UPDATE IN ANAESTHESIA

WORLD ANAESTHESIA

A journal for anaesthetists in developing countries

WORLD ANAESTHESIA

EDITORIAL

The WHO and UNICEF recently reported that in developing countries maternal mortality ratios range from 190 per 100,000 live births in Latin America and the Caribbean to 870 per 100,000 in Africa. Extremely high ratios of over 1000 per 100,000 live births are found in Eastern and Western Africa, where the UN have estimated that up to one of seven women die from pregnancy-related causes. The most common causes of death during pregnancy include haemorrhage, abortion, sepsis and obstructed labour.

Caesarean section is one of the commonest emergency operations and anaesthetists have a major role in the care of the obstetric patient in both theatre and the labour suite. This edition of Update reviews aspects of obstetric anaesthesia by authors from Nepal, South Africa and USA.

We also include two basic science articles, including a self assessment section and a review of some recent publications from international anaesthesia journals. We are aware that our readership is very varied and we always appreciate feedback on Update. Constructive criticism is essential to improve the journal. Update and

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our sister publication "World Anaesthesia" can both be found at our website http://www.nda.ox.ac.uk/wfsa

PHYSIOLOGICAL CHANGES ASSOCIATED WITH PREGNANCY

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Physiological and anatomical alterations develop in many organ systems during the course of pregnancy and delivery. Early changes are due, in part, to the metabolic demands brought on by the fetus, placenta and uterus and, in part, to the increasing levels of pregnancy hormones, particularly those of progesterone and oestrogen. Later changes, starting in mid-pregnancy, are anatomical in nature and are caused by mechanical pressure from the expanding uterus. These alterations create unique requirements for the anaesthetic management of the pregnant woman.

CARDIOVASCULAR SYSTEM

The pregnancy-induced changes in the cardiovascular system develop primarily to meet the increased metabolic demands of the mother and fetus.

Blood Volume increases progressively from 6-8 weeks gestation (pregnancy) and reaches a maximum at approximately 32-34 weeks with little change thereafter. Most of the added volume of

blood is accounted for by an increased capacity of the uterine, breast, renal, striated muscle and cutaneous vascular systems, with no evidence of circulatory overload in the healthy pregnant woman. The increase in plasma volume (40-50%) is relatively greater than that of red cell mass (20-30%) resulting in hemodilution and a decrease in haemoglobin concentration. Intake of supplemental iron and folic acid is necessary to restore hemoglobin levels to normal (12 g/dl). The increased blood volume serves two purposes. First, it facilitates maternal and fetal exchanges of respiratory gases, nutrients and metabolites. Second, it reduces the impact of maternal blood loss at delivery. Typical losses of 300-500 ml for vaginal births and 750-1000 ml for Caesarean sections are thus compensated with the so-called "autotransfusion" of blood from the contracting uterus.

Blood Constituents. As mentioned above, red cell mass is increased 20-30%. Leukocyte counts are variable during gestation, but usually remain within the upper limits of normal. Marked elevations, however, develop during and after parturition (delivery). Fibrinogen, as well as total body and plasma levels of factors VII, X and XII increase markedly. The number of platelets also rises, yet not above the upper limits of normal. Combined with a decrease in fibrinolytic activity, these changes tend to prevent excessive bleeding at delivery. Thus, pregnancy is a relatively hypercoagulable state, but during pregnancy neither clotting or bleeding times are abnormal.

Cardiac Output increases to a similar degree as the blood volume. During the first trimester cardiac output is 30-40% higher than in the non-pregnant state. Steady rises are shown on Doppler echocardiography, from an average of 6.7litres/ minute at 8-11weeks to about 8.7litres/minute flow at 36-39 weeks; they are due, primarily, to an increase in stroke volume (35%) and, to a lesser extent, to a more rapid heart rate (15%). There is a steady reduction in systemic vascular resistance (SVR) which contributes towards the hyperdynamic circulation observed in pregnancy.

During labor, further increases are seen with pain in response to increased catecholamine secretion; this increase can be blunted with the institution of labour analgesia. Also during labour, there is an increase in intravascular volume by 300-500 ml of blood from the contracting uterus to the venous system. Following delivery this autotransfusion compensates for the blood losses and tends to further increase cardiac output by 50% of pre-delivery values. At this point, stroke volume is increased while heart rate is slowed.

Cardiac Size/Position/ECG. There are both size and position changes which can lead to changes in ECG appearance. The heart is enlarged by both chamber dilation and hypertrophy. Dilation across the tricuspid valve can initiate mild regurgitant flow causing a normal grade I or II systolic murmur. Upward displacement of the diaphragm by the enlarging uterus causes the heart to shift to the left and anteriorly, so that the apex beat is moved outward and upward. These changes lead to common ECG findings of left axis deviation, sagging ST segments and frequently inversion or flattening of the T-wave in lead III.

Blood Pressure. Systemic arterial pressure is never increased during normal gestation. In fact, by midpregnancy, a slight decrease in diastolic pressure can be recognized. Pulmonary arterial pressure also maintains a constant level. However, vascular tone is more dependent upon sympathetic control than in the nonpregnant state, so that hypotension develops more readily and more markedly consequent to sympathetic blockade following spinal or extradural anaesthesia. Central venous and brachial venous pressures remain unchanged during pregnancy, but femoral venous pressure is progressively increased due to mechanical factors.

Aortocaval Compression. From mid-pregnancy, the enlarged uterus compresses both the inferior vena cava and the lower aorta when the patient lies supine. Obstruction of the inferior vena cava reduces venous return to the heart leading to a fall in cardiac output by as much as 24% towards term. In the unanaesthetised state, most women are capable of compensating for the resultant decrease in stroke volume by increasing systemic vascular resistance and heart rate. There are also alternative venous pathways, the paravertebral and azygos systems. During anesthesia, however, these compensatory mechanisms are reduced or abolished so that significant hypotension may rapidly develop.

Obstruction of the lower aorta and its branches causes diminished blood flow to kidneys, uteroplacental unit and lower extremities. During the last trimester, maternal kidney function is markedly lower in the supine than in the lateral position. Furthermore, the fetus is compromised by insufficient transplacental gas exchange.

Venous Distension increases approximately to 150% during the course of gestation and the venous ends of capillaries become dilated, causing reduced blood flow. These vascular changes contribute to delayed absorption of subcutaneously or intramuscularly injected substances. Distension of the extradural veins heightens the risk of vascular damage during institution of a regional block. The increased venous volume within the rigid spinal canal reduces the volume or capacity of the extradural and intrathecal spaces for local anaesthetic solutions. This will therefore increase the spread of injected drugs.

Clinical Implications. Despite the increased workload of the heart during gestation and labour, the healthy woman has no impairment of cardiac reserve. In contrast, for the gravida with heart disease and low cardiac reserve, the increase in the work of the heart may cause ventricular failure and pulmonary oedema. In these women, further increases in cardiac workload during labour must be prevented by effective pain relief, optimally provided by extradural or spinal analgesia. Since cardiac output is highest in the immediate postpartum period, sympathetic blockade should be maintained for several hours after delivery and then weaned off slowly.

> **TEACHING POINT**
> *There is a 30% reduction in volume of local anaesthetic solution required at term when compared to the non-pregnant woman, to achieve the same block.*
>
> *Aortocaval compression and its sequelae must be avoided. No woman in late pregnancy should lie supine without shifting the uterus off the great abdomino-pelvic vessels. During labour, the parturient should rest on her side, left or right. During Caesarean section and for other indications demanding the supine position, the uterus should be displaced, usually to the left, by placing a rigid wedge under the right hip and/or tilting the table left side down.*

RESPIRATORY SYSTEM

Changes within the respiratory system are of great significance to the anaesthetist.

Respiratory Tract. Hormonal changes to the mucosal vasculature of the respiratory tract lead to capillary engorgement and swelling of the lining in the nose, oropharynx, larynx, and trachea. Symptoms of nasal congestion, voice change and upper respiratory tract infection may prevail throughout gestation. These symptoms can be exacerbated by fluid overload or oedema associated with pregnancy-induced hypertension (PIH) or pre-eclampsia. In such cases, manipulation of the airway can result in profuse bleeding from the nose or oropharynx; endotracheal intubation can be difficult; and only a smaller than usual endotracheal tube may fit through the larynx. Airway resistance is reduced, probably due to the progesterone-mediated relaxation of the bronchial musculature.

Lung Volumes. Upward displacement by the gravid uterus causes a 4 cm elevation of the diaphragm, but total lung capacity decreases only slightly because of compensatory increases in the transverse and antero-posterior diameters of the chest, as well as flaring of the ribs. These changes are brought about by hormonal effects that loosen ligaments. Despite the upward displacement, the diaphragm moves with greater excursions during breathing in the pregnant than in the non-pregnant state. In fact, breathing is more diaphragmatic than thoracic during gestation, an advantage during supine positioning and high regional blockade.

From the middle of the second trimester, expiratory reserve volume, residual volume and functional residual volume are progressively decreased, by approximately 20% at term. Lung compliance is relatively unaffected, but chest wall compliance is reduced, especially in the lithotomy position.

Ventilation and Respiratory Gases. A progressive increase in minute ventilation starts soon after conception and peaks at 50% above normal levels around the second trimester. This increase is effected by a 40% rise in tidal volume and a 15% rise in respiratory rate (2-3 breaths/minute). Since dead space remains unchanged, alveolar ventilation is about 70% higher at the end of gestation. Arterial and alveolar carbon dioxide tensions are decreased by the increased ventilation. An average $PaCO_2$ of

32mmHg (4.3 kPa) and arterial oxygen tension of 105mmHg (13.7 kPa) persist during most of gestation. The development of alkalosis is forestalled by compensatory decreases in serum bicarbonate. Only carbon dioxide tensions below 28mmHg (3.73 kPa) will lead to a respiratory alkalosis.

During labour, ventilation may be further accentuated, either voluntarily (Lamaze method of pain control and relaxation) or involuntarily in response to pain and anxiety. Such excessive hyperventilation results in marked hypocarbia and severe alkalosis, which can lead to cerebral and uteroplacental vasoconstricton and a left shift of the oxygen dissociation curve. The latter reduces the release of oxygen from haemoglobin with consequent decreased maternal tissue oxygenation as well as reduced oxygen transfer to the fetus. Furthermore, episodes of hyperventilation may be followed by periods of hypoventilation as the blood carbon dioxide tension ($PaCO_2$) returns to normal. This may lead to both maternal and fetal hypoxia.

Oxygen consumption increases gradually in response to the needs of the growing fetus, culminating in a rise of at least 20% at term. During labour, oxygen consumption is further increased (up to and over 60%) as a result of the exaggerated cardiac and respiratory work load.

Clinical Implications. The changes in respiratory function have clinical relevance for the anesthesiologist. Most importantly, increased oxygen consumption and the decreased reserve due to the reduced functional residual capacity, may result in rapid falls in arterial oxygen tension despite careful maternal positioning and preoxygenation. Even with short periods of apnea, whether from obstruction of the airway or inhalation of a hypoxic mixture of gas, the gravida has little defense against the development of hypoxia. The increased minute ventilation combined with decreased functional residual capacity hastens inhalation induction or changes in depth of anaesthesia when breathing spontaneously.

GASTROINTESTINAL SYSTEM
Since aspiration of gastric contents is an important cause of maternal morbidity and mortality in association with anesthesia, an examination of the controversy surrounding gastrointestinal changes in pregnancy is justified.

Mechanical Changes. The enlarging uterus causes a gradual cephalad displacement of stomach and intestines. At term the stomach has attained a vertical position rather than its normal horizontal one. These mechanical forces lead to increased intragastric pressures as well as a change in the angle of the gastroesophageal junction, which in turn tends toward greater oesophageal reflux.

Physiological Changes. The hormonal effects on the gastrointestinal tract are an issue of debate among anaesthetists. Relaxation of the lower oesophageal sphincter has been described, but there have been differing views about the effect on motility of the gastrointestinal tract and the times at which it is most prominent. Many believe that there is also retardation of gastrointestinal motility and gastric emptying, producing increased gastric volume with decreased pH, beginning as early as 8-10 weeks of gestation. Recent studies, however, have shed a different light on the subject. Measuring peak plasma concentrations of drugs absorbed exclusively in the duodenum in both non-pregnant and pregnant volunteers, at different times of gestation, it was shown that peak absorption occurred at the same interval in all women with the exception those in labour. This suggests that gastric emptying is delayed only at the time of delivery.

Thus, the raised risk of aspiration is due to an increase of oesophageal reflux and decreased pH of gastric contents. The heightened incidence of difficult endotracheal intubations worsens the situation.

> **TEACHING POINT**
> *The gravida should be considered a to be a "full stomach" patient with increased risk of aspiration during most of gestation.*

Pulmonary Aspiration of gastric contents can occur either following vomiting (active) or regurgitation (passive). Aspiration of solid material causes atelectasis, obstructive pneumonitis or lung abscess, while aspiration of acidic gastric contents results in chemical pneumonitis (Mendelson's syndrome). The most serious consequences follow aspiration of acidic materials containing particulate

matter as may follow swallowing certain antacids such as magnesium trisilicate. Clear antacids such as sodium citrate (0.3 Mol) or bicarbonate should be used. While the incidence of pulmonary aspiration of solid food has decreased due to patient education, that of gastric acid has remained constant.

Clinical Implications. The danger of aspiration is almost eliminated when regional anaesthesia or inhalational analgesia is used. During general anaesthesia airway protection by means of a cuffed endotracheal tube is mandatory. Although awake intubation is safest, discomfort and the lack of patient cooperation and discomfort prevent it being the routine method for securing the airway. The endotracheal tube is placed immediately following loss of consciousness after induction of general anesthesia.

TEACHING POINT

Special precautions should be heeded, even when induction to intubation time is expected to be brief, to prevent the regurgitation:

a) *supine position with lateral tilt to minimise any increase in intragastric pressure*

b) *preoxygenation prior to induction then no positive pressure ventilation prior to insertion of the endotracheal tube to prevent distention of the stomach with gas (rapid sequence induction)*

c) *cricoid pressure (Sellick's maneuvre) during induction which is maintained until endotracheal tube placement in the trachea has been confimed. Cricoid pressure should be applied to the cricoid cartilage whilst supporting the back of the neck. This occludes the oesophagus, thus obstructing the path of regurgitation.*

The acidity and volume of gastric content can be reduced by pharmacologic interventions which may prove invaluable. Most importantly, a nonparticulate oral antacid, 30ml of sodium citrate 0.3 Mol or bicarbonate, should be given immediately prior to induction of general anesthesia to all women. In addition, if available, metoclopramide, 10 mg IV, should be administered 15 - 30 minutes before induction to promote gastric emptying and increase the lower oesophageal sphincter tone. This is especially beneficial in women in labour who have not been starved and and require emergency surgery. Lastly, histamine H_2 - receptor antagonist the night before and the morning of delivery may reduce secretion of hydrochloric acid (ranitidine 150mg orally).

METABOLISM

All metabolic functions are increased during pregnancy to provide for the demands of fetus, placenta and uterus as well as for the gravida's increased basal metabolic rate and oxygen consumption. Protein metabolism is enhanced to supply substrate for maternal and fetal growth. Fat metabolism increases as evidenced by elevation in all lipid fractions in the blood. Carbohydrate metabolism, however, demonstrates the most dramatic changes. Metabolically speaking, pregnant women live in a state of "accelerated starvation." First, nutritional demands of the growing fetus are met by the intake of glucose and, second, secretion of insulin in response to glucose is augmented. As early as 15 weeks of gestation, maternal blood glucose levels after an overnight fast are considerably lower than in the nongravid state.

Hypoglycaemia. Optimal blood glucose levels in pregnant women range between 4.4 to 5.5mmol/1 (80 to 100mg/dl). In healthy non-pregnant individuals, signs of hypoglycaemia usually begin when the blood glucose level declines to approximately 2.2mmol/1 (40mg/dl); in pregnant women, however, hypoglycaemia is defined as a concentration below 3.3mmol/1 (60mg/dl). Hypoglycaemia initiates the release of glucagon, cortisol and, importantly, catecholamines. In the anaesthetised state, however, these compensatory mechanisms, particularly the release of epinephrine (adrenaline), are blocked. Autonomic derangements in the form of hypotension and tachycardia tend to ensue during high regional blockade or deep general anaesthesia, which may mask the symptoms and signs of hypoglycaemia.

RENAL PHYSIOLOGY

Renal plasma flow and glomerular filtration rate begin to increase progressively during the first trimester. At term, both are 50-60% higher than in the non-pregnant state. This parallels the increases in blood volume and cardiac output. The elevations in plasma flow and glomerular filtration result in an

elevation in creatinine clearance. Blood urea and serum creatinine are reduced by 40%. The increase in glomerular filtration may overwhelm the ability of the renal tubules to reabsorb leading to glucose and protein losses in the urine. Thus, mild glycosuria (1-10gm/day) and/or proteinuria (to 300mg/day) can occur in normal pregnancy. There is also an increase in filtered sodium, but tubular absorption is increased by an increase in aldosterone secretion, via the renin-angiotensin mechanism (see Physiology of the Kidney page24). There is also a decrease in plasma osmolality. This is a measure of the osmotic activity of a substance in solution and is defined as the number of osmoles in a kilogram of solvent. In practice it indicates that the plasma concentrations of electrolytes, glucose and urea, fall if more water than sodium, for example, is retained. Over the whole period of gestation there is retention of 7.5L of water and 900mmol of sodium.

After the 12th week of gestation, progesterone can induce dilation and atony of the renal calyses and ureters. With advancing gestation, the enlarging uterus can compress the ureters as they cross the pelvic brim and cause further dilatation by obstructing flow. These changes may contribute to the frequency of urinary tract infections during pregnancy. The effect of postural compression of the aortic branches perfusing the kidneys has been discussed.

DRUG RESPONSES

The response to anaesthetic and adjuvant drugs is modified during pregnancy and the early puerperium. The most pertinent alteration is a reduced drug requirement, manifest in both regional and general anaesthesia.

Regional Anaesthesia. From the late first trimester to the early puerperium, a smaller dose of local anaesthetic is required to obtain the desired level of spinal or extradural blockade. During the last months of gestation, approximately two-thirds of the normal dose is adequate. This altered response, which is due to CSF and hormonal changes and an increase in volume of the epidural veins, subsides progressively in the early postpartum period.

General Anaesthesia. Induction and changes in depth of inhalation anaesthesia occur with greater rapidity in pregnant women than in non-pregnant subjects. Pregnancy enhances anaesthetic uptake in two ways. The increase in resting ventilation delivers more agent into the alveoli per unit time, while the reduction in functional residual capacity favors rapid replacement of lung gas with the inspired agent. In addition, there is a reduction in anaesthetic requirements, with a fall in the minimum alveolar concentrations (MAC) of halogenated vapors. When measured in ewes MAC was 25-40% lower in gravid as compared with nonpregnant animals.

The decreased functional residual capacity has a further effect on the management of general anaesthesia. As referred to earlier, the resultant reduction in oxygen storage capacity, together with the elevated oxygen consumption, leads to an unusually rapid decline in arterial oxygen tension in the apnoeic anaesthetised gravida.

There are also alterations in the response to intravenous agents, in particular prolongation of their elimination half-lives consequent to the greater distribution volume (resulting from the pregnancy-induced increase in plasma volume). Thus, the mean elimination half-life for thiopentone in gravid women is more than doubled in comparison with that in nongravid young patients.

Serum Cholinesterase. Serum cholinesterase levels fall by 24-28% during the first trimester without a marked change for the remainder of gestation. However, even lower levels (about 33% reduction) develop during the first 7 postpartum days. The decreased levels of the enzyme are still sufficient for normal hydrolysis of clinical doses of suxamethonium or chloroprocaine during gestation. Postpartum, however, approximately 10% of women will be at risk of a prolonged reaction to suxamethonium.

Clinical Implication. These altered drug responses must be taken into consideration whenever a patient is pregnant or in the early puerperium.

ANAESTHESIA FOR CAESAREAN SECTION

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Caesarean section (LSCS) is one of the commonest operations performed in the developing world and is often carried out in difficult circumstances. As with any operation, the anaesthetist should first think about all the problems that may occur as it is always better to be prepared for trouble than to be taken by surprise.

The problems concern 5 areas:

1. The patients

2. The surgery (and the surgeon!)

3. The drugs (both anaesthetic drugs and any taken by the patient)

4. Equipment

5. The anaesthetist

1. Problems with the patients

Caesarean section is often said to be the unique situation where the anaesthetist has to deal with 2 patients under the same anaesthetic. The health of the baby has to be considered as well as that of the mother.

Risks to the mother. Changes in maternal physiology are described elsewhere in this journal, as are problems associated with hypertensive disease of pregnancy. Any other significant concurrent disease, such as maternal diabetes or sickle cell disease, will have to be handled in the usual way. The important changes affecting anaesthesia are:

Pregnant women are at risk of hypoxia. They are more difficult to oxygenate than non-pregnant patients due to changes in their respiratory mechanics and they use the oxygen more quickly because of a higher metabolic rate. This situation can be made worse by other factors. Obesity makes control of the airway more difficult and interstitial fluid retention may make the larynx harder to visualise for successful intubation.

Although fluid retention is a feature of pregnancy, a more common problem is the risk of hypovolaemia either due to obstetric complications causing significant antepartum haemorrhage or, very commonly, prolonged labour leading to exhaustion and dehydration. This is particularly noticeable in the hot season.

The pregnant mother is at greater risk of pulmonary acid aspiration, as regurgitation of acidic stomach contents is more likely than in non-pregnant patients. This can lead to catastrophic aspiration pneumonitis.

The patient with hypertensive disease of pregnancy may have abnormal clotting function and multiple other complications of this disease.

Risks to the fetus include hypoxia and acidosis if placental blood flow is reduced. Since maternal blood pressure is maintained at the expense (if necessary) of placental perfusion, by the time a significant drop in maternal blood pressure has been measured the fetus has already suffered from reduced placental perfusion. The general condition of the fetus should be considered.

What is the state of the fetus preoperatively? How significant is any "fetal distress"? Is there an obstetric complication, such as cord prolapse, that puts the fetus at imminent risk and requires the quickest possible intervention? Are there more than one fetus?

Risks to mother and fetus. Both need to be protected from the "supine hypotensive syndrome" (aorto-caval compression). This occurs when the maternal inferior vena cava and, to a lesser extent, the aorta are compressed by the gravid uterus if the mother is allowed to lie on her back.

2. Problems with the surgery

Ask yourself the following questions:

Who is the surgeon, how experienced, how long does he expect to take and what incision is planned? Are blood and other intravenous fluids available? Is there a surgical complication such as placenta praevia that could cause serious intra-operative haemorrhage?

Does your surgeon lift the uterus right out of the abdominal cavity after delivery in order to suture it? (Under regional anaesthesia this is very uncomfortable and is rarely necessary.)

3. Problems with drugs

As with any patient, the pregnant woman may be taking drugs for concurrent diseases which have to be considered, e.g. steroids, anti diabetic medication. They may also be taking drugs that can react with anaesthetic drugs, e.g. antidepressant medication.

With all drugs, beware of the weight of the patient and try and weigh her if possible. Do not believe average doses quoted in textbooks but give drugs as mg/kg. This is particularly important in Asia where, in the authors' experience, fully grown women at term may only weigh 35 to 40kg.

There is a moderate reduction in psuedo-cholinesterase in pregnant women compared with the non-pregnant population (at least in Caucasians). This is more notable immediately post-partum. Although the initial dose of suxamethonium is the same, its effect may be prolonged. If suxamethonium has not been correctly stored it may not be fully effective.

Ketamine causes a rise in blood pressure. It should not be given to mothers with hypertension but is well worth considering if a mother is being resuscitated from hypovolaemia. Ergometrine, given to encourage uterine contraction immediately after delivery, frequently causes nausea and vomiting. It is better to use oxytocin in the awake patient having a regional or local anaesthetic.

Are all general anaesthesia including emergency drugs available?
Drugs used for the anaesthetic may affect the fetus. Anaesthetic drugs cross the placenta and therefore a "deep" anaesthetic will sedate the baby and risk birth apnoea. Narcotics and sedatives should not be given to the mother prior to delivery. Gallamine crosses the placenta and will affect the fetus. Other neuromuscular blocking agents are safe.

4. Problems with equipment

What anaesthetic equipment is available? Is there adequate oxygen , either in cylinders or as a functioning oxygen concentrator? Is the power supply reliable?

Does the sucker work and is there a back up manually operated sucker?

Does the table tilt and is there a suitable wedge available?

Is there a range of equipment for difficult intubation: introducers, a range of laryngoscope blades and handles and endotracheal tubes ?

Is there resuscitation equipment ready for the patient having a regional anaesthetic? What resuscitation equipment is ready for the baby?

What sterile needles are available for spinal anaesthesia?

Is there any monitoring equipment available ?

5. Problems with the anaesthetist

Finally, you should consider how experienced you are with any particular technique and how long you expect to take. *Can you obtain the help of another anaesthetist?* This is a good policy if you are expecting a difficult intubation or other problems. Lastly, and probably as important as anything else, *do you have a trained assistant? Do they know how to do cricoid pressure correctly? Are they strong enough to turn the patient on to her side if you get into trouble?*

Having considered all the potential difficulties, make a plan for your anaesthetic.

Plan for Anaesthesia

Preoperative preparation

Peroperative   induction
                           maintenance
                           recovery

Postoperative care

PREOPERATIVE PREPARATION

Visit the patient , take a history and examine them. Consider the state of the maternal cardiovascular system, whether or not you expect a difficult intubation and also the state of the fetus. Give antacid as described in the table 1. If ranitidine is not available, give antacid pre-operatively.

Explain the type of anaesthetic that you plan to use and what the patient can expect to happen before, during and after surgery. Try to gain the patient's, and her family's agreement for what you plan to do. If the patient is at high risk then this should be explained to the family concerned.

Table 1

Elective case	Ranitidine 150 mg orally the night before and 90 minutes pre op
	Sodium Citrate 30 mls orally immediately pre op
Emergency	Ranitidine 50 mg IV immediately decision made to operate
	Sodium Citrate 30 mls orally immediately pre op
High risk labour	Ranitidine 150 mg orally 6 hrly (e.g. Diabetic)
	Sodium Citrate 30 mls orally if proceeds to Caesarean section

Premedication should not be given because it will depress the baby's respiration and conscious level at birth. If naloxone is available for the baby (or nalbuphine as a second-best alternative), then pethidine may be given to the mother during labour. A good alternative to pethidine in labour is to use inhalational analgesia, either with a 50/50 mixture of nitrous oxide and oxygen (Entonox), or with trichloroethylene (Trilene) in air. Trilene is easy to use, cheap and effective. It can be used for analgesia in labour and other situations, e.g. change of burns dressings, setting of simple fractures. The simplest system for delivery is a draw over vapouriser (e.g. OMV) with some tubing, a face-mask and a one-way valve at the patient end. Note that it must never be used in the presence of soda lime. Provided about 1% Trilene is used (half way between max and min on a Cyprane inhaler scale) and the mother holds the mask herself, it is safe. If she begins to move from analgesia to anaesthesia then she will drop the mask and become fully conscious again.

ANAESTHESIA

Three anaesthetic techniques are possible:

1. Local infiltration anaesthesia with or without supplementation.

2. Regional anaesthetic

3. General anaesthetic

Before starting anaesthesia check the following:

● Antacids, sucker, availability of blood, oxygen and an assistant who can do cricoid pressure.

● Establish good IV access with a reliable large bore cannula and start an IV infusion of Normal Saline or Ringer's Lactate (Hartmann's solution).

● Place a wedge on the operating table, under the right side of the patient so that she is tilted to the left by 15 to 20 degrees.

● Always have at hand the drugs and equipment necessary to perform an urgent general anaesthetic or resuscitation of mother or child. Whatever technique you start with, you may end up giving a general anaesthesic.

1. Local Anaesthetic (LA)

The local anaesthetic infiltration is normally carried out by the surgeon. Work out the maximum safe dose of the drug being used and add adrenaline at the rate of 5 micrograms per ml of LA. This is a 1 in 200,000 solution of adrenaline (easily made by adding 0.1ml of adrenaline 1:1000 to each 20mls of LA).

If available give oxygen to the mother until delivery.

Using a 100mm needle two long bands of skin are infiltrated either side of the proposed incision. Keep the needle parallel to the skin and beware that the abdominal wall is very thin at term. Do not stick the needle into the uterus. After incising the skin, the rectus sheath is infiltrated. In order to anaesthetise the parietal peritoneum, a further 10mls of solution is injected under the linea alba, once it is reached and, lastly, 5mls is injected into the loose visceral peritoneum of the uterus at the point of the incision in the lower segment.

Reassure the patient and explain that after the local anaesthetic has been given, she will still feel certain sensations of touch. She may experience discomfort if the head is well engaged in the pelvis. However, the anaesthetic will prevent her feeling significant pain.

Supplementation is a problem because of the effects on the fetus and the first choice is to give nothing until the cord is clamped, after which small doses of narcotic or sedative may be used. Probably the safest supplementation is nitrous oxide in oxygen or Trilene in air (+/- oxygen), as described above.

Ketamine should be used cautiously, in as low a dose as possible, and only intravenously. In analgesic doses of 0.25mg/kg, it has little effect on the baby and, although it crosses the placenta easily, doses up to a total of 1mg/kg can be used. Full anaesthetic doses of 2mg/kg will sedate the baby and may cause chest wall rigidity which complicates resuscitation. If ketamine is used then diazepam or promethazine should be given to reduce the problems of hallucinations on emergence. These should only be given after the cord has been cut. Ketamine also causes contraction of the uterus and should probably be avoided in cases of significant fetal distress. Atropine may be needed in some adults with ketamine because of excess salivary secretions.

Other points with local and regional anaesthesia:
Atropine is also useful in combating the discomfort and nausea that some patients feel on surgical traction on the peritoneum.

If ergometrine is used to contract the uterus after delivery it will cause vomiting which may be awkward to manage in the supine position during surgery. If it is the only drug available give it very slowly intravenously, preferably with the infusion running. Oxytocin is better, either in a drip at 10-20 units in 1000mls running at 2 to 3mls/minute or a 5 to 10 units IV bolus slowly intravenously.

NB. if these drugs are mistakenly given prior to delivery, constriction of the uterus can be a catastrophe for the baby. There is no need to draw up the oxytocin until it is needed after delivery of the baby and on checking with the surgeon.

The main advantages of local anaesthesia infiltration are:

- It is safe, especially for mothers in poor condition and those who are hypotensive.

- There is a reduction in bleeding because of the adrenaline.

- It is a suitable technique for the single operator / anaesthetist although any supplementation is best avoided.

- It is inexpensive, requiring minimal resources.

The disadvantages are:

- It is not always a perfect technique and the

mother may experience considerable discomfort.

- It takes time to establish and gives less surgical exposure.

- It requires experience on the part of the surgeon.

It is probably most suitable when a reasonably experienced surgeon has limited anaesthetic backup.

2. Regional anaesthetic blockade
Either epidural (extradural) or spinal (subarachnoid) blocks may be used. A combined spinal + epidural technique is commonly practised in UK which has the advantages of a dense subarachnoid block, with the potential for topping up the anaesthetic via the epidural if necessary. In addition the epidural may be used for postoperative analgesia. This combined technique is rarely done in the developing world and will not be further discussed.

Epidural anaesthesia is commonly used in developed countries for analgesia during labour and can therefore easily be used to produce anaesthesia for Caesarean sections with larger doses of local anaesthetic. However, epidurals are technically more difficult to perform than spinal anaesthesia and require more specialised equipment, which is often not available in the developing world. There are significant and potentially fatal complications and they require experienced anaesthetists and midwives for their safe use.

The main advantage of epidurals is that they are suitable for prolonged use e.g., in labour and for post Caesarean section pain relief. Another indication for the experienced anaesthetist is as a choice in patients in poor condition since surgical analgesia can be established slowly with small repeated doses of local anaesthetic, thereby minimising cardiovascular instability. However, since equipment to perform epidurals is often not available, they are not always a practical technique for routine anaesthesia for Caesarean section. Anaesthesia takes longer to develop compared with subarachnoid block and is induced by using increments of either 2% lignocaine with 1:200,000 adrenaline or 0.5% bupivacaine. Note that 0.75% bupivacaine is not recommended for anaesthesia for LSCS. Since epidural anesthesia is not routinely used in many places it will not be further dealt with here.

Spinal anaesthesia has many advantages for anaesthesia for LSCS. The patient is awake and therefore her airway is safe. The baby is not sedated and is usually born in good condition providing hypotension is avoided. If the baby is born with a low Apgar score the anaesthetist is free to resuscitate the baby (unless he is also the surgeon!). With a little experience the technique is as quick as giving a general anaesthetic and provides good operating conditions with some reduction in surgical haemorrhage. It has the advantage of giving good pain relief for several hours after surgery and is straightforward to learn and teach. It is inexpensive and appropriate for virtually all cases except those with unresuscitated preoperative hypovolaemia and those with the specific contraindications of a bleeding disorder, sepsis at the site of injection or allergy to local anaesthetic. It should be avoided in a patient who is suspected of having raised intracranial pressure and patients with hypertensive disease of pregnancy should have clotting function checked. (See also Update in Anaesthesia No. 3)

Explain the technique and its advantages to the patient. There are widely varying views on spinal anaesthesia among different patient populations (and also between surgeons!). Ensure that the patient understands that pain sensation will be abolished but she should expect some pulling and pressure sensation during surgery which may be unpleasant but will be short-lived.

The preparations described above are necessary. i.e. antacids, IV access, wedge, sucker, assistant who can give cricoid pressure, full GA and resuscitation drugs and equipment for mother and child, oxygen until the baby is delivered and blood for transfusion. A sterile surface should be prepared for the procedure with all equipment for spinal anaesthesia available.

Technique:
- Measure a baseline blood pressure
- Pre-load the mother with one litre of normal saline or Ringer's lactate solution (Hartmann's) prior to performing the block.
- Have a vasopressor drawn up and diluted, ready for injection.
- The block can be performed either with the mother sitting up with her feet on a stool and her body bent forward over a pillow on her lap; or lying on her side. The spine should be well flexed. The injection should be at the level L2/3 or L3/4.

- **The injection should be performed with full sterile precautions.** The skin must be prepared with an alcoholic or iodine based skin preparation. The anaesthetist should be wearing sterile gloves and a face mask. A sterile drape should be placed over the patient if they are in the lateral position.

- Explain what will happen to the patient as this will help them to stay still.

- Inject local anaesthetic to the skin.

- While waiting for the local anaesthetic to take effect, draw up the correct dose of the spinal injection which you plan to use and leave it ready beside you on the sterile surface. Ensure that your drawing up needle touches only the inside of the ampoule that you are using. Check the name, concentration and expiry date of the spinal anaesthetic on two occasions. Almost all serious neurological complications of spinal blocks have been due to the wrong drug being injected into the subarachnoid space due to lack of vigilance. Record the batch number and date of expiry of the drug on your anaesthetic chart.

- For the mid-line approach, place a spinal needle introducer between the lumbar spines to a depth of 2 or 3 cms until it is firmly held by the interspinous ligament. If the proper introducer is not available, a size 19 gauge hypodermic needle can be used. Insert the spinal needle with the stillette through the introducer and advance steadily and carefully feeling for the slight extra resistance of the ligamentum flavum followed by the easing of resistance which occurs when the subarachnoid space is entered. This normally lies at a depth of about 4 - 6cms and you can check your progress from time to time by withdrawing the stillette and seeing if CSF flows back.

- On entering the subarachnoid space, hold the the hub of the needle firmly in place by resting the back of the left hand (for a right handed person) on the patient's back and holding the hub between thumb and forefinger. Carefully attach the syringe of spinal anaesthetic solution and withdraw gently on the plunger. CSF should flow back steadily and can be seen as "oily" streaks if a heavy solution is used. If all is well, steadily inject the anaesthetic solution and withdraw the needle and introducer. Apply a small dressing or sticking plaster over the puncture wound.

An alternative approach in difficult cases, or by choice, is the paramedian approach. In this case the local anaesthetic is infiltrated one finger's breadth lateral to the L3 or L4 spinous process. Place the introducer at right angles to the skin, followed by the spinal needle which is advanced straight in until it hits the lamina of the vertebra. Then angle it slightly medially and cephalad (towards the head) and "walk" it off the lamina aiming for the gap between it and the lamina above in the midline. Advance the needle until CSF is found then proceed to inject the local anaesthetic as described above.

Difficulties. If the patient experiences pain the introducer is probably not in the midline but is contacting the periosteum of an adjacent vertebra or in the muscle on one or other side of the ligament. The patient can tell you which side you are on, which will help you to redirect your needle. Withdraw the introducer and reposition it - it should be held firmly by the interspinous ligament. If it moves around freely, it is probably lying to one side of the midline and is not in the ligament. If the subarachnoid space is not found try re-positioning the patient and get your assistant to help flex the patient's back more (that's why they need to be strong!). Alternatively try a different space or the paramedian approach. If blood flows back when you remove the stillette it is probably due to minimal trauma to the small veins in the epidural space. Wait until clear CSF flows and then inject the spinal solution. If it doesn't clear reposition the needle slightly further in or try flushing with sterile saline. If blood continues to flow you must NOT inject the spinal anaesthetic but withdraw the needle and try again in a different space.

After injecting the local anaesthetic, lie the patient down on her right side for 2 or 3 mins then place her tilted to the left on a wedge ready for surgery. This helps ensure that the block spreads to both sides of the abdomen.

The height of the block can be assessed by testing for loss of temperature sensation using ice or cotton wool soaked with ether. Alternatively test gently for loss of pinprick sensation using a sterile needle. The block should be above the umbilicus and preferably towards the xyphisternum. The block works almost immediately, and you can allow the surgeon to proceed after 5 minutes.

Needles. One problem with spinals that has limited their use in the past is the occurrence of significant headache for 2 or 3 days following the procedure. This is due to leakage of CSF through the hole made by the needle and it is more common in pregnant women because the raised CSF pressure, due to dilated epidural veins, causes a bigger leak. The rate of post dural puncture headache is related to the size and design of the needle. Use the smallest gauge needle you have available, preferably 25 or even 26 gauge. If you have to use a 22 gauge spinal needle then you may find that the incidence of headache is unacceptable. When placing the spinal needle try to align the bevel of the needle along the body. This parts the fibres of the dura rather than cuts them and reduces the incidence of headache.

In recent years a new design of needle has been used which has an atraumatic "pencil-point" tip, instead of the standard cutting needle design. These reduce the rate of post puncture headache to less than 1% and are worth considering.

Some spinal needles can be reused, providing they are properly sterilised after each use. The best way of doing this is to use needles with metal hubs that can be re-autoclaved. Some plastic hubs stand up to autoclaving.

Drugs, ampoules and doses. You will have to use whatever drugs are available. A hyperbaric agent (local anaesthetic mixed with glucose) is most useful as it has a quick and predictable onset and usually produces a dense block. If you want to keep the level low and do a saddle block you will need the heavy solution. Solutions injected into the subarachnoid (or extradural) space should always be preservative free and taken from a single dose vial not a multi dose container. Where possible the ampoules should be sterile to make drawing up the solution easier. This can be achieved by buying sterile wrapped ampoules or by autoclaving glass ampoules of local anaesthetic. Never soak ampoules in sterilising solutions. If the ampoules are not sterile on the outside, draw up the drug carefully ensuring complete sterility.

Bupivacaine lasts longer and should be used if prolonged surgery is expected e.g. Caesarean section followed by hysterectomy.

The volume to use is controversial and has been discussed in this journal and elsewhere.

Table 2. Local anaesthetics suitable for spinal anaesthesia

Drug	Volume	Approximate length of action
Bupivacaine (Marcain) 0.5%. hyperbaric or plain	2-2.5 mls	2 - 3 hours
Lignocaine 5% hyperbaric +/- adrenaline 0.2ml of 1:1000	1.2 - 1.6 mls	45 - 90 minutes (with adrenaline)
Lignocaine 2% plain + adrenaline 0.2 ml of 1:1000	2 - 2.5 mls	60 - 120 minutes

Engorgement of epidural veins in pregnancy reduces the volume of CSF and hence a given volume of local anaesthetic will have a wider spread in the pregnant than in the non-pregnant female. The height of the block also depends on the size of the woman. Beware of the small stature of Asian women. At the same time, ethnic groups who are tall will require significantly higher volumes.

In the authors' experience, the following regimes have been effective, giving a block to T5 or T6.

1.2 to 1.4mls of heavy 5% Lignocaine in Nepali women (depending on height)

2.0 to 2.5mls of heavy 0.5% Bupivacaine in Caucasian women (less than 5 ft. to greater than 5ft 6 ins.)

Low spinal blockade. Some authors prefer a low block (T10 - around the height of the umbilicus) which is performed with the patient in the sitting position with a low dose of local anaesthetic (see letters Update in Anaesthesia no 6 & 7).

If the height of this or any block proves inadequate then it may be supplemented with an opioid after delivery. Ketamine, always starting with a low analgesic dose, or inhalational analgesia with air/ Trilene or nitrous oxide and oxygen are alternatives. Great care must be taken to avoid loss of consciousness and inadequate protection of the airway.

Management of hypotension. Sympathetic blockade occurs due to the action of the local anaesthetic on the sympathetic nerves which are easily blocked, often for several segments higher than the dermatomal action. Nearly all patients will have some fall in systolic BP (which is one of the signs of a successful block) and, furthermore, placental blood flow is reduced before maternal systolic BP falls. It is important to take preventative measures to minimise the fall in BP and to act quickly to treat it.

Preload the patient with IV fluid as described and measure the BP before the block and at least 5 minute intervals thereafter.

Make sure the left sided tilt is adequate.

If the systolic BP falls more than 20mmHg from the baseline then speed up the drip and give oxygen. If this does not reverse the hypotension then give a dose of vasoconstrictor (See table 3). These should be diluted and given in small bolus doses every few minutes until the hypotension is treated. Do not tilt the patient head down as this will potentially increase the height of the block. A feeling of nausea is often the first symptom of hypotension.

A good way to give the vasoconstrictor is to dilute it in a drip and start running it slowly as soon as the block is performed unless the patient is hypertensive. (e.g. Ephedrine 60mg in 500ml N Saline). However this is more expensive than giving bolus doses. Although these drugs cause vasoconstriction they increase blood flow to the placenta by raising the cardiac output and improving the placental perfusion pressure.

In a number of patients, the block will be high enough to cover the mid-thoracic sympathetic outflow to the heart (T4-T6) even when the correct dose is used. This prevents a compensatory increase in heart rate and may even cause a significant bradycardia. Severe falls in BP are sometimes seen and should be treated immediately. Give atropine (0.5mg) for bradycardia.

Table 3. Recommended vasopressors

Drug	Action	Initial/Follow-up dose
Ephedrine (best drug)	Vasoconstrictor and increases heart rate with some inotropic action	6-12/3-6mg
Methoxamine (Vasoxine)	Vasoconstrictor only (may see reflex bradycardia)	2-4/2mg
Phenylephrine	Vasoconstrictor only	200/100 micrograms
Adrenaline (use only if other drugs are unavailable)	Vasoconstrictor and increases heart rate ++ and inotropic action	0.5-1ml of 1:10,000 - make by diluting 1ml of 1:1000 to 10mls total with saline

High spinal blocks If the block is high then the patient may complain of tingling or even weakness of the upper limbs. Even though some of the intercostal muscles will be paralysed, the diaphragm is unaffected and these patients should be managed with a slight head-up tilt (to prevent a hyperbaric agent spreading higher), oxygen and reassurance. With these high blocks many patients will complain of an unpleasant feeling of not being able to take a full breath, however, they will be able to speak normally. If the patient gets difficulty in speaking with associated tingling in the arms this is indicative of a very high block which is beginning to affect the diaphragm. These patients are likely to need intubation and ventilation. Remember that a long acting muscle relaxant will not be required.

Total Spinal Very rarely, if too big a dose of local anaesthetic is given, there may be a "total spinal" with paralysis of all respiratory muscles and a respiratory arrest. There will be an associated loss of consciousness and severe hypotension and bradycardia. (This should really only be seen as a complication of epidural anaesthesia when a relatively large dose of local anaesthetic is injected into the subarachnoid space in error).

Immediate resuscitation along the normal lines of Airway, Breathing, Circulation; together with rapid intravenous fluids and large doses of vasoconstrictors will rescue the situation. This is one reason why full resuscitation drugs, equipment and skilled personnel must always be immediately available whenever a regional anaesthetic is given.

Nausea Apart from hypotension, this may be caused by traction on the peritoneum in which case a small dose of atropine may be helpful. Ergometrine will also cause nausea and is best avoided in the awake patient.

Sedation As previously discussed, none should be given prior to delivery, unless it is known that the baby is dead.

Caudal anaesthesia For completeness it should be mentioned that epidural anaesthesia via the sacral hiatus has been used to establish regional anaesthesia for LSCS. However, it gives an unpredictable spread of anaesthesia, slower onset and is not recommended.

3. General Anaesthesia (GA)

General anaesthesia will be necessary if there are contraindications to spinal anaesthesia or if you cannot encourage either the mother or the surgeon to do the operation with the patient awake. The main risk associated with general anaesthesia is that of airway control. There is a significant risk of aspiration of stomach contents and only 30mls of acid aspiration can cause a fatal acid pneumonitis (Mendelson's syndrome). General precautions previously mentioned must be observed.

Ensure good IV access, antacids and left lateral tilt. If it is an emergency with a significant risk of a full stomach then it is safest to pass a large bore nasogastric tube to drain the stomach. Remove the

tube before inducing anaesthesia. Check that the suction is working.

Always use a rapid sequence induction with pre-oxygenation and drugs given by bolus injection according to the patients weight. Cricoid pressure is maintained until the anaesthetist is satisfied that the airway is secure and both lungs are being ventilated.

If a draw-over circuit is being used then preoxygenate the patient from a reservoir bag filled from an oxygen cylinder or an oxygen concentrator (see page 48)

Monitor closely throughout. Measure the blood pressure at least every 5 minutes and the pulse continuously.

Common Problems Intubation is often more difficult than in the non-pregnant patient, especially if the mother is obese. Since oxygen consumption is high you cannot afford to take too long. Assess the difficulty of intubation beforehand and if you anticipate difficulties consider whether spinal anaesthesia should be your first choice or try and get an experienced colleague to help you.

Ensure there is a reliable, trained assistant. Prepare your different introducers, laryngoscopes and Magills forceps.

If there is not enough room for the laryngoscope handle because of a short neck or large breasts (or both!), try a child's laryngoscope handle with an adult blade or when using an adult handle, take off the handle, insert the blade and then re-attach the handle afterwards.

During maintenance of anaesthesia most anaesthetists continue the muscle relaxation using either a non-depolarising muscle relaxant or sometimes intermittent suxamethonium. This allows a light inhalational anaesthetic to be given. A standard anaesthetic should not be given before delivery as it will anaesthetise the baby. If you are using N_2O/O_2,give 50% N_2O only and add 0.5% halothane or 1 - 1.5% ether or 1% Trilene (Do NOT use Trilene with soda lime). If you are using air/oxygen give 50% oxygen if possible together with twice the percentage of volatile agent recommended above.

If you are not using a relaxant technique after intubation then ether breathed spontaneously has a good safety record provided that the baby is delivered quickly. Indeed, ether breathed spontaneously without intubation is a traditional technique which has a better than expected safety record. It is probably safer than having inexperienced practitioners attempting occasional intubations. In a series of 420 patients using open drop ether anaesthesia, there was no evidence of aspiration of gastric contents [1]. Light ether anaesthesia does not cause too much relaxation of the uterus.

After the baby is born you can revert to a standard anaesthetic and give narcotic analgesics. Avoid high concentrations of halothane (ideally no more than 0.5%) as it can increase the blood loss during surgery by relaxing the uterus.

Recovery Because of the risk of aspiration, the mother should always be extubated on her side, when she is awake and in full control of her airway.

Particular complications A failed intubation plan must be available and discussed regularly. Your colleagues should know what to do and it should be printed on a card and attached to the anaesthetic machine. The priorities are:

● Ensure full oxygenation of the mother at all times.

● Give only the initial dose of suxamethonium and do not attempt intubation too many times.

● If you have no success with introducers and other intubation aids then accept that you will not be able to intubate and ventilate the mother with a face mask until spontaneous respiration returns.

● Maintain cricoid pressure at all times.

● The safest thing to do is usually to allow the patient to wake up and perform the operation under a spinal anaesthetic.

If the airway is easy to maintain and the operation is urgent it may be necessary to consider proceeding under general anaesthesia (ether 4-6% or halothane 1-1.5%) without an endotracheal tube. An alternative is ketamine though this does not provide such good relaxation for the surgeon. In all cases maintain cricoid pressure until the operation is finished and the patient can be turned into the left lateral and head down position.

Remember that the baby will be sedated, and will probably require resuscitation, so get help.

Whatever happens, do not let your patient die or suffer brain damage just because you cannot intubate. If the airway is completely obstructed and mask ventilation impossible do an immediate cricothyroidotomy with a large IV cannula (see Update 7). This should be converted to a formal tracheostomy or the mother allowed to wake up and regain control of her own airway.

Acid aspiration If the patient vomits or regurgitates during induction of anaesthesia, the airway should be suctioned and the patient immediately placed in the left lateral head down position to prevent aspiration. Depending on the situation the patient may then be intubated or woken up.

Vomiting and regurgitation can also occur during recovery and it is imperative that patients are not left on their backs during this phase. Solid food causes immediate airway obstruction resulting in hypoxia, whilst liquid gastric contents cause an acid pneumonitis (inflammation of lung tissue). If aspiration occurs at induction then intubate the patient and clear the airways with suction. Consider bronchoscopy (if available) to remove solid food. Lavage of the airways is ineffective and not recommended. Ensure adequate oxygenation using added oxygen if available.

The diagnosis can usually be confirmed by listening with a stethoscope to the mid zone of the lungs at the tips of the scapulae 30 minutes after the event. Fine crepitations heard in this area is an early sign of aspiration pneumonia and, if the aspiration is significant, signs of hypoxia will develop. Within a few hours the chest Xray will show signs of aspiration pneumonitis. The patient will require supportive treatment with oxygen, chest physiotherapy and observation, particularly of respiration, for 24 - 48 hours. If respiratory function deteriorates then aggressive supportive treatment will be needed including high flow oxygen and, if necessary, intubation and ventilation. When there is no obvious deterioration, if the patient is well after 6 hours, clinically significant aspiration is most unlikely to have occurred.

In making the diagnosis, consider other causes of hypoxia and respiratory failure in late pregnancy, e.g. pulmonary oedema (fluid overload, cardiac failure, pre eclampsia / eclampsia), amniotic fluid embolism and pneumonia.

Some authorities recommend giving steroids immediately but opinion is divided on whether or not to give antibiotics since acid pneumonitis is initially a sterile condition without bacterial infection. Give broad spectrum antibiotics if solid matter has been inhaled or if signs of a secondary bacterial pneumonia develop after a day or two.

Intra-operative haemorrhage Good surgical technique should prevent the need for transfusion in most patients unless they are previously significantly anaemic and/or have been haemorrhaging in labour. Catastrophic haemorrhage can occur with certain placental problems (placenta praevia or abruption) or abnormalities (placenta accretia). All patients presenting for LSCS should be crossmatched for 2 units of blood before theatre. If haemorrhage is anticipated more blood should be crossmatched. Most theatres in UK store two units of group O negative blood (universal donor blood) for cases of severe unexpected haemorrhage.

A useful technique is normovolaemic haemodilution autotransfusion. Patients with a haemoglobin of at least 10gm% can have 2 units drawn off into transfusion bags just before surgery. These are replaced by 2 litres of normal saline or Hartmann's. The blood is kept nearby, labelled and is re-transfused at the end of surgery. Since it is fresh the clotting factors still function, unlike the situation with stored blood. All risks of stored blood are avoided. The technique has been well decribed [2].

Special circumstances - the operator anaesthetist. At some small hospitals the surgeon conducts the anaesthetic as well as performing the operation. This is a difficult situation, but is commonplace in many parts of the world. Techniques of anaesthesia used in this situation include local infiltration, spinal anaesthesia, epidural anaesthesia and ketamine. Whatever method of anaesthesia is used, expertise will develop with experience. A trained assistant must care for the patient throughout the operation to look after the airway and monitor the patient's vital signs. Regional techniques, during which the patient remains awake, are probably safer.

Summary
In most circumstances LSCS is a straightforward common operation, which requires little alteration to our normal anaesthetic practice. Preparation

should be thorough and problems anticipated before they occur. Plans should be prepared for emergencies such as a failed intubation or unexpected severe haemorrhage. Both general anaesthesia and regional anaesthesia may be associated with unnecessary mortality if they are not carried out carefully. In all cases keep reminding yourself of the 4 cornerstones which are easily forgotten: suction, cricoid pressure, left lateral tilt and close observation.

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THE ROLE OF THE ANAESTHETIST IN THE MANAGEMENT OF PRE-ECLAMPSIA

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Pre-eclampsia is a major cause of maternal mortality and morbidity, and fetal loss worldwide, but particularly in the third world.

Anaesthetists may be required to assist with pain management in labour, to provide anaesthesia for Caesarean Section and to assist in the Intensive Care Management of life-threatening complications which may arise from this condition.

DEFINITION

The cardinal features of this condition are hypertension and proteinuria, occurring for the first time after 20 weeks gestation. Pre-eclampsia is further classified into mild, moderate or severe groups. Mild pre-eclampsia is defined in a previously normotensive woman as a diastolic blood pressure in excess of 90mmHg with proteinuria of less than 0.3g/24hrs.

Severe pre-eclampsia is said to exist if one or more of the following is present:

- Systolic blood pressure > 160 or diastolic pressure > 110 mmHg on two readings 6 hours apart

- Rapidly increasing proteinuria (>3g/24hrs)

- Oliguria of < 400 ml/24 hours

- Evidence of cerebral irritability or visual disturbance

- Pulmonary oedema or cyanosis

Eclampsia is diagnosed with any degree of hypertension if convulsions occur.

AETIOLOGY

It is generally agreed that the essential disorder is utero-placental ischaemia, although the underlying mechanism for this has not yet been conclusively found. There is, however, a geographic and a socio-economic distribution with the condition being far commoner in developing countries, favouring either a genetic predisposition or a nutritional component.

PATHOPHYSIOLOGY

It is currently thought that a tissue factor is released from the ischaemic placenta affecting endothelial cells widely throughout the maternal circulation, resulting in occlusive spasm of arterioles involving:

Central Nervous System (CNS) CNS irritability is witnessed by headaches, visual disturbances, hyperreflexia and ultimately convulsions. The aetiology of this is more likely to be on the basis of vasospasm and hypoxia rather than cerebral oedema as was originally thought. Convulsions are not directly related to an elevation in blood pressure (as compared with hypertensive encephalopathy).

Cardiovascular System (CVS) The generalised arterial vasospasm leads to a decreased circulating blood volume with variable amount of tissue oedema. The systemic vascular resistance is increased as is the left ventricular stroke work index, leading to left ventricular strain. Consequently there may be left ventricular diastolic dysfunction with poor correlation between the central venous and pulmonary capillary wedge pressures.

Coagulation Up to one third of patients have thrombocytopaenia, and in severe cases platelet counts may fall rapidly. In addition, there appears to be a qualitative platelet dysfunction. Severe

cases may develop the HELLP syndrome, (Haemolysis, Elevated Liver Enzymes, Low Platelets), and disseminated intravascular coagulopathy.

Respiratory System Pulmonary involvement is uncommon until late in the course of the disease when pulmonary oedema and upper airway (especially laryngeal) oedema may occur. Pulmonary oedema occurs most frequently after delivery.

Liver There is reduced enzyme activity with raised liver enzymes particularly in the HELLP syndrome possibly due to areas of necrosis or ischaemia. Hepatic rupture is a rare, but often lethal, complication.

Kidneys The prevalence of proteinuria indicates glomerular involvement, probably on a vascular basis. Oliguria is more commonly due to hypovolaemia and decreased renal blood flow rather than primary renal pathology. Progress to acute renal failure is common, especially with hypotension and the HELLP syndrome. However, renal outcomes are generally good.

Feto-placental Unit The reduced placental perfusion results in a high prevalence of intra-uterine growth retardation. There is also a high incidence of abruptio placentae and preterm labour. Early delivery of the baby is often required and results in fetal prematurity.

MEDICAL MANAGEMENT
The management of these patients is both operative and non-operative. The anaesthetist should be involved in both facets of care in severe pre-eclamptics, as expertise in intensive care techniques, particularly cardiovascular monitoring and control and in the field of pain management can be invaluable.

AIMS to reduce maternal and foetal complications:
- Treat hypertension
- Control of convulsions
- Fluid therapy and treatment of oliguria
- Decision when to deliver
- Management of coagulation abnormalities

Control of Hypertension The aim is to keep the mean arterial pressure between 100 - 140mmHg (130/90-170/110mmHg). It is important to maintain

placental perfusion and reduction of the blood pressure to normal levels may be inappropriate.

Bed rest with avoidance of aorto-caval compression may be all that is required.

Vasodilatation should be preceded by volume expansion to avoid falls in blood pressure:

(a) Hydralazine is administered intravenously in 5mg increments followed by intravenous infusion at 5 - 20mg/h titrated against the blood pressure. This agent is a direct-acting vasodilator and is the most widely used drug for the control of pre-eclamptic hypertension. The onset time of dihydralazine is slow (about 15 minutes) and 20 minute intervals should be allowed between increments; if insufficient time is allowed, severe hypotension may occur.

Hypotension and tachycardia generally respond to infusion of fluids.

(b) Methyldopa is generally reserved for patients with an element of chronicity to their hypertension. It is used in standard doses but may cause drowsiness, depression and postural hypotension. It has a long history of safety in pregnancy used in the dose of 1 - 3g daily in divided doses.

(c) Nifedipine Although a logical choice, nifedipine has not been widely studied for use in PET. It's principal use has been in the acute management of very high blood pressures with 10 mg orally being the usual dose. Short-acting nifedipine is supplied as a bite-and-swallow capsule and is much more effective and reliable when used in this way, as opposed to the widespread practice of giving it sublingually.

(d) β blockers Fears of the effects of β blockade on the fetus makes the routine use of these agents in the at-risk pregnancy inadvisable. However, Labetalol has been used successfully in a small series of patients.

(e) Nitroprusside / Nitroglycerine(by continuous infusion) Nitroglycerine acts primarily on the venous capacitance vessels and is less effective following volume expansion. Nitroprusside, with it's rapid onset and brief duration, would appear an ideal agent, however, fears of cyanide toxicity in the foetus has limited its use to short term blood pressure control. There are also doubts as to its safety in the presence of raised

intracranial pressure, such as may occur in a patient who has had several convulsions.

Intravenous fluid therapy Some authors claim that plasma volume expansion per se can induce vasodilatation and reduce blood pressure, improving regional blood flow and optimising the effect of vasodilator drugs. However, in severe pre-eclampsia and especially post-delivery, left ventricular dysfunction combined with a low plasma oncotic pressure, can combine to produce a high incidence of pulmonary and cerebral oedema. In severe cases therefore, pulmonary capillary wedge pressure monitoring is mandatory when plasma volume expansion is contemplated. The absolute value of the central venous pressure is valueless as a guide to the risk of pulmonary oedema. However, careful titration of the fluid load against CVP response is a useful way of determining the ability of the ventricle to handle the volume imposed.

Management of convulsions Magnesium sulphate is now established as the agent of choice for the prevention of recurrent eclamptic convulsions. The place of magnesium infusions for the prophylaxis of convulsions in pre-eclamptic patients remains to be established. There is also no clarity in the literature as to the best agent for the termination of an eclamptic convulsion.

(a) **Magnesium Sulphate** is a potent cerebral vasodilator, as well as a powerful catecholamine antagonist. The therapeutic blood level lies between 2 and 4mmol/l. There are two commonly accepted dosage regimens:

The combined intramuscular and intravenous regime in which a 4gm intravenous dose, infused over 20minutes, is combined with a 10gm intramuscular injection followed by 5gm intramuscularly into each buttock every 4 hours thereafter.

The intravenous regime in which the 4gm loading dose is followed by a continuous infusion of 1 to 3 gm per hour to maintain the therapeutic level.

The major danger of magnesium infusion is neuromuscular blockade, which is a linear function of the plasma magnesium concentration. Neuromuscular monitoring by the hourly testing of patellar tendon reflexes is the standard method of determining the early onset of toxicity. If depression of the reflexes occurs, stop the infusion until the relexes return. Magnesium is exclusively excreted by the kidneys, and diminished renal function is a relative contraindication to the use of this ion.

(b) **Diazepam** is still widely used as the first line agent to terminate a convulsion and is given in 5 - 10 mg increments until effective. Diazepam infusions of 10mg/h have been used prophylactically but may produce excessive sedation with the consequent risks to the airway. Fetal depression, especially in a premature infant has been a major factor in the decline of the use of this drug. Magnesium is now the preferred agent.

(c) **Phenytoin** Although this drug was widely used in the past for the prevention and control of eclamptic convulsions, recent evidence no longer supports its use.

Prophylaxis for convulsions should be started with signs of cerebral irritability such as headache, visual disturbances, epigastric pain or hyperreflexia. Following a single eclamptic convulsion, prophylaxis with magnesium sulphate should always be instituted, unless there are major contraindications. Hypertension alone is not necessarily an indication for anticonvulsant therapy; convulsions may occur at moderately elevated blood pressures and blood pressure alone is a poor predictor of the likelihood of occurrence of a convulsion.

The decision to deliver. The obstetrician normally makes this decision in consultation with the paediatrician and in severe cases with the anaesthetist. It is often a balance between maternal morbidity and fetal viability. Commonly, the mother is presented for Caesarean Section at a time when her disease is most severe.

ANAESTHESIA and ANALGESIA
Optimisation If not already achieved by the obstetrician, the anaesthetist needs to ensure that the intravascular volume and renal function is optimised as well as the control of hypertension and the anti-convulsant therapy.

Labour analgesia In mild or moderate pre-eclampsia, the patient may be allowed to proceed with normal labour. Provided coagulation is normal, the early institution of an epidural block may often be useful in the management of these patients, both for the control of blood pressure and vasodilatation as well as reducing the stress response and catecholamine release that may be induced by pain.

It is also thought that regional anaesthesia improves placental intervillous blood flow. There is the possibility that concomitant magnesium infusion may increase the degree of hypotension that may accompany epidural blockade. However, this seems unlikely to be sufficiently severe to compromise placental blood flow, which may be selectively preserved by magnesium infusion.

Operative Management - anaesthetic technique General anaesthesia vs. regional anaesthesia - How to decide which?

The interest of both mother and fetus as well as the technical ability of the anaesthetist involved need to be considered. (A familiar technique is safer for all parties than a more technically correct method with which the anaesthetist is unfamiliar).

General anaesthesia is the only recommended technique in patients with diminished level of consciousness e.g. eclampsia or immediately post-ictal or the following problems:

- imminent eclampsia

- serious coagulation of abnormalities,
- anatomical problems interfering with insertion of the regional block
- sepsis at the site of the proposed regional block

The relative advantages of general and regional anaesthesia in pre-eclampsia are summarised in table 1.

Conduct of General Anaesthesia

a) Assessment of airway Prediction of airway oedema is not always possible but the presence of stridor and/or facial oedema may be a clue. The Mallampati score may change remarkably during labour, and should always be performed immediately prior to the performance of general anaesthesia. Post-convulsion, laceration of the tongue or mucosa may also be warning signs of a difficult intubation. In these cases, awake naso-tracheal intubation may be necessary. However, the unpredictability of airway difficulty in these patients behoves the anaesthetist to have the

Table 1.

	Regional Anaesthesia		**General Anaesthesia**	
	Advantages	*Disadvantages*	*Advantages*	*Disadvantages*
Airway	No intubation response No risk of failed intubation	No control	Control	Exaggerated intubation response Increased risk of failed intubation
Convulsions	Nil	No active control Risk of convulsion	Control	
Drugs and technique	No sedative drugs	Risk of convulsions Risk of high block		Maternal awareness Fetal depression
Speed	Spinals quick - 5-10 min	Epidural - slow 20-30 mins	Fast - less than 5 minutes	
Blood Pressure control	Lower catecholamines Less instability	Risk of hypotension	Less hypotension	Increased catecholamines Increases in BP, PAWP, CVP with intubation
Coagulation	No airway instrumentation	Risk of haematoma	Avoid spinal haematoma	Risk of airway haemorrhage

facilities available for a difficult or impossible intubation (introducers, laryngeal masks, surgical airway etc.) for every case.

b) Induction should be in accordance with standard obstetric practice:

Pre-oxygenation for at least three minutes followed by a rapid acting induction agent; thiopentone 4 - 5 mg/kg or etomidate 0.2mg/kg (not ketamine); and suxamethonium (1- 1.5mg/kg).

During this time, however, a method of reducing haemodynamic responses to laryngoscopy and intubation should be employed. Some of the methods used have proven injurious to fetal well-being, e.g. lignocaine, β blockers and the longer acting opioids. Vasodilators (nitroglycerine and nitroprusside) have been used but worries of fetal cyanide toxicity and maternal intracranial pressure have limited their use.

Alfentanil, given prior to the suxamethonium dose at 10mcg/kg produces obtunding of the pressor response with minimal fetal depression because of its short duration of action.

Magnesium sulphate, has a vasodilatory, as well as an anti-catecholamine action. Given as a 40 mg/kg bolus intravenously just after the induction, it can obtund the pressor response without excessive hypotension to follow (Remember this is a painful injection when given awake). MgSO4 and alfentanil can be used together in severe cases, with doses lowered accordingly (30mg/kg + 7.5mcg/kg), but if maternal risk is high (MAP (180) higher doses can be used (60mg/kg + 30mcg/kg). Hypotension following the intubation response is not uncommon, especially in combination with a volatile agent.

Lignocaine is less effective than alfentanil or magnesium. If it is to be used, a dose of 1.5mg/kg should be given intravenously 3-5 minutes before induction

Precurarisation, especially where magnesium has been used is not recommended, as it may lead to severe muscle weakness prior to induction. Remember that after magnesium, the usual fasciculations following suxamethonium may not occur, and a timed 60 seconds after suxamethonium administration should be allowed before attempting intubation.

Depolarising muscle relaxants should be used with caution, in smaller doses, and preferably with neuromuscular monitoring. It is our practice to use an infusion of suxamethonium, starting at 4 mg/min but also using a nerve stimulator to maintain relaxation sufficient to facilitate surgery.

Anaesthesia is best maintained with moderate to low concentrations (0.5 - 1 MAC) of Isoflurane (considering possible cerebral vasospasm and/or oedema), and a suitable opiate after delivery. We usually use 10 - 15mg morphine given immediately after delivery. Halothane may also be used, although isoflurane is preferable if there are signs of raised intracranial pressure

c) Extubation. Exaggerated CVS responses to extubation are often overlooked, but can be as severe and disastrous as those of intubation. MgSO4 and Alfentanil here are illogical and vasodilators, (β blockers (especially Esmolol), and possibly lignocaine may be used.

Conduct of Regional Anaesthesia

It has long been argued that spinal anaesthesia, except for the mildest of hypertension, is not suitable for PET, as precipitous hypotension may result. More recently though, several authors have studied the use of spinals for severe forms of the disease, with some good results. Although hypotension remains a problem, particularly in view of a conservative attitude to fluid loading, it has been shown that utero-placental flow is not diminished, presumably through arteriolar vasodilatation, and may even be enhanced.

It is our view that stable hypertensives on vasodilatory treatment (methyldopa, nifedipine, hydralazine) are good candidates for spinal anaesthesia, as they tend to drop their blood pressure less than those on no treatment. However, for the uncontrolled, newly diagnosed or severely hypertensive cases, an epidural seems to be the regional anaesthetic technique of choice, provided there is no need for rapid fetal delivery (abruptio placentae, severe fetal bradycardia). As with the spinal, the better the preoperative medical management (fluids + vasodilators) the less problem there is with hypotension. The possibility of running the epidural postoperatively, where a high percentage of cardiopulmonary complications can occur, make this an attractive option.

Conduct of epidural and spinal anaesthesia is according to that of regular practice:

a) Spinal Our guidelines suggest the use of a 25G or smaller pencil point needle, with 1.6 - 2.0 ml of "heavy" (with dextrose), bupivacaine 0.5% depending on the height and abdominal girth of the patient. Taller patients get a bigger dose, whereas heavier patients, with more pressure on their spinal space, need a smaller volume. The block height aimed for is T6, ideally.

b) Epidural The cannula is sited in L2/3 or L3/4 interspace and the standard test dose is used. The loading or main dose should be given in stages rather than as a single large bolus, so as to raise the height of the block slowly, again aiming for a T6 sensory level.

Our practice is to use 10mcg of fentanyl with the spinal and 50 - 100mcg with the epidural main dose. This has the effect of making the sensory component of the block denser.

Hypotension cannot be simply treated with a free hand in terms of crystalloid volume. A more balanced approach is to use some synthetic colloid (500ml starch solution) and crystalloid (Ringer's lactate 1000ml), as well as ephedrine in 5mg increments, as this will not adversely affect uterine blood flow.

POSTOPERATIVE CARE

Seventy percent of convulsions and pulmonary complications occur in the postoperative period in pre-eclampsia. Laryngeal oedema may worsen during the operative procedure and airway embarrassment, sufficiently severe to require re-intubation, may follow extubation. Anti-hypertensive therapy should be continued for as long as clinically indicated and anticonvulsant medication maintained for as long as the patient remains symptomatic. Invasive monitoring, if used intra-operatively, should be continued in the intensive care environment post-operatively. Good quality post-operative analgesia can contribute to the ease of management of these cases. Continued meticulous attention must be paid to fluid balance and the correction thereof in the presence of oliguria.

CONCLUSIONS

The management of the severe pre-eclamptic presents a considerable clinical challenge. The expertise of anaesthetists in the provision of pain relief, management of cardiovascular function, the control of fluid balance, management of respiratory function and familiarity with the drugs used makes them potentially key figures in the multi-disciplinary management of these patients. The provision of anaesthesia and analgesia for operative and non-operative delivery of these patients provide a particular clinical challenge requiring considerable skill and experience on the part of the anaesthetist.

Further Reading

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INTERSCALENE BRACHIAL PLEXUS BLOCK

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Interscalene block is the most proximal approach to the Brachial Plexus and is the most suitable block for proximal procedures on the arm or shoulder. The block is a paravertebral approach at the level of the cervical roots in the neck and can provide both brachial and cervical nerve blocks. The areas supplied by C8 and T1 nerve roots may prove difficult to block and this approach is therefore less suitable for surgery on the hand. Supplementary block of the ulnar nerve can provide the necessary analgesia for hand surgery.

Indications
- surgery to the shoulder or upper arm
- surgery of the hand (with peripheral nerve supplement)
- reduction of a dislocated shoulder, arm or wrist fractures

Anatomy After leaving their intervertebral foramina, the anterior primary roots of the cervical nerves (C5, 6,7,8,T1) course anterolaterally and inferiorly to lie between the anterior scalene and the middle scalene muscles which, respectively, arise from the anterior and posterior tubercules of the cervical vertebrae. The prevertebral fascia covers both the scalene muscles fusing laterally to enclose the brachial plexus in a fascia sheath. Between the scalene muscles, these nerve roots unite to form three trunks, which emerge from the interscalene grove to lie cephaloposterior to the subclavian artery as it courses along the upper surface of the first rib.

Preoperatively The patient is assessed for suitability for the block, the procedure is explained and consent obtained. Premedication may be given as indicated. The procedure should be conducted in the anaesthetic room or theatre after establishing venous access and monitoring of the patient. The drugs and equipment for resuscitation and airway management should be available and ready.

Equipment
- syringes 2mls, 10mls, 20mls and a 23 gauge butterfly needle with an extension
- antiseptic skin preparation solution
- a nerve stimulator (where available) and a 22 gauge 4cm body insulated short bevel needle with connecting tubing

Technique The patient should be in supine position, arms by the sides and head turned away from the side to be blocked. Downward displacement of the shoulder facilitates the palpation of the landmarks. The posterior border of the strenocleidomastoid muscle is made prominent by having the patient briefly lift their head. The interscalene groove can be palpated by rolling the fingers posterolaterally from this border over the belly of the anterior scalene muscles into the groove. The intersection of this groove with a transverse plane at the level of the cricoid cartilage is the point at which the needle should enter the skin and this is about the level of the sixth cervical vertebra (C6).
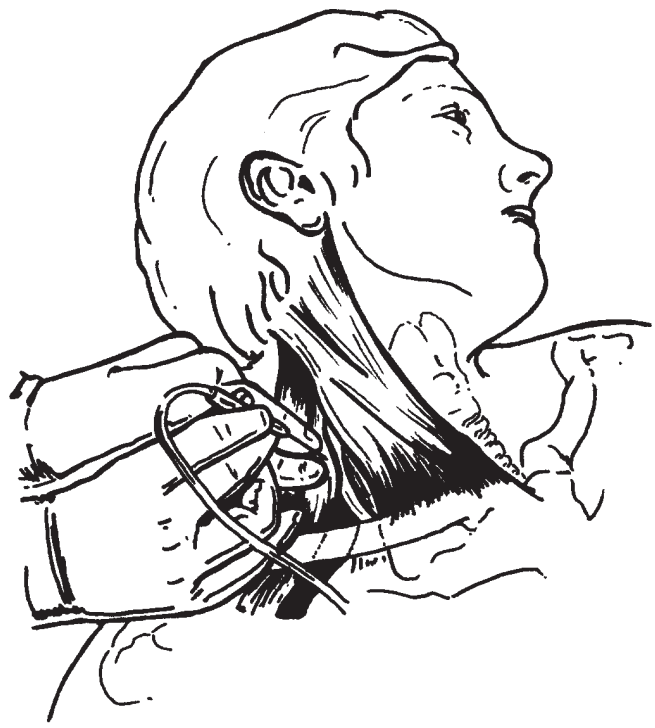
The skin on the side to be blocked is cleaned with an antiseptic and draped. A skin weal is made with lignocaine. A 22 or 23 gauge 4cm short bevelled needle is inserted perpendicularly to the skin with

a 45 degree caudad angle (towards the feet) and slightly posterior angle. The angle of approach is important to avoid accidental intravascular or intrathecal injection. The needle is then advanced carefully until a paraesthesia is elicited. A click may be detected as the needle passes through the prevertebral fascia. This usually occurs at the superficial level. The use of a nerve stimulator with a special insulated needle is very helpful in confirming the correct placement of the needle and performing the interscalene block accurately. Correct stimulation produces twitching below the shoulder. Stimulation of the diaphragm indicates too anterior an approach. Once paraesthesia is obtained, the needle is stabilised and after negative aspiration for blood, 20 to 30mls of the local anaesthetic solution is injected slowly and carefully.

Local anaesthetic solution Bupivacaine 0.375-0.5% solution may be used safely in the volumes between 20-40mls, but the maximum dose of 2 mg/kg should not exceeded. Other local anaesthetic agents like lignocaine or prilocaine may be used.

Complications

- Inadvertent epidural or subarachnoid injection is a potentially serious complication resulting from incorrect needle placement.

- Vertebral artery injection, this can result in convulsions and loss of consciousness.

- Phrenic nerve block is frequently produced, this complication precludes bilateral use of this technique.

- Recurrent laryngeal, vagus, and cervical sympathetic nerves are sometimes blocked.

- Pneumothorax is rare but can happen with deep placement of the needle and in unskilled hands.

PHYSIOLOGY OF THE KIDNEY

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The Functions of the Kidney
- Regulation of the water and electrolyte content of the body.
- Retention of substances vital to the body such as protein and glucose
- Maintenance of acid/base balance.
- Excretion of waste products, water soluble toxic substances and drugs.
- Endocrine functions.

Regulation of the water and electrolyte content of the body
The kidney allows a person to eat and drink according to their habits without changing the composition of their fluid compartments.

Renal Blood Supply is normally is about 20% of the cardiac output. Approximately 99% of the blood flow goes to the cortex and 1% to the medulla. The cortex is the outer part of the kidney containing most of the nephrons. The medulla is the inner part of the kidney and contains the specialised nephrons in the juxta-medullary region, immediately next to the medulla. These nephrons have a greater concentrating ability, the mechanism being explained below. The kidney is unique as it has two capillary beds arranged in series, the glomerular capillaries which are under high pressure for filtering, and the peritubular capillaries which are situated around the tubule and are at low pressure (figure 1). This permits large volumes of fluid to be filtered and reabsorbed.
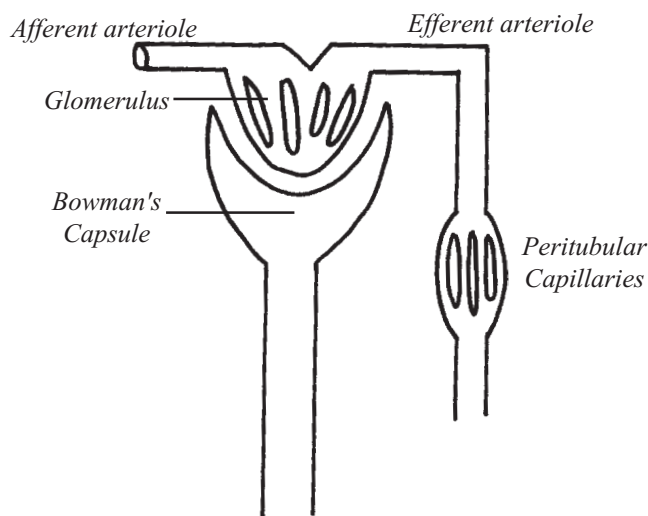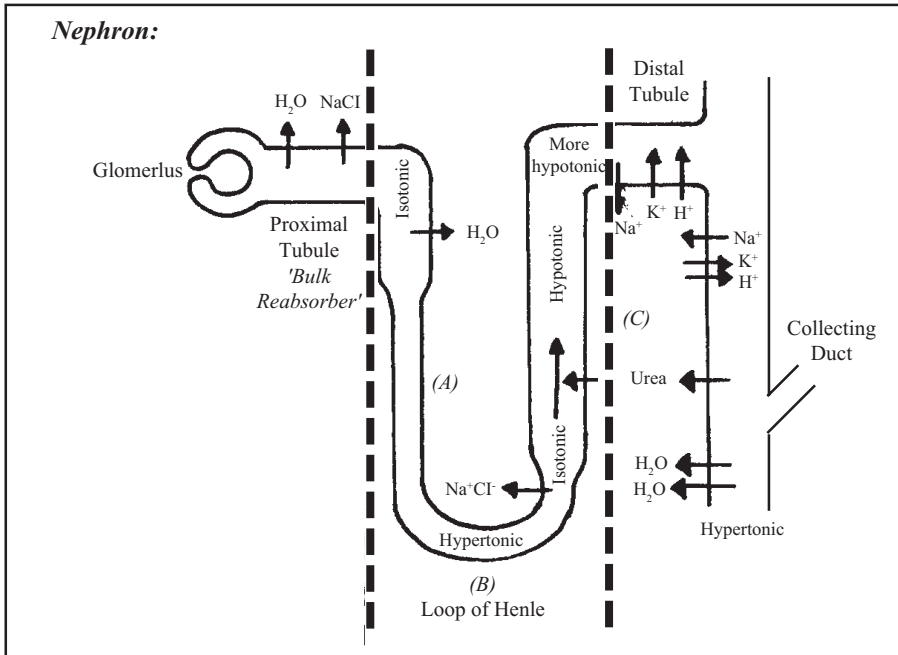

The Nephron: Each kidney consists of about one million nephrons. The nephron is made up of a glomerulus and its tubule (figure2). The tubule is

made up of a number of sections, the proximal tubule, the medullary loop (loop of Henle), and the distal tubule which finally empties into the collecting duct.

Nephron:

Urine is formed as a result of a three phase process - **simple filtration, selective and passive reabsorption and excretion.**
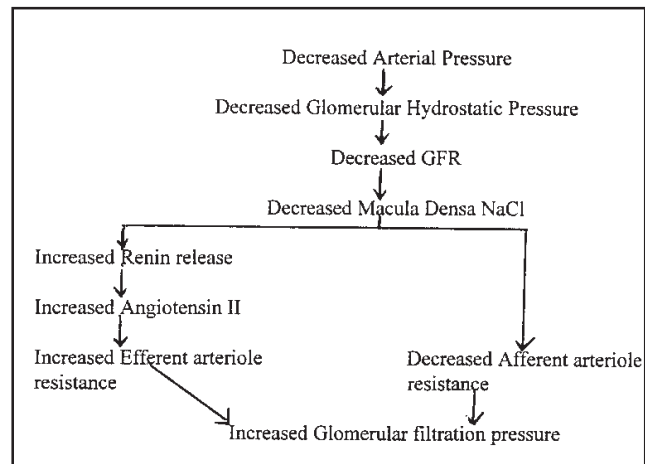
Filtration
Filtration takes place through the semipermeable walls of the glomerular capillaries which are almost impermeable to proteins and large molecules. The filtrate is thus virtually free of protein and has no cellular elements. The glomerular filtrate is formed by squeezing fluid through the glomerular capillary bed. The driving hydrostatic pressure (head of pressure) is controlled by the afferent and efferent arterioles, and provided by arterial pressure. About 20% of renal plasma flow is filtered each minute (125 ml.min^{-1}). This is the ***glomerular filtration rate*** (GFR).

In order to keep the renal blood flow and GFR relatively constant hydrostatic pressure in the glomerulus has to be kept fairly constant. When there is a change in arterial blood pressure, there is constriction or dilatation of the *afferent* and *efferent* arterioles, the muscular walled vessels leading *to* and *from* each glomerulus. This process is called **autoregulation**.

Autoregulation of GFR is achieved by autoregulation of renal blood flow and a feedback mechanism known as "glomerular tubular balance".

Glomerular Tubular balance. When there is a decrease in GFR, there is a resulting decrease in the fluid flow rate within the tubule. At the loop of Henle, there is greater time for reabsorption of sodium and chloride ions. Therefore there is a decrease in the number of sodium and chloride ions reaching the distal tubule which is detected by the macula densa. This in turn decreases the resistance in the afferent arteriole which results in an increase in renal blood flow. It also increases renin release from the juxtaglomerular apparatus which stimulates angiotensin II production causing constriction of the efferent arteriole.

These both act to increase the hydrostatic pressure in the glomerular capillary bed and return GFR to normal (*table 1*).

The **juxtaglomerular complex** consists of ***macula densa cells***, which are special distal tubular epithelial cells which detect chloride concentration and modified smooth muscle cells, ***juxtaglomerular cells***, in the walls of the afferent and efferent arteriole. These cells produce renin. **Renin** is an enzyme which converts the plasma protein angiotensinogen to ***angiotensin I.*** Angiotensin converting enzyme (ACE) which is formed in small

Table 1

quantities in the lungs, proximal tubule and other tissues, converts *angiotensin I* to *angiotensin II* which causes vasoconstriction and an increase in blood pressure. Angiotensin II also stimulates the adrenal gland to produce *aldosterone* which causes water and sodium retention which together increase blood volume.

This is a negative feedback system. In other words the initial stimulus is a fall in blood volume which leads to a fall in perfusion pressure in the kidneys. When blood volume, renal perfusion and GFR improve the system feeds back to switch off or turn down the response to the stimulus.

Selective and Passive Reabsorption

The function of the **renal tubule** is to reabsorb selectively about 99% of the glomerular filtrate.

The **Proximal Tubule** reabsorbs 60% of all solute, which includes 100% of glucose and amino acids, 90% of bicarbonate and 80-90% of inorganic phosphate and water.

Reabsorption is by either active or passive transport. Active transport requires energy to move solute against an electrochemical or a concentration gradient. It is the main determinant of oxygen consumption by the kidney. Passive transport is where reabsorption occurs down an electrochemical, pressure or concentration gradient.

Most of the solute reabsorption is active, with water being freely permeable and therefore moving by osmosis. When the active reabsorbtion of solute from the tubule occurs, there is a fall in concentration and hence osmotic activity within the tubule. Water then moves because of osmotic forces to the area outside the tubule where the concentration of solutes is higher.

The **Loop of Henle** is the part of the tubule which dips or "loops" from the cortex into the medulla, (*descending limb*), and then returns to the cortex, (*ascending limb*). It is this part of the tubule where urine is concentrated if necessary. This is possible because of the high concentration of solute in the substance or interstitium of the medulla. This high concentration of solutes is maintained by the *counter current multiplier*. A counter current multiplier system is an arrangement by which the high medullary interstitial concentration of solute is maintained, giving the kidney the ability to concentrate urine. The loop of Henle is the counter current multiplier and the *vasa recta* is the counter current exchanger, the mechanism being described below.

Actions of different parts of the loop of Henle

A   The **descending loop of Henle** is relatively impermeable to solute but permeable to water so that water moves out by osmosis, and the fluid in the tubule becomes hypertonic.

B   The thin section of the ascending loop of Henle is virtually impermeable to water, but permeable to solute especially sodium and chloride ions. Thus sodium and chloride ions move out down the concentration gradient, the fluid within the tubule becomes firstly isotonic then hypotonic as more ions leave. Urea which was absorbed into the medullary interstium from the collecting duct, diffuses into the ascending limb. This keeps the urea within the interstitium of the medulla where it also has a role in concentrating urine.

C   The thick section of the ascending loop of Henle and early distal tubule are virtually impermeable to water. However sodium and chloride ions are actively transported out of the tubule, making the tubular fluid very hypotonic.
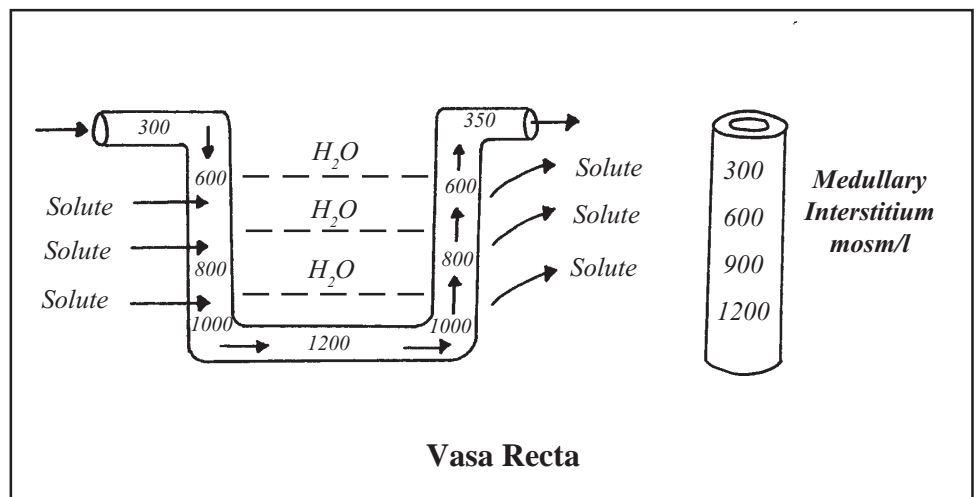
Vasa Recta

The **Vasa Recta** (figure 3) is a portion of the peritubular capillary system which enters the medulla where the solute concentration in the interstitium is high. It acts with the loop of Henle to concentrate the urine by a complex mechanism of counter current exchange. If the vasa recta did not exist, the high concentration of solutes in the medullary interstitium would be washed out. Solutes diffuse out of the vessels conducting blood towards the cortex and into the vessels descending into the medulla while water does the opposite, moving from the descending vessels to the ascending vessels. This system allows solutes to recirculate in the medulla and water, in effect, to bypass it.

Distal Tubule and Collecting Duct: The final concentration of urine depends upon the amount of **antidiuretic hormone (ADH)** secreted by the posterior lobe of the pituitary. If ADH is present the distal tubule and the collecting duct become permeable to water. As the collecting duct passes through the medulla with a high solute concentration in the interstitium, the water moves out of the lumen of the duct and concentrated urine is formed. In the absence of ADH the tubule is minimally permeable to water so large quantities of dilute urine is formed.

There is a close link between the hypothalamus of the brain and the posterior pituitary. There are cells within the hypothalamus, *osmoreceptors*, which are sensitive to changes in osmotic pressure of the blood. If there is low water intake, there is a rise in osmotic pressure of the blood, and after excess intake of water, the reverse. Nerve impulses from the hypothalamus stimulate the posterior pituitary to produce ADH when the osmotic pressure of the blood rises. As a result water loss in the kidney is reduced because ADH is secreted, and water reabsorbed in the collecting duct.

Acid/Base Function

- **Acid**: a substance that can release hydrogen ions in solution.

- **Base**: a substance that can accept hydrogen ions in solution.

- **Buffer**: a substance whose pKa (the pH at which half is in the ionised form and half unionised) is close to the pH of its environment. In those circumstances, addition or removal of hydrogen ions results in minimal change to pH, the purpose of the buffer.

- The pH is the negative log to base 10 of the hydrogen ion concentration [H^+] and indicates the acidity of the solution. The more acid the solution the **higher** the H^+ concentration but the **lower** the pH. The pH in the body is kept under tight control as almost all enzyme activities in the body are dependent on the pH being normal.

The lungs and kidneys work together to produce a normal extracellular fluid and arterial pH of 7.35-7.45 (34-46 nmol.l^{-1} H^+ concentration). Carbon dioxide (CO_2), when dissolved in the blood is an acid, and is excreted by the lungs. The kidney excretes fixed acid and performs three functions to achieve this:-

1. Tubular secretion of acid (figure 4): The buffer sodium bicarbonate, is filtered by the glomerulus and then reabsorbed in the proximal tubule. The sodium is absorbed by a sodium/hydrogen ion pump (Na^+/H^+) exchanging Na^+ for H^+ on the luminal proximal border of the tubular cell. A sodium/potassium pump (Na^+/K^+) forces Na^+ through the cell from tubular fluid in exchange for potassium.

Bicarbonate Reabsorption:
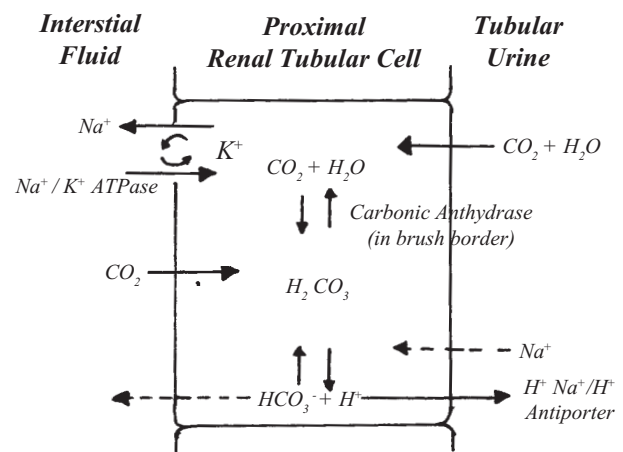
2. Glomerular filtration of buffers which combine with H^+ :

a) The majority of the filtered bicarbonate is reabsorbed (90% in the proximal tubule). The H^+, released as the Tubular Secretion of Acid (above), forms carbonic acid with the bicarbonate (HCO_3^-). $H^+ + HCO_3^- \rightleftharpoons H_2CO_3$

Carbonic anhydrase, found in the proximal tubular cells, catalyses the reaction to carbon dioxide (CO_2) and water (H_2O) (figure 4). The CO_2 diffuses into the cell where it again forms carbonic acid in the presence of carbonic anhydrase. The carbonic acid ionises to H^+ and HCO_3^-. The H^+ is then pumped out of the cell back to the lumen of the tubule by the Na^+/H^+ pump (1 above) and the sodium is returned to the plasma by the Na^+/K^+ pump (1 above). Water is absorbed passively.

b) Other buffers include inorganic phosphate (HPO_4^-), urate and creatinine ions which are excreted in urine as acid when combined with H^+ ions secreted in the distal nephron

3. **Ammonia is produced** enzymatically from glutamine and other amino acids, and is secreted in the tubules. Ammonia (NH_3) combines with secreted H^+ ions to form a non-diffusible ammonium ion (NH_4^-) which is excreted in the urine. Ammonia production is increased by a severe metabolic acidosis to as much as 700 mmol.day^{-1}.

Excretion of waste products
Filtration occurs as blood flows through the glomerulus. Some substances not required by the body, and some foreign materials (e.g. drugs) may not be cleared by filtration through the glomerulus. Such substances are cleared by *secretion* into the tubule and excreted from the body in the urine.

Hormones and the Kidney
Renin (see above) increases the production of angiotensin II which is released when there is a fall in intravascular volume e.g. haemorrhage and dehydration. This leads to:

● Constriction of the efferent arteriole to maintain GFR, by increasing the filtration pressure in the glomerulus.

● Release of aldosterone from the adrenal cortex

● Increased release of ADH from the posterior pituitary

● Thirst

● Inotropic myocardial stimulation and systemic arterial constriction

The opposite occurs when fluid overload occurs.

Aldosterone promotes sodium ion and water reabsorption in the distal tubule and collecting duct where Na^+ is exchanged for potassium (K^+) and hydrogen ions by a specific cellular pump. Aldosterone is also released when there is a decrease in serum sodium ion concentration. This can occur, for example, when there are large losses of gastric juice. Gastric juice contains significant concentrations of sodium, chloride, hydrogen and potassium ions. Therefore it is impossible to correct the resulting alkalosis and hypokalaemia without first replacing the sodium ions using 0.9% saline solutions.

Atrial Natruretic Peptide (ANP) is released when atrial pressure is increased e.g. in heart failure or fluid overload. It promotes loss of sodium and chloride ions and water chiefly by increasing GFR.

Antidiuretic Hormone (ADH) increases the water permeability of the distal tubule and collecting duct, thus increasing the concentration of urine. In contrast, when secretion of ADH is inhibited, it allows dilute urine to be formed. This occurs mainly when plasma sodium concentration falls such as following drinking large quantities of water. This fall is detected by the osmoreceptors (above). The hormones interact when blood loss or dehydration occurs to maintain intravascular volume. The flow diagram in Table 2 illustrates this.
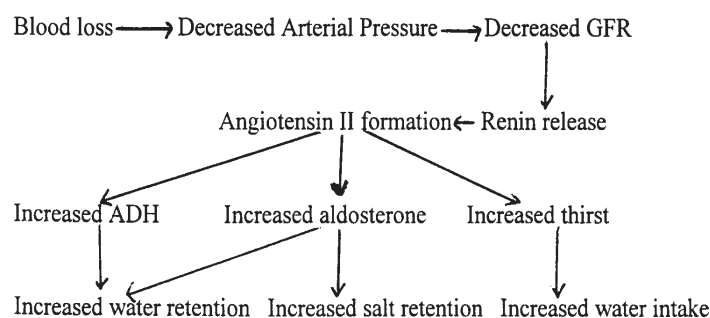
Other Substances Produced by the kidney
1,25 dihydroxy vitamin D (the most active form vitamin D) which promotes calcium absorption from the gut.

Erythropoietin which stimulates red cell production

Both of these decrease in renal failure.

Table 2. **The Kidney and maintenance of Intravascular volume**

Blood loss ⟶ Decreased Arterial Pressure ⟶ Decreased GFR

Angiotensin II formation ← Renin release

Increased ADH   Increased aldosterone   Increased thirst

Increased water retention   Increased salt retention   Increased water intake

NEUROPHARMACOLOGY - INTRACRANIAL PRESSURE AND CEREBRAL BLOOD FLOW

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ABBREVIATIONS

CBF   **Cerebral blood flow**
ICP   **Intracranial pressure**
CMRO$_2$ **Cerebral metabolic rate**
MAP   **Mean arterial pressure**
h   **Hour**
min   **Minute**

INHALATIONAL AND INTRAVENOUS AGENTS

Reviewing new pharmacological agents and their effects on the brain has been a considerable challenge. Over recent years a number of new agents, both inhalational and intravenous have been introduced. Their advantages and disadvantages are debated, their availability is not uniform, but their costs are undisputedly increased. Should they be included in this review? I believe they should, as a decision on the use of a drug can only be made after consideration of all the facts, pharmacological, economic and availability by the clinician. Previously, anaesthetists used inhalational techniques almost exclusively. Today it is different, with the important advances in intravenous drugs, a significant number of anaesthetists use intravenous techniques either routinely or with certain indications. Thus both inhalational and intravenous agents will be considered, with a discussion of clinical considerations where relevant.

INHALATIONAL AGENTS

The conventional understanding is that anaesthetic agents reduce neuronal function and so depress metabolic demands. This in turn reduces cerebral blood flow (CBF). However it is well recognised that volatile anaesthetic agents cause cerebral vasodilatation with an increase in CBF. This **direct** effect is caused by a reduction in the tension of the isolated cerebral artery muscle. Volatile agents also produce some uncoupling of the normal relationship between metabolism and blood flow. Usually, when cerebral metabolic rate (CMRO$_2$) decreases, local blood flow falls as there is a reduced requirement for oxygen delivery and carbon dioxide removal. This is the **indirect** effect. Volatile agents

uncouple or disconnect this relationship in a dose dependent way. The overall effect on cerebrovascular tone therefore, is the sum of both the direct vasodilatory effect and any indirect vasoconstrictor effect remaining.

The important consequence of this is that any dilatation in turn raises cerebral arterial volume and increases brain volume. When the brain is stiffer, in other words, compliance is reduced, intracranial pressure (ICP) will rise [1]. This was explained in the previous article to which the reader is referred and is demonstrated in fig 1. Note the different size of the rises in ICP which occur as the squashiness or compliance of the brain changes. At the left hand end, the brain is not stiff, the normal situation. Any change in cerebral volume results in a **small** increase in ICP. In contrast at the right hand end of the curve the brain is stiffer, due to oedema or a large space occupying lesion such as a blood clot, tumour or cyst. Note the **larger** increases in ICP at the right end of the curve in this situation.
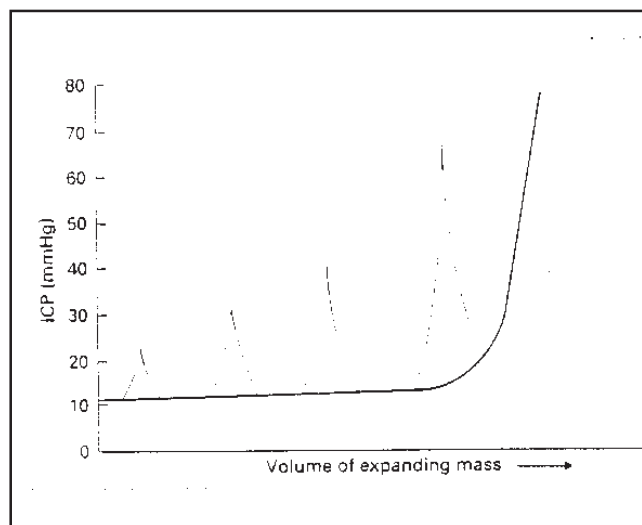

Fig 1   *The pressure-volume curve of the intracranial contents. As the volume of an expanding mass increases, ICP rises only slightly until the compensatory mechanisms are overcome. This point is reached at the elbow of the curve when further expansion of the mass causes a steep rise in ICP*

It is important to consider the influence on two other physiological mechanisms, autoregulation and CO$_2$-CBF relationship. The normal autoregulatory mechanism is gradually abolished as the concentration of the volatile agent is increased, CBF becoming blood pressure dependent. Thus as blood pressure rises, CBF increases and cerebral
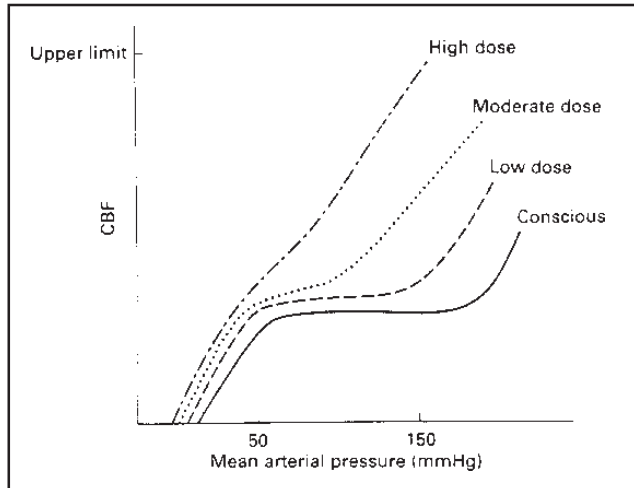
Fig . 2   Idealised curves of the effect of a progressively increased dose of a volatile anaesthetic agent on CBF autoregulation

Fig 3   Effects of anaesthetic agents on the CBF-CO₂ response curve. Idealised curves indicate the effects of carbon dioxide (CO₂) and volatile agents. The slope of the thin line indicates that CBF-CO₂ sensitivity is increased with agents which increase CBF, such as volatile agents.

vasodilation occurs. In contrast when blood pressure falls, there is no mechanism to sustain flow by reducing cerebrovascular resistance (fig 2).

The CO_2-CBF relationship is also affected by the volatile agents, the curve being shifted to the left. Hypocapnia is still able to reduce cerebral blood flow and therefore to oppose the vasodilation. However if CO_2 is allowed to rise, there is a much more rapid increase in CBF (fig 3).

TEACHING POINT

It is well recognised that following a head injury where the patient has lost consciousness briefly, a technique with spontaneous breathing with a volatile agent should NEVER be used. Following the head injury there will be some cerebral swelling because of contusion. Compensation will have taken place and thus the patient may not appear to have any significant decompensation. If an anaesthetic is required and halothene is given with the patient breathing spontaneously, there will be a rapid rise in ICP following cerebral vasodilatation induced by the combination of a raised CO_2, and a volatile agent. In addition blood pressure may fall as well, the combination dramatically reducing cerebral perfusion. Postoperatively this will be seen as persistent unconsciousness.
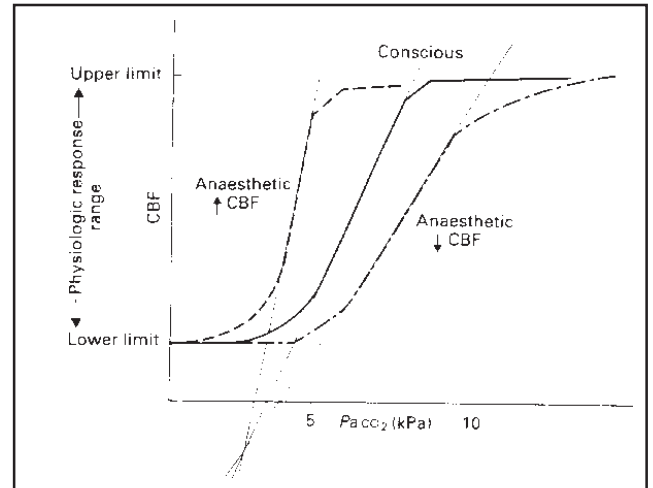

Halothane is a moderately insoluble agent, **blood:gas solubility 2.5** and a **MAC of 0.75%**. In the presence of cerebral swelling, it produces a large rise in ICP which can be prevented by hyperventilating the patient for 10 min before introducing it [2]. Cerebral autoregulation is reduced at 1% inspired concentration and abolished by 2% [3]. Halothane does not causes cerebral epileptic activity detected on the electroencephalogram (EEG). In practice therefore it is reasonably safe to use halothane in a hyperventilated patient up to a concentration of 0.5% in conjunction with nitrous oxide. It should be avoided, if possible, by using alternative techniques, before the dura is opened in patients who have severe intracranial decompensation [4] - in other words, massive brain swelling leading to unconsciousness preoperatively.

Enflurane has less effect on CBF and ICP than halothane. Like halothane it reduces $CMRO_2$ but it does cause cerebral epileptic activity, particularly when the patient is hypocapnic [5]. Epileptic activity is harmful as it induces a massive increase in cerebral metabolism, which in turn increases blood flow and hence cerebral swelling. Neurosurgery itself can also induce epileptic seizures postoperatively, and thus drugs which induce this process should be avoided. Finally the rate of production and resistance to reabsorption of CSF are increased by enflurane, making any increase in ICP associated with its use worse.

Isoflurane is a methyl ethyl ether with a **blood:gas solubility 1.4** and **MAC 1.2%**. It causes both respiratory and cardiovascular depression, the latter occurring predominantly due to a fall in systemic vascular resistance [6]. CBF and cerebral blood volume (CBV) are not affected by concentrations of 0.6-1.1 MAC isoflurane, but 1.6 MAC doubles CBF. Similarly it is only higher concentrations which cause an increase in ICP. There is less impairment of autoregulation and CO_2 reactivity when compared to halothane. A major property is the significant reduction in cerebral metabolic rate. There is evidence of uncoupling between the direct dilating effect and indirect vasoconstrictive effect, of isoflurane on the cerebral vasculature. Therefore up to 1.6 MAC the vasoconstricting effect predominates preventing cerebral blood flow from rising. As the inspired concentration rises, the direct vasodilatory effect overrides the indirect vasoconstrictor effect. However, in damaged or pathological brains the indirect vasoconstriction due to the depression in cerebral metabolic rate does not occur. Therefore small concentrations of isoflurane will cause some cerebral vasodilation although it is not as marked as with equipotent doses of halothane.

In clinical practice, it has been demonstrated that 1.1% isoflurane significantly increases intracranial pressure in patients with intracranial tumours with midline shift, despite hyperventilating the patient to induce a low CO_2 [7]. Despite these side-effects, isoflurane has become a useful drug for neuroanaesthesia because of its ability to reduce cerebral metabolic rate and to cause less vasodilation than other volatile agents available.

Sevoflurane is a new volatile agent with **MAC 1.7-2%**, and a low **blood:gas solubility 0.6** (isoflurane 1.4). It has similar properties to isoflurane on the brain, CBF, CBV and ICP [8,9]. A requirement of neuroanaesthesia is rapid recovery following surgery which may have lasted several hours. Low blood:gas solubility enables very rapid recovery even after many hours of surgery. Sevoflurane is also metabolised (5%), increasing blood fluoride concentration. So far no renal complications have been reported.

When sevoflurane is used with baralyme or soda-lime, in a circle system, Compound A, a toxic compound is produced. Again there have been no reports in man of problems, although some countries have set a minimum fresh gas flow of 2L/min. Sevoflurane is very expensive, but when used in a low-flow system is little more expensive than isoflurane. However this does require sophisticated monitoring, and there is the potential problem of complications arising from Compound A. Opinion is still divided on whether the use of low flow sevoflurane (<1L/min) is acceptable.

Desflurane is also similar to isoflurane when considering the brain. The **MAC is 5-10%** and **blood:gas solubility**, **0.4%**, also very low. Thus, like sevoflurane the main advantage to the neuroanaesthetist is rapid recovery. It too is expensive and is only economically viable if used in a sophisticated low-flow system. However in contrast to sevoflurane it is irritant to the tracheo-bronchial tree, requires a special vaporiser but is not metabolised or affected by soda-lime.

Ether (diethyl-ether) is still used as it is considered to be one of the safest agents because respiratory depression precedes cardiovascular depression. It is an irritant, soluble agent, **blood:gas solubility 12** (c.f. sevoflurane 0.6) and **MAC 1.92%**. Thus induction is prolonged and can be stormy. Recovery is also prolonged. Considering the brain, ether causes a biphasic response; at low concentrations, 2.4%, it will lead to some reduction in CBF and a significant fall in metabolic rate. However at higher concentrations, 4.5%, there is an increase in CBF with $CMRO_2$ rising towards baseline values [10]. Ether liberates catecholamines and it is believed that initially the increased sympathetic activity reduces CBF, but at higher ether concentrations the direct vasodilatory effect becomes dominant. The increased sympathetic activity also stimulates the brain with a risk of inducing epileptic activity, which both increases brain oxygen needs as well as increasing the risk of postoperative convulsions.

Thus, if there is a choice ether should be avoided. However, if there is no choice, then ether is better used as maintenance, following an intravenous induction, in a paralysed ventilated patient, in conjunction with a narcotic to reduce the ether requirements to a minimum.

Summary
Sevoflurane has great promise for neuroanaesthesia in those countries where it is available and can be

used with a low-flow system making it only slightly more expensive than isoflurane. The only question mark remains over the risk of the toxic substance, Compound A, which is formed when used with CO_2 absorber systems. Isoflurane is in general the "standard" volatile agent used for neuroanaesthesia. However the advantages of these different agents are relatively small and can be described as fine-tuning. Halothane or any of the less "ideal" agents are safe to use for neuroanaesthesia when there is no alternative *provided that close attention to basic details required for neuroanaesthesia are adhered to. It is this that makes a major contribution to reducing the disadvantages of less suitable agents.*

Nitrous oxide has been used for many years as a carrier gas and for its analgesic properties. It was believed that the effects on cerebral blood flow were minimal. However, in work with human volunteers nitrous oxide has been shown to cause a significant increase in cerebral blood flow acting synergistically with the volatile agents [11]. More importantly, the increase in CBF due to a combination of isoflurane and nitrous oxide is greater than simply increasing the volatile agent alone to provide the same MAC. As might be expected, an increase in ICP has been demonstrated when N_2O is used for patients with intracranial tumours.

This has lead a number of centres to omit N_2O from their technique. However nitrous oxide is not contraindicated for most neurosurgical procedures when there is minimal cerebral swelling. In addition, patients have to be anaesthetised with some agent and before omitting nitrous oxide the clinician who normally uses it, must ensure that the alternative technique does not produce worse conditions. Volatile agents with oxygen enriched air have been used to maintain anaesthesia. Alternatively an infusion of thiopentone has been used, but there is a significant problem of hypotension and accumulation. More commonly propofol infusions are used, but they are expensive and require sophisticated infusion pumps.

HYPNOTICS

Drugs which induce general anaesthesia are cerebral depressants with the exception of ketamine.

Barbiturates reduce CBF by both direct cerebral vasoconstriction and indirectly by a reduction in metabolism. Pierce in 1962 showed there was a dose dependent reduction in cerebral metabolic rate and cerebral blood flow which ultimately led to a reduction in cerebral blood volume [12]. It is this change in cerebral blood flow and cerebral blood volume which causes a fall in intracranial pressure and has been used therapeutically. Thiopentone is used for induction and has been used for maintenance as an infusion with nitrous oxide, but without narcotic supplements [13]. Depending on patient age, weight and general condition 500-1500mg of thiopentone is put into 500ml. The infusion is commenced at 1-3mg/min, titrated against patient reaction and finally stopped as the dura is closed. The infusion rates used ranged from 28 to 800mg/h, with a mean rate 300mg/h. However large doses of barbiturates must be used with caution in patients with raised ICP as they also cause a marked fall in blood pressure which will lead to a fall in cerebral perfusion pressure, and will accumulate, leading to prolonged recovery.

> **TEACHING NOTE**
> *It is more logical to consider each situation as it arises and the merits of the agents available. Many units, including our own, omit N2O when the conscious level of the patient is depressed due to serious intracranial decompensation.*

Propofol is an alkylphenol which has hypnotic properties and has a potency 1.8 times that of thiopentone. It has been solubilised in intralipid and causes both respiratory and cardiovascular depression. Propofol reduces CBF, CBV, ICP and cerebral metabolism. The drug causes a fall in blood pressure when it is given because of a reduction in both systemic vascular resistance and cardiac output. The drug is rapidly metabolised in the liver, even in those patients with cirrhosis. There is also some clearance in the urine. Anaesthesia, when maintained by a propofol infusion with either nitrous oxide and oxygen or oxygen enriched air, is followed by rapid recovery when used for not more than 3-4 hours. If used for many hours, some accumulation will occur (fig 5). One report related blood levels to the response to a supramaximal stimulus, analogous to MAC for inhalational agents. In the presence of 60% N_2O,

the propofol effector site concentration was 1.8 mg.ml^{-1} (95% confidence limits 1.4-2.34 mcg/ml; (14). Note the wide confidence limits, one of the factors that makes it more difficult to ensure the patient is not aware during a total intravenous technique.

> **TEACHING POINT**
> *Falls in blood and cerebral perfusion pressure are particularly risky in the elderly and emergency patient, when they will cause a significant fall in brain oxygenation. Any fall in blood pressure associated with the use of propofol can be avoided by reducing the bolus dose and titrating it cautiously in these vulnerable patients.*

A manual regimen has been described by Roberts et al [15] to achieve a maintenance level of propofol by commencing with a bolus, 1 mg/kg, and a rapid infusion rate (10 mg/kg/h for 10 min, 8m/kg/h for a further 10 min). This is subsequently reduced to the baseline maintenance rate of 6 mg/kg/h. This technique requires both narcotic supplements and N$_2$O. Propofol is a very useful agent for maintenance of anaesthesia of the neurosurgical patient, particularly when nitrous oxide is to be avoided. It should be noted that propofol infusions are expensive and require sophisticated infusion pumps. More recently the concept of Target Controlled Infusions (TCI) has been introduced where a specially designed syringe pump using a pre-programmed algorithm injects the drug at a rate necessary to achieve the blood level set by the clinician.

Ketamine is a derivative of phencyclidine that induces dissociative anaesthesia, and stimulates the cardiovascular system with minimal respiratory depression. However in patients with intracranial decompensation because of oedema or space occupying lesions, in contrast to other anaesthetic agents, it increases CMRO$_2$, CBF and ICP. These changes can be reduced by pre-treatment with hypocapnia, thiopentone or a benzodiazepine.

Following ischaemia the pathological mechanism which results in cerebral infarction involves the release of a number of neurotransmitters, a major one being N-methyl-D-aspartate (NMDA). Ketamine is a non-competitive antagonist at NMDA

> **TEACHING POINT**
> *All available evidence suggests that ketamine shoud be avoided if possible for anaesthesia for neurosurgery especially in those with raised ICP, or cerebral swelling leading to a decreased compliance or squashiness.*

receptors and may therefore offer protection from the adverse effects of cerebral ischaemia. This is a field under intensive research at present.

NEUROMUSCULAR BLOCKING DRUGS
Suxamethonium has been reported to raise intracranial pressure [16] in both man and animals. More recently other reports have failed to measure a rise in ICP in head injured patients following suxamethonium [17].

> **TEACHING POINT**
> *When presented with a patient who has a full stomach, safe establishment of the airway takes a priority. Despite the possible risks of raised intracranial pressure, in the emergency case, suxamthonium is the drug of choice for intubation.*

Pancuronium does not affect CMRO$_2$, CBF or ICP during induction of anaesthesia. It may cause arterial hypertension and tachycardia which may make intracranial conditions difficult during surgery.

Vecuronium is an intermediate acting non-depolarising neuromuscular blocking drug. Its principal advantage is that it has no significant effects on cardiovascular parameters. A preliminary report in neurosurgical patients confirmed that there was no effect on intracranial pressure or cerebral perfusion pressure [18]. Following an induction dose of 0.15mg/kg the onset time for neuromuscular blockade is approximately 140 seconds which is adequate for elective procedures.

Thus, in summary, vecuronium appears to have useful properties for inducing neuromuscular blockade prior to intubation in elective cases. It can be used to maintain neuromuscular blockade using an infusion technique. Alternatively, neuromuscular blockade can be maintained perfectly satisfactorily with the more established long acting or intermediate neuromuscular blocking drugs with the anaesthetist taking into account the unwanted side-effects.

> **TEACHING POINT**
> ● *Narctoics are required as an adjunct to the other agents to control the stimulation of surgery and the tube*
> ● *Narcotics will depress respiration, indirectly increasing CBF and ICP by raising PaCO$_2$*
> ● *Neurosurgical procedures are often long and require rapid but controlled emergence to assess the neurological status and ensure adequate respiration*
> ● *The ideal narcotic is one whose effects are predictable and can be controlled.*

NARCOTICS

It has been well established that narcotics given to conscious patients with some degree of intracranial decompensation will cause a rise in intracranial pressure. However, if ventilation is supported then the direct effect of narcotics in general on cerebral blood flow is minimal.

> **TEACHING POINT**
> *If a patient with trauma, which includes a head injury, is in severe pain this MUST be controlled. If there is major trauma the patient will probably be ventilated and thus narcotics can be used safely. If the patient is awake all other methods of practicable analgesia, such as local anaesthesia should be tried. However, if they are inadequate then small increments of morphine (1-2mg) can be given intravenously whilst the patient is given supplemental oxygen. It is stressed that supplemental oxygen will NOT prevent respiratory depression and a rise in arterial CO$_2$. It will only reduce the incidence and severity of transient desaturation. This can occur during sleep in patients receving narcotics. it is vital to observe changes in conscious level, neurological signs and respiration closely in an intensive care unit. This treatment is potentially hazardous but also essential for the patient. Thus it should only be carried out by those trained to recognise changes in cerebral status and to resuscitate the patient if required.*

Fentanyl is an established narcotic used in neuroanaesthesia, the peak effect occurring 4 minutes after injection and lasting for more than 15 minutes. Suppression of the cardiovascular response to painful stimuli can be achieved using 1.5-2.5 mcg/kg. There is no change in CBF or ICP, though if a large bolus is given a small decrease in arterial pressure can lead to a similar change in cerebral perfusion pressure. However it has been shown that fentanyl does accumulate and the duration of action can be up to 60 minutes or more if used over a period of time.

Pethidine is a synthetic opioid with similar properties to other narcotics, with additional atropine like effects. It can cause marked hypotension (minimised by slow intravenous injection), an important consideration in a patient with high ICP. The length of action is intermediate, 2h with a plasma half life 3-4hours. The usual intravenous dose is 0.5mg/kg. Pethidine has two drawbacks for neuroanaesthesia. It is metabolised in the liver to norpethidine, which is a convulsant. Norpethidine relies on excretion in the kidney, and thus this problem is particularly relevant in someone whose renal function is impaired. Secondly, pethidine is lipid soluble, thus if large doses are used, in a long and stimulating procedure for instance, the action can be prolonged.

In summary, pethidine is not an ideal agent, but if it is the only narcotic available then it is safe to use it, but with caution, while considering the properties mentioned.

Alfentanil is a more recent opioid which is less potent than fentanyl but has a very rapid onset of effect with shorter duration due to rapid excretion. Alfentanil has a very low volume of distribution and because it is not widely distributed throughout the body the amount required to produce an adequate effective concentration is less - hence its rapid excretion. This happens despite the clearance rate being similar to that of the more soluble opioid, fentanyl. It is analogous to the rapid emergence from the effect of an **insoluble** inhalational agent.

Suppression of the cardiovascular response to painful stimuli can be achieved with 10-30mcg/kg which will be effective within 1 min and last for 12 minutes. However, a bolus of this magnitude can cause a fall in arterial pressure, especially in elderly or emergency patients with a compromised circulation. This is very important when intracranial pressure is raised.

Initially there were reports that CBF, and ICP were increased by alfentanil. There appeared to be no explanation for this and the rises were not considered clinically significant. However these changes were accompanied by falls in arterial pressure which had a significant effect on cerebral perfusion pressure. Looking at the mechanism of autoregulation provides the answer for this otherwise unexplained rise in ICP. If autoregulation is functioning, as arterial pressure falls, a compensatory increase in CBF occurs, as explained in the previous article. The increase in CBF will happen because of a decrease in cerebral resistance, caused by dilatation of the cerebral arterioles. In turn this increases cerebral arterial blood volume, causes cerebral swelling and increases ICP if the brain is already enlarged. If this explanation is correct, when the opioid is given to patients with some cerebral swelling and blood pressure maintained by catecholamines, there will be **no** change in ICP. This study has been carried out by Werner [19] who noted that when BP was sustained there was no change in ICP following sufentanil, but when it was allowed to fall ICP rose. Although this work was carried out with sufentanil, another opioid with similarities to alfentanil, the mechanism is believed to be the same.

Remifentanil is an ultra-short acting esterase metabolised, mu-opioid receptor agonist. It is able to produce intense analgesia rapidly, and has potency similar to fentanyl. It has typical opioid effects of respiratory depression, bradycardia and skeletal muscle hypertonus. The major difference with this drug is that it is rapidly broken down by circulating and tissue non-specific esterases. Thus the ß half-life is 10-20 min with a plasma clearance of 3-4 L.min^{-1}. As recovery is so rapid, it is unaffected by the dose or the length of time that it has been given. The concept of context sensitive half-life has been introduced and will be considered below.

Early experimental work has shown that there is little difference between remifentanil and alfentanil on CBF or ICP. Studies in patients undergoing craniotomy have compared fentanyl, alfentanil and remifentanil [20,21]. A bolus administered over 1 min did not cause a significant rise in ICP (2-3 mmHg), but depressed blood pressure to an extent which was related to the dose (MAP 8 mmHg lower

dose). One of the problems with remifentanil is that the remaining analgesic which is very useful in the immediate postoperative period even for craniotomies does not occur. Therefore the problem of providing postoperative pain relief needs careful consideration.

Context sensitive half-time was discussed in a recent editorial [22] where it was defined as the time for plasma concentration to decrease by 50% after terminating an i.v. infusion designed to maintain a constant plasma concentration. Context refers to the length of the infusion. It has been demonstrated that the **context sensitive** half-time of both anaesthetic agents and opioids could differ markedly from elimination half-lives and that it is dependent on the duration of the infusion. The offset of action of a drug is not only a function of the elimination half-life but also of a number of complex factors which include the rate of equilibration between plasma and effector site, the method of administration and duration of infusion. It is also more relevant than simple elimination half-life data to clinical anaesthetists who give drugs over a period of time.

The method of administration can be either a continuous infusion or intermittent boluses. The context sensitive half-lives for different narcotics are illustrated (fig 4) and it can be seen that the half-life for remifentanil is unaffected by time. This would be expected, since there is no accumulation as it is rapidly metabolised. It is interesting to note the rapid increase in the context sensitive-half-life for fentanyl when used for more than a 2h period. When the drug is used over a long period of time, as it has a large volume of distribution and is fat soluble, a large amount is stored in the fat stores. It therefore takes a long time to be eliminated from the body, particularly as it is released slowly. Clinical experience supports this when fentanyl is used in long cases. Clinicians know that if they give large doses of fentanyl, even when the patient needs more analgesia, recovery will be slow. In contrast, although alfentanil does accumulate to some extent, it is predictable, appearing to level off at 60 minute context sensitive half-life after an infusion of 180 min. Thereafter longer infusions make little difference, again born out by clinical experience in long cases.
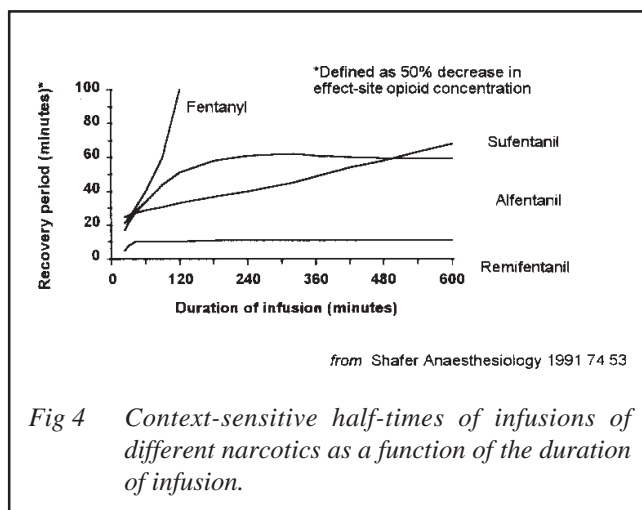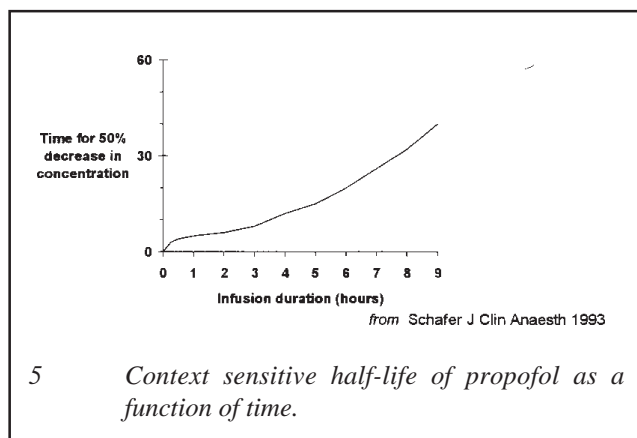
Fig 4   Context-sensitive half-times of infusions of different narcotics as a function of the duration of infusion.

When propofol infusions are used for more than 3-4 hours, recovery becomes longer. This can be understood by examining the context sensitive half-life (fig 5). Note that after an infusion of 6hours the context sensitive half-time is 20 minutes.

5   Context sensitive half-life of propofol as a function of time.

TEACHING POINT

The use of infusion techniques with newer narcotics are becoming more popular and enables the clinician to control the drug effect more accurately so that it peaks at maximum stimulation and wears off when no longer required. However, older drugs are still effective and have been used for many years successfully. Consideration of these new concepts will also help the clinican to use these drugs more accurately.

CONCLUSION

There are many new drugs available today which allow the clinician to improve techniques and skills. However, while these advances have some significance, the major benefits to the patient arise from careful assessment, and understanding of the problem with careful attention to basic principles of neuroanaesthetic management. These have been detailed previously but they can be summarised

- **Good clear airway**
- **Full oxygenation without hypercarbia**
- **Smooth induction with no coughing or bucking**
- **Careful monitoring of the patient**
- **Steady well controlled maintenance of anaesthesia**
- **Well controlled emergence and recovery**

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PREDICTION AND MANAGEMENT OF DIFFICULT TRACHEAL INTUBATION

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INTRODUCTION

During routine anaesthesia the incidence of difficult tracheal intubation has been estimated at 3-18%. Difficulties in intubation have been associated with serious complications, particularly when failed intubation has occurred. Occasionally in a patient with a difficult airway, the anaesthetist is faced with the situation where mask ventilation proves difficult or impossible. This is one of the most critical emergencies that may be faced in the practice of anaesthesia. If the anaesthetist can predict which patients are likely to prove difficult to intubate, he may reduce the risks of anaesthesia considerably. This paper reviews clinical techniques used for predicting difficulties in intubation and suggests different approaches to manage these patients.
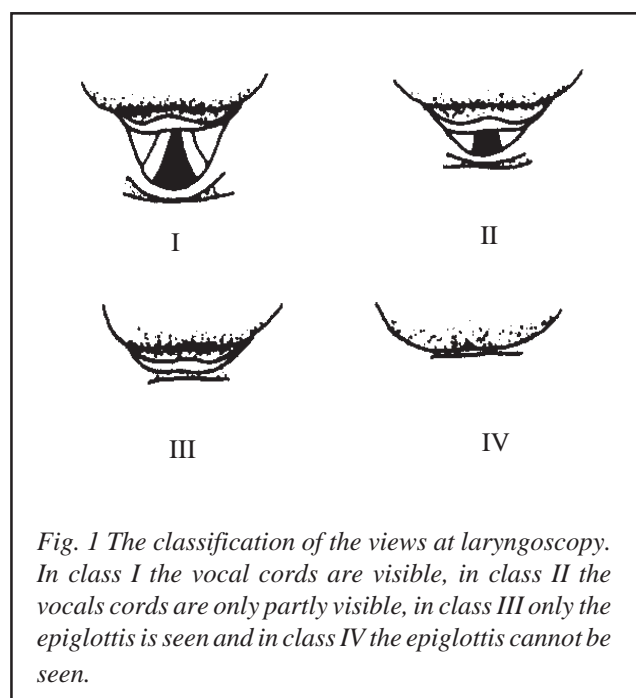

Fig. 1 The classification of the views at laryngoscopy. In class I the vocal cords are visible, in class II the vocals cords are only partly visible, in class III only the epiglottis is seen and in class IV the epiglottis cannot be seen.

There have been various attempts at defining what is meant by a difficult intubation. Repeated attempts at intubation, the use of a bougie or other intubation aid have been used in some papers, but perhaps the most widely used classification is by Cormack and Lehane [1] which describes the best view of the larynx seen at laryngoscopy (figure 1). This should be recorded in the patient's notes whenever an

anaesthetic is administered so there is a record for future use.

PREDICTING DIFFICULT INTUBATION

Tracheal intubation is best achieved in the classic "sniffing the morning air" position in which the neck is flexed and there is extension at the cranio-cervical (atlanto-axial) junction. This aligns the structures of the upper airway in the optimum position for laryngoscopy and permits the best view of the larynx when using a curved blade laryngoscope. Abnormalities of the bony structures and the soft tissues of the upper airway will result in difficult intubation.

History and examination

Pregnant patients, those suffering from facial/maxillary trauma, those with small mandibles or intra-oral pathology such as infections or tumours are all more likely to present difficulties during intubation.
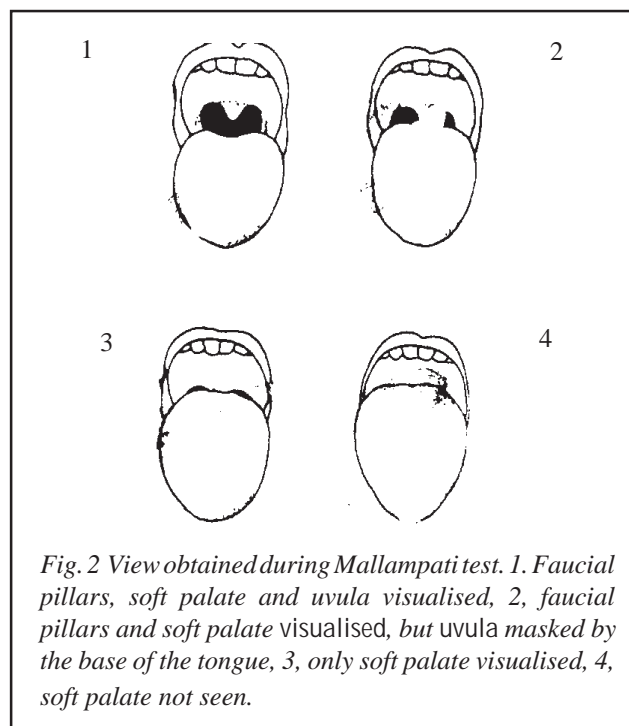
Patients who suffer with rheumatoid disease of the neck or degenerative spinal diseases often have reduced neck mobility making intubation harder. In addition spinal cord injury may result from excessive neck movements during intubation attempts. Poor teeth and the inability to open the mouth are obvious other factors as are obesity, and inexperience on the part of the anaesthetist.

Specific Screening Tests to Predict Difficult Intubation.

A history of successful or unsuccessful intubation during previous anaesthesia is obviously significant.

There a number of specific clinical assessments that have been developed to try to identify patients who will prove difficult to intubate.

Mallampati suggested a simple screening test which is widely used today in the modified form produced by Samsoon and Young [2]. The patient sits in front of the anaesthetist and opens the mouth wide. The patient is assigned a grade according to the best view obtained (figure 2). Clinically, Grade 1 usually predicts an easy intubation and Grade 3 or 4 suggests a significant chance that the patient will prove difficult to intubate. The results from this test are influenced by the ability to open the mouth, the size and mobility of the tongue and other intra-oral structures and movement at the craniocervical junction.

Fig. 2 View obtained during Mallampati test. 1. Faucial pillars, soft palate and uvula visualised, 2, faucial pillars and soft palate visualised, but uvula masked by the base of the tongue, 3, only soft palate visualised, 4, soft palate not seen.

Thyromental distance This is a measurement taken from the thyroid notch to the tip of the jaw with the head extended. The normal distance is 6.5cm or greater and is dependant on a number of anatomical factors including the position of the larynx. If the distance is greater than 6.5cm, conventional intubation is usually possible. If it is less than 6cm intubation may be impossible [3].

By combining the modified Mallampati and thyromental distance, Frerk showed that patients who fulfilled the criteria of Grade 3 or 4 Mallampati who also had a thyromental distance of less than 7cm were likely to present difficulty with intubation [4]. Frerk suggests that using this combined approach should predict the majority of difficult intubations. A 7cm marker can be used (eg a cut off pencil or an appropriate number of examiners fingers) to determine whether the thyromental distance is greater that 7cm.

Sternomental distance is measured from the sternum to the tip of the mandible with the head extended and is influenced by a number of factors including neck extension. It has also been noted to be a useful screening test for pre-operative prediction of difficult intubation. A sternomental distance of 12.5cm or less predicted difficult intubation [5].

Extension at the atlanto-axial joint should be assessed by asking the patient to flex their neck by putting their head forward and down. The neck is then held in this position and the patient attempts to

raise their face up testing for extension of the atlanto-axial joint. Laryngoscopy is optimally performed with the neck flexed and extension at the atlanto-axial joint. Reduction of movement at this joint is associated with difficulty.

Protrusion of the mandible is an indication of the mobility of the mandible. If the patient is able to protrude the lower teeth beyond the upper incisors intubation is usually straightforward [6]. If the patient cannot get the upper and lower incisors into alignment intubation is likely to be difficult.

Wilson et al [7] studied a combination of these factors in a surgical population assigning scores based on the degree of limitation of mouth opening, reduced neck extension, protuberant teeth and inability to protrude the lower jaw. Although their method can predict many difficult intubations, it also produces a high incidence of false positives (someone who is assessed as a likely difficult intubation, but who proves easy to intubate when anaesthetised) which limits its usefulness.

X ray studies Various studies have been used to try to predict difficult intubation by assessing the anatomy of the mandible on X ray. These have shown that the depth of the mandible may be important, but they are not commonly used as a screening test.

Preoperative assessment A combination of the above tests is better than using only one. The modified Mallampati, thyromental distance, ability to protrude the mandible and craniocervical movement are probably the most reliable.

Most patients without indicators of difficult intubation will prove easy to intubate under anaesthesia although occasional difficulties will occur. The majority of difficult intubations will be predicted by clinical assessment, but the tests will wrongly predict difficult intubation in some patients who will subsequently prove straightforward.

PREPARING FOR INTUBATION

Anaesthetists should be ready to deal with difficulties in intubation at any time. The correct equipment must be immediately available. This will include:

- laryngoscopes with a selection of blades
- a variety of endotracheal tubes

- introducers for endotracheal tubes (stylets or better, flexible bougies)
- oral and nasal airways
- a cricothyroid puncture kit (a 14 gauge cannula and jet insufflation with high pressure oxygen is the simplest and cheapest kit (see Update No 1996;6:4-5).
- reliable suction equipment
- a trained assistant
- laryngeal mask airways, sizes 3 & 4

The safety of laryngoscopy can be increased by preoxygenating the patient prior to induction and attempts at intubation. The anaesthetist should ensure that the patient is in the optimal position for intubation and must be able to oxygenate the patient at all times.

After intubation correct placement of the tube should be confirmed by:

- a stethoscope listening over both lung fields in the axillae
- observing the tube pass through the cords
- successful inflation of the chest on manual ventilation

Additional tests include the use of the oesophageal detector device (see Update No 1997;7:27-30) and a capnograph (if available).

Special techniques for intubation

When it is anticipated that a particular patient will present difficulties in intubation there are a number of options that need consideration. Regional anaesthesia is preferable to general anaesthesia whenever possible. However, in patients who require general anaesthesia with intubation, an awake intubation technique may be considered. This allows the patient to maintain their own airway and is the safest option.

Awake intubation under local anaesthesia

The aim with this technique is to anaesthetise the upper airway using local anaesthetic to allow tracheal intubation by a variety of techniques. This avoids the need for general anaesthesia and muscle relaxants to facilitate intubation. Either nasal or oral intubation may be performed, although the nasal route, despite the risk of haemorrhage, is often easier. The oral route is more stimulating and may be more difficult. The technique requires a co-

operative patient, and some experience on the part of the anaesthetist.

This technique may be performed using either a fibreoptic flexible bronchoscope or other fibrescope or using direct laryngoscopy. The patient is carefully prepared with a full explanation of why they are due to have awake intubation. Atropine 500mcg or glycopyrrolate 200mcg should be given intramuscularly half an hour before intubation to dry the mucous membranes, improving the action of the local anaesthetic and visibility. Oxygen at a rate of 2 - 3 litres/minute should be administered through a nasal catheter during the procedure (a suction catheter may be adapted for this purpose). The patient may be sedated gently during the procedure using small doses (2mg) of diazepam or other intravenous sedation. Small doses of opioid may also be helpful.

There are a variety of methods of producing local anaesthesia. Take care with the total dose of local anaesthetic used. A maximum of 4mg/kg lignocaine is usually recommended. Methods of producing anaesthesia include:

1. "Spray as you go" Lignocaine 2 - 4% is sprayed on to the mucosa of the upper airway as it exposed during the intubation process. This can be done via a special dispenser, or using repeated small boluses from a syringe with a cannula (not a needle) firmly attached. Some anaesthetists give an injection of 2mls of 2% lignocaine through the cricothyroid membrane. This anaesthetises the trachea and the under-surface of the vocal cords.

2. If nasal intubation is planned, cocaine (avoid in patients with ischaemic heart disease) is the preferred anaesthetic for the mucosa of the nose as it is an active vasoconstrictor and reduces the incidence of nasal haemorrhage. It is placed in the nose as a paste using cotton wool buds.

3. Nebulised lignocaine (4mls of 4%) is used by some anaesthetists and claimed to be a useful technique. However it usually requires topical supplementation and is not as good for nasal intubation.

After successful anaesthesia to the airway has been achieved, the patient may be intubated in a number of ways.

Oral intubation. Patients who are well prepared with good anaesthesia may often be intubated using a standard laryngoscope, but it is very stimulating unless preparation is excellent. If the laryngeal structures are easily seen during awake laryngoscopy general anaesthesia may be induced and the patient intubated conventionally. However patients who are difficult intubations will usually require a different technique. Awake fibreoptic intubation through the mouth is more difficult than through the nose due to the angulation involved in passing over the back of the tongue and round the epiglottis. In addition the patient may bite down on the endoscope unless a bite block is used.

Nasal intubation is the best method of awake intubation using a fibreoptic bronchoscope or other intubating fibrescope via the nose. The instrument is passed thorough the nose and into the trachea with an endotracheal tube mounted over it. After the trachea is entered the endotracheal tube is slid down over the scope into position. This equipment requires expertise and training and is not available in many parts of the world and will not be further considered in this article. However it should be remembered that there a variety of thin flexible fibreoptic scopes may be employed for awake intubation, including cystoscopes.

Some anaesthetists can perform a blind nasal intubation technique where a nasal endotracheal tube is gently passed through the nose towards the larynx. Breath sounds will be heard and the tube is guided in the direction of the loudest breath sounds by moving the patient's head until the larynx is entered. This technique requires a great deal of skill and expertise and is not feasible if the head and neck cannot be moved.

Retrograde intubation is a technique first described in Nigeria [8] for intubation of patients with cancrum oris. A wire or epidural catheter is passed through the cricothyroid membrane in a cephalad direction (towards the head) until it comes out of the nose or mouth. (In some patients it is necessary to grasp the catheter in the mouth using a pair of Magills forceps). At this point the patient has a wire running from within the trachea to the upper airway. An endotracheal tube is then inserted over this wire into the trachea from either the nasal or oral route. Ensure oxygenation is maintained throughout.

The bevel of the endotracheal tube should be posterior to make its passage into the larynx as

smooth as possible (figure 3). The tube may get obstructed at the level of the epiglottis or the vocal cords. There are a number of techniques to overcome this. The transtracheal wire may be used to guide a fibreoptic bronchoscope into the trachea and then the endotracheal tube placed over the scope. A larger hollow catheter may be placed over the wire into the trachea and the tube passed over the catheter. A disposable ureteric dilator which is hollow and has a tapered end is ideal for this. During the technique, if a nasal intubation is required but the wire comes out of the mouth, place another catheter through the nose, pull it out through the mouth, tie it to the wire, and then pull the wire up through the nose.
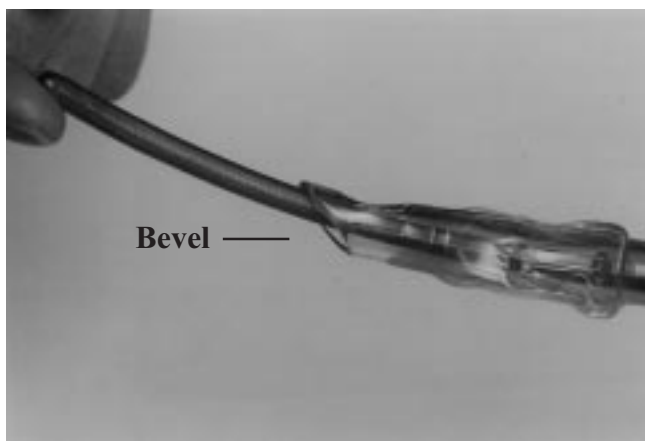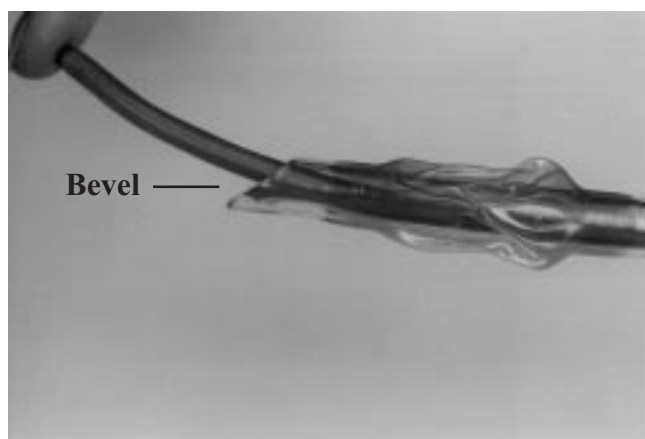

Bevel ————

Bevel ————

Figure 3. Bevel may stick on larynx if anterior
3b. Posterior bevel makes intubation easier

Retrograde intubation has recently been used successfully for traumatised airways when conventional techniques had failed [9], and there is a recent report suggesting that the membrane between the cricoid and first tracheal ring [10] can also been used.

Awake tracheostomy performed under local anaesthesia is the best solution when a patient is an impossible intubation, and regional anaesthesia is not a practical option. This is a straightforward technique, except in children, when sedation with ketamine has been used to facilitate this approach.

The Laryngeal Mask Airway (LMA - figure 4) is a common device in anaesthesia and can often provide a good airway in patients in whom intubation is difficult. Following insertion the anaesthetist may use it to maintain the airway during anaesthesia, or may use it as a route to allow tracheal intubation. A gum elastic bougie inserted down the laryngeal mask will often enter the trachea. A size 6 nasal tube may then be inserted through the mask, over the bougie, and the mask withdrawn if the tube enters the trachea. Unfortunately, on some occasions, the endotracheal tube is blocked by the fenestrations at the end of the laryngeal mask. The technique is best performed in conjunction with a fibreoptic bronchoscope.

Fig 4 The laryngeal mask airway

After insertion through the mask, the larynx may be identified and a bougie observed to pass through the chords. The scope and LMA are then withdrawn and an endotracheal tube passed in the normal fashion. Alternatively the scope may be introduced into the trachea and a size 6.0 tube inserted before the LMA is withdrawn A special intubating LMA has been produced to facilitate this manoeuvre (Intavent Medical UK).

The McCoy laryngoscope (figure 5) is designed with a movable tip which allows the epiglottis to be lifted and intubation often made easier [11]. It is manufactured by Penlon UK Ltd.

Fig 5 The McCoy Laryngoscope

A light wand is a long flexible device which has a bright light at the end and can be directed into the trachea with an endotracheal tube mounted over it [12]. When it enters the trachea the light can be seen shining through the skin. A darkened room is required, and the technique is more difficult in obese patients.

The Combi-tube (figure 6) is a tube which may be inserted blindly and used to ventilate the patient in an emergency [13]. It is designed in such a way that the tube can be used for ventilation whether it enters the oesophagus or the trachea. On insertion the tube normally enters the oesophagus, the large balloon is inflated and the patient is ventilated via the holes in the pharynx. If the tube is in the trachea then ventilation is carried out via the tube after the cuff has been blown up.

Oral gastroscopes can be used in the absence of a bronchoscope. The scope is used to find the larynx and direct a stylet into the trachea followed by an endotracheal tube. Alternatively a wire may be passed through the scope into the larynx and a tube passed over it.

Deep inhalational induction of anaesthesia using oxygen with halothane or ether is a technique that has been widely used for patients anticipated to be difficult intubations. If airway obstruction develops, anaesthesia may be turned off and the patient woken up. During induction, when the patient is deeply anaesthetised direct laryngoscopy is performed. If the larynx is visible the patient may be intubated directly or be given a muscle relaxant, and the patient intubated. If the larynx proves difficult to visualise, but the airway is easy to manage and face mask ventilation is straightforward, then a dose of

muscle relaxant (preferably suxamethonium) can be safely given. Intubation may then be attempted in the normal way; if it is unsuccessful, ventilation is continued using the face mask. If the airway becomes obstructed and cannot be cleared the patient is best woken up. If aspiration is a potential problem the induction is best carried out in the head down, left lateral position.

This method is the technique of choice to secure the airway in children with acute upper airway obstruction, particularly those with croup or epiglottitis.
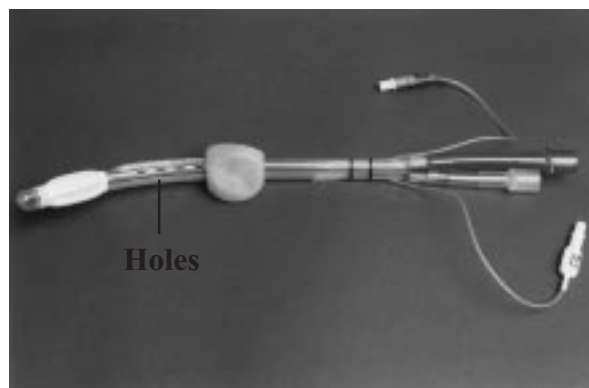

Fig 6 The Combi-tube

PLANNING ANAESTHESIA

When a difficult intubation is anticipated good planning is vital if anaesthesia is to be carried out safely. If general anaesthesia is essential and regional anaesthesia is not an option, the anaesthetist must decide whether the patient can be safely anaesthetised before an attempt is made at intubation. If the airway is likely to prove a problem then awake intubation is the best option before general anaesthesia is induced. **During general anaesthesia patients must never be given muscle relaxants unless the anaesthetist can be certain of being able to ventilate them.**

When the anaesthetist faces unexpected difficulty in intubation the priority is to ensure adequate mask ventilation and oxygenation of the patient. Multiple attempts at endotracheal intubation may result in bleeding and oedema of the upper airway making the task even more difficult. Often it is better to accept failure after a few attempts and move on to a pre-planned failed intubation sequence [14].

Failed intubation If intubation proves impossible the anaesthetist should consider whether to allow the patient to wake up and carry on surgery with regional anaesthesia, or whether to abandon the

surgery altogether. In situations where surgery is of an urgent nature it may be prudent to carry on the general anaesthetic under face mask anaesthesia if the airway is easy to maintain. If the airway is impossible to maintain and the patient is becoming hypoxic, an emergency cricothyroidotomy is required. If time allows an emergency tracheostomy can be considered.

Failure of face mask ventilation occurs when the patient has been anaesthetised and usually paralysed and face mask ventilation proves impossible. The priority is to ensure oxygenation by a number of emergency airway measures. The anaesthetist should attempt manoeuvres including chin lift, insertion of an oral and/or nasopharyngeal airway, and a jaw thrust procedure with both hands. If these techniques do not produce effective ventilation then an LMA should be inserted. (If an LMA is not available a Combi tube is another possibility). If there is still complete failure of ventilation then a cricothyroidotomy should be performed to deliver oxygen to the patient. Use a large intravenous cannula linked up to a high pressure oxygen system (as described in Update No. 6). There are commercially available devices for this purpose (Cook Critical Care Products). The cricothyroidotomy should be converted to an emergency tracheostomy as soon as possible (10 - 15 minutes maximum) or the patient allowed to wake up and regain their own airway.

Extubation of a patient who has been difficult to intubate should be performed with great care. There is a possibility that the patient may need re-intubation if there is a problem with extubation, and this may prove difficult or impossible. The patient should always be wide awake, co-operative and able to maintain their airway and ventilation before extubation is considered. If there are any doubts about the airway, the safest way to perform extubation is to insert a bougie or guide wire through the endotracheal tube and extubate the patient over this. The endotracheal tube may then be re-introduced over the bougie if the patient requires re-intubation. Some bougies are specially made for this (the Cook Critical Care endotracheal tube changer bougie) and have ports to insufflate oxygen through during the tube change.

CASE HISTORIES
A number of true cases histories are listed below to illustrate the management of difficult airway problems.

Case History 1
A 55 year old male was scheduled for resection of his parathyroid glands. He was known to be an impossible intubation as he had an immobile neck following spinal surgery and had previously had a failed attempt at a fibreoptic intubation. Tracheostomy was contraindicated because of the site of the surgery and an awake intubation was planned. He received atropine 500mcg premedication and then topical local anaesthesia to the mucosa of his upper airway. Midazolam 3mg was given for sedation, and 4 litres/minute of oxygen was administered throughout the procedure via a catheter in his left nostril. It was not possible to see the larynx with the fibreoptic bronchoscope. A retrograde intubation was carried out under local anaesthesia after placing a central venous line wire through the cricothyroid membrane. The wire came out through his right nostril and a size 7 nasal endotracheal tube was inserted over this into the trachea and the wire withdrawn. Anaesthesia was then induced, surgery performed and he was extubated wide awake at the end of the operation.

Case History 2
A child with an large nasopharyngeal tumour filling his mouth and nose was scheduled for surgical debulking (figure 7). There was clearly no possibility of a conventional oral endotracheal intubation and the patient received a tracheostomy under ketamine anaesthesia to secure the airway. He was given oxygen throughout the tracheostomy through his nose using a small face mask. Anaesthesia was continued via the tracheostomy and surgery performed.

Case History 3

A three year old girl was admitted to hospital with increasingly severe upper airway obstruction and a presumed diagnosis of epiglottis. She was taken straight to the operating theatre and anaesthesia was induced using oxygen and halothane. The anaesthetist maintained a degree of continuous airway pressure via the T-piece and after a prolonged induction he laryngoscoped the child to reveal a cherry red epiglottis. The cords were difficult to see until pressure on the chest revealed a bubble at the entrance to the larynx. An appropriate size of endotracheal tube was then inserted. The child was treated with antibiotics and was extubated in theatre 24 hours later when a leak had developed around the tube.

Case History 4

A 28 year old female who was known to be a very difficult intubation was scheduled for removal of a transplanted kidney. She was also known to be awkward to ventilate via a face mask, was very obese and had limited neck movements. Anaesthesia was induced after a successful fibreoptic awake intubation had been performed. During the operation it became impossible to ventilate the patient and after checking for problems with the circuit, airway and bronchi, it was decided that there was a problem with the endotracheal tube. Suction made no difference and the cuff was deflated and the tube pulled back in case it was against the carina. This did not improve the situation and so it was decided to change the endotracheal tube. The tube was removed but it was impossible to ventilate the patient via a face mask. A variety of techniques were tried, including most of the measures mentioned above. The surgeon carried out an emergency tracheostomy, but the patient suffered a hypoxic cardiac arrest. She later died from the complications of this episode. The cause of the difficulty in ventilation has never been satisfactorily explained. The endotracheal tube should have been changed over a tube changer or bougie. This would have minimised the risk of losing access to the trachea, which in this case proved fatal.

Further reading

In addition to the references in the text, articles written by Cobley and Vaughan and Biebuyck [15,16] are recommended. The technique of awake fibreoptic intubation has been well described by Telford and Liban [17].

Practical procedure

An emergency endotracheal tube introducer may be made using a reasonably stiff wire (such as from a wire coathanger) which is inserted inside a nasogastric tube [18]. The tip of the wire should be blunted or bent over before being placed inside the tube. The tube provides a non-traumatic covering for the introducer. The holes at the end could also be used to insufflate oxygen through if the device had to be used as a tube changer. It is easily diassembled for cleaning which should be done thoroughly.

Update in Anaesthesia No 7 described how to make a proper introducer for endotracheal tubes. Portex have recently introduced a range of inexpensive flexible bougies for intubation which have a hole down the centre. Although intended for single use, they could prove very useful to colleagues overseas.

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SELF ASSESSMENT IN NEUROANAESTHESIA

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Multiple Choice Questions - time allowed 30 minutes

1   Brain swelling causes

a)   a compensatory loss of CSF from inside the skull

b)   a reduction in cerebral arterial blood volume

c)   a reduction in cerebral venous blood volume

d)   an immediate rise in intracranial pressure (ICP)

e)   an estimated increase in ICP to 20 mmHg in a patient who has had a recent head injury which caused a brief period of unconsciousness

2   Cerebral venous blood volume is altered significantly by

a)   hyperventilating the patient

b)   placing the patient in a head-up position

c)   airway obstruction

d)   the patient coughing

e)   a fall in arterial blood pressure

3   Autoregulation

a)   is a central mechanism controlling ICP

b)   prevents a fall in cerebral blood flow (CBF) when there is a fall in arterial BP

c)   causes cerebral arterial dilatation when the arterial BP falls

d)   when the arterial BP rises to normal levels it leads to a fall in ICP in a patient with a swollen brain

e)   is unaffected by volatile inhalational agents

4   When the brain is stiff (low compliance) and enlarged, ICP

a)   rises only minimally when the patient coughs

b)   rises significantly with a small increase in arterial CO_2

c)   is unaffected by arterial desaturation (hypoxia)

d)   falls if the patient is put in the head-down position

e)   rises if the head is twisted to the left or right

5   Cerebral perfusion pressure (CPP)

a)   is satisfactory if more than than 70 mmHg in a patient with a head injury

b)   is calculated by adding mean arterial pressure (MAP) and ICP

c)   falls if arterial BP falls following induction of anaesthesia

d)   can be calculated by "guessing" ICP to be 20 mmHg after a head injury causing 5 min unconsciousness

e)   when low should be treated by infusing dextrose-saline solution

6   Cerebral blood flow

a)   is increased by acute hypocapnia (arterial CO_2 30 mmHg)

b)   changes affect ICP when brain compliance is low (brain stiffer or less squashy)

c)   is decreased by inhalation volatile agents

d)   is unaltered directly by opioids

e)   is decreased by the hypnotic agent thiopentone

7   Following a severe head injury, ICP will rise to damaging levels if

a)   the patient develops airway obstruction

b)   the patient becomes severely hypertensive

c)   the patient is allowed to breathe halothane spontaneously during an anaesthetic

d)   arterial hypoxaemia occurs

e)   the patient suffers severe pain from other injuries which is not treated

8   In a multi-trauma patient with a head injury, opioids

a)   can be used to treat severe pain

b)   cannot be given to a ventilated patient

c) can be given intramuscularly (IM) in the general ward

d) will cause a change to ICP in a ventilated patient whose blood pressure remains constant

e) will require the use of supplemental oxygen

9   Concerning inhalational volatile agents

a) the increase in ICP with halothane can be minimised by hyperventilating the patient

b) halothane is less soluble in blood than sevoflurane

c) recovery following anaesthesia with isoflurane is more rapid than after sevoflurane

d) during ether anaesthesia for neurosurgery, spontaneous respiration is acceptable

e) when the brain is swollen, if arterial blood pressure falls during halothane anaesthesia, it will not cause harm

10   Concerning intravenous agents

a) ketamine has no effect on ICP

b) thiopentone reduces ICP by direct cerebral vasoconstriction

c) a moderate fall in arterial BP following thiopentone in a patient with cerebral decompensation (raised ICP) need not be treated immediately

d) propofol does not effect cerebral metabolic rate

SHORT ESSAY QUESTIONS - Time 45 mins

1   A patient who has had a head injury 12h previously which caused a brief period of unconsiousness now requires an urgent general anaesthetic. Describe your management and technique with reasons.

2   What are the methods of pain relief which can be used in a patient who has a fractured ankle and a recent significant head injury.

Answers for the self assessment section can be found on page 49.

EXTRACTS FROM THE JOURNALS

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PREOPERATIVE FASTING

The duration of fasting before elective surgery remains an area of debate. Two editorials and one paper about fasting before surgery concluded that in patients scheduled for an elective operation the rule "nil per mouth after midnight" should be abandoned. Instead, both day-cases and inpatients, may take clear fluids by mouth up to 2 hours before surgery. There were no differences in gastric fluid volume or pH between patients who were allowed to drink free clear fluids until 2 hours before surgery (up to the time of premedication) and a control group who were fasted for 6 hours. However it should be noted that patients with factors likely to delay gastric emptying, such as pregnancy, trauma or opioid administration were excluded from the study. In addition, one would not advocate abandoning rapid sequence induction with cricoid pressure in those patients who come for emergency operations "with a full stomach".

1. Strunin L. How long should patients fast before surgery? Time for new guidelines. British Journal of Anaesthesia 1993;70:1-3

2. Brock-Utne JG.   Clear fluids, not breakfast, before surgery. Acta Anaesthesiologica Scandinavica 1996;40:549-53

3. Philips S et al.   Preoperative drinking does not affect gastric contents. British Journal of Anaesthesia 1993;70:6-9

MANAGEMENT OF RAISED INTRACRANIAL PRESSURE

A recent review by Pickard and Czosnyka [1] summarises the pathophysiology of patients with severe head injuries. Although the monitoring techniques described in the article may not be widely available for readers of Update the management strategies mentioned are very clear. The details on preventing intracranial hypertension in terms of general medical and nursing care is particularly useful.

The posture that the patient is nursed in is of great importance, bearing in mind venous drainage and cerebral perfusion pressure (see Update Number 8). Hypovolaemia should be avoided and a stable circulation maintained. Colloid, with an adequate plasma half life should be combined with careful electrolyte replacement.

Hyperpyrexia and hyperglycaemia should be avoided. Osmotic diuretics (mannitol) removes

water from both normal and oedematous brain, and its effects may be potentiated by adding frusemide. However it is crucial to avoid dehydration and hypotension. Mannitol should not be given if serum osmolarity exceeds 330mmol/l, or serum Na^+ exceeds 160mEq/l.

Aggressive hyperventilation may precipitate cerebral ischaemia. Systemic hypertension should not be treated directly with sodium nitroprusside. The majority of neurosurgical patients with hyponatraemia do not have inappropriate secretion of ADH and it is unwise to use fluid restriction to treat them even if they do. External ventricular drainage is a rapid procedure in a patient with hydrocephalus. There is a very restricted place for decompressive craniotomy following head injury. There is no benefit in using high dose steroids in head injury patients.

1. Pickard JD, Czosnyka M. Management of raised intracranial pressure. Journal of Neurology, Neurosurgery and Psychiatry 1993;56:845-858)

PRACTICAL TECHNIQUES IN DEVELOPING COUNTRIES

Dr David Williams
International Nepal Fellowship

Subarachnoid Saddle Block using Pethidine

Pethidine (Meperidine) is a synthetic phenylpiperidine derivative opioid agent with local anaesthetic and anticholinergic properties, and high lipid solubility. It is presented as a 5% solution (50mg/ml), which is slightly hyperbaric with respect to cerebrospinal fluid (specific gravity 1.026) [1]. These properties make it an ideal agent to use for subarachnoid anaesthesia if local anaesthetic agents are not readily available.

With the patient in the sitting position, a dose of 0.01 ml per kg body weight 5% preservative-free pethidine (i.e.0.5mg/kg) is diluted up to 2ml with sterile water or saline, and is slowly injected without barbotage into the subarachnoid space at the L3/4 level. The patient is kept in the sitting position for 5 minutes, then lies supine. This will give a satisfactory sensory block from S2 - S5 with an onset time of 4 - 8 minutes and duration of 1.5 - 2 hours; adequate for perineal surgery. Due to the action on spinal opiate receptors, there is good postoperative analgesia lasting for up to 5 hours; and motor block is of limited extent, facilitating early ambulation [2]. The cost per operation using pethidine is cheaper than heavy bupivacaine (100mg pethidine: 60 cents (US); 4ml 0.5% heavy bupivacaine: $1.60 (US) [3].

The potential side effects of intrathecal pethidine are related to its modes of action; namely local anaesthetic effects (motor & sympathetic blockade, hypotension, bradycardia), and opioid effects (sedation, respiratory depression, pruritis, nausea and vomiting) [4,5,6,7]. Dose related respiratory depression may occur with doses of >1mg/kg, and it is therefore recommended that premedication and perioperative administration of sedative drugs such as benzodiazepines should be avoided. Late onset respiratory depression is a risk of intrathecal opioid administration, and may occur several hours postoperatively. Although this is a well documented complication of intrathecal morphine administration, this problem has not been reported with pethidine due to the greater lipid solubility and hence reduced rostral spread of the latter agent [7]. However, it remains a potential complication, and therefore close monitoring in the postoperative period is mandatory, even though the patient may have full recovery of sensory and motor function. It is essential that only preservative free pethidine and diluents are used, as preservatives may cause arachnoiditis and irreversible neurological damage.

Larger doses of pethidine (up to 1 mg/kg) have been used to achieve higher levels of subarachnoid blockade up to T5 for urological, orthopaedic and gynaecological surgery [1,6,7], but the incidence of adverse effects is markedly increased. Positioning the patient supine without waiting for 5 minutes in the sitting position for the block to "fix" can lead to a higher and more variable level of block with increased adverse effects [6,7].

As with any subarachnoid block, blood pressure, heart rate, respiration and level of consciousness should be monitored continuously; and emergency drugs (fluids, atropine, vasopressors, naloxone) and equipment for intubation should be immediately available.

With these caveats in mind, saddle block using intrathecal pethidine is a cheap, safe and effective

alternative technique if local anaesthetic agents are unavailable or in short supply.

References:

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2. Acalovschi I et al. Saddle block with pethidine for perineal operations. British Journal of Anaesthesia 1986;58:1012-6

3. British National Formulary 35 ed (March 1998) pp205, 552

4. Khee WDN, Khor KE. Intrathecal pethidine: a word of caution. Anaesthesia 1993;48:276-7

5. Critchley LAH, Morley AP. Complications of intrathecal administration of pethidine. Anaesthesia 1996;51:711-2

6. Grace D, Fee JP. Anaesthesia and adverse effects after intrathecal pethidine hydrochloride for urological surgery. Anaesthesia 1995 Dec; 50(12):1036-40

7. Ngan Kee WD. Intrathecal Pethidine: Pharmacology and Clinical Applications. Anaesthesia and Intensive Care 1998;26:137-146

A Simple Reservoir System for Oxygen Concentrators

Domiciliary oxygen concentrators can produce high concentrations of oxygen from room air (up to 95%), but at the expense of a low flow rate (typically 1– 4 litres per minute). They may be used to increase the inspired oxygen concentration (Fi02) in a draw-over system and should be added using a reservoir attachment before the vaporiser inlet. During anaesthesia addition of 95% oxygen to the reservoir at a flow rate of 1 litre/minute will produce an FiO_2 of 35% - 40%; and a rate of 5 litres/min will produce an FiO_2 of up to 80% [1].

Augmentation of FiO_2 in this way is adequate for most circumstances. However, in certain situations, such as pre-oxygenation of an anxious hyperventilating patient, excessive entrainment of room air may occur with consequent reduction in FiO_2.

A simple solution to this problem is to prefill a large plastic sack (eg a bin-liner or clinical waste disposal sack) with concentrator "oxygen" and then attach this reservoir to the inlet side of the drawover system during preoxygenation. Avoid the risk of obstruction of the circuit from an empty sack by ensuring that the sack is removed as soon as preoxygenation and intubation is completed [2,3].

The greater FiO_2 afforded by this simple system enables more effective pre-oxygenation and hence an improved margin of safety during the period of intubation or extubation. It may be also be used in the lifesaving management of acute perioperative hypoxic events, such as cardiac arrest.

References :

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2. Johnson TW. Preoxygenation attatchment for the Triservice apparatus. Anaesthesia 1988;43:713

3.   Wilson, IH. Van Heerden, PV. Leigh J. Domiciliary Oxygen Concentrators in Anaesthesia: Preoxygenation Techniques and Inspired Oxygen Concentrations. British Journal of Anaesthesia 1990;65:342-345

An Improvised Precordial / Oesophageal Stethoscope

One of the most useful anaesthetic monitors is a precordial or oesophageal stethoscope. These give a continuous signal of heart rate, rhythm and breath sounds. By wearing the stethoscope in one ear only, the anaesthetist can continually monitor these parameters, whilst being able to communicate with colleagues in theatre.

Before leaving the UK to embark on a period of voluntary work, my hospital's Hearing Aid department moulded me a fitted ear-piece. A fine plastic tube was incorporated into the mould, which in turn snugly fitted and was glued into a short segment of 18Fr gauge tube.

The earpiece could either connected to an improvised precordial stethoscope (made from a 1 metre length of giving-set tubing attached to the head of a Littmann stethoscope and taped to the patient's chest) or an improvised oesophageal stethoscope (made from a second 18Fr gauge nasogastric tube, with the finger of a rubber glove securely tied and sutured to its distal end, to act as a diaphragm [1]). The combination could be worn comfortably and continuously.

This improvised system was a simple, cheap and effective method of ensuring continuous hands-free monitoring of vital signs during anaesthesia.

References

1) Dobson, MB. Anaesthesia at the District Hospital p33, WHO publications 1988

e)   the patient will recover rapidly when anaesthesia has been maintained by a thiopentone infusion

ANSWERS TO MCQ QUESTIONS

1   TFTFT

The normal compensatory mechanism for any brain swelling is a reduction in the volume of intracranial CSF, with squashing of the venous sinuses. This results in a reduction of venous blood volume in addition. However arterial blood volume is not altered. It is changed by autoregulation, arterial CO_2 and O_2 tensions as well as inhalational anaesthetic agents. As a result of compensation, ICP does not rise immediately. Normal ICP is 5-13 mmHg, it is sensible to "guess" ICP as being 20 mmHg after a head injury. This encourages the clinician to think always of, or to calculate cerebral perfusion pressure as arterial pressure changes.

2   FTTTF

Hyperventilating the patient has no effect on venous blood volume as this is a passive system unlike the arterial side. Thus positioning the patient, and anything that affects venous drainage or intra-thoracic pressure will alter cerebral venous blood volume. Similarly a fall in arterial blood pressure has no effect on venous blood volume

3   FTTTF

Autoregulation is a local mechanism which alters the tension in the arterial wall and so the calibre of the artery, controlling CBF. It is a basic mechanism which maintains a constant CBF over a range of arterial blood pressures, and so prevents a fall in CBF with a fall in arterial BP until the lower limit (50 mmHg MAP) is reached when CBF falls rapidly. By causing vasoconstriction, it alters arterial blood volume, which in a decompensated situation (swollen brain) can lead to a fall in ICP as arterial pressure rises over the normal range (50-150 mmHg MAP).

4   FTFFT

When the brain is swollen and stiff the system is working at the the right hand end of the brain volume - ICP curve (fig 2 Neurophysiology). Thus anything that will increase blood volume by even a small amount will increase ICP markedly. Coughing and the head-down position will increase cerebral venous volume. A rising arterial CO_2 and falling O_2 will increase arterial blood volume. Twisting the neck can obstruct venous flow in the great veins, hence patients with high intracranial pressure should be nursed with their head in the mid-line.

5   TFTTF

Cerebral perfusion pressure (CPP) is one of the most important variables to understand and have in mind when deaing with neurosurgical patients. Cerebral perfusion pressure is calculated by SUBTRACTING ICP from MAP.

$$CPP = MAP - ICP$$

Often ICP is not known, but it is reasonable to make a sensible guess that it is approximately 20 mmHg when the brain is swollen. This situation would exist for 3-5 days followng a significant head injury. Head trauma which is sufficient to cause unconsciousness, however brief is significant. CPP will fall if arterial pressure falls, but if the patients has a high ICP, it should be treated quickly with colloid, 0.9% saline (normal) or catecholamine boluses such as ephedrine 3-6 mg. It should never be treated with a potentially hypotonic glucose solution (5% Dextrose or 4% Dextrose/0.18% Saline). These solutions are contraindicated as they will exacerbate cerebral oedema.

6   FTFTT

CBF is decreased by arterial hypocapnia because of arterial vasoconstriction. It is also reduced by a direct vasoconstrictive action of the hypnotics, thiopentone and propofol. In contrast the inhalational volatile agents dilate the cerbral vessels so increasing CBF. The opioids have no direct effect, only increasing CBF if the patient who is breathing spontaneously becomes respiratorally depressed leading to a rise in arterial CO_2 tension.

7   TTTTT

If the airway of the patient with a severe head injury becomes obstructed, intra-thoracic pressure rise so increasing cerebral venous volume and ICP. Similarly respiratory failure may be present with a raised arterial CO_2 and low saturation (hypoxia). These will induce arterial cerebral vasodilatation, increasing cerebral arterial blood and then ICP. Volatile agents will increase CBF and ICP in their own right, but if a raised arterial CO_2 is present, the increase is greater. Spontaneous breathing during anaesthesia is always associated with some

respiratory depression. This combination would be severely damaging. In this situation anaesthesia can be maintained with halothane, but the patient needs to be hyperventilated to lower arterial CO_2. Pain will also increase CBF and ICP by a direct action on cerebral arteries. Therefore it should be treated.

8   TFFFT
A multi-trauma patient with a head injury is likely to be in severe pain. Pain can increase ICP, therefore it is not only humane to treat the patient it is intracranially beneficial. However if opioids can be avoided by the use of nerve blocks this should be done. If not suitable, then small intravenous doses of an opioid can be used with the patient's neurological status closely monitored in an intensive care or high dependency environment. In patients who are being ventilated, it is quite safe to use opioids. Provided blood pressure does not fall, there is no change in ICP. If BP falls, autoregulation induces cerebral arterial vasodilatation, which, in a decompensated state, will raise ICP. A patient who is breathing spontaneously, may also have a fall in arterial saturation. This would exacerbate the effects of a rising CO_2. Supplemental O_2 should be given whenever possible to reduce the risk of hypoxia, known to occur when a patient is under the effect of an opioid falls asleep.

9   TFFFF
The increase in ICP occurs because CBF and then arterial blood volume increases. Hyperventilation reduces arterial CO_2, this induces cerebral vasoconsriction which opposes the direct dilating effect of halothane on the cerebral vasculature. Sevoflurane is less soluble than both halothane and isoflurane. Therefore both induction and recovery are rapid. Any fall in blood pressure with halothane may be significant, but is especially important when the patient is decompensated (swollen stiff brain due to oedema, trauma or other pathology). In this situation blood pressure must be kept at control levels by preventing hypovolaemia and supporting the circulation with catecholamines either as boluses or as an infusion. The small doses required in a fully saturated, hypocapnic patient are unlikely to cause arrhythmias. The patient should be carefully monitored for them.

Ether is not an ideal agent for neuroanaesthesia, but if it is the only drug available the disadvantages can be reduced by hyperventilating the patient. Spontaneous breathing should not be allowed.

10   FTFFF
Ketamine increase ICP in patients who are decompensated. Thiopentone reduces CBF by a direct effect which leads to a fall in ICP. This effect is sustained by the reduction in metabolism, also a property of propofol. Any fall in arterial pressure when CPP is critical, the situation when the patient is decompensated with a raised ICP, must be treated immediately.

The rapid recovery following induction of anaesthesia with thiopentone is due to re-distribution of the drug from the brain to other parts of the body, the fat in particular. Therefore, when it is given as an infusion there is a significant risk that it will accumulate and cause a prolonged period of unconsciousness. This is not a major problem other than making neurological assesment difficult and creating the need for an intensive care bed with ventilation postoperatively.

SHORT ANSWER QUESTION POINTS
1.   A patient who has had a head injury 12h previously which caused a brief period of unconsiousness now requires an urgent general anaesthetic. Describe your management and technique with reasons.

Points to include in answers
1   Assess the patient for signs of intracranial decompensation, - Glasgow Coma Score, neurological signs: signs of other trauma, particularly the neck: concurrent clinical conditions and previous anaesthesia.

2   Note the physiology of brain swelling, the compensatory mechanism and problems of hypotension, hypoxia, hypercapnia and inhalational volatile agents.

3   Premedication: avoid, only use if the patient is very anxious and then cautious dose of sedative.

4   Describe monitoring patient, especially look for bradycardia and hypotension.

5   Describe induction, choice of agents is less important than the way they are used with an understanding of the complications, particularly

hypotension.

6   Maintenance with artifical ventilation. Again the choice of agents is less important than an adequate explanation of the properties that are useful and the side-effects and how they can be reduced.

7   Postoperative care should include: monitoring of neurological status, the signs of complications to be notes - increasing drowsiness, neurological signs, deteriorating Glasgow Coma Score: the problem off analgesia (see below)

8   Alternative techniques include nerve blocks, sciatic and femoral. They are useful but not reliable and if the situation gets out of control, the patient is worse off than if a good GA was used.

9.   Peridural techniques, epidural and spinal, are potentially risky. There is the problem of coning in the presence of cerebral swelling, if a significant leak in the dural sac occurs. In addition if an epidural technique is used, injection of local anaesthetic into the epidural space will transmit a rise of pressure up to the head.

2   What are the methods of pain relief which can be used in a patient who has a fractured ankle and a recent significant head injury.

Points to include in answer

1.   Use "mild" analgesics - oral or rectal. Paracetamol, and codeine phosphate are the main drugs. These preparations come as combination tablets. Alternatively, paracetamol can be given rectally and codeine by injection. Paracetamol should be given regularly initially. There is a risk of constipation with repeated doses of codeine.

2.   Non-steroidal anti inflammatory drugs are very effective analgesic drugs. However, they do reduce platelet effectiveness and should be avoided while there is a risk of intra-cranial haemorrhage, perhaps for 48h following the head injury.

3.   Local nerve blocks should be considered: femoral and sciatic nerve blocks. While they are unreliable for anaesthesia, they are effective for analgesia

4.   Opioids can be used as the final method when others methods have been found to be insufficient. Small doses of morphine, 1-2 mg or fentanyl, 10-20mcg, are acceptable. The patient should be nursed in an area with as much experience and nursing as possible, with facilities to intubate and ventilate the patient if necessary. Neurological status should be monitored: Glasgow Coma Score, conscious level, neurological deficits, with instructions to all medical staff if there is any deterioration. Supplemental oxygen should also be given.

LETTERS TO THE EDITOR.

Please submit letters to Dr I H Wilson, Editor, Update in Anaesthesia. We aim to publish those which we consider to be of interest to other readers.

Dear Sir,

Thank you very much for Update in Anaesthesia No8.

Update always contains useful and applicable articles, and I particularly welcomed this issue on Paediatric Anaesthesia. In my experience, the method described by Peter Bewes for using the EMO in children is very useful. I would add that I find it is often easier and safer to first give the child an IM or IV injection of ketamine and then continue with ether, either on a face mask or with intubation. This avoids some of the problems which can occur at stage 2 and makes it easier to breathe the child down so as to be able to intubate. After intubation it is possible to ventilate the child via the T piece with one hand, and then pump the bellows with the other if the theatre is short of staff. In this case a stethoscope strapped to the chest is mandatory.

Most places where I have worked have an EMO, an Oxford Inflating Bellows, ketamine and ether but very few have any kind of muscle relaxant. Another drawback with this method is that very few places have T-pieces, so I travel around with my own! In the area of Uganda in which I work, many of the hospitals have PAC vaporisers which can be used in the same way by replacing the EMO with the PAC.

Although it is best in such situations to use oxygen, unfortunately it is rarely available.

Please continue with Update, the advice contained is invaluable.

Yours sincerely,

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