Editorial

Estimating the demand for any publication naturally includes a large element of guesswork. When the potential readership is scattered among the most inaccessible parts of the globe, as in the case of Update in Anaesthesia, this factor becomes almost impossible to quantify.

Now that the first edition has been circulated and initial reactions have been assessed, it is already obvious that our first estimate of 1,000 copies per edition was too low. So many encouraging letters have already been received from anaesthetists in the developing world asking for extra copies that an additional 2,000 copies are now required for each edition. The editors of the journal ‘Tropical Doctor’ have kindly agreed to circulate copies with their next issue, so that a wider readership can be reached. The Janssen Laboratories have made a generous donation and the Overseas Development Administration of Great Britain have adopted Update as a project. There have been requests for French and Spanish translations, and it is hoped this challenge can be met.

Readers can help to contain distribution costs by ordering in bulk and arranging their own distribution locally, so that as many people as possible can benefit from this publication. Ideally this will be done through the Society of Anaesthetists within the country concerned.

As editors, we need to know if you would like to receive the journal, to which organisation we can send it in your country and what subjects you would like to see covered. The more criticisms and suggestions received the better the journal will become. Please write to Dr Iain Wilson, Department of Anaesthetics, Royal Devon & Exeter Hospital (Wonford), Exeter, EX2 5DW, Great Britain. We look forward to hearing from you!

Dr Iain Wilson

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Ruptured ectopic pregnancy is a common cause of massive intraperitoneal haemorrhage in developing countries. Many patients present with hypovolaemic shock, since considerable time may have elapsed between rupture and arrival at hospital. The mortality associated with hypovolaemic shock is very high in these countries due to the scarcity of donor blood available for transfusion.

This article is based on our experience in a district hospital in Malawi where we transfused the patient’s own blood (autotransfusion) collected from the peritoneal cavity in 25 cases of ruptured ectopic pregnancy. All of them survived without adverse effects.

**Method of autotransfusion**

After attention to the airway and breathing the hypovolaemic patient is rapidly infused with 0.9% sodium chloride solution or Ringer’s lactate on the way to the operating theatre where general anaesthesia is induced. The sterilised equipment for autotransfusion is prepared ready for use. On opening the abdomen a small initial peritoneal incision is made. The peritoneum is “tented” to avoid spillage (figure 1).

If the intraperitoneal blood appears fresh and of normal colour it is collected in small containers. After removing most of the fresh blood the peritoneal incision is enlarged and the bleeding site clamped. A few layers of sterile gauze are stretched over a large bowl into which the collected blood is filtered through the layers of sterile gauze (figure 2). This retains small blood clots and tissue.

The distal end of a giving set is cut off and dipped into the blood in the bowl. The proximal end is inserted into the rubber stopper of a transfusion vacuum bottle containing 120ml of acid-citrate/dextrose solution as anticoagulant (figure 3).

Once the bottle is full the blood is immediately retransfused into the patient via a standard blood-giving set (figure 4). An alternative method is to pour the blood into a jug and then through the gauze filters.
directly into the bottles (containing the anticoagulant), replace the rubber stopper and transfuse.

To avoid bacterial growth, blood for autotransfusions should be given at once and not stored. The equipment must be sterile.

2. Embolism, DIC or other coagulopathies
Although air and particle embolism were reported in early attempts at autotransfusion these were not encountered in our series. Emphasis is placed on thorough filtration of the recovered blood in order to prevent small particles from causing pulmonary embolism or disseminated intravascular coagulation and for this reason at least 3 layers of gauze should be used. There are no coagulation factors in the blood when it is retransfused and depletion of these will occur during the process.

Advantages
1. Safety
There is no risk of transfusion reactions caused by mismatching. Transmission of blood-borne diseases such as hepatitis, HIV, malaria, syphilis and tuberculosis is eliminated.

2. Availability
In contrast with donor blood, the patient's type of blood is instantly available and requires no cross matching. The technique is also suitable for patients with other causes of uncontaminated intraabdominal bleeding such as a ruptured spleen or liver and in cases of haemothorax.

3. Simplicity
The method of blood recovery is simple and practical even in remote hospitals. It does not require sophisticated equipment, electricity or storage facilities. Since there is no need for laboratory investigations such as typing and crossmatching, autologous blood is more quickly reinfused than donor blood.

If acid-citrate/dextrose is not available, it can be prepared by mixing 2 grams of sodium citrate and 3 grams of dextrose in sterile water to make a total volume of 120mls, which is sufficient for 1 unit of blood.

4. Acceptance
In some cultures blood donation is the source of fear or taboo, in others the infusion of blood is prohibited by religion. Therefore autologous blood transfusion may be a useful method of overcoming some of these objections.

Risks and complications
In a life-threatening situation, the advantages of autotransfusion far outweigh the potential disadvantages. The disadvantages include:

1. Infection or sepsis
Blood for transfusion should be discarded if there are signs of intraperitoneal infection such as an associated tubo-ovarian abscess. Blood contaminated by bowel contents or amniotic fluid from a ruptured uterus should not normally be used. Blood from the thoracic cavity may be used, provided the same precautions are taken.

There are many variations of emergency “salvage” autotransfusion. For example blood may be recovered from the peritoneal cavity with the aid of gentle suction (<40mmHg) before filtration. Another method utilizes a cell-saver which separates the red blood cells from plasma and white blood cells before retransfusion, but this is an expensive technique.

Figure 4
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Hypertension is a common disease and patients with this condition frequently present for surgery. Most hypertensive disease is idiopathic (no cause known), but around 10% of patients suffer from hypertension caused by renal, endocrine or pregnancy related disease.

Hypertension is usually diagnosed after a series of blood pressure measurements reveal pressures greater than 160/100. Due to the increasing rigidity of the arterial circulation, systolic blood pressure rises with age. In patients over 65 years a systolic of 170 to 180 is reasonable, if the diastolic pressure is normal. Single blood pressure readings are often misleading, as the process of hospital admission and blood pressure measurement may lead to a false, temporary elevation.

When dealing with hypertensive patients the following facts should be remembered:

The disease is usually symptomless but if untreated, hypertension may result in heart enlargement and failure, renal dysfunction and cerebrovascular accidents. It is important to look for such complications, as they may influence the choice of anaesthetic technique. The presence of renal disease, for example, may alter the choice and dosage of anaesthetic drugs, and evidence of recent myocardial ischaemia may necessitate delaying elective surgery.

Deaths related to hypertensive cardiovascular disease are reduced if the disease is adequately treated. Medication should be taken regularly and frequent review is essential.

Severe untreated hypertension (a diastolic pressure >120mmHg) in the perioperative period may lead to serious complications, such as myocardial infarction, left ventricular failure, cerebral haemorrhage, hypertensive encephalopathy, and renal failure.

Patients with untreated or inadequately treated hypertension develop marked swings in blood pressure with situations such as anaesthesia, blood loss or pain. They may undergo a profound fall in arterial pressure in response to induction and maintenance of anaesthesia. They also exhibit an exaggerated hypertensive response to stimuli such as laryngoscopy and intubation. They are prone to develop cardiac dysrhythmias and ischaemia during anaesthesia.

If diastolic pressure measured pre-operatively does not exceed 110mmHg, there is little evidence of increased cardiac complications.

Elective surgery

The blood pressure should be assessed before operation using the correct size of blood pressure cuff. If raised, it should be measured several more times over a few hours with the patient resting. An examination may reveal whether the hypertension has caused any cardiac, renal or neurological complications. Examination of the fundi may reveal vascular changes if there is severe hypertension. If available a full blood count, urea and electrolytes estimation, ECG and a chest X-ray are useful to help assess cardiac and renal function.

Patients with significant hypertension (diastolic pressures >110mmHg) should not undergo elective surgery until their hypertension has been adequately controlled. Usually this will take a few days. Some drugs used in the treatment of hypertension have anaesthetic relevance, and these are highlighted in table 1.

Patients with well-controlled hypertension should normally continue their medication up to, and including, the day of surgery. Premedication with benzodiazepines (eg diazepam 10-20mg, temazepam 20-30mg, or lorazepam 2-4mg) two hours prior to surgery will help to allay anxiety. Atropine should be avoided if possible, because of its tendency to cause tachycardia.

Adequate monitoring should be started prior to induction of anaesthesia. Continuous pulse measurement and frequent arterial pressure assessment are important. An ECG is useful to detect ischaemia and dysrhythmias. For major surgery, central venous pressure monitoring and measurement of urine output may be useful.

Anaesthesia

The aim is to ensure circulatory stability during surgery. All antihypertensive drugs should be taken preoperatively to assist in blood pressure control during surgery and recovery. Many drugs used in the treatment of hypertension are vasodilators or cardiac depressants. Since anaesthetic agents also have these actions, the effects on the circulation may be more noticeable.

Induction. Care should be taken not to cause a precipitous fall in arterial pressure at induction. The use of opioids, such as morphine or fentanyl, will reduce the amount of induction agent required. Thiopentone may be used provided it is given slowly, and titrated against response. Ketamine, which raises the arterial pressure and heart rate, is best avoided.
If tracheal intubation is necessary, the hypertensive response to laryngoscopy can be reduced by the use of intravenous opioids, lignocaine 1mg/kg intravenously, and an adequate depth of anaesthesia.

Maintenance. The use of opioids, which have minimal cardiovascular effects, will reduce the amount of volatile agents required. High concentrations of volatile agents can cause hypotension by decreasing the systemic vascular resistance and by depressing the myocardium. Nitrous oxide can be safely used. Local anaesthetic nerve blocks or infiltration are useful either on their own or to supplement general anaesthesia.

Hypertension during anaesthesia may reflect inadequate depth of anaesthesia, hypoxia or hypercarbia (raised blood carbon dioxide level due to inadequate ventilation). These factors should be corrected before treating blood pressure with antihypertensive drugs such as labetolol or hydralazine. If specific antihypertensive drugs are not available, small iv doses of chlorpromazine (around 5mg) may help.

Hypotension should be vigorously treated by reducing the depth of anaesthesia (if it is excessive), and correcting any hypovolaemia. Bradycardia should be treated using iv atropine. Remember that patients on beta blockers are particularly liable to develop bradycardia, and may develop marked cardiac depression with all anaesthetic agents including ether and ketamine. Occasionally, a small dose of a vasopressor may be required in patients not responding to this approach.

Normal intravenous fluid replacement should be given. Hypertensives tolerate hypovolaemia poorly.

Recovery. Coughing on the tracheal tube during emergence will increase arterial pressure. Opioids given during anaesthesia reduce this tendency. Hypertension may develop during the recovery phase. Hypoxia or inadequate breathing may be the cause. If the hypertension is due to bladder distension, a urinary catheter is indicated. If it is caused by inadequately treated pain, analgesics should be administered and the patient reassured. If no cause can be found, hypertension can be treated as suggested for emergency surgery (see below).

The patient should be returned to the ward only when the circulation is stable.

Regional anaesthesia
Spinal and epidural anaesthesia can cause unpredictable and profound arterial hypotension in poorly controlled hypertensive patients. Well controlled hypertensives, however, have a more predictable response. Local blocks e.g. brachial

<table>
<thead>
<tr>
<th>Mechanism of action</th>
<th>Examples</th>
<th>Relevance to anaesthesia</th>
</tr>
</thead>
<tbody>
<tr>
<td>Diuretics</td>
<td>Hydrochlorothiazide</td>
<td>May produce hypokalaemia resulting in dysrhythmias</td>
</tr>
<tr>
<td></td>
<td>Frusemide</td>
<td></td>
</tr>
<tr>
<td>Vasodilators</td>
<td>Hydralazine</td>
<td>Tachycardia (unpredictable when given IV)</td>
</tr>
<tr>
<td></td>
<td>Diazoxide</td>
<td></td>
</tr>
<tr>
<td>Central sympathetic depression</td>
<td>Clonidine</td>
<td>Rebound hypertension if withdrawn.</td>
</tr>
<tr>
<td></td>
<td>Methyldopa resepine</td>
<td>Slow acting.</td>
</tr>
<tr>
<td>Adrenergic neurone blockers</td>
<td>Guanethidine</td>
<td>Sensitive to vasopressors. Postural hypotension.</td>
</tr>
<tr>
<td>Beta blockers</td>
<td>Propranolol</td>
<td>Avoid in asthmatics and patients with heart failure. Cause bradycardias which usually respond to atropine.</td>
</tr>
<tr>
<td></td>
<td>Atenolol</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Labetalol (alpha also)</td>
<td></td>
</tr>
<tr>
<td>Alpha blockers</td>
<td>Phenoxybenzamine</td>
<td>Tachycardia.</td>
</tr>
<tr>
<td></td>
<td>Phentolamine</td>
<td></td>
</tr>
<tr>
<td>Calcium channel blockers</td>
<td>Nifedipine</td>
<td>Vasodilator. Cardiac depressant - avoid combining with beta blockers as extreme hypotension can occur.</td>
</tr>
<tr>
<td></td>
<td>Verapamil</td>
<td></td>
</tr>
<tr>
<td>Renin - angiotensin inhibitors</td>
<td>Captopril</td>
<td>Potentiate hypotensive action of anaesthetic drugs.</td>
</tr>
</tbody>
</table>

Table 1. Classification of drugs used in the management of hypertension
plexus blocks or ankle blocks, should always be considered in hypertensive patients, as the potential hazards of general anaesthesia are thereby avoided.

**Emergency Surgery**

If surgery is needed urgently in an uncontrolled hypertensive, efforts should be made to control the blood pressure before induction. Treat pain and anxiety with appropriate medication. The drugs listed in Table 2 may be used to carefully reduce the blood pressure to around 160/100. However, these drugs can cause unexpected hypotension which may result in stroke, blindness and myocardial ischaemia. The drugs should be given gradually, in small increments, while continuously assessing their effect.

During emergency surgery in untreated hypertensives, maintain careful fluid replacement. Note that a moderately low blood pressure in a normal patient (e.g., 90-100 systolic) may reflect more serious hypotension in the hypertensive.

### Table 2. Drugs used for emergency control of hypertension

<table>
<thead>
<tr>
<th>Drug</th>
<th>Action</th>
<th>Length of action</th>
</tr>
</thead>
<tbody>
<tr>
<td>Labetalol 10-200mg IV</td>
<td>Alpha &amp; beta blocker</td>
<td>1-4 hours</td>
</tr>
<tr>
<td>Propranolol 1-4mg IV</td>
<td>Beta blocker</td>
<td>1-2 hours</td>
</tr>
<tr>
<td>Hydralazine 5-20mg IV</td>
<td>Vasodilator</td>
<td>3-6 hours</td>
</tr>
<tr>
<td>Nifedipine 10mg sublingual or oral</td>
<td>Calcium channel blocker</td>
<td>2-5 hours</td>
</tr>
<tr>
<td>Diazoxide 30mg boluses IV (max 300mg)</td>
<td>Vasodilator</td>
<td>4-12 hours</td>
</tr>
</tbody>
</table>
Introduction

Caesarean section is a surgical procedure commonly performed in rural hospitals in developing countries. Those responsible for providing anaesthesia for such surgery must be fully aware of the basic principles involved.

Caesarean sections can be performed under general anaesthesia, regional anaesthesia or local infiltration. This article will consider only general anaesthesia: regional and local techniques will be described in a subsequent article.

When selecting the type of anaesthetic to be used, the anaesthetist must take into consideration his or her own experience and that of the surgeon, the condition of the patient, the degree of urgency and the availability of equipment and drugs. General anaesthesia has the advantage of speed, but introduces the risk of airway complications including aspiration of gastric contents or failed intubation. Regional anaesthesia, such as spinal or epidural block, avoids these risks, but takes longer to perform, and may cause undesirable hypotension secondary to peripheral vasodilation.

Physiological changes in late pregnancy

In the later stages of pregnancy some substantial physiological changes occur. The most important ones and their significance to the anaesthetist are:

1. When the patient lies on her back the pregnant uterus compresses the vena cava and the aorta and obstructs blood flow. Compression of the former leads to a diminished venous return and a fall in maternal cardiac output, and this together with the compression of the aorta will reduce the blood flow to the uterus, with undesirable effects on the fetus. In some women this “aorto-caval” compression may lead to hypotension (supine hypotensive syndrome).

2. There is diminished tone in the lower oesophageal sphincter and in later pregnancy the raised intra-abdominal pressure and altered gastro-oesophageal angle make gastric reflux more likely. In labour the administration of opioids markedly slows gastric emptying.

During induction of anaesthesia passive regurgitation of stomach contents into the pharynx may occur, and lead to aspiration pneumonia. This is likely if the pH of the stomach contents is less than 3 (very acidic) and more that 30 mls of fluid is aspirated. The mother may be protected from this complication by using local anaesthesia instead of general anaesthesia when possible, by reducing food intake in labour to minimise stomach contents and by a careful rapid sequence induction of anaesthesia using cricoid pressure (see figs 2 & 3). The stomach acid should also be neutralised if a Caesarean section is planned. An intravenous H2 blocker such as ranitidine or cimetidine is effective but takes an hour to work. An antacid such as 30mls of 0.3 Molar sodium citrate is reliable if given immediately before induction of anaesthesia and will last about 1 hour. A combination of both methods will protect for several hours.

The conscious patient can respond by improving her position, but under general anaesthesia this is impossible. Spinal or epidural anaesthesia considerably worsens the problem due to the sympathetic blockade produced.

By tilting the patient to the left by about 15 degrees the pressure from the uterus on the vena cava is reduced. This can be achieved by tilting the operating table or by placing a wedge under the patient’s right buttock (figure 1).

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Some anaesthetists pass stomach tubes prior to induction of anaesthesia to empty the stomach. Although this may remove some fluid it is rare for them to empty the stomach effectively, as solid material cannot generally be removed.

3. There is a greater increase in plasma volume than red cell mass causing dilutional anaemia. Cardiac output is increased. Patients with cardiac disease (either congenital or valvular) are at particular danger during pregnancy, due to their inability to adapt to these changes.

4. There is a decrease in the resting lung volume caused by pressure from the enlarged uterus, and there is an increase in the basal metabolic rate. Oxygen reserves are therefore diminished and hypoxia develops rapidly if airway problems occur.

5. Many drugs used in anaesthesia cross the placental barrier and may affect the fetus, particularly opioids such as morphine and sedatives such as diazepam. During anaesthesia these drugs should be avoided until the umbilical cord has been clamped.

Although this article is concerned with anaesthesia for Caesarean section, the general principles discussed are applicable to other operations in the pregnant patient.

Preparation. Caesarean sections are frequently performed as emergencies in unprepared patients. The procedure may be complicated by an unfasted patient, fetal distress, severe haemorrhage, pre-eclampsia etc. Prepare and check equipment for obstetric anaesthesia in advance, so that your apparatus and drugs are immediately to hand. This saves valuable time in an urgent case. Particular attention should be paid to the function of the laryngoscopes, the endotracheal tube and cuff, and the suction apparatus. As intubation may be difficult, it is a wise precaution to have an introducer and a smaller size of endotracheal tube ready. A trained assistant must be available at induction.

A relevant anaesthetic history is obtained from the patient and clinical examination carried out paying particular attention to the cardiovascular and respiratory systems. Any likely intubation problems should be identified. The blood pressure is measured and the haemoglobin result checked. It should be confirmed that blood has been sent for crossmatch and will be available for emergency transfusion if required.

The patient is positioned with the table tilted or with a wedge under the right hip. This should produce a lateral tilt of at least 15 degrees which helps to prevent aorto-caval compression. This position must be maintained until delivery. A large intravenous cannula is inserted and a reliable infusion established. An adequate supply of intravenous fluids should be available in case they are required at short notice. If there is clinical evidence of hypovolaemia (low blood pressure, rapid thready pulse, cold peripheries) this should be corrected with intravenous fluids prior to induction. Patients with Pregnancy Induced Hypertension (PIH) or eclampsia may require treatment for their high blood pressure prior to induction. Increments of hydralazine 5mg or labetalol 5-10mg intravenously may be given at 5 minute intervals until the diastolic pressure has been reduced to around 90-100mmHg. It should be remembered that beta blockers are contra-indicated in asthma.

Induction of general anaesthesia
The patient should be pre-oxygenated with 100% oxygen via a close fitting face mask for 3 minutes before induction. Thiopentone 3-5mg/kg or ketamine 2mg/kg is then injected, followed by suxamethonium 1.5mg/kg. Cricoid pressure is applied by the assistant as consciousness is lost and must be maintained until the anaesthetist is satisfied that the airway is secure. When the patient is fully relaxed intubation is performed. The lungs are not normally inflated by face mask prior to intubation as this may force gas into the stomach, raise the intragastric pressure and promote regurgitation. Only when the anaesthetist has confirmed the placement of the endotracheal tube and the cuff has been inflated is cricoid pressure released. If intubation cannot be performed, however, facemask ventilation will be necessary to maintain oxygenation. This situation is termed “failed intubation”. Always have a plan available in case this happens - a suitable plan is discussed at the end of this article.

Maintenance of anaesthesia
Anaesthesia can be maintained with a 50% mixture of nitrous oxide and oxygen, supplemented with a low concentration of a volatile agent in order to avoid the possibility of awareness. Halothane 0.5% is suitable. High concentrations of volatile agents should be avoided as they may decrease uterine tone increasing bleeding at operation and they may depress the neonate.
Further relaxation can be achieved by increments of suxamethonium (remember to give atropine before a second dose of suxamethonium) or the use of a non-depolarising relaxant. Most non-depolarising relaxants do not cross the placenta to any great extent, except gallamine which should be avoided until after the cord is clamped. After delivery oxytocin 10 units or ergometrine 500 micrograms is injected intravenously to contract the uterus. Ergometrine should be avoided in the presence of hypertension as it causes a rise in blood pressure. Once the umbilical cord is clamped an opioid such as morphine (5-15mg) can safely be given slowly intravenously. At this point the inspired oxygen concentration can be reduced to 30-35%.

In situations where no nitrous oxide is available an increased concentration of halothane (around 1%) should be given in oxygen until delivery. After the cord has been clamped an intravenous opioid should be administered and the concentration of halothane reduced to minimise relaxation of the uterus.

In many places diethyl ether is the main anaesthetic agent available, and it is also suitable for caesarean section. The patient may be paralysed and ventilated with a mixture of air, oxygen and 2-3% ether. Where muscle relaxants are in short supply many anaesthetists allow the patient to breathe air, oxygen and ether spontaneously following intubation. 4-6% ether is required for this purpose, although a higher concentration is needed initially until the patient is settled. If a spontaneously breathing technique is used it is wise to assist ventilation to improve the efficiency of respiration.

At the conclusion of surgery muscle relaxation is reversed, (or in the case of suxamethonium allowed to wear off), and the patient turned on to her left side in the head down position. The endotracheal tube is removed only when laryngeal reflexes have returned and spontaneous respiration has resumed. Oxygen is administered by face mask for at least 30 minutes following surgery, during which time the patient should remain on her side. The intravenous infusion is continued into the post-operative period to ensure adequate hydration and to retain venous access. Analgesia is prescribed, usually in the form of an opiate such as morphine or pethidine.

In extremely difficult circumstances
In some countries there are many anaesthetists working without oxygen or endotracheal tubes. However they are still faced with the challenge of providing anaesthesia for caesarean sections. People working in these difficult environments recommend the use of local infiltration anaesthesia or regional block (both of which will be covered in a future edition). Some anaesthetists attest to the use of ketamine by intravenous bolus injections, and others administer ether via a facemask. It is likely that techniques using local anaesthesia are the safest, providing proper precautions are taken, and the anaesthetists and surgeons are skilled in their use. Although several of these techniques are unconventional, careful attention to basic anaesthetic principles, such as airway care, monitoring of the colour and circulation, will ensure the safest anaesthetic available under the circumstances.

Failed intubation drill
A clear plan must be available in the event of failed intubation. There is a serious risk of hypoxia if the situation is mishandled. An appropriate course of action is as follows:

**Maintain cricoid pressure**
**Oxygenate using the facemask**

Turn the patient on to the left side into a head down position and allow her to wake up. Proceed with local anaesthetic block when the patient has regained consciousness.

If the operation is needed very urgently (eg for fetal distress or an antepartum haemorrhage), re-establish spontaneous respiration after the suxamethonium has worn off, and continue the anaesthetic under a facemask using nitrous oxide, oxygen and halothane or an ether based technique. If possible maintain cricoid pressure during the anaesthetic. If problems are encountered with the airway, it may be necessary to wake the patient up and use a regional technique.

At all times ensure that the patient is well oxygenated.

**Other measures**
If available a laryngeal mask may be useful for maintaining the airway in the event of a failed intubation. If severe airway obstruction develops during a failed intubation and none of the usual airway manoeuvres regains the airway, a cricothyroidotomy should be performed using a large intravenous cannula (at least 16 gauge). This should be connected to the anaesthetic circuit (use a Portex 3.5mm connector or other similar connector) and 100% oxygen delivered directly into the trachea, until the patient wakes up. This equipment should always be available for this event.
Digital nerve blockade is simple and easy to perform and provides useful analgesia for a variety of minor surgical procedures. The technique is essentially the same for fingers and toes.

Anatomy
In the hand, the common digital nerves are derived from the median and ulnar nerves and divide in the distal palm into paired volar (or palmar) branches. These run with the digital vessels on either side of the flexor tendon sheath of each finger and supply the lateral and palmar aspect of each finger together with the tip and nail bed area. The smaller dorsal digital nerves, derived from the radial and ulnar nerves, run on the dorsolateral aspect of each finger and supply sensation to the back of the finger.

In the foot, the digital nerves are the terminal branches of the tibial and peroneal nerves which, in their turn, are branches of the sciatic nerve.

Method
The patient’s hand is placed palm down and the skin cleaned. A 25g needle is inserted into the dorsal aspect of the web space between the fingers as proximally as possible and close to the phalanx (figure). The needle is advanced through the tissues until just below the skin on the palmar side.

After aspirating to ensure that a vessel has not been entered, 1-2ml of local anaesthetic is injected to block the volar branch and, as the needle is withdrawn, a further 0.5-1 ml is injected to block the dorsal branch. The nerves on the radial aspect of the thumb are best blocked by a subcutaneous wheal of local anaesthetic injected at its base.

Either 1% plain lignocaine or 0.5% plain bupivacaine (or other equivalent agent) can be used. On no account should adrenaline-containing solutions be used. The digital arteries are end arteries and ischaemia or necrosis can occur if adrenaline is injected.

The anatomy of the digital nerves in the foot is similar and a similar technique can be used to block them.
Anatomy

Penile block has been widely used for circumcision. Complications include inadequate block or, rarely, ischaemia. Techniques vary from injection below the symphysis pubis to ring block of the shaft.

The anatomy related to penile block is one of the least understood areas by anaesthetists. The key points are:

1. The triangular space lying deep to the fascia, bounded above by the symphysis pubis and below by the corpora cavernosa.

2. The fact that the fascia splits on its deep surface to form a vertical suspensory ligament of the penis which, in turn, divides to encircle the shaft of the penis.

3. The dorsal nerves and vessels lie deep to the suspensory ligament where it divides on the corpora cavernosa and are therefore in an enclosed space where they could be depressed if a large haematoma developed.

4. There are pear shaped, potential spaces on either side of the suspensory ligament which usually do not communicate directly (only 6% do).

Method

The safest technique is to inject an adequate volume of local anaesthetic bilaterally deep to the fascia into the pear shaped spaces on each side of the suspensory ligament. This avoids mid-line injection and therefore potential damage to the dorsal vessels and provides the maximum chance of diffusion into the nerves to block them. Injecting an adequate volume of local anaesthetic (estimated in children at 1ml + 0.1ml/kg on each side) should ensure that the ventral branch is also blocked so that a satisfactory block is achieved.

The technique involves inserting the needle until it touches the pubic symphysis. This gives a guide to depth. The needle is then withdrawn and redirected to pass below the symphysis and 3-5 millimetres deeper depending on the size of the patient. It is preferable to direct it slightly laterally into the pear shaped space and then to re-insert in on the other side depositing equal volumes on each side. Avoiding the midline injection reduces the chance of penetrating the dorsal vessels of the penis and causing haematoma. If a short beveled needle the fascia may be felt as a slight resistance when it is penetrated, but in small children this is not always felt as it is thin and may offer little resistance.

Figure 1. A lateral view of the penis in sagittal section showing the triangular space bounded by symphysis pubis, the membranous layer of superficial fascia and the corpus cavernosum. The needle should be inserted to hit the symphysis pubis (1) and then directed below it through the fascia into the space (2) where the local anaesthetic is deposited to block the dorsal nerves of the penis. X-X shows the section shown in Figure 2.

Figure 2. A transverse section through the triangular space shown in Figure 1 (X-X). This shows the membranous layer of superficial fascia and the suspensory ligament of the penis which divides to form another triangular space fusing with Buck's...
fascia through which pass the dorsal nerves, arteries and veins. The site of insertion of the needle is shown passing through the membranous layer of superficial fascia. The bilateral injections into the potential spaces shown allow the local anaesthetic to diffuse into the space with the nerves with minimal chance of damage to the dorsal nerves, arteries and veins.

The site of insertion of the needle is shown passing through the membranous layer of the superficial fascia. The bilateral injections into potential spaces shown allow the local anaesthetic to diffuse into the space with minimal chance of damage to the dorsal arteries. If an adequate volume of local anaesthetic is used the ventral branch which supplies the frenulum should be blocked. If in doubt a subcutaneous ring of local anaesthetic may be injected around the ventral side of the shaft of the penis.

This block is useful for circumcision and provides some post operative analgesia. It avoids potential problems of caudal anaesthesia for circumcision such as leg weakness or difficulty with micturition.

Bupivacaine or lignocaine are widely used for this block. It is vital that adrenaline containing solutions are never used, as severe arterial vasoconstriction can be produced causing ischaemia or necrosis of the penis. Bupivacaine may provide excellent post operative analgesia for up to 6 hours.

The diagrams accompanying this article were originally published in Anaesthesia and Intensive Care. We are grateful to the editor for allowing us to reproduce them.
Introduction
This is the first of two articles describing the maintenance of sphygmomanometers. The first deals with mercury type machines, and the second aneroid gauges.

A mercury sphygmomanometer is operated by inflating a rubber cuff placed around a patient’s arm until blood flow stops. The cuff pressure is measured via the mercury column.

The figure shows the parts of a mercury sphygmomanometer. The inflating bulb is used to inflate the cuff. It contains two one-way valves. Valve A allows air to enter the back of the bulb. When the bulb is squeezed this valve closes and the air is propelled through valve B to the cuff. Valve B stops the air going back into the bulb.

2. The cuff must be free of leaks.
3. The mercury should be clean and at the zero mark before use.
4. During cuff inflation the mercury should rise smoothly, and stop immediately inflation stops.

Problems with the mercury sphygmomanometer
Remember mercury vapour is poisonous. Any maintenance should be performed in an area of good ventilation. Store mercury in a plastic bottle with a little water placed on top of the mercury. Be careful not to inhale black mercuric oxide powder during cleaning procedures.

Black discoloration of the mercury. The mercury should be a clean silver colour. With time, a black powder (mercuric oxide), forms on the surface. A little black powder in the column does not matter. If there is a large amount, the mercury should be removed from the sphygmomanometer and the column and reservoir cleaned. Do this by laying the machine on its side with the reservoir downwards. Remove the column, ensure that you do not lose the leather disc at the top. Undo the reservoir top and pour the mercury out into a plastic bottle. Blow the reservoir and the leather disc in the reservoir top clean with compressed air and wipe with a cloth. (If you don’t have compressed air, take an old blood pressure inflation bulb, find a large bore needle, file off the tip and fit it to the inflation bulb. By squeezing the inflation bulb you will have a source of compressed air). Clean the inside of the column and replace it. Remember to replace the leather disc at the top and the washer at the bottom.

Replace the mercury in the reservoir to the zero mark. Use a syringe and needle to draw up the mercury from the plastic bottle. Keep the needle under the surface of the mercury to avoid returning any black powder. Replace the reservoir top with its sealing ring, connect the cuff and check the system is airtight by inflating the cuff until the mercury is at the top of the column. Check that it does not spill out—if this happens, the top leather disc is faulty or missing. (This disc should allow the air to pass in and out as the mercury rises and falls, but not allow mercury to escape.)

Mercury continues to rise slowly after stopping inflation. This is caused by the air at the top of the column failing to escape through the top leather disc quickly enough as the mercury rises up the column. When the sphygmomanometer is used, this fault may
result in abnormally high readings as the mercury falls more slowly than the cuff pressure, due to the faulty leather disc restricting the air entering the top of the column.

The cause of these faults is in the top leather disc. It is either too thick or dirty. It should be removed as described before and cleaned. Replace it and test the sphygmomanometer. If the fault persists, remove the disc. Holding it between your finger and thumb on a flat surface, gently scrape it with a scalpel blade. Turn it continually to ensure it keeps its round shape and take care not to put a hole in it. Refit.

**The mercury does not rise but the cuff inflates.** This indicates blockage at D in the figure or a kinked or obstructed tube.

**The cuff will not inflate or mercury rise.** This indicates that there is a leak. Check valves A, B and C, the rubber bladder, tubing and connections. Rubber bladders may be repaired with an ordinary bicycle tyre puncture kit. Valve B can usually be removed and cleaned. Valve A may be a small ball bearing which can be removed from its cage and, with care, cleaned and replaced. Valve C cannot normally be removed. After full assessment reassemble the sphygmomanometer and test.
**Preparation.** Sodium thiopentone (also known as thiopental or pentothal) is prepared by dissolving a yellowish powder in sterile water to provide a 2.5% solution (ie 25mg/ml). In this concentration 20mls of solution will contain 500mg. The solution should be used within 24 hours of preparation and kept cool. The solution is alkaline with a pH of greater than 10, and can be irritating and painful if accidentally injected into tissues. Because of the alkalinity, thiopentone should not be mixed in the same syringe as other drugs, as it may cause formation of a cloudy precipitate and inactivate the drug.

**Uses.** Thiopentone is a rapidly acting barbiturate. Its main use is for induction of anaesthesia. Following induction anaesthesia is usually maintained by breathing an anaesthetic vapour such as halothane. Thiopentone can be used as the sole anaesthetic agent for very brief procedures.

Thiopentone can also be used in Intensive Care patients with head injuries to control surges in intracranial pressure. As it posseses potent anticonvulsant activity it may be given to treat epileptic seizures that do not respond to other therapy.

**Main Effects.** An intravenous injection of thiopentone causes loss of consciousness within 15 to 30 seconds, and lasts for 5 to 10 minutes. The onset time of thiopentone is approximately the time it takes for the drug to travel from the vein in the arm to the brain. Induction of anaesthesia with thiopentone usually results in two or three deep breaths followed by a short period of breath-holding.

**Dose.** Healthy adults and children require around 3 to 7 mg/kg of thiopentone. Dose requirements are reduced following premedication, in the elderly and in those with compromised circulations (see adverse effects).

**Administration.** After 1-2 mls of thiopentone have been administered ask the patient whether the injection is painful. Pain would suggest extravascular or intra-arterial injection. Titrate the dose against effect; the loss of the eyelash reflex is a good guide to loss of consciousness. After an injection of thiopentone always flush the cannula with a little saline to reduce the chance of forming precipitations with the next drug.

**Advantages.** Thiopentone causes a rapid, smooth induction of anaesthesia, with little excitation or apnoea. Return of consciousness after thiopentone is rapid, with prompt return of airway protective reflexes.

Thiopentone decreases cerebral metabolism and cerebral blood flow. It does not have any direct toxic effects on the liver or kidney, but patients with liver or kidney disease may require a lower dose range than 3 to 7 mg/kg. Although it crosses the placenta it is a safe agent for induction in obstetric anaesthesia.

**Adverse effects.** Thiopentone directly depresses the contractile force of the heart, it increases heart rate, coronary blood flow, and the oxygen demand of the heart. Thiopentone also causes a decrease in venous tone, causing pooling of blood in the peripheral veins; this can cause hypotension in patients who are hypovolaemic (eg following haemorrhage).

Thiopentone has no analgesic properties, in fact in low doses it tends to heighten sensitivity to pain. It has poor muscle relaxant properties.

Although thiopentone has a relatively brief duration of action after a single dose, if repeated doses or an infusion of thiopentone are given the drug accumulates, and the more a patient is given, the longer it will take for him to wake up.

**Contraindications:**
1. Acute intermittent porphyria
2. Barbiturate allergy
3. Patients with a low circulating blood volume, such as after haemorrhage, are prone to severe hypotension with thiopentone.
4. Patients with cardiac disease (particularly those with stenotic heart valve lesions) are at risk from the cardiovascular depressant effects of thiopentone. The drug must be carefully titrated against effect.
5. Patients with partial airway obstruction should not be given an intravenous anaesthetic agent in case total airway obstruction develops.
6. In severe asthma it is thought that thiopentone may occasionally cause bronchospasm.

**Metabolism.** For many years it was thought that the short duration of action of thiopentone was due to its rapid metabolism. It is now known that thiopentone is metabolised quite slowly, and the rapid recovery is due to redistribution of the drug firstly into muscle and skin, and later into body fat stores.

Thiopentone is metabolised in the liver; less than 1% of the drug appears in the urine unchanged.
Anaesthesia affects respiratory function in different ways. Knowledge of respiratory physiology is necessary to understand these effects.

Physiological control systems involving the nervous system usually have three components. These are:

• a central controlling area
• an afferent pathway and
• an efferent pathway.

The neurones (nerve cells) of the controlling area integrate the information from other parts of the body and produce a coordinated response. This response from the central controlling area is carried to the various organs and muscles along efferent pathways. The input to the central controlling area is from the various sensors via the afferent pathways.

Central Controlling Area

The central controlling area for breathing, called the respiratory centre, is in the lower part of the brain stem, in the medulla oblongata. There are “inspiratory neurones” which are active during inspiration and inactive during expiration. Other neurones are active during expiration but not inspiration—the “expiratory neurones”. These two groups of neurones automatically maintain a rhythmic cycling pattern of inspiration and expiration. This automatic rhythm can be modified by the afferent information.

Afferent Supply

(1) Central chemoreceptors. Chemoreceptors are cells that respond to chemical stimuli. There are cells in the floor of the fourth ventricle (part of the brain stem) that respond to the acidity of the cerebrospinal fluid (CSF) and the output from these cells influences breathing. The acidity of any fluid is measured by the pH; this is related to the number of hydrogen ions in the solution. The normal pH of the body is 7.4; values higher than this represent alkaline conditions in the body, with a lower hydrogen ion concentration, and values of pH less than 7.4 represent acidic conditions, with a higher hydrogen ion concentration. The cells in the floor of the fourth ventricle respond to the pH of the CSF. An acidic CSF causes hyperventilation this is the reason for dyspnoea with conditions such as diabetic ketoacidosis. An alkaline CSF inhibits the respiratory centre. Carbon dioxide in the blood can rapidly diffuse across into the CSF, and there is a balance between the level of carbon dioxide, hydrogen ion and bicarbonate ion in the CSF. If the carbon dioxide in the blood increases (eg following exercise), then the carbon dioxide, hydrogen ion and bicarbonate ion concentrations increase correspondingly in the CSF. This increase in CSF acidity causes hyperventilation which lowers the carbon dioxide concentration in the blood. A low blood carbon dioxide level (hypocarbia) has the opposite effect and may occur, for example, following controlled ventilation during anaesthesia. This may delay the return of spontaneous breathing at the end of surgery.

(2) Peripheral chemoreceptors. The carotid and aortic bodies are small pieces of tissue that contain chemoreceptors which respond to the oxygen and carbon dioxide concentrations in arterial blood. The carotid body is the more important of the two and is situated at the division of the common carotid artery into the external and internal carotid arteries in the neck. The aortic body is found on the aortic arch. The information from the carotid body is carried along the glossopharyngeal nerve (the ninth cranial nerve) and the information from the aortic body is along the vagus nerve (the tenth cranial nerve), to the respiratory centre. The output from the carotid body is thought to provide information to allow immediate regulation of breathing, breath by breath, by the respiratory centre. In normal people, if the arterial blood reaching the carotid body has a partial pressure of oxygen of 10kPa (80mmHg) or a carbon dioxide partial pressure of more than approximately 5kPa, (40mmHg), then there is an immediate and marked increase in breathing. These limits can be modified by disease or age; for example, people with chronic bronchitis may tolerate an increased concentration of carbon dioxide or a decreased concentration of oxygen in the blood.

(3) Brain. Breathing can be influenced by other parts of the brain. We can all consciously breathe deeply and more rapidly (called hyperventilation), and this can happen, for example, before starting strenuous exercise. Intensely emotional situations, for example, distressing sights, will also cause hyperventilation. Hyperventilation is also part of the response to massive blood loss. This response is co-ordinated by the autonomic system in the hypothalamus and the vasomotor centre in the brain stem.

(4) Lung. There are various receptors in the lung that modify breathing. Receptors in the wall of the bronchi respond to irritant substances and cause coughing, breath holding and sneezing. In the elastic tissues of the lung and the chest wall are receptors that respond to stretch. The exact function of these receptors is not fully understood but are thought to be responsible for various reflexes that have been discovered in laboratory
studies of animals. There are stretch responses that occur when the lung and chest wall are distended and inhibit further inspiration. This is an obvious safety mechanism to avoid overdistension. Conversely, when the lung volume is low, then there are opposite reflexes. A small increase in lung size may stimulate stretch receptors to cause further inspiration. This can sometimes be seen in anaesthetised patients who have been given an opioid; spontaneous breathing may be absent or very slow, but if the patient is given a small positive pressure breath by the anaesthetist, then inspiration is stimulated and the patient takes a deep breath. This reflex may also have some function in newborn babies just after delivery, when small breaths may stimulate further inspiration.

There are also stretch receptors in the blood vessels in the lung. If these are stretched, as in heart failure, the response is to hyperventilate. The information from these receptors in the lung is carried to the respiratory centre along the vagus nerve.

**Efferents**

The efferent nerves from the respiratory centre pass down the spinal cord to the diaphragm, intercostal muscles and accessory muscles of inspiration in the neck. The diaphragm is supplied by the phrenic nerve that is formed in the neck from the spinal nerves, C3, 4 and 5. The intercostal muscles are supplied by the segmental intercostal nerves that leave the spinal cord between T1 and T12. The accessory muscles in the neck are supplied from the cervical plexus. During normal breathing, inspiration is an active muscular process. Expiration is passive and relies on the natural elasticity of the tissues to deflate the lung. The most important muscle for inspiration is the diaphragm. Any disease that affects the efferent pathways from the respiratory centre to C3, 4 and 5 and then the phrenic nerve to the diaphragm, may cause severe difficulty in breathing. Trauma to the cervical cord, above C3, is normally fatal for this reason.

**Anaesthetic drugs and respiration**

Opioid drugs, such as morphine or fentanyl, depress the respiratory centre’s response to hypercarbia. These effects can be reversed by naloxone. Volatile anaesthetic agents depress the respiratory centre in a similar fashion, although ether has less effect on respiration than the other agents. Volatile agents also alter the pattern of blood flow in the lungs, resulting in increased ventilation/perfusion mismatch and decreasing the efficiency of oxygenation. Nitrous oxide has only minor effects on respiration.

The depressant effects of opioids and volatile agents are additive and close monitoring of respiration is necessary when they are combined. When oxygen is not available respiration should always be supported during anaesthesia.
A 26 year old healthy male weighing 70kg was scheduled for circumcision under general anaesthesia. He had undergone one previous anaesthetic to allow manipulation of a broken bone.

The anaesthetist induced the patient using thiopentone 350mg. The patient rapidly became very wheezy and developed marked cyanosis despite the administration of oxygen. A student assisting with the anaesthetic commented that the pulse had become very weak and slow, and that a skin rash was developing.

**Questions**

1. What was the most likely diagnosis?
2. What is the management of the condition?

**Answers**

The patient developed acute cardio-respiratory collapse associated with severe bronchospasm, hypotension and a rash. The most likely diagnosis is an anaphylactic reaction to thiopentone. Anaphylaxis is a type of allergic reaction to a drug or other substance to which the patient has been previously exposed. During the previous exposure, an immune response to the substance developed resulting in immunoglobulin E (IgE) being formed against it. IgE binds to specialised immune cells known as “mast cells”. If the patient receives the same substance again, it is detected by the IgE which causes the mast cells to release histamine and other vasoactive mediators. These cause marked vasodilation, increased capillary permeability and smooth muscle contraction.

In a few circumstances patients develop a similar syndrome during the first exposure to a drug. This reaction is not propagated via IgE but through another immune mechanism and is known as an anaphylactoid reaction. Clinically anaphylactic and anaphylactoid reactions are indistinguishable and require exactly the same management.

**Clinical presentation of anaphylaxis**

The cardiovascular system suffers marked vasodilation and considerable plasma loss from the leaky capillaries. This results in tachycardia and hypotension. Occasionally the hypotension may be severe enough to require cardiac massage during resuscitation. The cardiovascular signs may be all that is seen in some patients with anaphylactic shock.

Examination of the respiratory system may reveal bronchospasm, which may be severe. Laryngeal obstruction from oedema can occur.

The skin may feature a raised erythematous type of rash, peripheral oedema (especially around the face) or cyanosis.

Other symptoms may include loss of consciousness, nausea or vomiting and abdominal pain.

**Management**

Patients with anaphylactic shock should recover completely if they are treated immediately. Deaths are usually related to delayed management of hypoxia or hypotension.

1. The airway should be cleared and a high concentration of oxygen administered by facemask. Intubation may be required for laryngeal oedema.
2. If the breathing is inadequate, for example from bronchospasm, the patient should be intubated and the breathing assisted.
3. The circulation should be supported by immediately inserting a large intravenous cannula and rapidly infusing intravenous fluid. Colloids (such as Haemaccel or Dextran) are thought to be more effective than crystalloids in this situation. Large volumes may be required. If a pulse cannot be palpated cardiac massage should be commenced.
4. Drugs. In all serious reactions adrenaline should be given intravenously. In adults give 1 or 2ml boluses of 1:10,000 adrenaline until an effect is seen. Remember that adrenaline only lasts a short time and repeated doses may be necessary. The usual concentration of adrenaline supplied in hospitals is 1:1000 which contains 1mg/ml. To prepare 1:10,000 adrenaline dilute 1ml of 1:1000 with 9mls of saline. (If no venous access is available give 0.5ml of 1:1000 adrenaline intramuscularly, or 10mls of 1:10,000 down the endotracheal tube). Adrenaline is the recommended drug as it will reverse the vasodilation and treat the bronchospasm.

Intravenous hydrocortisone (200mg) is usually recommended but only acts after about 2 hours. Although it has little effect in the emergency situation, it may prove useful with persistent bronchospasm.

Aminophylline (5mg/kg) may be given slowly intravenously if the bronchospasm does not respond to adrenaline alone. Salbutamol may also be used for this indication.

**Antihistamines** are of little use.
5. After the immediate crisis has been managed the patient should be carefully observed in a suitable area of the hospital, for example the intensive care unit or recovery room. They are likely to need continued management on the above lines for some hours.

**Follow up**

The patient should be warned of the problem that developed during the anaesthetic and the drugs used recorded. The patient will then be able to explain the problem to any anaesthetist they meet in the future.

In some centres the patient can be tested to assess which drug caused the reaction, however this is not generally available.

If you have to anaesthetise a patient who has had a reaction to a general anaesthetic in the past but does not know the drugs that were involved check what kind of surgery the patient had and predict the likely technique used. Avoid the drugs which you think might have been used, particularly thiopentone and muscle relaxants. Ketamine, nitrous oxide, volatile agents and local anaesthetics are usually safe.