not affected by these analgesic concentrations of these inhalational agents. Enflurane, however, causes long-term drowsiness so was never popular. Both the agents are expensive and since neither shows a significant advantage over entonox in terms of analgesia they are unlikely to be widely used on their own.

- **Desflurane** is the newest volatile agent to be applied in labour. The chief advantage of this agent is rapid onset and offset of action, however it is expensive and since it has not been shown to provide superior analgesia to entonox, it is unlikely to become a popular agent for labour analgesia.

**Systemic opioid analgesia**

Opioids have been used for anaesthesia in labour for hundreds of years. However, it was not until the early twentieth century that techniques deliberately employing the analgesic effects of the opioids gained major attention. Unfortunately, dosage and effect are limited by maternal and neonatal side-effects, so that only moderate pain relief could be obtained with these drugs.

- **Pethidine** has become the most commonly used and widely investigated systemic opioid in labour. It is principally a mu-agonist but of a low potency. Administered as hydrochloride in a dose of 75-100mg intramuscularly it reduces labour pain by about 25%. Delayed gastric emptying is a prominent feature. Respiratory depression is not usually observed in women who receive pethidine, because contractions continue to be painful and to provoke hyperventilation. However hypoxic episodes have been observed probably associated with significant underventilation between contractions. The major metabolite, nor-pethidine, is itself active, and has convulsant properties. Thus, pethidine may be inadvisable for use in fulminating preeclampsia or eclampsia, particularly in repeated doses.

- **Morphine** fell from favour in the first half of the twentieth century, in part because of its association with "twilight sleep" and in part because of its addictive side effects.

- **Meptazinol** is a mixed opioid agonist/antagonist, act primarily at the kappa receptor. It is given in a dose of 100-150mg intramuscularly every 2-4 hours. In high doses it has dysphoric side effects and also produce nausea and vomiting. The antagonist properties of meptazinol may cause withdrawal in parturients dependent on mu-agonists. It has a reduced potential to cause respiratory depression.

- **Buprenorphine** is a partial agonist acting selectively at mu-receptors. It is about 20 times as potent as morphine and has a high affinity for opioid receptors and slow dissociation from them. It has a capacity for self-antagonism, which tend to produce a biphasic time course of action. This may be observed for both analgesia and respiratory depression. It appears to have a long duration of action and though side effects are rare, when nausea and respiratory depression do occur they can be exceedingly persistent and difficult to reverse.

- **Nalbuphine** is a synthetic mixed mu-agonist/antagonist and a kappa-agonist. For analgesia in labour it is given in doses of 10-20mg intramuscularly. Maternal or foetal respiratory depression is less likely with nalbuphine due to the ceiling effect. The chief disadvantages of this drug are sedation and dysphoria.

- **Fentanyl** primarily acts on mu-receptors and is approximately 80-100 times as potent as morphine. It has a rapid onset action and shorter duration of action. The peak analgesic effect occurs within 5 minutes and the duration of effect is about 30 minutes after 1 mcg/kg administered intravenously. Fentanyl is principally bound to albumin which favours its transplacental transfer. For analgesia in labour 50-100mcg/hour is required, given in increments of 10mcg IV.

- **Tramadol** is a weak mu-agonist that has been prescribed in labour in doses of 50-100mg 4 hourly. The incidence of nausea is more common with tramadol than with pethidine or morphine[17].

- **Butorphanol** is a synthetic narcotic given as a 1-2 mg dose which lasts 3 to 4 hours. Neonatal respiratory depression is reported to be less than with pethidine[18].

**Patient-controlled analgesia**

Patient-controlled analgesia with intravenous administration of opioid analgesics was assessed for obstetric pain as early as 1970[19]. The patient's ability to control the analgesic administration may produce pharmacological as well as psychological benefits.

**Perineal infiltration, pudendal nerve block and paracervical nerve block**

Perineal infiltration with local anaesthetic solution is of no value for analgesia during labour, but is employed prior to episiotomy just before delivery of the baby.

Pudendal block is a relatively simple, safe and effective method of providing analgesia for spontaneous delivery, normally performed by the obstetrician. Pudendal block may not provide adequate analgesia for forceps delivery or when delivery requires extensive manipulation. 10ml of local anaesthetic solution (lignocaine 10mg/ml) containing adrenaline is injected, after appropriate aspiration.

Paracervical block serves to relieve the pain of uterine contractions, but because the pudendal nerves are not blocked, additional analgesia is required for delivery. Usually lignocaine is injected at 3 and 9 o'clock. Because these anaesthetics are relatively short acting, paracervical block may have to be repeated during labour. This technique has fallen out of favour because of the high incidence of foetal bradycardia and neonatal depression.

**Epidural and subarachnoid administration of local anaesthetics & opioids for labour analgesia**

Epidural anaesthesia is now widely recognized as the most effective form of pain relief technique for labour. Local anaesthetics alone were used for many years, but are now generally administered in lower concentrations in combination with opioids to provide effective, synergistic analgesia whilst reducing some of the unwanted side effects of local anaesthetics, such as motor block.

**Local anaesthetics**.

Bupivacaine has high protein binding and a long duration of effect. It is the most frequently used local anaesthetic for obstetric epidural analgesia. 10 ml of 0.25% bupivacaine (25mg) epidurally will normally provide good analgesia for approximately 90-120 minutes although repeated boluses may produce an increasing motor block. A spinal bolus of 2.5mg bupivacaine (1 ml of 0.25% bupivacaine, often diluted with 1 ml of 0.9% saline) will produce rapid onset of good analgesia for labour but this may only last 30 to 60 minutes.

- **Lignocaine** has a relatively short duration of action due to low lipid-solubility.

- It was suggested that ropivacaine produces less motor block than bupivacaine[20], but this may result simply from its relatively lower potency.

**Opioids**

Good analgesia can be achieved in labour with low doses of a combination of opioid and local anaesthetic. Side effects from neuraxial opioid administration include nausea, pruritus, urinary retention and respiratory depression. The respiratory depression may be delayed, particularly when less lipid soluble opioids are used such as diamorphine. Close observation of women who have received neuraxial opioids is important.

- A spinal dose of 15mcg of **fentanyl** added to local anaesthetic will improve the quality of analgesia. An epidural loading dose of 50mcg fentanyl will similarly enhance the effect of local anaesthetic. Continuous epidural infusion of up to 12ml per hour of 0.1% bupivacaine with 2mcg fentanyl per ml generally provides excellent pain relief in labour.

- Spinal **sufentanil**, in a dose of 10mcg, can be used to provide labour analgesia for over one hour.

- A single spinal dose of 100 to 300mcg of **diamorphine** added to local anaesthetic provides prolonged analgesia for up to 24 hours. 5 mg of diamorphine added to the epidural loading dose reduces the subsequent requirement of bupivacaine infusion[21]. Without this loading dose an infusion of up to 12 ml per hour of 0.1% bupivacaine with 50mcg diamorphine per ml gives excellent pain relief.

- Epidural **pethidine** produces good analgesia due to its partial local anaesthetic effect. 50mg administered 2 to 3 hourly.

**Patient-controlled epidural analgesia and combined spinal-epidural analgesia in labour**

- Patient-controlled epidural analgesia (PCEA)[22] allows immediate access to more epidural solution and creates flexibility, allowing self-titration of solution to acceptable analgesic endpoint throughout labour. As well as the psychological benefits of self-administration, staff workload should be less and reduced drug delivery might minimize side-effects and risks. The disadvantages of the technique include the delayed feedback loop associated with the slow onset of epidural solution and concerns about equipment, safety, monitoring and education.

- Combined spinal-epidural analgesia (CSEA) have evolved in an attempt to optimize the advantages of each separate technique. The advantages of this technique over epidural analgesia alone include more rapid onset of pain relief and good perineal analgesia despite much smaller drug doses. Motor block and its unwanted sequelae are reduced, thus improving maternal satisfaction. The disadvantages are an increased risk of complications due to two procedures as opposed to one.

**Non-narcotic analgesic techniques for labour**

- **Alpha 2-adrenergic agents**. These drugs have been used as it has been recognized that alpha 2-adrenoceptors can be found in the dorsal horn of the spinal cord and that their activation could produce analgesia.

- The **addition of adrenaline** to local anaesthetic solution intensifies and prolongs the neural blockade.

- **Clonidine** is a more selective alpha 2-agonist than adrenaline. It potentiates the action of spinal opioids. Clonidine does not enhance motor blockade. It does not lead to respiratory depression, pruritis or nausea. It can, however, produce hypotension, bradycardia and sedation after its administration epidurally.

- **N-methyl-D-asparate receptor antagonists**. Receptors for NMDA are thought to play a role in various physiological systems among which their role in enhancing pain transmission ('wind-up') is now well appreciated. Because wind-up and hyperalgesia is primarily a spinal cord phenomenon, it appears to be logical to administer NMDA antagonists spinally.

- **Ketamine** has been shown to have analgesic properties in various models. As this drug administered epidurally or intrathecally may not produce respiratory depression, urinary retention or pruritis, its clinical usefulness may be great. Spinal administration of ketamine does not cause motor blockade and arterial blood pressure and heart rate remained unaltered. Drowsiness, dizziness, horizontal nystagmus and dysphoria, however, are the major drawbacks. Recent studies suggest that ketamine may be a useful drug when used in combination.

- **Midazolam** has been demonstrated to have a direct spinal action. Recent studies have focussed on the mechanism of anti-nociception produced by midazolam. The initial step is interaction between midazolam and GABA leading to an increase in chloride flux into the neurone. Through an unknown process, anti-

nociception is then produced by activating a system which involves delta-opioid receptors[23]. A study in obstetric anaesthesia has shown that 1mg of intrathecal midazolam injected with bupivacaine at Caesarean delivery reduces post-operative morphine requirements[24].

**Effects of pain-relief on labour, mother and foetus/neonate**

Pharmacological pain relief in labour is frequently used. Maternal choice of pharmacological methods for analgesia in labour includes not only preferences for the route of drug delivery but also their efficacy and side effects for herself and her baby.

**Effect of epidural analgesia on labour.**

Epidural block induced prior to well-established labour may be followed by desultory labour. The precise role played by epidural analgesia in this phenomenon is not clear, because this sequence of events is also seen in its absence[25]. During the second stage of labour, epidural analgesia that provides effective pain relief, is likely to reduce appreciably maternal expulsive efforts. As a consequence, an epidural block could lead to delay or, less frequently, failure of descent of the presenting part and spontaneous rotation to the occiput anterior position, and hence an increased incidence of operative vaginal delivery as well as Caesarean delivery. However, mothers with high-risk, prolonged or difficult labours are more likely to request epidural analgesia. It is therefore difficult to prove whether the increased rate of interventional delivery seen in mothers with epidurals is the result of causation rather than association.

**Effects on mother**

- **Parenteral Opioids**. The inadequacy of analgesia associated with parenteral opioids is more likely to lead to hyperventilation. This will lead to a lowered maternal $PaCO_2$ that may produce a reduction in utero-placental blood flow. Relative overdose of opioids may lead to hypotension, which is further aggravated by posture and venocaval occlusion. Parenteral opioids cause delayed gastric emptying, which may already have been impeded by labour itself. Furthermore, opioids administered during labour cause nausea and vomiting, from a central action.

- **Paracervical block**. Minor effects such as vertigo, tinnitus and 'aura' have been reported[26]. Transient paraesthesia, numbness and anaesthesia of the leg can occur due to spread of anaesthetic solution to the sacral plexus[27]. Women may be sedated due to partial intravascular injection.

- **Pudendal block**. Unintentional overdosage or intravascular injection can induce dysrrhythmias and cardiovascular collapse. The needle used to place the pudendal block is a source of potential complication as it may unintentionally pierce and damage either the rectum, vagina or foetus. Haematoma in the ischaeorectal and paravaginal spaces have been described following pudendal block. The needle may serve as a vector, introducing bacteria into previously sterile spaces; abscess and periosteal infections have been reported in association with pudendal blocks[27,28].

- **Spinal opioids**. Nausea, vomiting, pruritus and urinary retention can occur with any mu-agonist given by the epidural or subarachnoid route. Respiratory depression (usually delayed) after subarachnoid and epidural opioid administration is a potentially serious complication. Post dural puncture headache severe enough to keep the patient bed-ridden is undesirable as she needs to take care of the neonate and to be mobilized to prevent thrombo-embolic complications.

- **Epidural analgesia with local analgesic**. Epidural analgesia in labour may be associated with maternal pyrexia and shivering not attributed to infection. The rise in temperature may be secondary to both vascular and thermoregulatory modifications induced by epidural analgesia[29].

**Effects on foetus/neonate**

- **Opioids**. The immature respiratory centre is more sensitive to the opioid analgesics. Thus, the analgesics cause respiratory depression after crossing the placenta. Opioid analgesics do not tend to have any primary effect on the cardiovascular system of the neonate but may cause bradycardia secondary to opioid-induced respiratory depression.

- **Paracervical block**.Transient foetal bradycardia is associated with this technique in a significant number of cases due to direct effects of the local anaesthetic on the foetus as a result of vascular constriction or uterine hyperactivity. Other pharmacological effects of local anaesthetics on the foetal heart are lengthening of the atrioventricular and intraventricular conduction times[30].

- **Spinal opioids**. Drugs administered to the women are transported rapidly to the uterus and cross the placenta. All commercially available opioids have low molecular weights and rapidly cross the placenta by diffusion.

  - **Morphine** - risk of neonatal depression with epidural morphine appears to increase with higher doses and shorter interval between dosing and delivery time because of higher maternal blood levels of morphine[31].
  - **Fentanyl** - neonatal depression has only been reported with very high repeated epidural doses[32].
  - **Alfentanil** - has been associated with neonatal depression[33].
  - **Butorphanol** - may be associated with low amplitude, high frequency sinusoidal like foetal heart rate pattern[34].

**Drug interactions**

Information on drug interactions specific to labour and delivery is both limited and complex.

- **Oxytocin**: tetracaine and chloroprocaine antagonize the uterotonic actions of oxytocins.

- **Nifedipine**: hypotensive effects of inhaled anaesthetics will probably be enhanced by this agent secondary to the peripheral vasodilating effect.

- **Epidural hydromorphone** in conjuction with intravenous droperidol cause profound respiratory depression to the parturient.

- **Chloroprocaine** administered into the epidural space decreases the subsequent effectiveness of both fentanyl[35] and morphine[36].

## Conclusion

Antenatal fear of pain in labour is common for many women. The measurement of pain in labour, albeit difficult, and its relief by analgesics or alternative methods, are central to care. Much time, effort and expense is expended in the control of pain. The experience of pain in labour, however, remains an overwhelmingly common experience despite the use of analgesia.

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# CHRONIC RENAL FAILURE AND ANAESTHESIA

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Chronic renal failure (CRF) and end stage renal disease (ESRD) are functional diagnoses characterised by progressive decrease in glomerular filtration rate (GFR). CRF occurs where GFR has been reduced to 10% (20ml/min) of normal function and ESRD when GFR falls below 5% (10ml/min). Patients with ESRD are dependant on renal replacement therapy (RRT) to survive. The relationship between serum creatinine and GFR is not linear (figure 1) and serum creatinine does not rise until GFR has fallen below 50%. In addition, renal tubular secretion of creatinine is increased at higher serum levels.

The incidence of ESRD in the developing world is difficult to estimate and ranges from 40 per million population (pmp) to 340 pmp. The prevalence of ESRD can be more accurately recorded as the number of patients receiving RRT. It ranges from 100 pmp to 600 pmp and can be related to a countries economic wealth. In comparison, the prevalence in the United States of America (USA) is 1191 pmp.

Glomerulonephritis is the main cause of ESRD worldwide (11% – 49%). Proliferative glomerulonephritis is more common in developing countries and may be secondary to endemic infections like streptococcus, schistosomiasis, and malaria. Focal segmental glomerulonephritis is also common in Africa, while IgA nephropathy is common in Asia and Pacific regions. Amyloidosis causes a smaller proportion of glomerular disease and again may be as a result of chronic endemic infections. Interstitial nephritis, secondary to renal stones, obstruction of the urinary tract, tuberculosis and various nephrotoxins, are responsible for up to 20% of ESRD. Diabetes mellitus and hypertension remain important factors in the aetiology of ESRD, but less so in the developing world than in the USA where they account for around 65% of ESRD.

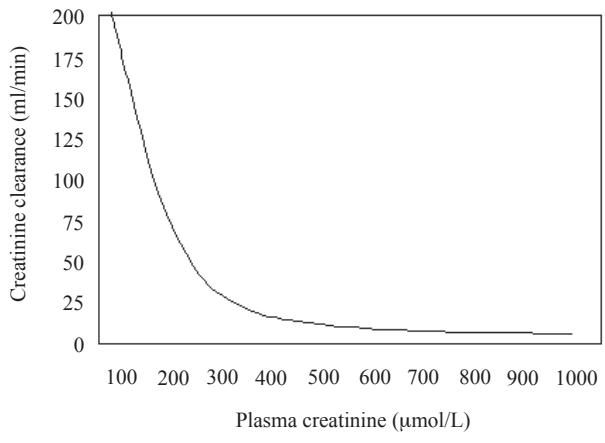


*Figure 1:*

*Table 1 Major identified uraemic toxins*

| | |
|---|---|
| Guanidine | Benzoates |
| Methlguanidine | Creatine |
| Phenols | Creatinine |
| Guanidinosuccinic acid | Tryptophan |
| Tyrosine | Aliphatic amines |
| Myoinositol | Glucuronoconjugates |

Patients with ESRD frequently manifest a wide range of pathological organ dysfunction either caused by the primary disease such as diabetes mellitus, the intrinsic pathological effects of uraemia or a combination of the two. Uraemia refers to the effects resulting from the inability to excrete products of the metabolism of proteins and amino acids. Some of the toxic products of amino acid metabolism are listed in table 1. The multi-organ effects of uraemia are also caused by the impairment of the wide range of metabolic and endocrine functions normally carried out by the kidney. This review will concentrate on the more common pathophysiological changes encountered of relevance to anaesthesia. Despite impressive medical development, the overall 4 year survival for patients with ESRD in the UK is only 48%.

**Fluid and electrolyte derangement**

**Sodium** - In a normal individual more than 25,000 mmol of sodium ions are filtered daily with less than 1% being excreted. CRF can therefore be associated with sodium retention, sodium wasting or normal sodium balance, and is further influenced by factors such as diuretic use and cardiac function. Most patients however demonstrate a mild degree of sodium and water retention whist the extracellular fluid volume remains isotonic. Ironically, the patient with CRF also has impaired renal concentrating mechanisms and thus extrarenal fluid losses such as vomiting, diarrhoea or pyrexia may rapidly cause hypovolaemia and hypotension.

**Potassium** - Adaptive processes increase potassium secretion in the distal nephron (collecting tubules) and also in the gut. Whilst a wide range of plasma potassium concentrations can be encountered, dependent on factors such as diuretic use, it tends to be elevated. Acute changes present the greatest threats to life. A range of drugs may cause acute hyperkalaemia such as beta-blockers, potassium sparing diuretics (spironolactone), angiotensin converting enzyme (ACE) inhibitors or angiotensin antagonists, non-steroidal anti-inflammatory agents and nephrotoxins such as aminoglycosides and cyclosporins. Extracellular acidosis causes an exchange of intracellular potassium for extracellular hydrogen ions in an attempt to

maintain electrical neutrality. In acute acidosis the serum potassium will rise 0.5mmol/l for each 0.1 unit decrease in pH. For this reason hypercarbia should be avoided during general anaesthesia.

The kidney handles magnesium in a similar way to potassium. Reduced excretion may cause hypermagnesaemia, muscle weakness and potentiate non-depolarising muscle relaxants.

**Acidosis** - Chronic metabolic acidosis is a common feature of ESRD. The inability to secrete protons or buffers such as phosphate, or to regenerate bicarbonate limits the clearance of hydrogen ions. Furthermore reduction in glutamine utilisation reduces ammonia production and secretion into the proximal tubule. Retention of organic anions causes a progressive increase in the anion gap and a further fall in plasma bicarbonate concentration. Although plasma bicarbonate concentrations rarely fall below 12-15mmol/l there is little reserve to counter acute acidosis caused by ketoacidosis or sepsis.

**Calcium, phosphate, parathormone and renal osteodystrophy** - Total plasma calcium concentration is reduced in CRF. Renal production of calcitriol ($1,25\text{-}(OH)_2D_3$) declines causing decreased intestinal absorption of calcium. Phosphate excretion is impaired as GFR falls below 20ml/min and hyperphosphataemia develops. As phosphate levels increase, calcium phosphate is deposited in soft tissues such as skin and blood vessels further lowering plasma calcium concentration. Hyperphosphataemia also has a negative effect on 1-$\alpha$-hydroxylase the enzyme responsible for renal calcitriol production. Both hypocalcaemia and hyperphosphataemia are potent stimuli to parathormone secretion, leading to hyperplasia of the parathyroid gland, and secondary hyperparathyroidism. This causes increased osteoclast and osteoblast activity causing osteitis fibrosa cystica. Patients usually tolerate hypocalcaemia remarkably well, as long as oral calcitriol is prescribed and calcium carbonate is used both as an intestinal phosphate binder and a source of calcium. The interrelationship between calcium, phosphate and parathormone in CRF is shown in figure 2.

Careful assessment of the pre-operative fluid and electrolyte status is needed. Dehydration can cause further renal impairment and some patients may benefit from pre-operative intravenous saline while fasting. Patients with oliguric ESRD usually have a fluid restriction imposed. This should be enough to cover insensible losses in addition to their urine volume. When planning fluid requirements the patient's normal daily fluid allowance should be considered and potassium containing fluids should be avoided. Hourly urine volume measurement and central venous pressure monitoring may be necessary pre-operatively. Blood pressure should be kept within the patient's normal range to maintain renal perfusion.

Patients awaiting dialysis may be hypervolaemic, and those recently dialysed may be hypovolaemic. A period of 4-6 hours should ideally elapse before anaesthesia to allow fluid compartment equilibration and the clearance of residual heparin. Vascular instability may complicate induction of anaesthesia. Indications for emergency pre-operative dialysis include:

- Hyperkalaemia ($K^+ > 6.0$ mmol/L)
- Fluid overload and pulmonary oedema
- Metabolic acidosis
- "Uraemic" toxicity and coma

**Haematological abnormalities**

A normochromic normocytic anaemia is a common finding in CRF. Decreased renal erythropoietin production reduces stem cell transformation into erythrocytes, while uraemic toxins reduce red cell life. Chronic upper GI tract losses and those from dialysis further compound the problem. Dietary deficiency in iron and folate also occurs. The introduction in 1989 of synthetic erythropoietin has revolutionised the management of anaemia in these patients, but a compensated relative anaemia is still to be expected. Rapid increases in haemoglobin concentration above 10g/dl often worsen hypertension and may precipitate heart failure. Compensatory mechanisms increase 2,3-

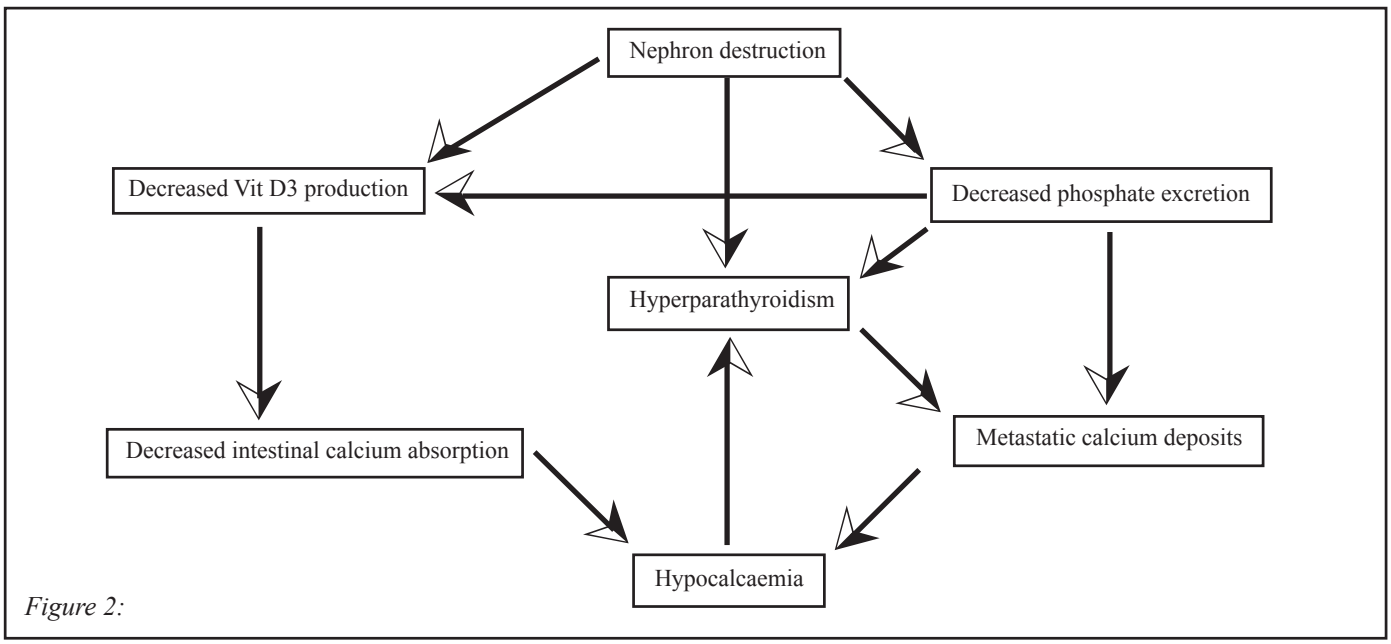


*Figure 2:*

diphosphoglycerate production and move the oxyhaemoglobin dissociation curve to the right thus enhancing oxygen delivery to the tissues.

**Coagulopathy**. Patients with CRF have a tendency to excess bleeding in the perioperative period. Standard tests of coagulation are usually normal (prothrombin time/INR, activated partial thromboplastin time) and platelet count is within normal limits. Platelet activity is however abnormal with decreased adhesiveness and aggregation, probably caused by inadequate vascular endothelial release of a von Willebrand factor/factor VIII complex, which binds to and activates platelets. Increased platelet release of β-thromboglobulin and vascular production of $PGI_2$ also contribute to the coagulopathy. Defects in platelet adhesion may also be related to excessive nitric oxide (NO) production. The plasma from patients with CRF has been shown to be a potent inducer of endothelial NO production.

If it is measured, bleeding time may be prolonged beyond the normal. Platelet dysfunction is not corrected by platelet transfusion, but if operative bleeding occurs, it can be improved by dialysis. Rapid improvements in coagulation require the use of cryoprecipitate or DDAVP (which enhances release of von Willebrand factor). DDAVP (0.3mcg/kg) is effective within 1-2 hours but has a short duration of only 6-8 hours, and is subject to tachyphylaxis. Intravenous conjugated oestrogens have a slower onset but a longer duration of action (5-7 days). The risks of bleeding complications should be considered when deciding to use regional anaesthetic techniques in CRF.

**Cardiovascular and pulmonary abnormalities**

**Cardiovascular abnormalities** are very common in CRF, and represent 48% of the causes of death in these patients. Systemic hypertension is the most common with an incidence approaching 80%, although it is often not a feature of sodium-wasting nephropathies such as polycystic kidney disease or papillary necrosis. Plasma volume expansion resulting from sodium and water retention is the most frequent cause of hypertension, and may be significantly improved by dialysis. Some patients may require beta-blockers, ACE inhibitors, alpha-blockers and vasodilators to adequately control their blood pressure. Alteration in the control of renin and angiotensin secretion may also contribute to hypertension in 30% of patients.

Ischaemic heart disease (IHD) is a frequent cause of mortality in patients with CRF. The incidence varies with patient subgroup, for example it is present in 85% of diabetics older than 45 years with CRF. Accelerated atherosclerosis results from decreased plasma triglyceride clearance, hypertension and fluid overload causing left ventricular hypertrophy and failure. The elevation in plasma triglyceride levels is caused by a defect in lipoprotein lipase activity and reduced lipolysis.

The incidence of metastatic calcific valvular heart lesions is increased. Aortic valve calcification occurs in up to 55% of patients, with aortic stenosis being present in 13%. Mitral valve calcification occurs in 40% (stenosis 11%). Elevation in the calcium/phosphate product and parathyroid hormone concentrations are the main cause. As a result of these lesions, bacterial endocarditis is much more common in dialysis patients than the normal population. Haemorrhagic uraemic pericarditis was often seen prior to the advent of effective dialysis, but is now uncommon and occurs in patients on inadequate dialysis regimen. If untreated, it may progress to pericardial tamponade with hypotension, elevated jugular venous pressure and signs of falling cardiac output. Pericardectomy may be required but should be reserved for those who fail to improve with immediate dialysis. Sudden death from acute cardiac arrhythmias is frequent and related to both IHD and electrolyte abnormalities.

**Pulmonary** complications are common in patients with CRF in the postoperative period. Fluid overload, malnutrition, anaemia, impaired humoral and cellular immune function and decreased surfactant production predispose patients to atelectasis and infection.

**Immune function**

Sepsis is a leading cause of death in patients with CRF. Inhibition of cell mediated immunity and humoral defence mechanisms occurs, with little improvement following the instigation of dialysis. There is an increased production of pro-inflammatory cytokines suggesting that activation of monocytes may play a role in uraemic immune dysfunction. Superficial infections are common in fistula and catheter sites, and wound healing is poor.

The incidence of viral hepatitis B has decreased somewhat following the introduction of erythropoietin and hepatitis B vaccination. There is also an increased incidence of hepatitis C infection in patients on haemodialysis and although there is often little effect on liver function it is of concern in patients undergoing renal transplantation and immunosuppression. Hospital staff must take precautions against blood borne viruses in these patients.

**Gastrointestinal abnormalities**

Gastrointestinal abnormalities are frequent with anorexia, nausea and vomiting contributing to malnutrition. Urea is a mucosal irritant and bleeding may occur from any part of the GI tract. Gastric emptying is delayed, residual volume increased and pH lowered. Peptic ulcer disease is common and most patients will receive proton pump inhibitors. The use of a rapid sequence induction technique needs be balanced against the risks of difficult intubation in chronically ill patients with poor dentition. Suxamethonium will increase the plasma potassium concentration by approximately 0.5mmol/L and this is not reliably prevented by precurarisation with a non-depolarising agent. Patients with diabetes mellitus have an increased incidence of difficult intubation and autonomic gastric paresis even in the absence of CRF. In practice, rapid sequence induction is restricted to patients who are inadequately fasted or have symptoms of gastric reflux and a low serum potassium.

**Neurological abnormalities**

Many patients with CRF have abnormalities in central (CNS) and peripheral nervous system function. CNS changes have a wide spectrum from mild personality alterations to asterixis, myoclonus, encephalopathy and convulsions. Peripheral neuropathy is common in advanced stages of the disease and is initially a distal "glove and stocking" sensory loss progressing later to motor changes. Both dialysis and renal transplantation may improve the neuropathy. The presence of a peripheral neuropathy should alert the anaesthetist to the presence of an

autonomic neuropathy with delayed gastric emptying, postural hypotension and silent myocardial ischaemia. Two types of neurological disturbances are unique to patients on dialysis. Dialysis dementia with dyspraxia, myoclonus and dementia occurs in patients on dialysis for many years and may be related to aluminium toxicity. The dialysis disequilibrium syndrome is associated with rapid initial reduction in plasma urea levels at the commencement of dialysis.

**Endocrine disturbances**

Changes in parathyroid function and lipid clearance have been noted above. Glucose tolerance is impaired, but there is a reduced requirement for exogenous insulin in diabetics, probably related to the reduced metabolism of insulin by the failing kidney. Patients with CRF have abnormalities of temperature regulation with reduced basal metabolic rate and a tendency to hypothermia. This may be important when assessing fever.

**Altered drug handling in CRF**

A wide range of pharmacokinetic changes occurs in drug handling in patients with CRF. The volume of distribution is usually decreased, but may be increased if there is fluid retention. Hypoalbuminaemia and acidosis increase the free drug availability of highly protein bound drugs. These changes may require an alteration in the loading dose of a drug. The doses of benzodiazepines and thiopentone may need to be reduced by 30% - 50%. Although the pharmacodynamics of propofol are unchanged in CRF and the metabolites lack sedative activity, changes in volume of distribution and mental state mean that a reduction in induction dose may also be appropriate. The elimination of highly ionised, water soluble drugs such as gallamine or atropine are partially or completely dependent on renal excretion and may be markedly reduced. However the duration of action of a single loading dose will be dependent on redistribution rather than excretion. Dialysis can only partially compensate for the loss of excretory ability of the kidney.

Most lipid soluble analgesics are metabolised by the liver to water soluble metabolites for renal excretion. Some of these metabolites may have far greater activity than the parent drug. Morphine is metabolised to morphine-6-glucuronide, a more potent analgesic and respiratory depressant. The interval between doses will need to be increased because of its reduced renal clearance. Metabolism of pethidine produces norpethidine, which can cause seizures. Although fentanyl undergoes hepatic metabolism and is not thought to have active metabolites, its clearance is decreased in severe uraemia. Alfentanil can be used as normal.

The elimination of volatile anaesthetic agents is not dependent on renal function and their activity is unaffected by CRF. The hepatic metabolism of both enflurane and sevoflurane will theoretically produce nephrotoxic fluoride ions and their use should be discouraged for prolonged durations. Metabolism of halothane produces fluoride ions when the liver is hypoxic but has been used safely in patients with renal disease. It has a greater myocardial depressant effect and causes more arrhythmias than other inhalational agents and caution should be observed when used in CRF patients with cardiovascular impairment. Isoflurane, although more expensive, may be the agent of choice as it undergoes less metabolism to fluoride ions. Nitrous oxide has little effect on the kidney. Older agents such as cyclopropane, ether and tricloroethylene are not recommended as they cause renal vasoconstriction.

Atracurium and cisatracurium are obvious choices for muscle relaxation. Around 90% is metabolised by ester hydrolysis and Hofmann elimination. Plasma cholinesterase activity is not thought to be affected by CRF and therefore mivacurium and suxamethonium (in the absence of hyperkalaemia) may be used. Limited doses of vecuronium and rocuronium are acceptable alternatives. Acidosis prolongs the action of all muscle relaxants except gallamine. The excretion of anticholinesterases and anticholinergic agents will be prolonged as they are highly ionised and water soluble.

Local anaesthetics are valuable agents for peri-operative pain control in CRF, but their duration of action is reduced secondary to acidosis. Maximum doses of local anaesthetics should also be reduced by 25% because of reduced protein binding and a lower CNS seizure threshold.

Non-steroidal anti-inflammatory drugs (NSAIDs) should be avoided in patients with CRF. NSAIDs inhibit the production of renal prostaglandins $PGE_2$ and $PGI_2$, which are responsible for maintaining renal blood flow during hypovolaemia and in the presence of vasoconstrictors, and could precipitate acute renal failure.

**Conclusions**

The incidence of chronic renal failure is increasing throughout the world. The effects of CRF are multiple and widespread beyond the confines of the kidney. The function of many organ systems of great interest to anaesthetists are adversely affected by a range of accumulated toxins. Great improvements in nephrology and transplantation mean that many more patients with CRF are living much longer. Their appearance on both renal and unrelated operating lists will continue to increase.

**Further reading**

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# THE EMERGENCY MANAGEMENT OF POISONING

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## Introduction

Drug overdose and poisoning is a common reason for presentation to emergency departments, accounting for 3-5% of attendances and causes over 2000 deaths a year in the UK. Drug overdose is the most frequent presentation of deliberate self-harm and this may complicate management. However most patients are young, otherwise medically well and managed appropriately the vast majority should recover fully.

This article aims to discuss the general principles behind the management of poisoning and then to review the specific treatment of the more common overdoses.

## General Principles

Many drugs in overdose (e.g. opiates, tricyclics, benzodiazepines) can cause significant depression of cerebral and cardio-respiratory function and emergency management should always start with a rapid **initial assessment** and **resuscitation** of the airway, breathing and circulation. Then, careful **history** and **examination** will in most cases give an indication as to the likely severity of the overdose and guide subsequent management. Treatment principles include strategies to reduce absorption, increase elimination, general supportive measures and where available, the use of specific antidotes to reduce toxicity. It is strongly recommended that, in cases where doubt exists regarding the degree of risk, or appropriate management, the Poisons Information Service be contacted. In the UK, they can be reached by dialing 0870 600 6266, 24 hours a day, or information may be obtained via their website www.spib.axl.co.uk

**History.** A detailed and reliable account of the drug or drugs taken should be sought; This should include the drug name, amount, preparation type, time of ingestion and the co-ingestion use of other substances such as alcohol or recreational drugs which might influence the patients clinical state or drug clearance. The presence of vomiting of tablets soon after overdose should be noted but does not preclude significant toxicity.

The medical, social, psychiatric and therapeutic drug history will help to identify high-risk patients and guide subsequent management.

The patient may well be uncooperative or unable to give these details and so a collateral and confirmatory history should be acquired from available sources (e.g. drug packets, ambulance crew, witnesses, suicide note, patient records, etc).

**Examination.** The **airway, breathing and circulation** should be reassessed and treated accordingly: Basic airway manoeuvers, simple adjuncts and/or cuffed **endotracheal intubation** may be required if the airway is compromised. The patient's level of consciousness may give an indication as to the toxicity of the overdose, risk to airway and guides the level of supportive care likely to be needed. It can be expressed on the 'AVPU' scale or as a formal GCS (although not designed for this purpose, it does give a reproducible score which is sensitive to subsequent changes.) A GCS equal or less than 8 (or responding to Pain only) increases the risk of airway compromise and endotracheal intubation is indicated unless rapid recovery is anticipated.

Careful attention should be paid to **respiratory function** particularly with sedative drug toxicity. This should include respiratory rate and tidal volume and the measurement of oxygen saturations using pulse oximetry. A low respiratory rate with decreased oxygen saturations may indicate hypoventilation but note that a normal saturation does not exclude hypercarbia or indeed hypoxia in carbon monoxide poisoning. If in any doubt, arterial blood gases should be measured. Tachypnoea can be seen with metabolic acidosis (tricyclics, methanol), anxiety, and stimulant drug overdose and as an early feature of salicylate poisoning (respiratory alkalosis). Supplementary oxygen via facemask should be given to all patients initially.

Many drugs exhibit **cardiovascular toxicity** in overdose (e.g. tricyclics, b-blockers, digoxin, lithium). This may manifest as hypotension and or cardiac arrhythmias. Pulse, blood pressure and ECG should be recorded, intravenous access established and initial fluid resuscitation given.

General examination may give corroborating evidence of significant ingestions or clues in unknown overdoses. Many drugs (SSRIs, tricyclics, phenothiazines) have serotonergic or anticholinergic effects with pupil dilatation, extra pyramidal movements, whilst opioid type drugs will cause sedation and pin point pupils.

## Further Management

Temperature, blood glucose (low in b-blocker, ethanol poisoning) and weight should also be recorded. Weight is important in calculating whether the patient is likely to have received a toxic dose and guides treatment e.g. in paracetamol overdose.

The examination should reveal any associated injury (accidental or deliberate self harm) or the presence of other substances, e.g. alcohol. Make an appropriate psychiatric examination of the patients mental state.

**Investigations** should be undertaken and may include appropriately timed drug levels when these will aid management e.g. paracetamol or lithium. In all cases of suspected overdose, it is recommended that a paracetamol and salicylate level be taken in addition to baseline biochemistry and haematology, as both of these poisons are associated with a lack of early clinical signs, and have specific therapeutic measures available to treat.

## Treatment

Supportive treatment of the cardio-respiratory and neurological systems should be given by standard Intensive Care methods.

Fitting should be controlled initially with intravenous diazepam (0.1- 0.3 mg/kg) or lorazepam (4mg in an adult, 0.05mg/kg in a child).

Gastric elimination, either by lavage or forced emesis may be useful, especially if the poison has been ingested less than 4 hours previously. In salicylate and tricyclic poisoning, forced gastric emptying may be useful up to 12 hours post ingestion. In the patient with disturbed level of consciousness, aspiration is a serious risk, and therefore in these patients, endotracheal intubation should be considered before emptying is attempted.

Drug absorption can be reduced by the use of activated charcoal, given either orally or nasogastrically. It is particularly useful for poisoning with benzodiazepines, tricyclics, anticonvulsants and antihistamines.

Drug elimination may be hastened by forced diuresis increasing renal clearance (if renal function is normal) or, in some cases, by consideration of dialysis.

**SPECIFIC POISONS**

**Alcohol** (Blood concentration of >4.5g/L (98mmol/L) is potentially fatal).

**Clinical features** – with increasing blood concentrations, features are progressive from ataxia, dysarthria, and nystagmus, to hypothermia, hypotension, stupor and coma. In severe cases, especially children, convulsions, respiratory depression and acidosis may occur.

**Hazards** – aspiration of vomit, hypoglycaemia (especially in children).

**Treatment** – General supportive

- Alcohol is rapidly absorbed from the gut, and therefore gut decontamination is unlikely to be of benefit.
- Hypoglycaemia should be treated as quickly as possible with oral glucose if the patient is awake, or otherwise intravenous 5% or 10% dextrose.
- If facilities allow, haemodialysis should be considered if blood concentration is greater than 5g/L, or arterial pH is <7.0.

**Paracetamol** (Ingestion of as little as 150mg/kg is potentially fatal).

**Clinical features** – often none. Occasionally nausea and vomiting, which is suggestive of liver damage if it persists beyond 24 hours and associated with right sub-costal tenderness.

**Specific Hazards** – Hepatocellular necrosis and liver failure. Liver damage is maximal 3-4 days after ingestion, and may be associated with hypoglycaemia, haemorrhage, encephalopathy, and death.

**Treatment Guidelines** - (knowledge of time of ingestion is vital).

Treatment with N-acetylcysteine must be started within 8 hours if maximal benefit is to be gained. Patients who regularly consume excessive alcohol, who are on long term carbamezepine, phenytoin, phenobarbitone, rifampicin or St. John's Wort, or who are likely to be glutathione deplete (e.g. HIV infection, eating disorders) are at higher risk of toxicity, and treatment should be started if >75mg/kg have been ingested.

**Presentation within 4 hours**

- Consider activated charcoal if more than 150mg/kg has been taken within the previous hour.
- 4 hours after ingestion, take a venous blood sample for plasma paracetamol level, as well as baseline biochemistry and haematology (including INR).
- If the patient is at risk according to the nomogram, start N-acetylcysteine at a rate of 150mg/kg over 24 hours.
- If the patient is not at risk, no treatment is necessary. If treatment was started within 8 hours of ingestion, the risk of liver or renal damage is minimal.

**Presentation within 4-8 hours**

- Gut decontamination is unlikely to be helpful.
- Take blood for plasma paracetamol level, and baseline investigation (as above). Treat with N-acetylcysteine according to the nomogram, and treat in all cases where ingestion has been >150mg/kg (75mg/kg in the at risk group). If the plasma paracetamol result is likely to be delayed, initiate treatment with N-acetylcysteine at 150mg/kg over 24 hours, and stop if necessary once the results become available.
- The patient should have their liver function tests (LFTs), INR, creatinine and bicarbonate checked 12 hourly until they return to normal. If they are abnormal, N-acetylcysteine should continue to be given at the above rate until they improve, or if they worsen, specialist advice should be sought.

**Presentation after 8 hours**

- Give N-acetylcysteine immediately unless certain that the ingestion has been less than 150mg/kg (75mg/kg in the high risk group). DO NOT wait for the result of the plasma paracetamol level. Patients presenting at this time are at high risk from liver damage, and whilst there is less evidence to support it, it is thought that the administration on N-acetylcysteine may still be beneficial.
- Take blood urgently for paracetamol level, ALT, creatinine, and INR.
- If the risk of liver damage is confirmed, continue administration of N-acetylcysteine, and keep the patient under observation for 3-4 days monitoring biochemistry and INR. Once they return to normal, the infusion may be discontinued. Liver and renal failure, if present, should be managed conventionally, with specialist advice being sought as necessary.
- Metabolic acidosis is a poor prognostic sign.

**Presentation after 24 hours**

- Measure plasma creatinine, ALT and INR, and venous acid/base balance/bicarbonate.
- If any of these are abnormal, consider treatment with N-acetylcysteine, and seek expert advice.
- For staggered overdoses, the risk of serious damage is minimal if <150mg/kg has been ingested in 24 hours. For all patients who have consumed more than this, consider treatment with N-acetylcysteine.

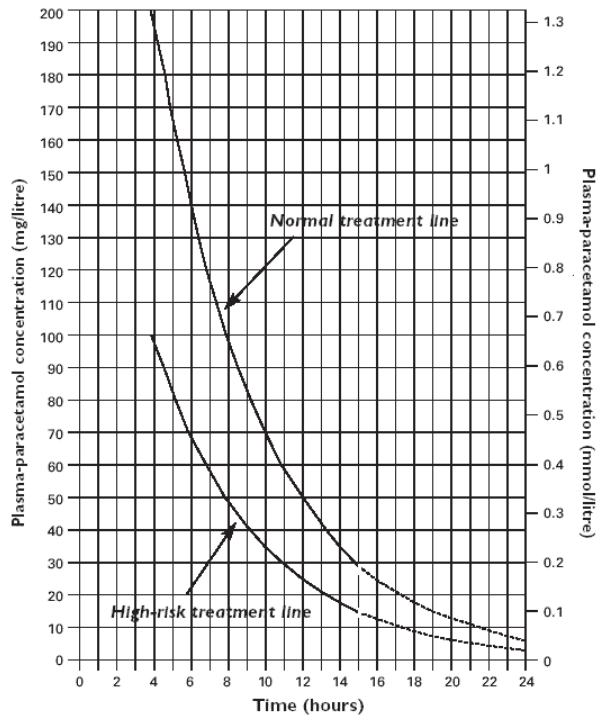


*Figure 1*

**Tricyclic antidepressants (TCAs)**

**Clinical features**. Toxicity is due to anticholinergic effects at autonomic nerve endings, and a quinidine-like effect on the myocardium. Peripheral signs include tachycardia, dry skin, dry mouth and dilated pupils. Central signs include ataxia, nystagmus, fitting, drowsiness and coma. There may also be increased tone and hyperreflexia. ECG features include lengthening of the PR and QRS intervals. Rarely, skin blisters are seen, which should be treated as burns.

**Specific Hazards.** Convulsions, coma and metabolic acidosis which exacerbates myocardial problems.

**Treatment guidelines**

- Activated charcoal (50g) by mouth or nasogastric tube is indicated if the patient presents within 3-4 hours of ingestion.
- Patients who are asymptomatic with normal ECGs at 6 hours are unlikely to develop late problems.
- Arrhythmias should be treated in the first instance by correction of hypoxia and acid/base disturbance.
- Sodium bicarbonate alters the binding of TCAs to the myocardium, and therefore (50mmol) should be given intravenously to an adult with ECG changes or arrhythmias, even in the absence of acidosis.
- Convulsions should be treated with diazepam or lorazepam, NOT phenytoin, as the latter, in common with TCAs, block sodium channels, and hence potentiates cardio-toxicity.
- Consider Glucagon 1mg IV every 3 minutes to treat refractory hypotension and myocardial depression.
- Prolonged resuscitation may be successful after cardiac arrest.

**Salicylates** - (Ingested dose of 500mg/kg is potentially fatal)

**Clinical Features** – vomiting, dehydration, tinnitus, sweating, vasodilation, hyperventilation. Less commonly haematemesis, renal failure, hyperpyrexia. Presence of CNS signs, e.g. confusion, coma, convulsions are commoner in children, but are an indicator of severe poisoning in all.

**Specific Hazards** - in adults, a mixed respiratory alkalosis and metabolic acidosis is usual. In children less than 4 years, a metabolic acidosis is seen, which increases salicylate transfer across the blood-brain barrier.

**Assessment of severity of poisoning** - Plasma concentrations of >350mg/L indicate salicylate intoxication. Most deaths in adults are associated with a level of >700mg/L. Risk factors for death include age (<10 years and >70 years), acidosis, CNS features, late presentation, and pulmonary oedema.

**Treatment guidelines**

- Give activated charcoal if >250mg/kg has been ingested within 1 hour.
- If >120mg/kg has been ingested, do a plasma salicylate level at least 2-4 hours after ingestion. A repeat sample (2 hours later) may be needed in patients with suspected severe poisoning, as there may be continued absorption.
- Arterial blood gas analysis is helpful. If a metabolic acidosis is present, and the serum potassium is normal, give intravenous sodium bicarbonate, as below, to cause an alkaline diuresis. If the potassium is low, correct this **before** giving the bicarbonate.

  - Salicylate concentration in adults >500mg/L (3.6mmol/L) - give 1.5L of 1.26% sodium bicarbonate (or 225mL 8.4%) over 2 hours

- Salicylate concentration in children (<5years) >350mg/L (2.5mmol/L) – give 1mL/kg 8.4% bicarbonate diluted in 0.5L 5% dextrose at 2-3 mL/kg/hr.
- Aim to achieve a urinary pH of 7.5-8.5, repeating treatment if necessary to achieve a falling plasma salicylate level.
- The previously used forced alkaline diuresis should not be used as it carries a significant risk of pulmonary oedema.
- In severe poisoning with evidence of cardiac or renal failure, haemodialysis is the treatment of choice.

**Ethylene glycol** (antifreeze, coolant, brake fluid)

**Fatal dose** is 100mL for a 70kg adult - inhalation and skin absorption are not serious.

**Clinical features.** Ethylene glycol is rapidly absorbed from the gut, and toxic features such as dysarthria, ataxia, nausea and vomiting start within 30 minutes of ingestion. Fits, coma and metabolic acidosis follow. Between 12 and 24hr after ingestion, cardiac failure, hypertension and respiratory distress occur, with progression to renal failure and hypocalcaemia.

**Treatment guidelines**

- Consider gastric lavage if the patient presents early. Forced vomiting is contraindicated.

- Measure acid/base status, and calcium. Correct metabolic acidosis with IV sodium bicarbonate. Large amounts may be needed, hence watch for hypernatraemia. Correct hypocalcaemia with IV calcium gluconate.

- Ethanol (alcohol) is the most widely used antidote, as it competes with ethylene glycol for alcohol dehydrogenase, which is responsible for the conversion of the ethylene glycol to its toxic metabolites. If the patient is conscious, give 2mL/kg of 40% alcohol (gin, vodka or whisky) orally.

- If the patient is unconscious, or acidotic, give an IV loading dose of 10% ethanol (7.5mL/kg in water), followed by an infusion as below.
  - Non-drinker/child – 66mg/kg/hour of ethanol (i.e. 1.65mL/kg/hr of 5% ethanol)
  - Average adult – 110mg/kg/hr (i.e. 2.76mL/kg/hr of 5% ethanol)
  - Chronic drinker - 150mg/kg/hr (i.e. 3.9mL/kg/hr of 5% ethanol)

- In severe poisoning with evidence of cardiac or renal failure, haemodialysis is the treatment of choice.

**Carbon Monoxide (CO)**

**Clinical features** are related in the main to tissue hypoxia as a result of impaired oxygen carrying capacity of haemoglobin. Therefore headache, nausea, irritability, agitation and tachypnoea, progressing to impaired consciousness and respiratory failure. A metabolic acidosis and cerebral oedema may develop in severe cases, and progression to multi-organ failure may ensue. Late complications, occurring weeks later in survivors of the acute exposure, may include psychiatric and Parkinson-like movement disorders. Pulse oximetry is unreliable in carbon monoxide poisoning, as it overestimates oxygen saturation.

Chronic carbon monoxide poisoning is less easy to diagnose, and usually occurs in more than one member of a household, associated with the use of gas heaters in under ventilated areas. The main symptoms are headache and flu-like symptoms.

**Treatment guidelines**

- Remove from exposure

- Give high dose oxygen in an attempt to displace CO from the haemoglobin, and hence improve oxygen delivery to the tissues

- Metabolic acidosis should be treated by improving oxygen delivery to the tissues. Sodium bicarbonate hinders this, and therefore should not be used.

- Consider mannitol 1g/kg if cerebral oedema develops.

- Measurement of carboxyhaemoglobin levels may give an indicator of severity of exposure (>20% indicates significant exposure), but correlation with clinical outcome is poor.

- In patients with coma, be alert to the possibility of longer term neurological damage.

- Hyperbaric oxygen is used in some specialist centres.

**Organophosphates –** see Update in Anaesthesia 19 (in press)

Supportive measures are vitally important. Avoid self contamination – wear protective clothing.

**Clinical features**. Organophosphates are readily absorbed through skin and the lungs. They bind irreversibly to acetylcholinesterase, hence lengthening the half-life of acetylcholine. Recovery only occurs with the synthesis of new acetylcholinesterase. Nicotinic effects (such as muscle weakness), and muscarinic effects (sweating, bronchospasm), predominate.

**Treatment guidelines**

- Prevent further absorption by removing source, including soiled clothing

- Give high dose oxygen

- Consider gastric lavage

- Give atropine (2mg for adults, 0.02mg/kg for children) IV every 10-30 minutes until there is improvement.

- IV diazepam (5-10mg for an adult, 0.02mg/kg for a child) to control twitching

- In severe poisoning, consider use of pralidoxime (a cholinesterase reactivator) in the first 24 hours.

- Intubation and ventilation is frequently required.

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# THORACIC ANAESTHESIA UPDATE

Dr Adrian Pearce, Guy's and St Thomas' Hospital, UK

Thoracic surgery is undertaken in only 30-40 units in the UK. National data is collected on number of cases and mortality. Core operations are lobar resection, pneumonectomy for malignant and non-malignant conditions, mediastinoscopy and mediastinotomy, bronchoscopy for diagnostic and interventional reasons, video-assisted thoracoscopic surgery (VATS) for drainage and investigation of effusions, management of air-leaks, management of empyema and operations on the chest wall. Some surgeons undertake both cardiac and thoracic surgery wilst others are dedicated thoracic surgeons. Lung volume reduction surgery and lung transplantation are specialised procedures usually rationalised to specific units or surgeons. Oesophagectomy is now undertaken more commonly by 'general' surgeons. Standard anaesthetic textbooks commonly contain a thoracic chapter.

## Bronchoscopy

Rigid bronchoscopy may be for diagnostic or interventional procedures. The latter include stenting, lasering and removal of foreign bodies. Propofol by manual or target controlled infusion has revolutionised the provision of anaesthesia particularly for longer procedures. Cardiovascular responses are easily controlled by alfentanil or remifentanil which both permit rapid awakening. Ventilation by the Sanders technique is still commonly used.

## Double lumen tube (DLT) sizes

Suitable PVC tubes are 35, 37, 39, 41 Fr. One suggestion is (for left DLT) to measure the tracheal width on the preop CXR since this predicts the diameter of the left bronchus: > 18 mm 41 Fr, > 16 mm 39 Fr, > 15 mm, 37 Fr and > 12 mm 35 Fr. This is specifically for Mallinckrodt tubes and one study which tested this predictive sizing didn't prove (in my view) it to be very useful.

## Left or right DLT

A lively debate continues [1, 2]. A right tube may be more expensive, is harder to place so that the right upper lobe is patent, requires fibrescopic checking and may not be possible to place when airway anatomy is abnormal. Early work detailing problems with right tubes has now been followed by studies suggesting that a right DLT placed and checked by fibre-endoscopy has no increased risk of complications. The indications for a right-sided tube are:

- Tumour in left bronchus
- Left pneumonectomy
- Left lung transplantation
- Left tracheobronchial disruption
- Left bronchial stent in situ
- Distorted left bronchial anatomy

## Airway trauma due to DLT

A recent review article [3] collected data from the world literature. Between 1972 and 1998, only 33 reports were identified. Reports involved 32 patients with red rubber DLTs and 14 patients with PVC DLTs. This may well just reflect the differing usage of these types of tubes. Authors were contacted to try and ascribe a cause for the airway trauma. A carinal hook, tube-tip irregularities, asymmetric cuff inflation, initial overinflation of the cuff and nitrous oxide induced cuff overinflation were the most common lessons. The bronchus was the most frequent site of injury, followed by tracheal damage followed by tracheo-bronchial tears. Almost half the published reports with PVC DLTs involved Japanese patients. Good practice recommendations to avoid airway trauma are:

- Choose the largest PVC DLT that will fit
- Remove the bronchial 'stylet' once the tip of the tube is past the vocal cords
- Advance the DLT the appropriate distance (based on height)
- Inflate both cuffs slowly and carefully
- Use a 3ml syringe to inflate bronchial cuff
- If nitrous oxide used – fill cuffs with saline or $N_2O/O_2$ mix
- If nitrous oxide used check cuff pressures intermittently
- Keep intracuff pressures < 30cm $H_2O$
- Deflate both cuffs before moving or repositioning tube
- Deflate bronchial cuff when not needed

## Checking DLT position

It is firmly established that the positioning of a DLT should be by visualisation of chest movement, auscultation, fibre-endoscopy[4] and a suitable pressure/volume/flow profile. A standard intubating fibrescope with nominal diameter 4mm will not pass down a 35 Fr and passes with difficulty down a 37 Fr DLT. Generally these smaller tubes are used in females: This gender discrimination should be overcome by purchasing a fibrescope designed for checking DLT e.g the Olympus LF-DP with a diameter 3.1mm. The sequence of checking is to pass the fibrescope down the tracheal limb to check that the correct bronchus has been intubated and to make certain that it is the correct depth. The bronchial limb of a right tube should always be checked to make certain that the slit in the bronchial cuff is opposite the right upper lobe bronchus. The right upper lobe has no constant anatomy and one should at least consider the desirability of inspecting the airway fibreoptically to make certain that the anatomy will suit a DLT. This can be done by passing the DLT through the vocal cords but not advancing beyond mid-trachea. The fibrescope is placed through the bronchial limb and

is advanced to inspect the anatomy and to guide the tube into position.

**Bronchial blockers**

Bronchial blockers are making a comeback particulary in the US (see Univent Tube on www.vitaidltd.com/Univent.htm or Arndt Endobronchial blocker set on www.cookgroup.com/cook_critical_care/blocker.html). A blocker is effectively a balloon on a suction catheter and is placed under direct or fibreoptic vision into the bronchus. The trachea is intubated with a normal single lumen tube and the blocker may pass within or outside the tracheal tube. When the blocker is inflated, that lung will not be ventilated. The 'suction catheter' element passing to the tip allows the isolated lung to deflate, or suctioning. A blocker might therefore be used instead of a DLT in a normal patient, be used when it would be difficult to insert a DLT (difficult intubation or paediatric) or when the airway anatomy is not suitable for a DLT. A blocker may also allow continued inflation of one lobe of the operated lung, for example if placed in the right bronchus intermedius, will allow right upper lobe ventilation when the operation is on the lower lobe.

An editorial[5] this year describes the characteristics of the ideal blocker;

- A balloon shape to stabilise it in the bronchus with low pressure/high volume inflation characteristics.
- Be flexible and easy to manipulate into mainstem or lobar bronchus.
- Have a channel for deflation and suction distal to balloon.
- Be adaptable for use internal or external to standard tracheal tube.
- Have a wide variety of sizes for adult and paediatric use.

Tips in the same editorial for insertion of a blocker are:

- Position deflated blocker after intubation when supine
- Particularly useful in left bronchus for left lung deflation
- Push blocker as far distally as possible to avoid migration out of bronchus
- Use preferentially for –'otomies' rather than –'oscopies' because lung deflation is slower
- Use a video camera on the positioning fibrescope to allow an assistant to help. It is a 4 handed task.

One study[6] compared left DLT with left and right bronchial blockers. Left blockers took longer to place but both left DLT and left blocker were satisfactory. Satisfactory lung deflation was obtained in only half of the right blocker cases.

**Physiology of one lung ventilation (OLV)**

Thoracic surgery is undertaken in the lateral position and the terms non-dependent and dependent lung indicate the operative collapsed lung, and continuously ventilated normal lung respectively. Clinically it is possible to identify four different periods of ventilation - TLV supine; TLV lateral, chest closed; TLV lateral, chest open and OLV. Classic teaching is that in TLV-lateral the dependent lung receives about 60% of the blood flow for gravitational reasons but only 40% of the total ventilation. When OLV is instituted all ventilation goes to the dependent lung and the non-dependent lung blood-flow (40% total) is reduced by 50% through mechanisms including hypoxic pulmonary vasoconstriction. This means that 20% blood flow still passes through the collapsed lung.

**Management of hypoxaemia during OLV**

- At initiation of OLV the $F_IO_2$ should be raised at least to 0.5, if not 1.0. This is simple, physiological and effective. The mechanisms diverting blood from the non-dependent lung take time and later in the period of OLV it is possible to reduce the $F_IO_2$.
- Check the position of the DLT to make certain that all bronchopulmonary segments of the dependent lung are being ventilated.
- PEEP to the lower lung sounds as though it should be effective and fits in with respiratory care given in ITU, but it isn't reliably effective in thoracic surgery and may cause the $PaO_2$ to fall. An excellent paper[7] details compliance curves on OLV and raises the likelihood that 'excessive' PEEP may produce deleterious increases in pulmonary vascular resistance in the dependent lung. Attempts should be made to individualise the ventilation of the dependent lung by considering tidal volumes, rate of ventilation, I:E ratios, pressure/volume controlled ventilation and amount PEEP artificially produced by the long, narrow lumens of the DLT.
- CPAP with 100% oxygen to the non-dependent lung is effective but inconvenient to the surgeon particularly in thoracoscopy.
- Manipulation of HPV. This is often cited by trainees as a most important factor but Conacher[8] argues coherently against the notion that anaesthetic agent induced failure of HPV is an important or even evident process in clinical anaesthesia in humans.

**Hypoxic pulmonary vasoconstriction[9]**

HPV was first described in 1946 by Von Euler and Liljestrand who found that when cats breathed an $F_IO_2$ of 0.105 the pulmonary artery pressure increased. Sympathetic or parasympathetic blockade did not affect this response, and one physiological explanation is that it derives from a direct effect of oxygen on pulmonary arterioles. HPV response is a function of both alveolar *and* mixed venous oxygen tensions and experimental elevation of mixed venous $PO_2$ above 13 kPa abolishes the process. When the lung is collapsed during OLV it is suggested that HPV decreases the blood flow to that lung by 50%.

**Effect of anaesthetic agents on HPV**

Early animal work demonstrated a marked (clinically relevant) inhibition of HPV by inhalational but not intravenous agents. It sounds credible that anaesthetic agents with a vasodilatory property would antagonise a physiological process involving vasoconstriction. However, current work supports at worst only a very modest (clinically borderline) impairment of HPV by modern inhalational agents. This revision has been supported by

various criticisms of previous work. An agent may impair the effects of alveolar hypoxia but any reduction in cardiac ouput would reduce mixed venous oxygen and promote HPV. A recent paper[10] examines the effects of increasing isoflurane or desflurane on various parameters during OLV in intact pigs and is worth reading to understand the difficulties of interpreting data. In humans, general anaesthesia with isoflurane is associated with a higher shunt fraction than propofol during OLV, but the prevalence of hypoxemia is similar. Recently[11] no difference in shunt fraction between sevoflurane and propofol could be demonstrated in humans during OLV.

**Modification of pulmonary blood flow during OLV**

Two approaches are to reduce PVR in the dependent lung or increase PVR in both (or a combination). Inhaled nitric oxide is a potent, short acting pulmonary vasodilator which is supplied only to the dependent ventilated lung during OLV and to regions of high ventilation within that lung. It has been studied in several studies and interestingly has little effect on oxygenation except perhaps in those patients with a low $PaO_2$ pre NO treatment. A specimen paper[12] investigated the effects of 40 ppm NO on 30 patients undergoing OLV in the standard lateral position. Mean shunt fraction increased from 14% during TLV to 42% with OLV but was not affected further by NO. Mean $PaO_2/F_1O_2$ (mm Hg) values decreased from 420 on TLV to 170 on one lung but did not change with NO. In patients with a shunt fraction > 45% on OLV, $PaO_2/FIO_2$ value increased from 84 to 104 with NO. Nitric oxide is a toxic agent and alternatives[13] are being investigated.

In humans[14] almitrine, an intravenously administered pulmonary vasoconstrictor available in France, prevents or limits OLV induced decreases in $PaO_2$ (this effect had previously been documented in a dog model). In patients anaesthetised with propofol/sufentanil and ventilated with 100% oxygen, an almitrine infusion at 8mcg/kg/min was started at initiation of OLV. With placebo the arterial oxygen tension fell from 430 to 178mmHg over 30 minutes, but with almitrine it fell only to 325mmHg. No changes in CO or PVR were seen with this dose of almitrine.

**Pain relief following thoracotomy**

A good editorial[15] a year ago considered the topical issues. There is as much debate[16,17] about the use or need for epidural analgesia as in other areas of major surgery. One particular local technique with a good record is paravertebral block insituted either by the anaesthetist percutaneously (see www.nysora.com for technique) or most easily with the catheter placed by the surgeon at the end of surgery. We have used this latter technique at Guy's/St Thomas' for many years, infusing lignocaine 0.5% at 10ml/hr continuously for 48hr on the ward. It is supplemented by NSAID and PCA morphine. Advocates[18] suggest that paravertebral blockade may be more effective with a lower complication rate than epidural.

**Post lung resection morbidity/mortality**

About 15% - 40% patients develop an arrhythmia which is usually atrial fibrillation, (usually) on the second or third postoperative day. This is more common in older patients[19]and is associated with a longer hospital stay and increased mortality. Various papers have suggested that other factors associated with the development of AF are ischaemic changes on the ECG, cardiac enlargement, abnormal preoperative exercise test, intraoperative hypotension, postoperative pulmonary oedema, right sided operation and pneumonectomy. It may be indicative of poor cardiovascular reserve. Avenues of research are whether this represents sympathetic stimulation or raised pulmonary artery pressures. Relative sympathetic stimulation may result from intraoperative damage to parasympathetic pathways. A study[20] comparing the effect of bupivacaine with morphine thoracic epidural analgesia on the prevalence of arrythmias in 50 patients showed that bupivacaine was associated with a lower incidence and duration of arrythmia than morphine. Postoperative administration of oxygen[21] to the third postoperative day did not affect the incidence of AF.

The overall mortality rate after pneumonectomy is 7-12% in large[22] or national studies. The causes being pneumonia, sepsis, cardiac causes, DVT/PE etc. A specific problem is post-resection pulmonary oedema. This is more common, and more serious, after pneumonectomy (2-4%) than lobectomy. Criteria are the combination of clinical respiratory distress and hypoxaemia, with diffuse shadowing on CXR in the absence of cardiac dysfunction, pneumonia, sepsis or aspiration.

Slinger provides a thoughtful review[23] of the condition and lists what is 'known':

- Symptoms on postoperative day 2-4
- Radiographic evidence at least 24 hr prior to clinical signs
- More common after right than left pneumonectomy
- High mortality (> 50%)
- Resistant to standard 'pulmonary oedema' treatment
- Associated with fluid overload but not clear cause-effect
- Histological picture of ARDS
- Associated with normal low PAOP and high protein oedema fluid

Alvarez[24] describes the onset and management of the condition in 5 patients. It is clear that overzealous fluid administration is not the cause but may exacerbate the condition. It appears to be due to capillary endothelial leak. Are steroids of use?

Data from the management of ARDS patients suggests that volutrauma may be an important adverse factor. Perhaps OLV should be pressure rather than volume controlled.

**Internet resources and thoracic anaesthesia**

An article[25] lists many sites. A starting point is the 'related sites' link at the Journal of Cardiothoracic and Vascular Anaesthesia on www.jcardioanesthia.com

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## JOURNAL REVIEWS

| | |
|---|---|
| Anesthesiology | Dr Mike Girgis |
| Anaesthesia | Dr Peter J. Shirley |
| Anaesthesia and Intensive Care | Dr Damaris K^hler |
| Anesthesia and Analgesia | Dr Yue Dong |
| Canadian Journal of Anesthesia | Dr Aneeta Sinha |
| British Journal of Anaesthesia | Dr Paul Sice |

**Relative Analgesic Potencies of Levobupivacaine and Ropivacaine for Epidural Analgesia in Labour.**
Polley LS, Columb MO, Naughton NN, Wagner DS, Van de Ven CJM and Goralski KH, **Anesthesiology** 2003;99:1354-8

In this study the relative analgesic potencies of epidural levobupivacaine and ropivacaine were assessed by determining their respective minimum local analgesic concentrations (MLAC). 105 parturients requesting epidural analgesia with ASA status I or II, singleton pregnancies greater than 36 weeks duration, vertex presentation and in active labour with cervical dilatation of 3-7cm at time of catheter placement were enrolled. The participants were allocated to receive either levobupivacaine or ropivacaine in a randomised double-blind prospective study. The concentration of local anaesthetic received was determined by the response of the previous patient in that group using an up-down sequential allocation technique. The first patient in each group received 20ml 0.10% wt/vol of the corresponding local anaesthetic and the dose was either increased or decreased by 0.01% wt/vol with each subsequent patient. Maternal and fetal haemodynamic data were recorded at 5 minute intervals. Pain was assessed at 10 minute intervals for the first 30 minutes using a 100mm visual analogue pain scale (VAPS). Sensory level and motor blockade were also assessed. A VAPS of 10mm or less was defined as effective and resulted in a 0.01% wt/vol decrement for the next patient. A VAPS of more than 10mm which responded to rescue with a 12ml bolus of 0.25% wt/vol of the same local anaesthetic, was defined as ineffective and resulted in a 0.01% wt/vol increment in drug concentration for the next patient. If the patient was not responsive to rescue local anaesthetic, the result was defined as a reject and the next patient received the same concentration.

In total 35 women were excluded for either not responding to rescue local anaesthetic, protocol violation, entering the second stage of labour before completion of the study, intravascular epidural catheter placement, patient withdrawing and feetal heart rate deceleration secondary to cord prolapse. This left 35 patients in each group, the demographic and obstetric characteristics of both groups being similar. There was no significant difference in maternal or fetal haemodynamics between the two groups. The MLAC was calculated using the Dixon and Massey formula and was 0.087% wt/vol (95% CI, 0.081-0.094%) for levobupivacaine and 0.089% wt/vol (95% CI, 0.075%-0.103%) for ropivacaine. The potency ratio was 0.98 (95% CI, 0.80-1.20) indicating that levobupivacaine and ropivacaine have similar potencies. There was no difference in time to onset of block, block duration, spread of block, or motor block between the two groups.

**New aspects of ventilation in acute lung injury.** Malarrkan N, Snook NJ, Lumb AB, **Anaesthesia** 2003;58:647-667.

This review starts by stating the definitions of Acute Lung Injury (ALI) and pointing out that ARDS (Adult Respiratory Distress Syndrome) is a severe form of ALI, with a greater impairment of gas exchange. Of those patients treated in intensive care units with ARDS, only about half survive, with old age and sepsis associated with poor outcomes. However, the mortality from ALI is slowly decreasing and only a minority of patients actually die as a direct result of their injured lungs; most succumbing to underlying pathology or multi-organ failure.

The initial treatment strategy, prior to tracheal intubation, should be to give supplementary oxygen, treat the underlying cause, optimise fluid management and consider non-invasive ventilation if available.

The authors suggest a primary ventilation strategy, based on the best current evidence and published guidelines. In particular 'protective' ventilation with tidal volumes of 6 - 8ml/kg and stepwise increments of positive end expiratory pressure (PEEP) to maximise benefit and limit cardiovascular side-effects (usually 10-15cm$H_2O$). The lowest fractional inspired oxygen ($FiO_2$) should be used to achieve a $PaO_2 \geq 8$ kPa, and respiratory rate adjusted to achieve $PaCO_2 \leq 8$ kPa. The plateau airway pressure should be kept < 35cm$H_2O$ by adjusting the respiratory rate and possibly accepting a slightly lower $PaO_2$ or higher $PaCO_2$.

If this primary strategy fails, then secondary strategies are suggested. These include inverse-ratio ventilation, high-frequency ventilation, prone positioning, inhaled nitric oxide and partial liquid ventilation. Of these, prone positioning and inverse-ratio ventilation are achievable with limited resources.

The review concludes with the idea that in the near future a 'one size fits all' approach, with one artificial ventilation technique for all patients, will be abandoned as subgroups of ARDS and ALI are recognised.

**Delivery times for caesarean section at Queen Elizabeth Central Hospital, Blantyre, Malawi; is a 30-minute 'informed to start of delivery time' achievable.** O'Regan M. **Anaesthesia** 2003;58:756-759.

The author introduces this paper by emphasising the limited health facilities in their hospital and the demands placed on it by poverty related illness. Their aim was to see how closely they achieved the 30-minute time line from informing the anaesthetist to the start of an operative delivery, as recommended by the Association of Anaesthetists of Great Britain and Ireland (AAGBI) and

Obstetric Anaesthetists Association (OAA). A questionnaire study was undertaken. The time from informing the anaesthetist until 'knife to skin' (I - KTS time) was divided into component times and reasons for any delays noted at each stage. The classification of urgency of the requirement to do the caesarean section was also noted and divided into four grades.

78 questionnaires were completed in a 3-week period in April 2002. 65 (83%) spinal anaesthetics were performed, 9 (12%) general anaesthetics and 4 (5%) generals following failed spinal. The anaesthetic was administered by a student in 47 (60%) cases, a clinical officer in 25 (32%) cases and a physician anaesthetist in 6 (8%) cases.

A 30 minute I - KTS was achieved in 69% of grade-1 (immediate threat to the life of the mother or fetus) and 27% of grade-2 (fetal or maternal compromise but not immediately life threatening). The authors do accept that these figures may be better than expected due to increased effort during the audit period, nevertheless they are impressive.

In 18% of grade-1 and 2 cases the I-KTS interval was over an hour. The anaesthetist was unavailable for more than 15 minutes in one grade-1 and seven grade-2 cases. In five of these, the anaesthetist was busy with another patient.

A median time of 12 minutes was recorded for actually anaesthetising the patient. No attempt was made to analyse delays in terms of anaesthetic technique, the unit policy being in line with the Seventh Annual Confidential Enquiry into Stillbirths and Deaths (CESDI) to recommend not repeatedly attempting spinal anaesthesia in the absence of significant risk factors for general anaesthesia.

**Anaesthesia and Isolated Systolic Hypertension - Pathophysiology and Anaesthesia Risk.** Wongprasartsuk P, Sear JW. **Anaesthesia and Intensive Care** 2003;31:619-628

Hypertension is regarded as an additional risk factor in anaesthesia. Attitudes towards the management of the hypertensive patient perioperatively have changed with time and continuation of antihypertensive therapy is now recommended.

Traditionally, therapy was focussed on controlling the diastolic rather than the systolic blood pressure. Recent clinical studies have led to a new emphasis on isolated systolic hypertension i.e. systolic pressures of >160mmHg with diastolic pressures of <90mmHg. It has been shown that a large difference between systolic and diastolic blood pressure is a greater risk factor for stroke or other cardiovascular events than isolated diastolic hypertension or elevation of both the systolic and diastolic pressures. This seems also to be true for the development of chronic renal disease secondary to hypertension.

The aggressive reduction of elevated systolic blood pressure to values <140 mmHg reduces the risk of stroke, coronary heart disease and other cardiovascular events dramatically (23-44% for stroke, 21-26% for CHD and 25-32% for other cardiovascular events). This seems to be especially true for patients aged 65 years and older.

Association between hypertensive disease and perioperative cardiovascular events is established and recommendations do exist concerning the lowering of diastolic blood pressure to values below 100 - 110 mmHg prior to elective surgery. However, there are as yet no recommendations on the management of isolated systolic hypertension. There is a great need for further studies using isolated systolic hypertension and increased pulse pressure as markers for perioperative risk.

**Allergies to local anaesthetics - the real truth.** Finucane BT **Canadian Journal of Anaesthesia** 2003; 9:869-874

Many of us face the situation of a patient claiming that they are 'allergic to local anaesthetics'. The author of this article discusses some important issues regarding this situation.

Allergic reactions to local anaesthetic (LA) agents are rare and are much less common with amide than with ester compounds. *Allergists believe that less than 1% of reported allergic reactions to LAs are immune mediated - amide linked immune reactions constituting a minute fraction.*

Allergy is only one of the causes of adverse events when performing local anaesthetic procedures. Epinephrine is frequently added to solutions and injection can result in a cardiovascular response of which the patient becomes aware. Inadvertent intravascular injection of LAs can result in systemic toxic reactions. These, along with vasovagal reactions and 'panic attacks', account for the vast majority of adverse reactions. Latex allergies should also be considered. Patients often emerge from these events interpreting 'a reaction to the LA' as an allergy.

Any patient suspected of true immuno- allergy to LAs should be referred for testing. A thorough history and review of records is important. Skin tests are performed using a control, a known histamine releaser and the LA. Small quantities of these substances are injected intradermally and the responses compared. Positive reactors are subjected to gradually increasing concentrations of the LA until full strength is reached. Intradermal testing is a useful primary screening test but results are often equivocal. *In vitro* testing of LA can be performed and is recommended in patients with a history of anaphylaxis. Cell cultures of lymphocytes are exposed to the suspected allergen. Proliferation of lymphocytes suggests allergy and a leucocyte histamine response adds weight to results.

The term allergy is used too casually. Both education and a strategy are required to ensure patients experiencing adverse events are appropriately investigated and given correct explanations. This would prevent patients being incorrectly labelled as 'local anaesthetic allergic', thereby losing the benefits of regional anaesthesia.

**Age-related iso-MAC charts for isoflurane, sevoflurane and desflurane in man.** Nickalls RWD and Mapleson WW**. British Journal of Anaesthesia** 2003;91:170-4

This study describes the development and use of age-related iso-MAC charts for isoflurane, sevoflurane and desflurane to assist the estimation of appropriate end-tidal concentrations for patients of different ages. Mapleson described the decrease in MAC for different volatile agents with age and calculated age-related MAC as a function of MAC at 40 yrs

$MAC_{age} = MAC40^{-10-0.00269(age - 40)}$

It was assumed that the clinical effects of nitrous oxide (N2O) and inhalational agents are additive. The fractional end-tidal concentration (FE') = k ( MACage, where k represents a multiple of MAC and by combining the above equations they produced graphs of end-expired anaesthetic concentrations for different ages to give multiples of MAC ranging from 0.6 - 1.6 in the presence of 0%, 50% and 67% $N_2O$.

*The graphs can be used to estimate the total age-related MAC value given the end-tidal anaesthetic and $N_2O$ concentrations or guide the clinician as to the end-tidal concentration appropriate for the patient's age to provide a desired MAC.*

They also allow a consistent MAC to be maintained when changing the fractional inspired $N_2O$ concentration. When changing from 67% $N2_2O$ to 0% in the elderly a 3-fold increase in end-tidal anaesthetic concentration will be required to maintain a similar MAC. The authors claim that colleagues found the charts helpful and easy to use.

There is a wide variation in MAC at extremes of age. An end-tidal concentration of isoflurane of 0.7 at 1 year and 0.2 at 80 will provide an estimated MAC of 1.0 in 67% $N_2O$. This difference in the required concentration of volatile agent is exaggerated by the age variation in MAC for $N_2O$, (133% at 1 and 81% at 80). Anaesthetic monitors do not make age-related MAC calculations and elderly patients may receive higher concentrations of anaesthetic agents than necessary.

MAC refers to a population of patients and care must be taken when extrapolating it to individuals. However, *it is probably the best estimation of brain anaesthetic concentration, once equilibrium with alveolar concentration has been reached, and therefore depth of anaesthesia currently available*. Taken into context with the pharmacological elements of a balanced anaesthetic, the degree of surgical stimulation and patients' physiological parameters during anaesthesia these charts provide a useful guide to depth of anaesthesia, especially at extremes of age.

**New light on intravascular volume replacement regimens: what did we learn from the past three years?** Boldt J. **Anesthesia & Analgesia** 2003;97:1595-604

Hypovolaemia is common in surgical, trauma, and intensive care unit (ICU) patients. Adequate intravascular volume replacement therapy may help to improve organ function and reduce patient morbidity or even mortality. There is still no widely accepted golden standard of adequate volume replacement strategy. The author undertook a key word MEDLINE search and reviewed 40 clinical studies published between 2000-2002.

The crystalloid / colloid debate continues. Crystalloids may have a negative influence on coagulation (hypercoagulability) and metabolic state (hyperchloremic acidosis) following large volume infusions and appear to have no beneficial effects on the microcirculation and organ perfusion.

Dextrans are not the first choice for volume replacement and gelatin was used in several recent studies without showing any severe negative effects.

Hydroxyethyl starch (HES) is the plasma substitute that has been studied most often but concerns remain about the effects of HES on renal function, coagulation and organ perfusion.

Because the studies did not have the same outcome endpoints it is hard to compare different treatments and the author doesn't give direct suggestions as to which agent is best. Colloids may have a better outcome compared to crystalloids. Different colloids have different therapeutic profiles but due to the lack of standard guidelines for choice of fluid and outcome measurement, it is very hard to draw a final conclusion based on this paper. Good prospective, double blinded studies on this topic are needed.

| Age | 1 MAC isoflurane | | 1 MAC sevoflurane | | 1 MAC $N_20$ |
|---|---|---|---|---|---|
| | 100% $O_2$ | 67% $N_2O$ | 100% $O_2$ | 67% $N_2O$ | |
| 1 | 1.5 | 0.7 | 2.3 | 1.1 | 133 |
| 20 | 1.3 | 0.55 | 2.1 | 0.9 | |
| 40 | 1.15 | 0.4 | 1.8 | 0.65 | 104 |
| 60 | 1.05 | 0.3 | 1.6 | 0.45 | |
| 80 | 0.9 | 0.2 | 1.4 | 0.3 | 81 |

# INDUCTION OF ANAESTHESIA IN PAEDIATRIC PATIENTS

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## Introduction

Induction of anaesthesia in children is achieved with broadly the same anaesthetic agents and techniques as are used in adults. However, there are some important differences in the pharmacology of the agents available when comparing adults and children. Also, the technical difficulties that are associated with small size and the psychological and behavioural issues due to immaturity may make induction of anaesthesia more challenging in the child compared to the adult.

Paediatric anaesthesia has been the subject of this journal before.1 Analgesic methods in children have also been discussed.2 In this short paper, the pharmacology of the induction agents will be covered. The different methods available for inducing anaesthesia will be discussed and the merits of each method compared. Techniques that are specifically designed to overcome difficult situations relevant to paediatric anaesthesia will be discussed.

There are several methods of anaesthetic induction: Gaseous induction, breathing a mixture of volatile anaesthetic agents until loss of consciousness is achieved; Intravenous induction, where an anaesthetic drug is injected intravenously in a dose sufficient to produce unconsciousness; Other, where an induction agent is given by a non-intravenous route, generally orally, rectally or intramuscularly, to produce loss of consciousness.

## Pharmacology - the intravenous agents

**Sodium Thiopentone**. First introduced in the 1930's, this barbiturate has been the mainstay of intravenous induction ever since. It is also known as pentothal or Nembutal. It is supplied as a yellow powder mixed with sodium carbonate and is dissolved in water before use. Solution in water results in an alkaline pH. The concentration in water is important as pain free injection is only reliable with a dilute solution (2.5% or 25mg/ml or less). The solution may result in severe tissue necrosis if injected extra-vascularly and this is worse with the more concentrated solutions. The induction dose of thiopentone is between 4-6 mg/kg in adults and 5-7 mg/kg in infants and children. Induction of anaesthesia is rapid and generally accompanied by minimal excitatory effects such as involuntary movement or hiccupping. Thiopentone is protein bound and highly lipid soluble. It's effect on the central nervous system, unconsciousness, is terminated by redistribution of the drug which results in recovery of consciousness about 5 minutes after induction by a single dose. If repeated doses are administered, recovery may be significantly prolonged. The drug is metabolized by the liver but the efficiency of this process has little bearing on the anaesthetic duration of action. Children handle thiopentone slightly differently from adults. The induction dose is higher. The elimination half time is reduced and this means that "hang-over" after thiopentone induction, is much less of a problem in children than in adults. When used clinically, anaesthesia is induced rapidly after injection. There is generally a short pause in respiration, but this rarely lasts more than a few seconds. Respiration then resumes and a volatile agent may be introduced whilst the patient is spontaneously breathing. Heart rate generally rises slightly on injection but there is vasodilation and a drop in cardiac output. This is clinically significant in hypovolaemic patients and those with intercurrent medical conditions but in otherwise healthy patients, is well tolerated. Cardiovascular compromise is less marked than with propofol. Hypersensitivity (allergy) is rare but generally very serious. The risk of anaphylaxis is quoted at 1:50,000 administrations but may carry a 50% mortality. The major specific contraindication is porphyria.

**Propofol** is a non-barbiturate intravenous anaesthetic agent introduced in the 1980s. It is presented as an aqueous solution in soya oil and egg phosphatide. In a dose of 2.5-4mg/kg, it rapidly induces anaesthesia. In this dose, excitatory movement is common and it is now common practice to use a higher dose in un-premedicated children. Typically, 4mg/kg is administered as a bolus, followed by aliquots of 0.5-1mg/kg to allow a smooth transition from propofol anaesthesia to a vapour based anaesthetic. Even in higher doses, there are more excitatory and involuntary movement than with thiopentone. Unlike thiopentone, repeat doses may be given without unduly affecting the quality of post-operative recovery. Indeed, it is used as a sedative for medium to long term use in the intensive care unit (not in children). An induction dose causes a more prolonged pause in respiration than thiopentone. With higher induction doses, this pause becomes longer and in clinical use, apnoea is frequently produced with this agent. Airway reflexes are depressed after an induction dose and it is said that airway instrumentation is facilitated more using this drug than with alternatives. The cardiovascular effects are dose dependent but a reduction in blood pressure is seen. In the hypovolaemic, this may be profound and dangerous. This effect is greater than with equivalent doses of thiopentone. Although less irritant than thiopentone in the event of extra-vascular injection, the chief disadvantage to the use of this agent in children is pain upon injection. This pain is quite severe and detracts from a smooth induction. It may be alleviated, but not prevented, by the co-administration of lignocaine in a dose of 1mg lignocaine/ml of propofol 1% solution. Propofol infusion syndrome may result if propofol, in high doses, is infused over long periods of time in children who are critically ill. Whilst the mechanism of this syndrome is not completely clear, it appears that this is a safe drug for use as an induction agent in children but it is not licensed as a intravenous sedative in children. In the UK, propofol costs three times more than equivalent dose of thiopentone.

**Etomidate** is a non-barbiturate induction agent that is used in doses of 0.3-0.4 mg/kg. It's use results in less cardiovascular depression than thiopentone and there is little or no depression in the respiratory rate or depth. It is associated with considerable

involuntary movement after injection and this makes induction much less "smooth" than with other agents. Etomidate is associated with pain on injection. Etomidate inhibits the synthesis of steroids by the adrenal gland and this finding has been used to explain a high mortality noted when this agent was used for sedation of young children on intensive care. Concern over it's inhibition of steroid synthesis, pain on injection and practical considerations relating to movement on induction have resulted in unpopularity of this drug for paediatric induction.

**Ketamine** is a non-barbiturate intravenous anaesthetic with many unusual and useful properties. Although anaesthesia is induced after an 2mg/kg intravenous injection, the presence of movement, opening of eyes and maintained respiration mean there is no clear "end-point" and it appears that induction is more prolonged than with thiopentone. However, there is preservation of heart rate and blood pressure at normal or supra-normal levels. Respiration is maintained at a higher rate and tidal volume. There is some preservation of airway reflexes during anaesthesia with this agent. The protective airway reflexes and increased respiration has lead to the popularity of this drug in circumstances where it may be used as a single agent, perhaps with limited access to anaesthetic equipment. However, it must be acknowledged that with overdosage, airway reflexes may be lost and respiration depressed and oxygen must always be available for it's safe use. An advantage to this drug is the versatility in the manner in which it may be given. Ketamine may be administered via the intravenous, intramuscular, rectal and oral routes. Drawbacks to it's use are excessive salivation and unpleasant dreaming. Excessive salivation may be improved with the use of an antisialogogue such as atropine. The dreaming may be reduced by co-administration of a benzodiazepine. Ketamine is relatively inexpensive.

**Benzodiazepines**. Midazolam and diazepam injection have been used as induction agents. The dose of each member of this class of drugs is very variable. Thus a dose of 0.05 to 0.5mg/kg of midazolam may be required to induce sleep. The time to peak effect is much longer than other induction agents and most anaesthetists find the best use for this class of drugs is as a pre-medicant. As a pre-medication, midazolam is widely used. It is given orally at a dose of 0.5-0.75mg/kg. It rarely produces deep sleep but renders a child placid and co-operative. Further it provides useful amnesia. Studies have demonstrated that midazolam may be used as premedication before day case surgery without delaying discharge.

**Pharmacology - volatile agents used for gaseous induction**

**Ether**. The original volatile anaesthetic agent and still much in use world-wide, the high solubility and irritant nature of this agent means it is not an easy induction method in children. Due to the difficulties encountered in obtaining other drugs and equipment, it is still used as a sole anaesthetic in many places but will not be discussed further here.

**Halothane** was introduced in the 1950s and rapidly became popular for maintenance and volatile induction of anaesthesia in both adults and children. It is administered through a dedicated vaporizer into a carrier gas. It has a MAC of 1.1% in infants and 0.6 in the elderly. The smell is non-irritant and not unpleasant. The most common method of inducing anaesthesia with halothane is to start with the patient breathing the carrier gas which might be a mixture of oxygen and air or nitrous oxide. Halothane is introduced at 0.5% and then the patient breathes 5 normal breaths, the concentration is increased by 0.5% for another 5 breaths until 5% halothane is reached. Once unconsciousness is produced, the concentration may be reduced to an appropriate level. Halothane is a respiratory depressant and tidal volume is reduced. Respiratory rate may actually be increased a little during halothane anaesthesia but the response to hypoxia or hypercarbia are attenuated. However, these effects are to a lesser extent than with other volatile agents with the exception of ether. The principal disadvantage of halothane is its potentiation of the arrhythmic effects of catecholamines on the myocardium. Arrythmias, particularly ventricular arrythmias, are more common with this agent than with other volatile agents. Hypercarbia causes release of catecholamines from the adrenal gland and the combination of mild airway obstruction, hypercarbia and halothane are a frequent cause of arrhythmia under anaesthesia. Usually, these are benign and respond to correction of the airway obstruction but in the presence of administered adrenaline (by sub-cut injection) these arrythmias may be dangerous. Post exposure hepatitis has been reported with halothane and extensively investigated. Its occurrence in children is not clear but is certainly very rare indeed.

**Enflurane and isoflurane** are both more pungent than halothane and have no advantages for gaseous induction of anaesthesia

**Sevoflurane** has been used in Japan since the 1970s. It is a volatile agent with a MAC of 2.3 in infants and 1.8 in adults. Its major advantage is that it has a smell which is non-pungent and it is possible to induce anaesthesia with high concentrations from the outset. It has no arrythmogenic effect. When compared to halothane, higher concentrations may be used earlier in induction, without complaint. Therefore, it appears to cause a swifter onset of anaesthesia. A disadvantage is that it is a more potent respiratory depressant than halothane and therefore, breath holding may occur before a truly deep stage of anaesthesia is reached. For this reason, some anaesthetists prefer halothane to sevoflurane when performing gaseous inductions for airway obstruction. The other major disadvantage to this agent is its high cost.

**Induction of anaesthesia**

**Gaseous induction of anaesthesia.** Anaesthesia is commonly induced in infants and children by means of a "gas induction." This is less commonly the case with adults, leading some anaesthetists to be unfamiliar or lacking in confidence with this method. Children, understandably, are reluctant to have a "needle" to put them to sleep. Many are aware of an alternative and will prefer this method. Infants may be very hard to cannulate prior to an intravenous induction, meaning that a gas induction becomes preferable. In nearly all cases, anaesthesia will be induced without an intravenous cannula in place and intravenous drugs will be hard to administer, particularly if the anaesthetist is working single handed.

Neonates may lie on the operating table and breathe from an anaesthetic mask attached to a T piece, or similar, low resistance

anaesthetic circuit. Older children will, if adequately informed, frequently behave very well and will lie on the operating table and accept a gas induction. Between these age groups, the skill of the anaesthetist must be fully employed to ensure a smooth induction. Infants and young children are often very reassured by the presence of a parent in the anaesthetic room. This may not be possible in some circumstances but if feasible, pays dividends. With the presence of a parent, the child may receive a cuddle whilst having a gas induction. An older child may be persuaded to co-operate by a parent. Various games may be employed to distract children enough for them to receive a gas induction. "Blowing up the balloon" will be familiar to most anaesthetists and is a very effective way of persuading children to "breathe the gas." Another useful technique is to use a strong smelling food substance and rub it in the face mask. Orange peel is a useful way of disguising anaesthetic gases. If rubbed on the mask, a "guess the fruit" game can be enjoyed whilst the child goes to sleep. Clear plastic face masks alleviate the claustrophobia associated with the black rubber face masks. If halothane is used, care should be taken to move up the concentrations incrementally, taking plenty of time to allow the child to breathe comfortably. Increasing the concentration too fast results in coughing. Many anaesthetists prefer to use 70% nitrous oxide before adding halothane as this means the patient is already partially anaesthetized before smelling the halothane. Sevoflurane is more forgiving in this respect. Once the patient is asleep, most anaesthetists switch to 100% oxygen as a carrier gas.

Once the child is asleep, any parents present should be invited to leave. A pulse oximeter may be applied if it has not already been positioned. The child should be disturbed as little as possible. Once asleep, the patient goes through an excitatory phase. If the child is moved about, for instance, to remove clothing, this is often the stimulus that provokes airway reflexes. The anaesthetist should continue holding the face-mask and child's airway, maintaining a clear airway and good ventilation using oxygen and a high concentration of volatile anaesthetic agent, until a deeply anaesthetized state is reached. At this point, the child may be moved to insert an iv cannula, to undress them, to apply other monitoring and to facilitate surgery. If an iv cannula is needed, this is the time to insert it. If a tourniquet is used, the insertion of an iv cannula may be achieved one handed whilst the anaesthetist holds the face mask. However, with harder subjects, another pair of hands, to hold the airway or perhaps, to cannulate the patient, will be invaluable.

The obvious question is what to do about any adverse events that occur before an iv cannula is inserted. The answer is that, with the exception of airway obstruction, all other problems are exceptionally rare. With the anaesthetist holding the patient's airway, he is ideally placed to diagnose and treat airway obstruction as it occurs. Hypoventilation is noted by decreased excursion of the breathing reservoir bag. Airway obstruction may be revealed by noisy breathing or increased work of breathing (increased chest excursion with decreased bag excursion). Normally, correcting the position of the patient's head corrects hypoventilation. Noisy breathing is often due to upper airway collapse during expiration and a small amount of continuous positive airway pressure (CPAP) will resolve it. This is generally applied by keeping tension on the reservoir bag with one hand. If hypoxia occurs, as evidenced by the pulse oximeter trace or by cyanosis, check that a maximum concentration of oxygen is being given. Increase the CPAP. Occasionally, an oro-pharyngeal airway is helpful but care should be taken that this is not inserted at too light a plane of anaesthesia. It is rare that serious laryngospasm can not be overcome with patience, CPAP, 100% oxygen and correct positioning. If the situation does not resolve consider other causes of airway obstruction and consider applying suction to the pharynx to clear any secretions which may be lying around the larynx. If it becomes necessary to paralyse the patient, after they are asleep, but before an intravenous cannula may be inserted, remember that suxamethonium may be given intramuscularly (5mg/kg) and will work within 2-3 minutes. Many anaesthetists prefer to intubate patients once they are deeply anaesthetized with a volatile anaesthetic agent alone. It has been advocated that suxamethonium may be given intramuscularly into the tongue. I have no personal experience of this. My feeling is that it might render a difficult airway worse by adding intra-oral bleeding to the clinical picture. I have no evidence for or aginst this route being any faster, or slower, than into any other muscle.

There are some circumstances when inhalational anaesthetic induction is not the method of choice. If a child already has a cannula in situ, perhaps for maintenance fluid therapy, then it is more appropriate to use this cannula. Many children express a preference for intravenous anaesthetic induction. Also, there are numerous occasions where a rapid sequence induction is indicated and here, inhalational induction is completely inappropriate.

**Intravenous induction**

The main problems with intravenous induction are pain on insertion of the cannula, a natural aversion of children to "needles" and difficulty in insertion. These are all relevant to adults but here we may reason with our patients and explain why it is necessary and why the cannula may be hard to place.

Children as young as five may well understand the reasons behind needing a cannula and may even understand that sometimes it is not easy to insert them and a second go might be required. Whatever the reasoning powers of the child, the whole process may be made much pleasanter by the application of topical anaesthesia to prevent the child feeling the cannula needle. EMLA (eutectic mixture of local anaesthetics) takes about one hour to become effective. If placed over a cannulation site for an adequate amount of time, it is very effective. Amethocaine gel works faster. Unfortunately, these drugs are not uniformly available and sometimes, the only sensible plan is to explain why a cannula is needed and to use the smallest gauge possible. If nitrous oxide is available, the child may breathe a mixture of nitrous oxide and oxygen whilst the cannula is inserted but this technique often seems to combine the worst aspects of both intravenous and inhalational techniques, the child getting a "nasty mask" and a "horrible needle!"

Insertion of a venous cannula may be easy if the veins are obvious. Sometimes, this is a difficult procedure. The task is harder when the child has a large amount of subcutaneous fat, a common situation in toddlers. Veins become smaller in cold, dehydrated and frightened children. A warm, well hydrated, comfortable child

should be our aim and parental presence or pre-medication may well help.

After insertion of an intravenous cannula, suitable monitoring can be attached and an intravenous induction agent injected. The choice of agent is described above but in a healthy child, the normal choice is between sodium thiopentone and propofol. Propofol undeniably results in less "hang-over" in the post-operative period. However, after one hour, this difference between sodium thiopentone and propofol becomes very subtle in children. Pain on injection is a considerable problem, especially when we have gone to such lengths to secure painless venous access. Therefore, unless immediate post-operative discharge is needed, my preference is still for sodium thiopentone. This drug is injected as a single bolus of 5-6 mg/kg, the child painlessly goes to sleep and after the briefest pauses, begins to breathe. Maintenance with a volatile agent may then be substituted without having to recourse to a period of positive pressure ventilation, with a bag and mask, as generally is the case when propofol is used.

**Rapid sequence induction**

The indication for a rapid sequence induction is the same in adults and children. If a risk of aspiration of gastric contents is foreseen, a rapid sequence induction should be performed.

The procedure for this is the same in children as in adults. A working intravenous cannula is mandatory. The patient should be monitored and positioned on a tilting trolley with suction readily available. Oxygen is administered via a close fitting mask for 3 minutes and anaesthesia induced. As the induction agent works, an assistant applies cricoid pressure to the cricoid ring, with one hand supporting behind the patient's neck. This manoevre seals the oesophagus and prevents material from the stomach and oesophagus reaching the pharynx. The traditional agents are sodium thiopentone 5mg/kg and suxamethonium 2-3 mg/kg.

In practice, this procedure presents several problems and it is rare to achieve such good pre-oxygenation in a child as with an adult. Good rapport and explanation works for middle sized children but in the younger or less co-operative, three or four good screams into the oxygen mask is often all that can be managed. In this situation, it seems sensible to delay administration of the short acting depolarizing muscle relaxant until a few breaths of oxygen are taken. In this technique, an attempt is made to pre-oxygenate the child pre-induction. The thiopentone is administered and sleep induced. Cricoid pressure is applied and the mask closely applied. The child should take a breath quite soon after the thiopentone and once this is seen, suxamethonium administered. The suxamethonium is effective more quickly than in adults at this dose.

A difficult situation is the child with a full stomach, needing emergency surgery, who can't be cannulated. Although not ideal, I think the most practical way forwards here is to induce anaesthesia by volatile induction with the patient in the lateral position. Once anaesthesia is induced, it should be easier to secure intravenous access, apply cricoid pressure, turn the patient supine and perform intubation.

**Other means of induction of anaesthesia**

In rare instances, induction of anaesthesia is most appropriately conducted using ketamine. Ketamine may be given intra-muscularly using a tiny needle and then reliably induces anaesthesia if used in a dose of 7.5mg/kg. Anaesthesia onset takes about 5 minutes. The intramuscular route seems more reliable than the oral route, for which, the dose range quoted in the literature is very wide.

**Conclusion**

The debates over which drugs and which methods are "best" to use to anaesthetize infants and children are popular at meetings of paediatric anaesthetists. The fact that these debates occur indicates that there are no answers. The skilled, confident anaesthetist, who is prepared to react flexibly and to adopt methods to suit the opportunities presented by a child and it's parents, will have most success.

**References**

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# CIRCUMCISION UNDER LOCAL ANAESTHETIC

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**Key Points**

Circumcision is a common procedure which can be carried out under local anaesthetic. The block is simple to perform and surgerycauses little patient distress and has few complications.

**Introduction**

Circumcision in the United Kingdom is normally performed under a general anaesthetic with a penile block used for postoperative analgesia. However, local anaesthesia for the penis is simple to achieve in most cases, and can give good intraoperative pain relief. There are three main approaches to achieving an effective block.

1. Eutectic mixture of local anaesthetics, EMLA (Lignocaine and prilocaine cream).

2. Dorsal penile nerve block.

3. Ring block

**Anatomy**

The penis is innervated by the left and right dorsal nerves, which are branches of the pudendal nerve. The dorsal nerve on each side passes under the inferior ramus of the pubis and penetrates the layer of superficial fascia to supply the skin and also gives a branch to the corpus cavernosus. The nerves on either side are separated by the suspensory ligament of the penis.

**Technique**

● **EMLA** The cream is applied to the prepuce on both the skin and mucosal sides if possible and a condom is placed over the penis to keep the cream in position. This is left in place for at least 45 minutes prior to the procedure. It should not be applied to bleeding areas. Before a formal block is administered the condom is removed and the area tested for sensation. In a number of patients, eg elderly diabetics, it will be possible to carry out the circumcision without further anaesthesia.

● **Dorsal Penile Nerve Block** For an adult a mixture of 0.5% bupivacaine 10mls and 1% lignocaine 10mls (both without adrenaline) is made up to a total volume of 30mls with normal saline. With the patient supine, a 27 gauge needle is inserted over the middle of the pubic arch at the base of the penis (see position marked A in figure 1a and b) until it contacts the pubic symphysis, it is then withdrawn slightly and redirected to pass below the symphysis to left or right of midline to a depth of 3-5 millimetres deeper than the depth to the pubic symphysis (see figure 1a). After aspiration to confirm no flash back 5-7mls of solution are injected depending on the size of the patient. Without bringing the needle out of the skin the procedure is repeated on the other side. The needle may then be withdrawn completely or withdrawn to skin and the dorsal part of a ring block performed.

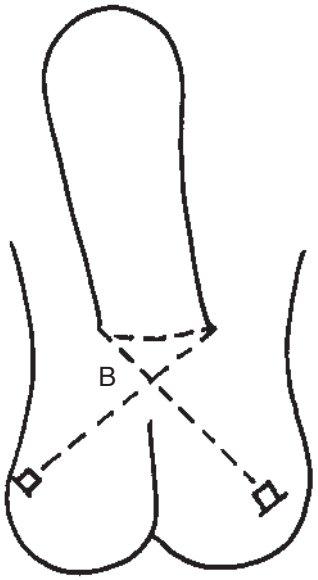


*Figure 1a: Cross-sectional view*

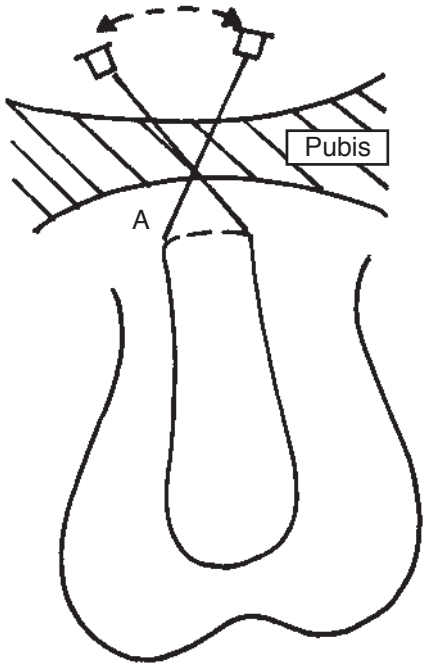


*Figure 1b: Anatomical view*

● **Ring Block** The same strength of solution as described above can be used to perform a circumferential subcutaneous injection at the base of the penile shaft using a 26 or 27 gauge needle. About 10mls of this solution should be enough to surround the base of the penis from two injection sites one ventrally (A on figure 1a and b) and one dorsally (B on figure 2).

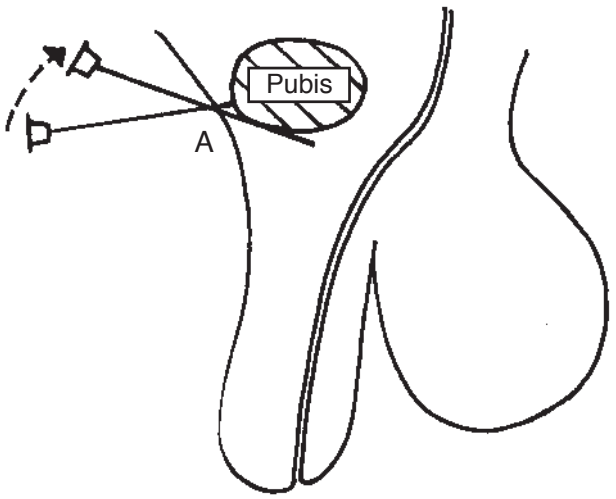


*Figure 2: Anatomical illustration showing entry point (B) of needle for administration of frenular block or ring block, the anaesthetic is infiltrated into the subcutaneous tissue*

**Choice of Anaesthetic Technique**

A combination of the three techniques is most likely to give effective cover. However EMLA is not commonly used.

By making up a 30ml solution at the beginning of the procedure, using 10mls for the dorsal penile block and 10mls for the ring block, there should be enough left over to inject additional solution should the block miss one particular area. When injecting during surgery it is better to inject using the ring block technique rather than directly into the affected area to avoid bulging of the subcutaneous tissues at the operative site, which makes identification of tissue planes more difficult. Any of these techniques could be combined with an oral or intravenous sedative in the anxious patient.

**Pitfalls and Complications**

Dorsal penile nerve block can miss the nerves to the frenulum and it is advisable to inject 1-2mls of anaesthetic at the base of the ventral aspect of the penis (marked B in figure 2) if a penile block without a ring block is being used.

An adequate amount of time must be left between injection of the anaesthetic agents and commencing the operation - normally 10-15 minutes, otherwise the anaesthetic will not have time to work.

Anaesthetics containing adrenaline should never be used because they cause arterial vasoconstriction, which may lead to ischaemia or necrosis of the penis.

A few cases of ischaemia have been described following dorsal penile block and this has been attributed either to using larger volumes of anaesthetic, or local haematoma formation causing compression of the dorsal penile artery.

Permanent numbness following the block is an uncommon complication and has been attributed to damage to the dorsal nerve of the penis.

**Conclusion**

Adequate local anaesthesia for circumcision can be achieved using a combination of simple techniques in the majority of patients and should probably be offered more frequently than is current practice. The combination of general anaesthesia supplemented with local anaesthesia to the penis is used routinely in many centres.

# FIRES AND EXPLOSIONS IN THE OPERATING ROOM

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The Operating Theatre/Room (OR) is a potentially dangerous place with regard to **fires** and **explosions**, due to the presence of:

- Flammable substances
- Oxygen and/or nitrous oxide;
- Sources of ignition (flames, sparks).

This article will explain how fires and explosions develop and the precautions, which, when acted upon will help to avoid this serious and sometimes disastrous event. The patient and staff may be injured or killed by a fire or explosion.

**Oxidation**

The basis of all combustions and explosions is **oxidation**, which is the chemical reaction between a substance and oxygen. Atoms are broken-up and rearranged to form a new compound with the production of energy, mainly in the form of heat but often associated with light, sound, pressure and electricity. This is an **exothermic oxidative reaction**.

**Oxidation can occur in a number of different ways:**

● **Biological oxidation**. Glucose, like a number of other carbohydrates, is oxidised in humans consuming oxygen and producing energy (3,700kcal/kg). This happens in a very slow, and well regulated manner, at a low and almost constant temperature through a series of enzyme reactions. The end point of the reaction is the production of carbon dioxide ($CO_2$) and water, plus energy, which is dissipated as heat or utilized in other reactions.

● **Combustion**. Alternatively wood under different conditions may **burn**, consuming oxygen and producing exactly the same amount of heat but in a much shorter time at a higher temperature. Similarly petroleum gases contained in the cylinders for kitchen use can burn in a controlled way in a stove to boil surgical instruments.

**Combustibles**

These are substances capable of reacting with oxygen to produce heat at high temperatures. Many combustible materials, which include alcohol, cotton fabric, wood and rubber, are present in the operating theatre. For complete combustion to occur there is an ideal proportion of fuel and oxygen, which is defined as a **stoichiometric mixture**. For instance, the stoichiometric mixture of diethyl ether vapour in oxygen is one mole (see Appendix 1) of ether (74g) and 6 moles of oxygen (192g) or about 14 % ether vapour in oxygen. In air, the stoichiometric concentration of ether is 3.4% and in nitrous oxide it is 8%.

In practice these exact proportions seldom occur. When the concentration of fuel is more than ideal, the mixture is described as "rich", with some fuel being left either unburned or incompletely oxidised into a range of compounds (e.g. carbon monoxide, or acetaldehyde in the case of ether). When the fuel concentration is less than the ideal, the mixture is described as "lean" with some oxygen left over. Whether oxidising sugar at body temperature or burning gas in a stove at a much higher temperature, the reaction normally proceeds until either the fuel or oxygen are finished. Moreover there is a balance between the energy produced and the energy, mainly heat, which is dissipated (escapes). The reaction is at an almost constant temperature, called an **isothermal reaction**. When heat production is faster than dissipation, heat will accumulate and the reaction can enhance itself to the point of an **explosion**.

**Flame**

Normally a flame remains confined to a fixed point and is called a **static** flame - the candle flame, gas burner and spirit lamp are examples. Of more interest is the **self-propagating flame**, again produced in a lean (1/10 stoichiometric) air mixture inside a tube. This describes a flame, which can travel along leaving behind the products of combustion, whilst the front of the flame heats fresh mixture which in turn ignites and becomes a flame itself. The process is called **deflagration**, which is generally a mild phenomenon but can become very dangerous if it comes in contact with an explosive mixture.

A very peculiar phenomenon is the production of a cool flame by the oxidation of very rich mixtures of certain volatile agents with air. Ether can form a cool flame at concentrations around 20% to 35% when heated to as little as 200°C. There is a small zone of oxidation (not a real burning) at a low temperature and with barely visible light. The cool flame travels along the mixture, eventually dying off. However the danger is that it can act as a powerful ignition source if it encounters an explosive mixture. In addition it may remain unseen until it is too late! A classic example occurs when ether is spilled on the floor and, because it is heavy, does not spread, forming a very rich localised mixture. A faulty electric plug could then ignite the ether mixture causing a cool flame, which then travels along the floor until it reaches a place where the mixture is explosive, such as the exhaust from an anaesthetic machine, which then explodes.

**Activation energy**

Energy, usually heat, is needed to start the reaction. This is called the **activation energy**, which can be provided by an open flame, sparks, hot plate or filament. The activation energy required to start a reaction varies very much, but for ether/oxygen mixture it is very little. In practice it is the temperature of the ignition source, which is measured. The **minimum ignition temperature** for the most inflammable mixtures of anaesthetic vapours in air lies between 400 and 500°C. Note in oxygen the **minimum ignition temperature** is some 50°C lower. In contrast, a cool flame may start at a temperature as low as 200°C in a rich mixture of ether in air (20 to 35%).

**Reaction Rate**

The reaction rate is directly related to the size of the activation energy. Thus the higher the activation energy, the more rapid the reaction rate, and the more likely an explosion. Other factors may influence the initiation and the rate of reaction.

**Temperature of the mixture**

The speed of the reaction is doubled when the initial temperature is raised by 10°C (**Arrhenius law**). If the heat generated at the beginning of a reaction is only partially dissipated, the small amount of heat left behind is sufficient to raise the temperature and thus the rate of the reaction. In contrast, when, adequate heat loss occurs, such as in large rooms, the reaction may come to a halt. A self propagating flame, as described above, produced by burning ether in air inside a tube can progressively increases its temperature and pressure causing a powerful deflagration with an explosion of the tube especially if the end is closed. If the tube contains an oxygen rich mixture, a much more powerful event can be produced. Fortunately this requires the combination of a very powerful ignition and a long tube. This cannot be produced in common anaesthetic machines.

**Limits of flammability**

If the mixture becomes too lean it cannot ignite. The **Lower Limit** (LL) for diethyl ether is 2.1% (vapour) v/v in either air or oxygen. There is also an **Upper Limit** (UL) where there is an excessive concentration of fuel for the oxygen present. The UL for ether in air is 36%v/v and 82% in oxygen.

**Nitrous Oxide**

Although $N_2O$ does not enter the biological oxidising processes, it is a powerful oxidant i.e. it strongly supports any combustion process. It is absolutely wrong to assume that it will prevent fires and explosions by dilution of oxygen. It is as effective as oxygen in producing explosive mixtures.

**Implications for Anaesthesia**

The conditions for flames and explosions require three essential components, a combustible substance, a source of ignition and oxygen. Despite the dangers described, in practice they rarely occur in the theatre, provided staff are careful, understand the mechanisms and take the appropriate precautions.

**Flammable Substances in the Operating Theatre.**

● **Diethyl Ether** burns slowly in air and is not easily ignited by a spark; mixtures with oxygen and/or nitrous oxide become explosive between approximately 1.5 and 40% v/v. Maximum detonability is approximately 15% in oxygen. Therefore hot wires and plates at 300°C, below the temperature of dull-red visible heat, are sufficient to start an invisible flame. Remember that ether vapour is denser than air and therefore sinks, spreading over the floor. Store ether in the dark to prevent auto-oxidation, which can make the simple shaking of the bottle enough to trigger an explosion. Anti-oxidants are added to "anaesthetic" or laboratory ether, reducing this risk.

● **Ethyl chloride** burns easily and explodes in oxygen or nitrous oxide or air - stoichiometric concentrations are respectively 25%, 14% and 6.5% v/v, with narrow ranges between LL and UL. Ethyl chloride is very dangerous.

● **Petroleum lubricants** - a sudden rise in pressure in a confined area such as a reducing valve or pressure gauge, can generate sufficient heat to ignite petroleum lubricants and cause an explosion.

● **Alcohol** burns very easily with an almost invisible flame, which is easily overlooked. It can easily soak drapes or swabs, which can then be ignited by a diathermy spark, especially in the presence of oxygen or nitrous oxide.

● **Natural** (generally methane or hydrogen) or anaesthetic gases inside body cavities (bowel, or alveoli) as well as swallowed ether in the stomach can explode or be ignited by the diathermy.

● **Propane, butane or other petroleum gases** for burners, stoves, lamps are very common sources of domestic and theatre disasters.

● **Modern volatile agents**. None of these (desflurane, isoflurane, sevoflurane) are inflammable; however they are expensive and not always available. Halothane, which is inexpensive and widely available, is also not inflammable, nor is the azeotropic mixture (halothane 66%-ether 34% - See Appendix 3).

**Source of ignition energy**

● **Extraneous flames** - any kind of open flames from candles, burners, matches, burning lamps etc. however small.

● **Hot wires and plates** - cautery, electric stoves, hot-wire spirometers, electric bulbs especially those for endoscopy or modern halogen lamps, glowing cigarette ashes.

● **Electrosurgical appliances** - these are powerful sources of ignition energy in the form of sparks, arcs or heat within the machine, the foot switch, diathermy spark, or faulty equipment. Sparks also happen in normally functioning switches, or when a live plug is pulled from a socket. Bad electrical contacts not only produce hot wires and/or arcs, but can also cause a fire themselves.

● **Static electricity** is a possible risk, though the risk is reduced in a humid environment. A fully antistatic equipped theatre is ideal but difficult to realise, small measures such as using black rubber antistatic tubing for the breathing tubing and scavenging will reduce the risk.

● **Compression energy** - gas escaping from cylinder may ignite lubricants.

**Clinical practice**

**Keep the anaesthetic mixture confined to the apparatus:**

● **Avoid open mask anaesthesia**. If an open mask technique is used and the head is not covered by drapes, and the room is well ventilated, only an area extending for some 25-30cm around any part of the patient's head should be considered dangerous. Gases passing under the towels toward the diathermy are potentially very dangerous.

● **Intubate / LMA whenever possible**. Semi-closed breathing systems (e.g. the Penlon EMO vaporiser or Tri-Service apparatus) contain the ether vapour.

● **Ensure good theatre ventilation**. In a well-ventilated room, the mixture is rapidly diluted to a safe concentration once it leaves the expiratory valve of the breathing system or ventilator.

● **Use a scavenging system** connected to the expiratory valve of the breathing system or ventilator to carry the ether mixture outside through a theatre wall.

● **The "zone of risk"** of fire or explosion was described by the Association of Anaesthetists of Great Britain and Ireland in 1971. It is defined as an "**area extending 25 cm around any part of the anaesthetic apparatus...**" This is because leakage from the apparatus is always possible, but careful maintenance of the apparatus will help to reduce leaks. Potential sources of ignition must not be put in this area.

● **Diathermy** is safe to use with ether and air providing you are not working on the head and neck or lungs. There is a risk of a fire, but if it is not used near the head and neck or lungs, the risk of a fire is very small.

● **Oxygen and ether combinations**. Oxygen may be required to maintain the patient's oxygen saturation during anaesthesia (Appendix 2),. Under certain conditions a mixture of ether and oxygen can result in an explosion, compared to a mixture of ether and air which can burn. Therefore added oxygen and a diathermy is very dangerous. **Do not use ether and oxygen with diathermy**. You must either switch off the oxygen, or switch off the ether, or ask the surgeon to switch off the diathermy. Never risk an explosion it could kill you !!

**Minimize the flammability**

● Use as little supplementary oxygen as needed to maintain saturation.

● If possible use non-flammable agents.

● Store all flammable substances out of the room and in a safe place.

● Gas and petroleum burners should be kept out of the theatre - **no open flames or fires.**

● Avoid nitrous oxide.

● Do not use lubricants on reducing valves, pressure gauges or other parts connected with oxygen or nitrous oxide cylinders.

● Dilute any flammable or explosive mixtures, which escape if the air in the room is changed as often as possible - **plenty of fresh air. NOTE** - air conditioners like those used for home or office do not change air, in fact they may be a source of ignition.

**Prevent ignition sources**

● Keep monitors, other electrical appliances and instruments 1.5m above the floor and at a safe distance from head of the patient (25cm)

● Beware of diathermy! Both the electrode and foot switches should not be allowed into contact with anaesthetic gases. Remember these may infiltrate the towels toward the surgical field.

● Keep all unnecessary electrical apparatus out of the room. The oxygen concentrator can be placed at a distance from the anaesthetic machine and connected by any long tube of 1cm diameter. Alternatively it can be mounted on the wall, 1.5m from the floor.

● All equipment should be properly earthed at a single point (not the water pipe!) with cables of large diameter and not welded.

● Any patient ventilator should be flame/explosion proof.

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To our knowledge no recent textbook of Anaesthesia deals with this topic, nor have we been able to trace the subject of explosions related to Anaesthesia in Internet. We are indebted to:

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**Appendix 1- A reminder of basic chemical terms and facts**

$2\ H_2 + O_2 = 2\ H_2O + 116$kcal. That is two moles (2g) hydrogen ($H_2$) plus one mole (32g) oxygen ($O_2$) produces two moles (36g) water ($H_2O$) and 116kcal of energy.

**Atomic weight**: the weight of the atom of an element compared to the weight of an atom of hydrogen, which in effect becomes the base unit; e.g. the oxygen atom weighs 16 times the atom of hydrogen - the atomic weight of oxygen is 16. Other examples include carbon 12, nitrogen 14 and sodium 23.

**Molecular weight**: the sum of the atomic weights of a compound: e.g. the gaseous oxygen molecule is formed by two atoms and therefore its molecular weight is 16x2=32; nitrous oxide ($N_2O$): (14x2) + 16= 44; diethyl ether (C4H10O): (12x4) + (10x1) + 16= 74

**Mole**: the molecular weight expressed in grams: 1 mole of oxygen = 32g; 1 mole of Ether = 74g One mole of any substance contains the same number of molecules. One mole of any gas ideally occupies a volume of 22.4 litres at

**Normal temperature and pressure** ($0^oC$ or $273^o$Kelvin: 760mmHg).

Combustion: When combustion is complete, the following reaction occurs:
1 mole fuel + b moles oxygen = products ($CO_2$ and $H_2O$) and Energy (heat, light etc).
"b" is the exact number of moles of oxygen required to completely oxidise completely one mole of fuel

**Density**: Ether vapour has a density of 2.56 with respect to air

**Appendix 2 - The ether-oxygen dilemma**

Ether is still considered a very valuable agent for inhalation anaesthesia, because it is non-toxic, efficient, and inexpensive. It is therefore rightfully still widely used in many parts of the world. However there are difficulties. As with any anaesthetic, there is impairment in pulmonary function, which may require an inspired oxygen concentration above 21% (together with assisted or controlled ventilation). Therefore oxygen supplement may be required, which increases the possible danger of fire or explosion.

**Appendix 3- Azeotropic Mixture**

Halothane 66% and diethyl ether 34% mixed together form an "Azeotrope" or a mixture where the molecules of the components form loose hydrogen bonds and cannot be separated by distillation in spite of different vapour curves. The halothane/ether Azeotrope can be vaporized with a halothane vaporiser and clinically useful concentrations are similar to those of this agent or around 1.5%. Induction is reasonably quick and not unpleasant and recovery more prompt than with ether. Due to the ether in the mixture, the Azeotrope retains powerful analgesic and relaxant properties and like ether it gives excellent cardiovascular and respiratory conditions. It is not explosive, can be easily transported and stored and may burn in oxygen only at concentrations over 10 %. The halothane/ether Azeotrope is an excellent anaesthetic which combines the best of the two parent substances. It is surprising that it does not have the recognition it deserves. This is possibly due to the poor development of anaesthesia relevant for developing countries whilst anaesthetists in affluent countries are submerged by a profusion of new molecules.

# THE HALOTHANE/ETHER AZEOTROPE - A RECONSIDERATION

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Halothane and diethyl ether, the two anaesthetics most commonly used in developing countries, have the unusual property that they form an azeotrope when mixed in the ratio of about 2 parts halothane to 1 part ether. An azeotrope is, according to the Encyclopaedia Britannica, "... a mixture of liquids that has a constant boiling point because the vapour has the same composition as the liquid mixture... The components of the solution cannot be separated by simple distillation." Thus, when the halothane ether azeotrope (**HE**) is placed in an anaesthetic vaporizer, the proportion of halothane and ether that emerges will remain 2:1 for all dial settings and all rates of carrier gas flow. In effect, the azeotrope behaves physically as though it were a single compound.

The nature of the chemical bond between the two substances has not been determined with certainty. However, the facts that the volume of the mixture is slightly less than the sum of the volumes of its constituents, that there is a slight exothermic reaction when the components are mixed, and that the boiling point of the mixture is higher than either of its components all support the proposition that chemical bonding does occur.

The first report of **HE** for anaesthesia was by Hudon[1] in 1958. Notable studies favorable to **HE** include those by Dobkin et al.,[2,3] Wyant et al.,[4,5] Bengtsson et al.,[6] and Kalman, et al.[7-9] The most comprehensive available review of **HE** is in the medical thesis of Kalman (Studies on the halothane-diethyl-ether Azeotrope. Linkoping University medical dissertation No. 417, Linkoping, Sweden, 1994). Despite favorable reports, **HE** has fallen into disuse in developed countries, probably because of the lack of any commercial marketing, (unjustified) concerns over flammability, and because halothane itself became unpopular.

Pharmacologically, **HE** shares the properties of its component parts. Its organ system effects are described briefly below:

**Circulation**

Patients under **HE** anaesthesia exhibit exceptional hemodynamic stability, probably because the ether moiety stimulates sympatho-adrenal activity and decreases vagal tone, which together tend to offset the direct myocardial depression of ether and halothane. Blood pressure tends to be stable, unless the level of anaesthesia is very deep or the patient is severely hypovolemic. Cardiac rate is well maintained, and arrhythmias are rare.[5] Crossover experiments in dogs at 2% concentration of halothane found that cardiac output, blood pressure, and heart rate were better preserved when the dogs also received ether from the azeotrope.[3]

Based upon a clinical series of over 6000 administrations, Wyant[4] suggested that **HE** offers a wider margin of safety than halothane alone that "makes the azeotrope a more desirable agent in the hands of those less skilled than the specialist anesthesiologist." This was confirmed in ventilated pigs, where the median ratio of the lethal concentration to the effective anaesthetic concentration (MAC) was 3.0.8

**Respiration**

The respiratory stimulating effect of the ether component of **HE** tends to offset the depressant effect of the halothane component, so that spontaneous respiration is well maintained at clinical levels of anaesthesia,[6,9] with rather increased respiratory frequency. In our experience the use of a moderate dose of an opioid (e.g., pethidine 1mg/kg) slows-down the frequency, somewhat improving respiration. Any tendency for hypoxia due to reduced alveolar ventilation can easily be overcome with a small amount of supplement of oxygen. Spontaneous ventilation under **HE** without supplemental oxygen cannot be recommended. Furthermore, it is important to note that **HE**, like other volatile anaesthetics, abolishes the ventilatory response to hypoxia[9] and can cause intrapulmonary shunt if atelactesis is present.

**HE** gas induction is faster and more pleasant than with ether, behaving much like halothane alone. It is not irritating to the airway, and it relaxes bronchial muscles, as do its parent substances. Bronchial secretion is not increased. However, salivation is increased, but not as much as with ether or ketamine.

From the point of view of respiration, recovery from **HE** anaesthesia may be safer than with modern anaesthetics. The azeotrope appears to break down in the body, and halothane, having lower blood solubility than ether, is excreted more rapidly during the early phase of recovery.[7] The remaining ether tends to preserve respiratory drive and give analgesia into the early postoperative period, eliminating the need for opioids and their concomitant respiratory depression.

**Hepatic function**

No change in liver function has been observed in the early postoperative period after **HE**.[10]

**Anaesthetic Potency**

The minimum alveolar concentration (MAC) for HE in man was found to be 0.71 vol.% (which has about 0.47% halothane), versus 0.65 % for halothane alone, suggesting that the ether component acts synergistically with halothane.[7] Thus, at a given MAC fraction, **HE** is less expensive to use than halothane.

**Flammability**

Flammability of a vapor is not simply a physical property of the material, but rather is dependent upon the source of ignition used, the carrier gas(es), the geometric configuration of the test chamber, temperature, and other factors. Boivin[11] used an electric filament and reported that **HE** was nonexplosive in concentrations of 10.7% or less in oxygen. Later, Brown[12] used a higher-energy electric spark in a recognized standard test apparatus. He

concluded that **HE** is nonflammable in air for all practical purposes, and that its lower limit of flammability in oxygen is 7.25%. Raventos, using a similar apparatus found a lower limit of 8.0% in oxygen.[13] The fact that ether alone has a lower limit of flammability of in oxygen of 2.1% suggests that the presence of halothane in the mixture does little to moderate the flammability of the ether moiety (e.g., 7.25% **HE** contains about 2.4% ether).

The flammability tests indicate that the OMV[6] and plenum-type vaporizers should not produce flammable concentrations of the mixture at any dial setting. However, caution should be used with non-calibrated vaporizers, they have not been formally tested as to the output concentration obtained at maximal settings. Like other anaesthetics, **HE** is more flammable in nitrous oxide mixtures than in oxygen/air mixtures[13]

Comparative properties of the azeotrope and its components are summarized in the table below.

**Tips for use of HE**

The only additional equipment required is a graduated measuring cup or cylinder. Although the azeotrope is nonflammable in air, the ether component is flammable, so the azeotrope should be prepared in a well-ventilated area, away from sources of ignition. A large quantity can be prepared in advance, because the azeotrope is stable for at least 4 months when kept out of bright light.[11] It can be administered with the OMV,[6] any plenum-type vaporizer designed for halothane, or a non-calibrated Boyle's Bottle-type of vaporizer. Because the OMV lacks temperature compensation, the concentration emerging from it at any given dial setting will diminish by about half over two hours.[6] **HE** should not be placed in an EMO ether vaporizer, as the thymol from the halothane will ruin it.

Inhalation induction with **HE** is neither unpleasant nor unduly long and is similar to that of halothane. With any calibrated vaporizer, the dial setting should be increased progressively to a dial setting of 4-5 and kept there for some minutes after loss of consciousness, carefully attempting from time to time to insert the laryngoscope if intubation is to be done without a muscle relaxant. Surgery can commence after 5-10 minutes.

The typical dial setting for maintenance is 1 to 2 (corresponding roughly to 0.9 to 1.5% or 1.3 to 2 MAC). However, owing to the wide margin of safety, higher concentrations can be used, when necessary.

Muscle relaxation is fairly good, probably due to the relaxing properties of ether. Nondepolarizing muscle relaxants are potentiated, and can be used sparingly for abdominal surgery and not at all for extra-abdominal interventions. The azeotrope, like its component parts, will relax uterine muscle and should be used only in low concentrations during Caesarean section.

In the absence of opioids, muscular tone, blood pressure, pupil size, eyelid reflex, spontaneous respiration depth and rate (if allowed) all help in assessing the depth of anaesthesia.

*Table 1*

| | Halothane | Diethyl Ether | Azeotropic Mixture |
|---|---|---|---|
| Molecular weight | 197.4 | 74.1 | 158 (calculated) |
| Boiling point @ 760 mmHg or 100 kPa (°C) | 50.2 | 34.6 | 52 |
| Vapor pressure @ 20°C (kPa [mmHg]) | 32.1 [243] | 49.1 [373] | 28.4 [216] |
| Liquid density | 1.86 | 0.72 | 1.48 |
| MAC (% v/v) | 0.65 - 0.75 | 2.0 | 0.71 |
| Working concentration (% v/v) | 0.85 - 1.5 | 3 - 7 | 1.0 - 1.5 |
| Flammability | No | 2% to 80% in O2 | 7% to 67 % in O2 |
| | | 2% to 36% in Air | not in Air |
| Mask induction | Acceptable, quick | Unpleasant | Acceptable, not long |
| Clinical signs of depth | No | Yes | Similar to ether |
| Induction/recovery time | Short | Long | Short |
| Awake analgesia | +/- | +++ | ++ |
| Muscular tone | — | Decreased | Decreased |
| Cardiovascular function | Decreased | Stimulated | Stimulated |
| Respiratory function | Decreased | Maintained | Maintained |

Awakening time, even after a long procedure, is about 10-15 minutes.[6] Postoperative nausea and vomiting are said to occur in about 10% if patients.[4]

## Conclusion

The azeotrope of halothane and ether appears to share many of the virtues of its constituent parts, while ameliorating their undesirable properties. It is less expensive than halothane alone and may be a safer in the hands of less experienced anaesthetists. It should be considered for routine use in developing countries.

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# ANAESTHESIA FOR HIP REPLACEMENT

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## Arthritis of the Hip

In the simplest terms, the hip is a ball and socket joint. The ball is formed by the upper end of the femur, and the socket by part of the pelvis called the acetabulum. The ends of the bones are covered with a smooth layer of cartilage, which allows nearly frictionless and painfree movement. When the cartilage is damaged by arthritis, joints become stiff and painful. Arthritis may affect 2% of the population and causes include:

- Osteoarthritis (OA) - A degenerative disease affecting the articular surface of one or more joints, usually due to aging or repetitive joint trauma. Some populations show an extremely high incidence of OA. An example of endemic OA includes Mseleni Joint Disease, found in the Tsonga people of East Africa, and in Mseleni, Northern Kwazulu-Natal, South Africa.
- Inflammatory - rheumatoid arthritis is characterized by an immune-mediated joint destruction with chronic and progressive inflammation of the synovial membranes
- Infective
- Congenital dislocated/shallow hip

Treatment of arthritis includes:

- Anti-inflammatory and analgesic treatment.
- Physiotherapy to maintain movement and flexibility.
- Joint replacement surgery if arthritis is severe and significantly affects activity.

## Hip Replacement Surgery

Joint replacement is a common surgical procedure with a high success rate. Total hip replacement (THR) involves:

- dislocation and removal of the femoral head.
- reaming of the acetabulum and insertion of a prosthetic plastic or ceramic acetabular cup.
- reaming of the femur with insertion of a femoral component (metal or ceramic femoral head, and metal stem) into the femoral shaft (with or without cement). Metals used include stainless steel, alloys of cobalt and chrome, and titanium. Wear-resistant polyethylene (plastic) is used for socket replacement. Bone cement (with or without antibiotics) may be used to anchor the prosthesis into the bone. Joint replacements implanted without cement are designed to fit and lock into the bone directly.

## Hip Replacement Options

- **THR, also called total hip arthroplasty (THA)**. The hip socket and ball of femur is replaced, with a metal or ceramic ball, on a stem fitted into a cup with a plastic liner.
- **Revision of Hip Replacement**. A re-operation on a previously performed hip replacement which has failed or become loose. Part or all of the previous implant is removed and replaced with a new one. This may be a prolonged operation with significant blood loss.
- **Bilateral Hip Replacement**. Both hips are replaced simultaneously. This operation has a longer recovery time and requires a higher level of fitness preoperatively.
- **Hip resurfacing (Birmingham hip resurfacing; BHR)**. The ball of the femur is 'resurfaced' with a metal shell rather than being removed and replaced. This preserves more of the patient's own bone and produces a more anatomical load bearing on the femur. The socket is replaced as in a traditional replacement procedure, without cement.
- **'Girdlestone' Procedure**. Usually if revision hip replacement is not an option, the loosened prosthesis is removed altogether. Scar tissue develops between the upper end of the femur and the hip bone and allows the person to move with little pain. However, the femur is shortened, the leg is weak, and walking with the aid of a stick or crutches is usually necessary.

## Preoperative Assessment For Hip Replacement Surgery

There is no single standard anaesthetic. An anaesthetic plan should be formulated that will optimally accommodate all aspects of the patient and planned surgical procedure (primary THR versus complex revision THR). Assessing patients preoperatively includes a pertinent history, a physical examination, and any indicated laboratory tests.

Most patients presenting for hip surgery are elderly, and somewhat frail. A thorough preoperative assessment is necessary, but indiscriminate cancellation or delay is inappropriate.

## History

- Current problems and activity - What type of patient are we dealing with? Young/old/active/inactive/lucid? Personality, activity and age often dictate the type of anaesthetic.
- Underlying medical fitness, and review of organ systems - especially in the elderly. Enquire about respiratory and cardiovascular problems. Debilitating and limited joint mobility prohibit assessment of exercise tolerance, potentially masking underlying coronary artery disease (CAD) and lung problems.
- Drug history - warfarin, aspirin, nonsteroidal anti-inflammatory drugs (NSAIDS - have side-effects of GIT bleeding, renal toxicity and platelet dysfunction) has implications for the anaesthetist with regard to neuraxial blocks, and also the surgeon. Some patients are on immunosuppressants and steroid supplementation may be required. A high proportion of elderly patients are on cardiovascular treatment, particularly beta-blockers and ACE-inhibitors.

● Allergies - enquire specifically about antibiotic allergies, and check the type of cement being used. Aseptic loosening of cemented THRs has been linked to allergies to some of the components of the cement.

● Surgical history, previous anaesthetics - anaesthetic records may detail difficulties in spinal/epidural insertion, airway assessment with intubation, and other problems encountered.

● Family history

**Examination**

● Ideally blood pressure should be optimised preoperatively. Although poorly controlled hypertension increases the likelihood of perioperative silent myocardial ischaemia, direct evidence of a worse outcome in these patients is lacking. However, if there are associated cardiac risk factors, these need to be investigated appropriately preoperatively.

● General - weight (Body Mass Index) and shape of back may determine type of anaesthetic used. Pallor, dehydration and oedema will guide investigations needed and necessary interventions required preoperatively.

● The cervical spine should be carefully assessed for pain during movement, and restricted movements. Under anaesthesia and particularly during intubation attempts, excessive movement of the abnormal neck must be avoided. In OA the spine may be involved causing nerve root compression. In RA the cervical spine and temperomandibular joint can be involved. Atlantoaxial subluxation, which can be diagnosed radiologically, may lead to protrusion of the odontoid process into the foramen magnum during intubation, compromising vertebral blood flow and compressing spinal cord or brainstem. Intubation should be performed with neck stabilization, and in some patients an awake intubation technique will be required. Involvement of the TM joint can limit jaw mobility. Regional anaesthesia may prove practical in these patients.

● Systemic review - Heart, lungs, extremities and neurological examination. In RA multiple joints including small joints of the hands, wrists and feet may be involved in patients with RA, therefore insertion of invasive catheters and even gaining IV access are a challenge. Make sure the patient can tolerate lying flat if a simple spinal is used. Neurological assessment is also important regarding confused patients (lying still).

**Laboratory Evaluation**

Most patients are elderly and should have as routine:

● Full blood count or haemoglobin level

● Creatinine and electrolytes (if available)

● ECG for symptomatic patients, and routinely over 60 years

● Group and save or two units packed cells crossmatched depending on the base-line Hb, type of hip procedure, size and weight of the patient. (Sometimes only 'O negative' blood available in a rural set-up)

Other tests may be indicated:

● Clotting studies (if on Warfarin)

● Blood gas/ lung function tests

● Chest x-ray

● Urinalysis

● Blood glucose

**Choice of Anaesthetic**

Hip replacement can be performed under general, spinal or epidural anaesthesia, and a combination of techniques is often used. An anaesthetic plan should be made for each patient taking account of the patient's physiological state, including any medical and surgical illnesses, the planned procedure, drug sensitivities, previous anaesthetic experiences, and psychological makeup.

Recent reviews show that anaesthetic technique makes no difference to operative mortality[1,2,3]. In the recently published Cochrane Database Systemic Review, Choi et al[4] reviewed the evidence comparing the efficacy of epidural analgesia with other postoperative modalities for pain relief following hip or knee replacement. The authors conclude that epidural analgesia may be useful for postoperative pain relief following major limb joint replacements, however, the benefit may be limited to the early (four to six hours) postoperative period. The current evidence is insufficient to draw conclusions on the frequency of rare complications from epidural analgesia, postoperative morbidity or mortality, functional outcome or length of hospital stay.

These reviews suggest that a variety of appropriate anaesthetic techniques can be used. The choice will depend on a number of factors including patient choice, the skills of the anaesthetist, the surgical procedure, the facilities, including postoperative care, funds available and location of the hospital.

The advantages of regional techniques include:

● Reduced blood loss, reducing the need for transfusion

● Avoids effects of general anaesthesia on pulmonary function

● May avoid intubation

● Good early postoperative analgesia

● Reduced incidence of postoperative venous thrombosis and pulmonary embolism (sympathectomy-mediated increase in blood flow, and amelioration of the hypercoagulable state associated with surgery)

● Lower cost

● Simple technique in rural set-up

Spinal and epidural anaesthesia have proved to be extremely safe when correctly managed; however, there is still a risk of complications. Adverse effects range from self-limiting back pain to debilitating permanent neurological deficits and even death. The anaesthetist must therefore have a good understanding of the anatomy involved, be thoroughly familiar with the pharmacology and toxic dosages of the agents used, diligently employ aseptic technique, and anticipate and quickly treat problems.

The advantages of general anaesthesia include:

- Easier for patients that cannot tolerate lying flat
- Safer in patients with fixed output states like aortic stenosis, where maintenance of normal sinus rhythm, heart rate and intravascular volume is critical. (Remember these patients need cardiology review preoperatively. Echocardiography can determine the size of the stenosed orifice, the transvalvular gradient and peak blood flow velocities distal to the obstruction.)
- May be safer for patients with ischaemic heart disease as stable cardiovascular conditions may be easier to maintain
- Patient preference

**Anaesthesia**

Anaesthesia should be planned depending on the surgery which may be Primary THR, revision THR, bilateral THR, resurfacing technique or 'girdlestone' procedure.

**Premedication**

Consider relevant premedication if necessary. Often explanation and reassurance is all that is needed. Pre-emptive analgesia (paracetamol or NSAIDS) if appropriate.

**Monitoring**

All patients should be fully monitored with blood pressure (NIBP usually, direct arterial is indicated in high risk patients undergoing difficult surgery), pulse oximetry and ECG. Capnography, inspired oxygen, volatile agent analysis and airway pressure monitoring are indicated for a general anaesthetic.

**Intravenous lines**

A reliable 14-16G cannula should be inserted. If a lateral position is anticipated, use the lower arm, as this leaves the upper arm free for a BP cuff or direct arterial pressure measurement. CVP is indicated in high risk patients undergoing revision surgery.

**Warmth**

Keep the patient warm with a forced air warmer or equivalent and remember to warm IV fluids. Maintaining normal body temperature during hip replacement surgery has been shown to reduce blood loss.[5]

**Spinal anaesthesia**

A simple THR is particularly amenable to spinal anaesthesia and this can be supplemented with sedation or general anaesthesia, a decision which may be partly influenced by the patient's request.[6]

- Check for any contraindications to SAB.
- Preload with IV fluids prior to performing a spinal. Monitor blood pressure closely.
- Single-shot spinal (2.5-3.5ml bupivicaine 0.5% plain) under sterile technique. In 'younger' patients diamorphine (0.25mg) may be added for more prolonged anaesthesia. 10-25mcg fentanyl is an alternative.
- Target-Controlled-Infusion (TCI) propofol with a target of 1.0-3mcg/ml is useful sedation for the lateral position, using facemask supplemental oxygen. However, some patients may be uncomfortable due to pain from arthritic shoulders and other joints. Intermittent doses of midazolam, cautious opioids or $O_2$-$N_2O$/isoflurane via the face-mask may be useful. On occasions induction of GA is required, using a LMA.
- For the supine position in a patient who wishes to be asleep during surgery, consider an LMA with a light GA to maintain the airway.
- The addition of intrathecal opioid helps cover the longer duration of surgery necessary for a more complex primary hip replacement. It is a suitable technique for up to 3 hours of surgery. Alternatively, or for longer cases, a combined spinal/epidural technique can be used.

**General Anaesthesia**

- GA (rather than sedation) may be combined with an epidural for any complex primary operation because of the prolonged surgical time. An LMA, or endotracheal tube and IPPV, may be considered. The epidural should be topped up incrementally to avoid the combination of a high spinal block and IPPV resulting in reduced venous return and hypotension.
- Using an epidural postoperatively will necessitate inserting a urinary catheter (which also helps monitor fluid balance). This is best performed at the time of surgery.
- A femoral 3:1 block or a psoas lumbar plexus block plus lateral cutaneous nerve of thigh block can be used to supplement GA if central neuraxial blocks are contraindicated.
- Aim to maintain blood pressure at an adequate level based on preoperative readings. In elderly patients with vascular disease hypotension should be treated immediately.
- Intra-operative antibiotic prophylaxis will be required.
- Ensure adequate IV loading prior to cementing of femoral component. Hypotension can occur on pressurisation of the cement into the femur, usually due to vasodilatation and direct myocardial depression from the monomer. The transient hypotension does not correlate with the level of monomer in the circulation, but with deficit in blood volume.

**Postoperative**

- The surgeon usually prefers the patients to be placed on their bed in the supine position with the legs abducted using a pillow to prevent dislocation of the prosthesis. Anaesthesia techniques which lead to rapid recovery of airway control and patient cooperation is therefore an advantage.
- Patients are usually mobilized at 24-48 hours and simple IM/subcutaneous opioids with regular paracetamol or NSAIDs are usually sufficient for postoperative analgesia in a simple THR. If an epidural has been inserted, a postoperative infusion can be used but needs to cease prior to mobilization. PCA is a suitable alternative if pain relief is needed for an extended period.

**Special considerations**

- To some extent position (lateral or supine) dictates anaesthetic technique. Sedation with an oxygen facemask is much simpler in the lateral position where the airway is better maintained. If supine, then sedation should either be light enough to maintain airway reflexes or anaesthesia with an LMA should be considered.

● Blood loss varies with different types of bone structure and levels of inflammation. It is also affected by anaesthetic technique. The average loss in a simple THR is 300-500 ml. A similar amount may be lost in the drain and tissues postoperatively. Blood transfusion is relatively uncommon during surgery in patients with an adequate preoperative Hb. Group and saved serum is acceptable if cross-matched blood can be provided within 30 minutes. The Hb should be checked 24 hours postoperatively, and treated with either transfusion or iron supplements if indicated. The decision to transfuse is multifactorial and includes general fitness, continuing surgical losses, and local practice. In complex revision hip surgery perioperative blood transfusion is frequently required and blood loss can be substantial. Two units of cross-matched blood should be available in theatre with the ability to obtain more within 30 minutes. (Blood recovery and autologous transfusion using a 'Bratt' device or similar is often practical.) These complex hip procedures should not be done in a rural set-up. Oxygen therapy is advisable in most patients overnight and for those with cardiorespiratory disease 48 hours as nasal spectacles 2-3lpm.

**Life-threatening intraoperative complications**

Bone cement implantation syndrome - Methylmethacrylate (MMA) cement interdigitates within the interstices of cancellous bone, and strongly binds the prosthetic device to the patient's bone. Mixing polymerized MMA (PMMA) powder with liquid MMA monomer causes polymerization and cross-linking of polymer chains. This exothermic reaction leads to cement hardening and expansion against prosthetic components. The resultant intramedullary hypertension can cause embolization of fat, bone marrow, cement, and air into the femoral venous channels. The residual monomer can also cause vasodilatation and a decrease in systemic vascular resistance, thought to be the cause for the transient hypotension often seen with cement insertion. The release of tissue thromboplastin may trigger platelet aggregation, microthrombus formation in the lungs, and cardiovascular instability as a result of circulation of vasoactive substances.

The clinical manifestations of this syndrome include:

- Hypoxia (increased pulmonary shunt)
- Hypotension
- Dysrhythmias (including heart block and sinus arrest)
- Pulmonary hypertension
- Decreased cardiac output.

Strategies to minimize the effects of this complication include:

- Increase inspired oxygen concentration prior to cementing
- Maintaining normovolaemia, monitor blood loss carefully
- Surgeons vent the distal femur to relieve intramedullary pressure
- Use uncemented femoral component

**Perioperative haemorrhage** - a revision THR may be associated with significant blood loss. Blood loss depends on many factors including the experience and skill of the surgeon, the surgical technique used, and the type of prosthesis chosen. Some ways of decreasing intraoperative bleeding include:

**Summary of hip replacement anaesthesia**

| | **THR** | **Revision of THR** |
|---|---|---|
| Procedure | Prosthetic replacement of femoral head and acetabulum | Revision of previous THR - may include one or both components |
| Time | 2 hours | 2-6 hours |
| Postoperative pain | +++ | +++/++++ |
| Position | lateral or supine | lateral or supine |
| Blood loss | 300-500 ml, G&S | 1 litre, occasionally considerably more, crossmatch 2 units |
| Practical techniques | Spinal, with or without sedation or GA +LMA; GA + ETT with nerve block or epidural or opioids | Epidural or combined spinal/ epidural with sedation or GA/LMA, or IPPV + ETT + epidural or opioid<br>Arterial line +/ - CVP may be indicated for complex revision or high risk patient. |

● Avoiding hypertension and tachycardia during anaesthesia

● Regional anaesthesia (may be due to vasodilatation of the venous and arterial vascular systems leading to redistribution of blood flow)

● Maintaining normal body temperature

**Thromboembolism** - Venous thromboembolism is a significant cause of morbidity and mortality following hip-replacement surgery. Strategies minimizing the risk:

● Regional anaesthesia (spinal or epidural)

● Intermittent leg-compression devices

● Low-dose anticoagulant prophylaxis - If central neuraxial blockade is planned, ensure that the final preoperative dose is timed appropriately. Bleeding and compression neuropraxia is a potential complication of regional anaesthesia in patients who are anticoagulated or with clotting abnormalities. The recommendations allow a 12 hour interval between low molecular weight heparin and epidural/spinal injection. Avoid any further dose for 4 hours post block. This also applies for removal of epidural/spinal catheters.

**Reaction to the antibiotic in Antibiotic-loaded bone cement (ALBC)**

Commonly used antibiotics in cement include:

| | |
|---|---|
| Tobramycin | Cefazolin |
| Gentamycin | Cefotaxime |
| Vancomycin | Cefamandole |
| Ticarcillin | Erythromycin |
| Nafcillin | Clindamycin |
| Cefalothin | |

ALBC is extremely rare and

● Adverse reactions are lower than those among patients receiving systemic antibiotics[7]

● Some surgeons believe that the primary reasons for avoiding the indiscriminate use of ALBC include the occurrence of an allergic or toxic reaction to the antimicrobial agent and emergence of antibiotic-resistant bacteria[8]

**An example of THR surgery in rural South Africa**

On 8 August 2003, Dr Victor Fredlund from Mseleni Hospital in Northern KwaZulu-Natal, RSA, received the Pierre Jacques Award, the Annual Rural Doctor of the Year Award. Dr Fredlund has been working at Mseleni Hospital since 1981 and has been Medical Superintendent there since 1985. A notable achievement has been the establishment of a programme of THR surgery for the local community.[9]

Mseleni Joint Disease (MJD) is a particularly disabling form of destructive OA which occurs in the Mseleni area, creating the necessity for hip replacements in many people. In view of the impossibility of getting large numbers of patients into a programme for hip replacement surgery at the tertiary referral centre in Durban, 350 km away, Dr Fredlund established a programme for hip replacement surgery at his rural district hospital.

MJD affects 1 in 2 women, and by the age of 50, 50% of the 75,000 people who live in the area suffer from some form of arthritis, which usually begins causing pain in their twenties. In an area where mobility is essential, and walking is the only means of transport, it is vital that these patients have access to surgery.

There are almost 900 patients on the MJD clinic register, but Fredlund says there are probably about 2000 more people living in the community who could benefit from the operation. Fredlund has performed about 200 operations, and his postoperative results compare favourably to those from the most advanced hospitals worldwide, with only 1% developing post-operative complications. Because of the cost, and lack of resources, Mseleni does not offer revision therapy, but there is the option of a 'Girdlestone' procedure. Spinal anaesthesia is the safest and most affordable option of anaesthesia.

The Mseleni Hip Clinic is run jointly by the South African Red Cross and the Department of Health, and the hip replacement programme is supported and sponsored by various means. The SA Red Cross Air Mercy Service (AMS) facilitate travel for the dedicated and committed teams of volunteer orthopaedic surgeons and anaesthetists who regularly perform hip replacement operations for this impoverished community.

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# LARGE AIRWAY OBSTRUCTION IN CHILDREN - PART 1: CAUSES AND ASSESSMENT

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Opening and maintaining the airway is fundamental to the treatment of all emergency situations in paediatrics, as in adults. All resuscitation algorithms start with ABC (Airway, Breathing, Circulation) and must be qualified in trauma to include cervical spine control. The commonest cause of paediatric airway obstruction is still the child with depressed conscious level who is not positioned properly or whose airway is not opened adequately by Basic Life Support manoeuvres. Airway foreign bodies are also common and may need rapid intervention. The pattern of infective causes of airway obstruction has changed since the introduction of vaccination programmes against Haemophilus influenzae type B. There has been a marked reduction in the incidence of epiglottitis, with a relative predominance now of viral croup and bacterial tracheitis, usually caused by Staphylococcus aureus.

## Why are Children at Increased Risk from Airway Obstruction?

There are anatomical, physiological and developmental reasons for children to be particularly susceptible to airway obstruction.

The nares, upper and lower airways are smaller in absolute terms in children. Resistance to air-flow (and thus the work of breathing) increases during quiet, laminar flow breathing in inverse proportion to the fourth power of the radius. A small decrease in radius of the airway increases markedly the resistance to breathing. This is even more noticeable during crying when air-flow is turbulent as resistance is then related to the fifth power of the radius. An example of this amplification effect in the upset child is to compare the increase in airway resistance when the airway narrows from 4mm to 2mm: in the quiet child the airway resistance increases 16-fold but when the child cries the increase is 32-fold.

The infant has a relatively large tongue and the larynx is situated relatively high in the neck, with the epiglottis at the level of C1 at birth, C3 in the infant and C6 from puberty. The laryngeal inlet appears to lie more anteriorly because of its high position. In the infant, the epiglottis is long and omega shaped and angled away from the long axis of the trachea. The larynx is funnel shaped and is narrowest at the level of the cricoid ring compared with the cylindrical adult conformation, which is narrowest at the level of the vocal cords. The airway is more compressible as cartilage support components are less well developed. Thus, extrinsic pressure from haematomas, neoplasms, vessels or enlarged heart chambers may more readily compress the airway. The collapse of the laryngeal inlet during inspiration is a feature of laryngomalacia and the collapse of the trachea and/or bronchi during expiration occurs in tracheo-bronchomalacia. If the intrathoracic airways are narrowed from whatever cause, the extra work of inspiration and of expiration leads to large swings in intrathoracic pressure and the potential for gas trapping and hyperinflation behind the obstructed airway causing further compression of small airways. During forced expiration efforts, the intrathoracic airways may collapse down exacerbating the gas trapping effect.

Hyperinflation and gas trapping also impair the function of the diaphragm which is unable to contract so efficiently from its optimal length. In infants the diaphragm has a smaller proportion of contractile elements and fewer fatigue resistant muscle fibres. The rib cage is cartilaginous and more compliant so the diaphragm anchor points are more mobile, leading to wasted inspiratory work and the clinical sign of recession of the chest wall. The chest wall shape in cross-section is circular in the infant compared with the eliptical shape in the older child and the ribs are attached perpendicular to the vertebral column compared with the acute angle of attachment in the older child. This means that the contribution of the "bucket-handle" movement of the rib cage to inspiration is minimal in small infants and also the elastic recoil effect is much less during expiration. The intercostal muscles and accessory muscles of inspiration are also less well developed. Thus, the small infant is very reliant on the diaphragm's contribution to inspiration and thus has few reserves when work of breathing has to increase. This is on top of the already high basal demands placed on the infant respiratory system by the higher rate of metabolism in early life.

The small absolute size of airways in children means that secretions, small airway constriction, oedema or compression more readily lead to airway closure and either atelectasis or gas trapping. The interalveolar pores and bronchoalveolar channels do not develop until the ages of 1 year and 8 years respectively so collateral ventilation is not an option around an area of obstruction by these mechanisms.

Thus anatomical, physiological and developmental factors conspire to make the child susceptible to airway obstruction and is exacerbated in disease states (Table 1).

## What are the Symptoms and Signs of Airway Obstruction?

### Signs of foreign body aspiration

Sudden onset of respiratory compromise associated with coughing, gagging, choking, aphonia or stridor suggests foreign body aspiration and this may necessitate emergency basic life support measures for the choking child. Signs of gas trapping behind a foreign body ("ball-valve effect") may be seen with hyper-resonance of the hemithorax, loss of percussion dullness over the liver, surgical emphysema, tracheal deviation and unequal breath sounds. It is particularly important to think of the possibility of pneumothorax and actively exclude it and treat it promptly. Pneumomediastinum, pneumopericardium and pneumoperitoneum may be seen. Collapse or consolidation of lobes or lungs with bronchial breathing, widespread crackles and expiratory wheeze may all be elicited depending on the cause, site and duration of the airway obstruction.

*Table 1: Some causes of large airway obstruction in children*

| |
|---|
| Depressed conscious level |
| Foreign body |
| Infection |
| ● Viral: croup, papillomatosis |
| ● Bacterial: epiglottitis, tracheitis, tonsillitis, abscess |
| ● adjacent to airway |
| Trauma |
| Thermal injury |
| Congenital abnormalities: choanal atresia, choanal stenosis, micrognathia, macroglossia, laryngomalacia, laryngeal web |
| Neoplasm: haemangioma, lymphoma, mediastinal mass |
| Peripheral neurological disease |
| Neuromuscular disease |
| Iatrogenic: subglottic stenosis, post-intubation stridor, neck haematoma |
| Anaphylactoid reactions |

**Signs of increased work of breathing**

The increased effort of breathing caused by airway obstruction may produce an increase in respiratory rate for age. A rate >50bpm in an infant and >30bpm in a child may be considered abnormal. However, of even more concern would be respiratory distress associated with a normal respiratory rate, bradypnoea or apnoeic spells which indicate decompensation and exhaustion.

A "see-saw" pattern of chest and abdominal breathing movements is seen in airway obstruction. This sign occurs earlier in younger infants. Recession of the intercostal spaces, subcostal region and sternum are also seen early in young infants and reflect the forces generated by vigorous contractions of the diaphragm and the compliant chest wall. If recessions are seen in older children they indicate severe airway obstruction. Use of the accessory muscles of inspiration (sternomastoids, scalene muscles and intercostals) is associated with tracheal tug, suprasternal and supraclavicular recessions and nasal flaring. Often the child sits upright and may adopt the "tripod" position to improve the mechanical advantage of these muscles in moving the chest wall and that of the diaphragm. In the small infant, an opisthotonic posture may be seen in airway obstruction and head bobbing is a sign of accessory muscle contraction in the infant. Lack of effort associated with deteriorating conscious level may indicate exhaustion and decompensation.

Expiratory grunting is often noted in infants with respiratory distress who are trying to generate auto-CPAP or expiratory braking at laryngeal level to maintain a residual lung volume at end expiration.

Stridor during inspiration is usually a sign of airway obstruction at supraglottic or laryngeal level but can occur in tracheal obstruction also. Stridor during expiration is usually a sign of intrathoracic airway obstruction. Prolonged expiration with wheeze is usually a sign of small airways obstruction as in bronchiolitis or asthma but can occur in large airway obstruction especially due to foreign body or if there is an underlying anatomical abnormality. The volume of stridor or wheeze does not correlate with the degree of airway obstruction. Indeed, the most ominous sign is the "silent chest" where obstruction is so severe that no gas flow is occurring.

**Signs of ineffective breathing**

Cyanosis, depression of conscious level, slow respiratory rate, the silent chest despite vigorous respiratory efforts or lack of adequate respiratory effort, apnoeic spells and bradycardia are most worrying signs of ineffective breathing.

**Secondary effects of airway obstruction**

Airway obstruction may produce hypoxaemia and hypercarbia. Tachycardia, sweating, confusion, restlessness, agitation, anxiety, dyspnoea, inability to speak, peripheral vasoconstriction with pallor or mottling, cyanosis, decreased conscious level, apnoeic spells and bradycardia may occur. Generalised convulsions may occur secondary to hypoxaemia. Hypertension and bounding pulses may be felt and pulsus paradoxus of greater than 20 mmHg may be elicited in older children. Chronic airway obstruction may cause chest wall abnormalities, pulmonary hypertension, right heart failure and obstructive sleep apnoea syndrome.

**What investigations are helpful?**

The assessment of the child in order to identify and manage airway obstruction is a clinical one. Do not try to examine the child's throat. The pulse oximeter is a very helpful, non-invasive and atraumatic monitor of arterial oxyhaemoglobin saturation and heart rate. However, the readings need interpretation in context with the clinical picture as they are affected by poor perfusion, movement, ambient light and carboxyhaemoglobinaemia (as may occur in smoke inhalation injury) and are less accurate at values below 70%.

Radiology should not be used in the child in extremis before intervening but in the less acute situation may help elucidate chest signs, such as pneumothorax, consolidation, collapse, foreign body, steeple sign in croup, mediastinal mass, etc. It should be carried out at the bedside. Lateral soft tissue neck films are seldom indicated but may show a foreign body, thumb sign of epiglottitis, prevertebral or a retropharyngeal abscess. CT and MRI scanning have no place in emergency management but are very helpful in cases such as haemangioma, mediastinal mass, or abscess adjacent to the airway.

The process of obtaining arterial, capillary or venous blood gases is likely to cause undue distress which will worsen airway obstruction. In the obtunded child intervention should be immediate and should not await blood gas results. For less severe cases, trends in carbon dioxide levels, pH and oxygen values may be helpful in guiding treatment and in reinforcing the need to intervene. Chronic airway obstruction leads to a respiratory acidosis which induces renal compensatory mechanisms with retention of bicarbonate and a metabolic alkalosis reflected in a high serum bicarbonate level and often a near normal arterial pH.

**Making the diagnosis**

Some features of the history and examination may be particularly helpful in pointing to a specific diagnosis and they are summarised in Table 2. These clinical signs are suggestive only as each disease process has a spectrum of severity. In individual cases it can be difficult to differentiate between the infective causes and foreign body aspiration. Severe tonsillitis or abscesses near the airway can produce similar symptoms and signs. Oedema of the face, periorbital tissues, tongue and peripheries is suggestive of angioneurotic oedema or anaphylactoid reactions.

**Part 2: Management**

**Importance of rapid clinical assessment, minimal disturbance and rapid intervention**

An assessment from the end of the bed with minimal disturbance should be possible in most cases with the child sitting in the parent's arms and the child should be allowed to adopt the posture in which they are most comfortable. Clinical assessment and a concise history as described in part 1 should allow identification of the need for intervention. A pulse oximeter probe is relatively atraumatic to apply. Gentle physical examination of the chest seeking actively for the important signs described in part 1 should be possible. In some cases, the need for immediate intervention will be obvious. In others, measures to buy time to enable experienced help to arrive may be appropriate. It is often stated that attempts at venous access should not be made as they will upset the child - this is a reasonable view. However, some argue that, in the less ill child and with topical local anaesthesia or ice analgesia of the skin and a skilled paediatric venepuncturist, this is not an issue. Some experienced paediatric anaesthetists are of the view that, in the hypercarbic, obtunded child with a hyperdynamic circulation, establishing venous access is relatively simple and appropriate. Despite this debate, the classical approach of not attempting venepuncture is recommended for the non-specialist anaesthetist.

*Table 2: Differentiation between croup, tracheitis and epiglottitis*

| | **Croup** | **Tracheitis** | **Epiglottitis** |
|---|---|---|---|
| Cause | Viral | Staphylococcus aureus Streptococcus | Haemophilus influenzae B |
| Age | 6m - 3y | Any age | 2 - 6y |
| Onset | Gradual | Gradual | Sudden |
| Pyrexia | Mild | >38 | >38 |
| Abnormal sounds | Barky cough, stridor | Barky cough, stridor | Muffled, guttural cough |
| Swallowing | Normal | Difficult | Very difficult with drooling |
| Posture | Recumbent | Sitting | Tripod position |
| Facies | Normal | Anxious | Anxious, distressed, toxaemic |

**Severity scoring system**

A scoring system for croup (table 3) is helpful in assessing severity, response to therapy and of the need for intervention.

| *Table 3: Croup score* | | | |
|---|---|---|---|
| | 0 | 1 | 2 |
| Breath sounds | Normal | Harsh, rhonchi | Delayed |
| Stridor | None | Inspiratory | Insp. + Exp. |
| Cough | None | Hoarse cry | Bark |
| Retractions/ flaring | None | Flaring + suprasternal retractions | Flaring + suprasternal + intercostal retractions |
| Cyanosis | None | In air | In 40% oxygen |

**When is immediate intervention required to open the airway?**

Immediate intervention is needed in the choking child or if the child is apnoeic or exhausted and making ineffective respiratory efforts. A silent chest or no stridor (either in the child making maximum effort to breath or in the exhausted child) are very sinister indications of complete airway obstruction. The simultaneous treatment priorities are oxygenation, opening the airway, improving failing respiratory efforts and relieving tension pneumothorax.

**Can I "buy time" prior to intervening to secure the airway?**

Steroids improve airway patency in croup, haemangioma, lymphoma and some mediastinal masses. They can be given orally, parenterally or by nebuliser. Prednisolone 4mg/kg orally, or dexamethasone 0.6mg/kg intravenously or intramuscularly, or budesonide 1-2mg by nebuliser are favoured as initial therapy with maintenance by repeated nebuliser therapy or oral prednisolone or parenteral dexamethasone at one quarter of the initial dose every 8-12h for up to 48h. Prompt administration of steroids often pre-empts the need for intubation in most cases of moderate or severe croup.

Nebulised adrenaline 1:1000 standard solution at a dose of 0.5 ml/kg (maximum 5ml) diluted if necessary with 0.9% saline to a total volume of 5ml will give a dose of 2-5mg in most cases. It reduces mucosal oedema and acts very rapidly but when stopped may give rise to a rebound worsening of airway obstruction. It is a useful temporising measure. ECG monitoring is recommended although dysrhythmias are seldom a problem.

CPAP - Continuous positive airway pressure acts as an effective splint for the collapsible, compressible paediatric upper airway and can be delivered to infants and children by a well fitting facemask (especially using the Jackson-Rees T-piece circuit) and, in babies, by nasal cannulae, nasal prongs or a nasopharyngeal airway. It is a very useful measure particularly during the preparation phase prior to intubation and during inhalational induction of anaesthesia. It can be very useful in the management of chronic airway obstruction. A development of CPAP is bi-level CPAP or BIPAP; it is becoming increasingly popular in the management of children.

Prone position +/- nasopharyngeal airway can be useful in babies with congenital upper airway abnormalities where the tongue is relatively large eg hemifacial microsomia, Pierre-Robin syndrome, or Treacher-Collins syndrome. The tongue falls forward from the posterior pharyngeal wall and often improves the airway. In conjunction with nasal CPAP and /or a nasopharyngeal airway, the child may improve markedly with this simple manoeuvre.

Helium is less dense than air or oxygen and gas flow tends to be more laminar which reduces the work of breathing. However, it is not readily available, is expensive and dilutes the inspired oxygen concentration. Breathing a helium-oxygen mixture may be helpful in buying time.

**How should I intervene to open and secure the airway?**

The algorithm in Figure 1 is a useful guide. If possible, call for expert help.

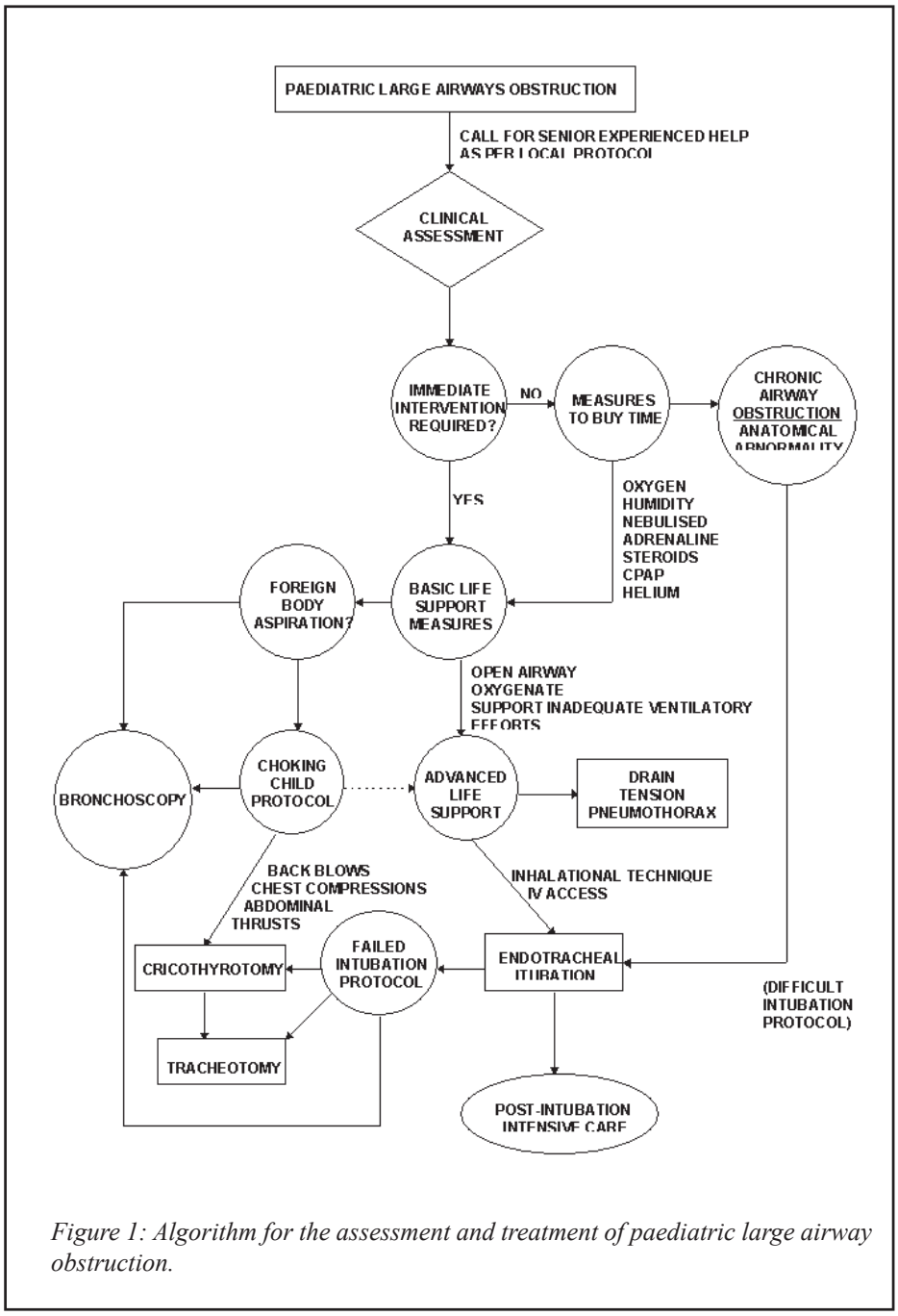


*Figure 1: Algorithm for the assessment and treatment of paediatric large airway obstruction.*

**Basic life support manoeuvres and the choking child**

Head tilt, chin lift and jaw thrust are the first basic steps. Physical methods of clearing the airway should only be used if the diagnosis of foreign body aspiration is clear and dyspnoea is increasing rapidly or apnoea has occurred. Do not use finger sweeps as this may push the foreign body further down the airway and may impact it in the laryngeal inlet. Do not try to examine the throat. In infants, immediately carry out five back blows with the heel of the hand with the infant lying prone and head down along your arm which should be resting along your thigh. If the obstruction remains, turn the baby supine and give five chest thrusts as for cardiac compression but more slowly and repeat airway opening manoeuvres, expired air ventilation and cardiac compressions as appropriate. In older infants and children, five back blows with the child prone across the lap and up to five abdominal thrusts exactly in the midline with the child standing, kneeling, sitting or supine should be used.

**Advanced life support manoeuvres**

Oxygen 100% should be administered by self-inflating bag and mask or anaesthetic T-piece circuit, depending on familiarity. The latter has the advantage that CPAP can easily be applied and the transition from spontaneous to controlled ventilation is simple. It is very important to actively exclude tension pneumothorax and, if present, to intervene with a needle, cannula or drain. This can be inserted under local anaesthesia in the conscious child giving careful attention to technique. Some distress may be caused to the child but this is transient and justifiable in the emergency situation. In the context of a tension pneumothorax secondary to acute airway obstruction, it is very wise to drain the pneumothorax prior to inducing anaesthesia. However, circumstances may dictate simultaneous intervention to drain the pneumothorax and induce anaesthesia to secure the airway if rapid decompensation is occurring.

**Endotracheal intubation technique**

It is better to have the child in an intensive care or operating theatre environment. Senior experienced staff only should attempt intubation unless circumstances mandate a life-saving attempt. Some recommend calling for assistance from the ENT team in case a bronchoscopy or tracheostomy is needed. A wide range of paediatric airway and intubation equipment should be to hand and checked.

Inhalational induction of anaesthesia with halothane (increasing gradually to 5%) in oxygen, starting with the child in the position in which they are most comfortable is recommended. Some experts now prefer sevoflurane (starting with 8%) in oxygen but experience in children with critical airway obstruction is limited and sevoflurane is intrinsically less potent making it difficult to gain a sufficient depth of anaesthesia to allow intubation. Remember, alveolar ventilation may be severely compromised and uptake of volatile agents may be very slow. It may take up to 10 minutes to reach a sufficient depth of anaesthesia. As the depth of anaesthesia increases and the patient is moved to the supine position, airway obstruction may occur and it is wise to change position gradually and to add CPAP to splint the airway open. Once the pupils become constricted and central, wait for a further 30 breaths and then perform laryngoscopy and orotracheal intubation. Change to a nasotracheal tube once the child is stabilised and fix securely with tape or a tube holder and fixation system (eg Tunstall, Burtles, Secure-ET).

If intubation is difficult or impossible, a number of techniques using the flexible fibreoptic or rigid bronchoscope are possible and very occasionally blind or retrograde intubation techniques may be employed. The use of a nasopharyngeal airway or the laryngeal mask airway may be helpful in certain cases. All these techniques are for the expert only.

In certain cases of impossible intubation where the child is rapidly deteriorating, the safest option may be to consider an emergency cricothyrotomy using a cannula, 3mm endotracheal tube connector, a T-piece or self inflating bag and oxygen source. Remember that there must also be a patent expiratory pathway to avoid barotrauma. Carbon dioxide levels will tend to rise with this technique. Jet ventilation is not recommended in children due to the risks of overpressure and barotrauma. In other cases, particularly of severe anatomical abnormalities, the safest option may be an emergency tracheostomy under mask anaesthesia and or local infiltration analgesia.

**When should I not intervene?**

If you are inexperienced with advanced life support measures in children, you should try to maintain oxygenation, airway patency and ventilatory support with basic measures until experienced staff arrive. Advanced life support interventions should be carried out by the most experienced staff present. However, you may have to intervene in the extreme situation to save the child's life. If it is available, it is vital that expert help is called as early as possible to manage children with airway obstruction.

**How should I manage the child after the airway is secured?**

It is important to ensure that the artificial airway is fixed securely and is correctly positioned. A post-intubation chest X-ray is useful for checking tube position and identifying lower respiratory tract or pulmonary parenchymal changes. Indications for controlling ventilation rather than allowing spontaneous ventilation are; small diameter endotracheal tube, child with signs of septicaemia, lower respiratory or lung disease, child who has sustained a hypoxic insult, very abnormal, inflamed or oedematous airway, traumatic intubation and need to transport the child to another hospital. Sedation, analgesia and muscle relaxation should be given as appropriate. Some children develop post-intubation pulmonary oedema which requires ventilatory support with PEEP and diuretic therapy.

Antibiotic therapy is indicated for likely organisms - the third or fourth generation cephalosporins are favoured with some preferring flucloxacillin for staphylococcal tracheitis. Duration of intubation varies widely from 18-24 hours for acute epiglottitis to days or weeks for those with lung involvement, severe disease and pre-existing congenital anomalies. Some children may require formal investigation of their airway eg endoscopy, foreign body removal or reconstructive surgery.

**Conclusion**

Large airway obstruction in children is a common emergency. If available, senior experienced help should be summoned immediately. Therapy is guided by clinical assessments. All consultants should be competent in the performance of basic life support measures for the choking child and for opening the airway, oxygenating the child and supporting inadequate ventilation. Measures to buy time can be very helpful in croup and where the airway anatomy is abnormal. Advanced life support measures are for experienced staff but you may have to intervene immediately to save a child's life. The results of correct management are excellent.

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## ASPECTS OF MYOCARDIAL PHYSIOLOGY

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**Introduction**

This is the first of two articles covering aspects of myocardial physiology which are important to candidates for Primary FRCA.

**Cardiac action potentials**

Action potentials (APs) are sequential changes in transmembrane potential that occur as a result of activity of ion channels, this results in the propagation of electrical impulses in excitable cells. The heart has a multicellular structure but behaves like a syncytium because the individual muscle cells communicate with their neighbours through gap junctions which provide low resistance pathways for easy movement of action potentials between cells. The cardiac action potential is much longer than those of nerve or skeletal muscle (~250ms compared with ~ 1-3ms) This is due to a prolonged plateau phase caused by calcium ions in cardiac muscles. Two types of action potential occur in the heart:

The fast response - found in heart muscle and Purkinje fibres (figure 1). The resting heart muscle potential of cardiac muscle and Purkinje fibres is ~ -90mmV (interior negative to exterior). An AP is initiated when the membrane is depolarised to a threshold potential (~-65mV). The initial depolarisation originates from transmission from an adjacent cell via gap junctions.

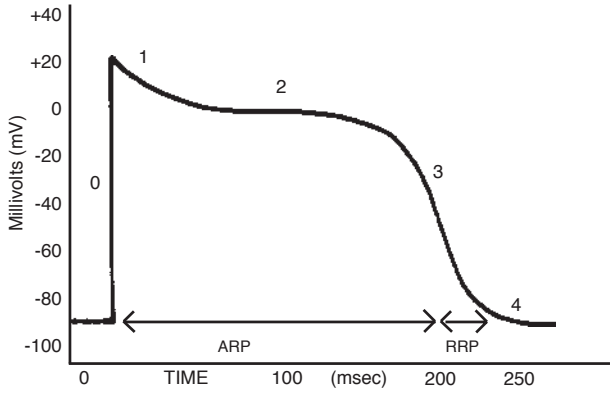


*Figure 1. The Purkinje fibre action potential*

Phase 0 - Rapid depolarisation - the inward current caused by opening of fast $Na^+$ channels becomes large enough to overcome the outward current through $K^+$ channels resulting in a very rapid upstroke. T-type (transient) $Ca^{2+}$ channels open at negative membrane potentials of -70mmV to -40mV causing $Ca^{2+}$ influx.

Phase 1 - Early incomplete repolarisation - due to inactivation of fast $Na^+$ channels and efflux of $K^+$ ions.

Phase 2 - Plateau phase - a period of slow decay mainly due to $Ca^{2+}$ entering the cell via L-type (L=long lasting) $Ca^{2+}$ channels which are activated slowly when the membrane potential is more positive then ~ -35mV. There is also slow closure/inactivation of some of the $Na^+$ channels. Reduced $K^+$ outward current continues. Calcium entry during the plateau is essential for contraction; blockers of L-type $Ca^{2+}$ channels (e.g. verapamil) reduce force of contraction.

Phase 3 - rapid repolarisation - $Ca^{2+}$ influx declines and the K+ outward current becomes dominant, with an increased rate of repolarisation.

Phase 4 - Electrical diastole - resting membrane potential is restored.

The slow response (figure 2) - found in pacemaker tissues; for example Sinoatrial and Atrioventricular nodes. These cells sponaneously depolarise and are said to have automaticity.

Phases 1 and 2 are absent. There is no depolarisation plateau.

Phase 4 - Pacemaker potential - The cells have an unstable resting membrane potential during phase 4; they gradually depolarise from ~-60mV to a threshold of ~-40mV due to a slow continuous influx of $Na^+$ ions and a decreased efflux of $K^+$ ions. A $Ca^{2+}$ current due to the opening of T-type (transient_ $Ca^{2+}$ channels completes the pacemaker potential.

Phase 0 - Depolarisation - when the membrane potential reaches threshold potential fast (L-type_ calcium channels open, causing $Ca^{2+}$ influx and an AP is generated.

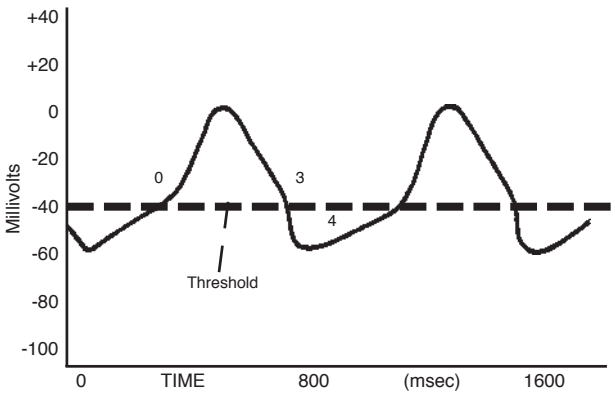


*Figure 2. The Sinoatrial node action potentials*

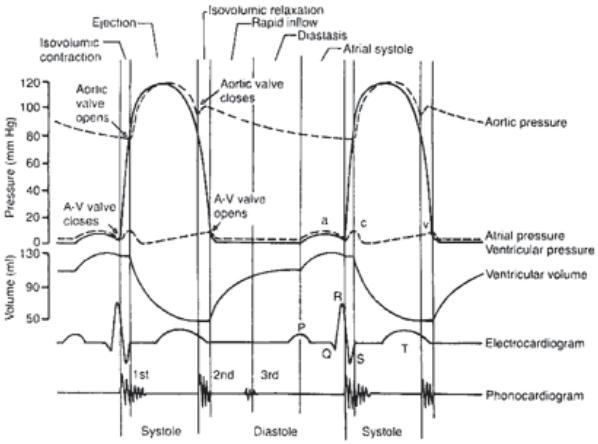


*Figure 3. Events of the cardiac cycle.*
*Re-printed from: Guyton AC, Textbook of Medical Physiology, 8th edition, © 1991 with permission from Elsevier.*

Phase 3 - Repolarisation - due to efflux of $K^+$.

Noradrenaline and adrenaline (mediated via β1 - receptors) increase the slope of phase 4 by increasing $Ca^{2+}$ influx, therefore increasing the heart rate. $Ca^{2+}$ influx also increases the force of contraction. Acetylcholine (mediated via M2 receptors) decreases the slope of phase 4 by increasing $K^+$ efflux and causing hyperpolarisation (increased negativity within the cells). This makes the conduction tissue much less excitable so it takes longer to spontaneously reach the threshold level. This results in a decrease in heart rate. The intrinsic rate of the SA node is 100 beats/minute however, vagal tone decreases this to ~70 beats/min.

**Refractory periods**

During the absolute refractory period (ARP) (figure 1) the cardiac cell is totally inexcitable. During the following relative refractory period (RRP) there is a gradual recovery of excitability. A supramaximal stimulus can elicit an AP in the RRP. This AP, however, has a slower rate of depolarisation, a lower amplitude and shorter duration than normal and, therefore, the contraction produced is weaker. Peak muscle tension occurs just before the end of the ARP and the muscle is halfway through its relaxation phase by the end of the RRP. The long refractory period protects the ventricles from too rapid a re-exitation which would impair their ability to relax long enough to refill sufficiently with blood. Unlike skeletal muscle, two contractions cannot summate and a fused tetanic contraction cannot occur.

**The Cardiac Cycle**

The cardiac cycle refers to the relationship between electrical, mechanical (pressure and volume) and valvular events occuring during one complete heartbeat.

**Passive filling (early diastole)**

The atria and ventricles are relaxed, ventricular pressure is zero. The atrioventricular (AV) valves are open and the semilunar valves are closed. Blood flows from the great veins into the atria and ventricles (from higher pressure to a lower pressure.) About 80% of ventricular filling occurs during this phase.

**Atrial contraction (late diastole)**

A wave of depolarisation beginning at the sinoatrial (SA) node, spreads across both atria, and reaches the AV node - the P wave of the ECG. The atria contract and atrial pressures increases producing the wave of the central venous pressure trace. Blood continues to flow into the ventricles and ventricular pressure increases slightly. The atrial contribution to ventricular fillinf increases as heart rate increases, as diastole shortens and there is less time for diastolic filling. Ventricular volume (EDV) = volume of blood in the ventricle at the end of diastole. Arterial pressure is at its lowest at this stage of the cycle.

**Isovolumetric ventricular contraction (early diastole)**

The action potential is conducted through the AV node, down the bundle of His, across both ventricles and ventricular depolarisation occurs - the QRS complex of the ECG. Ventricular contraction causes a sharp rise in ventricular pressure, and the AV vavles close (first heart sound) once this exceeds atrial pressure, preventing backflow into the atria. Ventricular pressure increases dramatically with no change in ventricular volume. During this initial phase of ventricular contraction pressure is less than in the pulmonary artery and aorta, so the outflow valves remain closed - the ventricular volume does not change. The increasing pressure causes the AV valves to bulge into the atria, resulting in a small atrial pressure wave - the c wave of the central venous pressure trace.

**Ejection (systole)**

The semilunar valves open as evntricular pressure exceeds aortic blood pressure. Approximately two thirds of the blood in the ventricles is ejected into the arteries. Flow into the arteries is initially very rapid (**rapid ejection phase**), but subsequently decreases (**reduced ejection phase**).

**Stroke volume** (SV) = volume of blood ejected from each ventricle in a single beat.

**Ejection fraction** = SV/EDV. Arterial blood pressure rises to its highest point - systolic blood pressure. During the last two thirds of systole before the AV valves open again, atrial pressure rises as a result of filling from the veins - the v wave of the central venous pressure trace. Active contraction ceases during the second half of ejection, and the ventricular muscler repolarises - the T wave of the ECG. Ventricular pressure during the reduced ejection phase is slightly less than in the artery, but blood vontinues to flow out of the ventricle because of momentum. Eventually the flow briefly reverses, causing closure of the outflow valve and a small increase in aortic pressure, the **dicrotic notch**.

**Isovolumetric relation (early diastole)**

The ventricles relax and the ventricular pressure falls below arterial blood pressure. this causes the semilunar valves to close - the second heart sound. The ventricular pressure falls with no change in ventricular volume. When ventricular pressure falls below atrial pressure, the AV valves open and the cycle begins again.

*Figure 4a. Re-printed from: Smith JJ, Kampine JP, Circulatory Physiology - The Essentials 3rd edition, with permission from Lippincott, Williams and Wilkins. Figure 4b re-printed from Aaronson PI, Ward PT, The Cardiovascular System at a Glance, 1st edition with permission from Blackwell Publishing Ltd.*

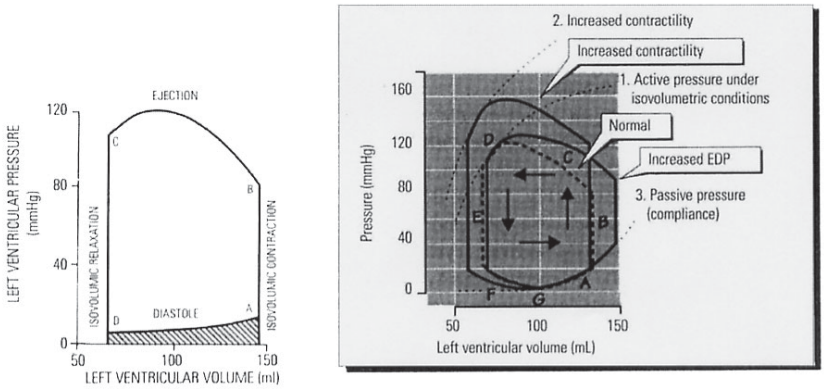


*Figure 4a. Left ventricular pressure-volume loop showing left ventricular volume and pressure changes during a single heart cycle in a normal adult at rest. Figure 4b. The pressure-volume loop is affected by the contractility and compliance of the ventricle, and factors that alter refilling or ejection (e.g. CVP, afterload).*

X descent of CVP trace - results from atrial relaxation and downward displacement of the tricuspid valve during ventricular systole.

Y descent of CVP trace - due to atrial emptying as the tricuspid valve opens and blood enters the ventricle.

**The pressure volume loop**

This represents the events of the cardiac cycle. the cardiac cycle proceeds in an anticlockwise direction. (A) End diastole, (B) aortic valve opening, (C) aortic vavle closure, (D) mitral valve opening. EDV and end systolic volume (ESV) are represented by points A and C respectively. The area close by the loop represents the stroke work (since work = pressure x volume).; The pressure - volume curve in diastole is initially quite flat, indicating that large increases in volume can be accommodated by only small increases in pressure. However, the ventricle becomes less distensible with greater filling, as evidenced by the sharp rise of the diastole curve at large intraventricular volumes.

**Coronary circulation**

The heart is supplied by the right and left coronary arteries. They arise separately from the aortic sinus at the origin of the ascending aorta, behind the right and left cusps of the aortic valve. The right coronary artery (RCA) runs forward between the pulmonary trunk and right atrium, to the AV sulcus. As it descends to the lower margin of the heart, it divides into posterior descending (interventricular) and right marginal branches. The left coronary artery (LCA) runs behind the pulmonary trunk and forward between it and the left atrium. It divides into the circumflex, left marginal and anterior anastomoses between the left and right posterior descending branches, but these are not enough to maintain perfusion if one side of the coronary circulation is acutely occluded. The LCA supplies mainly the left ventricle and septum and left atrium. The RCA supplies mainly the right ventricle and right atrium, SA node (in 60%) and AV node (in 80%). The 'dominant' supply to the heart is usually determined by the artery that forms the posterior descending and supplies the major arterial supply to the posterior inferior wall of the LV and to the AV node. The RCA is dominant in 70% of individuals, the LCA is dominant in another 20% and the flow delivered by each main artery is approximately equal in the remaining 10%

**Venous drainage**

Venous drainage is mainly via the coronary sinus and anterior cardiac vein which both empty into the right atrium. Some venous blood empties directly via the Thebesian veins and small venules into all heart chambers. Venous blood entering the left side of the heart will cause a small reduction in the $O_2$ content of systemic arterial blood.

**Control of the coronary circulation**

The heart at rest receives about 5% of the cardiac output. Coronary blood flow is ~250ml/min. $O_2$ extraction by the myocardium at rest is very high (65%) compared to other tissues (35%). Therefore, the myocardium cannot compensate for reductions in blood flow by extracting more oxygen from heamoglobin. Any increases in myocardial $O_2$ demand must be met by an increase in coronary blood flow. The three main factors influencing coronary flow are:

1. Mechanical, mainly external compression and perfusion pressure
2. Metabolic
3. Neural

**Coronary artery compression and blood flow**

Coronary blood flow is unique in that there is interruption of flow during systole (mechnical compression of vessels by myocardial contraction. Coronary blood flow occurs predominatly during diastole when cardiac muscle relaxes and no longer obstructs blood flow through ventricular vessels. Conversely, right coronary arterial flow rate is highest during systole, because th aortic pressure driving flow increases more during systole (from 80 to 120mmHg) that the right ventricular pressure which opposes flow (from 0 to 25mmHg). As about 80% of the total coronary arterial flow occurs during diastole, a pressure around the aortic diastole

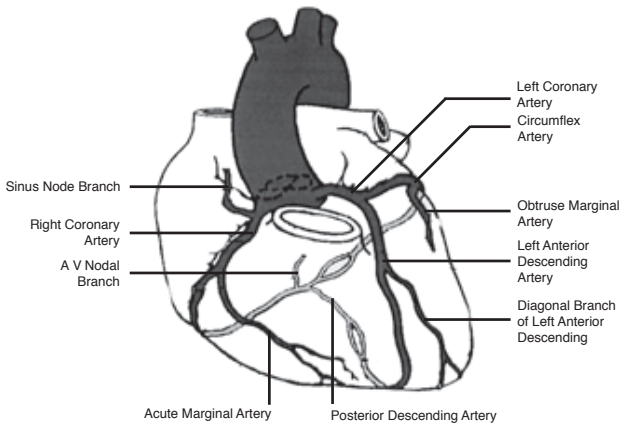


*Figure 5. Coronary arteries*

pressure becomes the primary determinant of the pressure gradient for coronary flow. CPP = arterial diastole pressure - LVEDP. Increases in heart rate that shorten diastole time for coronary blood flow are likely to increase oxygen consumption more than elevations in blood pressure, which are likely to offset increased oxygen demands by enhanced pressure-dependent coronary blood flow. The myocardium regulates its own blood flow (autoregulation) closely between perfusion pressures of 50 and 150mmHg. Beyond this range, blood flow becomes increasingly pressure dependent. This autoregulation is due to a combination of myogenic and metabolic mechanisms.

**Metabolic factors**

The close relationship between coronary blood flow and myocardial $O_2$ consumption indicates that one or more of the products of metabolism cause coronary vasodilation. Hypoxia and adenosine are potent coronary vasodilators. Others factors suspected of playing this role include $PCO_2$, $H^+$, $K^+$, lactate and prostaglandins. Under normal conditions, changes in blood flow are entirely due to variations in coronary artery tone (resistance) in response to metabolic demand.

**Neural Factors**

The coronary arterioles contain α1-adrenergic receptors which mediate vasoconstriction, and β2-adrenergic receptors which mediate vasodilation. Sympathetic stimulation generally increases myocardial blood flow becuase of an increase to metabolic demand and a predominance of B2-activation.

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