WORLD ANAESTHESIA

UPDATE IN ANAESTHESIA

A journal for anaesthetists in developing countries

WORLD ANAESTHESIA

No 8 1998

ISSN 1353-4882

EDITORIAL

Millions of children are anaesthetised each year. Some are fortunate enough to be looked after in dedicated paediatric centres, undergoing surgery and anaesthesia with fully trained specialists. At the other end of the spectrum there are places where children do not receive surgery because of a lack of trained anaesthetists or suitable equipment.

Almost all anaesthetists, particularly those working in developing countries, expect to care for children regularly. This edition of Update in Anaesthesia contains a review of paediatric anaesthesia which we hope will prove practical. It discusses the differences between anaesthetising children and adults and details some of the techniques and equipment required. There is also an article on caudal anaesthesia, a commonly used regional technique to reduce postoperative pain.

We know that our readers work in a variety of circumstances, and we are anxious to ensure that our topics are covered at a realistic level. We would be delighted to receive correspondence regarding our reviews, particularly where specific techniques can be described or recommended for those working with minimal resources.

Update in Anaesthesia is now available on the internet and can be accessed as described on p24. The articles can be downloaded directly to your computer and you can use any of the material for teaching or studying purposes. When doing this please acknowledge Update in Anaesthesia.

We have recently made a few alterations in the way Update is produced and hope to publish every six months in the future. There will be some changes in the way material is presented and we would appreciate feedback on which areas of anaesthesia should be covered. There are many experienced anaesthetists who read our publication and we would benefit from your comments.

Iain Wilson
Roger Eltringham

Contents: No 8

PAEDIATRIC ANAESTHESIA REVIEW

Dr Lyn Rusy
Medical College of Wisconsin Anaesthesia Department, Childrens Hospital of Wisconsin
9000 W Wisconsin Ave PO1997
Milwaukee WI 53201
USA

Dr Elmira Usaleva,
Research Institute of Obstetrics & Paediatrics, Rostov on Don, Russia

This article outlines the essential principles of safe paediatric anaesthesia covering the basics of anatomy, physiology and pharmacology emphasising the differences which exist between adults and children. It assesses how principles used in adult anaesthesia may be adapted to paediatric anaesthesia and includes suggested techniques, monitoring methods, regimens for fluid management and new advances in paediatric pain management. It is divided into three sections; physiology, pharmacology and practical considerations.

PHYSIOLOGY

One of the most important differences between paediatric and adult patients is oxygen consumption which, in infants may exceed 6 ml/kg/min, twice that of adults. There are physiological adaptations in paediatric cardiac and respiratory systems to meet this increased demand.

Cardiovascular. The cardiac index (defined as the cardiac output related to the body surface area to allow a comparison between different sizes of patients) is increased by 30-60 percent in neonates and infants to help meet the increased oxygen consumption. Fetal haemoglobin (which is present in fetal life and up to 3 months following birth) is not able to deliver oxygen to the tissues as efficiently as normal haemoglobin because the oxy-haemoglobin dissociation curve is shifted to the left causing oxygen to be released less readily. Neonates have a higher haemoglobin concentration (17 g/dl) and blood volume and this together with the increased cardiac output compensates for the decreased release of oxygen from haemoglobin in the tissues. Replacement of fetal haemoglobin with adult haemoglobin begins at 2-3 months of age and this period is known as physiological anaemia as haemoglobin concentrations may fall to 11 g/dl. Anaemia sufficient to jeopardise oxygen carrying capacity of the blood is possible if the haemoglobin concentration is less than 13 g/dl in the newborn and less than 10 g/dl in the infant under 6 months of age.

Neonatal myocardium has a large supply of mitochondria, nuclei and endoplasmic reticulum to support cell growth and protein synthesis but these are non-contractile tissues which render the myocardium stiff and non-compliant. This may impair filling of the left ventricle and limit the ability to increase the cardiac output by increasing stroke volume (Frank Starling mechanism). Stroke volume is therefore relatively fixed and the only way of increasing cardiac output is by increasing heart rate.

The sympathetic nervous system is not well developed predisposing the neonatal heart to bradycardia. Anatomical closure of the foramen ovale occurs between 3 months and one year of age. Arterial blood pressure increases with age.

Table 1 depicts many of these cardiovascular differences.

Age	Neonate	Infant	1yr	5yr	Adult
0_2 Consumption (ml/kg/min)	6	5	5	4	3
Systolic BP (mmHg)	65	90	95	95	120
Heart Rate (beats/min)	130	120	120	90	77
Blood Volume (ml/kg)	85	80	80	75	70
Haemoglobin (g/dl)	17	11	12	13	14

Respiratory. There are some special features peculiar to the paediatric airway. The head is relatively large with a prominent occiput, the neck is short and the tongue is large. The airway is prone to obstruction because of these differences. Infants and neonates breathe mainly through their nasal airway, although their nostrils are small and easily obstructed. The larynx is higher in the neck (more cephalad), being at the level of C3 in a premature infant and C4 in a child compared to C5-6 in the adult.

The epiglottis is large, floppy and U shaped. The trachea is short (approximately 4-9 cm) directed downward and posterior and the right main bronchus is less angled than the left. Right mainstem intubations are therefore more likely. The glottic opening (laryngeal opening) is more anterior and the narrowest part of the airway is at the cricoid ring. (In the adult airway the narrowest point is the vocal cords). The diameter of the trachea in the newborn is 4-5mm. Since the resistance to airflow through a tube is directly related to the tube length and inversely related to the fourth power of the radius of the tube, tracheal oedema of just 1mm can dramatically increase resistance to breathing.

The size of the endotracheal tube is critical, as one that is too large will exert pressure on the internal surface of the cricoid cartilage resulting in oedema which could lead to airway obstruction when the tube is removed. An uncuffed endotracheal tube which has an air leak around it when positive pressure is applied to it should be used in children under 10 years of age. An uncuffed tube provides a larger internal diameter compared with a cuffed tube. In general the internal diameter of endotracheal tube related to age is as follows:

Premature	2.5 - 3.0 mm
Neonate to 6 months	3.0 - 3.5 mm
6 months - 1 year	3.5 - 4.0 mm
1 - 2 years	4.0 - 5.0 mm
> 2 years Use the formula	$4 + \frac{\text{Age}}{4}$

Because of the higher position of the larynx and the shape of the epiglottis intubation may be easier in infants and young children using a straight bladed laryngoscope to elevate the epiglottis rather than a curved Mackintosh blade in the vallecula. Although the length of the trachea varies, in most infants up to one year of age, if the 10cm mark of the endotracheal tube is at the alveolar ridge (gums), the tip of the tube is just above the carina. With older children an easy formula of

$\frac{\text{Age}}{2} + 12$ will provide a guide to which mark should be at the lips. After intubation the tube should be secured carefully to the maxilla rather than the mandible which is mobile and the position of the tube checked by auscultation and capnography if this is available.

Alveolar minute ventilation is increased to meet the increased oxygen demands. Carbon dioxide production is also increased in neonates but a normal $PaCO_2$ level (blood CO_2 level) is maintained by the increased alveolar ventilation. Tidal volume is similar for adults and children on a ml/kg basis, so that the increased alveolar ventilation is achieved by an increase in respiratory rate. All of these factors give the lungs less reserve of oxygen so that a well oxygenated infant with upper airway obstruction can become cyanotic in a matter of seconds. Control of ventilation is immature in neonates and responses to hypoxic conditions are unpredictable, sometimes resulting in periods of apnoea. Ex-premature babies are at risk of apnoea following general anaesthesia up to 52 weeks gestational age and should be closely observed for 24 hours post operatively.

Infants have relatively soft chest walls compared with the more rigid chest wall of older children and adults. This results in intercostal and sternal recession in small children with airway obstruction. The diaphragm is responsible for most of the ventilation in this group and anything tending to decrease its efficiency, such as a distended abdomen, may cause respiratory problems for the infant.

Renal system and fluid balance. The neonatal kidney is characterised by a decrease in glomerular filtration rate, sodium excretion and concentrating ability. These values slowly approach those of the adult by 12 months of age. Consequently, the infant cannot handle a large water load and may be unable to excrete electrolytes.

The extracellular fluid volume (ECF) is equivalent to about 40% of the body weight in neonates as opposed to 20% in adults. This difference has disappeared by the age of two years. The increased metabolic rate of infants results in a faster turnover of extracellular fluid. An interruption of the normal fluid intake can therefore rapidly lead to dehydration and careful attention to intra-operative fluids is mandatory. Fluid requirements can be considered as **maintenance fluids and replacement fluids.**

Maintenance fluid requirements are calculated on an hourly basis depending on the body weight. A suitable way of working this out is as follows: 4 ml/kg for the first 10 kg, adding 2 ml/kg for the second 10 kg and 1 ml/kg for each kg over 20kg.

Example of maintenance fluid calculation:

8kg child
8kg X 4mls/kg = **32mls/hour maintenance**

12kg child (is 10kg + 2kg)
10kg X 4mls/kg = 40mls/hr
+2kg X 2mls/kg = 4mls/hr
Total = 40 + 4 = **44mls/hour maintenance**

25kg child (is 10kg + 10kg + 5kg)
10kg X 4mls/kg = 40mls/hr
+10kg X 2mls/kg = 20mls/hr
+ 5kg X 1ml/kg = 5mls/hr
Total = 40+20+5= **65mls/hr maintenance**

The maintenance fluid replaces the fluid that the child would normally have been drinking. After most minor or moderate surgery children will return to drinking fairly quickly and make up any deficit. However after major surgery or when there are pre-existing fluid deficits intravenous maintenance fluids will be required. A regimen with 30% of the fluid as normal saline and 70% as dextrose 5% is suitable for this purpose. Alternatively 4% glucose in 0.18% saline may be used.

Replacement fluids. Patients undergoing surgery may also need intravenous fluid to replace abnormal losses of fluid from bleeding or "third space" loss and any pre-existing deficits. "Third space" loss refers to fluid which is lost from the circulation during surgery. Some of this fluid forms oedema in the area of the operation, some may be lost into the bowel and there may also be losses from evaporation. In general the more major the surgery the more replacement fluid will be required. These losses are commonly replaced by balanced salt solutions such as Hartmanns solution. Colloid solutions are sometimes used when losses are heavy.

Body surface surgery (eg a hernia), or surgery involving a distal extremity will result in only minor fluid losses and replacement fluids will only be needed in the event of significant blood loss. Abdominal or chest surgery will have much greater requirements for fluids and possibly blood. In general abdominal surgery will need extra fluid to replace these third space losses at around 10mls/kg/hour for each hour of surgery.

Pre-existing deficit. Patients who are already fluid depleted preoperatively need replacement in a volume proportional to the degree of dehydration estimated on the basis of the history and clinical signs: Dry skin and mucus membranes represents a 5% deficit. Cool peripheries and loss of skin elasticity, depressed fontanelles and eyeballs and oliguria represents a 10% deficit. A hypotensive moribund patient, unresponsive to pain represents a 15% deficit. The volume required to replace this deficit is calculated as the percent deficit times 10mls/kg. Whenever possible fluid deficits should be corrected prior to surgery, though time is always limited with emergency or urgent surgery. If venous access is impossible fluids can be administered via the intra-osseous route (Update in Anaesthesia Number 5, page 17).

Replacement of blood. Blood volume varies according to age (Table 1). In general blood replacement is required when the haematocrit drops below 25% (around a Hb of 8g/dl) or when the estimated blood loss exceeds 20% of the calculated blood volume. Lesser degrees of blood loss can be replaced by colloid, such as gelatin, dextran or albumin or crystalloid solutions. If crystalloid is used then a volume of three times the estimated blood loss should be given, usually in the form of lactated Ringers (Hartmanns solution) or 0.9% saline. Try to warm iv fluids for children in theatre if they are receiving replacement fluids.

Calculation of blood loss is best done by collecting and measuring suction blood during the procedure. Swabs may be weighed on a simple pair of kitchen type scales. If the weight of the dry swabs is subtracted from the total weight then the extra grams can be taken to indicate the number of mls of blood on the swab. The swabs should be weighed before they dry, because of inaccuracies due to evaporation.

Temperature regulation. Maintenance of body temperature may be a major problem even in warm countries. Neonates and infants have a large surface area to volume ratio and therefore a greater area for heat loss, especially from the head. There is an increased metabolic rate but insufficient body fat for insulation and heat is lost more rapidly. Infants

less than three months of age do not shiver and rely primarily on non-shivering thermogenesis to generate heat. The heat is produced in the brown fat and occurs only in infants. The fat is located primarily around the scapula, in the mediastinum and around the adrenal glands and kidneys. It is important to maintain a warm environment to minimise heat loss. For the very small neonate, operating room temperatures must be increased much to the dismay of the surgeon in heavy operating room scrub suits! In addition to warming the environment, heat loss may be reduced by wrapping the limbs and head in wool or foil, placing the child in a heating blanket, warming and humidification of inspired gases and warming of intravenous fluids. Very occasionally, in a hot theatre environment, children may become too hot, particularly if they were pyrexial preoperatively. Atropine or ether anaesthesia may increase this tendancy. The temperature can be measured in theatre using a simple thermometer.

PHARMACOLOGY

Changing factors during development determine the response of the paediatric patient to various drugs. These include those affecting pharmacokinetics (absorption, distribution and elimination) and those affecting pharmacodynamics (the effect of the drug on the body).

Inhalational agents. Both induction and emergence from anaesthesia are more rapid in children than in adults. This is probably because of a smaller lung functional residual capacity per unit body weight and a greater tissue blood flow, especially to the vessel rich group (brain, heart, liver and kidney). The vessel rich group in adults is 10 percent of body weight versus 22 percent in neonates.

Anaesthetic potency has traditionally been measured by the minimal alveolar concentration (MAC) required to prevent response to a surgical incision. The anaesthetic requirements of paediatric patients vary according to age. In general the MAC of inhalational agents are greatest in the young and decrease with age. However neonates require lower concentrations of volatile anaesthetics than infants. For example the MAC in preterm neonates for halothane is 0.87 percent compared to 1.20 in older infants. MAC decreases to 0.9 by 3 years of age and then progressively declines with age reaching 0.76 in adults. There are slight increases at the time of puberty. The reasons for this apparently greater anaesthetic requirement in infants are unclear, but may reflect interaction of many factors such as residual elevated progesterone and/or endorphin levels as well as an immature central nervous system. Thus, there is nearly a 30 percent greater anaesthetic requirement for infants to obtain the same depth of anaesthesia. It should be emphasised that there is a smaller margin of safety between adequate anaesthesia and severe cardiovascular depression for the infant and child compared to the adult. This is because the cardiac output in infants is largely dependent on heart rate. Some of the myocardial depression of volatile anaesthetics can be offset by the administration of vagolytic agents such as atropine.

Nitrous Oxide is used as a carrier gas to supplement more potent inhalational agents. It is virtually odourless and makes the introduction of more pungent agents more acceptable. As it is relatively insoluble it rapidly achieves equilibrium with alveolar concentration leading to rapid induction and recovery. In the recovery period rapid diffusion into the alveoli may reduce alveolar concentrations of oxygen (diffusion hypoxia) and high concentrations of oxygen should be given for 5 - 10 minutes following its administration. Because it is very diffusible it rapidly equilibrates with gas filled body cavities and should be avoided in such conditions as pneumothorax.

Brief Review of Halothane. Halothane is a halogenated hydrocarbon. The carbon-fluoride bonds are responsible for its non-flammable and non-explosive nature. During preparation it is mixed with thymol as a preservative and is stored in amber colored bottles to retard spontaneous oxidative decomposition. It has a MAC of 0.76 and vapor pressure of 243mmHg. Circulatory effects include dose dependent reductions in blood pressure and cardiac output often associated with reductions in heart rate. It is non-pungent and therefore allows for a smooth inhalational induction. Halothane causes an increased respiratory rate and decreased tidal volume which results a rise in $PaCO_2$. Halothane increases the susceptibly of the heart to the arrhythmic effects of adrenaline (epinephrine) injected by the surgeon. Although children are less likely that adults to exhibit this effect, doses of adrenaline should be kept under 10mcg/kg when using halothane.

Around 20% of halothane is metabolised by oxidative metabolism in the liver. Rarely hepatic dysfunction (sometimes known as "halothane hepatitis") is diagnosed when other causes of hepatic impairment have been excluded. Certain factors increase susceptibility, including repeated exposures to halothane and obesity. Extremely rarely, severe and occasionally fatal liver damage may occur. However, in patients with liver problems, halothane has undoubtedly been wrongly incriminated in many patients when a more detailed investigation would have cleared the anaesthetic from any blame. The mechanism of hepatic dysfunction in unknown but several theories exists, including metabolic, hepatic oxygen deprivation and immunological.

Enflurane is less useful as an induction agent as it may cause breath holding, coughing and laryngospasm. It may cause cardiovascular and respiratory depression and should be avoided in epileptic patients especially when controlled ventilation is used as it lowers the threshold for seizures.

Isoflurane has a pungent odour and induction is characterised by breath holding, coughing and laryngeal irritation. It is more useful for maintenance of anaesthesia and causes ventilatory and cardiovascular depression similar to halothane.

Sevoflurane is a recently introduced inhalational agent which has the advantage of a pleasant non-irritating odour and a low blood/gas solubility coefficient. Consequently induction is both rapid and smooth and it is an ideal agent for inhalational induction but is considerably more expensive than halothane.

Ether has a high blood gas solubility ratio and a pungent odour. Consequently inhalational induction takes a long time and may be associated with respiratory irritation. It causes minimal respiratory and cardiovascular depression and is therefore extremely safe. It is flammable in air and explosive in oxygen.

Intravenous Anaesthetics. An immature blood brain barrier and decreased ability to metabolise drugs may increase the neonate's sensitivity to barbiturates and opioids. Lower doses may be required to produce the desired pharmacological effects. Children less than 6 months old are susceptible to the respiratory depressant effect of opioids and when used, the infant's breathing should be monitored. On the other hand, older, healthy children require higher doses of thiopentone to achieve intravenous induction of anaesthesia (5-7 mg/kg in children versus 3-5 mg/kg in adults).

Propofol is a new intravenous anaesthetic agent that is being used in paediatric anaesthesia. It is dissolved in a soyabean emulsion and is associated with rapid recovery and reduced nausea and is therefore popular for daycase anaesthesia. Induction of anaesthesia may be associated with pain on injection which can be prevented by mixing lignocaine with the propofol. Induction doses of 2-5 mg/kg may be associated with apnoea, a reduction in arterial blood pressure and cardiac output, similar to thiopentone. As always, with use of such drugs, cardiac and respiratory function need to be monitored. Propofol is safe for patients with acute intermittent porphyria and does not trigger malignant hyperthermia.

Ketamine is a phencyclidine derivative that is widely used in paediatric anaesthesia. An induction dose of 1-2mg/kg produces dissociative anaesthesia characterised by open eyes and nystagmus. Muscle tone is preserved but not sufficiently to maintain laryngeal reflexes. Blood pressure is maintained due to sympathetic stimulation and it is the agent of choice in shocked patients. It does not produce significant depression of ventilation, upper airway tone is well maintained and it causes bronchodilation and is recommended for asthmatics. It causes a rise in intra-ocular and intracranial pressure. Hallucinations may occur in the recovery period but these can be minimized by the administration of benzodiazepines and the provision of a quiet recovery environment. It causes salivation in children, and should be used in combination with an antisialogogue such as atropine 0.02mg/kg. It can also be given intramuscularly in a dose of 5-10mg/kg. (See also Update No4 for a full review of ketamine.)

Muscle Relaxants. Neonates and infants are more sensitive than adults to non-depolarising muscle relaxants. Initial doses, however are similar in both age groups because the increased extracellular fluid volume and volume of distribution in younger patients means that less drug actually reaches the neuromuscular junction. This increased sensitivity

combined with decreased glomerular filtration rate and hepatic clearance can result in prolonged duration of muscle relaxants in neonates. The dose of neostigmine per kg required for antagonism of non-depolarising muscle relaxants is similar in children to adults. A combination of either atropine 0.02mg/kg or glycopyrrolate 0.01mg/kg with neostigmine 0.05mg/kg is suitable.

Neonates and infants require more suxamethonium on a body weight basis to produce comparable degrees of skeletal muscle paralysis, 2 mg/kg for infants versus 1 mg/kg for adults to obtain acceptable conditions for intubation. Again, this change in drug requirement reflects dilutional effects of the increased extra cellular fluid volume and volume of distribution of younger patients. When suxamethonium is contraindicated, one of the newer non-depolarising agents, rocuronium, allows intubation almost as rapidly.

Table of drug doses

Thiopentone	5-6mg/kg standard induction dose
Suxamethonium	1-2mg/kg (2mg/kg in infants)
Atropine	0.02mg/kg
Ketamine	1-2mg/kg IV 3-5mg/kg IM "sedation" 8-10mg/kg IM anaesthetic dose 8mg/kg rectally
Curare	0.5 mg/kg
Atracurium	0.5 mg/kg
Pancuronium	0.1 mg/kg
Vecuronium	0.1 mg/kg
Neostigmine	0.05mg/kg

PRACTICAL CONSIDERATIONS

Preoperative Assessment. Every patient should be visited by the anaesthetist prior to surgery, preferably in the presence of the parents in order to obtain a history, perform a physical examination and evaluate laboratory data in addition to estimating the patient's response to hospitalisation. Parental anxiety and fears concerning surgery and anaesthesia are very real and may be transmitted to the patient. To prepare the child psychologically for elective surgery, educational booklets and a clear explanation are useful. Parents are informed of what to expect, possible risks and the anaesthetic plan. If a parent wishes to accompany the child to the induction room they should be warned in advance what to expect and arrangements made for someone to escort them from the room once induction of anaesthesia has occurred.

Fasting. Safe anaesthesia depends on the patient being fasted. However, numerous studies have shown that the traditional prolonged period without clear liquids prior to anaesthesia is unnecessary. Fasting periods can safely be shortened in normal infants and children who feed frequently during the day. A baby who is used to feeding every two hours will come to surgery less distressed and better hydrated if he or she has been allowed to have clear liquids closer to the time of surgery. Most hospitals have developed more liberal fasting guidelines along the following lines:

Newborn to 12 months:
No formula or breast milk 4 hours before surgery
Clear liquids up to 2 hours before surgery

Over 1 year:
No formula, milk or solid food 6 hours before surgery
Clear liquids up to 2 hours before surgery

(A clear fluid is defined as a fluid through which print can be seen. Remember breast milk is not a clear fluid.)

Common conditions relevant to anaesthesia
What is the anaesthetist to do if the parents tell you the child "has a cold" (upper respiratory tract infection)? Children, especially those having ENT type procedures such as ear tubes or tonsillectomy, often have clear rhinorrhea (nasal discharge) and procedures should not be cancelled on this basis alone. Purulent rhinorrhea, productive cough and fever indicate the cold is a more systemic problem and elective surgery is better postponed for one to two weeks. Ask the parents how the child with a cold has been acting. Is he or she eating and playing normally or is it associated with general malaise and fatigue? These are the cases best postponed. Performing an anaesthetic in the presence of a systemic upper respiratory tract infection may result in laryngospasm and ventilatory problems with hypoxia, all easily avoided if the procedure is simply postponed until the child is well.

Asthma is a common disorder (3-5% of the population) resulting in airway hyperreactivity in

response to a variety of stimuli. Active bronchial asthma is usually characterised by reversible narrowing of airways resulting in audible wheezing on auscultation of the chest. Obstruction of airflow leads to changes in lung volumes, chest wall mechanics, and altered distribution of ventilation and perfusion resulting in hypoxemia and hypercarbia. Pulmonary function tests show the ratio of FEV1:FVC is less than 80 percent.

The sympathetic nervous system plays a major role in maintaining normal bronchial tone and treatment often includes beta-adrenergic agonists, theophylline, anticholinergics, glucocorticoids (in severe cases) and cromoglycate. Beta-adrenergic agents are usually given in the form of inhalers or nebulisers producing bronchodilatation by activation of beta-2 receptors thus avoiding the undesirable beta-1 cardiac effects.

Theophylline and aminophylline produce bronchodilation by inhibiting phosphodiesterase, the enzyme that breaks down cyclic AMP. Anticholinergics produce bronchodilation through their antimuscarinic action and also dry up airway secretions. Glucocorticoids (steroids) have anti-inflammatory effects as well as membrane stabilization and are used in severe cases. Beclomethasone is used as an inhaled steroid and produces fewer systemic side effects.

Management of anaesthesia for the asthmatic patient includes preoperative assessment to determine the severity of the asthma. Elective cases are best avoided in the presence of active wheezing. The goal during induction and maintenance of anaesthesia in the asthmatic is to avoid unnecessary stimulation of the non-anaesthetised airway which may result in bronchospasm. Regional anaesthesia is a good choice as any manipulation of the airway is avoided but is often not practical in children. If general anaesthesia is needed, a smooth induction and emergence is the goal, using drugs such as the volatile anaesthetics, non-histamine releasing opioids or ketamine to establish a depth of anaesthesia that will depress hyperreactivity of the airway. Ketamine is the only intravenous agent with bronchodilating properties.

Intraoperative wheezing should be treated by deepening the anaesthetic and by using inhaled beta-2 agonists by aerosol (salbutamol / terbutaline). Severe bronchospasm can be treated with intravenous aminophylline using a 6mg/kg loading dose followed by 0.5-0.9mg/kg/hour by infusion. Cardiac dysrhythmias during aminophylline need to be carefully monitored. With refractory bronchospasm, adrenaline may be required - in this situation halothane should be turned off. Hydrocortisone (3mg/kg 6 hourly) may also prove useful but acts slowly producing an effect after about 2 hours.

Epilepsy. Seizures represent abnormal synchronisation of electrical activity of the brain and may be localised or generalised. Grand mal seizures are most common and are characterised by loss of consciousness followed by clonic/tonic motor activity. The condition is usually controlled with daily doses of anticonvulsants including phenytoin, phenobarbitone, carbamazepine and valproic acid.

Preoperatively, the anaesthetist needs to determine how active the disease is, i.e., is it well controlled on medications and when was the last seizure? Adverse side effects of the medications can be determined clinically (ataxia, dizziness, confusion and sedation). Anticonvulsants should be ideally continued pre and post operatively to maintain therapeutic levels. Fortunately, most have a prolonged half-life so missing one or two doses is not critical.

Anaesthetic agents which may provoke epilepsy include ketamine, enflurane and methohexitone. Phenytoin and carbamazepine may increase the dose requirements for non-depolarising muscle relaxants due to hepatic microsomal enzyme induction.

Sickle Cell Disease is an inherited disorder resulting from the formation of abnormal haemoglobin (HbS). HbS differs from normal adult haemoglobin (HbA) in the substitution of valine for glutamic acid at the sixth position of the beta chain of haemoglobin. HbS has lower affinity for oxygen as well as decreased solubility. Upon deoxygenating, HbS polymerizes, causing the cells to take on a sickle shape and obstruct vessels.

Painful vaso-occlusive crises result which are thought to be due to micro infarcts in various tissues resulting in joint pain, chest pain and abdominal pain. Aplastic crises can produce profound anemia when red cell production is exhausted. The red cell survival is only 10-20 days compared to the 120

days of a normal red cell, resulting in chronic anaemia. The diagnosis is confirmed with haemoglobin electrophoresis. Update in Anaesthesia No. 4 contains a full discussion of Sickle Cell disease.

Optimal preoperative preparation includes hydration, control of infections and ensuring an acceptable haemoglobin concentration. Preoperative simple or exchange transfusion may be necessary to achieve a haemoglobin concentration of 10g/dl or greater with 40-50% of normal HbA present.

Intraoperative problems that might promote sickling should be avoided including dehydration, hypothermia, hypoxaemia, hypotension and acidosis. An inspired oxygen tension of 50 percent is desirable, if possible. Many clinicians also avoid the use of tourniquets as these may enhance sickling distal to the tourniquet. The same principles apply in the postoperative period. Supplemental oxygen, optimal pain control, pulmonary physiotherapy, hydration and early ambulation are all desirable.

Pre-medication should be prescribed according to the needs of the patient. Providing that a good rapport has been established with the child and parents most children do not require pre-medication. Sedatives should be reserved for those who are unduly anxious. Oral midazolam 0.75mg/kg administered 30 minutes prior to induction is very suitable. When prepared from the parenteral form the bitter taste can be reduced by adding a teaspoonful of paracetamol elixir. Other commonly prescribed pre-medications are oral trimeprazine 3mg/kg and diazepam 0.25mg/kg. Intramuscular pre-medications are traumatic for children and should be avoided whenever possible

Atropine or glycopyrrolate can be administered orally (or intramuscularly) preoperatively or given iv on induction of anaesthesia. Certain anaesthetic drugs, particularly suxamethonium and halothane may cause vagally induced bradycardia. This effect is more prominent in infants under three months of age. A vagolytic dose of atropine (0.03 mg/kg) provides complete protection against vagal cardiac slowing or other cardiac arrhythmias in infants under 6 months of age. These seemingly large doses are well tolerated by infants. In general, if atropine is needed, many anaesthetists prefer to give it intravenously in the operating room. As depth of anaesthesia is often judged by changes in heart rate, the resultant tachycardia from atropine may make it more difficult for the anaesthetist. Flushing of the face, delirium, restlessness and agitation may occasionally occur in the recovery room following atropine or scopolamine (hyoscine).

A recent advance is EMLA (a Eutectic Mixture of Local Anaesthetic) prepared with prilocaine and lignocaine as an emulsion in a white cream. It produces skin anaesthesia after it has been in place for one hour under an occlusive dressing and has proved useful for painless venous puncture even in very young children. Systemic absorption of the drug is well below toxic levels even after extensive application of the cream. However, methaemoglobinaemia may be produced by the metabolism of prilocaine in the young infant or the older infant after repeated application or where it has been placed on broken skin. Another local anaesthetic cream has recently been introduced based on amethocaine (Ametop).

Basic Anaesthetic Techniques

The induction room. There are advantages in anaesthetising children in a dedicated induction room outside the operating theatre away from distracting sights and sounds. Anxiety may be reduced if a parent accompanies the child. Anaesthesia can frequently be induced with the child sitting on the parent's lap. When induction is over the child can then be transferred to the table and the parents escorted from the room by a nurse.

Equipment checks. Since children can deteriorate rapidly during anaesthesia it is especially important to check that all drugs and apparatus are ready prior to induction. In particular there should be two laryngoscopes, suction apparatus, a range of endotracheal tubes and masks. The Rendell-Baker mask is designed to fit closely around the face to minimise the dead space although some anaesthetists prefer to use a clear mask which allows the child's colour to be checked during induction. Atropine and suxamethonium should be instantly available in case unexpected laryngospasm or other airway problems develop causing hypoxia and bradycardia.

Induction of anaesthesia is generally by intravenous or inhalational methods. Since the introduction of topical local anaesthetic preparations intravenous induction has increased in popularity.

In the absence of suitable veins induction can

generally be achieved rapidly using one of the potent inhalational agents. Nitrous oxide may be added to oxygen as the carrier gas to speed induction but the proportion of oxygen must be kept at >30% to reduce the possibility of hypoxia. The application of a face mask may be unacceptable to the patient in which case the anaesthetic can be gradually introduced via the cupped hand of the anaesthetist held initially away from the patients face.

An inhalational induction of anaesthesia is usually very easy to perform with halothane which is not irritating to the airway. The induction is started with 70% nitrous oxide (if available) and 30% oxygen for a few breaths. The volatile anaesthetic is then gradually introduced, increasing a half percent with every three breaths. More rapid increases should be avoided as coughing or even laryngospasm may develop. A calm voice is helpful from the anaesthetist. Once anaesthesia is obtained, an intravenous cannula or needle may be inserted and drugs can be administered as needed for the surgery. Muscle relaxants are often used to facilitate intubation. If laryngospasm occurs before an intravenous catheter is placed, positive airway pressure should first be used in an attempt to cure the spasm. If this is not successful and hypoxia develops, suxamethonium administered sub-lingually 2mg/kg or intramuscularly 4mg/kg (preceeded by atropine if possible to prevent bradycardia) should be used.

Once anaesthesia has been induced veins usually become more prominent and an indwelling intravenous cannula should be inserted as soon as possible. In babies and children less than six months intubation is advisable due to the difficulty of maintaining an airway. Because of the problems of absorption atelectasis, falling functional residual capacity and hypercapnia it is common practice to ventilate all children under 20kg for anything but very short procedures. The laryngeal mask airway is now manufactured in paediatric sizes and provides an alternative form of airway management but they are not suitable for controlled ventilation in small children because of the danger of gastric dilatation.

Muscle relaxants are often used to facilitate intubation. If suxamethonium is selected atropine must be drawn up ready to administer in case of bradycardia. If a second dose of suxamethonium is required it should always be preceded by atropine (0.02mg/kg) since bradycardia is common with repeat administrations.

It is important to flush an indwelling intravenous cannula with saline following the administration of any drug to prevent any residue in the dead space of the cannula being inadvertently injected when the cannula is next used.

Occasionally the anaesthetist is confronted by an unruly and hysterical child who will not co-operate with either of the above methods of induction. While an IM injection of ketamine (3-5 mg/kg) is possible, it is often easier and less traumatic to gown up the parent and bring them to the induction or operating room with you to be present with the child until an inhalational induction is performed. However, there must be sufficient staff to allow someone to escort the parent out of the operating room after anaesthesia is induced. If intramuscular ketamine is the only option, secretions in the airway can be kept to a minimum if glycopyrolate (0.01mg/kg) is added to the injection. Alternatively atropine may be given after anaesthesia develops.

Anaesthesia Breathing Systems

Many anaesthesia ventilators designed for adults cannot reliably provide the low tidal volumes and rapid respiratory rates required for infants and small children. Unintentional delivery of large tidal volumes to a small child can generate large airway pressures and cause barotrauma (damage to the lungs due to excessive inspiratory pressure).

Spirometers are less accurate at small volumes and delivered tidal volumes may be reduced when adult anaesthesia breathing systems are used due to compression of the gas in long breathing tubes with high compliance. Dedicated paediatric anaesthesia tubing is usually shorter and stiffer and the smaller tidal volumes can be better delivered manually with a 500ml or 1000ml breathing bag.

The efficiency of breathing circuits is measured by the fresh gas flow required to eliminate CO_2 rebreathing. Mapleson circuits are lightweight, and inexpensive. The Mapleson A circuit (Magill circuit) is very efficient during spontaneous ventilation if fresh gas flow is equal to the patients minute ventilation. It is inefficient during controlled ventilation and requires a high gas flow to prevent rebreathing. The Mapleson D is more efficient than the A system with controlled ventilation requiring

a fresh gas flow of 1 to 2 times minute ventilation. The Bain circuit is a co-axial Mapleson D system.

The Ayres T-Piece (Mapleson E) functions in a similar way to the Mapleson D circuit, but because there are no valves and very little resistance to breathing it has proved very suitable for children under 20kg. The version most commonly used is the Jackson-Rees modification which has an open bag attached to the expiratory limb (classified as a Mapleson F system). Fresh gas flows of 2-3 times minute volume should be used to prevent rebreathing during spontaneous ventilation, with a minimum flow of 3 litres/min. During controlled ventilation the litres of fresh gas required per minute can be calculated by giving 1000 ml + 100 mls/kg. The minimum flow should be 3 litres/min. (A full discussion of breathing systems can be found in Update No. 7.)

In general circle systems are bulky with increased resistance making them less suitable for spontaneously breathing children.

Drawover systems have slightly increased respiratory resistance compared with standard continuous flow apparatus. There are 2 ways of using drawover apparatus for small children.

The systems can be converted to continuous flow by connecting a continuous flow of oxygen to the upstream side of the vaporiser. A T piece can then be connected in the normal fashion. Ensure that there are no leaks.

There are differences in the performance of the different drawover vaporisers when supplied with a continuous flow of gas. In this mode, the Oxford Miniature Vaporiser (OMV) requires 4 - 6 litres / minute of fresh gas flow to work efficiently. The EMO requires 8 - 10 litres /minute flow to perform predictably. Lower flows should not be used with this technique.

An alternative technique when using the OMV is to make a paediatric drawover circuit using a AMBU Paedivalve and small AMBU inflating bag. The child's respiration should be supported or controlled throughout, but this vaporiser has been reported to work well at small tidal and minute volumes.

Monitoring techniques for paediatric patients should be similar to those of adults undergoing comparable types of surgery. Standard monitoring includes close observation by the anaesthetist, precordial or oesophageal stethoscope and blood pressure. Where facilities allow the presence of more advanced monitors increases the safety of anaesthesia. These include non-invasive blood pressure, pulse oximetry, temperature, end tidal CO_2 and ECG, all of which should be placed on induction of anaesthesia . There is no doubt that the best monitor available is the pulse oximeter which was discussed in detail in the 5th edition of Update in Anaesthesia.

The stethoscope (precordial or oesophageal) is a most valuable monitoring device and should be used continually in all paediatric patients. It is inexpensive, reliable, does not require any external source of power or maintenance. It provides information on the cardiovascular system (heart rate and rhythm, intensity of heart sounds) and respiratory system (respiratory rate, the presence of secretions, pulmonary oedema and bronchospasm). It also gives instant warning of ventilator disconnection.

Blood pressure measurement requires the correct size of the cuff. A cuff that is too large will give artificially low readings and one that is too small will give readings that are falsely high.

In seriously ill children who are undergoing more extensive surgery with potential for fluid losses, blood pressure should be closely monitored. The central venous pressure may be assessed to help determine the blood volume status. Hourly, urinary output with a Foley catheter is also useful and should exceed 0.5 ml/kg/hr. Hypoglycemia occurs frequently in seriously ill children or very young infants and should be treated with 1-3 ml/kg of a 20% glucose solution intravenously over 5 minutes. Excessive glucose replacement should be avoided as an osmotic diuresis resulting in dehydration may result from hyperglycemia.

All monitors as well as a reliable intravenous infusion should be secured in position before the commencement of surgery after which access to the patient may be difficult.

A Common Intra-operative Problem

Bradycardia in paediatrics often means hypoxia and restoring adequate ventilation and oxygenation may be all that is needed to restore heart rate. Bradycardia from vagal influence, as seen in eye

muscle surgery, can be treated by asking the surgeon to relieve tension on the eye muscle and giving intravenous atropine. Bradycardia resulting from increased intracranial pressure is treated with hyperventilation, diuretics and surgical release of the pressure.

Basic Postoperative Care

Infants and children generally recover faster than adults from anaesthesia and surgery. The immediate postoperative care is as critical as the intra-operative care and the child should be taken to a recovery area with trained staff. The anaesthetist should report to the recovery room personnel any intra-operative problems that occurred. The airway should be maintained to assure adequacy of ventilation and oxygenation and any unexpected findings reported to the anaesthetist. Vital signs should be taken frequently in the first hour and pain treated. The child may return to the ward when the observations are stable, he is fully conscious and his pain is controlled.

Occasionally croup or subglottic oedema after endotracheal anaesthesia manifests itself in the first few hours after extubation. Mild croup results in a hoarse cough, more severe croup may cause labored respiration, sternal recession, anxiety and inadequate ventilation. Mild cases will resolve over time and may only require extra observation and possibly some humidified oxygen. Nebulised adrenaline (5mg) by facemask is used in more severe cases. It is the vasoconstrictor effect of the adrenaline that relieves the oedema but the effect may be short lived. The patient should be monitored very closely as it may recur after the treatment and may require a second dose. Steroids may or may not be useful and a single, large dose of i.v. dexamethasone (4mg for infants, 8mg for children) can be used. If the above treatment is ineffective, preparations should be made for reintubation using a smaller endotracheal tube. This complication may be prevented by using the correct size of endotracheal tube at induction of anaesthesia.

Post operative pain management

Methods of treating postoperative pain in children include the use of systemic analgesics and local anaesthetic agents. The systemic analgesics can be divided into non-opioids and opioids. This area was discussed more fully in Update No 7.

1. Non-opioid analgesics (for mild or moderate pain)

a) paracetamol (acetaminophen) 15mg/kg 6 hourly

b) NSAIDS - this group of drugs has become extremely popular for treating post operative pain in children as they are effective with few side effects and produce an opioid sparing action. They should be avoided in patients with coagulopathy (because of a tendency to prolong postoperative bleeding), nephropathy, gastropathy and asthma. Diclofenac 1-3mg/kg per day in divided doses is widely used. It is also available as a suppository. NB: Aspirin should not be used for children under 12 years because of the association with Reye's syndrome.

2. Opioids (for severe pain)

Opioids may be administered by IM, IV or oral routes. Children are sensitive to opioids and doses should be reduced accordingly. They should not be given to children <5kg.

Suggested doses intramuscularly:

Morphine: 0.1-0.2mg/kg 4 hourly (may be given subcutaneously also)

Pethidine: 1 - 1.5mg/kg 4 hourly

Slow IV administration avoids the need for painful intramuscular injections, but the child should be closely observed whilst this is given.

Codeine is also a useful drug and may be given orally or intramuscularly in a dose of 0.5-1mg/kg 6 hourly. Codeine should not be given intravenously.

Local anaesthetic techniques

Local wound infiltration with bupivacaine 0.25% at the conclusion of surgery is very effective and is extremely simple and safe. It reduces the need for additional measures. Other regional blocks are used in specific situations eg intercostal blocks following thoracotomy, ilio-inguinal and ilio-hypogastric nerve blocks following hernia repair and orchiopexy, dorsal nerve blocks of the penis or caudal blocks following circumcision. Caudal anaesthesia is described on page 14.

Unusual diseases in paediatrics

Pyloric stenosis. Hypertrophy of the muscle of the pyloric sphincter causes obstruction leading to persistent vomiting. It occurs in about one in every 500 live births and usually manifests itself around 2-6 weeks of age. Persistent vomiting results in loss

of hydrogen ions with compensatory attempts the kidney to maintain a normal pH by exchanging potassium for hydrogen. It is the "hypo" disease with the result being a dehydrated infant with hypokalaemia, hyponatraemia, hypochloraemia and metabolic alkalosis. It is not a surgical emergency and the infant should first be rehydrated with intravenous fluid therapy including sodium chloride with potassium supplements for 24-48 hours.

When the infant presents for surgery with normal electrolyte values, the likelihood for aspiration is still high and the stomach should be emptied with a nasogastric tube before induction. Anaesthesia should be induced using a rapid sequence pattern with atropine, thiopentone, suxamethonium and cricoid pressure until the position of the endotracheal tube is confirmed. Postoperative respiratory depression is common in these patients, possibly as a result of CSF alkalosis and the use of opioids should be avoided. The postoperative pain is easily treated with paracetamol and infiltration of local anaesthetic at the time of surgery. The infants are usually happy to feed 3 - 6 hours after the surgical procedure.

Epiglottitis. Symptoms of epiglottitis include an acute onset of difficulty in swallowing, high fever and inspiratory stridor in 2-6 year old children. Children suffering from epiglottitis typically remain upright and dribble saliva. It is an emergency and treatment includes antibiotics and intubation of the trachea until the swelling has subsided with medical therapy. Any attempt to visualise the epiglottis should not be undertaken until the child is in the operating room with appropriate airway equipment for endotracheal intubation and instrumentation for tracheostomy. (It goes without saying that personnel capable of using these instruments need to be present!) Induction and maintenance of anaesthesia for intubation of the trachea is with halothane in oxygen. Muscle relaxants are avoided as skeletal muscle relaxation may result in total upper airway obstruction. Use an endotracheal tube half a size smaller than calculated and have a variety of sizes available, including a stylet. The swelling usually improves in one to two days after appropriate antibiotic therapy. Extubation of the trachea is performed in the operating room after direct laryngoscopy confirms reduction of swelling of the epiglottis. The presence of a leak around the endotracheal tube is used as a sign that swelling has resolved enough for the child to breathe on their own without the presence of an endotracheal tube.

Down's Syndrome (trisomy 21) occurs in about 0.15 % of live births. The condition is associated with mental retardation, congenital cardiac anomalies (atrial and ventricular septal defects), duodenal atresia, hypotonia, small mouth, subglottic stenosis, hypoplastic mandible and protruding tongue. Patency of the upper airway may be difficult to maintain after onset of unconsciousness for the above reasons but intubation is usually not difficult. Manipulation of the neck during intubation should be done cautiously as 10% of these patients have associated asymptomatic atlanto-axial instability.

Prematurity. Six percent of infants in the United States of America are born prematurely, before 37 weeks gestational age. During the last 3 months of gestation, organs are still developing, both structurally and developmentally. When an infant is born prior to term, these organs are asked to function fully when they may not be ready. Preterm infants are less able to maintain body temperature, suck, swallow, eat and even maintain ventilation adequately. As a result, birth asphyxia predisposes them to central nervous system damage, retinopathy of prematurity, intraventricular haemorrhage, respiratory distress syndrome, bronchopulmonary dysplasia, anaemia, apnoea, patent ductus arteriosis and necrotizing enterocolitis.

Anaesthesia for premature infants is often difficult because they may have multisystem disease and respond poorly to anaesthesia. It is important to gather as much information as possible prior to anaesthesia; birth history, ICU problems, laboratory data, radiological findings, state of hydration and nutrition and clotting status. It is advisable to have a second anaesthetist in the operating room to help. The operating room needs to be warm, 35-37 degrees centigrade and an infrared heater, if available, should be placed over the operating room table. Fluids are warmed and heating blankets may also be used. Oxygen requirements should be dictated by the neonate's needs. Using 100 percent oxygen to a neonate who does not need it only predisposes that baby to development of retinopathy of prematurity.

Conclusion

It has not been possible to cover all aspects of paediatric anaesthesia in a single article but it is hoped that this overview of the main principles will prove useful for those called upon to anaesthetise paediatric patients. More detailed accounts of particular techniques and the management of specific paediatric problems will appear in future editions.

References

1. Ryan, J.F., Cote, C.J., Todres, I.D., Goudsouzian, N. A Practice of Anaesthesia for Infants and Children. 1st ed. Grune and Stratton Inc. Orlando,Fla., 1986.
2. Stoelting, R.K., Miller, R.D. Basics of Anaesthesia. 2nd edition. Churchill Livingston Inc. New York., 1989.
3. Gregory, G.A. Paediatric Anaesthesia. 3rd edition. Churchill Livingston Inc. New York, 1994.
4. Barash, P.G., Cullen, B.F., Stoelting, R.K. Clinical Anaesthesia. 2nd edition. J.P. Lippincott Co. Philadelphia, 1989.
5. Procter, L.T., Gregory, G.A. Paediatric Anaesthesia. Current Opinion in Anaesthesiology 1995;8:3, 221-3.
6. Steward, D.J. Paediatric Anaesthesia. Current Opinion in Anesthesia 1993; 6:507-8.
7. Litman, R.S. Gastric volume and pH in children. Anaesth Analg 1994;79:482-85.
8. Skues, M.A., Prys-Roberts, C. The pharmacology of Propofol. J. Clin. Anaesth.1989;1:5.
9. Meakin, G. Drugs in Paediatric Anaesthesia. Current Opinion in Anesthesiology 1994;7:3,251-6.
10. Houck CS., Wilder RT, McDermott J, et al. Intravenous ketorolac in children following surgery: Safety and cost savings with a unit dosing system. Anaesthesiology 1993;79:1139A.
11. Murat I. New inhalational agents in Paediatric Anaesthesia: desflurane and sevoflurane. Current Opinion in Anesthesiology 1996;9:3,225-8.

The authors would like to thank Mia S L Wyatt for her secretarial assistance during the preparation of this review.

CAUDAL EPIDURAL ANAESTHESIA

Dr Bela Vadodaria and Dr David Conn
Department of Anaesthetics,
Royal Devon and Exeter Hospital, Exeter

Introduction

Caudal anaesthesia has been used for many years and is the easiest and safest approach to the epidural space. When correctly performed there is little danger of either the spinal cord or dura being damaged.

It is used to provide peri and post operative analgesia in adults and children. It may be the sole anaesthetic for some procedures, or it may be combined with general anaesthesia.

Indications

Anaesthesia and analgesia below the umbilicus. Paediatric patients do not generally tolerate surgery under regional anaesthesia alone. However in the very young a caudal block may be adequate to carry out urgent procedures such as reduction of incarcerated hernias, allowing return of normal bowel function prior to surgical repair. Anaesthesia can be provided for superficial operations such as skin grafting, perineal procedures, and lower limb surgery. A general anaesthetic will often be required in addition. Pain relief will extend into the post operative period. The duration of the block can been prolonged by the addition of an opiate (pethidine 0.5mg/kg) to the local anaesthetic. The possibility of delayed respiratory depression from epidural opiates needs taken into account, and patients should monitored in an intensive care or high dependency unit for 24 hours following their administration.

Obstetric analgesia for the 2nd stage or instrumental deliveries. Care should be taken as the foetal head lies close to the site of injection and there is real risk of injecting local anaesthetic into the foetus.

Chronic pain problems such as leg pain after prolapsed intervertebral disc, or post shingles pain below the umbilicus.

Contraindications

Infection near the site of the needle insertion.

Coagulopathy or anti coagulation.

Pilonidal cyst

Congenital abnormalities of the lower spine or meninges, because of the unclear or impalpable anatomy.

Anatomy

The caudal epidural space is the lowest portion of the epidural system and is entered through the sacral hiatus. The sacrum is a triangular bone that consists of the five fused sacral vertebrae (S1- S5). It articulates with the fifth lumber vertebra and the coccyx.

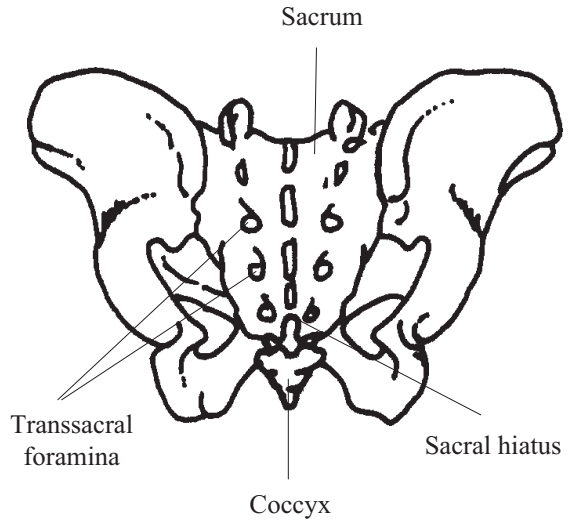


Fig. 1. Anatomy of the sacrum and coccyx

The sacral hiatus is a defect in the lower part of the posterior wall of the sacrum formed by the failure of the laminae of S5 and/or S4 to meet and fuse in the midline. There is a considerable variation in the anatomy of the tissues near the sacral hiatus, in particular, the bony sacrum. The sacral canal is a continuation of the lumbar spinal canal which terminates at the sacral hiatus. The volume of the sacral canal can vary greatly between adults.

The sacral canal contains:

1. The terminal part of the **dural sac**, ending between S1 and S3.

2. The five sacral nerves and coccygeal nerves making up the **cauda equina**. The sacral epidural veins generally end at S4, but may extend throughout the canal. They are at risk from catheter or needle puncture.

3. The **filum terminale** - the final part of the spinal cord which does not contain nerves. This exits through the sacral hiatus and is attached to the back of the coccyx.

4. **Epidural fat**, the character of which changes from a loose texture in children to a more fibrous close-meshed texture in adults. It is this difference that gives rise to the predictability of caudal local anaesthetic spread in children and its unpredictability in adults.

Choice of drug and dosage.

Choose the drug with the longest duration of action and the fewest side effects. Drugs that are commonly used include Lignocaine 1% and Bupivacaine 0.25%, although higher concentrations may be needed for muscle relaxation. Drugs used for epidural injections should come from single use ampoules and be preservative free.

Various regimes have been produced to calculate the appropriate dose of local anaesthetic, the doses vary widely:

1. Armitage recommends bupivacaine 0.5ml/kg for a lumbosacral block, 1ml/kg for a thoraco-lumber block, and 1.25ml/kg for a mid thoracic block. He recommended the use of 0.25% bupivacaine for the block up to a maximum of 20ml. For larger volumes he recommended adding one part of 0.9% NaCl to three parts local anaesthetic to produce a 0.19% mixture.

2. Scott calculates the dose from the child's age or weight. If the child is of average weight for its height both figures will be the same. If the child is overweight use the figure based on age to avoid the possibility of overdose.

Weight in Kg.	Age (in years)	Dose(ml) 0.25% bupivacaine for a block to T12	Dose(ml) 0.25% bupivacaine for for a block to T7
12.5	2	4	6
15	3	5	7.5
16	4	5.5	8
17.5	5	6	9
20	6	7	10.5
22.5	7	8	12
25	8	9	13.5
27.5	9	10	15
30	10	11	16.5

Scott's lower doses are more likely to produce analgesia to the expected height, whereas Armitage will get anaesthesia. Dosages for adults are 20-30 ml for a block of the lower abdomen and 15-20ml for a block of the lower limb and perineum.

Care is needed to avoid the use of toxic doses of drugs for high blocks. The recommended maximum dose of Bupivicaine is 2mg/kg or Lignocaine 4mg/kg. These dosages are the maximum for a correctly injected dose. If the drug is mistakenly injected intravenously very small dosages may cause serious toxicity.

Technique

The patient is prepared as for general anaesthesia,

1. He/she should be fasted
2. All appropriate equipment for resuscitation must be available. Equipment for intubation, airway suction and drugs to stop fitting ie thiopentone 2-5mg/kg or Diazepam 0.2-0.4mg/kg.
3. An intravenous cannula should always be inserted in an upper limb, in case of accidental intravenous injection, or profound sympathetic blockade from a high epidural block.
4. The procedure must be carried out with a **strict aseptic technique**. The skin should be thoroughly prepared and sterile gloves worn. **Any infection in the caudal space is extremely serious**.
5. There are three main approaches: the prone, the semi-prone, and the lateral. The choice depends on the preference of the anaesthetist and the degree of sedation of the patient.

The prone position is often easiest in the adult, as fat tends to move away from the mid-line and landmarks are easier to find. However, there could be difficulty if urgent access to the airway is required. The caudal space is made more prominent by asking the patient to internally rotate their ankles (fig. 2).

The semi-prone position is preferred for the anaesthetised or heavily sedated patient as the airway is easier to control in this position, while still allowing reasonably easy access to the sacral hiatus.

The lateral position is often used in children, as the landmarks are easier to find than in adults. Care should be taken to avoid over flexing the hips (as for lumber epidurals) as this can make the landmarks more difficult to palpate.

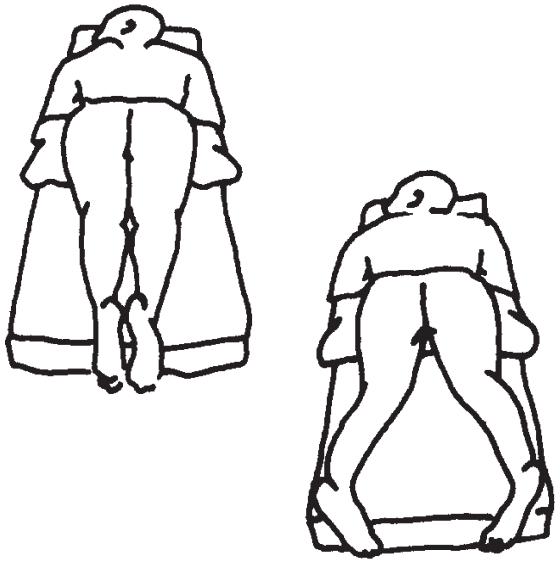
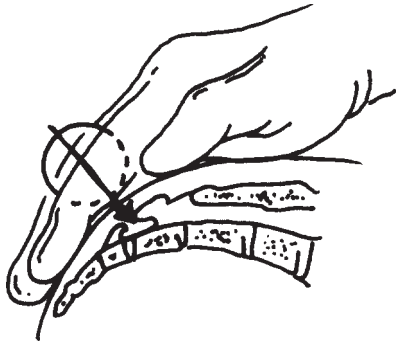


Fig. 2. Position (a) causes contraction of the glutecal muscles. Position (b) allows relaxation of glutecal muscles.

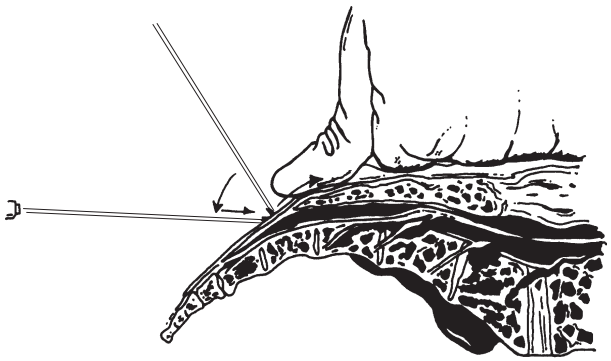
6. The landmarks are palpated. The sacral hiatus and the posterior superior iliac spines form an equilateral triangle pointing inferiorly. The sacral hiatus can be located by first palpating the coccyx, and then sliding the palpating finger in a cephalad direction (towards the head) until a depression in the skin is felt. (In an adult the distance from the tip of the coccyx to the sacral hiatus is approximately the same as the distance from the tip of their index finger to their proximal inter phalangeal joint)!



As there can be a considerable degree of anatomical variation in this region confirmation of bony landmarks is the key to success. The needle can

penetrate a number of different structures mimicking the feel of entering the sacral hiatus. It is important to establish the midline of the sacrum as considerable variability occurs in the prominence of the cornua, causing problems unless care is taken.

7. Once the sacral hiatus is identified the area above is carefully cleaned with antiseptic solution, and a 22 gauge short bevelled cannula or needle is directed at about 45^0 to skin and inserted till a "click" is felt as the sacro-coccygeal ligament is pierced. The needle is then carefully directed in a cephalad direction at an angle approaching the long axis of the spinal canal.



Care should be taken not to insert the needle too far as the dura lies at or below the S2 level in the child.

8. The needle should be aspirated looking for either CSF or blood. A negative aspiration test **does not exclude** intravascular or intrathecal placement. Care should always be taken to look for signs of acute toxicity during the injection. The injection should never be more than 10 ml/30 seconds.

Further tests to confirm the correct position include gently moving the tip of the needle from side to side. The needle will feel firmly held. Introduction of a small amount of air will **not** produce subcutaneous emphysema, and will be heard as a "woosh" sound if a stethoscope is place further up the lumbar spine. Light blood staining is not uncommon and indicates entry into the sacral canal. There should be no local pain during injection. Tingling or a feeling of fullness that extends from the sacrum to the soles of the feet is common during injection.

9. A small amount of local anaesthetic should be injected as a test dose (2 – 4mls). It should not produce either a lump in the subcutaneous tissues, or a feeling of resistance to the injection, nor any systemic effects such as arrhythmias, peri-oral tingling, numbness or hypotension. If the test dose does **not** produce any side effects then the rest of the drug is injected, the needle removed and the patient positioned for surgery.

In the post-operative period, motor function must be checked and the patient should not be allowed to try and walk until complete return of motor function is assured. The patient should not be discharged from hospital until he/she has passed urine, as urinary retention is a recognised complication.

Complications

Intravascular or intraosseous injection. This may lead to grand mal seizures and/or cardio-respiratory arrest.

Dural puncture. Extreme care must be taken to avoid this as a total spinal block will occur if the dose for a caudal block is injected into the subarachnoid space. If this occurs then the patient will become rapidly apnoeic and profoundly hypotensive. Management includes control of the airway and breathing, and treatment of the blood pressure with intravenous fluids and vasopressors such as ephedrine.

Perforation of the rectum. While simple needle puncture is not important, contamination of the needle is extremely dangerous if it is then inserted into the epidural space

Sepsis. This should be a very rare occurrence if strict aseptic procedures are followed.

Urinary retention. This is not uncommon and temporary catheterisation may be required.

Subcutaneous injection. This should be obvious as the drug is injected.

Haematoma

Absent or patchy block.

Conclusion

Caudal block is an easy and safe technique which can be used provide anaesthesia and postoperative analgesia for a wide range of surgical procedures.

When performed carefully complications are rare.

INTRACRANIAL PRESSURE AND CEREBRAL BLOOD FLOW

Dr. F.J.M.Walters
Consultant Anaesthetist
Frenchay Hospital, Bristol UK

INTRODUCTION

The physiological changes that maintain cerebral blood flow (CBF) and accommodate alterations in brain volume are relatively simple to understand. Following trauma or in the presence of major intracranial disease additional changes occur. Major advances in the care of patients with major neurosurgical problems have been developed over the last 10 years. These advances have evolved from a sound understanding of basic physiological rules and the pathological process of different disease situations as well as the pharmacology of anaesthetic drugs. Successful management of these patients relies on a clear understanding of these physiological mechanisms and of the added effect of anaesthesia and the manipulation of arterial pressure, CO_2 and O_2 tensions. Poor anaesthetic technique which allows coughing, straining, hypotension, exaggerated hypertension, hypoxia and hypercarbia will seriously damage the brain. Better results can be obtained by careful monitoring of the patient and attention to simple details than by complex pharmacological interventions. It is the purpose of this article to explain these factors and how an understanding of them can be applied to patients following head trauma or intracranial disease.

The brain is only able to withstand very short periods of ischaemia, unlike the kidney, liver or muscle. Thus cerebral blood flow must be maintained to ensure a constant delivery of oxygen and glucose as well as the removal of "waste" products. Maintenance of cerebral blood flow depends on a balance between the pressure within the skull, intracranial pressure (ICP) and the arterial pressure of the blood, mean arterial pressure (MAP). It is important to maintain a constant blood flow. Thus when blood pressure falls, physiological mechanisms attempt to maintain flow to prevent ischaemia. This process is autoregulation and is explained in detail later. Similarly, when blood pressure rises, the same mechanism stops the blood flow from increasing to excessive levels. If this did occur, cerebral oedema could develop and the brain would enlarge because of the increase in cerebral arterial blood volume.

A number of terms will be used in this article and are defined:-

ICP intracranial pressure is the pressure within the rigid skull.

CBF cerebral blood flow is the flow of blood through the brain, important for delivery of oxygen and removal of "waste" products

CPP cerebral perfusion pressure is the effective pressure driving blood through the brain. It is discussed in detail later

INTRACRANIAL PRESSURE

> *Teaching point*
> High intracranial pressure (ICP) will cause internal or external herniation of the brain, distortion and pressure on cranial nerves and vital neurological centres. Cerebral perfusion will be impeded and operating conditions difficult or impossible. Loss of CSF and reduction of venous blood volume act to compensate for increases in brain volume. Once these mechanisms are exhausted, any further increase, however small, will cause a large increase ICP.

The principle constituents within the skull are brain (80%), blood (12%) and CSF (8%). The total volume is 1600ml. The skull is thus a rigid fluid filled box. If the volume of the contents of a rigid fluid-filled container increase, the pressure inside will rise considerably unless some fluid is able to escape. So it is with the skull and brain within it. If the brain enlarges, some blood or CSF must escape to avoid a rise in pressure. If this should fail, or be unable to occur there will be a rapid increase in ICP from the normal range (5-13 mmHg). If there is an increase in the volume of either the brain or blood the normal initial response is a reduction in CSF volume within the skull. CSF is forced out into the spinal sac. Thus the pressure within the skull, ICP, is initially maintained. If the pathological process progresses with further increase in volume, venous blood and more CSF is forced out of the skull. Ultimately this process becomes exhausted, when the venous sinuses are flattened and there is little or no CSF remaining in the head. Any further increase in brain volume then causes a rapid increase in ICP. This chain of events is represented by the sequence in

Fig 1a and 1b.

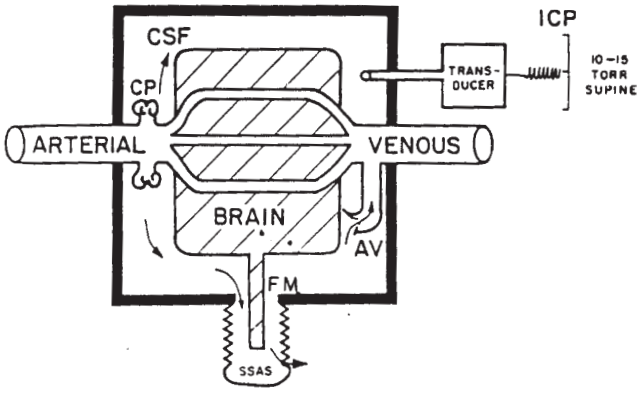


Fig. 1a. Schematic representation of normal intracranial contents SSAS= spinal subarachnoid space, FM = foramen magnum, AV = arachnoid villi, CP = choroid plexus. Arrows indicate direction of CSF flow, heavy lines the skull

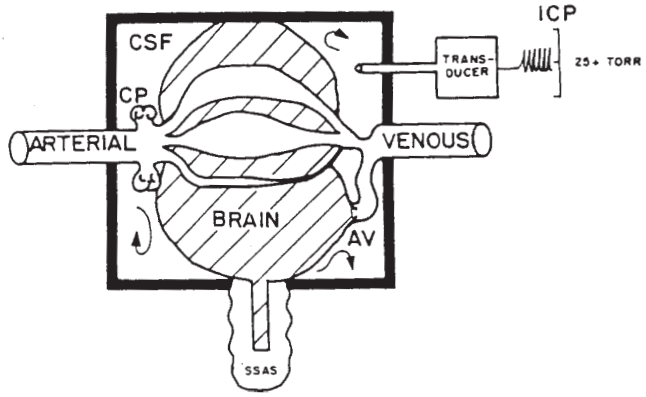


Fig. 1b. Schematic representation of contents of skull during raised intracranial pressure

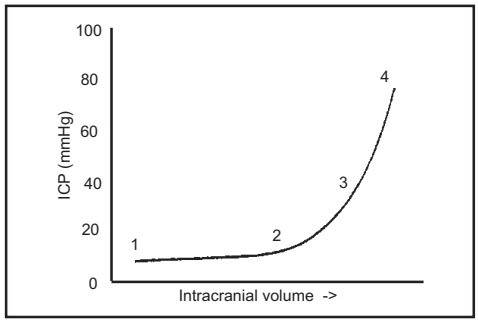


Fig. 2. Brain volume-intracranial pressure relationships: 1-2 compensation phase; 3-4 decompensation phase

The pressure changes within the skull are drawn in the classical curve Fig. 2 which indicates an increase in volume with little change in pressure until a certain point is reached when a further small change in volume results in a large increase in pressure: 1-2 compensation phase; 3-4 decompensation phase.

It is interesting to note that this classic curve represents the alterations in pressure when the volume of a single compartment within the skull, in this case CSF, changes. Therefore it is a CSF-pressure volume curve. In practice when the enlargement of the brain is due to a tumour or haematoma the curve is less steep. Pressure gradients develop within the brain substance and the compliance or "squashiness" of the tumour is different from that of brain leading to this altered curve.

Cerebral swelling leads to herniation of the brain either internally, when the temporal lobe is pushed down onto the mid-brain through the tentorium incisura or externally, with the cerebellar peduncles being forced down through the foramen magnum. This causes torsion of the brain stem and a reduction of local cerebral blood flow as the unrelenting rise in ICP opposes arterial pressure. Ultimately cerebral perfusion pressure falls to a point when there is no cerebral blood flow, no cerebral perfusion and death. The rise in ICP may be accelerated because of acute hydrocephalus. This is caused by brain-stem torsion leading to sudden obstruction of CSF flow.

The volume of blood contained within the venous sinuses is reduced to a minimum as part of the compensatory process. However, should free flow of venous blood be impeded by a number of simple causes (Table 1) then this increase in volume of the venous system in a critically swollen brain will lead to a rapid rise in ICP. In practice, it is imperative to ensure that when the patient is in the supine or lateral position that a head up tilt to a maximum of 30^{o} is obtained. This improves venous drainage with minimal effect on arterial pressure [1].

Table 1. Non-Pathological causes of raised ICP

Increased Venous Volume	
	Coughing
	Obstructed airway
	Head-down position
	Obstructed neck veins
Cerebral oedema	
AVOID	Hypotonic IV solutions
	5% Dextrose, Dextrose-Saline, Hartmann's Solution
USE	0.9% Normal Saline
Increased CBF	
	Anaesthetic drugs - *see next article*

Venous drainage is passive and thus maximised by ensuring there is no pressure on, or kinking, of the neck veins. In addition the higher the head, the greater the effect of gravity on the flow of venous blood. However, as the head is raised, the gravitational effect on the arterial pressure at the brain is also increased. This is a disadvantage as it reduces the pressure of blood perfusing the brain. The best compromise is the position described above of 30°.

Teaching point
If the patient is lying in the supine position, and it is necessary to turn the head laterally, a sand bag should be placed under the shoulder to reduce the pressure of the sternomastoid on the jugular vein. When patients with severe head injuries are nursed or transported it must be with a 30° head up tilt, and the blood pressure maintained.

The extent of the change in ICP caused by an alteration in the volume of intracranial contents is determined by the compliance or "squashiness" of the brain. In other words if compliance is low, the brain is stiffer or less "squashable". Therefore, an increase in brain volume will result in a higher rise in intracranial pressure than if the compliance were high. Compliance affects the elastance or "stretchiness" of the walls of the ventricles. When the elastance is reduced the walls are stiffer. Therefore there is a greater change in pressure for a given alteration in brain volume. If a catheter is inserted into a lateral ventricle via a burr hole, this can be assessed by injecting 1ml of saline and observing the change in intracranial pressure. After the injection, if the rise in pressure is more than 5 mmHg then the patient has become has become decompensated and is at the right hand end of the pressure-volume curve (Fig 2).

CEREBRAL PERFUSION PRESSURE

Cerebral perfusion pressure (CPP) is defined as the difference between mean arterial and intracranial pressures. Mean arterial pressure is the diastolic pressure plus one third of the pulse pressure (difference between the systolic and diastolic). MAP is thus between systolic and diastolic pressures, nearer diastolic. It is used as it is the best value to estimate the "head of pressure" perfusing in the brain:

CPP = MAP - ICP

Normal cerebral perfusion pressure is 80 mmHg, but when reduced to less than 50 mmHg there is metabolic evidence of ischaemia and reduced electrical activity. There have been a number of studies on patients with severe head injuries which have shown an increase in mortality and poor outcome when CPP falls to less than 70 mmHg for a sustained period [2,3]. Continuous monitoring of jugular venous bulb saturation is another tool used to monitor the adequacy of the cerebral circulation when it is at risk. Jugular venous bulb saturation is the oxygen saturation of venous blood in the jugular bulb which is at the base of the skull. The normal range is 65%-75%. If blood flow to the brain is reduced below a critical point there is a fall in venous saturation. As the flow of blood and delivery of oxygen is reduced, the brain, in order to maintain its oxygen supply, extracts more oxygen from the blood, leading to a fall in venous oxygen saturation.

Teaching point
Cerebral perfusion pressure (CPP) = MAP - ICP

Inadequate CPP (less than 70 mmHg) has been shown to be a major factor in the poor outcome of patients with raised ICP. Assessment of CPP is vital and possible either by measurement of both ICP and MAP (mean arterial pressure - see text) or by measuring MAP and making a reasonable estimate of ICP. During anaesthesia therefore, if ICP is raised a fall in blood pressure must be avoided or treated quickly by volume replacement or catecholamines whichever is relevant.

More specifically, when CPP is inadequate the oxygen saturation of jugular venous blood falls (normal range 65%-75%) because of increased oxygen extraction. Does the jugular venous bulb measurement give an indication of the minimum level for CPP? Chan [4] in another study of head-injured patients showed that when CPP was below 70 mmHg, there was a rapid decrease in jugular venous bulb saturation. It was concluded that when CPP was less than 70 mmHg cerebral perfusion was insufficient.

In the head injured patient, CPP should not fall below 70 mmHg.

Therefore continuous consideration of changes in CPP are vital when anaesthetising patients who may have raised ICP and a fall in arterial pressure occurs as a result of anaesthetic agents or blood loss. Ideally ICP should be monitored, but often this is impossible or impractical. However a reasonable estimate can be made in head injured patients who are not sedated: (Drowsy and confused (GCS 13-15)ICP=20 mmHg, Severe brain swelling (GCS <8) ICP=30 mmHg).

Teaching point
The following example illustrates the point. A 28-year old patient who has had a recent head injury where he was unconscious briefly, requires urgent abdominal surgery. He is confused, restless and drowsy. It would be reasonable to estimate his ICP to be 20 mmHg. Following induction of anaesthesia his systolic arterial pressure (SAP) falls to 80 mmHg. In this situation MAP will have fallen to 65 mmHg and therefore CPP will have fallen to less than 45 mmHg, significantly below the critical value of 70 mmHg with a significant risk of causing cerebral ischaemia and a poor cerebral outcome.

CEREBRAL BLOOD FLOW

The normal cerebral blood flow is 45-50ml $100g^{-1}$ min^{-1}, ranging from 20ml $100g^{-1}$ min^{-1} in white matter to 70ml $100g^{-1}$ min^{-1} in grey matter. There are two essential facts to understand about cerebral blood flow. Firstly, in normal circumstances when the flow falls to less than 18-20ml $100g^{-1}$ min^{-1}, physiological electrical function of the cell begins to fail. Secondly, an increase or decrease in CBF will cause an increase or decrease in cerebral arterial blood volume because of arterial dilatation or constriction. Thus in a brain which is decompensated as a result of major intracranial pathology, increases or decreases in CBF will in turn lead to a significant rise or fall in ICP. The physiological factors which can alter CBF and hence ICP are listed in Table 2. There are also a number of drugs which can induce arterial dilatation, the most well known being high concentrations of volatile agents. These will be discussed in detail in a subsequent article.

Autoregulation
CBF is maintained at a constant level in normal brain in the face of the usual fluctuations in blood pressure by the process of autoregulation. It is a poorly understood local vascular mechanism. Normally autoregulation maintains a constant blood flow between MAP 50 mmHg and 150 mmHg. However in traumatised or ischaemic brain, or following vasodilator agents (volatile agents and sodium nitroprusside) CBF may become blood pressure dependent. Thus as arterial pressure rises so CBF will rise causing an increase in cerebral volume. Similarly as pressure falls so CBF will also fall, reducing ICP, but also inducing an uncontrolled reduction in CBF.

Teaching point
There are a number of physiological factors which affect or change cerebral blood flow (CBF). Rises in CBF due to hypoxia, hypercapnia (raised blood CO2) and high concentrations of volatile agents will cause a rise in ICP once the normal compensating mechanisms have been exhausted. Poor anaesthetic technique during which hypoxia, hypercapnia and hypotension occur will seriously damage the critically ill brain further.

Table 2. Physiological causes of raised ICP

Hypoxia
Hypercapnia
Pain
Low Cerebral Perfusion Pressure
Exaggerated Hypertension

More recent work has shown that following trauma autoregulation may still be functioning. Bouma reported that it was present in up to 69% patients with head injuries[5]. In this situation if CPP falls below the critical value of 70 mmHg, the patient will have inadequate cerebral perfusion. Autoregulation will cause cerebral vasodilatation leading to a rise in brain volume. This in turn will lead to a further rise in ICP and induce the vicious circle described by the vasodilatation cascade (fig 3a) which results in cerebral ischaemia. This process can only be broken by increasing the blood pressure to raise CPP, inducing the vasoconstriction cascade (fig 3b). This explains why the maintenance of arterial blood pressure at adequate level by careful

monitoring and rapid correction if it falls is so important.

Carbon dioxide causes cerebral vasodilation. As the arterial tension of CO_2 (fig 4) rises, CBF increases and when it is reduced vasoconstriction is induced. Thus hyperventilation can lead to a mean reduction in intracranial pressure of about 50% within 2-30 minutes [6]. When $PaCO_2$ is less than 25 mmHg (3.3kPa) there is no further reduction in CBF. Therefore there is no advantage in inducing further hypocapnia as this will only shift the oxygen dissociation curve further to the left, making oxygen less available to the tissues. Acute hypocapnic vasoconstriction will only last for a relatively short time (5 hours). While hypocapnia is maintained, there is a gradual increase in CBF towards control values leading which will lead to cerebral hyperaemia (over-perfusion) if the $PaCO_2$ is returned rapidly to normal levels [7]. When long term ventilation is required, only mild hypocapnia (34-38 mmHg: 4.5-5.1 kPa) should be induced. Worse outcome was reported in patients after head injuries at 3 and 6 months in those who had been hyperventilated to low $PaCO_2$ levels for long periods [8].

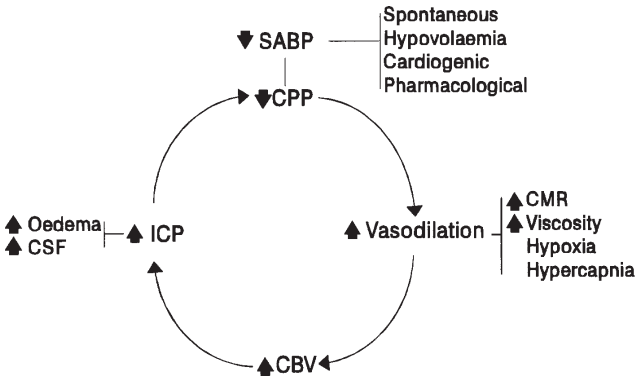


Fig 3a Vasodilatory cascade

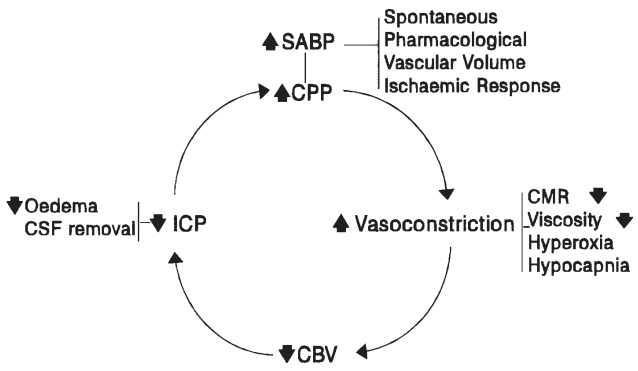


Fig 3b Vasoconstriction cascade

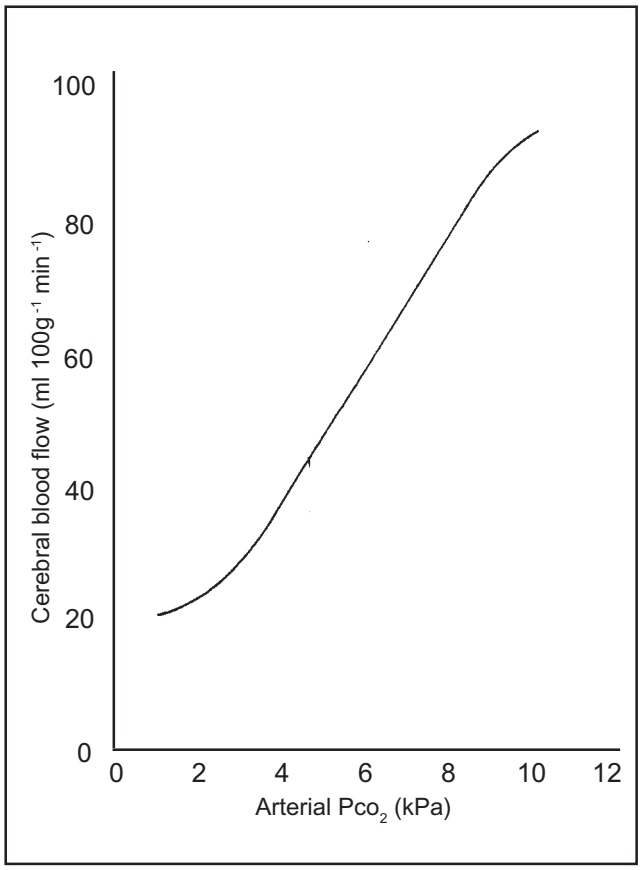


Fig 4 Schematic representation between cerebral blood flow and arterial carbon dioxide tension

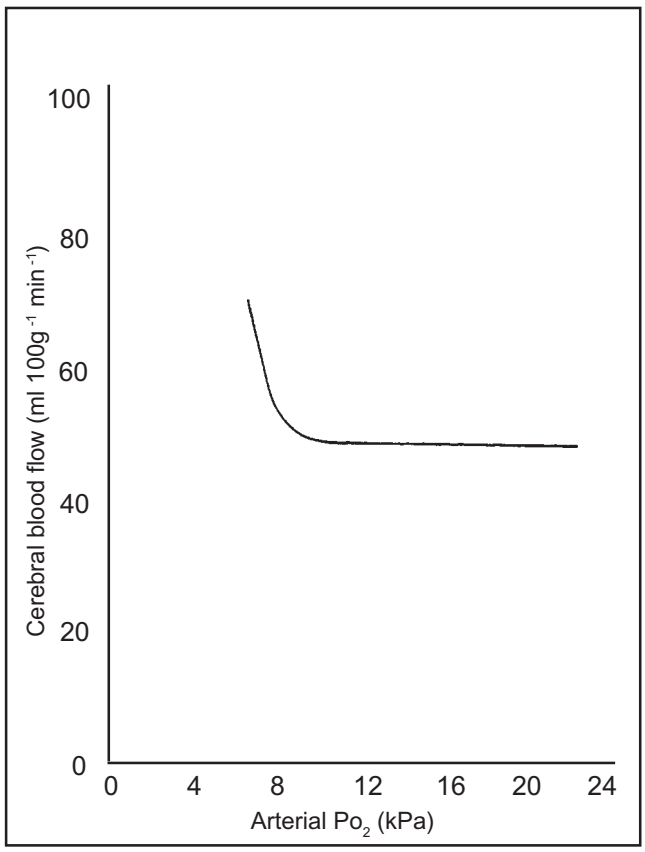


Fig 5 Schematic representation between cerebral blood flow and arterial oxygen tension

Teaching point
When there is an acute rise in ICP, for example after an acute head injury ICP can be reduced by hyperventilation to lower arterial CO_2 tension. This technique is used during neurosurgery to reduce brain size to improve access for the surgeon. In **CONTRAST** only mild hyperventilation should be used for long term ventilation of patients as described above.

Oxygen. Low arterial oxygen tension also has profound effects on cerebral blood flow (Fig 5). When it falls below 50 mmHg (6.7 kPa), there is a rapid increase in CBF and arterial blood volume.

APPLIED PHYSIOLOGY - HEAD INJURY

Following acute head injury, the term "secondary injury" has been described. The primary injury is due to the actual trauma. The secondary brain injury is caused by ischaemia due to the combination of rapid brain swelling and hypotension. Any patient who is unconscious can easily develop an obstructed airway and become hypoxic, hypercapnic, possibly hypotensive and have a rise in intrathoracic pressure. Considering the physiological changes described already, the process that results in cerebral swelling and raised ICP is clear. In addition, pain from other injuries, despite the patient being unconscious, will cause an increase in CBF as a result of both the hypertensive response and local dilatation in the relevant sensory area of the brain. Thus in the initial management of the acutely head injured patient who is unable to maintain his airway, intubation and hyperventilation should be instituted following an intravenous anaesthetic agent, and an opiate, to attenuate the response to intubation. The dose must be carefully chosen to avoid hypotension in a patient who may also be hypovolaemic.

Teaching point
In an article on the management and resuscitation of patients with serious head injuries Gentleman et al [9] noted that over an 11 year period there was significant reduction in mortality, (45% to 32%) and an increase in patients making a good recovery, (42%-58%) associated with a reduction in hypoxaemia and hypotension during treatment.

Hypotension must be treated aggressively with a rapid infusion of colloid or blood and if, necessary, intravenous (ephedrine 3-6 mg, methoxamine 1-3 mg).

REFERENCES

1. Durward Q J et al. Cerebral and cardiovascular responses to changes in head elevation in patients with intracranial hypertension. J.Neurosurgery 1983; 59: 938-944.
2. McGraw C P. A cerebral perfusion pressure greater than 80 mmHg is more beneficial. Intracranial Pressure VII. Edits. Hoff J T & Betz A L. 839-841. Springer-Verlag, Berlin. 1989
3. Rosner M J, Rosner S D & Johnson A H. Cerebral perfusion: management protocol and clinical results. J.Neurosurgery 1985; 83: 949-962.
4. Chan K H, Miller J D, Dearden N M, Andrews P J D & Midgley S. The effects of changes in cerebral perfusion pressure upon middle cerebral artery blood flow velocity and jugular bulb venous oxygen saturation after severe brain trauma. J.Neurosurgery 1992; 77: 55-61.
5. Bouma G J, Muizelaar J P, Handoh K & Marmarou A. Blood pressure and intracranial pressure - volume dynamics in severe head injury: relationship with cerebral blood flow. J Neurosurgery 1992; 77: 15-19.
6. Ruben B H. Intracranial hypertension in Advances in Anaesthesia. Edit. Gallagher T J. p.1, London Year Book Medical Publishers Inc. 1984.
7. Jones P W. Hyperventilation in the management of cerebral oedema. Intensive Care Medicine. 1981; 7: 205-207.
8. Muizelaar J P et al. Adverse effects of prolonged hyperventilation in patients with severe head injury: a randomised clinical trial. J.Neurosurgery 1991; 75: 731-739.
9. Gentleman D., Dearden M., Midgeley S. & Maclearn D. Guidelines for the transfer of patients with serious head injury. British Medical Journal 1993; 307: 547-552

ANAESTHESIA FOR EYE SURGERY

Andrei Varvinski, Archangelsk Medical Academy, Troytski, 51 Archangelsk, 163061 Russia

Roger Eltringham, Gloucestershire Royal Hospital, Gloucester, United Kingdom

Surgery on the eye can be performed under either local or general anaesthesia. In the previous issue of Update techniques for local anaesthesia were described. In this article the principles of general anaesthesia for eye surgery are outlined.

General anaesthesia for eye surgery presents a number of special considerations for the anaesthetist. Patients are frequently at the extremes of age and in the case of the elderly concomitant medical conditions are not uncommon, particularly diabetes and hypertension. Drugs used in ophthalmology may influence the course of the anaesthetic. For example agents used in the treatment of glaucoma may include timolol, a beta blocking agent, or phospholine iodide which has anticholinesterase properties and may prolong the action of suxamethonium.

The anaesthetist must be familiar with the factors which influence intra-ocular pressure (IOP). This is the pressure within the eyeball and is normally in the region of 10-20mm Hg. When the surgeon is operating within the globe (eg cataract surgery) it is important that the anaesthetist controls the IOP. A rise in the IOP will impair the operating conditions and may cause an expulsion of intraocular contents with permanent damage to the eye. On the other hand a mild reduction in IOP will improve operating conditions for the surgeon.

A rise in IOP can generally be attributed to one or more of the following: pressure from outside, an increase in the volume of blood in the vessels within the eye or an increase in the volume of aqueous or vitreous humor.

Factors increasing IOP include:

1. External pressure eg face mask
2. Raised venous pressure, eg by coughing, straining, vomiting
3. Raised arterial pressure
4. Hypoxia and hypercarbia which cause vasodilation of intraocular blood vessels
5. Suxamethonium - the precise mechanism is unknown but may be due to contraction of extraocular muscles during fasiculation or dilation of blood vessels. The effect is maximal at 2-4 minutes returning to normal within 7 minutes.
6. Ketamine

Factors lowering IOP include:

1. Reduced venous pressure, eg. head up tilt.
2. Lowered arterial pressure - at systolic pressures <90mm Hg IOP is proportional to the blood pressure.
3. Hypocarbia by constricting choroidal vessels.
4. Intravenous induction agents (except ketamine).
5. Inhalational agents (the fall in IOP is proportional to the inspired concentration).
6. Non-depolarising muscle relaxants.
7. Reduction in aqueous volume, eg by acetazolamide which inhibits production.
8. Reduction in vitreous volume, eg by mannitol which exerts an osmotic effect.

A suitable technique for intraocular surgery in adults is as follows:

Premedication. Oral diazepam 0.1 - 0.2mg/kg. Heavy sedation with opiates is best avoided because of the dangers of respiratory depression and hypercarbia.

Induction: Thiopentone 4mg/kg
Suxamethonium 1mg/kg

Laryngoscopy. This should not be performed until the patient is fully paralysed in order to avoid gagging or coughing. Topical anaesthesia to the larynx and trachea with 4% lignocaine will reduce the incidence of coughing especially if the head is moved.

Maintenance. N20:02:halothane 0.5 - 1% (or equivalent concentration of other volatile agent). Vecuronium 0.1mg/kg administered before the effects of suxamethonium have worn off. IPPV to produce moderate hypocarbia.

Position. Head up tilt to reduce venous pressure

Monitoring. ECG, oximeter, capnograph and peripheral nerve stimulator should be used if available. In situations where full monitoring is not available it is probably safer to administer atropine or glycopyrrolate routinely with the

induction of anaesthesia to prevent the bradycardia which may occur during manipulation of the eye due to the oculocardiac reflex.

Reversal. Continue volatile agent until reversal is complete and spontaneous respiration is resumed using atropine or glycopyrrolate and neostigmine as required.

Extubation. This should be accomplished with the patient on their side. An anti-emetic may be administered to minimise the incidence of post operative vomiting.

Post Operative Analgesia. Morphine 0.1mg/kg if required. No food or drink should be administered for 3 hours to reduce the possibility of aspiration of gastric contents.

If muscle relaxants are unavailable and the patient breathes spontaneously the depth of anaesthesia must be increased to prevent coughing or straining against the tube. This technique however carries the additional disadvantages of subsequent hypercarbia, hypotension and a prolonged recovery period. Some anaesthetists use a reinforced laryngeal mask with spontaneous respiration for some ophthalmic operations.

Penetrating Eye Injury

When the globe has been penetrated the IOP is reduced to atmospheric pressure. An increase in IOP during induction may cause expulsion of intraocular contents and permanent damage to the eye.

If the repair is carried out as an emergency procedure the patient must be assumed to have a full stomach and requires a rapid sequence induction. During pre-oxygenation care must be taken not to exert pressure on the eye by the face mask. Suxamethonium is theoretically contra-indicated as it causes a rise in IOP. The anaesthetist must however weigh the risk to the eye against the risk of aspiration of gastric contents.

If intubation is anticipated as being uneventful a large dose of a non-depolarising relaxant (eg vecuronium 0.15mg/kg) may be substituted for suxamethonium and a modified rapid sequence induction performed. Care must be taken to allow time for full muscle paralysis to occur before laryngoscopy is attempted meanwhile, cricoid pressure is maintained.

If, however, difficulties are anticipated with intubation suxamethonium should be used to provide the best intubating conditions as rapidly as possible despite the theoretical risk to the eye. In practice the risk is minimised by the prior administration of an induction agent which reduces IOP.

Once intubation has been achieved anaesthesia is conducted using the general principles described above.

Strabismus Surgery

During surgery for the correction of strabismus traction on the extra ocular muscles may cause sudden and profound bradycardia via the oculo-cardiac reflex mediated by the vagus nerve. This effect is also occasionally seen during other forms of eye surgery eg retinal detachment.

When this occurs the surgeon must be immediately alerted as the normal heart rate is generally restored when traction is released. If this does not occur an intravenous bolus of atropine 0.02 mg/kg may be required. It is important that the anaesthetist carefully monitors the heart rate throughout the operation, preferably using an audible warning device so that the surgeon is aware of changes in heart rate. Intravenous access must be assured and atropine drawn up and ready for immediate use. In paediatric patients with their increased vagal tone the prophylactic administration of atropine before the commencement of surgery is advisable.

Examination Under Anaesthesia

Although endotracheal intubation is required for most patients requiring general anaesthesia for eye surgery, examination of the eye in children can often be provided satisfactorily via a face mask. If the naso-lacrinal duct is to be irrigated steps should be taken to avoid respiratory obstruction due to laryngospasm. This can be achieved either by intubation or positioning the patient with a pillow under the shoulders to divert irrigation fluid away from the larynx.

Ketamine can also be used for examination of the eye but pre-medication with atropine is essential to prevent laryngospasm caused by excessive secretions.

ANAESTHESIA IN CHILDREN USING THE EMO SYSTEM

Mr Peter Bewes, Continuing Medical Education, c/o Department of Surgery, Makerere University Medical School, PO Box 4127, Kampala, Uganda

In some places, by necessity, children undergo anaesthesia with inadequate facilities. Transport to another unit is frequently not an option and the following article describes a method to adapt the EMO system for use in children under 15kg. The technique requires anaesthesia training and should not be attempted by someone without the ability to intubate.

The concept is to use the EMO with a T piece and continuous flow. Two people are required; the anaesthetist who anaesthetises the child using a standard T piece and an assistant who pumps the Oxford inflating bellows continuously so that there is always a flow of ether in air +/- oxygen through the T-piece. This minimises the work of breathing for the child and ensures adequate amounts of ether are produced by the EMO.

The Oxford inflating bellows should have the magnet removed from the flap valve and the T piece connected to the bellows outlet. The T piece can be used with a mask or endotracheal tube (fig. 1).

Induction of anaesthesia using ether. With a larger child you may be able to get good co-operation by "talking the patient to sleep" as you administer the anaesthetic, but in babies it is often easier to wrap them in a blanket to keep the arms and legs still while the baby cries and inhales the anaesthetic ether.

Ether is a very strong smelling vapour that stimulates a lot of secretions. In order to prevent problems give atropine in appropriate doses (around 0.02mg/kg) intravenously or intramuscularly before induction of anaesthesia.

The strong smell of ether frightens many patients, so introduce it gradually. I like to let them breathe through the mask for a few breaths before attaching it to the T piece. This allows time to check that the mask fits the face properly and reassures the patient that they are not being suffocated. Then tell the patient that they will smell a strange smell (not unpleasant) and, with the ether concentration lever on the EMO set to transit, connect up the face mask to the T-piece. At this point start your assistant pumping the Oxford inflating bellows 8 times per minute to produce a near continuous flow of gas until the end of the operation when anaesthesia is finished.

Initially the child will detect the residual vapour from the rubber tubing, and will get used to the smell without the feeling of being poisoned! After a minute or two, nudge the ether concentration lever from the transit position to give a weak concentration of ether. Reassure the child that the "nice smelly stuff" will make him feel sleepy. Keep the face mask snugly fitted to the patient's face, so that there is no leak. Slowly nudge the concentration up to 2%, always allowing at least four cough-clear breaths between nudges, and never increase the concentration by more than one half per cent. If the patient coughs or objects, nudge it back one step and wait for six clear breaths (without swallowing or breath-holding) and talk reassuringly to the patient saying all is well - then increase the ether concentration slowly. All the time keep talking gently, quietly and confidently until you have reached an ether concentration of around 10%.

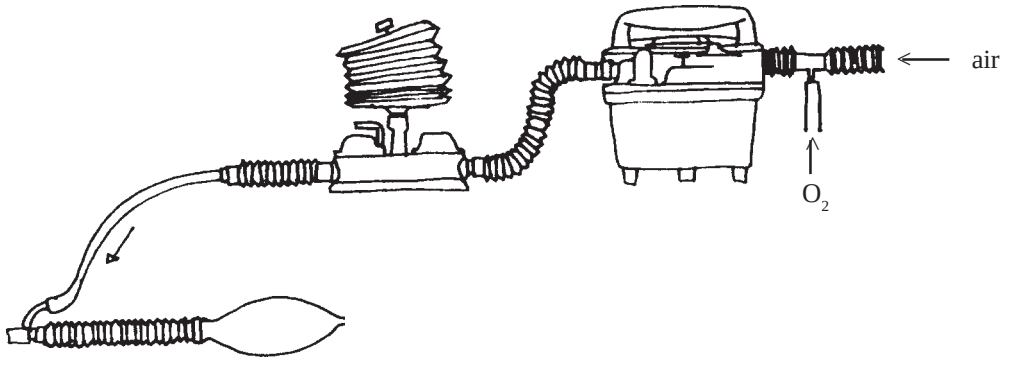


Fig.1. A slow steady pumping action will provide a near continuous flow of ether vapour.

Keep your finger on the patient's pulse during the entire induction, or better still have a paediatric stethoscope attached to his chest. Keep listening and watching the chest to make sure that air is going in and out freely. In stage 1 of anaesthesia, the eyelash reflex goes. In stage 2 there may be some struggling, and the patient will need reassured while an assistant holds their limbs. The patient may vomit at this stage and need to be turned on their side and have their pharynx cleared with suction. During this stage the pupils may dilate, and breathing begins to become irregular. In stage 3 (the stage of surgical anaesthesia) there are 4 described planes. The first is plane 1 during which the respiration becomes more regular but the pupils are not central. The second is plane 2 when the pupils are central and it is safe to insert a Guedel airway. With deeper anaesthesia the pupils may begin to dilate (although the atropine may mask this).

Minor surgery can be done in plane 1 (incision of abscesses etc.). Most general surgery can be performed in plane 2. In plane 3 there is progressive paralysis of the intercostal muscles, which can be detected by careful observation of the pattern of breathing. If it becomes "see -saw" in character, with paradoxical movement of the chest (falling instead of rising on inspiration) anaesthesia is becoming too deep and the face mask should be removed for a few breaths whilst reducing the ether concentration to a more satisfactory maintenance level. In general this is 6 to 8% for surgical procedures that do not require any muscular relaxation, or nearer 8-10% for intra-abdominal procedures requiring relaxation. Be careful not to overdose the patient.

Stage 4 of ether anaesthesia is marked by shallow respiration and if it occurs should be treated immediately by removing the mask and reducing the ether concentration. Be prepared to assist ventilation and if respiratory arrest occurs switch the ether setting to "Transit" and ventilate the patient via the T-piece.

During the operation continue to monitor the child closely paying particular attention to his colour, airway, respiration and cardiovascular signs. Towards the end of the operation allow the patient to lighten by adjusting the ether setting towards 2% or less. With abdominal surgery do not do this too early as closure may prove difficult. At the end of the operation the patient should be breathing air and the face mask may be removed. Ensure that the airway is completely clear before handing the patient over to the nurse in the recovery position.

Practical points: Aim at a steady level of anaesthesia and do not allow the patient to get too deep. Never let the surgeon start until you are ready. If laryngeal stridor develops, allow a few breaths of air and then continue more slowly. However, if spasm develops during surgery, it means the patient is too light for the procedure. Ask the surgeon to stop and deepen anaesthesia until the stridor stops. Add some oxygen to the circuit if possible.

The safety of this technique is considerably increased by adding oxygen to the inspired mixture. It should be put into the circuit upstream (before) of the EMO vaporiser using some sort of reservoir. Around 1 litre/minute is adequate during maintenance. During induction this should be increased to around 3 to 4 litres/minute.

The technique may also be used with intubation and controlled ventilation. This is the preferred technique for longer procedures and in smaller children. If muscle relaxants are used then an intravenous induction may be performed, less ether will be required for maintenance and recovery will be faster.

Editor's note: The EMO can also be used with a T piece by connecting the vaporiser inlet to a source of continuous gas flow (eg Boyles machine, oxygen cylinder etc). Connect the T piece to the outlet of the EMO. The EMO does not work so efficiently when used as a plenum vaporiser and needs a continuous fresh gas flow of 10 litres/minute to produce adequate concentrations of ether.

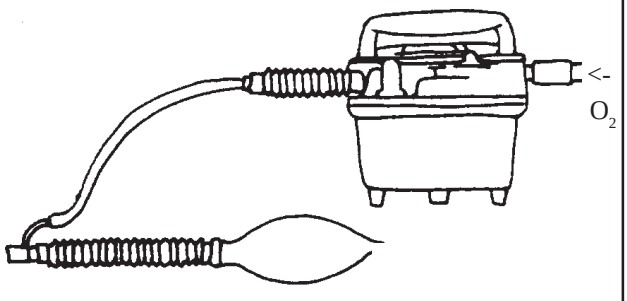


Fig.2.

UPDATE ON THE INTERNET

Dr Michael Dobson
Consultant Anaesthetist.

You can now receive an electronic version of Update in Anaesthesia - you can read it on your computer screen (using free software which we provide). You can use the electronic version whether or not your computer is connected to the internet.

The electronic version of Update in Anaesthesia contains almost all the articles ever published in Update, together with illustrations. The software allows you to search, read and print out selected material, or to print whole issues if you wish. You can make as many reprints as you want whenever you need them. This should be of considerable help to teaching centres.

You do not need the latest and most expensive sort of computer to make use of Electronic Update. A 286 PC should be adequate. If you need technical advice please write to Dr. Mike Dobson, Nuffield Dept of Anaesthetics, John Radcliffe Hospital, Oxford OX3 9DU, UK or send an email request to michael.dobson@ndm.ox.ac.uk

We will need to know a few details about your computer model and operating system [e.g. DOS, Windows 3.x, Windows 95] to ensure that we send the correct software for you to use. If you don't have internet access but do have a computer we can post you the same materials on floppy disk with instructions for use (this offer applies only to addresses in developing countries).

If you have an internet connection you can access Update at

http://www.nda.ox.ac.uk/wfsa/

This site also has links through which you can download the necessary free software to use Update off-line, as well as downloading the issues themselves. (To download a complete issue -about 300k - via a telephone link will take 2-3 minutes)

If you have a local contact or (for example) British Council library, with internet access, you can ask them to do the downloading and printing for you. Update is supported by the UK Department for International Development (formerly ODA)

Electronic Update has been available for only a few months, but has already been used by anaesthetists in 56 different countries.

PRACTICAL PROCEDURE FOR DEALING WITH STICKING OMV'S

Mike Yeats, Equipment Maintenance, Derriford Hospital, Plymouth

The Oxford Miniature Vaporiser is designed for drawover anaesthesia and rarely give problems. Occasionally the pointer becomes stiff to rotate due to deposits of thymol left behind by halothane. The following simple procedure should be carried out once a month or whenever the control lever become stiff to operate.

Place a bung in the inlet side of the OMV, turn the vaporiser on to its side and pour in some some ether or methylated spirits or halothane whilst moving the pointer to and fro. The vaporiser should be completely filled and allowed to stand for 5 minutes before emptying. If you are using ether or halothane perform this procedure outside to avoid inhaling the fumes. Empty the fluid out of the vaporiser and blow it through with air for 10 - 15 minutes. If this procedure does not free the lever the next issue of Update will describe how to deal with a completely jammed vaporiser.

Sponsored by: World Federation of Socieities of Anaesthetists
Typset by: Clinical Graphics, Royal Devon & Exeter Healthcare NHS Trust, Exeter, UK.
Printed in Great Britain by: Media Publishing
Correspondence to: Dr I H Wilson, Anaesthetics Dept., Royal Devon & Exeter Healthcare NHS Trust, Barrack Road Exeter, EX2 5DW, UK.
Subscriptions to: Dr Ray Sinclair, Department of Anaesthesia, Royal Truro Hospital(Treliske) Truro, Cornwall, UK.