Editorial

Welcome to Update in Anaesthesia number 20! This edition has been produced by the generous support of the International Relations Committee of the Association of Anaesthetists of Great Britain and Ireland. The Publications Committee of the World Federation of Societies of Anaesthesiologists would like to thank the AAGBI for their generosity and assistance.

This edition covers a number of topics which the editorial team hope will prove practical to our readers. In particular the articles on ketamine, fluid balance and servicing a seized OMV have all been specifically requested. Please continue to send requests for articles.

Readers of Update will be delighted to hear that a series of on-line tutorials are being produced on the website www.anaesthesiauk. com . These are available free of charge, and in time will form an entire curriculum of anaesthesia based topics. The tutorials are produced on a weekly basis, and cover a range of topics including clinical anaesthesia, obstetric and paediatric anaesthesia, basic sciences, regional techniques and intensive care. The tutorials can be downloaded from the website or received weekly by email by contacting carol@world-anaesthesia.org We look forward to feedback on this new venture.

The WFSA is also trying to increase the numbers of books and journals donated to developing world anaesthetists. If you are working without access to books and journals, and would like to request a book donation, please email carol@world-anaesthesia. org with details of yourself and your hospital.

Please inform us if you change address in order to keep our records up to date

Best wishes

Iain Wilson
Editor

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VASOPRESSORS FOR SUB-ARACHNOID ANAESTHESIA IN OBSTETRICS

Dr Ian Johnston, Department of Anaesthesia ,Raigmore Hospital, Inverness, University of Aberdeen
Medical School *Corresponding author. E-mail: igj@doctors.net.uk*

Despite recent studies[1] which suggest that spinal anaesthesia may not be the safest option for the fetus when caesarean section is required, it has, for many years, been the preferred technique for the majority of anaesthetists[2]. This is primarily due to the benefits conveyed to the mother. There are, however, a variety of complications and side effects associated with central neuraxial blockade in the pregnant patient, the commonest being maternal hypotension which is believed to occur in up to 95% of patients[3,4,5]. How important is this, and what should we be doing to prevent it?

Severe maternal hypotension gives rise to a reduction in utero-placental perfusion resulting in fetal bradycardia and acid-base abnormalities[6,7] and if prolonged, may lead to neurobehavioural changes in the newborn[8]. Although it has been suggested[9] that the duration of the period of hypotension is as important as the actual numerical value and, if transient, may be of little consequence, this concern indicates that it should be avoided or promptly treated. In the mother, hypotension leads to reduced cerebral blood flow which is associated with light-headedness and nausea and vomiting[10] . This, alone, may cause great discomfort and spoil what should be one of the most pleasurable experiences for the patient. Good control of the maternal systolic blood pressure will minimise, or even abolish, these unpleasant side effects[11,12].

Causes of hypotension

Spinal anaesthesia adequate for caesarean section will provide sympathetic nerve blockade up to T5 causing a fall in systemic vascular resistance (SVR). The normal reaction to this, in order to minimise the ensuing hypotension, is a reflex increase in heart rate and cardiac output. Denervation of the splanchnic autonomic ganglia (T5-11), however, also causes a significant venodilatation of the mesenteric bed with an increase in venous capacitance. This reduces the venous return to the extent that the reflex increase in cardiac output may be compromised or even abolished. These factors are compounded by the reduced venous return attributable to aorto-caval compression in the third trimester of pregnancy.

Spinal anaesthesia extending above T4 directly affects the cardiac sympathetic innervation, thereby attenuating the compensatory tachycardia and so a high spinal block may further reduce the heart rate.

Harrop-Griffiths[13] has suggested that another reflex (Bezold-Jarisch) may contribute to episodes of maternal hypotension in this setting. This reflex involves baroreceptors, in an under filled left ventricle, mediating a neural response which leads to increased parasympathetic activity over-riding the sympathetic tone. This reflex (although not fully understood) may explain why vasopressors and anticholinergic agents sometimes do not produce the expected results and also may explain the bradycardia seen in mothers in whom the spinal block is well below the T3-5 dermatomes.

Avoidance of hypotension

If no preventative measures are taken during caesarean section under spinal anaesthesia, the incidence of hypotension is reported as 92%[14]. Conservative measures include the avoidance of aorto-caval compression (left lateral tilt), and intra-venous fluid "pre-loading".

Aorta-caval compression does not only reduce maternal venous return and intervillous blood flow[15], but it also increases the spread of the spinal block, thereby compounding the issue. The deleterious effects of this have been recognised for many years and numerous papers have been published suggesting the degree of lateral tilt required to minimise them[16]. Although the majority recommend a 15 degree tilt to the left, this may prove to be inadequate and 20 degrees, or greater, may be required[17]. (It is recognised that anaesthetists are poor judges of the degree of tilt of the patient and frequently overestimate it [18]!) A full lateral position is much more effective than the lateral tilt in preventing hypotension[19] but only serves to delay its onset until the patient is repositioned for surgery.

As the reduction in cardiac preload due to mesenteric venodilatation is the root cause of the hypotension, it would appear logical to prevent this by expanding the circulating blood volume. Despite the fact that preloading the circulation with intra-venous fluids increases the cardiac output[20], its effects on maternal blood pressure are unreliable. However, colloids have been shown to be more effective than crystalloids when combined with leg wrapping[21]. There is, nevertheless, little evidence to suggest that either fluid type improves fetal well being, and some authors do not recommend their routine use[22]

Even with the careful use of these conservative measures, the incidence of hypotension may remain as high as 80-95%. To significantly improve this, vasopressors are indicated.

Vasopressors

Pressor agents raise the blood pressure, usually by vasoconstriction but also by increasing cardiac output. Vasoconstriction may be the result of central stimulation, direct action on the smooth muscle of the arterioles or venules, or by stimulation of the adrenergic receptors. In practice, the most commonly used drugs are the sympathomimetic agents which exert their effects via the adrenergic receptors. These may act **directly** on the receptor or **indirectly** by inducing the release of noradrenaline which then acts on the receptors. Because of their mode of action the indirectly acting drugs may exhibit tachyphylaxis (decreasing effect with repeated doses) on repeated administration.

The adrenergic receptors can be divided into alpha and beta (and dopaminergic) which are then further divided into sub-types 1 and 2. Alpha-1 receptors are distributed throughout the vascular smooth muscle and stimulation of them causes vasoconstriction. The alpha-2 receptors are located in the central nervous system and may cause sedation and analgesia. Beta-1 receptors are present in the heart and stimulation of them produces positive inotropic and chronotropic effects whereas beta-2 receptors are found in the bronchi, vascular smooth muscle and myometrium all of which are relaxed on stimulation. Stimulation of alpha-1 and beta-2 receptors will, therefore produce opposing effects on the vasculature smooth muscle.

Phenylephrine is a direct acting, potent alpha-1 agonist with no beta activity. It, therefore, causes a rapid increase in systemic vascular resistance and blood pressure. Metaraminol exhibits both direct and indirect activity. It has some beta activity but acts predominantly via the alpha-1 receptors so also increases systemic vascular resistance. Ephedrine is a potent alpha and beta agonist, acting both directly and also indirectly. Its effects on vascular resistance are less pronounced than the other alpha agonists but it also increases cardiac output thereby maintaining blood pressure.

Ephedrine was introduced into Europe in 1923 and by 1927 was being used to treat the hypotension associated with spinal anaesthesia. There was, however, a great reluctance to use vasopressors in the pregnant patient due to their effects on the uterine artery and Crawford (1966), and others[23,24], thought that the maintenance of maternal blood pressure by the action of vasopressors was responsible for foetal asphyxiation. Evidence supporting the use of ephedrine, however, came from the seminal paper by Ralston & Shnider (1974) in which uteroplacental blood flow (UBF) and fetal acid-base status were measured in pregnant ewes following the administration of equipotent doses of ephedrine, metaraminol, mephentermine and methoxamine[25]. When given in sufficient dose to raise the maternal arterial pressure by 50%, ephedrine had little effect on UBF and fetal arterial pH whereas the alpha-agonists caused a marked reduction in UBF. This study led clinicians to believe that ephedrine was the most efficacious agent in the prophylaxis and treatment of maternal hypotension for over 30 years. There are now, however, doubts about the relevance of this paper to humans. Quite apart from the inter-species variation, the ewes were studied in the standing position, were not anaesthetised and were rendered hypertensive by the infusion of vasoconstrictors. The animals also received doses of alpha-agonists greater than those used in clinical practice!

The fall in arterial pressure following spinal anaesthesia is caused partly by a reduction in systemic vascular resistance (SVR) but, predominantly by a reduction in cardiac output secondary to venodilatation and a decreased venous return. Pure alpha-agonists will correct the fall in the SVR and prevent venodilatation, thereby maintaining cardiac preload and output and preserving arterial pressure. A combined alpha- and beta- agonist has a lesser effect on SVR and venous capacitance so the patient remains vasodilated and venous return is reduced. Cardiac output, however, may rise following beta- mediated increases in myocardial contractility and heart rate, offsetting the fall in SVR.

These responses have been identified in clinical practice[26] and subsequently repeated in numerous studies. Following epidural anaesthesia for caesarean section, minimal changes in SVR were measured but there was a significant reduction in end diastolic volume leading to a compensatory tachycardia. Both ephedrine (alpha and beta agonist) and phenylephrine (alpha-agonist) corrected the fall in arterial pressure and maintained cardiac output at pre-epidural levels. Ephedrine, however, achieved this at the expense of an increase in heart rate whereas phenylephrine returned the heart rate to pre-epidural levels, suggesting that it is better at maintaining pre-load and stroke volume. Capacitance vessels in the splanchnic bed are more sensitive to vasoconstrictors than resistance vessels[27] so it is possible that careful use of low dose alpha-agonists may regain the majority of capacitance function, before there is a significant increase in vascular resistance, thereby preventing uterine vasoconstriction. These investigations support the theory that alpha-1 agonists should be superior to mixed alpha and beta agonists in preventing hypotension following spinal anaesthesia. They, therefore, should improve the condition of the mother but does this necessarily imply that they will improve the condition of the fetus?

The mature placenta is a high capacitance, low pressure organ with no autoregulatory and little vasoconstrictor

ability[4]. Pregnancy is associated with a reduced response to alpha- agonists particularly in the uterine arterioles[28]. Further studies on pregnant ewes[29] demonstrated that a gradual occlusion of the uterine arteries failed to produce fetal acidosis until the blood flow was reduced by over 60%. Uteroplacental perfusion pressure should, therefore, be dependant on the systemic arterial pressure.

Currently accepted signs of foetal compromise are, unfortunately, inadequate and unreliable. Though prevention of hypotension with phenylephrine has been shown to produce a higher umbilical artery(UA) pH than with ephedrine[30] this reflects both metabolic and respiratory acidosis, the latter of which may occur without fetal hypoxia. The more relevant measurement of standard base deficit, indicative of prolonged hypoxia, does correlate with neonatal outcome, but only at a magnitude unlikely to be seen in comparative studies. Doppler studies (pulsatility index) do not actually measure fetal well being or uteroplacental blood flow, but vascular resistance in the distal uterine bed which is dramatically affected by the physiological changes following spinal anaesthesia[31] and, therefore, subject to great variation. Apgar scores are highly subjective and poor predictors of neurological outcome and fail to demonstrate a correlation with maternal blood pressure. We cannot, therefore, be absolutely confident about the effects of drugs on the fetus!

A survey of obstetric anaesthetists practicing in the UK showed that over 95% still use ephedrine as their vasopressor of choice in caesarean sections[32]. This would suggest that, despite recent evidence to the contrary, they still are concerned about the potential effects of other agents on uterine blood flow. Both ephedrine and the alpha-1 agonists would appear to be **relatively** effective in maintaining maternal arterial pressure and many studies report there to be no significant difference in fetal outcomes when comparing the use of the various agents. Meta-analysis, however, has shown that ephedrine is associated with a more severe umbilical artery acidosis than the alpha- blockers phenylephrine and metaraminol, although this is inconclusive evidence with regard to the foetal compromise (vide supra).

This acidosis may be associated with an increase in umbilical arterio-venous CO_2 difference, suggesting an increase in fetal metabolic rate by direct beta-adrenergic stimulation[33]. Ephedrine readily crosses the placenta and fetal blood concentrations are approximately 70% of maternal. This has been associated with an increase in fetal heart rate and beat-to-beat variability[34]. Excessive administration is also associated with serious maternal cardiac arrhythmias particularly in the presence of a high spinal block and increased vagal tone.

Phenylephrine, while as effective as ephedrine in restoring maternal arterial pressure, does not affect the fetal circulation although it may cause a reflex bradycardia in the mother. Atropine rapidly restores the maternal heart rate without inducing a tachycardia, but this, itself crosses the placenta and may affect fetal haemodynamics[35]. A more suitable alternative may be glycopyrralate which has a more prolonged effect without crossing the placenta. Both of these anticholinergic agents may also reduce the incidence of nausea in the mother.

As the diagnosis of hypotension is retrospective, should we be administering vasopressors prophylactically to prevent rather than treat? Disadvantages of this are causing hypertension when the expected fall in pressure does not occur and inducing tachyphylaxis when using ephedrine. The response times to boluses of either phenylephrine (27s) or ephedrine (78s) is sufficiently rapid to warrant prophylactic treatment unnecessary[36] although the former would appear to be superior in its onset of action. Neither intramuscular nor intravenous ephedrine , when given prophylactically, demonstrate an improvement in neonatal outcome compared with their use in treating, and are not recommended[5].

In the absence of recognised vasopressors, alternative strategies must be used to minimise the fall in maternal blood pressure. In many situations increasing fluid preload is preferred but this, as has been shown, has limited efficacy. Adrenaline is frequently used for maintaining blood pressure in the critical care situation. It does this by its potent action on alpha-1 and 2, and beta-1 and 2 receptors. In low doses the beta effects predominate causing an increase in cardiac output but with a fall in systemic vascular resistance. As the dose increases, so does the alpha-1 activity leading to a rise in vascular resistance and an increase in blood pressure. This, unfortunately, predisposes the patient to cardiac arrhythmias and the beta-2 effects may causes a reduction in uterine tone at a time when there is an increased likelihood of haemorrhage. Adrenaline is, therefore, not a particularly suitable drug for the maintenance of maternal blood pressure but may be used in the emergency situation when other methods have failed. The drug should be titrated to effect and may be injected in 0.3 - 0.5ml. aliquots of 1:10,000, but a safer technique is controlled infusion of a more dilute solution e.g. 1:20,000

Conclusion

There is no doubt that avoiding maternal hypotension, following spinal anaesthesia for caesarean section, is important for the well being of the mother and fetus.

The patient should be placed in the lateral position while establishing the block and returned to a left lateral tilt

position (using a wedge in preference to lateral table tilt) for surgery. Although there is little evidence for the use of an intravenous pre-load, an infusion of 1,000mls of crystalloid may be given either before administering the spinal or while waiting for the block to be established.

Blood pressure should be monitored at regular and frequent intervals. Falls in maternal systolic pressure below 100mmHg or greater than 15% of baseline should be treated with a vasopressor.

Ephedrine has been the vasopressor of choice for the last 30 years. Despite increasing evidence that alpha 1-agonists may be more effective and less harmful than was once believed, it remains so! There is probably little difference in the efficacy of the various alpha-1 agonists, and many have been tried, but due to the limited number now being manufactured, we are generally limited to a choice between phenylephrine and metaraminol.

Ephedrine, phenylephrine and metaraminol may all be used to maintain an adequate maternal blood pressure and uteroplacental perfusion following spinal anaesthesia. The choice is frequently made on the grounds of the maternal heart rate - those with a tachycardia are given phenylephrine or metaraminol, and those in whom the pulse is less than 60/min., ephedrine. Some regard ephedrine as the safer agent as it is the one with which we are most familiar and, along with metaraminol, require a simple dilution to 10mls. Phenylephrine, conversely, requires a double dilution technique or, alternatively, dilution to a large volume (both 100mls and 500mls have been recommended) which has led to confusion, errors and overdosage. On the grounds, however, that the alpha- agonists are more specific in their action on the splanchnic venous bed (the primary cause of the hypotension) and probably cause fewer biochemical disturbances in the fetus than ephedrine, we should, perhaps be reviewing our thoughts on this 30 year old habit and ensuring a scrupulous technique in our drug preparation and administration!

Case Report

A 35 year female, para 2+0, was admitted at 37 weeks gestation for elective caesarean section delivery of her twins. Early delivery had been agreed due to increasing discomfort from her large abdominal mass, marked peripheral oedema, varicosities on her legs and haemorrhoids. Her previous obstetric history consisted of a spontaneous vaginal delivery followed, two years later, by an emergency caesarean section, for acute foetal distress, under epidural anaesthesia. Apart from the symptoms caused by her pregnancy she was in good health with a normal blood pressure. Having discussed the options available she had decided upon spinal anaesthesia for the procedure.

Following oral premedication with ranitidine 150mg and metoclopramide 10mg, a 16G intravenous cannula was inserted in her left forearm and 1000mls Hartmann's Solution infused. Blood pressure (BP) and heart rate (HR) were measured at 130/80 mmHg and 90 beats/min. With the patient in the sitting position, and under aseptic conditions, a 25G pencil-point tipped spinal needle was inserted through the dura by a midline approach at the L3-4 interspace. Having confirmed free flow of CSF, a mixture of 2.2mls 0.5% hyperbaric bupivacaine and 300mcg diamorphine was slowly injected. On completion of the injection the patient was immediately placed in the supine position, with a right lateral tilt, for 2 minutes. Her BP and HR were measured immediately and at 2 minute intervals.

The recording at 2 mins was 110/75 and 95/min. She was then rolled over to a left lateral tilt position to minimise aorto-caval compression. At this point she began to complain of feeling "light-headed" and her BP/HR were found to be 95/75 and 85/min. Metaraminol 0.5 mg was administered IV, but the patient remained "light-headed" and now complained of feeling nauseated. BP/HR were 85/45 and 85/min. A further 1mg metaraminol was injected but the patient became pale and clammy and unwell. ECG showed a sinus bradycardia of 40 beats/min. with multiple ectopic beats although pulse oximetry showed oxygenation to be satisfactory.

The heart rate was restored with glycopyrrolate 0.2mg but the patient's condition did not improve until she was rolled into a complete left lateral position. She then felt significantly better and her recordings were 125/80 and 115 beats/min. The height of the block was assessed, initially by cold (ice cube) and found to be T6 bilaterally. To enable the surgeon to proceed, the patient was again repositioned (with lumbar wedge) into a 20 degree left lateral tilt (with side supports to prevent her from rolling off the table). Although no longer nauseated, she again began to feel "light-headed" not withstanding her BP/HR measurements of 110/65 and 100beats/min. It was suspected that despite the significant lateral tilt, her excessively large uterine contents were contributing to continuing aorto-caval compression.

It was found impossible to increase the tilt and still allow surgical access. A further increment of metaraminol 0.5mg increased the BP to 135/80 without affecting the heart rate and improved the patient's condition. The surgeon was informed of the situation and delivered twin 1 rapidly. At this point she again began to feel much better and her HR fell to 85/min. although her BP remained at 135/85. Twin 2 took a further 8 minutes to deliver but cardiovascular recordings remained stable throughout this period. Following delivery the operating table was restored to the level position with no untoward effect on the mothers comfort. Syntocinon 5iu was administered by slow intravenous injection followed by an infusion of 10 iu./hour for 4 hours. Blood pressure fell slightly to 110/75 with a significant fall in heart rate to 65/min. Apgar scores at 1 and 5 minutes were 9 and 10 for twin 1 and 7 and 10 for twin two. Umbilical cord pH's were not measured. Closure of the abdomen proceeded uneventfully.

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FLUID MANAGEMENT FOR EMERGENCY LAPAROTOMY IN RURAL HOSPITALS

Dr Aleksandra Bojarska FCA RCSI, Consultant Anaesthetist, alexbojar@hotmail.com

Introduction

Abdominal emergencies are a frequent cause of death in sub-Saharan Africa. Using experience gained in hospitals in several different African countries and analysing perioperative data I have developed a protocol, which I believe could be useful for colleagues in other African hospitals. This protocol does not involve any expensive monitoring and is based on structured clinical assessment, simple measurements and treatment options, which are available in any hospital.

Anaesthesia in many African hospitals is provided by experienced and competent non-physician anaesthetists. They are rarely involved in preoperative management and frequently only meet the critically ill patient in theatre. Preoperative management is usually prescribed by a surgeon or general physician.

In spite of the correct surgical diagnosis, the critical condition of these patients is frequently not recognised. Profound dehydration, hypovolaemia, hypoxia and acidosis are often overlooked and not corrected. As a result perioperative mortality in this group of patients is very high. These patients sometimes require more then 10 litres of intravenous fluids in first 24 hours (excluding intra-operative fluids) and medical and nursing staff may lack the confidence to infuse such volumes. However treating this group of patients with aggressive resuscitation reduces the mortality significantly.

The purpose of this article is to encourage the perioperative team (anaesthetists and non-anaesthetists) to provide effective pre-operative preparation of critically ill patients.

Patient presentation

Patients presenting with abdominal emergencies often come to hospitals in Africa very late, especially if payment for treatment is required. They have often been sick for some days with a perforated or obstructed bowel. It is extremely important to establish the duration of their disease, as this can give some idea of the degree of dehydration and electrolyte imbalance. Reasons for dehydration include:

- no oral intake especially in children
- vomiting/ diarrhoea
- fever
- high environmental temperature
- third space loss (fluid in the body which is not available to the circulation for example oedema, ascites or other collections).

When taking the history enquire specifically about the first three and also the colour and amount of urine over the last day as profound dehydration and hypovolaemia will result in oliguria or even anuria. Consider the possibility of drug or herb ingestion by asking about visits to the local healer or ingestion of local or traditional medications. A green, leafy gastric content on naso-gastric (NG) tube insertion is suggestive even if the patient is not toxic.

> **Keypoint: Every patient with an 'acute abdomen' is severely dehydrated unless proven otherwise**

Physical examination

The ABCD framework should be used for both examination and initial management.

Airway is not usually a problem but should be checked in every patient.

Breathing - an increased respiratory rate (RR) is an early warning sign caused by acidosis or hypoxia and is often ignored. Tachypnoea can also be caused by pain, anxiety or pyrexia. Check the oxygen saturation and record respiratory rate regularly.

Tip: *make sure that nurses have a watch!*

Circulation - the cardiovascular system is usually significantly compromised due to hypovolaemia. Assess:

- heart rate (HR)
- blood pressure (BP)
- pulse - is it weak or well filled?
- capillary refill time; make sure it is done properly - press for 5s (count to 5) then release the pressure and count refill time. This sign is very accurate in children and young adults, less reliable in very anaemic or old patients. *Tip*: *teach nurses how to do it - you do not even need a watch for this!*
- core - peripheral temperature gradient - check the difference between the temperature of the trunk, which is usually hot (pyrexia) and the extremities which are cold (vasoconstriction). This is a very good indicator of

the intravascular volume; especially useful to observe the trend - the difference should reduce during resuscitation.

● degree of dehydration - severe thirst, decreased skin turgor, dry tongue, sunken eyes, sunken fontanelle in a newborn. However, decreased skin turgor or sunken eyes may be masked by oedema resulting from hypoalbuminaemia.

Disability - assess the mental status; adult patients can be apathetic occasionally agitated; children can fluctuate between being apathetic and agitated.

Document all of your findings on an appropriate chart.

> **Key point - it is important to realise that these patients do not only have an abdominal problem, but multiple organ impairment.**

Figure 1.	*Common problems in an emergency laparotomy*
Cardiovascular	● Hypovolaemia ● Dehydration ● Sepsis and septic shock
Respiratory	● Hypoxia ● Tachypnoea ● Atelectasis
Blood	● Anaemia ● If septic - potential coagulopathy
Renal	● Oliguria or anuria due to acute renal failure
CNS	● Decreased level of consciousness, confusion ● Anxiety ● Pain ● Possibility of intoxication
Gastro-intestinal	● Full stomach ● Abdominal distension ● Bowel perforation or obstruction
Metabolic	● Pyrexia ● Hypothermia ● Acidosis ● Electrolyte disturbance ● Hypoglycaemia

Management

The main purpose of the preoperative treatment is to optimise the patient's condition and maximise their chance of survival.[1,2] Early effective resuscitation improves oxygen delivery to the tissues and reduces mortality in this group of patients.[3]

Preoperative resuscitation obviously takes time but long delays before surgery should be avoided as early surgical management improves the outcome in septic patients. The preoperative plan should be discussed between the surgical and anaesthetic teams to achieve the right balance between providing adequate resuscitation and the risk of delaying surgery. Most patients will benefit from 2 - 4 hours preoperative resuscitation. The best area to carry out resuscitation is ICU/HDU if available.

Make a management plan following the ABC framework.

Airway and Breathing

Provide oxygen with the face mask at 2-4 l/min

Circulation

● insert iv cannula, preferably 16G or 2x 18G

● take a sample for Hb, electrolytes and consider crossmatching (see below).

● Infuse first litre of normal saline or Hartmann's rapidly over 15 min. During the following hour give 2000mls, watching clinical signs.

● insert Foley catheter; measure and record the initial amount and colour (concentration) of urine in the bag and discard it.

● If the initial Hb is < 12g/dl and the patient is severely dehydrated and hypovolaemic, crossmatching is essential as the patient is most probably severely anaemic, and the "normal" Hb level is due to haemoconcentration

Further treatment

● request urgent surgical opinion.

● if abdominal X-ray requested by surgeon, and patient is very sick make sure that he/she is transported to X-ray department on a stretcher or wheel-chair and iv fluids are continued. If possible accompany the patient.

● antibiotics prescribed should be administered iv as soon as possible

● insert NG tube

● check temperature

> **Keypoint - make sure that patients are not operated on while still hypovolaemic, hypoxic, and oliguric.**

Further management

Assess the patient after each 1 - 2 l of fluids. Whenever possible warm the fluids even if patient is pyrexial. Use either crystalloids and colloids, but avoid glucose 5 or 10%. Hartmann's solution seems slightly better then normal saline as it results in less hyperchloraemic acidosis - see page 15. The correct volume of fluid is more important then the type. When the initial resuscitation is completed, potassium containing fluids (20mmol KCl /litre) may be used providing there is an adequate urine output.

Tip: *No glucose 5% or 10% for preoperative fluid resuscitation!*

Assessment of progress of resuscitation involves assessment of:
- HR
- BP
- capillary refill time
- RR
- Improving peripheral temperature
- filling of neck veins
- urine output

After 4-5 l of IV crystalloid (or 1.5 - 2 l of colloid) it may be worth repeating the Hb level to assess whether a blood transfusion is likely to be required.

In case of severe anaemia (Hb < 4g/dl), which is frequently accompanied by hypoproteinaemia, there is a significant risk of pulmonary oedema. In such cases blood should be transfused in early stages of fluid resuscitation.

It is important to explain management to all staff involved or the IV infusion will be slowed down as soon as you leave the bedside. An anaesthetist needs to supervise the rapid resuscitation and not leave it to ward nurses.

What about measuring central venous pressure (CVP)?

CVP measurement provides valuable information in selected cases, especially if urine output is low despite infusing large amount of fluids. However it is frequently unavailable in rural African hospitals because of a lack of expertise with invasive monitoring. Even if it is available one has to weigh benefits against risks. Repeated assessment of capillary refill, cardiovascular observations and filling of the neck veins usually give reasonable indication of intravascular volume.

What about inotropes?

Adrenaline and noradrenaline are frequently used in developed countries to treat sick patients with abdominal emergencies. I believe however, that there is much less requirement for their use in sick patients treated in rural hospitals. These patients, who are predominantly young and previously fit, respond very well to simple, adequate fluid resuscitation, provided adequate volumes are given.

From my experience, patients who do not respond to fluid resuscitation and require an inotrope infusion have a very high mortality and are often in an irreversible clinical situation. Inotropes can also divert attention from providing adequate fluid resuscitation by increasing the blood pressure without adequate volume expansion.

In cases of septic shock, adrenaline or noradrenaline can be used provided adequate fluid administration has been achieved.

Electrolyte imbalance

Often it is not possible to measure electrolytes. K^+ levels are important as cardiac arrhythmias may result from hypo or hyperkalaemia. Sick patients are normally depleted due to K^+ loss from diarrhoea and third space losses. However, anuric patients are at risk of hyperkalaemia. After initial resuscitation, when the patient is passing good volumes of urine, it is justified to add 20 -40mmol of KCl to each litre of IV fluids.

Anaesthesia

The correct timing of anaesthesia and surgery depends on the underlying problem. Resuscitation should be as complete as possible, but delay dramatically increases the risk to the patient in cases of peritonitis or bleeding.

Ideally following resuscitation and before anaesthesia, the patient will be stable with a pulse less than 100/min, a blood pressure greater than 90 systolic, established urine output and good capillary return.

Patients require general anaesthesia with intubation and ventilation. Diligent preparation is extremely important. On top of the usual routine preparation and equipment check there is enough oxygen for a long case, adequate amounts of IV fluids (warmed) and high volume suction. Empty the urine bag and suction NG tube.

Very sick patients are frequently hypotensive immediately after induction. Make sure there is a large bore IV line through which you can infuse fluids fast.

Prepare "emergency" drugs:
- ephedrine or metarminol or other vasopressor, ready and diluted in the syringe
- atropine

In case of high risk patients I also prepare diluted adrenaline in two concentrations:
- 1 mg of adrenaline (1ml) diluted to 10 ml (concentration 1:10 000, 100mcg/ml)
- 1ml of the above solution diluted to 10 ml (concentration 1:100 000, 10mcg/ml)

Induction

Preoxygenation is followed by rapid sequence induction with cricoid pressure. Thiopentone or ketamine can be used. In hypotensive patients, ketamine is a better choice. This should be followed by suxamethonium, non-depolarising muscle relaxant and an opioid analgesic. The cricoid pressure is absolutely mandatory as regurgitation is almost guaranteed. It is worth teaching everybody in theatre how to do it.

Maintenance

In hypotensive patients maintenance with a ketamine infusion (500mg ketamine/500ml of fluid) has some advantages over halothane which can cause hypotension and arrhythmias, especially in patients with electrolyte imbalance.

It has been suggested that keeping inspired oxygen level around 80% intra-operatively and for 2 hours after surgery might reduce the incident of wound infection and post operative nausea and vomiting (PONV).[4,5]

If hypotension follows induction of anaesthesia, it should be treated with rapid infusion of fluids and ephedrine or adrenaline boluses. If hypotension does not respond to vasopressor, adrenaline is indicated. I usually give 1 - 2ml boluses of the more dilute adrenaline solution and change to more concentrated only if I use the whole 10 ml syringe within relatively short time (around 15 minutes).

During anaesthesia make sure that the patient receives an adequate amount of fluids and use ephedrine or adrenaline as your second line of treatment. In some patients an adrenaline infusion during anaesthesia is useful to prevent hypotension and can be continued postoperatively (5mg adrenaline/500ml). In septic patients who are unresponsive to inotropes, hydrocortisone 100 mg should be considered.[6]

Normothermia during and after surgery improves recovery, decreases oxygen consumption (increased by shivering), reduces wound infection and decreases blood loss. Intravenous fluids should be warmed, as patients always cool down during surgery, especially in air-conditioned operating theatres. This can be achieved by putting the fluids into a simple water bath. Using hot-water bottles (wrapped in cotton sheets) and applying them to armpit and groins can also help to warm up patients.

Appropriate antibiotics should be administered pre- or intra-operatively.

Post-operative period

Patients are best managed in a recovery area, and then in an Intensive Care Unit (ICU) or High Dependency Unit (HDU) if possible. In many hospitals in rural Africa this is an area where good nursing care is available, but there are often no ventilators or infusion pumps. Supplementary oxygen (3-4 litres/minute) should be continued for the first 24 hours if available.

Careful monitoring of basic physiological parameters (RR, HR, BP, oxygen saturation, urine output, temperature) is essential over next 24 hours. Signs such as tachypnoea, tachycardia, hypotension, hypoxia, oliguria, changed mental state or hypothermia should trigger immediate review by the medical staff.

Adequate pain control should be established. This is usually achieved by intravenous opioid in recovery followed by intramuscular injections when required. Paracetamol suppositories can be a valuable addition. NSAID suppositories should be used only in patients with good renal function.

Intravenous fluid requirements will remain high in the immediate post-operative period. Patients will continue to have third space loss and residual fluid deficit from the preoperative period. Therefore fluid requirements will be above the maintenance amount of 3 litres per day. Often 4 - 6 litres are required in the first 24 hours and should be given as Hartmanns or Normal Saline. Although the calculated fluid balance will be positive, increased insensible losses (fever, tropical environment), fluid loss from drains and continuing third space losses due to the underlying pathology result in continued fluid deficit in the circulation. Adding 20mmol of potassium to each 1000ml bag of fluid is recommended, providing the urine output is adequate. The daily requirement of potassium is 70 - 100mmol.

Summary

Finding the right balance between appropriate pre-operative resuscitation but not delaying surgery unnecessarily seems to be the key to successful treatment of sick patients with abdominal emergencies.

Appropriate assessment and management by medical and nursing staff is based on interpretation of simple parameters which do not require any sophisticated monitoring equipment, and can be achieved in any rural hospital in Africa.

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PERIOPERATIVE FLUID BALANCE

Dr W A English[1], Mrs R E English[2], Dr I H Wilson[3], [1]Department of Anaesthesia, Bristol Royal Infirmary, Bristol; [2,3]Royal Devon and Exeter NHS Foundation Trust, Exeter

Fluid balance is an important area of perioperative medicine. If managed incorrectly it is a significant cause of morbidity and mortality. This article will discuss:

- aims of adult perioperative fluid therapy

- fluid and electrolyte physiology

- assessment of hydration and volume status

- different fluid preparations

- controversies in fluid management

- guidelines for fluid management in surgical patients

AIMS OF FLUID THERAPY

Perioperative fluids are required to maintain adequate:

- hydration

- blood volume and oxygen delivery

- renal function

- electrolyte balance

- splanchnic and hepatic circulation

FLUID AND ELECTOLYTE PHYSIOLOGY

Fluids and electrolytes are present in a number of "compartments" in the body, according to their chemical composition. Plasma is the fluid component of the blood surrounding the red cells, intracellular fluid is the fluid within the body's cells, and interstitial fluid is the fluid found between the cells, outside of blood vessels.

Water is present in plasma, interstitial and intracellular fluid volumes and passes freely between compartments, under the influence of osmotic pressure gradients. The interstitial fluid volume and plasma volume together make up the extracellular volume (ECF). Water accounts for 60% of adult body weight (total body water = 42 litres for a 70kg adult). Of the 42 litres, 3, 11 and 28 litres are found in the plasma, interstitial and intracellular compartments respectively. Osmolality is kept constant between all compartments by the movement of water by osmosis.

The ECF contains most of the sodium in the body, with equal sodium concentrations in the interstitial fluid and plasma. Sodium and water can pass freely through capillary membranes whilst albumin (the most important oncotically active constituent of the ECF) does not. Albumin is unequally distributed in the intravascular and interstitial compartments (normal concentrations of 40g/l and 10g/l respectively) and is excluded from the intracellular compartment. This distribution helps to retain fluid within the plasma due to the osmotic effect of albumin. The predominant intracellular anion is potassium.

This information predicts the distribution of infused fluid. Sodium free water e.g. glucose 5%, will be distributed throughout the total body water as it freely crosses cell membranes. Less than 10% of the infused volume remains in the plasma.

An infusion of a crystalloid solution, with a sodium concentration of approximately 140mmol/l, will be

distributed throughout the ECF as the sodium and water will move freely across the capillary membrane, but will not enter cells. Therefore around a third of infused normal 0.9% saline or Hartmann's solution remains in the intravascular volume. Colloid solutions are retained primarily within the plasma volume due to the effect of their albumin like content, providing an added osmotic (or oncotic) effect. However over time colloids leak across the capillary membrane entering the interstitial fluid space, or are metabolised.

NORMAL FLUID AND ELECTROLYTE REQUIREMENTS

Average adults in temperate climates lose between 2.5 and 3 litres of water per day (1300-1800mls urine, 1000mls insensible loss from lungs and skin, 100mls in the faeces). Normally fluid enters the body orally although around 200mls/day is produced from metabolic processes. Average adults lose about 1.5mmol/kg/day of sodium ions and 1 mmol/kg/day of potassium ions in the urine.

If a patient is nil by mouth then normal daily requirements may be provided by:

1000mls	Normal saline 0.9%
1500-2000mls	Dextrose 5%
60 mmol	KC1

ASSESSMENT OF HYDRATION STATUS AND INTRAVASCULAR VOLUME

Fluid management in the perioperative period involves maintaining the intracellular and extracellular fluid volumes.

Dehydration reflects loss of water. This may come from extracellular fluid (ECF) and intracellular fluid (ICF) depletion. Sodium is usually lost at the same time, giving rise to hyper- or hyponatraemia, depending on the relative degrees of loss. If ECF osmolality rises, water passes from the ICF into the ECF by osmosis. Predominantly water loss is therefore shared by ECF and ICF, whilst water and sodium loss is mainly from the ECF. This explains why fluid losses from fever and lack of intake (mainly water loss) may be tolerated for longer than severe vomiting or diarrhoea (water and sodium loss).

Hydration status and intravascular volume is assessed by the patient's history, examination, test results and response to intravenous fluid administration.

History may detail causes of perioperative dehydration and intravascular volume depletion such as inadequate oral intake, vomiting, diarrhoea, bowel preparation, haemorrhage, burns, drain losses and third space losses.

Physical examination will include: pulse rate, arterial blood pressure, respiratory rate, urine output, JVP, capillary return and mucous membranes. The physical findings for various degrees of intravascular volume loss and dehydration are given in Tables 1 and 2. Table 1 also provides guidelines for the type of replacement fluids that should be used for each category of hypovolaemic shock. Choice of replacement fluid for dehydration will depend on which fluid compartments have been depleted. Initially electrolyte solutions such as normal saline or Hartmann's are used to replace ECF losses. Initial fluid boluses in moderate to severe dehydration should be 10-20ml/kg followed by reassessment of the patient and further fluids guided by clinical signs.

Clinical signs of hypovolaemia may be more difficult to determine during anaesthesia. The same physical variables should be measured, together with an estimation of ongoing intraoperative losses. Tachycardia alone is an insensitive marker of hypovolaemia. A normal pulse rate, blood pressure and CVP of 6-12mm Hg suggest adequate blood volume. Arterial blood pH and serum lactate are useful indicators of effective resuscitation. Urine output falls with hypovolaemia, and an output of 0.5 to 1.0ml/kg/hr during anaesthesia suggests adequate renal perfusion and intravascular volume. The physiological response to fluid administration, via repeated fluid challenges is a practical part of volume assessment.

Advanced monitoring of fluid status may be necessary for some patients with pre-existing pathology undergoing complex surgical interventions. Techniques utilised include CVP measurement, pulmonary artery catheterisation or newer cardiac output monitoring such as oesophageal Doppler ultrasound.

In perioperative care the emphasis is to use IV fluids to maintain the circulating volume and tissue oxygen delivery. There is good evidence that patients undergoing major surgery in a dehydrated, un-resuscitated state do worse than those who have received adequate IV fluid preoperatively. These patients are unable to react to the stress of the surgery and underlying illness, develop intracellular hypoxia and organ dysfunction.

In emergency patients, the time available for resuscitation is always limited, but early effective resuscitation may be lifesaving.

Laboratory investigations are useful, particularly the haematocrit, urea and electrolytes. An initially raised haematocrit (or Hb) may reduce substantially after fluid replacement.

FLUID PREPARATIONS

There are three types of intravenous fluids: crystalloids,

Table 1: Grades of hypovolaemic shock secondary to intravascular volume loss, their physical findings and suggested replacement fluids for a 70kg man

	Class I	Class II	Class III	Class IV
Blood loss (ml)	Up to 750	750-1500	1500-2000	> 2000
% blood volume lost	Up to 15%	15-30%	30-40%	> 40%
Pulse rate (/min)	<100	>100	>120	>14
Blood pressure	Normal	Normal	Decreased	Decreased
Pulse pressure	Normal	Decreased	Decreased	Decreased
Respiratory rate	Normal	Increased	Increased	Rapid
Urine output (ml/hr)	>30	20-30	5-15	Negligible
CNS / mental state	Slight anxiety	Mild anxiety	Anxious, confused	Confused, lethargic
Fluid replacement	Crystalloid	Crystalloid / colloid	Crystalloid / colloid and blood	Crystalloid / colloid and blood

Table 2: Grades of dehydration, relating to the % body weight lost and the resulting physical signs.

	Mild <5%	Moderate 5-10%	Severe >10%
Pulse rate	Normal	Increased	Increased
Blood pressure	Normal	Normal	Decreased
Respiratory rate	Normal	Normal	Rapid
Capillary return	<2 seconds	3-4 seconds	>5 seconds
Urine output	Normal	Decreased	Negligible / absent
Mucous membranes	Moist	Dry	Parched
CNS / mental state	Normal / restless	Drowsy	Lethargic / comatose

colloids and blood products. A useful principle is to "replace what is lost". This requires knowledge of the constituents of both the fluids lost and of intravenous fluids. Tables 3 and 4 contain the relevant information.

Crystalloids

Crystalloids are solutions of crystalline solids in water. In general they contain sodium in similar concentrations as found in the plasma (e.g. normal saline, Hartmann's). They are rapidly and evenly distributed throughout the extracellular space, with only 25-30% remaining in the intravascular compartment. When used to maintain circulating volume they are given in a volume equivalent to three times the estimated blood losses. Crystalloids which contain a lower concentration of sodium than plasma (i.e. 5% glucose or 0.18% saline with 4% glucose) are distributed throughout the total body water after the glucose has been metabolised.

Colloids

Colloids are suspensions of high molecular weight particles, derived from gelatin (gelofusine, haemaccel), protein (albumin solutions) or starch (hetastarch) and are prepared in solutions of saline or glucose. Colloid solutions remain longer intravascularly than crystalloids prior to being metabolised or excreted (plasma half-lives: gelofusine or haemaccel 4 hours, hetastarch 24 hours, albumin solutions 5-10 days). They should be given in a volume equivalent to the estimated blood loss. Potential disadvantages of their use include increased cost, anaphylactic reactions and risk of infection with human albumin.

Blood

Oxygen delivery to the tissues is primarily a function of haemoglobin level, haemoglobin oxygen saturation and cardiac output. Ensuring an adequate haemoglobin level and intravascular volume is therefore vital for

Table 3: Composition of body fluids

Types of fluid	Na (mmol/l)	K (mmol/l)	Protein (g/l)
Serum	135-145	3.5-5.3	70
NG losses and vomit	60-120	5-10	Minimal
Diarrhoea	80	25-40	Minimal
Third space losses	135-145	3.5-5.3	10-20

Table 4: Composition of various intravenous fluids

(Concentrations are given in mmol/l, molecular weights are given in Daltons)

Fluid	mOsm/l	Na	Cl	Lactate	Ca	K	Solute	pH
N saline 0.9%	308	154	154					5
4% glucose 0.18% saline	284	30	30				40g/l glucose	4.5
5% glucose	278						50g/l glucose	4.2
Hartmann's	278	131	111	29	2	5		6
Gelofusine	274	154	120		0.4	0.4	40g/l gelatin MW 30,000	7.4
Haemaccel	301	145	145		6	5	35g/l gelatin MW 35,000	7.3
HAES 6%	308		154	154			60g/l starch MW 130,000	5.5
HAES 10%	308		154	154			100g/l starch MW 200,000	5.5
HAS 4.5%	270-300	100-160	100-160		2	2	45g/l albumin MW 64,000	6.8
HAS 20%	135	50-120	<40			2	200g/l albumin MW 64,000	6.8
Dextran 40	278						100g/l dextran MW 40,000 50g/l glucose	5

oxygen delivery. Transfusion of red cells is indicated when haemoglobin levels fall, or are expected to fall, to around 7.5 g/dl in fit patients. Patients with underlying ischaemic heart disease may need higher levels (>9g/dl). Transfusion of one unit of packed red cells (volume 300ml Hct 60-70%) will raise Hb by 1 to 1.5g/dl.

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FLUID CONTROVERSIES
Crystalloids versus colloids
Controversy still exists about the roles of crystalloids and colloids in perioperative fluid therapy. In 2004 a well designed randomised controlled trial involving nearly 7000 ICU patients was published comparing the effect of fluid resuscitation with albumin or saline on mortality[1]. No significant difference was demonstrated between the albumin and saline groups for the primary outcome

measure of 28 day all cause mortality. The study concluded that albumin can be considered safe in the heterogeneous population of adult ICU patients, but no clear advantage over saline was identified.

Potential advantages of colloids. If membrane permeability is intact, colloids preferentially expand plasma volume rather than interstitial fluid volume which may result in lower fluid requirement and less peripheral and pulmonary oedema.

Potential disadvantages of colloids. These include greater expense, allergic reactions (gelatins), infection risk (HAS), coagulopathy (dextrans and starches), impaired cross matching (dextrans), and reduction in ionised calcium (HAS).

All colloids leave the plasma volume to enter the interstitial space and the long term benefits of colloids are unclear. In disease states associated with increased alveolar capillary permeability (sepsis, ARDS), infusion of colloid may aggravate pulmonary oedema. Similarly capillary leak of infused colloid in head injuries may cause increased cerebral oedema and increased intracranial pressure.

Potential advantages of crystalloids. Crystalloids are inexpensive and non-allergic. They are more effective at replacing depleted ECF and are not associated with transmission of infection, impairment of coagulation or cross matching.

Disadvantages of crystalloids. Crystalloids exert short lived haemodynamic effects in comparison to colloids. When used for massive fluid resuscitation they invariably produce peripheral oedema and occasionally pulmonary oedema.

Most anaesthetists use a mixture of crystalloids and colloids based on the individual patient being managed and the clinical situation.

Normal saline versus Hartmann's solution.
Normal saline contains a higher concentration of chloride than plasma (154 versus 135 - 145mmol/l). As a consequence of this high chloride load, large volumes of normal saline may cause a hyperchloraemic metabolic acidosis. Whilst the dangers of this temporary saline induced metabolic acidosis are probably minor, it causes a base deficit on arterial blood gases, which may be misinterpreted as a metabolic problem. A recent study confirmed that hyperchloraemic metabolic acidosis can be prevented by using balanced solutions containing physiological concentrations of chloride such as Hartmann's solution[2].

PERIOPERATIVE FLUID MANAGEMENT
Fasting Policy
Current preoperative fasting guidelines for elective surgical patients state that elective patients may take clear oral fluids until two hours prior to the anaesthetic and surgery. Oral fluids alone are suitable for many patients undergoing anaesthesia and surgery. The Association of Anaesthetists of Great Britain and Ireland recommends these minimum fasting periods based on the American society of Anaesthesiologists (ASA) guidelines:

- 6 hours for solid food, infant formula, or other milk.

- 4 hours for breast milk

- 2 hours for clear (non particulate) and non-carbonated fluids.

Intravenous fluid
Perioperative fluid therapy is divided into replacement of pre-existing losses, provision of maintenance fluids and replacement of intraoperative and postoperative losses. Many factors alter the amount of fluid required in the perioperative period (Table 5). Wide variations in the impact of these factors exist between individuals. Fluid regimes must therefore be individualised for each patient.

REPLACEMENT OF PRE-EXISTING LOSSES
The fluid deficit to be replaced is the maintenance fluid requirement (multiplied by the hours since last oral intake) added to preoperative external and third space losses.

Table 5: Factors affecting perioperative fluid requirements
- Patient size, weight, body composition
- Preoperative fluid losses, hydration and volume status
- Co-morbid diseases, particularly sepsis, renal, cardiac and hepatic impairment
- Normal maintenance requirements
- Fever
- Temperature of environment
- Anaesthetic technique
- Type of operation
- Duration of operation
- Operative losses
- Neuro-hormonal stress response
- Postoperative losses
- Speed of return to oral intake

Using the information in Tables 3 and 4 choose the most appropriate replacement fluid.

Normal maintenance requirement of fluid and electrolyte can be estimated at 1.5ml/kg/hr and is usually

replaced with normal saline or Hartmann's and 5% glucose. 4% glucose plus 0.18% saline is a satisfactory alternative.

Abnormal insensible losses must be included. Pyrexia increases insensible loss by 20% per degree Celsius rise in body temperature. Note normal insensible loss for an adult in a temperate climate is 1000ml/day. Fluid lost in this way is exhaled water vapour and electrolyte rich sweat.

Abnormal preoperative external losses are often from the gastrointestinal tract. As shown in Table 3 this fluid contains electrolytes and depletes the ECF. It is best replaced with crystalloid of similar composition, either normal saline or Hartmann's. Diarrhoeal losses may contain high concentrations of potassium.

Third space losses describes the loss of fluid from the intravascular compartment secondary to increased capillary permeability found in conditions such as sepsis and trauma. Some of this fluid forms oedema in the surgical field, some is lost into the bowel lumen and some into the peritoneal cavity. These losses contain equivalent electrolyte concentrations but a lower protein concentration compared to extracellular fluid. These losses are best replaced during resuscitation by a combination of normal saline or Hartmann's and colloids.

The fluid volume required to replace pre-existing losses is difficult to estimate as the losses are hard to measure accurately. Third space losses are notoriously difficult to assess and may continue to increase until the patient starts improving. Therefore fluid replacement must be dynamic, based on the patient's response, and not simply based on one set of observations or predictions. Rate and volume of fluid administration must be adjusted to achieve physiological goals that suggest correction of intravascular volume and hydration status (refer to tables 1 and 2).

Blood loss is replaced initially with 3ml of normal saline or Hartmann's per ml of blood lost. Colloid may also be used in a ratio of 1ml colloid per ml blood lost. After the Hb falls to less than 7.5g/dl blood transfusion may be required.

PERIOPERATIVE LOSSES
During the operation, anaesthesia and surgery affect fluid balance. General anaesthesia causes significant vasodilatation and varying degrees of myocardial suppression. Positive pressure ventilation will reduce venous return and cardiac output. Vasodilatation produced by sympathetic blockade following spinal and epidural anaesthesia will also reduce preload and blood pressure.

Surgery has a number of influences on intraoperative and postoperative fluid balance. These include bleeding, third space losses, evaporative losses from exposed surfaces, fluid sequestration in obstructed or adynamic bowel, patient

positioning, and the neuro-hormonal or stress response. The physiological stress response to surgery or trauma causes a rise in the levels of circulating catecholamines, aldosterone, cortisol and antidiuretic hormone (ADH). This catecholamine and steroid release results in water and sodium retention post-operatively. Because relatively more water is retained than sodium there is a risk of post-operative hyponatraemia, particularly if an excess of non-sodium containing fluids are given.

Third space losses caused by alterations in endothelial function leading to fluid extravasation are very variable. Major influences are the extent of the surgery and the presence of any complications such as sepsis.

PLANNING A FLUID REGIME
Minor body surface surgery
Patients undergoing elective minor surgery often do not need supplementary intravenous fluids perioperatively unless there are additional factors suggesting a delay in return to oral intake or poor preoperative hydration. Some anaesthetists start IV fluids even in this group to ensure optimum hydration and as a route for drug administration, but there is no clear benefit.

Intermediate surgery *(eg bilateral inguinal herniae)*
More significant surgery is associated with a longer postoperative recovery and in some patients significant delays in re-establishing oral intake, particularly in the elderly and those with postoperative nausea and vomiting. Fluids may be given only during surgery or continued for a few hours postoperatively.

Major surgery *(including all laparotomies)*
All patients require supplementary IV fluid during and after surgery. The amount required is dependent on the patient's condition, the type of surgery, anaesthesia and the degree of the stress response. Estimate the preoperative losses and perioperative maintenance requirements and then add an amount to replace additional losses in theatre (estimated blood loss, third space, evaporation etc). Construct a regime which should then be adjusted according to physiological targets.

Long, complex bowel surgery will require much more intravenous fluid replacement than a simple hysterectomy. Blood loss may be similar in both operations, but the bowel surgery is likely to be followed by more tissue swelling, oedema and bowel dysfunction (third space). A large wound will result in more evaporative losses (and cooling), and further third space losses. Giving exact figures for specific operations is not possible.

All laparotomies will require replacement of preoperative losses, and then an estimated volume added for intraoperative replacement. Most laparotomies will therefore receive 10 - 20 mls/kg/hour of Hartmann's

during surgery as a baseline, increased for larger operations (20-30mls/kg/hr). This fluid replaces maintenance, third space losses and evaporative losses, but estimated blood loss will need to be replaced in addition. Fluids must be targeted against physiological parameters.

During straightforward surgery in fit patients physical assessment combined with measurement of blood loss is sufficient to guide fluid therapy (i.e. pulse rate, arterial blood pressure, respiratory rate, urine output, JVP and capillary return). Measurement of haemoglobin concentration using a HaemoCue is also useful.

Patients with significant underlying disease, or those undergoing operations during which large volume shifts are anticipated, should have supplemental invasive monitoring. Suggested physiological goals to be achieved are given below:

- Normal pulse rate (<100/min)

- Normal blood pressure (within 20% of normal)

- Urine output 0.5-1ml/kg/hr

- CVP 6-12 cm H_2O

- Normal pH, PaO_2, base excess, serum lactate

- Haemoglobin > 7.5 g/dl - fit patients; >9g/dl in patients with ischaemic cardiac disease.

- Where advanced monitoring, such as oesophageal Doppler is used, measurement of flow is possible and fluids may be targeted to maintain cardiac output.

POSTOPERATIVE LOSSES

Postoperative losses should be replaced in a similar way to preoperative losses. Maintenance requirements, abnormal insensible losses, visible external losses (via NG tube, vomiting, lower GI tract, urinary tract and drains), third space loss and concealed blood loss should all be measured or estimated. Because of the risk of hyponatraemia during this phase, Hartmann's solution or a combination of 2 litres of normal saline with 1 litre of glucose per day are appropriate maintenance regimens. As during other times in the perioperative period, fluid administration should be regularly reassessed and adjusted according to physiological goals.

COMMONLY ENCOUNTERED POSTOPERATIVE PROBLEMS

Hypotension

Postoperative hypotension is most commonly due to hypovolaemia but may also be caused by sepsis or cardiac dysfunction. Early treatment is needed to prevent subsequent vital organ dysfunction. Initial steps in management of patients with postoperative hypotension are assessment of the intravascular volume status and fluid replacement where appropriate.

Fluid resuscitation should be titrated to clinical end points of arterial blood pressure, heart rate, urine output, skin perfusion, together with indices of tissue perfusion such as blood lactate concentrations. Invasive monitoring may be required.

Once fluid resuscitation is adequate, if hypotension is still an issue, inotropic or vasopressor therapy may be required. If sepsis is suspected noradrenaline (0.02 - 0.8 microgram/kg/min) is the first line agent for increasing blood pressure in a patient with clinical signs of shock and hypotension not responsive to aggressive fluid challenge. Dopamine (2 - 10microgram/kg/min) and adrenaline (0.02 - 0.8 microgram/kg/min) are alternatives. Adverse effects of gut hypoperfusion and lactic acidosis are common with adrenaline. Prior to commencing vasopressors, patients must be fully volume resuscitated with the CVP within the normal range. For patients with cardiac failure dobutamine (2 - 15microgram/kg/min) is an alternative, but if used during anaesthesia often results in vasodilatation and hypotension.

Low urine output

Postoperative urine output should be at least 0.5ml/kg/hr. Oliguria and renal impairment may be caused by pre-renal, renal or post-renal problems.

Pre-renal and post-renal problems predominate in the postoperative period. Common pre-renal causes include hypovolaemia, cardiac dysfunction and septic shock. Post-renal causes of urethral prostatic and bladder neck obstruction are all relieved by urethral catheterisation. Renal failure may be caused by renal disease such as acute tubular necrosis, glomerular or interstitial nephritis, and diabetic nephropathy. It is also caused by toxicity from myoglobin, radio contrast media and certain drugs (NSAIDS, ACE inhibitors and aminoglycoside antibiotics).

Management of low postoperative urine output is aimed at treating or removing any underlying cause of renal impairment and ensuring adequacy of intravascular volume and renal perfusion via restoration of adequate blood pressure and volume. Complications of renal impairment include fluid retention, causing peripheral and pulmonary oedema, hyperkalaemia and metabolic acidosis. These may be severe enough to warrant renal replacement therapy.

Hyponatraemia

Extracellular water and electrolytes are under hypothalamic control. Osmoreceptors in the anterior hypothalamus maintain plasma osmolality between 280 - 295mmol/kg by secretion of ADH. ADH or vasopressin reduce renal diuresis and cause retention of water. Hyponatraemia is a serum sodium of less than 135mmol/l and confusion

followed by convulsions and coma occurs in severe cases (<115mmol/l).

Preoperative low serum sodium may be the result of sodium loss or water excess. Hyponatraemia with decreased extracellular volume is caused by sodium and water loss through the kidneys (i.e. during the diuretic phase of renal failure, diuretic excess, osmotic diuresis secondary to hyperglycaemia and Addisons disease.). Other routes causing water and sodium loss include diarrhoea, vomit, small bowel obstruction and burns.

Postoperatively hyponatraemia is mostly associated with water overload due to an over reliance on dextrose-containing fluids and ADH secretion as part of the stress response to surgery. It may also be a feature of nephrotic syndrome, cardiac failure and hepatic failure. The syndrome of inappropriate ADH secretion (SIADH) is diagnosed by finding a concentrated urine (sodium > 20mmol/l) in the presence of hyponatraemia (serum sodium < 125mmol/l), or low plasma osmolality (<260mmol/l) and the absence of hypovolaemia.

Treatment of hyponatraemia is of the underlying cause. If a patient is not dehydrated and renal function in normal, then sodium of more than 125mmol/l rarely requires treatment. If the serum sodium is less than 125mmol/l and the patient is not dehydrated, then water restriction of 0.5 - 1 litre per day is indicated. If the patient is dehydrated, then normal saline is normally used. Gradual increase in serum sodium helps to avoid heart failure and neurological complications (central pontine myelinosis).

Hyperkalaemia

Hyperkalaemia (K+ > 5.3mmol/l) occurs in severe dehydration, renal failure, diabetic ketoacidosis, excess potassium therapy, transfusion of large volumes old blood, severe tissue damage (rhabdomyolysis, burns) and malignant hyperthermia. K+ > 6.5 mmol/l may cause fatal cardiac arrhythmias and therefore needs urgent treatment. Temporary treatment measures include:

- Intravenous fluids if the patient is dehydrated.

- 10mls 10% calcium gluconate to improve cardiac rhythm stability.

- 25 units of actrapid insulin IV with 50mls of 50% dextrose to increase the transport of potassium into cells - monitor blood sugar regularly)

- Sodium bicarbonate 50mls of 8.4% to correct any acidosis and shift potassium into cells.

- 5mg nebulised salbutamol

- Sulphonate resins 15g orally 6 hourly or 30g rectally to aid excretion of potassium by intestinal route.

These measures provide a temporary reduction in K+. Management priority is to try to treat the underlying cause and ensure optimal hydration. A minority of patients require urgent renal replacement therapy in the form of dialysis or haemofiltration.

Hypokalaemia

Hypokalaemia is often due to diuretics or diarrhoea. Other common causes in the post operative period include GI dysfunction and insufficient intake (normal daily requirement is 1mmol/kg). Symptoms are lethargy, weakness, ileus and cardiac arrhythmias. Treatment is with potassium chloride supplementation, preferably given orally if tolerated. Intravenous therapy should be given at a rate no more than 40mmol/hr with a concentration no more than 40mmol/l, unless given via a CVP line with ECG monitoring in place.

CASE HISTORIES

The three case histories given below illustrate some of the principles and variations in perioperative fluid requirements. For each case a brief description is given including relevant background information.

Fluid management for an elective laparoscopic cholecystectomy

- Fit 43 year old female, weighing 80kg.

- Clear oral fluids until 2 hours preoperatively.

- Total blood loss approximately 30mls.

- Operation duration 1 hour.

Fluid regime

During surgery there was no significant blood loss and tissue oedema and third space losses are miniml. Therefore fluid replacement only needs to cover pre-operative deficit and maintenance requirements. These are approximately:

- Normal hourly maintenance = 1.5ml/kg/hr = 120 mls/hr

- Pre-operative deficit =2 hours x 120 mls = 240 mls

- Maintenance fluid during operation = 1 hour = 120mls

- Allowance for third space loss / evaporation 5mls/kg = 400mls

- Total: maintenance + replacement = 760mls Hartmann's

- Ongoing postoperative maintenance fluids (until tolerating oral fluids) = 120mls/hour.

In most operations of this type fluid balance is not exact and most anaesthetists would give a litre of Hartmann's (or saline 0.9%) during surgery followed by a further litre of Hartmann's and then glucose 5%, both at 120mls/hour. However the method of calculating a suitable fluid regime is demonstrated.

Fluid management for an elective bowel resection
- Fit 70 year old man, weight 80kgs
- Preoperative Hb 12g/dl
- Has been nauseated overnight and had no oral intake for 8 hours.
- Received enema to prepare bowel resulting in diarrhoea overnight.
- Total blood loss: 1000mls
- Operation duration 3hours.

Fluid regime

Preoperative deficits and maintenance fluids need replacing, but in addition there has been significant blood loss and third space losses. As only one third of infused crytalloids remain in the intravascular compartment, crystalloid replacement volume should be 3 times the blood volume lost. This patient would have a blood volume of around (70mls X 80kg = 5600mls). Measured blood lost is around 20% of the blood volume and so the postoperative Hb will be around 9 - 9.5g/dl which is acceptable.

- Normal maintenance rate = 80kg x 1.5ml/hr = 120ml/hr
- Pre-operative deficit = 8 hours x 120mls = 960mls
- Maintenance fluid during operation = 120mls x 3 hours = 360mls
- Other losses include diarrhoea of an unknown amount - estimate 1000mls.
- Replacement of 1000mls blood loss
  - can be with 1000mls of colloid or 3000mls of Hartmann's.
- Third space losses estimated = 10ml/kg/hr for first hour then reducing to 5mls/kg/hr thereafter = 1600mls
- Total - approximately 6000mls over 3 hours

As can be seen this patient requires a substantial volume of IV fluid and it is difficult to replace volumes accurately. Following trends in clinical signs as described above will assist fluid therapy. Inadequate volume replacement results in hypotension, poor tissue perfusion and cellular dysfunction. Although it is important not to fluid overload patients, this is rarely a problem perioperatively. In unfit patients undergoing this degree of surgery, CVP monitoring is useful.

Postoperatively the patient will require higher that normal fluid volumes as fluid will tend to gather in the bowel which will not function, and oedema in the area of surgery will sequester fluid. Fluid replacement should be primarily with Hartmann's or saline 0.9% during the first 24 hours and K+ should be added on the following day. Fluid replacement should be reassessed every 4 hours using the clinical signs described earlier.

Fluid management for an emergency laparotomy
- 65 year old female with occasional angina weighing 70kgs
- Pain for 24 hours, no oral intake during this time
- Pulse 120/min, BP 90/60, poor capillary refill
- Preoperative Hb: 16g/dl, Na 143, K5.4, creatinine 153, Urea 10.4
- Scheduled operation: laparotomy and bowel resection for faecal peritonitis.

This patient is very complex and fluid management will be difficult. Estimating preoperative losses is difficult as she has had no fluid for 24 hours and also will have a lot of intra-abdominal fluid due to her faecal peritonitis. Her Hb and urea are raised with her existing fluid deficit. Fluid replacement will be based on her clinical signs, invasive monitoring and the response to volume loading.

Preoperative intervention:	1 litre colloid and 2 litres crystalloid over 2 hours. Two large IV cannulae CVP line Urinary catheter Arterial line
Intraoperative blood loss: Operation duration 2 hours	700mls

Calculations

This is a complex case and calculations will be very inaccurate. In addition to maintenance fluids and replacement of blood loss, there are uncertain preoperative deficits and significant third space losses relating to the surgery and sepsis (estimate as 10-20 mls/kg/hr)

Titrate fluids to restore HR, BP, capillary refill, CVP and urine output to normal levels. Watch the Hb level as resuscitation continues as it is likely to drop. Postoperatively this lady will require increased fluids, and with a recovering peritonitis will have large fluid shifts due to loss of fluid into the abdomen.

- Preoperative deficit = uncertain. Management as described under preoperative resuscitation, based on physiological response of patient.
- Maintenance rate over the 2 hours of surgery titrated to maintain observations at a normal level.
- Replacement of 700ml intraoperative blood loss = replaced by colloid or crystalloid.
- Replacement of third space losses during the 2 hour operation will depend on the physiological signs intraoperatively.

- Postoperatively the losses will continue and fluid requirements will be high and difficult to assess, but are likely to add 50% to the maintenance requirements for the first 2 days.

SUMMARY

Fluid balance is an important area of perioperative medicine. The primary goals are to maintain an effective circulating volume, preserve oxygen delivery to the tissues and maintain electrolyte homeostasis. Appropriate management requires careful consideration of pre-, per- and post-operative factors on an individual patient basis. Basic knowledge of the physiology relevant to fluid management is required. This includes the constituents and features of commonly used intravenous fluids and an understanding of the neuro-hormonal response and third space fluid loss. Well-informed decisions and management plans made on an individual patient basis reduce patient morbidity and mortality.

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LETTER TO THE EDITOR

Dr Aleksandra Bojarska FCA RCSI, Locum Consultant Anaesthetist

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Sir,

I read with interest an article by Dr Banks and Dr Levy "Retained placenta: anaesthetic considerations". It is an excellent summary of British anaesthetic practice used in case of retained placenta. I use the same practice and teach it to trainees while working in the UK.

However, Update in Anaesthesia is designed for much broader audience and targets especially anaesthesia providers in developing countries. It is a pity that this excellent article only marginally included the practice widely used in many hospitals.

The most commonly used technique for removal of retained products of conception, and manual removal of placenta, in a rural hospital in Africa is ketamine anaesthesia. This is usually provided by intravenous ketamine 50 - 100mg, atropine 0.5mg and diazepam 5 - 10mg, with the patient breathing supplementary oxygen by facemask. Ketamine was only briefly mentioned in the article under the heading of sedation. However, the method above is not sedation, but anaesthesia.

Ketamine has an excellent safety record even if used in obstetric patients who theoretically never have "an empty stomach". The other alternative technique, which could be considered is spinal anaesthesia. General anaesthesia would be reserved only for rare cases where the uterine relaxation is absolutely necessary.

The risks of general anaesthesia in the rural African hospital are significant. It is frequently provided by the sole nurse anaesthetist who might not have anybody around to assist and provide cricoid pressure. For a number of reasons, failed intubation is relatively common. An anaesthetist usually manually ventilates the patients which does not allow him to have "hands free" for example in case of need for blood transfusion.

I believe, the risks of general anaesthesia in this environment far outweigh the risks of ketamine anaesthesia and it should not be recommended as the first line anaesthetic method.

While working in various "anaesthetic environments" we realise that there are no ultimate anaesthetic techniques which are always right, and that the practice has to be applied to the local circumstances. However, the principle of safety remains and our duty is to provide the safest technique available.

Dr Aleksandra Bojarska FCA RCSI
Locum Consultant Anaesthetist
alexbojar@hotmail.com

CHANGES IN MATERNAL PHYSIOLOGY DURING PREGNANCY

Dr Bernhard H Heidemann, FRCA, Consultant Anaesthetist, The Royal Infirmary, Edinburgh
e-mail: b.heidemann@ed.ac.uk

Medical Terms

Gestation	Period between conception and birth
Pregesterone/Oestrogen	Female hormones
Erythropoietin	Hormone secreted to increase the numbers of red blood cells
Trimester	A period of 3 months - during pregnancy there are three trimesters
Gravid	Pregnant - "Gravid uterus" means pregnant uterus
FEV^1	Forced Expiratory Volume in 1 minute
FRC	Functional Residual Capacity - *see Update in Anaesthesia (2000) 12;42*
Parturient	A patient who is about to or is giving birth
Palmar erythema	Reddening of the palm of the hand

Maternal physiology undergoes many changes during pregnancy. These changes, which are largely secondary to the effects of progesterone and oestrogen, begin as early as 4 weeks gestation and are progressive. In the first 12 weeks of pregnancy progesterone and oestrogen are produced predominately by the ovary and thereafter by the placenta. These changes both enable the fetus and placenta to grow and prepare the mother and baby for childbirth.

Haematological

Red cell mass, white cell count and platelet production are all increased during pregnancy. The rising white cell count during pregnancy, which peaks after delivery, can make diagnosis of infection difficult. Platelet production is increased, but platelet consumption increases more, causing the platelet count to fall to low normal values. Renal erythropoietin production increases leading to a 20% increase in red cell mass.

Increased concentrations of progesterone and oestrogen directly act on the kidney causing the release of renin. This activates the aldosterone-renin-angiotensin mechanism leading to renal sodium retention and an increase in total body water. Plasma volume increases by 45% and as this increase is relatively greater than the increase in red cell mass, maternal haemoglobin concentrations falls from 150 g per litre pre-pregnancy to 120 g per litre during the 3rd trimester (Figue 1). *This is termed physiological anaemia of pregnancy.*

The increased circulating volume offers protection for mother and fetus from the effects of haemorrhage at delivery. Knowledge of this is important for the anaesthetist as it can delay the onset of classic signs and symptoms of hypovolaemia. It is very easy to be misled into thinking that, even in the presence of considerable volume loss, it does not need replacement. This is wrong, it being essential to replace the measured loss, and to be aware that more volume may have been lost than the blood pressure and pulse might indicate. By two weeks post partum the haematological changes have mostly reverted to pre-pregnancy status.

Cardiovascular

Oestrogen and progesterone mediated relaxation of vascular smooth muscle in pregnancy cause vasodilatation reducing the peripheral vascular resistance by 20%. Consequently systolic and diastolic blood pressures fall. A reflex increase in heart rate by 25% together with a 25% increase in stroke volume, results in a 50% increase in cardiac output. During labour cardiac output may increase further by up to 45%. Cardiac contractility remains unchanged. The rise in cardiac output is facilitated by anatomical changes, namely left ventricular hypertrophy and dilatation.

In the supine position the gravid uterus can compress the inferior vena cava. This will reduce venous return to the heart resulting in a decrease of cardiac output, maternal blood pressure and placental perfusion. The descending aorta can also be compressed by the uterus causing a reduction in uterine blood flow. Aortocaval compression must be considered as a cause of maternal hypotension from the end of the 1st trimester onwards, though it typically occurs after 20 weeks gestation.

Figure 1: Respiratory changes
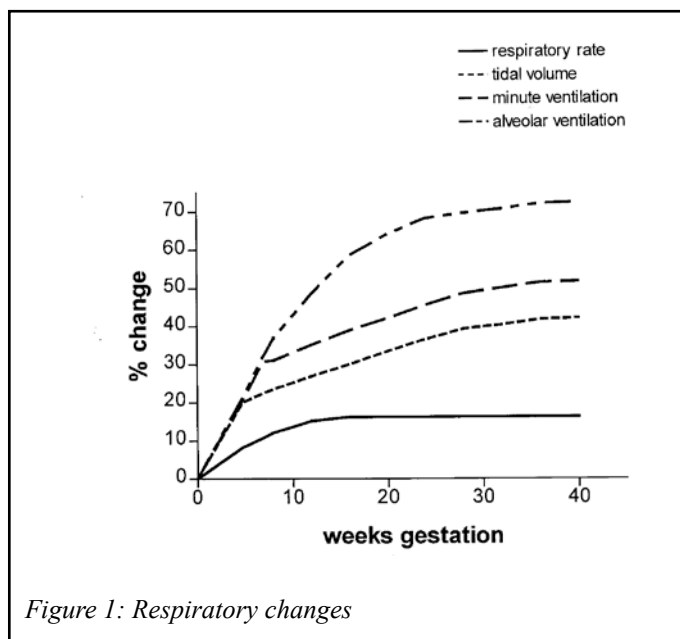
The maternal compensatory mechanism for aortocaval compression comprises of an increase in sympathetic tone causing vasoconstriction and tachycardia and diversion of blood flow from the lower limbs through the vertebral plexus and the azygos veins to reach the right heart. In 10% of parturients this is inadequate to maintain blood pressure in the supine position and hypotension may be severe enough for the mother to lose consciousness. Obstetricians and anaesthetists should be aware that fetal hypoxia due to aortocaval compression may occur in the asymptomatic mother. Intravenous and inhalational anaesthetic agents, causing a reduction in stroke volume and cardiac output, and neuroaxial blockade (spinal/epidural), causing sympathetic blockade, increase the risk of supine hypotension. Whenever possible pregnant patients should adopt a full lateral position. When supine position is required they should be tilted to the left or have a wedge inserted under their right hip.

Coagulation
Plasma levels of fibrinogen and all clotting factors, except XI and XIII, gradually increase during pregnancy inducing a hypercoagulable state. An increase in fibrinolysis is reflected in increased concentrations of antithrombin III, plasminogen, and fibrin degradation products. Platelet activity and consumption are both increased but platelet function remains normal in pregnancy. None of these changes are reflected in a routine clotting screen, which will show values around normal. Platelet function, as assessed by thromboelastography, remains normal while the platelet count is greater than 100×10^9 per litre. A platelet count of greater than 80×10^9 per litre is regarded as safe for the use of neuroaxial blockade by many. Thromboembolic complications remain a common source of morbidity and mortality associated with pregnancy.

Respiratory System
Changes in the respiratory system may be categorised as anatomical and physiological. The anatomical changes make upper airway obstruction and bleeding more likely during mask anaesthesia and may make tracheal intubation more difficult. There is approximately a 7-fold increase in failed intubations in parturients at term. The anatomical changes include capillary engorgement and oedema of the upper airway, pharynx, false cords, glottis and arytenoids. There is also an increase in chest diameter, to allow increased minute ventilation, and an enlargement of the breasts, which can make laryngoscopy with a standard Macintosh blade more difficult.

The gravid uterus progressively displaces the diaphragm cranially reducing diaphragmatic movement in late pregnancy, particularly in the supine position. Inspiratory reserve volume is increased but vital capacity, total lung volume and FEV1 remain unchanged. A decrease in both residual and expiratory reserve volumes causes a 20% reduction in functional residual capacity, which in turn causes airway closure in 50% of parturients at term in the supine position. Thus, pre-oxygenation is less effective in the term parturient and desaturation is likely to occur much faster than in the non-pregnant patient. A pre-oxygenation period of 3 - 5min is the standard recommendation. Some of the changes to respiratory physiology are illustrated in Figure 2.

Bronchial and tracheal smooth muscle relaxation are a result of increased progesterone concentrations. This often causes the symptoms of asthma to lessen in pregnancy. $PaCO_2$ falls and then levels off at 4.1kPa (31mmHg) by the end of the first trimester. This is caused by a 10% increase in the respiratory rate, secondary to progesterone mediated hypersensitivity to CO_2, and an increase in alveolar and minute ventilation, secondary to increased respiratory rate and tidal volume. PaO_2 rises to 14 kPa (105mmHg) during the 3rd trimester but then falls to less than 13.5 kPa (101mmHg) at term because increased oxygen consumption is no longer fully compensated for by the rise in cardiac output. Thus, the alveolar arterial oxygen gradient increases. In some parturients this may be worsened by aortocaval compression and closure of dependant airways. At term (40 weeks gestation), oxygen consumption and carbon dioxide production are increased by 60% above non-pregnant values.

Renal
As a result of the changes in the cardiovascular system, renal plasma flow and glomerular filtration rate increase in pregnancy. This results in an increase in urea, creatinine and urate clearance and excretion of bicarbonate causing plasma concentrations to be less than in the non-pregnant population. The activities of renin-angiotensin, aldosterone

Figure 2: Haematological changes
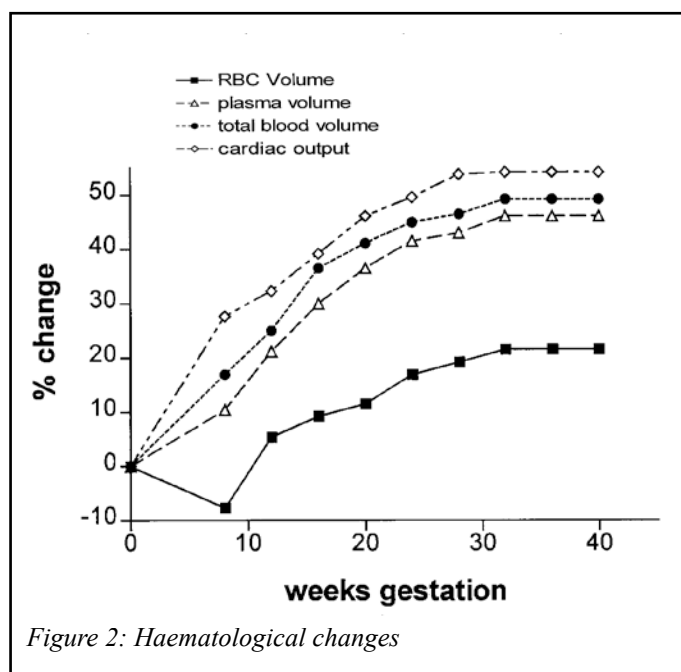
and progesterone are increased leading to increased water retention and a decreased plasma osmolality. Glycosuria can be observed in 40% of parturients secondary to reduced reabsorption of glucose. Urinary tract infections are more common in pregnant patients due to urinary stasis from progesterone mediated ureteric smooth muscle relaxation.

The changes in renal physiology increase the volume of distribution for drugs and those that are renally excreted may have to be given in higher than normal dosages and may have prolonged action.

Acid Base regulation
Increased minute ventilation leads to a decrease in $PaCO_2$ producing a respiratory alkalosis and a left shift of the oxyhaemoglobin dissociation curve. A 30% increase in 2-3 DPG has the opposite effect on the oxyhaemoglobin dissociation curve with an increase of the P50 from 3.5 kPa to 4 kPa (26-30mmHg). The respiratory alkalosis is compensated by increased renal bicarbonate excretion so that plasma hydrogen ion concentrations remain essentially unchanged.

Pain in labour causes maternal hyperventilation associated with an acute left shift of the oxyhaemoglobin dissociation curve. This increases the affinity of maternal haemoglobin for oxygen and consequently oxygen delivery to the fetus decreases. If labour becomes prolonged and is also painful, basic metabolic rate increases and O_2 extraction does as well. Under these circumstance there will be less O_2 available to the fetus, as cardiac output cannot be further increased to match the increased O_2 demand. In this situation regional analgesia is useful as it prevents the increase in BMR and further hyperventilation secondary to the pain. Effective regional analgesia largely abolishes the detrimental effects of a painful labour on the fetus.

Hepatic
Plasma concentrations of γ-GT, ALT, AST, and LDH are high normal or slightly elevated and clinical signs of liver

Table 1: Ventilation in pregnancy and labour			
	pregnancy	**labour**	**Non-pregnant**
respiratory rate [per min]	15	22 - 70	12
tidal volume [ml]	480 - 680	650 - 2000	450
$PaCO_2$ [kPa] (mmHg)	4.1 (31)	2 - 2.7 (15-20)	5.3 (40)
PaO_2 [kPa] (mmHg)	14 (105)	13.5 - 14.4 (101-108)	13.3 (100)

Table 2: Renal function		
plasma level	**non-pregnant**	**pregnant**
creatinine [μmol per litre]	73	50 - 73
urea [mmol per litre]	4.3	2.3 - 4.3
urate [mmol per litre]	0.2 - 0.35	0.15 - 0.35
bicarbonate [mmol per litre]	22 - 26	18 - 26

disease like spider naevi and palmar erythema may occur during normal pregnancy making diagnosis of liver disease during pregnancy more difficult. Plasma concentrations of alkaline phosphatase are increased 3-fold as a result of placental production. Pregnant patients are more likely to develop gall-stones as increased progesterone concentrations cause a decrease in cholecystokinin release and a reduction of the contractile response to cholecystokinin. Succinylcholine may lead to prolonged neuromuscular blockade secondary to a 25% fall in plasma cholinesterase concentrations at term and a further 8% fall three days postpartum (post delivery). This is compounded by an increased volume of distribution at term but not usually clinically significant. Never the less, standard or increased doses of succinylcholine are recommended in pregnancy. Succinylcholine sensitivity in females who are heterozygote for an abnormal cholinesterase gene may be unmasked due to a 25% decrease in hepatic protein synthesis in pregnancy.

Gastro-intestinal System

Increased intra-abdominal pressure by the gravid uterus, displacement of the gastric axis and progesterone mediated reduction in lower oesophageal sphincter tone cause gastro-oesophageal reflux in as many as 80% of term parturients. Whilst pregnancy does not seem to cause increased gastric volumes and delayed gastric emptying, both of these are features of labour. The administraion of opioids for labour analgesia further accentuates this. Pregnant women are therefore at risk of developing Mendelson's syndrome (aspiration pneumonitis) especially on induction of general anaesthesia, which reduces upper oesophageal sphincter pressure. Strategies for the prevention of this may include the administration of H_2 blocking drugs, neutralization of gastric contents with non-particulate antacids, e.g. sodium citrate, and the use of a rapid sequence induction with cricoid pressure, when administering general anaesthesia to pregnant women. At 24 - 48 hours postpartum the changes in the gastro-intestinal system are thought to have reverted to normal.

Endocrine

In the non-diabetic pregnant woman carbohydrate loads will cause a greater than normal increase in plasma glucose levels facilitating placental glucose transfer. This is due to increased insulin resistance caused by placental hormones (mainly human placental lactogen). Insulin production is also increased during pregnancy.

Maternal hyperglycaemia in diabetic pregnant women induces an increase in fetal insulin production, as insulin does not cross the placenta. Neonatal hypoglycaemia may follow as the carbohydrate load falls immediately after birth. Since insulin also acts as a growth hormone maternal diabetes is associated with fetal macrosomia.

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KETAMINE: A REVIEW

Dr Carl Stevenson, Medecins Sans Frontieres Anaesthetist

Ketamine was first synthesized in 1962 at the Parke Davis Lab by Calvin Stevens. The original name for Ketamine was 'CI581'. 1965 saw the first accounts of the recreational use of ketamine - Professor Edward Domino described it as a potent psychedelic drug and coined the term 'dissociative anaesthetic'. In 1966 ketamine was patented by Parke-Davis for use as an anaesthetic in humans and animals. Ketamine was then used as a field anaesthetic by the U.S.A. during the Vietnam War.

Ketamine provides dissociative anaesthesia - a combination of profound analgesia with superficial sleep. This state is characterised by spontaneous ventilation, relative preservation of airway reflexes and haemodynamic stability, and explains why ketmaine has remained the anaesthetic drug of choice in the developing world and for mass casualties in the field.

Ketamine, a phencyclidine derivative, is soluble in water and is prepared with the sodium salt benzethonium chloride as a preservative. It is a basic compound and is dissolved in a solution of pH 3.5-5. The ketamine molecule contains an asymmetrical carbon atom with two optical isomers (enantiomers). The S(+) isomer is about three times more potent and longer acting as an anaesthetic than the R(-) isomer; the latter is thought to be the cause of some of the undesirable side effects.

Central Nervous System

Ketamine's site of action appears to be primarily in the thalamus and limbic systems, acting as an N-methyl-D-aspartate (NMDA) receptor non-competitive antagonist. It does not suppress respiratory drive unless high doses are used, or smaller doses given rapidly. The eyes often remain open with a slow nystagmic gaze along with preservation of the corneal and light reflexes. Glucose uptake in the auditory and somatosensory systems is reduced, suggesting selective deprivation of these senses (the thalamic and limbic regions show increased uptake. Ketamine produces high-amplitude slowing of the EEG, and case reports of successful treatment of status eplilepticus exist.

Historically, anaesthetists have regarded ketamine as contraindicated in patients with brain injury as the drug may increase intracranial pressure and alter haemodynamics. The previously accepted explanation was that the rise in mean arterial pressure caused a rise in cerebral perfusion pressure and therefore intracranial pressure. Recent evidence suggests that a rise in intracranial pressure may not always occur. Experimental data have shown ketamine to decrease cerebral infarct volume and improve outcome in experimentally head injured rats. Antagonism of the effects of extracellular neurotoxic glutamate may be a mechanism of action. An alternative explanation is that the NMDA antagonists simply induce cerebral vasodilatation, so improving perfusion of the watershed areas adjacent to the injury. One study[1] has compared sedation with midazolam/sufentanil and midazolam/ketamine in brain-injured patients and found both combinations effective. Importantly, no significant differences were observed between the two groups in the mean daily values of

Pharmacokinetics of ketamine	
Absorption	Well absorbed orally, nasally, rectally and intramuscularly
	Oral bioavailability 20%
Distribution	20-50% protein bound in plasma
	Volume of distribution 3L/Kg
	Distribution half life is 11 mins
	Recovery primarily due to redistribution from brain to periphery
Metabolism	N-demethylation & hydroxylation of the cyclohexylamine ring in the liver
	Some metabolites are pharmacologically active
Excretion	Urinary excretion of conjugated metabolites
	Clearance 17ml/kg/min
	Elimination half life 2.5 hours

intracranial pressure and cerebral perfusion pressure, and the numbers of intracranial pressure elevations were similar in both groups. When ketamine was administered to adults with traumatic brain or spinal cord injury, systemic haemodynamics were unaltered but the effects on intracranial pressure were not reported.[2]

Cardiovascular System

Blood pressure and heart rate are frequently raised after administration of ketamine. The drug causes direct depression of the myocardium and vasodilatation on direct exposure to smooth muscle. Despite this, due to central nervous system stimulation, haemodynamic stability is maintained. A rise in noradrenaline levels is detectable in the blood after ketamine administration, and this pressor response can be blocked by α- and β-adrenoceptor antagonists and sympathetic ganglion blockade. Ketamine also inhibits catecholamine uptake at sympathetic nerve terminals. Pulmonary vascular resistance can rise and an increase in pulmonary shunting can occur in patients with cardiac septal defects.

Respiratory System

Apnoea is unusual unless ketamine is administered rapidly or another respiratory depressant drug (e.g. an opioid) is given. Airway reflexes and skeletal muscle tone are relatively preserved, but salivary and tracheobronchial secretions are increased. Aspiration is still a potential hazard despite the retention of protective reflexes. Ketamine has a bronchodilator action that may be mediated either via an increase in blood catecholamines or by its direct smooth muscle relaxant effect. A valuable property for patients with asthma.

System Effects	
CVS	↑ heart rate, ↑ blood pressure ↑ CVP, ↑ CO, baroreceptor function is maintained and dysrhythmias are uncommon
RS	Bronchodilation, ↑ RR, relative preservation of airway reflexes
CNS	↑ Cerebral blood flow/metabolic rate and intraocular pressure
AS	Nausea and vomiting, ↑ salivation
GU	↑ uterine tone
Other	Emergence delirium/dreams/ hallucinations

Stereoisomers of ketamine

The S(+) isomer is 3-4 times more potent as an analgesic with a faster clearance and less side effects than the R(-). Thus, S+ ketamine has been studied more recently.

Preservative free S(+) ketamine with a low dose of bupivicaine intrathecally has been shown to provide a more rapid motor and sensory block, shorter duration of action and less motor blockade in elderly males.

The addition of preservative free caudal S(+) ketamine to bupivicaine prolongs the duration of postoperative analgesia. IV S(+) ketamine combined with a plain bupivicaine caudal provides no better analgesia than caudal bupivicaine alone, suggesting that the main analgesic effect of caudal S(+) ketamine is due to a neuroaxial rather than a systemic effect.

In another study[5] the duration of analgesia was significantly longer in a ketamine/ropivicaine caudal anaesthesia group than in a plain ropivicaine group. The subjects in the ropivicaine alone group needed significantly more doses of postoperative analgesia. There were no significant differences between the groups in the incidence of postoperative nausea, vomiting, sedation, emergence delirium, nightmares, motor block or urinary retention.

In addition S(+) ketamine 1.0mg/kg for caudal block in children can produce surgical and postoperative analgesia equivalent to that of bupivacaine.6

Ketamine and Obstetric Anaesthesia

Ketamine supports maternal blood pressure which may be important in the face of significant maternal hypotension. There is a dose dependent rise in maternal blood pressure that makes ketamine less suitable for use in pre-eclampsia. In elective caesarean section, ketamine 1mg/kg does not increase maternal blood pressure at induction or intubation any higher than with thiopentone 4mg/kg.

Ketamine very rapidly passes through the placenta, and ketamine levels in cord blood exceed the levels in the maternal venous blood as early as 1 min 37s after the injection. The ketamine levels in cord blood reach a maximum in the period 1 min 37s to 2 min 5s after the injection.

Apgar scores are similar in neonates delivered abdominally after induction of anaesthesia with ketamine 1mg/kg or thiopentone 3mg/kg. Induction with ketamine 2mg/kg is associated with neonatal depression and increased uterine tone.

Ketamine 10 - 20mg iv has been used to provide analgesia for labour and can be repeated every 2-5 minutes but should not exceed 1mg/kg in 30 minutes and the total dose should not exceed 100mg.

With regards to the teratogenic effects of ketamine in early pregnancy, no specific reproductive studies have been performed in humans, but in rats 120mg/kg im failed to produce teratogenic effects. In contrast, large doses produced neural tube defects in chick embryos.

In the U.S.A. ketamine has been given the FDA fetal risk category C - i.e. should only be given if the benefit outweighs the risk.

Ketamine as an additional analgesic
Ketamine has been studied in combination with other analgesics with varying results. When ketamine, 0.25mg/kg was given to patients whose pain was poorly controlled with i.v. morphine, pain scores were improved.[4] The ketamine treated patients also experienced reduced nausea and vomiting than those receiving placebo. In contrast, when patients undergoing anterior cruciate ligament repair received S(+) ketamine as well as opioids, no benefit was seen.[3] Ketamine may also reduce acute tolerance to morphine.

Ketamine as a premedicant
Ketamine has been used in the premedication of children using different routes and doses. Oral ketamine 8mg/kg has been shown to be an effective premedicant for in-patient children although recovery from anaesthesia is longer. Lower doses of ketamine have been studied when combined with benzodiazepines. For example oral ketamine 3mg/kg combined with oral midazolam 0.5mg/kg as premedication did not significantly alter recovery times after sevoflurane anaesthesia compared to midazolam alone.

The use of intranasal ketamine is gaining popularity as a premedicant and as an analgesic in the emergency room. In children, 3mg/kg, diluted to 2ml with saline (1ml per nostril) can allow pleasant and rapid separation from parents, cooperative with monitoring and of mask inhalation induction and also has not been shown to cause prolonged recovery or delayed discharge home. Plasma concentrations of norketamine peak about 2 hours after the administration of nasal ketamine (slower than rectal ketamine). Nasal doses of 3mg/kg produce a large enough plasma concentration to produce analgesia and sedation but not anaesthesia.

Premedication for ketamine anaesthesia
Atropine or glycopyrrolate are often given intravenously before the induction of ketamine anaesthesia to reduce secretions. Glycopyrrolate is the better choice due to its lower psychotropic and chronotropic effects (it is a quaternary ammonium compound which does not cross the blood brain barrier). The increase in heart rate at intubation has been shown to be significantly higher following atropine than following glycopyrrolate.

Oral clonidine premedication (5mcg/kg) has been shown to reduce the hypertensive response to ketamine.

Other areas of interest
- Some studies suggest that ketamine provides better intubating conditions than thiopentone after administration of a neuromuscular blocker

- A ketamine/midazolam combination provided better surgical conditions (collapsed intestinal loops) and better recovery during prolonged abdominal surgery when compared to halothane/nitrous oxide/oxygen anaesthesia.

- Prophylactic low-dose ketamine (0.5mg/kg i.v. 20 mins prior to the end of surgery) was found to be effective in preventing postoperative shivering.

- Ketamine reduces the need for inotropic support in septic patients. In animal models of endotoxic shock, ketamine reduces pulmonary damage by enhancing haemodynamic stability and reducing pulmonary hypertension and extravasation.

In some difficult situations non-anaesthetists have been trained to use ketamine. In Nepal, 679 cases of ketamine anaesthesia for simple ophthalmic procedures were successfully carried out by paediatricians with experience in paediatric resuscitation. Also the use of ketamine anesthesia has been described at high altitude, by primary-care physicians without a specialist training in anesthesia. At a low dose of 2.0 mg/kg, ketamine produced a dissociative anaesthesia whilst not depressing the hypoxic drive, or interfering with the pharyngeal or laryngeal reflexes. Supplemental oxygen was useful in the recovery phase for the less acclimatized individuals. However it is important to remember that ketamine is an anaesthetic drug, and as such should not be seen as simply a sedative agent.

The laryngeal mask airway is not easy with ketamine anaesthesia since ketamine maintains some degree of laryngeal and pharyngeal tone.

Using IV ketamine
- Premedicate with an antisialogogue (e.g. atropine 10-20mcg/kg)

- Anaesthesia: give 1-2mg/kg in small increments initially to avoid episodes of apnoea. For example 30mg boluses every 60 secs to a total of 100mg in a 70kg man. Onset is rapid (1-2 mins) with a duration of 10 minutes. Anaesthesia can be maintained by repeated boluses 0.5mg/kg every 15-20 minutes or by continuous infusion 2-4 mg/kg/hr.

- (Add 500mg of ketamine to 500ml of a crystalloid solution. Spontaneous ventilation = 1 drop/kg/min (4mg/kg/hr); Controlled ventilation = 0.5 drop/kg min (2mg/kg/hr). The infusion is stopped roughly 30 minutes prior to the end of surgery.[7]

● Diazepam 0.1-0.2mg/kg helps to reduce intraoperative movement and also limits postoperative delirium.

● Analgesia - 0.5mg/kg produces rapid and profound analgesia

Using IM ketamine

● Ideal for children and for painful repeated procedures. Atropine can be mixed with ketamine for a single injection.

● Anaesthesia: 6-8mg/kg. Onset is gradual over 5-10 minutes and is preceded by intense analgesia. IM titration of maintenance doses is difficult but 5 mg/kg every 30min is usually adequate. An easier technique is to prolong anaesthesia using IV supplements.

● Analgesia - 2-4mg/kg. Onset is again 5-10 minutes

Monitoring the patient during anaesthesia

● Careful monitoring of the airway is vital. Secretions can cause obstruction and although airway reflexes are preserved laryngospasm and aspiration can still occur. It is the author's practice to use a chin lift procedure to improve the airway whilst detecting the pattern of breathing on the palm of the hand.

● In the presence of normal respiratory physiology a supply of oxygen is preferable but not essential.

● Pulse oximetry is a valuable tool if available.

● Due to the cardiovascular effects of ketamine, hypotension is less common than when using other general anaesthetics. In the absence of a sphygmomanometer, palpation of the rate and quality of the pulse is effective.

Judging depth of anaesthesia

● Assessing depth of anaesthesia is difficult when using ketamine as there are few obvious signs. Spontaneous movement and eye opening may occur during adequate anaesthesia but are more common during subanaesthetic doses. It is worth noting that i.v. induction is rapid (30-60 sec slower than thiopentone) but that intense analgesia will be present in subanaesthetic doses.

Patients suitable for ketamine

● Children - nausea, vomiting and hallucinations are less common in children

● Burns (repeated painful procedures), trauma, radiotherapy

● Shocked patients

● Status asthmaticus

Patients unsuitable for ketamine

Avoid ketamine with:

● Hypertension

● Ischaemic heart disease

● Pre-eclampsia

● Raised intracranial pressure

● Open eye procedures

● Acute porphyrias

Difficulties during ketamine anaesthesia

● Excess secretions

● Preserved muscle tone - airway manipulation and surgical access can be difficult

● Assessing depth of anaesthesia

● Spontaneous muscle movement and "grunting"

● Irregular respiratory pattern with episodes of apnoea particularly in children under 12 months and following rapid iv induction

● Prolonged recovery time +/- nausea/vomiting/ hallucinations

Case report - analgesia for a multiply injured patient

An 18 yr old male was brought in by relatives following an attack in the early hours of the morning. There is an obvious compound fracture of the humerus, in addition to multiple lacerations to the abdomen and scalp. The patient
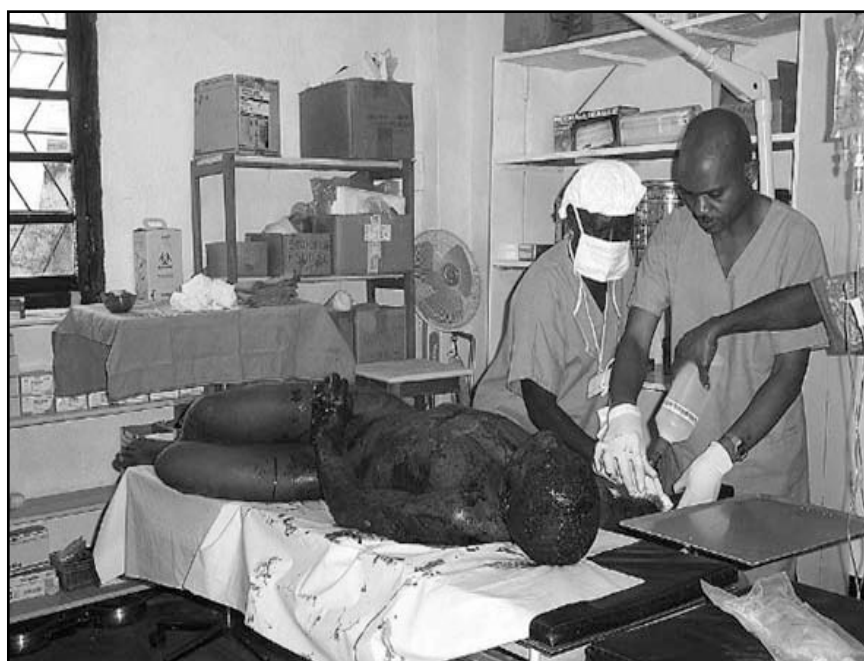

Figure 1.

is severely distressed and in obvious pain. The only anaesthetist available has been delayed at a security road block. The non-medically trained staff have been taught to use ketamine. They administer an analgesic dose of 210mg i.m. mixed with atropine and after 5 -10 minutes are able to splint the fracture, wash out wounds and apply pressure to the scalp lacerations before more experienced staff arrive.

Case report - an obstetric emergency
An emergency caesarean section for placental abruption is scheduled in a remote district hospital. The patient has had significant blood loss and has a weak thready pulse with a rate of 120/min. There is no working oxygen concentrator or laryngoscope available.

Large bore access is obtained and fluid resuscitation is initiated. The patient is placed in a 15° left lateral tilt to avoid aorto-caval compression. An induction dose of ketamine 70mg i.v. is given in 10-20mg aliquots to avoid periods of maternal apnoea plus 600mcg of atropine is given i.v. to reduce oral secretions and therefore aid airway management.

Chin lift and jaw thrust are used to help maintain the airway whilst the exhaled breath on the palm of the hand helps the nurse to monitor the depth, rate and pattern of breathing. Further boluses of ketamine 10-20mg i.v. are given to maintain anaesthesia, although occasional limb movements and eye opening are noted. After clamping of the cord diazepam 5-10mg is administered i.v. to reduce emergence phenomena. Post operatively the patient is placed in the left lateral position in the recovery room.

Case report - anaesthesia for laparotomy
A young man is scheduled for a laparotomy following a bullet wound to the abdomen. Heart rate, blood pressure and oxygen saturation are stable.

Anaesthesia is induced using atropine 600mcg i.v. plus ketamine 100mg i.v. (in 20mg boluses every 60 seconds). An appropriate dose of a neuromuscular blocker is given followed by endotracheal intubation.

Anaesthesia is maintained by a continuous infusion of ketamine:

> 500mg of ketamine is added to 500ml of a crystalloid solution; (controlled ventilation)
> 0.5 drop/kg/min = 2mg/kg/hr[7]

The infusion is stopped and diazepam 10 mg administered roughly 30 minutes prior to the end of surgery. Following reversal of neuromuscular blockade and an awake extubation the patient was nursed on his side in the recovery area.

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6) Marhofer P. S(+) ketamine for caudal block in paediatric anaesthesia. British Journal of Anaesthesia 2000;84(3):341-5

7) Vreede E. Field Anaesthesia - Basic Practice. MSF publication.

Figure 2.

QUESTIONS FROM THE ZAMBIAN ANAESTHESIA REFRESHER COURSE, KITWE 2005
Dr Jo Thorpe, Glasgow

Are husbands allowed in the operating room during Caesarean Section; if yes, what are the advantages?

Husbands are encouraged to come into the operating room during Caesarean section in Britain, as long as the section is under epidural or spinal anaesthesia. Theatre clothes are worn and a screen is erected to hide the view of the wound. Practice varies whether he comes in before insertion of the block or after the block has been confirmed as satisfactory but before surgery commences. If, at any stage, a general anaesthetic becomes necessary, the husband or partner is usually asked to leave. A debate last year at an Obstetric Association of Anaesthetists' Controversies meeting on whether partners should be allowed if Caesarean section were to be under general anaesthesia showed only a minority of anaesthetists in favour, with little change after debate[1].

The perceived advantages are

● Emotional support for the mother. An early audit study[2] of maternal concerns in the theatre environment and satisfaction during this event showed that mothers talked to family more than to staff about their worries. In theatre, husbands / partners scored more highly than all others in the provision of moral support.

● Sharing of a life event. The involvement of the husband / partner in decision making and in the birth process was emphasised in an Audit Commission report[3].

● Paternal bonding with the baby. When appropriate after delivery, the baby is brought over to be held by the parents. One could argue that the mother should have first call, having carried the baby for 9 months!

In the event of a husband or partner not being able or unwilling to come into theatre, another person who is emotionally close to the mother (patient's mother, sister or close friend) may be allowed instead.

Some disadvantages are that there could be pressure on the husband / partner to conform though he may be reluctant for some reason. It could be awkward persuading the husband to leave if difficulties arise, though this should be agreed in advance. In U.K., the anaesthetist is rarely required to look after the baby. In an environment where looking after the baby is the norm, the anaesthetist may have three potential patients to attend as it is not unknown for husbands to feel queasy or to faint.

1) Robinson N and Smiley R. Controversies: Partners should be allowed to stay in the operating theatre during Caesarean section. International Journal of Obstetric Anesthesia 2004; 4: 251-6

2) Kennedy BW, Thorp JM, Fitch W and Millar K. The theatre environment and the awake patient. Journal of Obstetrics and Gynaecology 1992; 12:407-11

3) Audit Commission. First Class Delivery; improving maternity services in England and Wales. Abingdon: Audit Commission Publications; 1997.

Can the Glasgow Coma Score be used in a patient with head injury intoxicated with alcohol?

Yes, the Glasgow Coma Score (GCS) can be used in any patient as an assessment of "conscious level". In a case where alcohol and pathology may co-exist e.g. possible head injury in a patient who has been drinking alcohol, it would be expected that the scores would improve with falling alcohol levels and that non-improvement would suggest underlying pathology.

There are several important points to note about the Glasgow Coma Scale:-

● The worst possible score is 3 not zero.

● The best response is noted.

● GCS 8/15 provides less information than the three components of the Scale E2 V3 M3.

● If impossible to assess the best response, e.g. the patient is sedated and / or intubated, this is documented - not scored as "1" for no response.

1) Teasdale GM, Jennett B. Assessment of coma and impaired consciousness. Lancet 1974;2:81-4.

2) Teasdale GM, Murray L. Revisiting the Glasgow Coma Scale and Coma Score. Intensive Care Medicine 2000;26:153-4.

Is there a different Glasgow Coma Score for Paediatric Patients?

The assessment tool below is the one used at the Institute of Neurological Sciences in Glasgow, the home of the Glasgow Coma Scale. A modification of the adult scale was suggested by Australian workers[1,2] to take account of normal developmental milestones and after discussion with Glasgow.

The age column indicates the best that can be expected for that age range, e.g. 12 months to 2 years

- Verbal scale - recognizable words are expected - so best is 4.

- Motor scale - the infant will usually locate pain but not obeys commands - best is 4.

"Orientation" for children is defined as awareness of being in hospital

	Age		Coma scale
Eyes open		Spontaneously	4
		To speech	3
		To pain	2
		None	1
Best verbal response	> 5yrs	Orientated to place	5
	1-2yrs	Words	4
	6-12 mths	Vocal sounds	3
	6 mths	Cries	2
		None	1
Best motor response	>2 years	Obeys commands	5
	6mths- 2 yr	Localises pain	4
	6mths	Flexion to pain	3
		Extension to pain	2
		None	1

Normal aggregate score for children	
0-6 months:	9
> 6 - 12 months:	11
> 1 - 2 years :	12
>2 - 5 years:	13
> 5 years:	14

1. Simpson D, Reilly P. Paediatric Coma Scale. Lancet 1982; 2:450.

2. Reilly P, Simpson D and al. Assessing the conscious level in infants and young children: a paediatric version of the Glasgow Coma Scale. Child's Nerv Syst 1988; 4:30-3.

THE LARYNGEAL MASK AIRWAY

Tim Cook BA MBBS FRCA , Ben Walton MB ChB MRCP FRCA, Royal United Hospital, Bath, UK
E mail: timcook@ukgateway.net

Few advances in anaesthesia have had such a dramatic impact as the introduction of the classic Laryngeal Mask Airway (cLMA) to anaesthesia in 1988. This was the first really effective alternative to facemask or tracheal intubation for airway maintenance during anaesthesia. The cLMA rapidly became the standard alternative to tracheal intubation and is the first of the modern supraglottic airway devices (SADs) [also referred to by some as extraglottic airway devices: EADs]. Many anaesthetists, though not all, initially restricted the LMA to anaesthesia for minor extremity surgery with spontaneous breathing, but is interesting to note that in the Dr Archie Brain first publication on the cLMA reported its use in 21 patients, of whom 16 underwent gynaecological laparoscopy with controlled ventilation. Since then the indications for the LMA have evolved rapidly and there is now a far more liberal attitude to indications for laryngeal mask use. It is estimated that over 200 million anaesthetics have now been administered using a cLMA.

The LMA has continued to develop since 1988 and seven alternative laryngeal masks have been developed and marketed. This practical review summarises the role of the various devices. The 'family' of LMAs now includes the standard or classic LMA (cLMA), the flexible/reinforced LMA (fLMA), the Intubating LMA (ILMA) and the ProSeal LMA (PLMA). Single-use versions of the cLMA, fLMA and ILMA are now available (although largely unevaluated).

In modern anaesthesia the LMA family has a role in routine anaesthesia, planned difficult airway management and in rescue of the obstructed or 'lost' airway.

The Classic Laryngeal Mask Airway (cLMA)

The cLMA was developed by Dr Archie Brain (figure 1) from 1981-1988 and released in 1988. During development over 100 prototypes were tested in more than 6000 patients. Its introduction was revolutionary: despite considerable scepticism at the time of its introduction, within a year every single hospital in the United Kingdom had purchased the cLMA.

The cLMA consists of a transparent silicone tube with a small oval-shaped silicone mask at the distal end. The mask has an anterior cuff, with pilot balloon and its posterior surface is semi-rigid to prevent folding. Across the distal end of the airway tube are two flexible bars that prevent the tongue impeding insertion, and the epiglottis
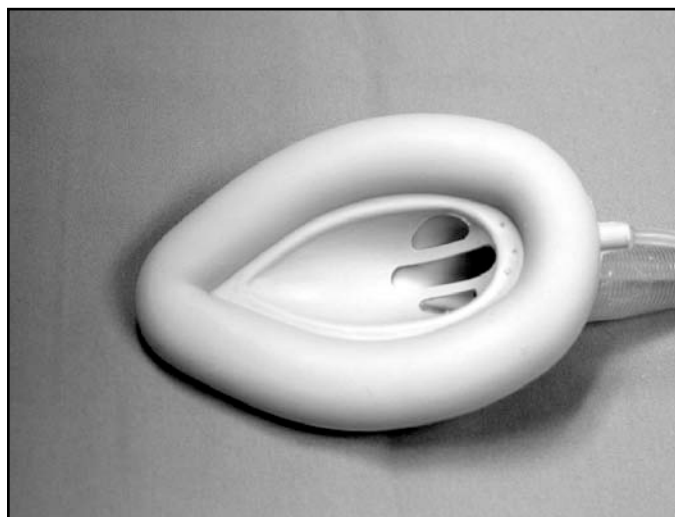

Figure 1a,b and c: Classic LMA

causing obstruction after placement. The lateral cuff lies against the pyriform fossa and the upper cuff lifts the base of the tongue. The mask is held in a stable position by the hypopharyngeal constrictor muscles laterally and cricopharyngeus inferiorly. When correctly placed the LMA lies with the airway orifice facing anteriorly over the glottis, the tip at the origin of the oesophagus and the cuff encircling the laryngeal inlet (figure 2). The cLMA cuff forms a low pressure seal allowing controlled or spontaneous ventilation. Correct positioning relies on getting the tip of the mask to the upper oesophageal sphincter, behind the cricoid cartilage.
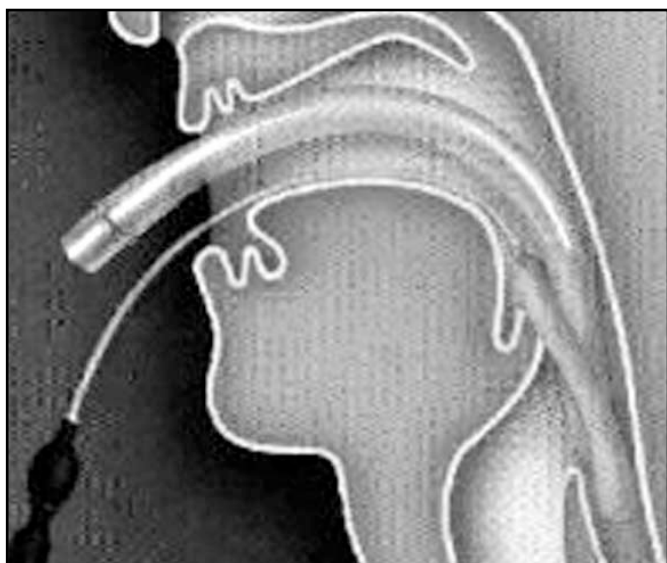

Figure 2

Size selection, pre-use checks and cleaning
The cLMA is supplied in 7 sizes from 1-6 suitable for practically all size patients (table 1). Size selection is generally based on weight for children and in adults the default should be a size 4 for women and a size 5 for men. If one size does not 'fit', it is often worth trying a different size based on clinical judgement. As an approximate rule,

LMA sizes	
Weight	**Size**
<5kg	1
5-10kg	1.5
10 - 20kg	2
20 - 30kg	2.5
30 - 50kg	3
Adult female	2 - 4
Adult male	4 - 5

where patient size suggests one of two mask sizes might be used (eg a patient of 20kg or 30kg) mask performance is frequently better with the larger mask. Mask insertion may be slightly more difficult but airway seal and performance is often superior.

The cLMA is designed for repeated use. The manufacturers guarantee it will perform well up to 40 times based on research showing alterations in the characteristics of the silicone used in it's construction. However some hospitals use the cLMA for up to 100 uses without problem, although the manufacturers do not support this practice.

Checking the laryngeal mask carefully before use is vital. This includes:

● A full visual inspection to ensure the cuff, connector and tubing appear normal

● Exclude foreign bodies within the tube lumen

● Deflate the cuff fully and observe - if there is a leak in the cuff, it will start to re-inflate

● Gentle inflation to ensure pilot cuff and valve function

● Gentle folding over of the tube to detect damage and weakening of the airway tube.

After use the cLMA must be decontaminated (washed and dried) to remove any visible debris (including brushing the interior of the airway tube) then sterilised in accordance with local practice. The cLMA is designed to be autoclaved. There have been concerns that routine cleaning does not remove all protein deposits from laryngeal masks, but there is no evidence to date that this represents an important infection risk. Supplementary cleaning for 20 minutes with potassium permanganate 2mg/L eliminates approximately 90% of protein deposits from cLMAs, although this is not routine in most hospitals. Once cleaned the cLMA should be stored in a sterile fashion.

Routine use
A major advantage of the cLMA, over many other SADs, is that it is readily inserted, even in inexperienced hands, and routinely allows 'hands free' airway maintenance during anaesthesia. In one study of over 11,000 patients the cLMA provided a safe and secure airway in 98.5% of cases. There are a number of advantages for the cLMA including better oxygenation compared to facemask anaesthesia and reduced haemodynamic instability, anaesthetic requirements and sore throat compared to tracheal tube anaesthesia.

Case selection
Despite its versatility the cLMA requires careful case selection to ensure patient safety.

The cLMA is most suited to peripheral and minor surgery in healthy patients who are breathing spontaneously. This is a good place to start and learn. With experience the strengths and limitations of the cLMA are learnt. Obese patients, and those with a history of gastro-oesophageal reflux are less suitable.

Insertion

A greater depth of anaesthesia is needed for insertion of a cLMA than for a guedel airway but less than that needed for tracheal intubation. Depth of anaesthesia is crucial to success as movement during insertion often leads to an imperfectly positioned mask.

Before insertion the patient should be unresponsive with a relaxed jaw and should not response to jaw thrust. However, insertion of the cLMA does not need use of muscle relaxants. Propofol is the most suitable induction agent as it profoundly suppresses airway reflexes and enables cLMA insertion without coughing or movement. If propofol is not available, insertion after thiopentone induction is aided by deepening anaesthesia with volatile agents or topical local anaesthesia to the oropharynx. Whichever induction agent is used, giving a rapid onset opioid (eg fentanyl or alfentanil) prior to induction improves mask insertion. If necessary the cLMA can be inserted under topical anaesthesia.

Insertion is carried out with the patient in the laryngoscopy position ('sniffing the morning air') and may be made easier by an assistant performing jaw thrust during insertion. The cLMA cuff should be fully deflated and the posterior surface well lubricated with a water-based lubricant before insertion. The anaesthetist stands behind the supine patient with one hand stabilising the patients head and neck and the other holding the cLMA. This is best done by placing a hand under the patients occiput and slightly extending the upper cervical spine. The cLMA is held like a pencil at the junction of mask and tube. The route the cLMA should follow mimics the route of a bolus of food during insertion. The cLMA is advanced

Figure 3a and b

Step 1   **Step 2**

posteriorly (as if towards the occiput) along the hard palate and is allowed to follow the posterio-superior aspects of the airway. When the cLMA 'stops' during insertion, the tip has reached cricophayngeus (the upper oesophageal sphincter) and should be correctly positioned. Insertion should take place in one smooth movement to ensure this 'end point' is identified. In the authors' experience meticulous attention to insertion technique is rewarded by improved mask performance and reduced complications. For children and when the standard insertion technique fails partial inflation of the cuff, or initial insertion with the LMA rotated, akin to insertion of a Guedel airway, may help insertion.

The cuff should be inflated prior to connection to the anaesthetic breathing circuit. Five simple tests assist in confirming correct positioning of the cLMA:

1. A definite end point is noted during insertion.

2. The cLMA rises slightly out of the mouth as the cuff is inflated.

3. The anterior neck bulges slightly as the cuff is inflated.

4. The black line on the back of the cLMA remains in the midline.

5. The LMA cuff is not visible in the mouth.

The amount of air 'recommended' for cuff inflation by the manufacturer varies with cLMA size (Table 2). It is important to note that the recommended volumes are maximum volumes. Usually no more than half this volume is required. The volume required is that to achieve a low pressure seal with the airway. The pressure in the cuff should not be above 60 cmH$_2$O. This pressure can be detected at the pilot balloon with an anaeroid barometer and with experience is easily estimated clinically. Over-inflation may increase the risk of pharyngolaryngeal complications, including nerve injuries (glossopharyngeal, hypoglossal, lingual and recurrent laryngeal) and occasionally causes airway obstruction.

Once the cLMA is inserted, head and /or neck movement make little difference to the position of the cLMA though may cause changes in the intra-cuff pressure and airway seal. Nitrous oxide if used will diffuse into the cLMA cuff until the intracuff partial pressure equilibrates with the anaesthetic gas mixture. This leads to increases pressure in the cuff in the first 30 minutes of nitrous administration. Excessive cuff pressures can be avoided by intermittently palpating the pilot cuff.

After insertion patency of the airway should be tested by gentle hand ventilation. Remember the cLMA cuff produces a low-pressure seal around the larynx and airway
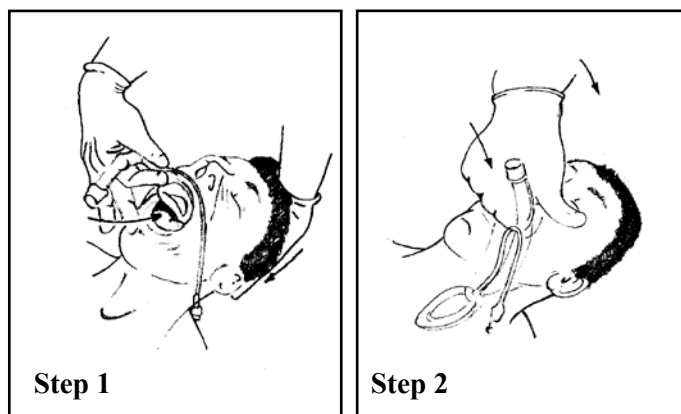
pressures above this seal will cause anaesthetic gases to leak from the airway. Gentle, slow hand ventilation should produce chest rise without airway noise or audible air leak followed by unobstructed exhalation (the anaesthetic reservoir bag rapidly refills). Oxygen saturation should remain stable and the capnograph trace should be square waved. A sloped capnography trace indicates airway obstruction - check for airway noise and raised airway pressure. Similarly if the reservoir bag does not refill normally this indicates either a large leak (does not refill at all) or partial airway obstruction (slow refilling). Where a self-inflating bag is used this useful clinical sign is lost. If there is a large gas leak or airway obstruction the cLMA should be removed and reinserted.

The cLMA should be secured with tape or tied in such a manner that it is prevented from migrating outwards. When the anaesthetic circuit is attached, ensure that its weight is prevented from pulling on the cLMA as this will lead to displacement.

A bite block is recommended, and a roll of gauze between the lateral teeth is effective.

This helps secure the device particularly in edentulous patients and prevents biting down during recovery from anaesthesia leading to airway obstruction, dental damage and damage to the cLMA.

When controlled ventilation is used peak airway pressures in slim adults and most children is usually no more than 10-14cmH$_2$O. Pressures above 20 cmH$_2$O should be avoided, as not only does gas leak out of the cLMA, but this exceeds oesophageal sphincter pressure. At low airway pressures gas leak is out of the mouth but at higher pressures gas enters the oesophagus and stomach increasing the risk of regurgitation and aspiration later in the case.

For small children and infants spontaneous breathing through the cLMA for prolonged periods is probably not advisable. The cLMA increases airways resistance and access to the airway for clearance of secretions is not as good as via a tracheal tube. Fortunately controlled ventilation in this group is often easy as children generally have lungs with high compliance and the airway seal with a cLMA is generally slightly higher in children than in adults.

During the maintenance phase of anaesthesia the cLMA usually provides a clear airway and adjustment of position is rarely necessary. However occasionally misplacement can occur, particularly if anaesthesia becomes light or the patient moves. The anaesthetic circuit reservoir bag should be visible and full monitoring with appropriate alarms should be used throughout anaesthesia to ensure this infrequent event is detected should it occur. If the patient's position needs to be altered it is wise to disconnect

the airway during movement. When the repositioning is complete, reattach the anaesthetic circuit and recheck the airway.

At the end of surgery the cLMA should be left in place until the patient has woken up and is able to open his/her mouth to command, at which time protective airway reflexes have normally recovered. Suction of the pharynx and turning the patient at this time are generally unnecessary and may lead to stimulation and increase airway complications such as laryngospasm. When the patient can open their mouth the cLMA can be removed: most secretions will come out at this time and any additional secretions or blood can be suctioned out at the time of removal if the patient cannot clear them. In children the timing of removal is less clear: some studies suggest a higher rate of complications when the cLMA is removed awake, and some when it is removed 'deep'. Certainly removing it in a 'light' patient who is not fully awake increases problems and should be avoided. If the cLMA is removed deep, watch out for airway obstruction and hypoxia. If removed awake be ready for coughing and laryngospasm.

Avoiding regurgitation and aspiration

The cLMA does not offer protection against pulmonary aspiration of regurgitated stomach contents and it is unwise to use the cLMA in any patient in whom there is an increased risk of regurgitation. This includes the following patients: non-starved patients, emergencies, those with symptomatic hiatus hernia or gastro-oesophageal reflux and the grossly obese. Few patients have 'no risk' of aspiration and it is rational to divide patients into those with low, moderate or high risk of aspiration. The cLMA is a device to use only in those with a low risk of aspiration. The cLMA forms a low pressure seal with the airway and if excessively high pressures are applied to the cLMA during controlled ventilation, increasing volumes leak from the mask into the oesophagus and thence into the stomach increasing the risk of regurgitation.

The risk of serious lung injury, when aspiration occurs, is reduced by neutralising stomach contents (H$_2$ blockers, proton pump inhibitors and antacids all do this) but the best way to avoid this problem is by careful selection of cases, and avoiding use of the cLMA in patients at obvious increased risk of regurgitation / aspiration.

Good practice with the cLMA is as follows:

- select cases carefully

- insert the cLMA at an adequate depth of anaesthesia

- optimise correct placement by using the recommended insertion technique and checks of correct positioning

- avoid high airway pressures both when 'bagging' the patient before the cLMA is inserted

- after insertion use slow low pressure ventilation

- secure the cLMA to prevent misplacement during use

- unless necessary return to spontaneous breathing as soon as possible

- avoid movement of the cLMA during surgery unless unavoidable

- allow the patient to recover until able to open the mouth to command before removing the cLMA

Clinical application and areas of controversy

The cLMA has now been used in a much wider range of clinical settings than when first introduced. Although there remains considerable debate over what is appropriate or safe use of the cLMA even amongst experienced users, in many countries it is now used for up to two thirds of all anaesthetics. For some it has replaced tracheal intubation where this was previously thought to have been the mandatory 'gold standard' eg the paralysed patient, during laparoscopic surgery or advanced life support. However it is important to note that success in cases such as these is dependant on careful case selection, avoiding use when there are contraindications, and employing meticulous technique. It is unwise to use the cLMA for complex and unusual cases until considerable experience is gained with more routine and straightforward cases. Assessing the risk of aspiration is vital for the suitability of the cLMA in situations where intubation would normally be used.

Despite widespread use of the cLMA during controlled ventilation, there remains considerable debate as to the appropriateness of the technique where aspiration is thought to be a risk. There is a potential for the cLMA to disrupt the integrity of the upper oesophageal sphincter and it is also thought possible that an LMA may also reduce lower oesophageal sphincter competence (due to its presence stimulating swallowing reflexes). Laparoscopic procedures are particularly controversial with concerns over increased risk of aspiration from raised intra-abdominal pressure, lithotomy position and increased airway pressures. Despite these concerns there is evidence that the cLMA is widely used both for controlled ventilation, and during gynaecological laparoscopy, with a low incidence of complications. However it is likely that the Proseal LMA (see below) is a better choice for all cases where controlled ventilation is required and the anaesthetist believes a laryngeal mask is indicated.

Post-operative sequelae

These are usually minor. Sore throat is variously reported in 5% to 30% but is almost always minor, transient and less of a problem than after tracheal intubation. Blood is visible on the cLMA in up to 20%, but detectable in microscopic amounts in up to 80%. Rarer complications include nerve injury due to prolonged mucosal compression leading to neuropraxia. Hypoglossal, glossopharyngeal, lingual and recurrent laryngeal nerve palsies have been reported.

Difficult airway

The cLMA is also an important tool in the management of the difficult airway and as an aid to endotracheal intubation. Much of the published literature on its use in management of the difficult airway is anecdotal and has not been subjected to robust controlled trials.

The cLMA has three roles in management of the difficult airway:

1. Use in patients in whom tracheal intubation is difficult or impossible (ie avoidance of tracheal intubation)

2. Rescue of the airway when ventilation and or tracheal intubation fail.

3. As a conduit for assisting tracheal intubation and allowing oxygenation during attempts.

When considered as an alternative to tracheal intubation it is important to note that physical features that predict difficulty with laryngoscopy and intubation do not predict difficulty with cLMA placement. Importantly however mouth opening is critical. If mouth opening is less than 2.5cm cLMA insertion is very difficult and if below 2.0 cm it is impossible.

The cLMA will rescue an obstructed airway in more than 90% of cases. This is particularly so when the obstruction is supraglottic. For this reason it can be argued that a cLMA should always be available (and the anaesthetist familiar with it!) wherever anaesthesia is provided. When cricoid pressure is applied this obliterates the space that the tip of the cLMA must enter when correctly placed. So when the cLMA is inserted (indeed for all laryngeal masks) it is important to reduce or release cricoid pressure during insertion. Once the device has been place cricoid pressure can be resumed.

The bowl of the cLMA lies over the glottic in more than 90% of cases making it a suitable device for attempting to instrument the trachea when conventional methods fail. Narrow endotracheal tubes (ET) and bougies have been reported for this use. Importantly the cLMA allows oxygen and ventilation to continue uninterrupted during these attempts. Relatively long and narrow tubes are needed, particularly if an attempt is then made to remove the cLMA (table 2). There is a danger of the tube catching on the bars of the mask as it passes and if the tube is too short it may not reach the trachea, or the tracheal tube cuff may sit at or outside the glottis. The glottis in an adult is usually approximately 3cm beyond the bars of the cLMA so an endotracheal tube must be advanced approximately

6-7cm beyond this to ensure the tube tip and cuff are inserted far enough.

Blind techniques with an endotracheal tube or bougie may be successful but fail far more often than they succeed. Where available fibreoptic-guided techniques increase success rates to close to 100%. A specifically designed semi rigid tube of 80 cm length and with internal and external diameters of 4.6mm and 7.0mm respectively (the Aintree Intubating Catheter, AIC), solves many of the problems associated with cLMA-guided tracheal intubation in adults. The AIC is mounted on a fibrescope and then placed in the trachea. It is long enough to allow removal of both fibrescope and cLMA before railroading an ET (>7.0mm id) over it. There is increasing experience and published data with the AIC demonstrating a high level of success. Where this is not available narrow bore uncut tracheal tubes may be used instead. It is worth remembering that the tracheal tube will need to extend a considerable distance beyond the cLMA grilles in order to reach and pass through the vocal cords. The length of tube needs to be even longer if it is cuffed, in order to avoid inflation of the cuff within or above the vocal cords.

Developments of the cLMA
The inventor of the cLMA has produced three different laryngeal masks since the cLMA. These are the flexible LMA (fLMA), Intubating LMA (ILMA), and the ProSeal LMA (PLMA). In addition single-use versions of the cLMA (the LMA-Unique (LMA-u), flexible LMA and ILMA have been recently developed. The most recent innovation is an ILMA with built in fibreoptics allowing intubation under direct vision, the C-trach.

Table 2 Ability to pass an endotracheal tube though a variety of laryngeal masks			
cLMA size 1	3.5 id	distance to bars	108mm
size 1.5	4.5 id		135mm
size 2	5.0 id		140mm
size 2 1/2	6.0 id		170mm
size 3	6.5 id		200mm
size 4	6.5 id		205mm
size 5	7.0 id		230mm
fLMA (not recommended for this technique)			
size 2	3.5 id*	distance to bars	140mm **
size 2 1/2	3.5 id*		205mm**
size 3	5.5 id		237mm
size 4	5.5 id		237mm
size 5	6.0 id		284mm
PLMA size 3	6.0 id	distance to bowl	190mm
size 4	6.5 id		190mm
size 5	6.5 id		195mm
ILMA	7.0-8.0 mm special ILMA tubes	distance to epiglottic elevator	160mm

Notes
1.   These are the sizes that can be passed through the LMA without force when well lubricated.
2.   These figures can only be regarded as a guide as different manufacturers ETs have different external diameter for same ID.
3.   This data relates to Portex blue line ETs for which external diameter ranges from 1.3- 2.6mm greater than internal diameter.
4.   This data only applies to cLMAs made by Intavent Orthofix or the LMA company.
5.   If using a cuffed ETT use a size 0.5 mm less.
*6.   *A tube larger than this cannot pass the proximal end of the LMA, at the site of the 15mm connector*
*7.   **This is longer than an uncut ETT of this size, so not suitable for this technique*
The cLMA varies in price but costs approximately US $120.

The flexible laryngeal mask airway (fLMA)

The mask of the fLMA is identical to that of the cLMA but the airway tube is wire-reinforced. This ensures that movement of the proximal end of the tube is less likely to be transmitted to the mask end and increases flexibility allowing it to be positioned away from the surgical field without dislodging the mask. The fLMA is particularly useful in head and neck, maxillofacial and ENT surgery where the flexible nature of the airway stem allows the anaesthetic circuit to be moved out of the surgical field while still allowing surgical access. The fLMA provides good protection of the larynx from secretions and blood above the device and has become popular for nasal and intraoral surgery, including tonsillectomy.

The fLMA has a longer and narrower airway tube than the cLMA (table 2). This increases the resistance of the tube and work of breathing through it: however this is not a problem in practice as the resistance is still comparable to a tracheal tube. The fLMA is not MRI compatible because of the metal in the reinforced airway tube.

The fLMA is supplied in sizes 2-5. Insertion of the fLMA can be more difficult than the cLMA due to the flexibility of the airway tube: the mask can rotate 180º on its axial plane (leaving the mask orifice facing backwards) without this being detected at the circuit end. Good insertion technique is likely to reduce this and a number of techniques are described to assist insertion, including guiding the fLMA into place by opening the airway with a laryngoscope. A specific insertion device the 'Flexiguide' that is inserted into the fLMA and grips it from inside has been shown to aid insertion.

The long narrow airway tube makes the fLMA the least suitable laryngeal mask for accessing the trachea when this is required. As a result the fLMA has only a limited role in the management of the difficult airway.

The fLMA costs approximately 30% more than the cLMA and is recommended to be used 40 times.

The Intubating Laryngeal mask airway (ILMA)

The ILMA (or Fastrach) is a laryngeal mask designed specifically to enable tracheal intubation. Although it is possible to intubate via a cLMA the long narrow airway tube creates some difficulties. The airway tube of the ILMA is rigid, shorter and of a wider diameter then the cLMA. At the proximal end of the ILMA airway tube there is a metal handle, which assists insertion and is critical for aiding intubation. This allows insertion and manipulation of the device without the operator having to place their fingers in the patient's mouth. At the mask end the bars of the mask are replaced by an 'epiglottic elevator', a semi rigid bar attached to the mask at only one end. Unlike other laryngeal masks the ILMA is designed for insertion with the patient's head and neck in the neutral position.

The ILMA is produced in sizes 3-5, and is supplied with a re-useable silicone bullet-tipped tracheal tube; the ILMA tube. The ILMA tubes range in size from 6.0-8.0 mm id, with all sizes of tubes passing through all ILMAs. The ILMA is inserted by holding the handle and then advancing the tip of the mask into the airway in a smooth arc in the curve of the mask (and airway) until it stops.
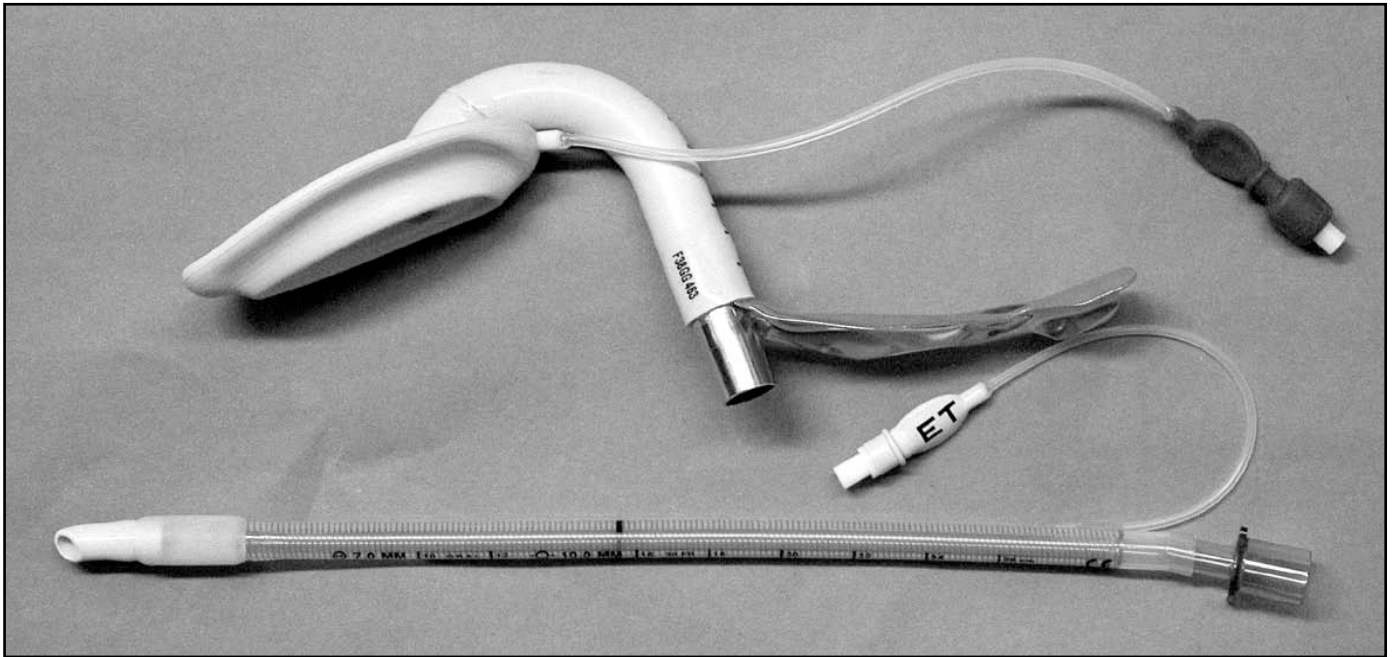
Figure 4: Flexible LMA
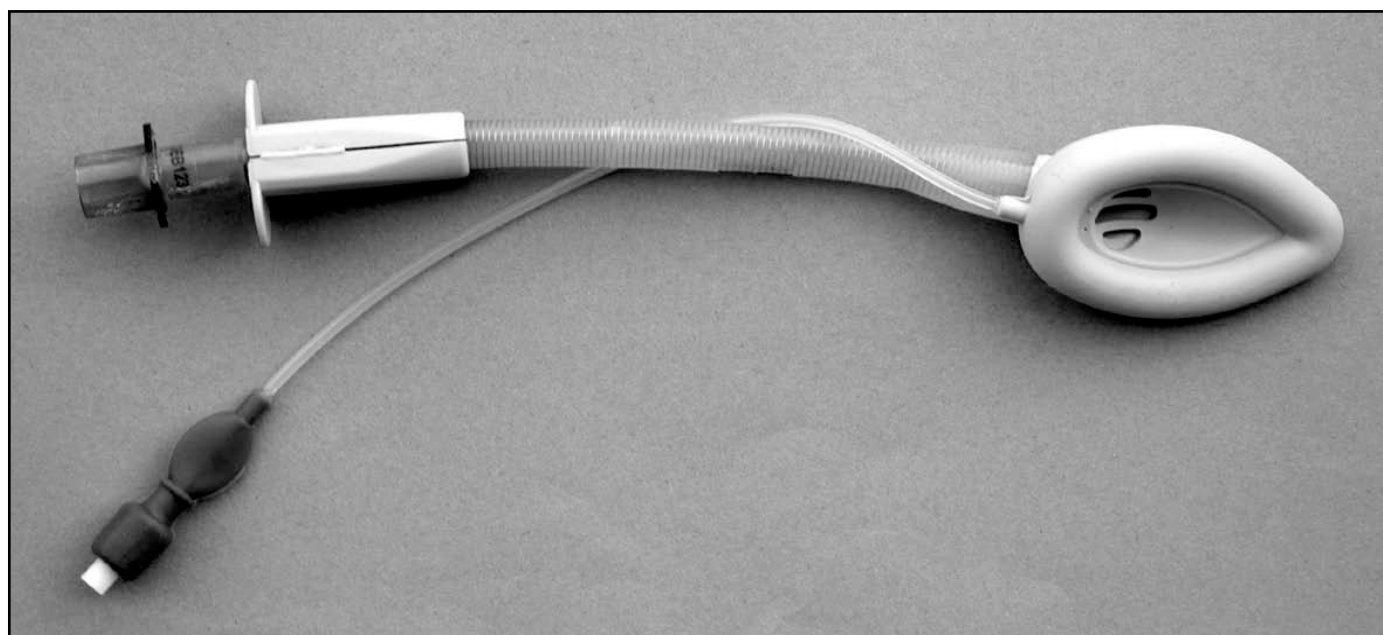
Figure 5: ILMA and ILMA tracheal tube

When blind intubation is to be attempted the anaesthetic circuit is attached and hand ventilation started while manoeuvring the ILMA handle to find the position where ventilation of the lungs is easiest (lowest resistance, highest compliance). In this position the ILMA orifice is most likely to lie opposite the larynx. Helpful movements include partial withdrawal or deeper insertion, lifting the mask to press against the larynx (named the Chandy manoeuvre after its originator), and an out/in manoeuvre to overcome epiglottic downfolding. Once correctly positioned the ILMA tube is gently advanced through the ILMA. Tube markings assist correct positioning. As the tube tip passed through the distal end of the airway tube it pushes the epiglottic elevator bar outwards, which lifts the epiglottis out of the path of the tracheal tube (TT). The ILMA tube is then rotated and advanced gently. Resistance usually indicates failure to intubate the trachea. As this is a 'blind intubation technique' it is important to use only gentle force and to confirm successful intubation (with a capnograph or Wee oesophageal detector) and exclude bronchial intubation. Fibreoptic and light-guided techniques increase intubation success with the ILMA.

The ILMA should not be used for those patients with contraindications to laryngeal mask use. When advancing the TT, resistance to passage should never be overcome by force. A soft tipped TT is recommended. The ILMA should not be used in patients with upper oesophageal pathology. Fatal oesophageal perforation has been reported when these precautions were not followed.

After intubation it is recommended to remove the ILMA for all but the shortest procedures, as the rigid airway tube exerts high pressures on the surrounding mucosa. Removal of the ILMA is achieved by using a 'tube stabiliser' (or the hub of a 5ml syringe) to stabilise the ILMA tube while the ILMA is withdrawn over it. Sore throat and hoarseness, though usually mild, are more frequent after use of the ILMA than the cLMA.

The ILMA has an important role in management of both anticipated and unexpected difficult intubation. It is suitable for managing patients with cervical spine injury and may be used during cardiopulmonary resuscitation. The ILMA achieves an airway seal between that of the cLMA and the PLMA.

Several studies have evaluated the success rates of intubation with the ILMA. First attempt blind intubation success may be as high as 75% but in pooled studies is 66% and second attempt 22%. Overall success is approximately 95% when studies including patients with easy and difficult airways are included. Intubation success is increased to approaching 100% when a fibreoptic or light-guided technique is used. In the largest series of difficult airways, of almost 300 patients, overall success was 97%, increasing to 100% when fibreoptic guidance was used. If this is combined with four other studies then for patients with known or predicted difficult airways the overall intubation success rate is 92%, with 62% intubated at the first attempt.

Haemodynamic responses to intubation via the ILMA are similar to conventional intubation with a laryngoscope. However despite this success it is unlikely that the ILMA will enter mainstream practice for routine tracheal intubation.

There is evidence that individuals who are naive to both devices achieve greater success with ILMA than cLMA insertion. There is probably a learning curve of approximately 20 intubations though this may improve by training using a Manikin.

In addition to its role in difficult intubation, the ILMA is popular in some areas for assisting intubation in patients with cervical trauma. The patient can be left in the neutral position during ILMA insertion. It is usually necessary to take off the hard collar and stabilise the head with manual in line stabilisation (MILS) during insertion. Cricoid pressure also impedes ILMA placement and needs to be reduced or removed during insertion. Once the ILMA is inserted, cricoid pressure may be reapplied without altering function. Insertion of the ILMA and intubation through it cause minimal movement of the cervical spine.

Finally the ability to insert the ILMA from behind, in front or from the side of the patient and with the patient supine, lateral or even prone means that the ILMA is a suitable airway for insertion during extraction of patients who are entrapped.

The ILMA is an expensive device costing approximately $500 and may be reused up to 40 times. A single use ILMA has recently been marketed but its performance is unevaluated. The most recent development of the ILMA is one with an integrated camera in the bowl communicating with a video screen close to the handle. For those with up to $7000 this will allow the ILMA to provide intubation under direct vision without need for a fibrescope.

The ProSeal Laryngeal mask airway (PLMA)
The PLMA was introduced in 2000. It is designed with three improvements in mind:

1. Improved performance during controlled ventilation

2. Improved safety regarding aspiration

3. An ability to diagnose misplacement of the device tip.

The PLMA has a softer, larger deeper bowl than the cLMA. The mask cuff extends over the back of the device pushing it forward when inflated. There is a drainage tube passing from the tip of the mask, through the bowl to run parallel to the airway tube attached to it by an integral bite block.

When correctly positioned the drain tube of the PLMA lies at the top of the oesophagus encircled by cricopharyngeus and the bowl lies over the airway. These tubes therefore allow continuous passage to or from the gastrointestinal or respiratory tract, respectively, to the outside world. Further the gastrointestinal and respiratory tracts are functionally separated. As such, unlike the other laryngeal masks, which are simple airway tubes, the PLMA can be considered as a form of 'artificial larynx'.
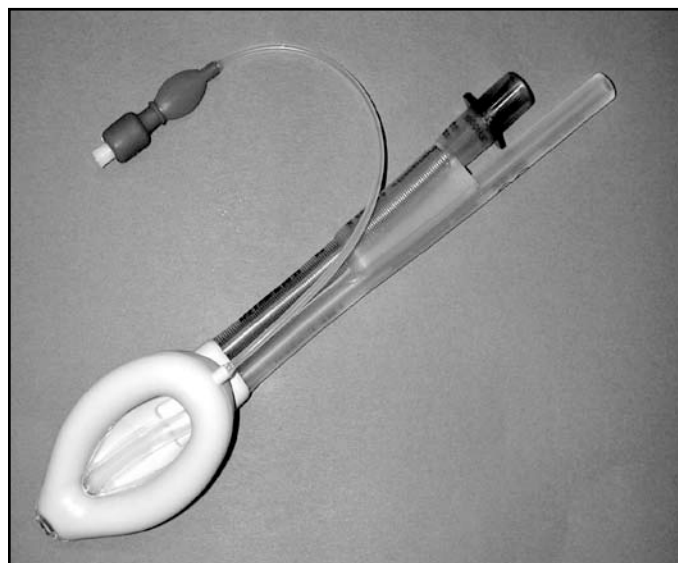

Figure 6: PLMA

The PLMA may be manually inserted (like the cLMA) or by attaching a metal introducer (rather like the ILMA insertion technique). Finally when insertion is difficult it may be railroaded via the drain tube over a bougie placed in the oesophagus. The latter technique is the most invasive but appears to be the most successful and least likely to lead to misplacement (there is a tendency for the PLMA to fold over during insertion if insertion technique is not meticulous). It is perhaps the technique of choice when first time insertion success is critical, such as when managing a difficult airway. The drainage tube allows reliable insertion of an orogastric tube. In fact inability to pass a gastric tube via the PLMA drain tube nearly always means the PLMA is misplaced with the posterior of the mask folded backwards.

A series of post insertion tests are recommended to confirm correct positioning of the device:

1. When correctly positioned the PLMA should allow leak-free ventilation with square wave capnography and airway pressures below 20 cmH$_2$O. Gel placed on, and occluding, the proximal drain tube should not be displaced when a pressure of 20 cmH$_2$O is applied to the airway; this tests separation of the gastrointestinal and respiratory tracts and fails most often when the PLMA is not pushed in far enough, allowing gas to pass directly from the airway tube up the drain tube.

2. Usually no more than one third of the bite block should be visible as this also suggests incomplete insertion.

3. Pressing on the chest should not displace the drain tube gel; if it does it suggests the drain tube tip has entered the glottis, though airway obstruction is likely to coexist.

4. Pressure at the suprasternal notch should lead to the drain tube gel bulging outwards. This tests that the drain tube is
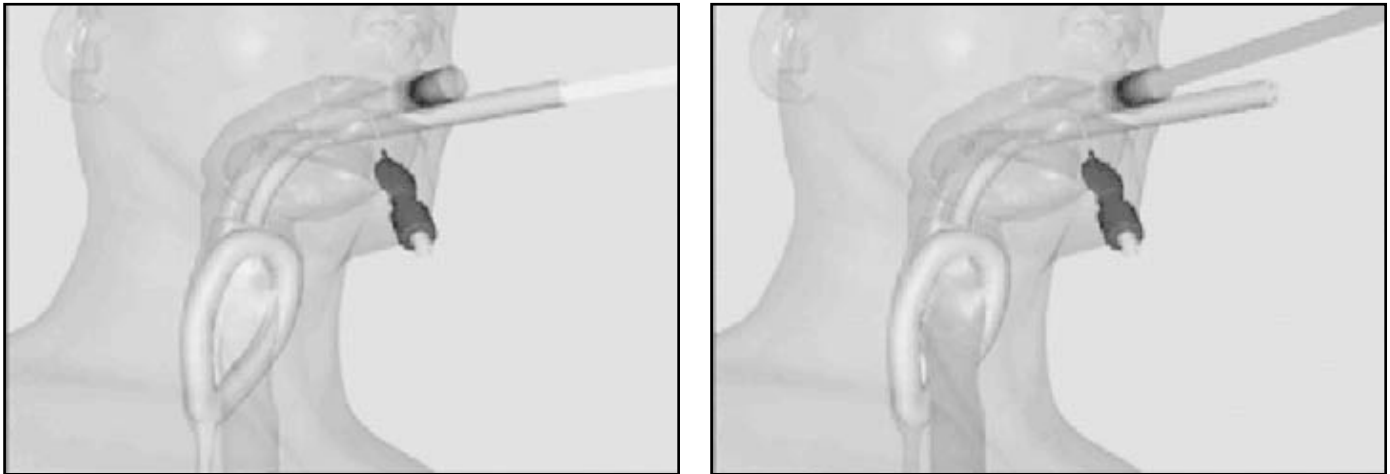
Figure 7a and b: PLMA positioning and GI vs RS route. Thanks to Intavent for illustrations.

not bent over; suprasternal notch pressure is transmitted to the oesophagus and then to the drain tube, unless the tip is folded over. If in doubt attempting to pass an orogastric tube to the tip of the drain tube, or beyond, will identify whether it is folded over. In practice these short tests can be carried out in a matter of seconds to confirm correct positioning and function of the PLMA, with further attention only necessary if a test is not 'passed'.

Importantly the drain tube allows confirmation of correct placement, or diagnosis of the type of misplacement of the PLMA: a feature not possible with other laryngeal masks.

Changes to that mask shape and size added to the posterior cuff increase the average airway seal by >50% to above 30 cmH$_2$O.

There is good theoretical and performance evidence to support the view that compared to the cLMA the PLMA reduces gas leak, gastric inflation and increases protection from regurgitated gastric contents. However this is entirely dependant on correct positioning of the device.

So do these modifications make the PLMA a superior device? On the positive side there is increased seal pressure (allowing a wider range of patients to be ventilated) and increased safety against aspiration. The PLMA also exerts less pressure against the mucosa than either cLMA or ILMA for a given insufflation pressure so reducing the potential for mucosal trauma and damage. These advantages must be balanced against slightly greater difficulty in insertion of the PLMA. When combining a large number of studies, first time insertion success with the PLMA was 85% and with the cLMA was 93%. In addition when the PLMA is used during spontaneous ventilation there are reports of minor complications including partial airway obstruction and oesophageal breathing via the drain tube, leading to oesophageal distention and even the possibility of gastric

distension. These complications are generally overcome by using controlled ventilation, or passing an orogastric tube.

The PLMA is the most recently introduced laryngeal mask. It has advantages over others when controlled ventilation is used. Whether its advantages in terms of safety extend to other clinical circumstances is as yet unproven. It is possible that it will become the standard laryngeal mask in time because of increased safety, but this will require more research and is far from certain.

The PLMA has no grill or elevator bar across the airway opening to the mask. This offers the possibility of an unimpeded view and access to the larynx when managing the difficult airway. In these circumstances improved ventilatory performance and reduced risk of aspiration are also likely to be benefits. The vocal cords appear to be visible through the PLMA as frequently as through the cLMA. The airway tube of the PLMA is shorter and slightly narrower than that of the same size cLMA and similar problems exist for direct tracheal access. There is as yet little written on the use of the PLMA for management of the difficult airway but one area of interest is for airway rescue after failed rapid sequence induction where its characteristics appear very suitable. Several reports have been published, and the PLMA appears in the UK Difficult Airway Society Guidelines for this situation as one of the options.

The PLMA cost approximately 10% more than a cLMA and is recommended for up to 40 uses.

Single-use laryngeal masks
Several laryngeal masks are now available in single use versions. A PVC single use cLMA was introduced in 1998, the fLMA followed in 2003 and the ILMA in 2004. The cLMA came 'off patent' in 2003 and there are now at least six manufacturers who make similar devices based closely

on its design. All of these except one are made of PVC rather than silicone. The move towards single use devices accelerated following the recognition that prion diseases (causing variant Creutzfelt Jacob or 'mad cow' disease) were potentially transmissible despite current disinfection and sterilising techniques. The extent of this risk is at present unproven and unquantified, but is considered exceptionally low. In addition to clinical considerations a change from reusable to single-use devices has impacts on cost, storage and environmental factors. Evidence for the effectiveness or safety of single-use laryngeal masks is so far limited, and it is too early to say whether they will perform less well, as well, or better than the cLMA. It is difficult to recommend, on the basis of evidence, whether a change to single use laryngeal masks is sensible at this time, and if so which device to chose.

Summary
The family of laryngeal masks have revolutionised airway management in anaesthesia in many parts of the developed world. In many countries the cLMA is now 'the standard' airway for anaesthesia, with other devices including the endotracheal tube only being used when there is a specific indication. In addition the laryngeal masks have important roles in difficult airway management, in which they may be used as an alternative to tracheal intubation, may rescue the lost airway and may aid alternative intubation techniques. Each laryngeal mask has strengths and limitations. It is only by understanding these, selecting (or excluding) patients and mask type carefully and by meticulous attention to technique that the role of the laryngeal mask may be extended safely to the benefit of both patient and anaesthetist.

Further reading

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2. Caponas G. Intubating laryngeal mask airway. A review. Anaesth Int Care 2002; 30: 551-69

3. Cook TM, Lee G, Nolan J. The ProSealTM Laryngeal Mask Airway: A review of the literature. Can J Anaesth 2005: 52: 739-60.

STUDY INTO HARMONISATION OF DRUG CONCENTRATIONS TO PROMOTE SAFE PRACTICE

Dr Melinda Lyons & Dr Dan Wheeler, Cambridge University Hospitals NHS Foundation Trust, Cambridge UK

Dear All,
We would appreciate help in a research project we are conducting into the different ways the concentration of drugs in solution are expressed around the world.

Previous research in our department has shown that there is considerable confusion about solutions when their strength is expressed as a ratio or percentage. We are interested to hear from practitioners in different countries about the number of terms used to describe different drugs.

We would therefore be very grateful if you could please email us the answers to the questions below, giving your name, position and country.

Which of the ways listed are the drugs below described in your country?

If any further information or clarification is required, please contact one of the research team listed below. In addition, if you think this could be more appropriately answered by another organisation or person in your country, would you please pass on this message or provide us with their contact information.

Many thanks indeed for your time.

Kind regards,

Dr Melinda Lyons, Clinical Scientist. Email: mnl24@cam.ac.uk and **Dr Dan Wheeler**, Clinical Lecturer. Email: dww21@cam.ac.uk, University Department of Anaesthesia, Box 93, Addenbrooke's Hospital, Hills Road, Cambridge, CB2 2QQ

Tel: +44 1223 217889   Fax: +44 1223 217887

Epinephrine (adrenaline)	: ratio (e.g. 1 in 1,000), percentage (%) or mg/ml?
Lidocaine (lignocaine)	: ratio (e.g. 1 in 1,000), percentage (%) or mg/ml?
Atropine	: ratio (e.g. 1 in 1,000), percentage (%) or mg/ml?
Sodium bicarbonate	: ratio (e.g. 1 in 1,000), percentage (%) or mmol/l, mg/ml?
Dopamine	: ratio (e.g. 1 in 1,000), percentage (%) or mg/ml?

MULTIPLE CHOICE QUESTIONS
Dr K Holmes, Torbay Hospital, UK

Please answer True or False to the following statements:

1. Regarding storage of donated blood:
a. Blood storage in CPD-A preserves but does not anti coagulate the blood
b. SAG M gives blood a shelf life of 14 days
c. Red blood cells must be stored at around - 4 degrees Celsius
d. Levels of 2,3 DPG can become depleted in stored blood
e. Potassium levels increase with duration of storage

2. The following are true of "MAC":
a. It is the mean alveolar concentration
b. It is proportional to the oil-gas solubility co-efficient
c. It is proportional to the blood-gas solubility co-efficient
d. The MAC of halothane is 1.0
e. The MAC of enflurane is 1.6

3. With regard to cricoid pressure:
a. It was first described by Simmons
b. The pressure that should be applied is 10 Newtons
c. It is contra-indicated in active vomiting
d. It is useful in patients with a pharyngeal pouch
e. It was first used in paediatric anaesthesia

4. Awake fibreoptic intubation:
a. Is indicated in the management of the bleeding airway
b. Is indicated in acute upper airway obstruction
c. Can be performed using a sedation
d. Is contra-indicated in patients with an unstable C spine injury
e. Can be performed orally and nasally

5. Breathing circuits:
a. A Mapleson B is used as a co-axial circuit
b. A Mapleson D is only used for resuscitation
c. Mapleson D circuits are more efficient for spontaneous ventilation
d. Flows with the Ayres T piece should be 1.5 times the patient's minute volume for spontaenous respiration
e. The Lack circuit is more efficient for spontaneous ventilation than controlled ventilation

6. Absolute contra-indications to regional anaesthesia in obstetrics include:
a. HELLP syndrome
b. Essential thrombocythaemia
c. Clopidogrel therapy
d. Hypertensive disease of pregnancy
e. Systemic infection

7. Regarding nerve blocks:
a. With axillary blocks, the nerve most often missed is the radial
b. Interscalene blocks are reliable for hand surgery
c. Intercostal nerve blocks may produce high systemic levels of local anaesthetic
d. Blocks at the elbow can be associated with ulnar nerve compression
e. Four approaches to the sciatic nerve have been described

8. Sciatic nerve blocks:
a. Should be performed using bupivacaine with adrenaline
b. Using the Raj approach require the patient to lie in the left lateral position
c. Can be performed using an anterior approach
d. Can block the whole lower limb alone
e. Always block the posterior cutaneous nerve

9. In neonatal resuscitation:
a. Cardiac compressions should be started if the heart rate is less than 60
b. Ratio of ventilations to compressions is 3:1
c. Cardiac compressions are performed with the heel of the hand
d. Apgar scoring looks at the colour of the baby
e. The commonest reason for continued bradycardia is failure of lung ventilation

10. In preoperative assessment of the airway:
a. Mallampati class III and IV are associated with a higher incidence of difficult laryngoscopy
b. The thyromental distance should be less than 6.5cm
c. An underbite will make intubation more difficult
d. Sternomental distance should be more than 12.5cm
e. It is possible to predict all difficult intubations

11. Regarding physiological changes in pregnancy:
a. The increase in cardiac output is mainly due to an increase in heart rate
b. The anatomical dead space is increased at term
c. Gastric acidity increases in the third trimester
d. Gastric emptying is delayed in pregnancy
e. By the third trimester, total blood volume decreases

12. The following are true of blood groups and transfusions:
a. ABO antibodies usually cross the placenta
b. Delayed haemolytic transfusion reactions can cause jaundice at 5-7 days post transfusion
c. 50% of people secrete ABO group substrates in their bodily fluids
d. Rhesus antibodies are formed naturally, without external exposure
e. ABO group incompatibility transfusions are most often due to clerical error

13. In paediatric anaesthesia:
a. The dose of atropine is 20mcg/ kg
b. A size 4.5 tube would be suitable for an average 6 year old
c. The dose of adrenaline in an arrest would be 0.1mg/ kg
d. Suxamethonium is given at 1.5mg/kg in infants
e. Propofol infusions are recommended for long term sedation

14. In the head-injured patient:
a. Careful maintenance of the mean arterial pressure is paramount
b. The CO_2 should be reduced to below normal range
c. Tying the endotracheal tube tightly in place can cause venous congestion
d. The patient should be positioned in a 30 degree head down position
e. Mannitol 20% should be given as soon as possible

15. Concerning adequate reversal of neuromuscular blockade:
a. Measurement of tidal volume is a reliable guide
b. A sustained head lift for 5 seconds is sufficient
c. A train of four (TOF) ratio of < 0.7 is inadequate
d. Hypokalaemia is antagonistic
e. A vital capacity of 10ml/ kg can be used as a measure of reversal

16. With regard to vaporizers:
a. Plenum vaporizers can usually be refilled whilst in use
b. Drawover vaporizers have a high resistance when used in a circuit
c. The Goldman is a plenum vaporizer
d. The desflurane vaporizer has a back-up battery
e. An isoflurane vaporizer could be a Tec 6

17. The following are methods of volatile agent analysis:
a. Infrared spectroscopy
b. Raman scattering
c. Mass spectrometry
d. Paramagnetic analyser
e. Raleigh spectroscopy

18. Concerning patient temperature control in theatre:
a. The greatest core temperature drop occurs in the first hour
b. The greatest heat loss is due to radiation
c. All heat loss occurs in the first hour
d. Warmed and humidified gases have little effect on temperature maintenance
e. Thermocouples are commonly used to measure patient temperature.

19. The following statements about pre-eclampsia are true:
a. It is hypertension, proteinuria and pathological oedema at greater than 20 weeks gestation
b. It causes hypovolaemia and a reduction in systemic vascular resistance
c. It increases the risk of airway oedema
d. It can be treated with intravenous magnesium
e. If the blood clotting test is normal, no other blood tests are required before epidural insertion

20. With regard to cigarette smoking and general anesthesia:
a. Smoking can reduce available oxygen in haemoglobin by up to 25%
b. Abstinence from smoking for 6 hours can usefully increase arterial oxygen levels
c. 6 weeks of smoking abstinence leads to a reduction in respiratory mucus production
d. The cardiovascular effects of smoking are caused by nicotine
e. Smokers are less likely to suffer from post-operative

ANAESTHESIA AND BABIES

Isabeau Walker, Consultant Paediatric Anaesthetist, Great Ormond Street Hospital

Age is an important risk factor when discussing anaesthesia morbidity and mortality. The risks of anaesthesia are greater in neonates and infants, even in expert hands[1,2]. This presentation will consider the physiology of the neonate and the premature infant in an attempt to explain this particular vulnerability. In addition to the dramatic physiological changes in the transition from intrauterine to extrauterine life at birth, the newborn infant undergoes a period of rapid maturational development, particularly in the first few months of life. Recent research indicates that experiences during this period of rapid development may have lifelong effects[23].

Physiology of the neonate

The full term neonate is defined as a child born between 37-40 weeks gestation and less 1 month of age, the premature neonate a child born before 37 weeks gestation, the infant a child from 1 month to 1 year of age. Prematurity is defined as a gestational age of less than 37 weeks - extreme preterm infants are born between 23 weeks (limit of viability) and 27 weeks. Premature infants may be low birthweight (<2.5kg), very low birth weight (VLBW) (<1.5kg) or extremely low birth weight (ELBW) (<1.0kg).

Cardiovascular function [3]

Transitional circulation. Fetal life is characterised by the presence of fetal shunts (ductus arteriosus, sinus venosus and foramen ovale). These enable blood to bypass the non-aerated lungs such that in the foetus, less than 10% of the right ventricular output passes through the lungs. Pulmonary vascular resistance (PVR) is high, systemic vascular resistance (SVR) low. At birth, the situation is reversed - clamping of the umbilical vessels results in a dramatic increase in SVR at the same time as the first breaths are taken and PVR drops. Pulmonary blood flow increases, pulmonary venous return and pressure in the left atrium increase; the flap valve covering the foramen ovale closes. The arterial duct constricts in response to oxygenation, later fibrous occlusion occurs over several weeks. Patent arterial duct (PDA) is seen in 50% of VLBW infants due to inadequate constrictor muscle in the immature duct and deficient metabolism of prostaglandins that maintain ductal patency. Left to right shunting results and is a risk factor for respiratory distress syndrome, intraventricular haemorrhage, nectrotising enterocolitis and poor outcome.

Pulmonary vessels in the fetus are well muscularised when compared to similar arteries in the adult. The oxygen tension in fetal lung is 3kPA; the high PVR in fetal life results mainly from hypoxic pulmonary vasoconstriction. Physiological control of PVR is also modulated by the balance between endothelial derived factors such as endothlin[1] and leucotrienes (vasoconstrictor) and nitric oxide (NO) and prostaglandins (vasodilator). At birth, the newborn lung is exposed to oxygen and vasodilatory mediators and the PVR is dramatically reduced. Further reduction of PVR takes place due to involution of smooth muscle in the pulmonary arterial walls. The PVR remains relatively high in neonates (estimated PVRI 3-5 Wood units/m^2) only reaching adult levels (PVRI 0.8-1.9 Wood units/m^2) at about 2 months of age. The relatively high PVR in neonates explains why infants with significant left to right shunts may not become symptomatic until a few months of age. The pulmonary vasculature of the neonate remains sensitive to vasoconstrictor effects, for instance due to hypoxia, hypercarbia and acidosis. Severe pulmonary hypertension may ensue, with reopening of the fetal shunts (foramen ovale/ductus arteriosus), profound hypoxia and cardiovascular compromise. Conditions which predispose towards this condition, Persistent Pulmonary Hypertension of the Newborn (PPHN), include congenital diaphragmatic hernia, meconium aspiration, asphyxia, hypoxia and sepsis. Management includes optimising oxygenation and ventilation (avoiding over/underinflation, loss of synchronisation), sedation, inotropic support, inhaled nitric oxide and high frequency oscillatory ventilation (HFOV) and occasionally extracorporeal membrane oxygenation (ECMO).

Immature myocardium. The right ventricle and left ventricles in term neonates are symmetrical with the right ventricle forming the apex of the heart. Chamber proportions change in response to the haemodynamic workload of the heart - by three months the left ventricle is dominant, as in adults. The cardiac myocytes multiply in number in the first seven months of life (hyperplasia), thereafter cardiac weight increases due to hypertrophy of cardiac myocytes.

The immature myocyte is spherical in shape with disorganised intracellular contractile elements, immature sarcolpasmic reticulum and disorganised supporting extracellular matrix. The myocardium therefore has limited functional reserve, is dependent on extracellular calcium for contraction (ionised hypocalcaemia is poorly tolerated) and is relatively stiff and non-compliant. Functional reserve

increases in the first few days of life with an increase in stroke volume. The Frank-Starling relationship regulates cardiac output as in adults and neonates do increase cardiac output with careful volume loading, especially after the first few weeks of life. Contractility in neonates is high due to high sympathetic tone, especially around the time of birth, and this also explains the high resting heart rate of neonates. Cholinergic innervation is also well developed at birth and vagally mediated cardiac reflexes are well developed even in premature infants. Analysis of heart rate variability indicates that parasympathetic control becomes more important with age (reduction in heart rate). Heart rate is an important determinant of cardiac output and the heart rate should be maintained in the normal range (120-180bpm, term neonate). Afterload is also a major determinant of left and right ventricular output. The SVR in a healthy term neonate has been estimated to be 244mmHg/l/min (mean 723mmHg/l/min in healthy young adults).The neonatal heart is exquisitely sensitive to increases in SVR or PVR. Ventricular interdependence may exaggerate the effects of increased afterload. Neonates respond to inotropes in a predictable manner - dopamine, dobutamine and adrenaline are commonly used, milrinone especially useful after cardiac surgery.

Cardiovascular immaturity results in neonates being more sensitive to the negative inotropic effects of anaesthetic agents than older children. Volatile anaesthetics all reduce myocardial contractility due to an effect on intracellular calcium release; contractility decreases in a dose dependent manner, the effect more marked with halothane than isoflurane or sevoflurane. The infant baroreceptor reflex is poorly developed and abolished by anaesthesia. Atropine may counteract the reduction in cardiac output seen with volatile agents and will protect against vagally mediated reflexes.

Respiratory function[4,5]
Control of ventilation. Periodic breathing is a feature of newborn infants. The ventilatory response to hypercapnia is blunted in the first few weeks of life. Neonates respond to hypoxia by a brief increase in ventilation followed by apnoea. The apnoeic response to hypoxia is probably due to respiratory muscle fatigue or upper airway obstruction. However, by three weeks of age in the term infant, with maturation of the chemoreceptor centres, hypercapnia and hypoxia cause a sustained increase in breathing, as seen in adults.

Anaesthetic agents depress ventilation in a dose dependent manner. Neonates are thus at risk from postoperative apnoeas, especially if born prematurely, especially if anaemic. There is little evidence with respect to term neonates, but it generally accepted that the risk of postoperative apnoea after routine minor surgery is low at

44 weeks post conception. However, in premature neonates the probability of postoperative apnoeas decreases to less than 1% only at 60 weeks post conception[6].

Lung development. Development of the lung starts early in embryonic life, but continues well into childhood. Of note, airway branching is completed at 16 weeks gestation terminal sacs first appear at 24 weeks, surfactant producing type II pneumocytes appear between 24-26 weeks, capillary networks surrounding the terminal sacs at 26-28 weeks. Alveolar development begins at 32 weeks reaching 10% of the adult number at birth. Alveolar development is complete by 18 months of age.

Respiratory mechanics. The newborn lung is small in relation to body size. The metabolic rate is high, tidal volumes small but similar per kg body weight as adults (7ml/kg), thus the respiratory rate is high (30-40 breaths per minute) and there is little respiratory reserve. Lung compliance is low and the ribs soft and elastic; chest wall compliance is higher compared with adults. Chest wall stability increases by about 1 year of life. Thus the distending pressures on the lung are low and the newborn infant is prone to lung collapse, especially under general anaesthesia. Anaesthetic agents also depress the pharyngeal dilator muscles leading to upper airway obstruction. PEEP or CPAP and the appropriate airway support should be utilised in the anaesthetised infant. The diaphragm is the predominant respiratory muscle in neonates but is more easily fatigable than in adults. Ventilation under anaesthesia should be at least assisted and abdominal distension should be avoided.

Respiratory distress syndrome (RDS), bronchopulmonary dysplasia (BPD) and chronic lung disease (CLD) occur in the premature neonate due to the immaturity of lung development, surfactant deficiency and adverse lung mechanics, compounded by ventilator induced lung injury and oxygen toxicity. Strategies to avoid BPD include use of CPAP, surfactant, HFOV, avoiding volutrauma, hypocarbia and reducing oxygen exposure[22].

Oxygen transport. Infants have a high metabolic requirement for oxygen (6-8ml/kg/min vs 4-6ml/kg/min in adults). Tissue oxygen delivery is achieved by a relatively high cardiac output (300ml/kg/min vs 60-80ml/kg/min in adults) and high respiratory rate (30-40bpm). However, oxygen transport oby haemoglobin shows developmental changes with time.

Fetal haemoglobin (HbF) is suited to the hypoxic conditions found during fetal life. It has a low P50 (18-20mmHg vs 27mmHg in adults) and allows effective tissue oxygenation of the fetal tissues. However, at birth HbF still forms 70-80% of total haemoglobin - relatively poor tissue oxygenation is compensated for by a relatively high

haemoglobin concentration. HbA2 production increases from birth, being the dominant haemoglobin by the first few months of life. The P50 continues to rise during infancy to levels higher than found in adults, probably reflecting increased levels of[2,3] diphosphoglycerate (2,3 DPG) during a period of rapid growth. Coupled with a relatively high cardiac output, tissue oxygen delivery is extremely efficient in infants compared to adults. These factors affect the triggers for transfusion or the haemoglobin level at which a child should be considered significantly anaemic:

	P50 (mmHg)	Hb required for equivalent tissue oxygen delivery (g/dl)		
Neonate<2 months	24	17.6	14.7	11.7
Infant >2 months	30	9.8	8.2	6.5
Adult	27	12	10	8

Hepatic function and drug handling.
The liver in the newborn infant contains 20% of the hepatocytes found in adults and continues to grow until early adulthood. The liver is the principle site of drug metabolism, some evidence of which can be found in fetal life, albeit at low levels. Phase I processes (metabolic, e.g. the cytochrome P450 system) are significantly reduced at birth whilst phase II processes (conjugation) may be well developed (sulfation) or limited (glucuronidation). Paracetamol is excreted by sulfation in the neonate, glucuronidation in adults. In general, drug effects are prolonged in neonates and drugs should be titrated to effect, given by bolus rather than infusion, or plasma levels monitored as appropriate. Maturation of enzymatic processes increases over the first few weeks of life and the half-life of drugs such as morphine reaches adult levels at 2 months of life. However, infants require significantly less morphine than older children, especially in the first week of life[7]. Plasma protein binding is reduced in neonates (low levels of a1-acid glycoprotein) and drugs that are plasma protein bound (such as local anaesthetics) may demonstrate increased toxicity in infants.

Children have been described as 'therapeutic orphans' in that many drugs, especially new drugs, have not been studied in this age group - hopefully this will be rectified in future.

Infants have reduced hepatic stores of glycogen and immature gluconeogenic enzyme systems. Coupled with a high metabolic rate, this makes them susceptible to hypoglycaemia following starvation. Blood sugar should be measured during surgery and glucose containing solution continued if the child has been hypoglycaemic or receiving parenteral nutrition preoperatively

Renal function[8]
Nephrogenesis is completed at 36 weeks gestation and no further nephrons are produced (impaired nephrogenesis in premature infants has been related to hypertension in adult life). Further increase in renal mass is due to the growth of tubules. The GFR at term is low and reaches adult values only at 2 years of age. Renal autoregulation of blood flow is functioning in neonates, albeit at lower levels of blood pressure. Creatinine at birth reflects the mother's creatinine and falls to reflect renal function of the infant by 1 week of age. Tubular function matures over the first few months of life; infants usually produce urine that is isotonic to plasma, but if required, can concentrate their urine to achieve an osmolality of 500-700mOsmol/kg H_2O. Adult values (urinary osmolality typically 1200-1400mOsmol/kg H_2O) are reached by a year of age. Infants tolerate fluid restriction poorly. The neonate's limited renal function is appropriate to the period of rapid growth after birth - growth has been termed the 'third kidney'. However, in the postoperative (catabolic) infant, renal insufficiency may become apparent and the neonate does not handle fluid or sodium overload.

Fluid and electrolyte balance[9]
The extracellular fluid compartment is expanded in neonates, with total body water representing 85% of body weight in premature babies, 75% of body weight in term babies, compared to 60% body weight in adults. Contraction of the extracellular fluid compartment and weight loss in the first few days after birth is a normal physiological process, due in part to a diuresis induced by atrial naturetic peptide (ANP) secondary to increased pulmonary blood flow and stretch of left atrial receptors. After this period of negative water and sodium balance, water and sodium requirements increase to match those of the growing infant. Fluids should therefore be restricted until the postnatal weight loss has occurred. Liberal fluid regimens in the first few days of life have been shown to be associated with worse outcomes in premature infants (increased patent ductus arteriosus, necrotising enterocolitis and death)[24].

Of interest to anaesthetists, the expanded extracellular fluid compartment results in an increased volume of distribution of commonly used drugs and increased dose requirements, despite increased sensitivity (muscle relaxants, intravenous induction agents).

Temperature control[5]

Thermoregulation in the neonate is limited and easily overwhelmed by environmental conditions. Heat production is limited and there is a greater potential for heat loss (high body surface area to body weight ratio, increased thermal conductance, increased evaporative heat loss through the skin). The newborn infant is able to vasoconstrict to reduce heat loss and to increase heat production through brown fat metabolism (non shivering thermogenesis, inhibited by volatile agents), however this is at the expense of increased oxygen consumption and the possibility of increased complications. The preterm baby is particularly vulnerable in this respect as the immature skin is thin and allows major heat (and evaporative fluid) losses. The principle of anaesthesia in these infants is close liaison with the neonatologist and minimal handling. Surgery is frequently performed in the neonatal unit for this reason.

Central nervous system, nociception and the stress response

The brain forms 10% -15% of body weight at birth, only 2% of body weight by the age of 8 years. The brain is reliant on glucose for metabolism but the child is also able utilise ketones under normal conditions. The CMRO2 is higher in young children due to the demands of growth and autoregulation of cerebral blood flow is present, even in premature neonates.

The lower limit for cerebral autoregulation in neonates is not known, but is thought to be around a cerebral perfusion pressure of 30mmHg. Appropriate mean arterial blood pressure for premature neonates are controversial but it generally accepted that the mean arterial pressure equates to the gestational age of the child during the first day of life, rising to a minimum of 30mmHg by 3 days.

Survival of extreme preterm infants has improved considerably in recent years, but this has been associated with high levels of disability. Marlow et al followed the progress of the cohort of children born in the UK at less than 25 weeks during 1995. At 30 months of age, 24% of survivors had severe disabilities; at 6 years of age, 21% had severe disability, and when compared with their classmates, 41% had significant cognitive impairment[20]

A major determinant of cerebral impairment is intraventricular haemorrhage (IVH), particularly complicated by ventricular enlargement, parenchymal infarction or cystic periventricular white matter injury (PVL). Major IVH usually occurs within the first few days of life. Factors that have been shown to reduce the incidence of IVH or later neurodevelopmental delay include: delayed delivery to allow the administration of prenatal maternal steroids, postnatal surfactant to reduce

lung disease, neuromuscular paralysis to avoid ventilator asynchrony, avoidance of hypotension or fluctuating blood pressure, avoidance of morphine infusions in hypotensive infants, early indomethacin to encourage duct closure in PDA, and the avoidance of postnatal steroids. It has been estimated that there is one additional case or cerebral palsy for each 7 infants treated with dexamethosone. Avoidance of postnatal dexamethosone is currently the single most important factor to improve neurological outcomes in premature infants[21]

Developmental aspects of pain[10]

Neonates, including premature neonates, show well developed responses to painful stimuli. Indeed, the foetus shows a stress response (and behavioural changes) to nociceptive stimulation from 18-20 weeks gestation, which can be attenuated by the administration of fentanyl[12]. It has long been known that attenuation of the stress response to surgery improves postoperative morbidity and mortality.

The neonatal period is characterised by marked sensitivity to sensory stimuli of all types, with low thresholds of response to mechanical and noxious stimulation. The nociceptive responses of neonates are significantly different to adults; at birth, a noxious stimulus (eg heel prick), will elicit an exaggerated movement of the whole body and movement of all four limbs. These responses are less pronounced after 29-35 weeks post conception, but repeated stimulation results in sensitization.

The process of maturation of the nociceptive system is complex and involves interactions between the peripheral and central nervous systems, changes in receptor, ion channel and neurotransmitter expression and the effects of neurotrophins. Experimental evidence has shown widespread, functional opioid receptors in the spinal cord of newborn animals (rather than located to lamina I and II of the spinal cord as in adult life). It appears that there is a great deal of neuronal fine tuning during early neonatal life which may be influenced by the activity of endogenous opioids. However, this raises the question of the long term effects of exogenous morphine administration to neonates at the time of neuronal plasticity[11].

Long term effects of early pain experiences[23]

Animal and human work has indicated that early pain experiences can have long term effects, possibly through developmental changes in the nociceptive circuitry. For instance, painful procedures induce behavioural changes in preterm infants in NICU[13] Infants who undergo circumcision without analgesia show exaggerated responses to later immunisation compared to controls[14]. Infants who undergo major surgery in the first three months of life exhibit greater analgesic requirements, stress response and pain scores when undergoing repeat

surgery compared to controls that have not had previous surgery[15].

Long term effects of early exposure to anaesthetic agents

Recent work from Olney et al has investigated the effects of exposure of the developing brain to drugs that block NMDA receptors or potentiate GABA receptors. Anaesthetic drugs commonly used in paediatric practice (midazolam, nitrous oxide, isoflurane) were administered to 7 day old rats for 6 hours. They were found to cause widespread apoptosis (programmed cell death) with deficits in hippocampal synaptic function and persistent memory/learning impairments[16]. Similar findings had been previously noted with ketamine[17]. The relevance to clinical practice is at present unclear - indeed, surgery without effective anaesthesia and pain relief would equally have significant adverse effects. Others have questioned the experimental conditions used and found that smaller doses of ketamine that those used by Olney do not induce apoptosis in rat pups[18]. The jury is still out and animal models may not accurately reflect the situation in humans, but clearly, there is room for more research![19]

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Erratum: In Update in Anaesthesia number 19, PERIOPERATIVE FLUIDS IN CHILDREN:

Page 36 (page 1 of article) paragraph 2 should read:
Breast milk may be given to within 3 hours, formula milk to within 4 hours.

Page 37 (page 2 of article), paragraph 2: The word "in-vitro" should be substituted for "in-vivo".

SERVICING A SEIZED OXFORD MINIATURE VAPORISER
Dr Mike Dobson, Nuffield Dept of Anaesthesia, Oxford, UK.

The Oxford Minature Vaporizer (Penlon Ltd) is widely used, suitable for drawover use and very popular in the developing world. It is simple, robust, and can be used to deliver almost any volatile anaesthetic agent, mostly used with halothene.

Like any piece of equipment, it needs servicing from time to time. As with much apparatus, keeping it clean is one of the best ways of looking after it. This article deals with the problem of accumulation of materials inside the vaporizer, which make it increasingly difficult to move the control pointer.

It is inevitable that some dirt and dust from the atmosphere gets drawn into the vaporizer during normal use; you may be able to reduce this by fitting a dust filter on to the air entry point of your breathing system, but make sure it is a coarse filter with low resistance, as the patient has to breathe through it. However clean the air, over the course of time, residues from the anaesthetic agent will accumulate inside the vaporizer, and it is these sticky residues that are the main problem. They consist of substances added by the manufacturer to the anaesthetic agent I order to protect it from undergoing chemical change. Halothane is the most reactive of volatile anaesthetics, and has the largest amount of preservatives and anti-oxidants added to it. (An OMV used only with isoflurane will be a lot less sticky than one used with halothane).

If your vaporizer is getting stiff, a 'quick-fix' is to insert a bung on the inlet side of the OMV, trun the vaporizer on to its side and pour in some ether, methylated spirits or halothane whilst moving the pointer to and fro. If you put another bung on the other side you can give the vaporiser good shake without spilling liquid. Leave the vaporizer to stand for 5 minutes before emptying. If you are using the ether or halothane perform this procedure outdoors to avoid inhaling the fumes. Empty the fluid out of the vaporizer and blow it through with air for 10-15 minutes.

This is only a short term solution - in order to clean out the sticky residues properly, there is no alternative but to partly dis-assemble the vaporizer and clean it by hand. The instructions below are taken from the manufacturer's service manual. Do not start unless you have had some training, and ideally have had a trained person demonstrate the process in front of you.

Let's assume you are confronted with an OMV on which the pointer control is either very sticky, or completely stuck.

Before you start, find a clear, well-lit space to work in. Sweep the floor first, and it will then be much easier to find any small items that you accidentally drop. Spend five minutes looking carefully at the vaporizer, comparing it with the pictures below, and identifying the parts. Have a pencil and paper so that you can draw what you see as you work - this will make it much easier when you have put things back together. Have a small dish or saucer to store parts like screws that can roll away and get lost.
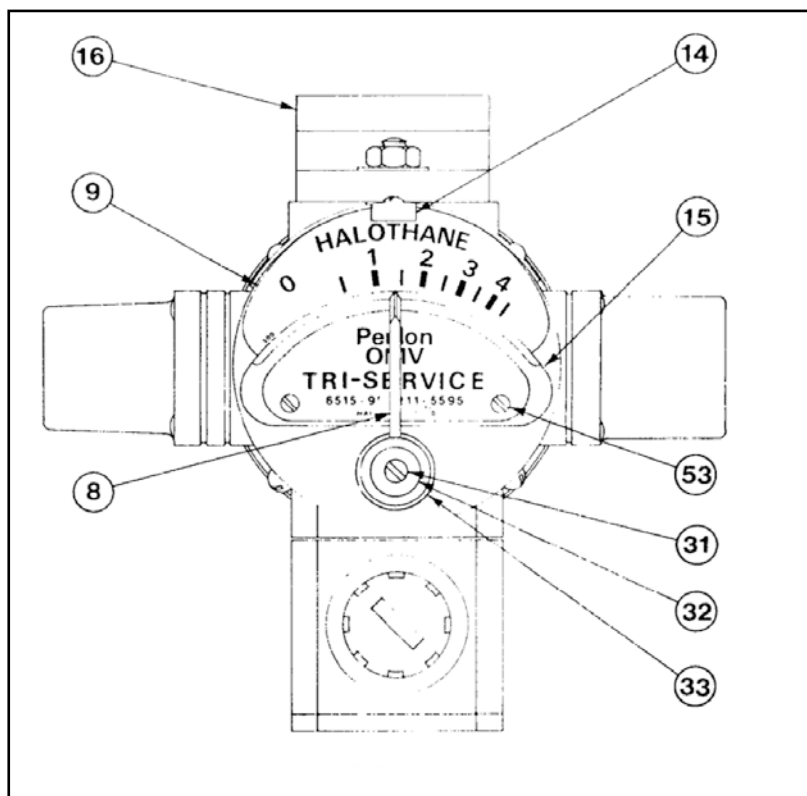
Figure 1

What you have to do:
Most air going through the vaporizer passes straight across the top, but a small, variable amount is diverted into the vaporising chamber. The by-pass tube has holes at the bottom, which are party or totally occluded by the metal cylinder (slider) that slides from side to side under the control of the pointer. Sticky residue between the cylinder and the by-pass channel makes it hard for the cylinder to move inside the channel. You have to take the cylinder out, clean it and the inside of the by-pass tube, and put it back. You do not need to do anything inside the vaporising chamber itself. No special tools are needed - just a set of small screwdrivers. The whole procedure can be completed in about half an hour.

1. *Remove the pointer* by unscrewing the screw (31) and washer (32), then lift off the pointer and the washer beneath it (33). Remove the scale, which is held in place wither by a clip (shown) or 2 small screws. Note the engraved 'degree' scale underneath the halothane scale - you will need to refer to this later.

2. *Remove the lid* - take out 2 screws (53) on the top and 3 around the edge of the lid, and carefully lever the lid off the body.

3. Once you have removed the lid the vaporiser will look like this. Put the pointer back on for a moment, and observe how the cog and rack transmit movement to the sliding cylinder in the by-pass channel. The vaporiser pictured below has a right-to-left flow (which is normal for draw-over use) but some exist with a left-to-right flow, and these have an extra cog wheel mounted on a plate. Identify which sort of vaporiser you have - the principles of working on them are the same.
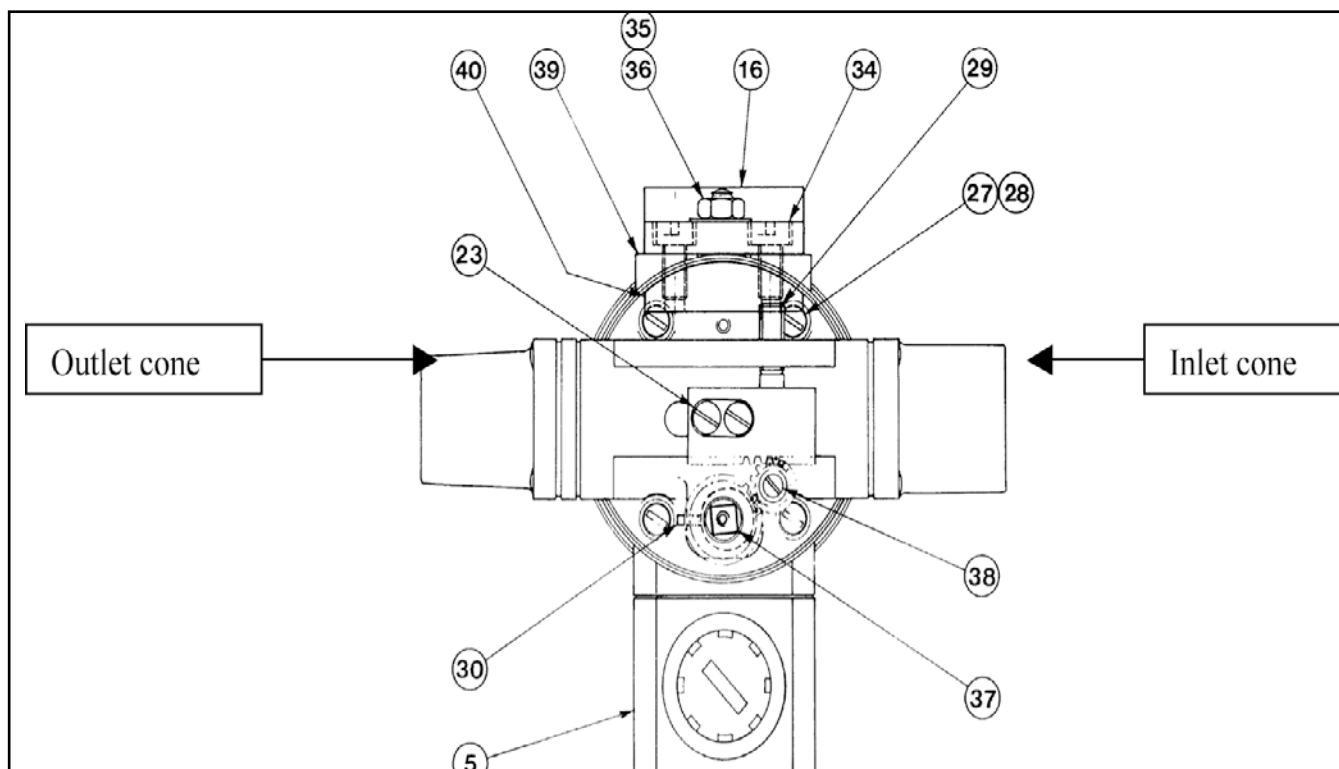

Figure 2

4. *Dismantle the regulator assembly:*

Remove the inlet & outlet cones (4 screws each (25 % 26) and obturator assembly (19).

Notice that the obturator can only be fitted to one end of the vaporiser, because of the small locating pins on the vaporiser body.

5. *Remove 2 screws* (23) *securing the rack* (20). First make a mark across the point where the cogwheel and rack teeth meet (this will help you to reassesmble). Lift off the rack and spacers. Retain the plastic sleeves inside the spacers. Note the relative positions of the ports and the direction of the internal cone of the slider.

Figure 3

Mark here

Rack

Cogwheel

Inlet port
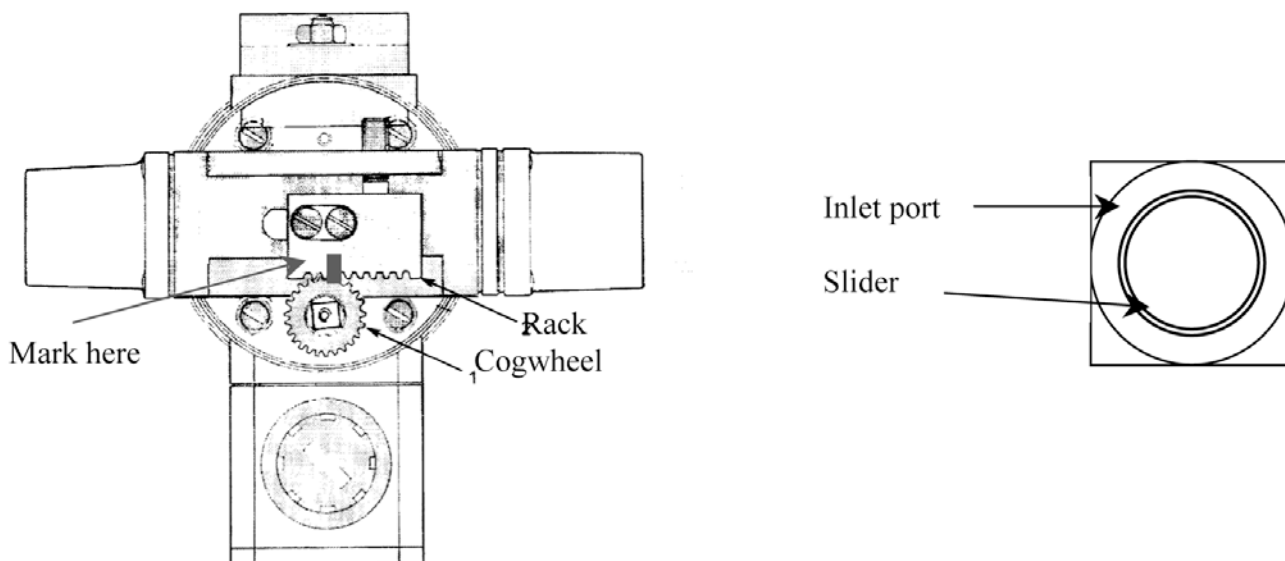
Slider

6. *Remove the slider valve* by pushing from the outlet end of the regulator housing. If it is stiff, use a piece of wood or plastic to push it, so as not to damage the metal surfaces. If you cannot move the slider, apply some penetrating oil around the edge inside the bypass channel. Do this with the vaporizer on its side, so the oil can sweep down.

7. If you do not have penetrating oil, use a 50:50 mixture of sewing machine or bicycle oil and ether. Leave the vaporiser like this for 2 hours - if it still will not move, apply more oil at the other end. Notice as you take it out that there is a right and a wrong way to put the slider back ! Check the picture below to see how the inside of the slider relates to the obturator.

8. *Soak the assembly* in ether or alcohol to dissolve deposits, then wash the slider valve using cleaning liquid and a soft cloth. NEVER use abrasives such as sandpaper. You may use metal polish ("Brasso") to remove stubborn dirt, but make sure you remove all polish residues.

9. *Before re-assembling, lubricate* the moving surfaces with a VERY small amount of "Vaseline" (petroleum or petrolatum jelly). Do not use oil or other lubricants as they dissolve in halothane!

10. *Re-assemble the regulator* assembly by reversing step 5. Check the correct location of ports, direction of cone, and reassemble with spacers and crews, lining up the marked teeth on the rack.
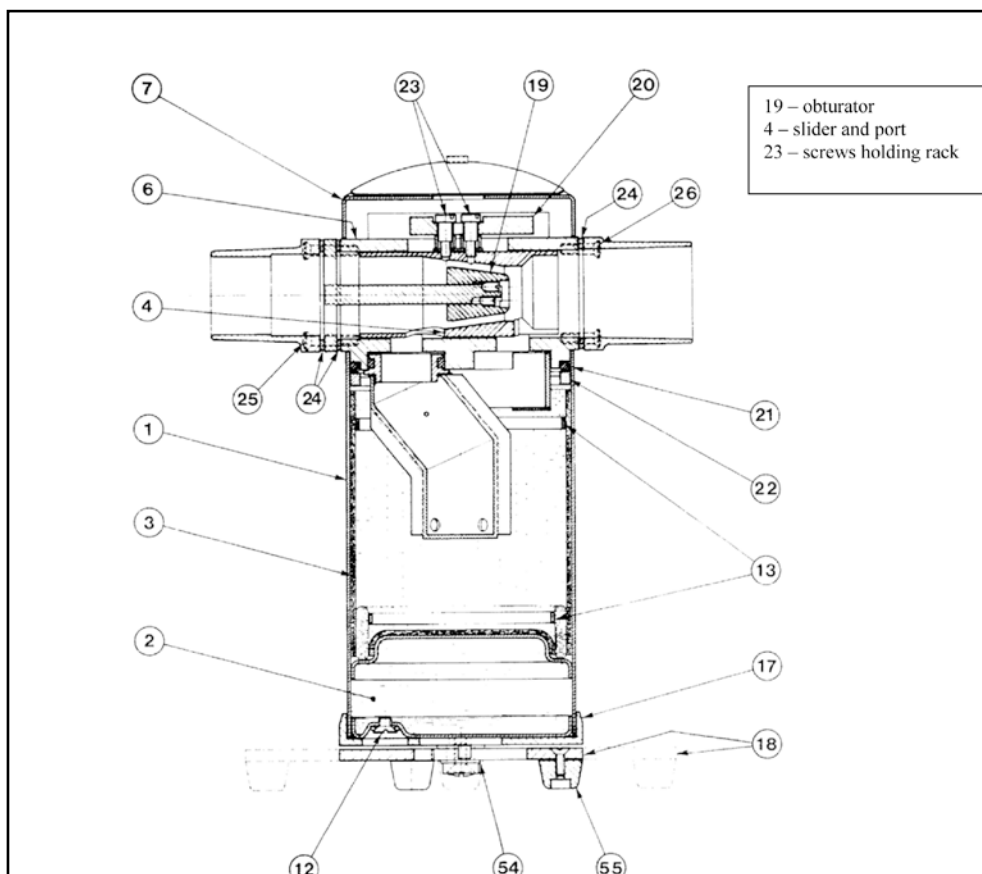

19 – obturator
4 – slider and port
23 – screws holding rack

Figure 4

11. *Reset the vaporiser* The relative positions of the slider and pointer may have moved. To reset you will need a special gauge, which you can obtain from the manufacturer, but if you have a vernier gauge, hacksaw, and file it is easy to make your own from a piece of thin metal sheet:

Figure 5: Setting Gauge.

(Measurements in inches. $^1/_8$inch = 0.125 inches = 3.175mm)

12. If you have marked the cog/rack as suggested in step (5), you can miss out the rest of this step, as you can realign them accurately in their original positions, and go to step (14). Otherwise, before replacing the lid, loosely reattach the pointer to check that the cog and rack are in roughly the right positions. The pointer should have a range of movement corresponding to where the scale will go. Now replace the lid and check the exact setting using the special tool. With the pointer set at maximum concentration, insert the calibration tool at the air inlet and let it slide along the 'floor' of the passageway until it drops into the port leading down to the vaporising chamber.
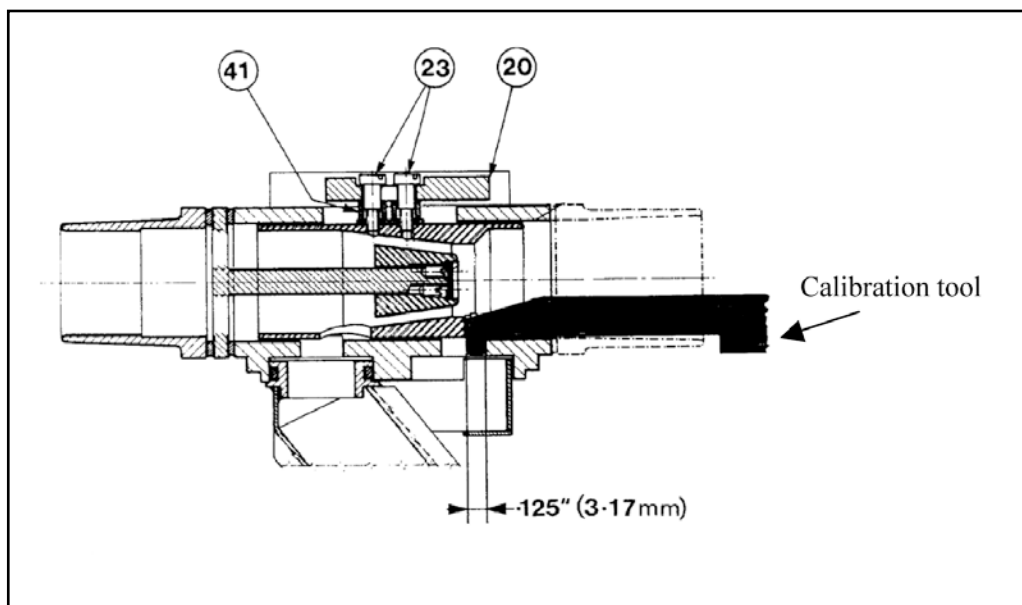
13. Gently turn the concentration pointer "down" until the sliding valve lightly grips the tip of the gauge. The pointer should now be at 35° as shown on the scale engraved on the top of the vaproiser. If not, you have not replaced the pointer or cog in the right place, and will need to readjust it. The mark you made in step 6 should help you avoid this error.

If the pointer does not point at 35°, you need to re-align the cog and rack - if you are one 'tooth' out, the pointer will be 7.5° away from 35°, if 2 cogs, 15° away etc.

14. Complete the re-assembly of the vaporiser lid, scale, pointer and mountings. The lid screws are particularly fiddly and short, so use a suitable small screwdriver and be very careful not to cross-thread them.

The complete Penlon workshop manuals for both the EMO and OMV vaporisers are on the "Anaesthesia Resource 2" CD which is available free of charge from "TALC" (www.talcuk.org). Unfortunately due to a technical error the OMV manual is not accessible from the "index" page of the CD, but you can find it easily by carrying out a search/ find command for "OMV".

ANSWERS TO MCQS

1.

F CPD-A (citrate, phosphate, dextrose, adenine) is used to anti coagulate and preserve donated blood collected into sterile plastic bags.

F SAG M (saline, adenine, glucose and mannitol) gives blood a shelf life of up ton 35 days.

F Red blood cells should be stored at 2-6 degrees celsius

T Levels of 2,3 DPG fall rapidly with increasing duration of storage and this contributes to the reduced oxygen carrying capacity of blood when it is transfused

T Red cell lysis causes an increasing potassium level in stored red cells

2.

F MAC is the minimum alveolar concentration-(of volatile at equilibrium) at which 50% of subjects do not respond to a standard surgical stimulus (at STP)

F It is inversely proportional to the oil-gas solubility and

F not related to the blood-gas solubility coefficient- this relates to speed of onset

F The MAC of halothane is usually quoted as less than 1.0, often as 0.7-0.8

T

3.

F Cricoid pressure was described by Sellick

F The experimentally suggested pressure is around 35 Newtons

T It could result in oesophageal rupture if applied during active vomiting

F It will not prevent tracheal soiling in pharyngeal pouch- the pouch could be emptied prior to induction by the patient or using a nasogastric tube

F It was first described in obstetric anaesthesia

4.

F Fibreoptic intubation can be impossible in the presence of bleeding

F It can be useful in upper airway obstruction and subsequent difficult airway

T Many operators use sedation to facilitate awake fibreoptic intubation

F It may be useful in this situation

T

5.

F Mapleson A and D (Lack and Bain) circuits are used co-axially, not B

F The Mapleson D is seen in many anaesthetic rooms as its co-axial version as the Bain circuit. The Mapleson C has been used for resuscitation eg in A&E

F Mapleson D circuits are actually more efficient when used for controlled ventilation (require < 1 x minute volume) rather than spontaneous.

F The Ayres T piece requires 2-3 x minute volume

T The Lack is a co-axial Mapleson A.

6.

F HELLP syndrome (Hypertension, elevated liver enzymes, low platelets) is associated with pre-eclampsia. The low platelets can be a relative contra-indication to regional anaesthesia/ analgesia, depending on the actual level.

F This is an elevated platelet level. Patients may be on aspirin or other drugs affecting platelet function.

T Clopidogrel should be ceased 7-10 days prior to interventions such as epidurals as it will give a high risk of bleeding and could cause epidural haematoma.

F If HELLP syndrome is not present.

F This is a relative contra-indication, depending on clinical need and condition.

7.

T The radial nerve lies behind the axillary artery in the axilla. Transfixion of the artery technique can be more reliable.

F Interscalene blocks, whilst valuable in shoulder surgery, are known to often leave areas of the hand unanesthetised.

T This is probably due to the close relationship of the intercostal vessels to the nerves.

T

T The supine approach (Raj), the anterior (Beck), the posterior (Labat) and the lateral (Ichiangi).

8.

F It is suggested that sciatic nerve blocks are performed without adrenaline, to avoid compromise of the vascular supply of the vulnerable sciatic nerve.

F This is the Labat approach.

T True.

F The femoral nerve should also be blocked.

F Only the posterior approach will reliably block this nerve.

9.

T

F It is 3 compressions to 1 ventilation.

F They are performed just below the internipple line with either 2 finger compressions or using the thumbs to compress with the hands encircling the chest.

T A pink baby scores 2, a baby with some peripheral cyanosis 1, a pale or grey baby 0.

T This is true. Good oxygenation and ventilation should normally improve the heart rate in neonates.

10.

T They will not predict all difficult intubations.

T It should be more than 6.5 cm.

F It is usually an overbite that suggests a more difficult intubation.

T

F This is not possible, although many can be predicted with careful clinical examination and use of old notes!

11.

F There is an increase in stroke volume more than heart rate.

T This is true, due to progesterone-related airway dilatation.

F Acidity does not increase.

F Gastric emptying is delayed in labour.

F Total blood volume increases.

12.

F Usually they are Ig M and do not.

T It can also cause fever and anaemia.

F The figure is closer to 70%.

F Rhesus antibodies require exposure from an external source- eg Rhesus immunisation in pregnancy.

T This is true- eg sample labelling, errors in patient label checking.

13.

T

F The formula is age/4 plus 4, so for a 6 year old a size 5.5 tube would be expected.

F It is 10mcg/ kg which is 0.01mg/kg

F In infants it is more often 2mg/ kg.

F Propofol is not recommended as a long-term infusion for children after a numder of deaths in intensive care related to accumulation of fatty deposits from lipids used in the propofol formulation.

14.

T This is to maintain cerebral perfusion against an increased intracranial pressure.

F CO_2 is usually maintained at the lower end of the normal range.

T True. The tube is therefore often taped into position.

F The preferred position to try and reduce ICP is to have the patient up to 30 degrees head up, to encourage venous drainage.

F Mannitol is usually reserved for delaying imminent coning rather than first line treatment.

15.

F This is not.

T This is often taken as a reliable clinical sign of adequate reversal.

T TOF ratio should be 0.7.

T This is true.

T Vital capacity has been used.

16.

T Only the desflurane vaporizer can be refilled like this.

F Plenum vaporizers have a high resistance. Drawovers do not.

F It is a drawover.

T This is because the vaporizer must be heated for adequate function.

T The Tec 6 is the desflurane vaporizer.

17.

T

T

T

F This is used to analyse oxygen levels, other gases are diamagnetic.

F

18.

T There is distribution of heat from core to periphery.

T There is also heat loss from evaporation and convection and some conduction.

F Heat will continue to be lost for longer than this.

F A great deal of energy is required for the body to warm and humidify dry cold gases, hence the widespread use of HME filters.

F Thermistors are used, thermocouples are not normally used clinically.

19.

T

F There is an increase in systemic vascular resistance, giving hypertension with intravascular depletion.

T

T

The association with HELLP syndrome and low platelets means that the full blood count should be checked to ensure platelet levels are safe for epidural insertion.

20.

T By carbon monoxide substituting for oxygen in haemoglobin, as it has a much greater affinity. This can occur in heavy smokers.

F 12 hours is the suggested time frame.

T

T Causing sympathetic stimulation, tachycardia etc.

T This is apparently true.

Dear Reader

If you would like to receive **Update in Anaesthesia** please write to: Mrs Carol Wilson, Pound Cottage, Christow, Exeter, EX6 7LX, UK. The fax number is + 44 1392 402472. Alternatively, you can contact Carol by email carol@world-anaesthesia.org

When writing please include your name, address, email (if available), your title and role, and a few details about your hospital and work. If you would like extra copies of Update to distribute to other anaesthetists, please let us know how many you require, and the names of the readers.