UPDATE IN ANAESTHESIA

UPDATE IN ANAESTHESIA

WORLD ANAESTHESIA

No 17 2003
ISSN 1353-4882

Editorial

Welcome to Update in Anaesthesia - Number 17!

We are looking forward to meeting any readers who are in Paris at the World Congress of Anaesthesiologists 17 - 23 April 2004. The World Federation of Societies of Anaesthesiologists (WFSA) will have a stand in the exhibition section, which will be a convenient meeting point for members of World Anaesthesia. We hope to have CDROMs containing back copies of Update for distribution for those with computer access.

Update 17 has a variety of articles including a self-assessment section. I am delighted that we have also gained two new Assistant Editors - Keith Allman and Laurie Barker from UK who have helped with this edition.

Please remember to contact us if you would like to submit an article, or would like us to cover any particular theme. A contact address is given on the back page.

If you would like access to a French, Spanish, Russian or Mandarin edition of Update - the addresses are given below.

Happy reading!

Iain Wilson
Editor

Contents: No 17

Contacts

Russian Edition:- Andrei Varvinski, Dept. of Anaesthesia, Torbay Hospital, Torquay, Devon, U.K.
Email: avarvinski@hotmail.com
Website: www.ua.arh.ru

Spanish Edition:- Oscar Gonzales, Rio Parana 445, Bo Felicidad - Lambare, Paraquay
Email: ojgam@conexion.com.py

French Edition:- Michel Pinaud. Service d'anaesthesia, Hotel Dieu, 44093 Nantes Cedex 1, France
Website: www.sfar.org/update/updatechapo.html
Mailing list email: reltringham@clara.net

Mandarin Edition:- Jing Zhao, Dept. of Anaesthesia, Peking Union Medical College Hospital, No. 1 Shuai Fu Yuan, Beijing 100730, Peoples Rep. of China

POSTOPERATIVE NAUSEA AND VOMITING

Dr Gordon Yuill, Dr Carl Gwinnutt, Department of Anaesthetics, Hope Hospital, Eccles Old Road, Salford, Manchester, UK. M6 8HD

Introduction

Postoperative nausea and vomiting (PONV) are among the most common adverse events following surgery, anaesthesia and opioid analgesia. Although usually of minor medical impact, they can cause a lot of distress, lead to delayed hospital discharge and increased use of resources. The aetiology of PONV is multifactorial, involving physiological, pathological and pharmacological factors. It is more common in women than men and in younger patients. It's incidence is difficult to estimate, but may be as high as 60-70%, particularly when the "older" anaesthetic agents are used, compared to around 30% when using anaesthetics such as propofol and isoflurane. Despite the use of such agents, it may remain high in certain high-risk groups of patients.

As there is no "magic bullet" that will prevent PONV in all patients, the clinician should attempt to identify and minimize contributing factors and employ any anti-emetic techniques available to them in those patients considered to be high-risk.

The aims of this article are to:

- Define nausea and vomiting
- Outline the anatomy and physiology of nausea and vomiting
- Identify who is at greater risk of PONV
- Review the pharmacological and other associated techniques used to manage PONV

Definitions

- Nausea is the subjective sensation of the need to vomit.
- Vomiting is a forced expulsion of stomach and gastrointestinal contents through the mouth.

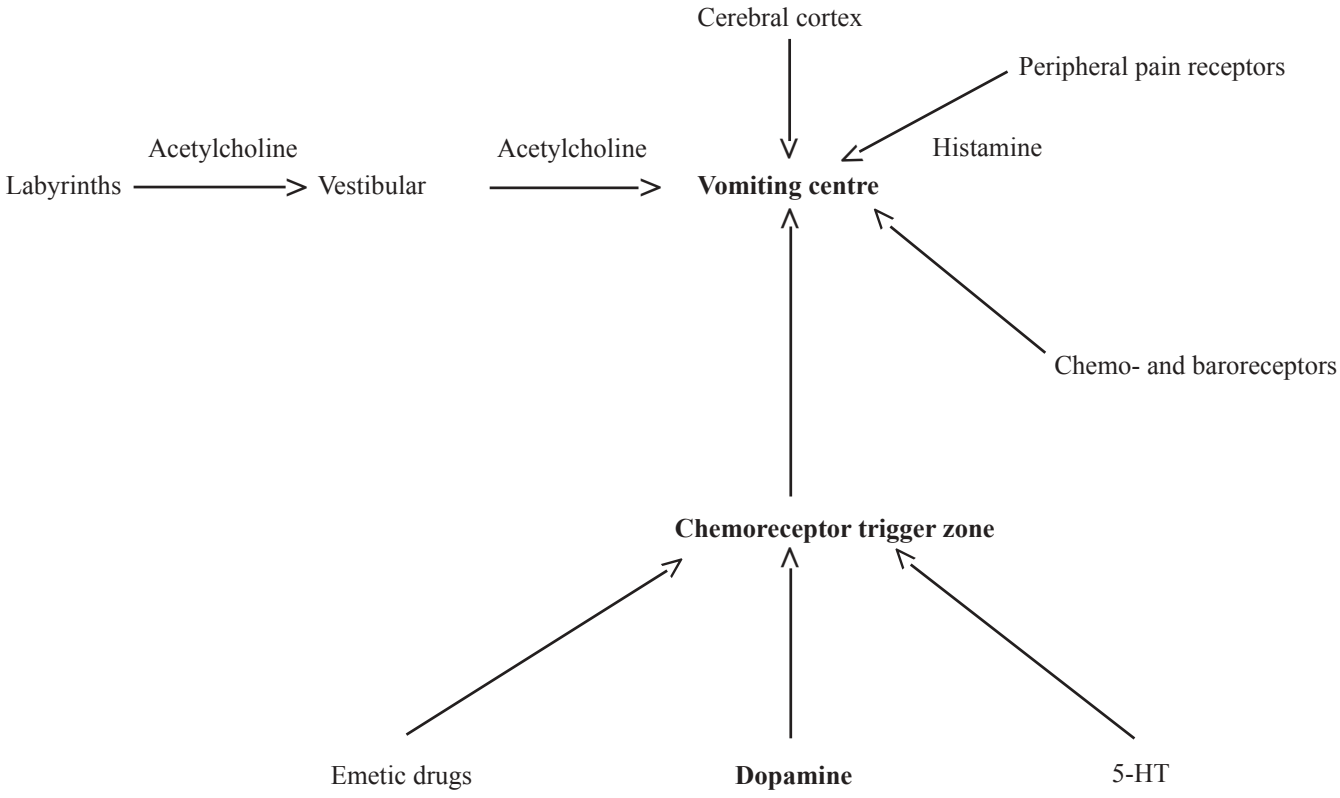


Figure.1 Anatomy and physiology of the vomiting centre and the chemoreceptor trigger zone

Anatomy and Physiology

It is generally accepted that an indiscrete area located in the lateral reticular formation of the medulla, known as the vomiting centre, is responsible for controlling and coordinating nausea and vomiting. A complex range of interactions occurs here, between the reticular formation, the nucleus tractus solitarius and certain autonomic nuclei, especially of the vagus nerve. The centre also receives a wide range of afferent inputs (figure 1); from receptors in the gastrointestinal tract, peripheral pain receptors (responsible for the nausea that may accompany trauma), the nucleus solitarius (involved in the "gag" reflex), vestibular system (involved in motion sickness), the cerebral cortex and the chemoreceptor trigger zone (CTZ). The neurochemistry of the vomiting center is complicated, with some 40 neurotransmitters being implicated. Two that are believed to be particularly important are acetylcholine and histamine hence drugs that antagonize these substances have a central effect on PONV.

The CTZ is a group of cells situated close to the area postrema on the floor of the fourth ventricle. This area is extremely vascular and is situated outside the blood-brain-barrier making it vulnerable to circulating drugs and toxins. It is thought to have a major impact on the activity of the vomiting centre. The CTZ is also sensitive to systemic stimuli and is linked with the control of blood pressure, food intake and sleep. Two important neurotransmitters located here are dopamine and 5-hydroxytryptamine (5-HT, serotonin) and antagonists of these will have an indirect effect on the vomiting center (Figure 1) to reduce nausea and vomiting.

Thus antagonists of four neurotransmitters, acetylcholine, histamine, dopamine and 5-HT, have acquired much interest in the development of the pharmacological treatment of nausea and vomiting, and most of the currently used anti-emetic drugs are antagonists at one of these receptors.

Vomiting is the result of a complex reflex and the combination of the autonomic and motor nervous systems, with efferents from the vomiting center relaying to the vagus and motor neurons that supply the abdominal muscles. The vomiting process starts with deep inspiration, reversed peristalsis moving contents from the upper small bowel into the stomach and an increase in salivation. The glottis closes to protect the airway, the breath is held and the gastric sphincter and oesophagus relax. The muscles of the abdominal wall and thorax contract, and the diaphragm descends vigorously, increasing the intra-abdominal pressure and the gastric contents are ejected into the oesophagus and out through the mouth.

Who is at Risk of PONV?

As already indicated, the aetiology of PONV is multi-factorial. Specific factors that are known to increase the risk relate to:

- The patient
- The type of surgery performed
- The anaesthetic technique used

Patient Factors. The following groups have been identified as having a greater requirement for postoperative anti-emetic drugs:

- Female patients
- Patients with a history of motion sickness
- Patients with a previous history of PONV
- Patients who are non-smokers

Surgical Factors. The following types of surgery are associated with a higher incidence of PONV:

- Gynaecology
- ENT
- Strabismus surgery
- Breast surgery
- Laparoscopy
- Laparotomy
- Craniotomy

Anaesthetic Factors. The following anaesthetic techniques are associated with an increase in PONV:

- The administration of opioids intra and postoperatively
- The use of nitrous oxide
- The use of volatile inhalational anaesthetics (e.g. ether)
- Some intravenous anaesthetics (e.g. ketamine and etomidate)

Postoperative factors. Pain, anxiety, hypotension and dehydration all contribute to nausea and vomiting.

Not surprisingly, females undergoing gynaecological procedures where opioids are used as part of the anaesthetic technique, have one of the highest incidences of PONV (up to 70%)! By taking into account the above factors and availablility of local resources, a start can be made to try and reduce PONV.

Pharmacological Treatment of PONV

It is interesting to note that of the drugs generally referred to as anti-emetics and used in the management of PONV, some have more anti-nausea and less anti-vomiting effects, whilst others have less anti-nausea and more anti-vomiting effects.

Pharmacological treatment of PONV is common, using a wide range of drugs, but with variable efficacy. The drugs are generally grouped according to the type of receptor at which they act, usually as an antagonist.

The following text describes the various groups of drugs conventionally used in the treatment of PONV, and their contra-indications. Doses and routes of administration can be found in Table 1.

Anticholinergic (antimuscarinic) drugs

Anticholinergic drugs that can cross the blood-brain barrier, will act directly on the vomiting center and have anti-emetic properties. This is the oldest group of drugs used to treat nausea and vomiting, although this was not their original intention. Atropine was used to block the vagal effects of chloroform and later used for its drying effect on salivary secretions during ether anaesthesia. It was subsequently replaced by hyoscine

Table 1. Anti-emetics; doses and routes of administration

Drug	Group	Dose, Route & Frequency
Atropine	Anticholinergic	0.3 - 0.6mg im or iv, 30 - 60mins pre-op
Hyoscine	Anticholinergic	0.2 - 0.4mg sc or im, 6 hourly 1mg transdermal patch, lasts upto 72hrs
Cyclizine	Antihistamine	50mg orally, im or iv, 8 hourly
Promethazine	Antihistamine	25mg orally, 100mg max in 24hrs
Prochlorperazine	D_2 antagonist	12.5mg orally or im, 6 hourly 25mg rectally as initial dose 3mg buccal preparation is available
Droperidol	D_2 antagonist	0.5 - 1.25mg iv, 8 hourly 2.5 - 5mg orally, 8 hourly
Metoclopramide	D_2 antagonist	10mg im or iv, 6 hourly
Domperidone	D_2 antagonist	10 - 20mg orally, 60mg max in 24hrs 60mg rectally, 4 - 8 hourly
Ondansetron	5-HT_3 antagonist	4 - 8mg orally, im or iv, 24mg max in 24hrs 16mg orally, 1hr pre-op as a one-off dose
Granisetron	5-HT_3 antagonist	1mg iv, 2mg max in 24hrs
Dexamethasone	Corticosteroid	6 - 10mg iv, preferably in combination (see text)

(Scopolamine). Both are still used to treat nausea and vomiting, with hyoscine being the more potent and effective. They are most effective against motion sickness, labyrinthe disease, vestibular disorders, after surgery in the posterior fossa and to counter the emetic effects of opioids. However, as a result of antimuscarinic actions, side effects include sedation, dry mouth, blurred vision and urinary retention, all more common after hyoscine. Contraindicated in closed angle glaucoma.

Antihistamines

This group of drugs is similar to the above in that they act on the vomiting center antagonizing the histamine (H_1) receptors. They are effective in the treatment of motion sickness, the management of labyrinthe disorders and to counter the emetic effects of opioids. In the UK, the most commonly used drug is cyclizine. Alternatives include promethazine. Side effects include mild sedation along with antimuscarinic effects. Cyclizine is contraindicated in acute myocardial infarction as it can aggravate severe heart failure and counteract the beneficial effects of the opioids. The sedative effects of antihistamines are additive with that produced by anaesthetic agents, and hence care must be taken with their use. Promethazine is said to have a slight antanalgesic effect.

Dopamine antagonists

There is a wide range of drugs that antagonize dopamine (D_2 receptors) at the CTZ and therefore have antiemetic properties. These include the phenothiazines, butyrophenones, metoclopramide and domperidone.

Phenothiazines. Prochlorperazine (Stemetil) is more commonly used in the UK as an anti-emetic than chlorpromazine, due to the latter is more marked sedation and drowsiness. Both can produce extrapyramidal side effects and acute oculogyric crises can occur with high doses and prolonged treatment. The neuroleptic malignant syndrome (catatonia, cardiovascular instability, hyperthermia and myoglobinaemia - mortality in excess of 10%) has been reported in association with prochlorperazine.

Butyrophenones. This group of drugs was originally developed to treat major psychoses (eg schizophrenia) and includes haloperidol and droperidol. The latter was widely used as a component of "neurolept anaesthesia", but associated with unpleasant side effects including extrapyramidal symptoms, hypotension, hypothermia and unpleasant hallucinations. However, in much smaller doses it has been shown to be a very effective anti-emetic when administered orally or intravenously. Unfortunately, it is no longer manufactured for use in the UK. Droperidol is pharmaceutically incompatible with thiopentone and methohexitone.

Metoclopramide. In addition to having an effect on the CTZ, metoclopramide has prokinetic actions on the gut, promoting gastric emptying and increases the barrier pressure of the lower oesophageal sphincter (by about 17mmHg). Although widely used as an anti-emetic evidence for its efficacy in treating PONV is limited. It is perhaps best reserved for use preoperatively in those cases where there is evidence for delayed gastric emptying or patients at risk of gastro-oesophageal reflux. Extrapyramidal side effects can occur. Not recommended following gastrointestinal surgery involving anastamoses.

The neuroleptic malignant syndrome has been reported in association with metoclopramide.

Domperidone. Similar to metoclopramide, but does not cross

the blood-brain-barrier and therefore not associated with sedation or extrapyramidal side effects. Not effective against motion sickness. It can cause cardiac arrythmias in large doses.

5-HT_3 receptor antagonists

This is the most recently introduced (and therefore the most expensive) group of anti-emetics available. Although it is thought that their main action is to antagonize 5-HT_3 receptors that are found in a high concentration in the CTZ, they may also have a peripheral effect. Ondansetron is the most commonly used and appears effective when used orally preoperatively and intravenously for PONV. It is well tolerated with few side effects, headache being the most commonly reported. It appears to produce greater anti-vomiting effects than anti-nausea effects. Recently, more potent 5-HT_3 receptor antagonists have been introduced mainly for use in chemotherapy induced nausea and vomiting, but granisetron has been shown to be effective in treating PONV.

Other drugs used for anti-emesis

Dexamethasone has been shown to be anti-emetic in a dose of 10mg in adults. It appears to be of most use in combination with a 5-HT_3 receptor antagonist, working via an additive or even synergistic effect.

Which drug shall I use?

When using drugs to prevent or treat PONV, find out what is available, then consider what to use, when to use it and how to use it.

The first thing is to know what is available to use. For instance, droperidol is being removed from the formulary in the UK, and 5-HT_3 receptor antagonists are too expensive to be considered in many parts of the World.

Everything else being equal it has been shown that ondansetron is as effective as droperidol, and that both are more effective than metoclopramide. However the administration of one receptor antagonist will reduce the incidence of PONV by only 30%, but a combination anti-emetic therapy (typically a 5-HT_3 receptor antagonist with droperidol or dexamethasone) can achieve a response rate of up to 90%. What to use will also depend upon what are you treating. Ondansetron has more anti-vomiting than anti-nausea properties, dopamine receptor antagonists have more anti-nausea properties than they do anti-vomiting properties.

When should drugs be prescribed? It has been shown that the "number needed to treat" (i.e.; the number of patients you need to treat before the treatment is effective for one of them) with anti-emetics ranges from 5 for ondansetron to more than 10 with metoclopramide. Thus if everyone received anti-emetic prophylaxis, 80% of patients would still be at risk of PONV. The benefit of this has to be weighed up against the exposure to potential side-effects.

It can be seen therefore that the prevention and treatment of PONV using drugs alone is not especially effective. It would appear to make sense to identify those patients at high risk and firstly try to use an anaesthetic technique or anaesthetic agents associated with less nausea and vomiting and supplement this with anti-emetic agents as required .

Use of anti-emetics in pregnancy

The use of anti-emetics in pregnancy is controversial because of the risks of fetal teratogenicity, especially during the first trimester. Unfortunately, the incidence of nausea and vomiting in pregnancy is extremely high (nausea affects between 75 - 85% of women, and vomiting about 50%). In its most severe form, hyperemesis gravidarum, it can lead to dehydration, hyponatraemia, hypokalaemia, metabolic hypochloraemic alkalosis, ketonuria and loss of bodyweight. Although initial management consists of intravenous fluid and electrolyte replacement, anti-emetic therapy often has to be added.

Evidence - based reviews suggest that pyridoxine (vitamin B6), antihistamines (H_1-blockers), phenothiazines, ginger root extract and acupuncture are safe for use in in pregnancy with varaiable efficacy. Metoclopramide, droperidol and ondansetron may be effective, but there are insufficient safety data to recommend them as first-line therapy.

Anaesthesia as a cause of nausea and vomiting in labour commonly relates to the hypotension associated with the use of central neural blockade. In these situations nausea and vomiting are often an indication of cerebral hypoperfusion, secondary to hypotension. It can usually be overcome by elevating the blood pressure to normal limits by the judicial use of fluid boluses or vaoconstrictor drugs such as ephedrine or phenylepherine. Surgery may also contribute, particularly exteriorizing the uterus or excessive traction on the bowel or mesentery. Although atropine may help the answer lies in the hands of the surgeon!

Non-pharmacological treatments of PONV

Ginger root has been postulated as an anti-emetic but a systematic review of the available evidence has only shown it to be as effective as metoclopramide, and not significantly different from placebo.

Acupuncture at the Pericardium 6 point (5cm proximal to the palmar aspect of the wrist, between the flexor carpi radialis and palmaris longus tendons) has been shown to be effective in treating early PONV, with a number needed to treat of 5.

Perioperative hypnosis has been demonstrated to reduce PONV following breast surgery.

The choice of anaesthetic technique in the prevention of PONV

It has already been stated that anaesthetic factors contribute to PONV. From the above list, the ideal anaesthetic technique would consist of avoiding opioids, nitrous oxide and volatile anaesthetic agents, and result in no pain, anxiety, hypotension or dehydration!

- **Omitting nitrous oxide**. Fifteen percent of patients receiving nitrous oxide will experience nausea and vomiting and omitting it from a general anaesthetic has been proven in three systematic reviews to reduce the incidence of PONV. This was also most pronounced in high-risk patients, with a number needed to treat of 5. However, there was also an increase in the incidence of awareness.

- **Omitting reversal of neuromuscular blockade**. Neostigmine increases salivation, lower oesophageal and gastric tone, gastric

acid output and lower gastrointestinal tract motility, thus nausea and vomiting may occur. Omitting anticholinesterase drugs at the end of surgery may decrease the incidence of PONV, but only in doses greater than 2.5mg of neostigmine. There is a concomitant risk residual neuromuscular block with all the attendant risks.

- Propofol appears to possess intrinsic antiemetic properties, possibly by the antagonism of dopamine D_2 receptors. It has been used in the treatment of refractory nausea and vomiting in chemotherapy patients. When used for induction and maintenance there is a reduction in the incidence of PONV, with a number needed to treat of 5. Induction alone has no influence. Total intravenous anaesthesia with propofol is an expensive option, both in terms of the cost of the propofol itself, and the equipment required.

- Ether is one of the most emetogenic of all the inhalational anaesthetic agents. It has been reported as being associated with an over-all incidence of PONV in excess of 80% of patients. It seems to be worse when high inspired concentrations are used, or when administered over prolonged periods. Ether is therefore best avoided, but if it has to be used, then the lowest concentrations for the shortest period of time should be used.

- Regional Blockade is a useful technique in the prevention of PONV. When used as the sole technique, opioids can be avoided thereby reducing the risk of PONV. If a technique is used with an in-dwelling catheter (for example an epidural), opioids may also be avoided postoperatively. If a regional technique is used in combination with general anaesthesia, both opioids and nitrous oxide can still be avoided and therefore provides a better bet than general anaesthesia using opioids. However, it is essential when using regional anaesthesia, in particular any form of central neural block to avoid hypotension and ensure hydration, otherwise perioperative nausea and vomiting will still be an issue.

Therefore an anaesthetic technique that reduces anxiety by using an appropriate premedicant along with reassurance at the preoperative visit, avoids opioids and pain using alternative analgesics for example local or regional anaesthesia and substitutes total intravenous anaesthesia for nitrous oxide and volatile anaesthetic agents, avoids reversal of neuromuscular blockade and results in a warm, well hydrated normotensive patient, would be ideal to minimise the incidence of PONV, especially in the high risk patient.

Cost-effectiveness of anti-emetics

The result of cost-effectiveness trials has shown that treatment of PONV with ondansetron is more cost-effective than prophylaxis with ondansetron in all patients. However, the pre-emptive treatment of high risk patients with droperidol is cost-effective and leads to greater patient satisfaction.

Future development

Future development in anti-emesis is looking at the neurokinin 1 (NK-1) receptor, where substance P is the natural ligand. This receptor is found in the nucleus tractus solitarius and the area

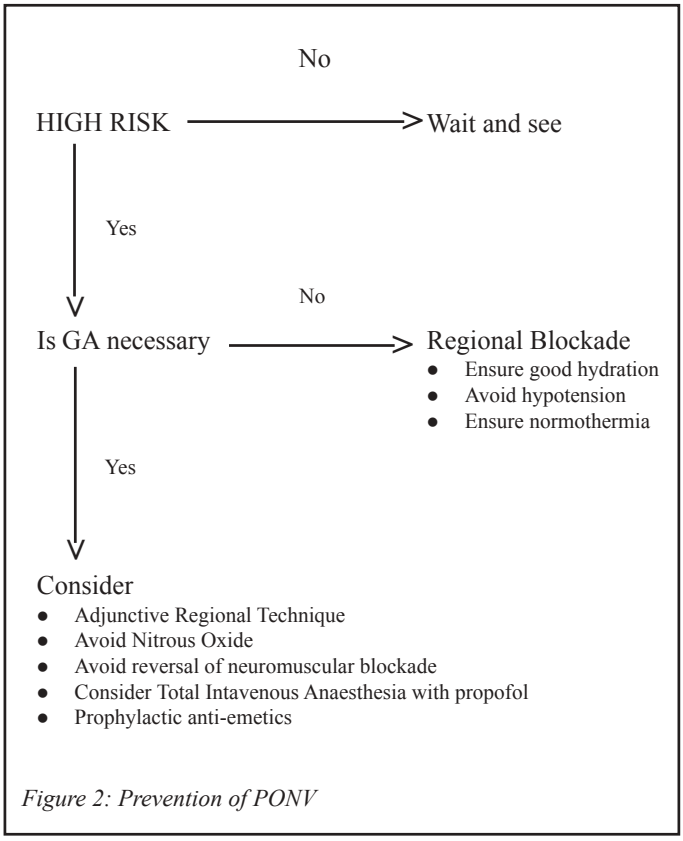


Figure 2: Prevention of PONV

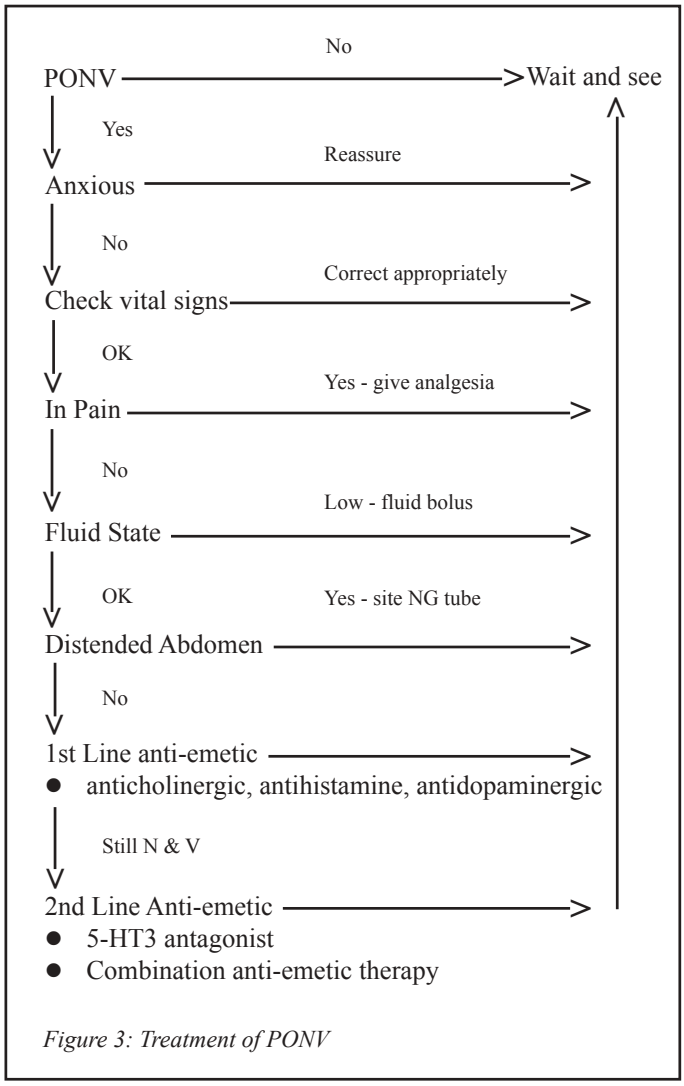


Figure 3: Treatment of PONV

postrema, as well as the peripheral nervous system. Early studies of NK-1 antagonists have been promising, especially in combination with ondansetron.

Development of new non-opioid analgesia will help to decrease PONV in the future.

Conclusion

Despite an improvement in the incidence of PONV with modern pharmacology and techniques, there is still no "Gold Standard". This affects both patients and research, such that a new anti-emetic strategy has no ideal to compare with. Thus we are left with numerous approaches to the problem, with no satisfactory answer. When considering the problem on a World scale where funds are limited, it would appear that the best strategy is first to identify the high risk patients. Once these patients are known an anaesthetic technique should be employed that avoids as many emetogenic factors as possible, combined with combination anti-emetic drug therapy. Thus providing a balanced anti-emetic technique (Figure 2). It would appear that for low risk patients, the most cost effective plan is to "wait and see", and treat as required (Figure 3).

Further Reading

Calvey NT, Williams NE. Premedication and antiemetic drugs. In Calvey NT, Williams NE. Principles and practice of pharmacology for anaesthetists. Third Edition. Oxford: Blackwell Science. 1997: 448-479

Jewell D, Young G. Interventions for nausea and vomiting in early pregnancy (Cochrane Review). In: The Cochrane Library, Issue 3, 2002. Oxford: Update Software

Magee LA, Mazzotta P, Koren G. Evidence-based view of safety and effectiveness of pharmacologic therapy for nausea and vomiting of pregnancy (NVP). American Journal of Obstetrics and Gynecology. 2002:**185**:S256-61

Neal MJ. Drugs used in nausea and vertigo (anti-emetics). In Neal MJ. Medical pharmacology at a glance. Third edition. Oxford: Blackwell Science. 1997: 66-7

Sasada M, Smith S. Drugs in Anaesthesia and Intensive care. Second Edition. Oxford University Press. 1997

Tong J, Gan MB. Postoperative nausea and vomiting - can it be eliminated? Journal of the American Medical Association. 2002:**287**:1233-6

Tramer MR. A rational approach to the control of postoperative nausea and vomiting: evidence from systematic reviews. Part 1. Efficacy and harm of anti-emetic interventions, and methodological issues. Acta Anaesthesiology Scandinavia 2001:**45**:4-13

Tramer MR. A rational approach to the control of postoperative nausea and vomiting: evidence from systematic reviews. Part 2. Recommendations for prevention and treatment, and research agenda. Acta Anaesthesiol Scand 2001: **45**: 14-19

AIDS TO TRACHEAL INTUBATION

Dr. Sasanka S. Dhara, Staff Anaesthetist, Royal Hobart Hospital, Tasmania, Australia

Using an endotracheal tube to secure a patient's airway is still the gold standard. Most routine orotracheal or nasotracheal intubations are performed with the help of a laryngoscope that has a curved or straight blade. Other adjuncts such as external laryngeal pressure, a bougie, a stylet or a pair of Magill's forceps may also be used.

Difficulties encountered during intubation can be due to a number of factors and may be difficult to predict. It is important to have a strategy prepared and to be familiar with the equipment. This will help to avoid potential morbidity or mortality from the sequelae of hypoxia and/or cardiovascular incident that may result from a failed intubation.

The anaesthetist must be familiar with the major decision making components of the difficult airway algorithm. These are as follows:

- recognition of a difficult airway
- positioning a patient for airway manipulation
- awake intubation techniques
- techniques for anaesthetised patient with a difficult airway
- techniques for the patient who cannot be ventilated or intubated
- confirming the position of the endotracheal tube
- extubation or tube change for a patient with a known difficult airway

Over the years many attempts have been made to address various factors responsible for difficult intubations and this has resulted in a number of different techniques. It is best to use affordable, safe and useful adjuncts that are best suited to your particular anaesthetic set up.

Direct laryngoscopic orotracheal intubation

Secure intravenous access, pre-oxygenate and then induce the patient. A source of oxygen and facilities for mask ventilation should be present. Unconscious patients may obstruct their airways and the mechanism for this may be:

- the relaxed soft palate falling onto the posterior pharyngeal wall
- relaxed muscles on the floor of the mouth letting the tongue fall back against the posterior pharyngeal wall
- the epiglottis getting stuck in the glottic inlet

Various devices may be used to overcome mechanical airway obstruction:

- An **oropharyngeal airway** is useful although care should always be taken when inserting it to avoid damaging the patient's teeth and oral soft tissues.
- A **nasopharyngeal airway** may be better tolerated than an oropharyngeal airway in a patient recovering from general anaesthesia because it causes less salivation and coughing. It may cause nasopharyngeal bleeding on insertion.
- The **Laryngeal Mask Airway (LMA)** can be used as a primary airway in an unconscious patient and it has also been used for emergency airway management although it does not protect the airway from regurgitated stomach contents.
- The **Cuffed Oropharyngeal Airway (COPA)** is a modified oral airway with an inflatable cuff mounted at its distal end.
- **Laryngeal Tube (LT)** is a short S-shaped tube with two cuffs: a small oesophageal cuff at the distal end that blocks entry into the oesophagus and reduces the likelihood of gastric inflation, and a large pharyngeal cuff to stabilise the tube and to block the naso- and oropharynx. There is a ventilation hole between the two cuffs that aligns with the larynx. The laryngeal tube is blindly inserted and positioned by the 'teeth marks' on the stem of the tube. It has been used as an alternative to ventilation with a facemask or LMA.

When ready to intubate, have the following ready:

- A pillow or a padded ring to elevate the head by 8-10 cm. This manoeuvre helps to align the laryngeal and pharyngeal axes. For obese and pregnant patients, a pillow under the shoulder blades to elevate the upper thoracic spine may help with better visualisation of the larynx during direct laryngoscopy.
- A working laryngoscope handle with a choice of two blades.
- Reliable suction.
- Endotracheal tube with another that is half a size smaller. A syringe to inflate the cuff.
- Intubating stylet and Eschmann gum elastic bougie
- A pair of Magills forceps.
- Local anaesthetic spray and lubricant gel.
- Tape or tie to fix the endotracheal tube.
- Stethoscope to confirm the correct positioning of the endotracheal tube.
- Throat pack when the surgical operation involves areas such as the nasal passage, mouth, tongue and pharynx.
- Routine patient monitors.
- An assistant to help with intubation. In addition to passing the laryngoscope, the endotracheal tube or the suction, the assistant may have to help with the application of external laryngeal pressure or retraction of right angle of mouth for better visualisation of the laryngeal inlet. External laryngeal pressure is applied on to thyroid cartilage in a backward, upward direction

and can help the operator to visualise the glottis. It is not the same as cricoid pressure.

Some of the common causes of difficult direct laryngoscopy are:

● Improper positioning. Too much neck extension will result in difficulty in finding the laryngeal inlet while too much flexion will make it difficult to introduce the laryngoscope into the mouth.

● Insufficient muscle relaxation.

● Positioning of the laryngoscope blade. No tongue should be visible on the right side of the blade.

● Identification of structures. Finding the epiglottis is the key to the laryngeal inlet.

● The position of the tip of the blade. If the tip is not placed far enough into the vallecula, the view of the larynx will be closer to a grade 3 view and if it is too far into the oesophagus, the whole larynx will be missed (a common problem in neonatal intubation).

● Excessive force applied during cricoid pressure will make laryngoscopy difficult.

● The best person to find the optimal external laryngeal pressure for best view of the larynx is the person performing the intubation. Ask the assistant to put their fingers in place on the larynx and then move the assistant's hand. When the best view has been obtained the assistant can then the pressure in the right place.

Adjuncts to facilitate intubation

● **Laryngoscope handles**: A short handle may help to insert the laryngoscope blade into a patient's mouth when a normal blade is awkward to use due to the presence of large breasts in an obese or pregnant patient.

● **Blades**: The most commonly used blade in adults is the Macintosh blade. Straight Miller blades are commonly used when intubating children. The polio blade was designed for intubation of patients in an "iron lung" and may still be useful today if large breasts pose a problem.

● **Adaptors**: These adjuncts have been developed to fit between the handle and the blade of a laryngoscope to change the angle between them. They may help to visualise an anterior larynx.

● **Special laryngoscopes**: A McCoy laryngoscope has a manoeuvrable tip that is controlled by the operator (Figure 1). The tip moves anteriorly and lifts up the epiglottis. It is reputed to convert the Cormack and Lehane laryngoscopic view from 3 to 2 and from 2 to 1. A rigid bronchoscope can also be used to visualise the larynx and to place an introducer that can then be used as a guide for the endotracheal tube.

● **Stylet**: A malleable metal wire covered with plastic is used to give a tracheal tube curved shape and rigidity (Figure 2). It should be used with care as it may cause airway trauma.

● **Introducer**: This is a firm guide to lead a tracheal tube into the larynx. An example of an introducer is a gum elastic bougie that has a slightly angulated tip (coude tip) (Figure 2) and a plastic exchange catheter that has a hollow lumen to deliver oxygen through it. An introducer is especially useful when only part of the larynx is visualised or when only the epiglottis can be seen.The anaesthetist slides the angled tip of the introducer under the edge of the epiglottis and into the larynx where the tracheal rings can be felt. If they cannot be felt, the introducer may have entered the oesophagus. The endotracheal tube is then guided over the introducer and into the trachea whereupon the introducer is withdrawn.

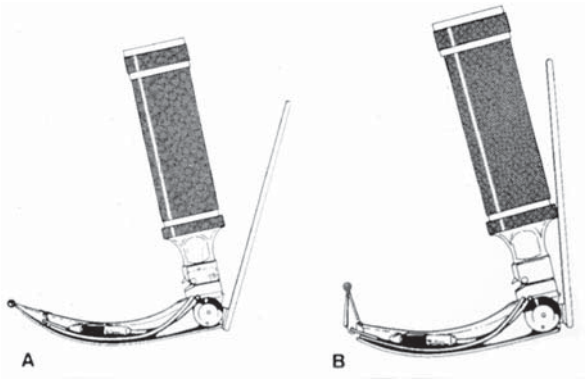


Figure 1. McCoy laryngoscope

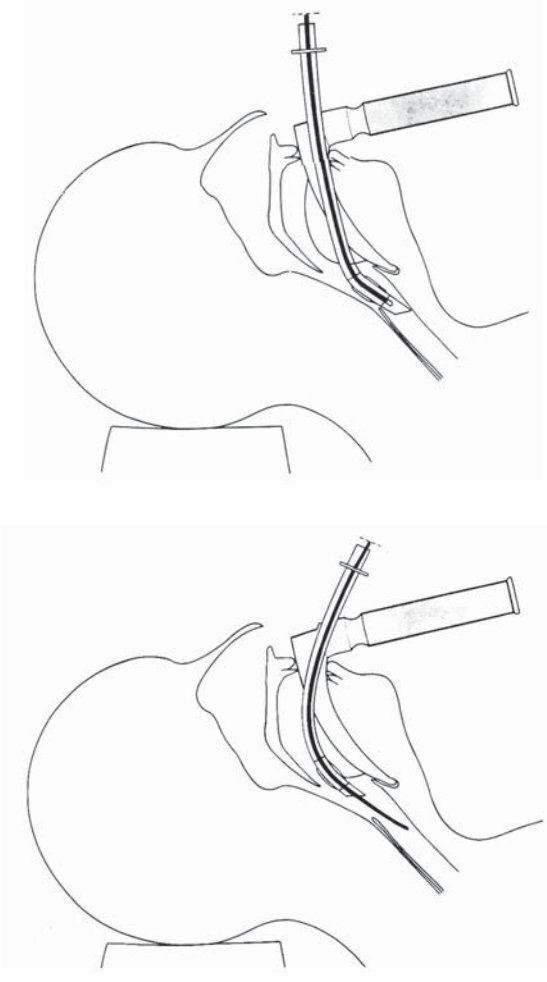


Figure 2a. Intubation using stylet. 2b Intubation using a bougie

Difficulties may be encountered when attempting to guide the endotracheal tube over the introducer in which case the following scenarios should be considered:

- Is the introducer in the airway to an adequate depth?
- Is the difference between the external diameter of the introducer and the internal diameter of the tube too great? A softened and well lubricated tube of a smaller size (typically 6, 6.5 or 7mm) will follow the guide (typically a gum elastic bougie) better as it does not "hang up" or drag the introducer out of the airway. This may happen with a larger and stiffer tracheal tube. A reinforced tube is easier to guide than a standard endotracheal tube because it is softer.
- Is the larynx too anterior? In this case, pulling the tongue forward is a useful manoeuvre to guide the tube in the right direction.
- Is the muscle relaxation sufficient?
- Is the larynx too small for the tube? In which case use a tube half a size smaller.
- Is the tube stuck at the anterior commissure? Twisting the tube on its axis anticlockwise to 90 degrees so the bevel faces posteriorly may help the tube to pass through the larynx.

Predicting a difficult intubation

Methods of predicting a potentially difficult intubation have been developed. Mallampati described an assessment with the patient sitting opposite the anaesthetist. The patient is asked to open his mouth and extend his tongue. The view obtained gives the anaesthetist an indication of the difficulty that is likely to be encountered. This system is not foolproof.

Changing an endotracheal tube

If it is necessary to change an endotracheal tube that is in place:

- Secure intravenous access and have anaesthetic and resuscitation drugs present as well as the airway adjuncts previously discussed.
- The patient should be appropriately sedated and paralysed.
- Pre-oxygenate for 3 minutes prior to changing the tube because this will give you a bit more time if the tube change proves to be difficult.
- Suction the oropharynx so that you have a clear view.
- Insert an introducer down the tube and remove the tube, leaving the introducer in situ. Then pass another tube over the introducer and remove the introducer. A smaller tube may be required if the patient has been intubated for some time as the airway may be oedematous.
- Confirm that the tube is correctly positioned by observing chest movement, auscultating the chest with a stethoscope and using capnography if available.

"Can't ventilate, can't intubate"

- If the patient is difficult to intubate, stop trying and return to bag and mask ventilation. If you are able to ventilate the patient then consider any adjuncts or procedures that may help you.
- If you are unable to ventilate the patient in spite of the adjuncts mentioned previously, call for help. Wake the patient up if appropriate or prepare for an emergency cricothyroidotomy.
- A 14G intravenous cannula or a cricothyroidotomy cannula is inserted through the cricothyroid membrane and oxygen under pressure is administered into the patients lungs. This is called transtracheal jet ventilation (TTJV).
- The oxygen supply is from the wall or a cylinder. It is connected to a pressure regulator and a jet actuator that is then connected to the cricothyrotomy cannula via a luer lock system (Figure 3). Remember that the oxygen is under high pressure and that the patient is at risk of barotrauma when using this method of ventilation. Adjust the driving pressure carefully and make sure that there is no obstruction to airflow on exhalation.
- Jet ventilation works mainly by bulk flow of oxygen, but a considerable volume of air is entrained from the open glottis (venturi effect).

Mallampati's classification

Class 1	The soft palate, faucial pillars and uvula are all visible
Class 2	The soft palate and faucial pillars are visible, but the uvula is obscured by the base of the tongue
Class 3	Only the soft palate is visible

Cormack and Lehane's classification

Grade 1	Most of the glottis is seen. No difficulty.
Grade 2	Only the posterior part of the glottis is visible. Pressure on the larynx may improve the view. Slight difficulty.
Grade 3	The epiglottis is visible, but none of the glottis can be seen. A bougie may be used. There may be severe difficulty.
Grade 4	Not even the epiglottis is visible. This situation usually arises with obvious pathology. Intubation may be impossible without special techniques.

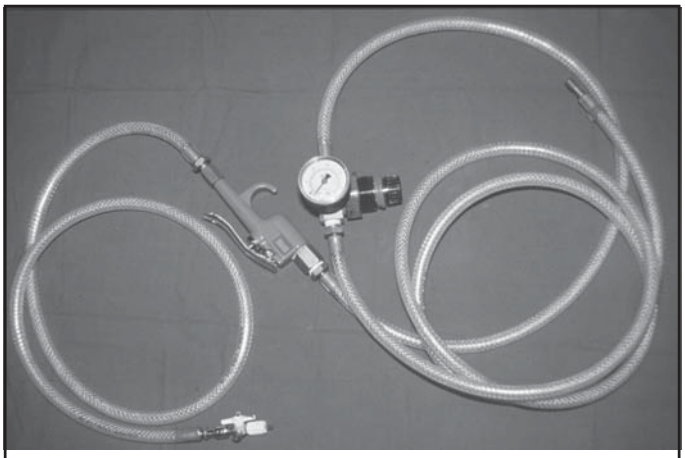


Figure 3. Assembled apparatus for jet ventilation

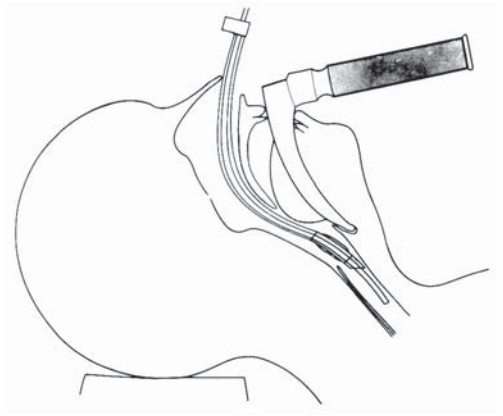


Figure 4. Suction catheter through nasotracheal tube

● Oxygenation is the main concern and this is achieved by using smaller tidal volumes, a higher respiratory rate (20-40/minute) and a longer I:E ratio (1:4).

● The emergency oxygen flush from the anaesthetic machine may be used as a source of pressurised oxygen by connecting a non-compliant tubing system to the common gas outlet with a 15 mm endotracheal tube connector. However, it should be noted that most modern machines are fitted with a safety valve to prevent overpressure of the backbar and therefore may not be suitable for this purpose.

● A 7.5 mm tracheal tube connector fitted tightly into the barrel of a 3ml luer lock syringe can accommodate a self-inflating bag on one end and the cricothyroid cannula on the other. Some oxygen can be transferred by squeezing the bag hard, but this is not ‘ jet ventilation’.

● These are only temporary measures.

For nasotracheal intubation using direct laryngoscopy

● Local anaesthetics can be sprayed into the nasal passage eg.cocaine 4-10% (maximum 1.5mg/kg) which has the advantage of having vasoconstrictor properties or lignocaine 2-10% (maximum 3mg/kg).

● Vasoconstrictor drugs eg.phenylephrine or pseudoephedrine nasal spray are helpful in reducing nose bleeds.

● Soften the endotracheal tube by immersing it in warm clean water.

● Insert the endotracheal into a nostril at an angle perpendicular to the face and exert gentle pressure until the tube is visualised at the back of the oropharynx. Guide the tube into the larynx using a pair of Magill‘s forceps if necessary. Rotating the bevel of the endotracheal tube so that it faces posteriorly allows smoother passage of the tip across the laryngeal inlet.

For emergency intubation

● A rapid sequence induction (RSI) is always performed in emergency situations, when a patient is not starved or when reflux is present. A rapid sequence induction consists of preoxygenation for 3 minutes, intravenous administration of a predetermined dose of an induction agent eg.3 - 4mg/kg of thiopentone and a rapidly acting muscle relaxant such as suxamethonium 1-1.5mg/kg.

● Cricoid pressure is applied by an anaesthetic assistant as soon as the patient loses consciousness. The pressure applied on the cricoid ring occludes the oesophagus against the body of the 6th cervical vertebra and thus prevents regurgitation of stomach contents up into the oropharynx.

● The assistant’s hand applying cricoid pressure may obstruct the introduction of the laryngoscope into the mouth. It requires a careful sideways insertion of the blade into the mouth. A laryngoscope with short handle is useful in this situation.

● The cricoid pressure is taken off only after confirmation of correct placement of endotracheal tube with the cuff inflated.

Awake intubation

Indications for awake intubation include:

● Upper airway obstruction

● A known or suspected difficult intubation

● Patient with an unstable cervical spine fracture where any traction on the neck should be avoided

● Full stomach. This technique is used in the United States of America.

● Respiratory failure in extremis where anaesthetic induction may bring about the patient’s immediate demise

The procedure for anaesthetising the airway is as follows:

● Give oxygen to the patient throughout the procedure eg.nasal prongs and ensure that all routine monitoring is in place with intravenous access secured.

● Administer a drying agent intravenously, for example atropine 400-600mcg or glycopyrrolate 200-400 mcg.

● Administer sedation to make the patient comfortable without compromising patient safety. For example, a benzodiazepine eg.midazolam 0.5-2mg along with a short acting opioid eg.fentanyl 50mcg. Although both drugs have specific antagonists available, care should be taken not to depress respiration too much.

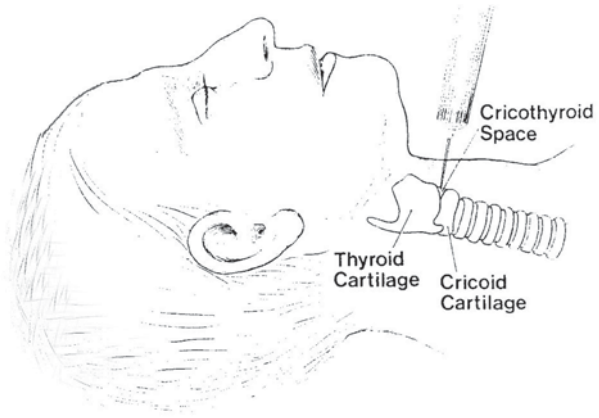


Figure 5. Transtracheal injection

● Local anaesthesia of the upper airway is achieved as follows: Surface analgesia is achieved with 2-4% lignocaine (maximum 3mg/kg) applied to the mouth, tongue, pharynx and nasal passages by spraying, gargling or inhaling a nebulised form. Cotton tipped pledgets soaked in the same solution may be used for analgesia of the nasal passage. A translaryngeal injection through the cricothyroid membrane provides analgesia to the area below the vocal cords. To perform this injection, identify the cricothyroid membrane and confirm that the tip of the needle is in right place by free aspiration of air in a saline-filled syringe before the injection is made (Figure 5). 2-4ml of lignocaine 4% is used because the higher concentration penetrates the mucosa more efficiently. As soon as the injection is performed the patient will cough and the needle should be withdrawn swiftly to prevent any damage.

● Have the equipment ready for the chosen technique eg.fibreoptic bronchoscope or retrograde intubation set.

● Plan for the procedure and have a rescue plan in case it fails.

Indirect laryngoscopy

● **Flexible fibreoptic scope**: This instrument has changed the whole management of a difficult airway by allowing indirect visualisation of the larynx. It is made up of coated glass fibres that transmit light and images. These fibres are delicate structures and are easily damaged so the fibreoptic scope should always be handled with care. It may contain aspiration channels that can be used for suctioning secretions, insufflating oxygen or instilling local anaesthetic. One does require training in its use. Disadvantages include: poor images in the presence of bleeding or excessive secretions, initial and subsequent maintenance costs and the need for adult and paediatric sizes.

● **Rigid indirect laryngoscope**: This instrument uses fibreoptics to visualise the glottis and contains a channel for the endotracheal tube. It is expensive, learning to use it takes considerable time and success rates vary.

Guided blind techniques

These techniques require the use of a physical guide to lead the endotracheal tube into the glottic inlet.

Laryngeal mask and Intubating LMA:

The laryngeal mask is probably the most important invention in anaesthesia in recent times. The LMA has been used as a conduit to reach the larynx by passing a bougie, a fibreoptic bronchoscope or sometimes a smaller tracheal tube through it. In these situations the LMA is usually left in situ until the end of the anaesthetic.

The Intubating LMA (ILMA) is a preformed, anatomically-shaped metal tube that is fitted with the usual LMA cuff (Figure 6). A specially designed tube is passed through the ILMA and into the larynx. Tube position is checked before removing the ILMA and leaving the tube in situ.

Augustine guide:

This device consists of an anatomically-shaped, disposable, plastic channelled guide with a special stylet. It combines features of pharyngeal airway, stylet, bougie and oesophageal detector device. The tube is loaded over the guide and the holllow stylet is used to find the trachea. The stylet position is confirmed by injecting air with a syringe and auscultating the stomach (oesophageal detector device). The tube is then guided over it.

In order to use it, mouth opening must be normal. It is more traumatic than normal laryngoscopy though cervical spine mobility during the procedure is minimal.

Retrograde intubation:

This technique was first reported by D.J.Waters in 1963. The basic technique consists of passing a retrograde guide through the cricothyroid membrane. This is then taken out from mouth or nose and an endotracheal tube is guided over it. There are many reports of retrograde intubation using various techniques and equipment:

● The retrograde guide may be an epidural catheter or a vascular guidewire (such as those used for insertion of a central venous

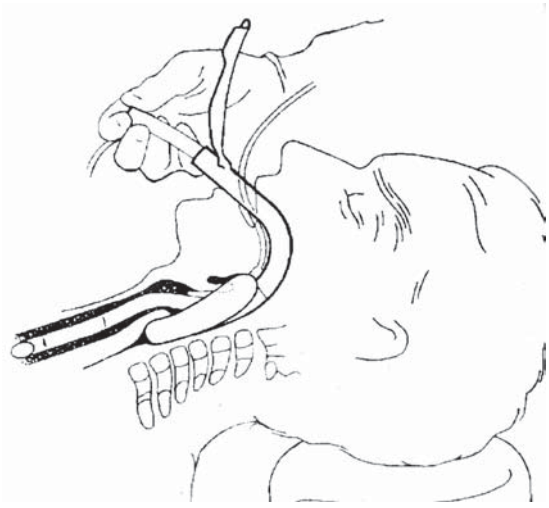


Figure 6. Intubating LMA

catheter) that is firmer and also the 'J' tip is less traumatic when moved inside the airway.

● A cricothyroid puncture is performed using a 16G intravenous cannula. It is important to make sure that the guide wire passes through the cannula easily. Once positioned, the cannula should be left in place, even after the retrograde guide wire has been passed through and positioned. The cricotracheal space rather than cricothyroid space has been advocated by some because it is less vascular and the depth of insertion is longer thus preventing the endotracheal tube from becoming dislodged when the retrograde guidewire is withdrawn and tracheal intubation is attempted.

● An anterograde guide such as a 14-16 FG suction catheter may be used and passed over the retrograde wire so that the endotracheal tube, when railroaded, has a better guide to follow rather than the thin and easily flexible retrograde guidewire. It is important that the anterograde guide is inserted to an adequate depth to prevent it from becoming dislodged when the patient coughs or when the retrograde guide is removed. The cough reflex is usually well suppressed from the transtracheal instillation of local anaesthetic solution. The retrograde wire is then removed and an endotracheal tube guided over the anterograde guide which is removed once correct placement has been confirmed.

● The procedure can be performed awake with appropriate application of local anaesthesia to the airway. Sedation or a small dose of induction agent will make the procedure more comfortable for the patient.

● The technique is very useful when all else has failed but it can also be used as a planned procedure. It does not require expensive equipment and with basic knowledge of anatomy, the technique can be performed easily. Contraindications are few, but include infection or tumour in the area and clotting disorders. Unlike fibreoptic bronchoscopy, the presence of blood in the airway does not hinder the procedure.

Lighted stylets or lightwands:

This method involves using a malleable stylet with a light at its tip that is placed inside the endotracheal tube. The stylet is bent to a L-shape and the patient is positioned with his head fully extended. The lightwand is passed in the midline over the tongue. Abrupt transillumination occurs when the lighted tip passes the epiglottis enters the larynx. The stylet is then removed.

Blind techniques

The intubation is performed without a direct or indirect view of the glottis and techniques are blind nasal and tactile oral.

Blind Nasal:

This procedure may be performed in an awake patient with conscious sedation and local anaesthesia to the airway or in an anaesthetised patient breathing spontaneously. The head is positioned as for direct laryngoscopic intubation and a softened, well lubricated endotracheal tube (typically 6.5-7.5 mm in adults) is gently passed through the nostril of choice until it reaches the pharynx. Then the chin is lifted forwards and the other nostril is occluded. If the patient is awake he is asked to close his mouth and breathe deeply. Alternatively, in an anaesthetised patient, the tube is advanced slowly while listening for breath sounds at the end of the tube. Capnography is extremely useful in this situation. Breath sounds or a capnography trace indicate that the tube has passed into the trachea. Blind nasal intubation is a very useful technique because it does not need expensive equipment and can be performed anywhere.

Tactile oral (Blind oral intubation):

This technique was first reported in 1880 by William MacEwen. It is performed by palpating the larynx while guiding the tube into it.

Conclusion

In real life scenarios, these techniques may be used in combination. It will depend on the problem, the resources available and the expertise of the anaesthetist. These factors should be considered carefully so that the best technique is chosen.

Further reading

1. Fibreoptic Endoscopy and the Difficult Airway. A. Ovassapian. Second Edition, 1996. Lippincott-Raven.

2. Airway Management. Hanowell and Waldron, 1996. Lippincott-Raven.

EDUCATION 2 - TUTORIAL TEACHING

Dr Kester Brown, President WFSA, Melbourne , Australia

Tutorial teaching is ideally conducted in small groups of 4 - 10 people, and has the advantage of being interactive between students and tutor. It is an ideal method of teaching when students already have a significant amount of knowledge. Students should have been notified of the topic of the tutorial beforehand to allow preparation, so that they are ready to discuss and gain from the session.

Everybody should be encouraged to participate. This sometimes requires the tutor to suppress those who always want to talk, and encourage those who tend to remain silent.

How can we achieve this? At the start of the tutorial the tutor should explain that everyone is expected to contribute and that students should feel free to express their ideas. Occasionally the tutor may have to ask talkative or disruptive students to hold their answers, while others give their opinion. This gives the everyone a chance to participate and provides a more balanced session.

The quieter people may have to be encouraged to contribute. This may involve asking them simple questions at first which they can answer, or it may need a way of showing that everyone can participate without embarrassment as fear of this is the commonest reason for holding back. A question can be asked with a numerical answer. Several people can be asked for the answer. If different answers are offered then one can put them to the vote. Some may still not participate. Not everyone is prepared to take the chance of being wrong. Take another vote. Then give the correct answer and show that you do not mind if they did not know the answer as long as they do when you have finished. An example is to ask how many mmols of chloride are in a litre of normal saline, usually in relation to a question on vomiting or pyloric stenosis. This approach often breaks down any barriers and facilitates participation.

There are teachers who threaten and belittle the students if they do not know something. This usually results in the student either being sure he knows the next time or not coming at all. It can be effective in the short term, but it is not conducive to creating an enjoyable atmosphere where the students think rather than learning by submission. Often a resentment develops between the teacher and the student.

What is the role of the tutor? The main role is to act as a catalyst and guide to the discussion. Many people do this by planning a series of questions which are worked through in order to generate more detailed discussion. The tutor tends to dominate the group in this approach.

A better approach is to let the students contribute more to the overall plan. This can be achieved very effectively by giving them a question which covers much of the topic of the tutorial. They have five minutes at the beginning to write a summary answer. The advantages of this are that their brains are fully activated at the start, they then have comments to make when asked, and they are contributing their ideas. The tutor then uses their summaries to work out with them a logical answer with all the points included. The detail can be discussed by the students with the tutor adding comments where appropriate and acting as a catalyst during the discussion. This summary answer approach has other advantages. It can be used in bigger groups when several questions can be posed and different groups each answer one.

Ultimately, if a group meets repeatedly they can run their own tutorial and the tutor then becomes more of a facilitator, a resource person clarifying contentious issues or filling in points that they do not know. This technique uses some of the principles of self directed learning. The tutor can also highlight the key points. Some students have difficulty discerning what is important from detail. The tutor can also explain the practical relevance of information which has been brought out in the discussion.

There are some key tactics in conducting the tutorial. Do not point to someone and then ask a question. Why? The unfortunate individual has a sympathetic response before even hearing the question. Cardiac output increases, tachycardia develops, sweating begins and the cardiac output is largely redistributed to muscle where no thinking takes place. The mind can go blank. The remainder of the class sigh with relief and relax. It is much better to ask the question, give a few seconds for the students to think about their answer and then ask someone to answer. The stress on the individual is much reduced and they have had time to think of the answer.

Do not go round the class in sequence asking questions. The person answering is stressed, the next person is becoming apprehensive and the sympathetic response is beginning to activate, while the others can sit back and relax and even go into "mental neutral"! Ask questions in a random order so that the students all have to remain alert throughout.

To summarise, ask a question and give the students time to prepare a summary answer. Take the main ideas from several students - they may vary in approach. Write them down to refer back to. Then try to put the main points into a logical order and develop each with further discussion. At the end there should be a comprehensive, logical answer which the student can use for a written or oral examination or as a framework for teaching others at a later date.

Tutorial teaching can be fun. It is lively because the speakers are changing frequently. It gives students a chance to put forward ideas that are sometimes new to the tutor. These experiences are stimulating and therefore contribute to the teacher's continuing education as well.

CAUDAL EPIDURAL ANESTHESIA FOR PEDIATRIC PATIENTS: A SAFE, RELIABLE AND EFFECTIVE METHOD IN DEVELOPING COUNTRIES

Alice Edler MD, MA (Education), Assistant Professor of Clinical Anesthesiology, Vinit G.Wellis, MD, Assistant Professor of Anesthesiology, Department of Anesthesiology, Section of Pediatric Anesthesiology, Stanford University School of Medicine, 3300 Pasteur Dr., Rm. H3580, Stanford Ca. 94305 USA

Introduction

In all areas of anesthesia, safety and efficiency are valued goals, and in developing countries additional challenges due to shortages of anesthetic drugs, supplies and monitoring equipment may be present. Caudal epidural anesthesia in developing countries, can in combination with general anesthesia or alone provide safe, reliable and efficient analgesia and / or anesthesia for both high risk and general pediatric surgical patients.

These techniques can be easily learnt and may be modified to extend analgesia into the postoperative period (with the addition of opioids or continuous techniques) or replace general anesthesia in circumstances where either the equipment or general anesthetic techniques are not available. The following manuscript will describe the pharmacological and physiologic basis of caudal epidural anesthesia, techniques for administration, monitoring, and specific modifying techniques of caudal epidural anesthesia and a discussion of complications and contraindications to caudal epidural anesthesia in pediatric patients.

History

Although spinal anesthesia was used in pediatric anesthesia as early as the 1940's, reports of successful pediatric caudal epidural anesthetics initially came from developing countries and in 1967, Fortuna reported a series of 170 patients between the ages of 1-10 years who received caudal epidural anesthesia for surgical procedures of the lower abdomen and lower extremities. These results showed that caudal anesthesia either alone or in combination with general anesthesia was well tolerated with little in the way of respiratory depression or cardiovascular changes.

A further series from Zimbabwe reported 500 pediatric caudal epidural anesthetics. The reported success rate was high (close to 90%), again being well tolerated by the patients, with little in the way of respiratory or cardiovascular problems, but with major failures due to incomplete block or restlessness during surgery. As these patients were only sedated, not anesthetized this could have accounted for some of the technical difficulties in positioning the caudal epidural needle and the restlessness during the surgery.

Today pediatric caudal epidural anesthesia is a well-accepted technique commonly used in combination with general anesthesia or occasionally as the sole anesthetic in high-risk patients.

Indications

Caudal epidural anesthesia in children can be used in:

- **Lower abdominal surgery**: (incision below the umbilicus - T10 sensory level) especially perineal, genitourinary or ilioinginual surgery.
- **Lower extremity surgery (hip, leg and foot)**: though at times it is difficult to achieve a satisfactory block to the distal 1/3 of the foot.
- **Newborn and premature infants**: If used as the sole anesthetic, caudal epidural anesthesia reduces the risk of respiratory depression from residual neuromuscular blockade (pancuronium) and inhalation anesthetics. Post-operative apnea associated with general anesthesia, is reduced with caudal anesthesia but not abolished.
- **Neuromuscular disease such as muscular dystrophy**. There is a high incidence of postoperative respiratory failure due to a combination of general anesthesia and muscle weakness. Caudal epidural anesthesia indicated for lower extremity surgery (very common in these patients).
- **Malignant hyperthermia**: it is generally accepted that all local anesthetic agents are considered safe.

Contraindications

The contraindications for caudal epidural anesthesia are similar to those for spinal or lumbar epidural anesthesia.

- **Coagulation disorders**: Bleeding abnormalities are an absolute contraindication to caudal epidural anesthesia. These abnormalities can be due to disorders of coagulation factor activity (such as Hemophilia, ITP, tumors, or DIC from sepsis) or from the administration of anticoagulants such as heparin or Warfarin. If there are any questions about the coagulation status, the anesthetist should perform a bleeding time test and confirm that

Table 1: Formulas to calculate drug volumes (mls) for single shot caudal epidural block (see also table 3)

Local Anesthetic*	Dose (mls)	Estimated Sensory Level
1 % Lignocaine	0.06ml/segment	Mid-Thoracic (T8)
0.25% Bupivicaine	1ml/kg	Mid-Thoracic (T8)
0.175% Bupivicaine	1.25 ml/kg	Mid Thoracic (T8)

* All solutions are containing epinephrine 1:200,000

Table 2 Maximum Recommended Doses of Local Anesthetics for Regional Anesthesia

Drug	Mg/kg (with epinephrine)	Duration (minutes)
Lignocaine	4 (7)	45 -180
Bupivicaine	2 (3)	180-600
Tetracaine	1.5	180-600
2 Chloroprocaine	8 (10)	30-60
Procaine	8 (10)	60-90

the bleeding time is normal. Bleeding time is a simple laboratory procedure that can be done at the bedside and gives results within 5 minutes. Another laboratory test to consider is INR. This is a more sophisticated test and may not be available at all hospitals.

- **Infection**: Caudal epidural anesthesia should not be used if there is an active infection at the site of injection either at the skin surface or below. This includes active cellulitis, pilonidal/ perirectal abscess, and meningitis. Even in the absence of localized infection, the caudal region has a higher bacterial count than the lumbar epidural space.
- Unstable blood pressure and/or heart rate
- Patient or parent refusal
- Congenital anatomic anomalies of the spinal cord or vertebral bodies - in cases of Spina Bifida, caudal epidural anesthesia should not be attempted as the spinal cord may be tethered within the spinal canal.
- Scoliosis is not an absolute contraindication to caudal epidural anesthesia though scolisis may make caudal epidural anesthesia technically more difficult to achieve.
- The dose of bupivicaine must be carefully controlled in patients with decreased cardiac function, as is often the case of patients with muscular dystrophy.

Anatomy and technique of caudal anaesthesia

The sacral hiatus (SH) in an infant or young child is easily identified because the landmarks are more superficial. The sacral hiatus is formed by failure of fusion of the fifth sacral vertebral arch. The remnants of the arch are known as the sacral cornu, and are located on either side of the hiatus. (See figure 1) The coccyx lies caudal to/lower than the sacral hiatus. Drawing an equilateral triangle by connecting the two posterior superior iliac spines (PSIS) usually locates the sacral hiatus at the apex. Palpation of the sacral hiatus at the apex of this inverted triangle should identify the puncture site. Alternatively, the anesthetist can palpate the convexity of the coccyx and then move cephalad to palpate the concave sacral hiatus to identify the puncture site. (See figure 2).

In young children, the epidural space can be easily reached by the caudal epidural approach with less risk of dural puncture than with thoracic or lumbar epidural approaches. There is minimal risk of cord injury at the level of the sacrococcygeal ligament so general anesthesia or heavy sedation is not often required to prevent the child from moving. However, the dural sac can extend to the level of third or fourth sacral vertebrae in the newborn and therefore care must be taken to avoid an inadvertent intrathecal injection. The sacrococcygeal ligament binds the sacral hiatus posteriorly, superiorly by the sacral cornu and the fused arch of the sacrum. There is considerable variation in the anatomy of the sacral hiatus, which may account for the small percentage of caudal epidural block failures. In addition, there is considerable variation in the angle of the sacral canal. Adult patients of African descent, have a steeper angle of entry into the sacral canal, therefore making the angle of initial needle place different than non-African women. (See figures 4 and 5).

Technique

Caudal epidural block can be performed in the prone or lateral decubitus position. The first step is to identify the sacral hiatus. It is essential that the skin over the caudal area is cleaned with an iodine or alcohol (70%) containing solution, which is allowed to dry. Then, using sterile technique, the caudal epidural space is entered using a short 23-gauge needle or a 22-gauge IV catheter. (See figure 3). The needle is inserted at a 60-degree angle and the needle is advanced until a “pop” is felt. (See figure 4) The needle is then lowered to a 20-degree angle and advanced an additional 2-3 mm to make sure the bevel is in the caudal epidural space, (See figure 5) if using a cannula withdraw the stylet and advance the cannula into the caudal space. Do not advance the needle or cannula any more than is necessary.

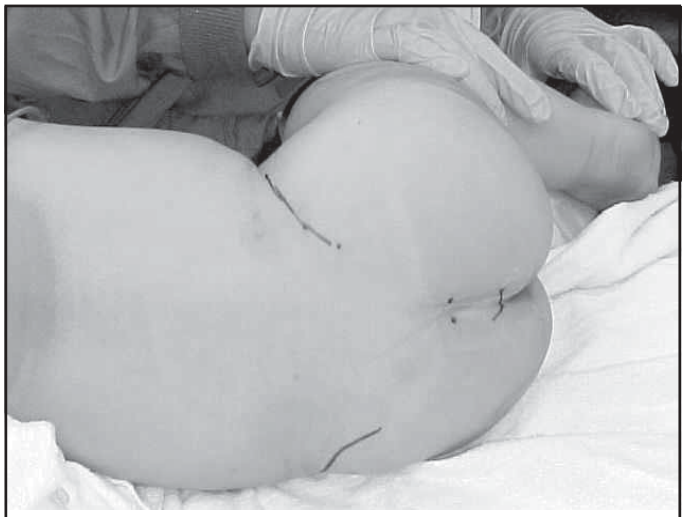


Figure 1: Positioning and Anatomy of Sacral Hiatus

Advancement of the cannula rather than the needle may reduce the incidence of inadvertent dural or vascular puncture and easy progression of the cannula is a good prognostic sign of success. Test aspiration should be gentle as vessel walls can easily collapse producing a false negative. If no blood or CSF is aspirated then the appropriate amount of local anesthetic is injected in small amounts, with repeated aspirations throughout the injection. An epinephrine containing test dose can be used to exclude intravascular injection. The most important test for correct placement (not including intravascular placement) is ease of injection. If the local anesthetic solution can be injected with little resistance, it is mostly likely in the correct space. If there is initial resistance or resistance develops over the course of the injection, the injection should be stopped and the needle location reassessed. There will be some increase in resistance as the potential space of the caudal epidural space is expanded, but this should be minimal.

If the hands are positioned as seen in figure 4, subcutaneous bulging, indicating subcutaneous injection will be detected by the thumb. Injection of air to confirm identification of the caudal space should be avoided because of the risk of air embolus. If the angle of insertion is too shallow, the needle may go subcutaneously. This produces an incomplete block and can cause pressure necrosis if large volumes of solution, especially epinephrine-containing solutions, are injected subcutaneously. If the angle of insertion is too steep, the needle may actually penetrate the vertebral bodies resulting in intraosseous injection and possible osteomyelitis.

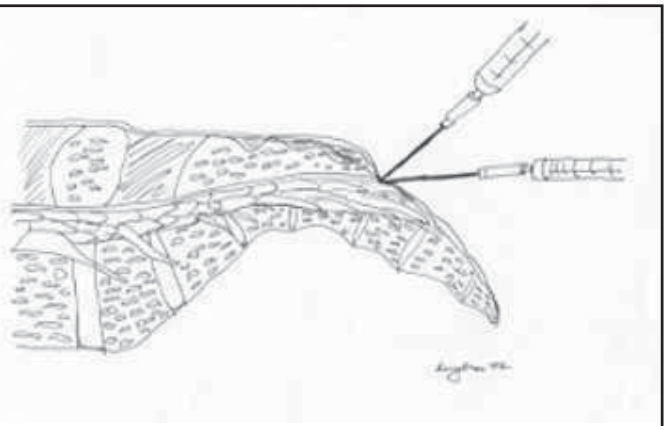


Figure 2: Anatomic Representation of Caudal Epidural Space

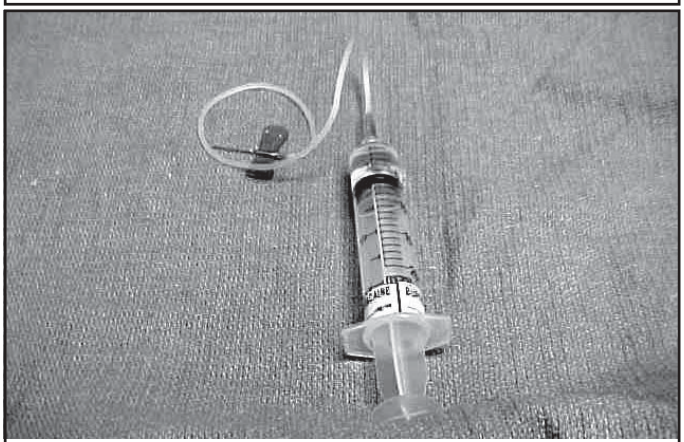


Figure.3: Syringe and Needle attachment for Caudal Epidural

'Single shot' technique: Caudal epidural anesthesia can be used as a "single shot" technique providing anesthesia limited by the duration of the local anesthetic that is chosen. This "single shot " may be repeated at the end of the surgery to prolong the analgesic effect into the postoperative period. Single shot caudal epidural anesthesia has a reported high success rate, frequently over 90%.

'Continuous / catheter' technique: an indwelling catheter can be placed to provide anesthesia of longer duration than single dose of local anesthesia would allow. Another advantage of an indwelling caudal epidural catheter is the ability to thread the catheter to a higher location in the epidural space and therefore achieve a higher, more localized block with less local anesthesia dose. Because of the close proximity of the perineum and likelihood of infection, a caudal catheter should not be left in situ ideally for longer than 36 - 48hrs.

Monitoring

Monitoring for caudal epidural anesthesia must include monitoring of ventilation, oxygenation, and circulation at least every 3 minutes. These can be accomplished with automated equipment such as automated blood pressure monitor, ECG, pulse oximeter, and capnograph. If automated monitoring equipment is not available, vital signs can be just as well be monitored with a sphygmomanometer, pericardial stethoscope and a finger on the temporal pulse. A means of temperature monitoring, such as an axillary thermometer is also needed if the anesthetist expects changes in body temperate due to loss of heat from surgery or cold operating theaters.

Local anaesthetic drugs and additives

Local anesthetics can be divided into two classes of compounds, Amides and Esters. The amides undergo metabolism by the liver, and the esters are hydrolyzed primarily in the plasma by cholinesterase. These different routes of metabolism are important in pediatric patients who may have immature liver function, especially neonates. Neonates have lower levels of alpha1 acid glycoprotein and albumin, 60% and 30% respectively compared to adults. This causes a reduction in the binding of protein bound drugs, such as amide local anesthetics, increasing the free (unbound fraction) thus increasing the possibility of toxic effects (max dose bupivacaine in neonate 1.5mg/kg). The volume of distribution of local anesthetics is larger in children than adults, which results in lower peak plasma levels, but this is counteracted by the reduction in the rate of elimination of local anesthetics in children.

Table 3. Armitage (1989) 0.25% Bupivacaine - Bupivacaine 0.19% for volume in excess of 20mls (one part 0.9% sodium chloride + three parts 0.25% Bupivacaine)

Segmental level of operation	Dose ml/kg
Lumbo-sacral	0.5
Thoraco-lumbar	1.0
Mid-Thoracic	1.25

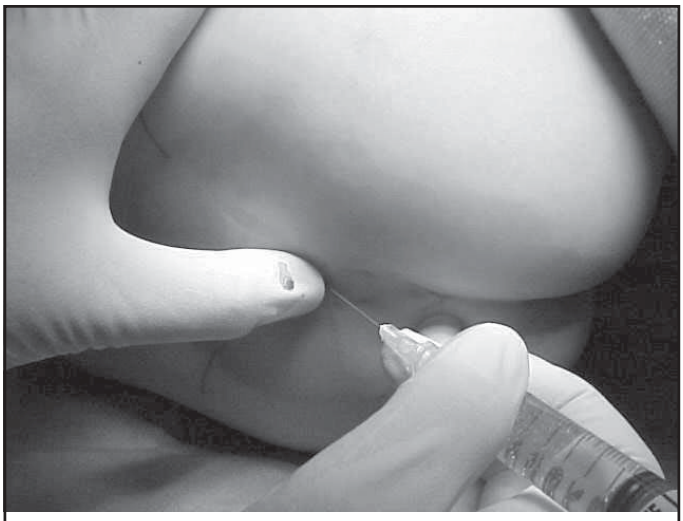


Figure 4: Initial Insertion

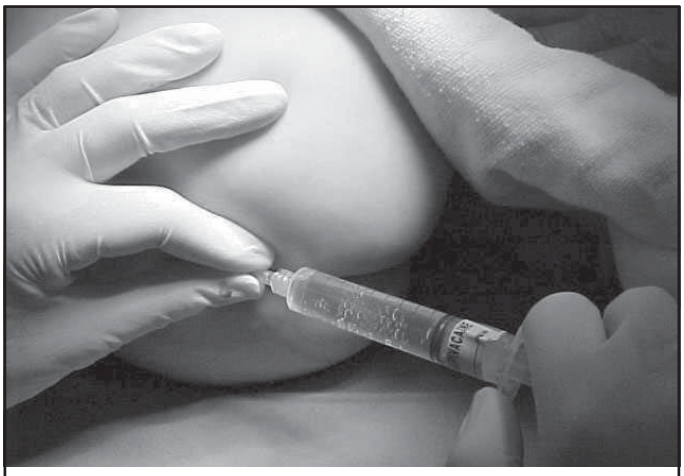


Figure 5 Repositioning and Completion of Epidural Injection

The maximum recommended doses of local anesthetics in children older than 4 weeks (doses should be reduced in neonates), are lidocaine 3mg/kg, (6mg/kg with adrenaline), bupivacaine 2-2.5mg/kg (addition of adrenaline will delay peak plasma level but will not extend duration). ***Note: malnutrition may also decrease albumin concentration causing reduced protein binding and increased free (unbound fraction) drug - increasing toxic effects.***

The spread of local anesthetic injected into the caudal epidural space in children less than 7yrs of age is predictable and correlates well with age and weight encouraging the use of formulae or normograms.

- **Bupivacaine**: provides reliable, long-lasting anesthesia and postoperative analgesia. An easy rule is 1ml/kg of Bupivacaine 0.25% with epinephrine 1:200,000 provides 3-6 hours of anesthesia for all procedures below the umbilicus. In Infants, less than 2.5 kg a more dilute solution is used (0.125% / 0.19 %) and the volume can be increased to remain below the toxic dose range. See table 1 - 3.

- **Adrenaline (epinephrine) 1:200,000**: combined with the local anesthetic solution can be used as a test dose. If the injection mistakenly occurs into a blood vessel, either vein or artery, the heart rate should increase more than 10 beats in 10 seconds after injection when epinephrine is added. However, this test dose is not 100% conclusive of intravenous injection.

- **Opioids** prolong analgesia in infants and children. Epidural opioids should be reserved for surgery in which catheterization is required and all children should be admitted to an area of the hospital where close monitoring and observation will take place. A dose of 50mcg/kg of preservative free morphine or diamorphine 30mcg/kg can be added to the local anesthetic solution. This will provide 12 to 24 hours of analgesia but can produce urinary retention, nausea, and itching and respiratory depression. However, a dose of 33microgramg/kg of preservative free morphine in the caudal epidural solution can provide prolonged analgesia, with less risk of delayed respiratory depression.

- **Clonidine**: (α2-adrenergic agonist, with spinal analgesic action). A dose of 0.5-1.0 microgram/kg improves the quality and duration of analgesia with bupivacaine without causing significant bradycardia or respiratory depression, lasting for up to 12 hours. Doses greater than 1 microgram/kg are often associated with increased sedation.

- **Ketamine**: an NMDA antagonist. In doses of 0.25 - 1.0 mg/kg, causes significant prolongation of postoperative analgesia, when compared to 0.25 % bupivacaine alone . There is no increase in adverse effects including delayed motor strength, time to micturation, postoperative sedation or postoperative nausea and vomiting. Though in doses higher than 0.5 mg/kg, the neuroleptic effects of ketamine appear to be more of a problem. Preservative free ketamine should be used at all times if possible, although animal studies have been performed using ketamine with benzothonium chloride demonstrating no histologic or pathologic changes in the spinal cord or roots but these have not been confirmed in human subjects.

Complications

The complications for caudal epidural anesthesia can be classified as:

- **Failed or incomplete block**. Between 5- 25 % of caudal epidural blocks can be considered “failed or incomplete”, this includes a number of different problems.

The anesthetist may not be able to identify the anatomic landmarks and are therefore unable to insert the caudal needle into the epidural space. This occurs frequently in small children with anomalies of those structures originating from the urogential ridge such as hypospadias, imperforate anus or chloacal atresia. In some of these children, it is impossible to palpate the sacral hiatus.

Also as the child advances in age the sacral plate tends to flatten out, making the insertion of the needle through the sacrococcygeal ligament more difficult. By far the easiest insertion is in the child below the age of 7 years. Though caudal anesthesia can and is accomplished in the older patient, it is technically more difficult.

- **Unilateral block**: less common than with lumbar epidural because the sacral/ caudal epidural space is bigger and requires more volume to fill. Patchy and one-sided blocks are rare with caudal epidural anesthesia but can result from too rapid injection of local anesthesia dose. Local anesthetics should be injected

slowly over 2 minutes after test dose. It is not infrequent though to have too low a block. This is a result of insufficient local anesthetic volume. Remember that the volume of the dose is often limited by total mg/kg dose of the local anesthetic that is chosen and must remain less than the toxic dose for that drug. (See table 2.) One solution to this problem is to give a more dilute solution. Decreasing the concentration allows the anesthetists to give a larger volume of local anesthetic, though it may decrease the intensity and duration of the block.

In my practice, I limit caudal epidural anesthesia to children who are still small enough for their mothers to carry. In this way, if the child is released from the hospital and still has some motor weakness of the lower extremities, the parent can carry the child and I am less fearful that the child will fall and injure himself.

- **Local anesthetic toxicity**

Intravascular injection: Even though most local anesthetics have close to 100% bio-availability from the epidural space, they are absorbed over time. Intravascular injection allows immediate bioavailability of the total dose of the local anesthetic with consequent systemic toxicity if the peak plasma concentrations are with the toxic range. Peak concentration is lower if drugs are injected slowly. As the extradural veins have no valves, local anesthetic can enter the cerebral circulation by retrograde flow, producing convulsion at doses lower than recommended maximum safe doses. If large volumes of local anesthetic are given (>10mls) the anesthetist should aspirate again in the middle of the injection as the expansion of the potential epidural space can displace the tip of the needle. The anesthetist should be aware of potential intravascular injection throughout the injection.

Absorption / overdose: If either incorrect dosing or volume is injected then absorption of the local anesthetic will result in a rise in plasma level over time into the toxic range (not immediately as with intravascular injection). It is important to strictly follow the guidelines of local anesthetic dose given in table 2.

- **Dural puncture (intrathecal injection)**. The spinal cord typically ends at the first lumbar vertebra in the adult but can be as low as the third or fourth lumbar vertebra in the neonate and premature infant, with the corresponding dural sac extending 1-2 vertebral segments below this. There is considerable variation in the level of termination of the spinal cord. If unintentional dural puncture is performed, a large dose of local anesthetic is injected intrathecally. This will produce a 'total spinal block', characterized by sudden apnea, unconsciousness and dilated pupils. There is usually little in the way of haemodynamic disturbance in young children and babies.

- **Intraosseous injection**. An intraosseous injection is equivalent to an intravenous injection.

- **Penetration of the sacrum**. In infants and young babies, the vertebral bodies can be soft due to incomplete calcification and the anesthetist can pass the needle into the body of the sacrum and through the vertebral body into the pelvis damaging either pelvic viscera or aorta.

- **Bleeding and infection**: haematoma and abscess formation are very uncommon after caudal epidural anesthesia but can result in serious and permanent neurologic damage involving the spinal cord or cauda equina.

Conclusions

Caudal epidural anesthesia is a safe and effective method of anesthesia in pediatric patients. It can be used as the sole anesthetic agent or combined with general anesthesia to reduce both intraoperative anesthetic requirement and postoperative need for additional analgesia. The addition of opioids, clonidine or ketamine can significantly enhance and prolong the anesthetic effects, even when used in minimal amounts and can reduce the need for postoperative narcotics in some of the sickest and smallest of children. However, as with all regional anesthetic techniques, extreme diligence should be taken to insure sterility and avoid intravascular injection or toxicity due to overdose of local anesthetic solutions.

Further Reading

1. Update in Anaesthesia 1998 No. 8

BRONCHOSCOPY FOR A FOREIGN BODY IN A CHILD

Dr P Dix, Exeter, UK

Introduction

Inhalation of a foreign body (FB) is a potentially life threatening event, with boys in the age range 1 to 3 years most at risk. Resistance to gas flow is related to the fourth power of the radius, so a small reduction in airway radius in a child will result in a large increase in resistance to airflow. Inhalation of an organic FB may result in airway hyperreactivity and mucosal oedema. The occurrence of oedema in addition to the physical presence of the FB results in a rapid increase in airway resistance. Coupled with the high oxygen consumption of infants and small children, hypoxia may rapidly occur.

Presentation

The presentation may be acute, with symptoms and signs of laryngeal or tracheal obstruction (cough, choking, respiratory distress, cyanosis, stridor, tachypnoea), or with signs of obstruction of a main bronchus (respiratory distress, tachypnoea, wheeze or absent breath sounds on the affected side). There may be no clinical findings, even following a clear history of FB inhalation. The presentation may be more insidious, with chronic cough, chest infection, signs of mixed upper and lower airway collapse and consolidation affecting one or more lobes. Air trapping might be seen radiographically on expiratory films, due to a "ball valve effect". Initially an air bronchogram may be seen, with later evidence of atelectasis distal to the obstruction.

The history may help in the diagnosis, for example sudden onset of respiratory distress while playing with small objects, but FB aspiration should be considered in every child presenting with cough or stridor, in the absence of clear symptoms and signs pointing to another aetiology. It should also be remembered that an oesophageal FB, usually hypopharyngeal, may present with respiratory distress due to external compression of the trachea.

Preparation, investigation and examination of the child

To a large extent this will be dictated by the clinical condition of the child. If time allows the usual preoperative assessment of the child should be made, with particular attention to examination of the airway and chest. The presence of an inspiratory wheeze (glottic or supraglottic) or expiratory wheeze (infraglottic) may help to localise the site of a FB. If the child is stable a chest radiograph may be helpful in localising the FB, although the majority of FBs will not be radio-opaque. In the acute situation few other investigations are indicated. The child should be starved according to the recommended guidelines, but this will clearly not be possible in a child with acute respiratory distress. Sedative premedication should not be used.

Anaesthesia

General anaesthesia will be required to perform bronchoscopy. The anaesthetic machine and other equipment should be checked, especially suction equipment. A range of sizes of endotracheal tubes should be available, in case intubation is urgently required, bearing in mind that the presence of airway oedema reduces the tracheal diameter.

Monitoring including pulse oximetry, ECG, non-invasive blood pressure, and capnography should be applied. Intravenous access should be secured prior to induction, but if the child is distressed this can be performed immediately after induction.

A senior anaesthetist and ENT surgeon should be present at induction, along with the most skilled assistant available. Inhalational induction is recommended using either sevoflurane or halothane in 100% oxygen. There is much debate about the relative advantages of halothane and sevoflurane. Sevoflurane causes less airway irritation and is more cardiovascularly stable than halothane. Halothane has a longer lasting anaesthetic effect, allowing more time for airway manipulation without fear of the child becoming too lightly anaesthetised. In many locations halothane is more readily available than sevoflurane. Halothane is associated with more cardiovascular instability than sevoflurane, especially arrythmias, which are worsened in the presence of hypercapnia and high circulating levels of catecholamines. The choice will be dictated by personal experience and preference, and also local availability. Ether is still used in some centres.

Spontaneous ventilation is recommended, although occasionally it might be necessary to assist ventilation with gentle mask ventilation. Spontaneous ventilation reduces the risk of hyperinflation of the lung and pneumothorax, and is also less likely to dislodge the FB distally. After induction , a cannula should be sited, if it has not been sited prior to induction, and nitrous oxide should be discontinued. When the child is deeply anaesthetised, which can take a long time due to the reduced airflow, laryngoscopy should be performed, and the larynx and trachea sprayed with 4% lignocaine (maximum dose 4mg/kg). After a few minutes, the ENT surgeon can perform rigid bronchoscopy.

During bronchoscopy, anaesthesia should be maintained by connecting a T-piece to the sidearm of the Stortz bronchoscope. Intubation should not be performed prior to rigid bronchoscopy, due to the risk of dislodging or fragmenting the FB, with a risk of complete airway obstruction. If desaturation during bronchoscopy of one lung occurs, the bronchoscope can be withdrawn into the trachea to allow re-oxygenation of both lungs, before a further attempt at bronchoscopy is made. The telescope might also need to be removed from the bronchoscope to allow adequate gas flow. During bronchoscopy careful observation of chest movements should be made.

After removal of the FB the airway can be maintained using a face mask, endotracheal tube or laryngeal mask. The anaesthetic is discontinued, 100% oxygen is administered, and the child observed carefully until awake and extubated.

Postoperatively, the child must be monitored for signs of stridor and airway obstruction due to oedema. Humidified oxygen is recommended for 24 hours, and if worsening stridor occurs, nebulised adrenaline 1:1000 may be useful (0.5ml/kg, maximum 5ml). Dexamethasone 250mcg/kg i.v. at induction, followed by 100mcg/kg 6 hourly for 24 hours has also been recommended.

The use of flexible fibreoptic bronchoscopy is described for the retrieval of bronchial FB. In general this is not recommended, as rigid bronchoscopy has several clear advantages - complete airway control, a better view of the bronchial tree, and larger channels through which to pass instruments and withdraw FBs. However, in older children, the rigid bronchoscope only allows limited access to the upper lobes and more distal airways.

Types of bronchoscope

Two types of rigid bronchoscope are available. The older Negus bronchoscope is the original rigid bronchoscope, and has a tapered shape. It is no longer used in most UK centres.

The Stortz ventilating bronchoscope is the most commonly used rigid bronchoscope in most centres. Bronchoscopes are available in a variety of sizes and lengths from 2.5mm internal diameter. This allows safe examination of all children, including neonates. The Hopkins rod lens telescope is inserted through the lumen, allowing a clear view of the endobronchial tree. A wide range of instruments are available to enable retrieval of a FB, as well as other therapeutic procedures.

A T-piece circuit is attached to the sidearm of the bronchoscope to allow delivery of oxygen and anaesthetic gases during the procedure. The presence of the telescope, with the viewing end occluded, results in a closed system, through which spontaneous or controlled ventilation may occur. The system is open when the telescope is removed, allowing only spontaneous ventilation. The telescope occupies a significant proportion of the bronchoscope, through which expiration must occur. It may be necessary to remove the telescope periodically to allow adequate breathing through the lumen of the bronchoscope, especially when using smaller diameter bronchoscopes.

A Sanders injector can be attached to the sidearm to enable controlled ventilation, when the telescope is not being used. It uses the Venturi principle to entrain oxygen-enriched air. The entrainment of air means that it is not possible to deliver high oxygen concentrations, which may not maintain adequate patient oxygenation. Anaesthesia is maintained using intravenous anaesthetic agents. In smaller children barotrauma can easily occur, so this may not be the technique of choice.

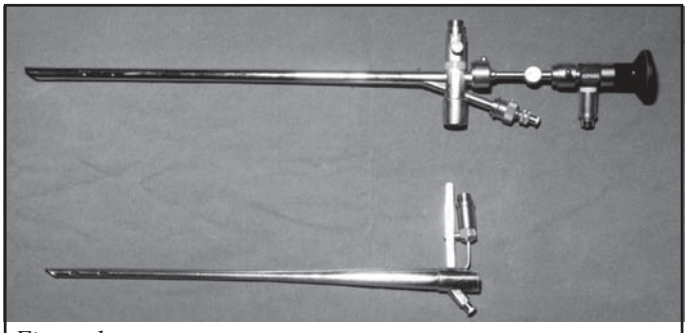


Figure 1:

Case history

A 4 year old boy presented to the emergency department with a history of coughing and choking while he was lying on his back playing with some coins. He had a short spell of cyanosis which lasted a few seconds. He reported "swallowing" a coin.

On examination he was upset, but well. He was apyrexial, and had a respiratory rate of 20/minute with no recession. Chest auscultation revealed reduced breath sounds in the right lower lobe. A chest Xray showed a radio-opaque sphere in the area of the right main stem bronchus. Ametop local anaesthetic cream was applied to both hands.

The child was transferred to the operating theatre. The anaesthetic machine, suction equipment, and laryngoscopes were checked. The consultant ENT surgeon was present. A pulse oximeter and ECG leads were applied, and a 22G cannula was sited. Atropine 10mcg/kg and dexamethasone 250 mcg/kg were given i.v. Anaesthesia was then induced using 3% halothane in oxygen breathed spontaneously via a face mask and Ayres T-piece circuit. After several minutes, when deep anaesthesia had been achieved, laryngoscopy was performed and the cords sprayed with 2mls of 4% lignocaine. A 3.5 rigid bronchoscope was introduced into the trachea. The coin was retrieved uneventfully from the right main bronchus, and the bronchoscope was withdrawn from the airway. The halothane was discontinued, and the airway maintained with a face mask until the child was fully awake.

Humidified oxygen was given overnight on the ward, and the child was monitored with pulse oximetry. The child was discharged the next day.

Learning points

1. Airway obstruction by a foreign body reduces tracheal gas flow, and prolongs the time taken to induce anaesthesia using an inhalational induction.

2. Organic foreign bodies can cause airway oedema and hyperreactivity, which may be worsened by anaesthetic gases.

3. A foreign body may be dislodged at any time, resulting in complete airway obstruction. For this reason, spontaneous ventilation must be maintained, and rigid bronchoscopy performed prior to intubation.

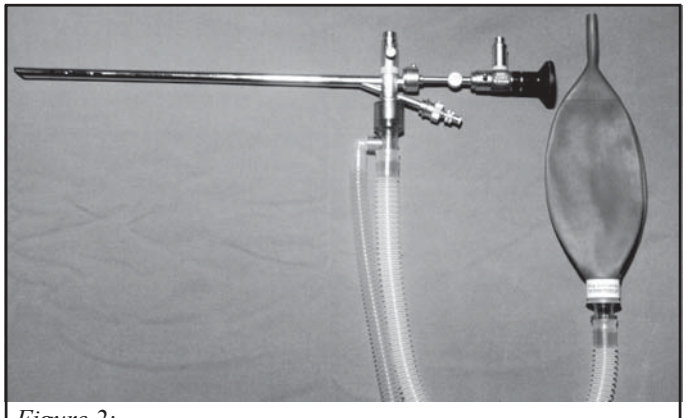


Figure 2:

FROM THE JOURNALS

Journal Skimmers: Anaesthesia - Dr Peter J. Shirley; Anesthesia and Analgesia - Dr Yue Dong; Anesthesiology - Dr Mike Girgis British Journal of Anaesthesia - Dr Paul Sice; Canadian Journal of Anesthesia - Dr Aneeta Sinha

Plasma paracetamol concentrations after different doses of rectal paracetamol in older children. A comparison of 1g vs. 40mg/kg. Howell TC, Patel D *Anaesthesia* 2003;**58**:69-73.

This paper compared the levels of paracetamol reached following rectal administration in healthy children to see if therapeutic levels (>10μg/ml) were present. The authors make initial observations that paracetamol is often given rectally to children in accident and emergency departments, operating theatres and intensive care units for both antipyretic and analgesic effects. They also note that sub-therapeutic doses of paracetamol have been given in the past and that initial rectal doses of 35 - 45mg/kg are now recommended. Whilst the therapeutic range for analgesia is not well established other work has demonstrated little or no analgesic benefit below a plasma level of 10μg/ml.

All children were undergoing elective spinal surgery and were excluded if paracetamol had been administered within 24 hours or if they were on regular paracetamol before attending hospital. Children over 25kg were enrolled and randomised to receive either a dose of 1g or 40mg/kg rectally. Commercially available suppositories in 60, 120, 240 and 500mg preparations were used to deliver as close to the required dose as possible. No suppository was cut, as it is recognised that paracetamol is not distributed evenly throughout each suppository.

Twenty-two children were included and plasma paracetamol levels were measured at 2, 3, 4 and 5 hours post-dose. Blood was taken from an indwelling central line inserted as part of the anaesthetic for the surgical procedure. The mean plasma level at 2, 3, 4 and 5 hours in the group receiving 1g paracetamol did not achieve a therapeutic level, whereas the mean plasma level in the 40 mg/kg group was in the therapeutic range at all times (three children in this second group did have plasma levels that fell just short of the therapeutic range). When subjected to statistical analysis there was a significant difference between the two groups ($p = 0.01$). The toxic level of plasma paracetamol is considered to be 120μg/ml; the maximum level reached in this study was 45μg/ml.

The conclusion reached is that 1g of rectal paracetamol is insufficient in children weighing >25kg and that doses of 40mg/kg should be used. This dose will result in plasma paracetamol levels well below those associated with toxicity. Paracetamol is also known to have a morphine sparing effect of >36% when administered correctly.

The paper concludes with questioning the effectiveness of rectal doses of 1g of paracetamol in adults and asks whether doses of 2g would be more appropriate. However, they point out that this inference cannot necessarily be acted on, as adults metabolise paracetamol differently to children and are more prone to hepatoxicity. More work would be required to establish the appropriate dose in adults.

Volatile agents to avoid ventilating asthmatics
Baigel G. *Anaesthesia and Intensive Care* 2003;**31**:208-10

Six patients with severe asthma (five of them with a previous history of mechanical ventilation) and with severe dyspnoea, tachycardia, hypertension and blood gases indicating the need for immediate intubation and mechanical ventilation were recruited. All patients were receiving six hourly hydrocortisone, salbutamol and ipratropium bromide nebulisers, intravenous aminophylline as bolus and infusion and terbutaline infusions. Ketamine was not being given.

Instead of intubating the patients, they were given Halothane 0.5% (five patients) or Sevoflurane 0.25% (one patient) in oxygen. Administration was via a facemask connected to a Bain circuit using a Tech 3 vaporizer. Patients were asked to hold the facemask themselves, thus avoiding over-sedation and maintaining verbal contact.

All patients responded immediately and showed improved blood gases after 15 minutes. The wheeze disappeared rapidly, the heart rate and respiratory rate returned to near normal.

The vapour was administered for between 45 and 173 minutes, and five patients needed only this one period of treatment during their hospital stay. One patient needed intubation later during a second attack.

The bronchodilatory effect of halothane is well recognised, although the mechanism of action is still unclear. A direct β-mimetic effect as well as a blockade of vagal reflexes is possible (although the effect is not attenuated by β-blockers). A second mechanism is the sedating and therefore anxiolytic effect of low concentrations of Halothane. The same mechanisms are likely to apply for Sevoflurane.

Except for one episode of ventricular ectopics in one patient (with high blood levels of aminophylline) no complications were observed. The author therefore judges the therapy a safe and effective method to prevent intubation and mechanical ventilation in severe asthma attacks.

Lidocaine sprayed down the endotracheal tube attenuates the airway-circulatory reflexes by local anesthesia during emergence and extubation. Daelim Jee and So Young Park *Anesthesia and Analgesia* 2003;**96**:293-297

The authors designed a study to compare the reflex response after lidocaine spray through the endotracheal tube and intravenous administration during extubation. Seventy-five patients receiving a standard anaesthetic were divided into three groups: controls, those receiving 1mg/kg 2% lidocaine sprayed down the ET 5 min before extubation and those given 1mg/kg 2% lidocaine IV 3 minutes before extubation. Patients BP, HR, number of coughs and rate of coughing were recorded from 5 min before until 5 min after extubation.

The group receiving ET lidocaine spray during extubation had significantly less numbers of coughs per patient during extubation compared to controls and the IV group (4.5 +/- 3.7 vs. 10.2 (6.0 and 7.8 (+/-4.6, $p<0.01$ and $p=0.06$). Although the cardiovascular parameters (systolic/ diastolic blood pressure, heart rate) changed compared to baseline levels, those in the ET group increased less compared with control and IV groups.

Lidocaine is relatively safe and has various applications. ET spray is not a new idea. Careful monitoring of cardiovascular and respiratory vital signs is needed to exclude any potential adverse effects (systematic toxicity). This study shows that lidocaine can be used topically to prevent extubation reflex responses (especially coughing).

Effects of postoperative non-steroidal anti-inflammatory drugs on bleeding risk after tonsillectomy. *Anesthesiology* 2003;**98**:1497-1502

The authors performed a literature search for pertinent studies published between 1966 and 2001. The quality of each study was assessed using the Jadad composite scale and only those that were randomised, double-blind and with a score greater than 3 were included. Outcome measures were the need for surgical electrocautery to stop postoperative bleeding, or postoperative bleeding requiring a change in management i.e. admission to the emergency department, readmission to hospital, or blood transfusion. Bleeding was defined as primary if it was within 24hrs after surgery, and secondary if it occurred later. They found 90 articles in total of which only 20 were randomised controlled trials and of those only seven met the selection criteria to be included. All seven were published in or after 1995 and included a total of 505 patients. Perioperative NSAIDs used were ketorolac, ketoprofen or ibuprofen. Placebos received saline, paracetamol, codeine, morphine or meperidine (pethidine).

Of the 243 controls, 13 (5.3%) had primary or secondary postoperative bleeding, 7 of these being secondary. Of the 262 patients who received NSAIDs 24 (9.2%) had postoperative bleeding (odds ratio 1.8; 95% CI 0.9-3.4), 15 of these being secondary. Only two of the control patients (1 primary and 1 secondary) required re-operation for haemostasis, compared with 11 in the NSAID group (5 primary and 6 secondary).

This produces a significant difference in the rate of reoperation for haemostasis: 0.8% for controls compared with 4.2% for those given NSAIDs (odds ration 3.8; 95% CI 1.3-11.5; P=0.02).

This study suggests that NSAIDs increase the incidence of re-operation for post-tonsillectomy bleeding five fold. 'Number needed to harm' was 29 (95% CI 17-144) for re-operation despite some patients receiving only a single dose of NSAIDs ie the use of NSAIDs in 29 patients would result in haemorrhage severe enough to require reoperation in at least one patient. Reoperation for haemorrhage from tonsillectomy site bleeding is associated with a high risk of morbidity related to pulmonary aspiration and difficult intubation. It was concluded that conventional NSAIDs should not be used after tonsillectomy. The authors also suggested that since specific COX-2 inhibitors do not inhibit platelet aggregation in vitro, these may provide similar pain relief without the associated risk of bleeding.

Prevention of postoperative nausea and vomiting after spinal morphine for Caesarian section: comparison of cyclizine, dexamethasone and placebo. Nortcliffe SA, Shah J and Buggy DJ. *British Journal of Anaesthesia* 2003;**90**:665-70

This study investigated the efficacy of cyclizine 50mg, dexamethasone 8mg and placebo in preventing postoperative nausea and vomiting (PONV) after intrathecal morphine for Caesarian section.

ASA I and II patients presenting for elective Caesarian section were randomised to receive one of the antiemetics or placebo. Following ranitidine premedication, anaesthesia to the T4 dermatome was established with hyperbaric bupivicaine 0.5% 2ml, with 10mcg of fentanyl and 0.2mg of preservative-free morphine. All patients also received intravenous crystalloid (with ephedrine boluses for hypotension) and rectal diclofenac. The primary outcome measure was the incidence of nausea in the first 24hrs. Nausea severity, incidence of vomiting and VAS pain assessments were also collected at 3,6,12 and 24 hrs postoperatively.

30 patients per group completed the trial, the groups being closely matched. The incidence of PONV, and the need for prochlorperazine rescue antiemetic, were all significantly reduced in the cyclizine group. The incidence of nausea was 67% in the placebo group compared with 60% for dexamethasone and 33% for cyclizine. The incidences for vomiting were similar, and rescue antiemetic was needed for 4 patients after cyclizine, compared with 17 after dexamethasone. Overall patient satisfaction scores were significantly higher after cyclizine.

This study reiterated the high incidence of PONV after intrathecal morphine. It is interesting that dexamethasone was little better than placebo having been shown to be effective after general anaesthesia and epidural morphine, and the group speculate a different mechanism of PONV for the epidural and intrathecal routes.

Spinal anaesthesia is commonly employed worldwide for Caesarian section and although intrathecal morphine can provide good analgesia up to 24hrs postoperatively, PONV is common. Cyclizine, which is widely available, well tolerated and cheaper than the new 5-HT3 antagonists, has been shown to significantly reduce this problem and improve patient satisfaction.

Dexamethasone reduces postoperative vomiting and pain after paediatric tonsillectomy. Elhakim M, Ali N, Rashed I, Riad M K, Refat M *Canadian Journal Of Anesthesia* 2003;**4**: 392-397

The aim of this study was to evaluate the effects of a single dose of dexamethasone on the incidence and severity of postoperative vomiting and pain in children undergoing electrocautery tonsillectomy under a standardised general anaesthetic.

In a double-blinded study, 120 patients were randomly allocated to receive either dexamethasone 0.5 mg/kg (maximum 8 mg) IV or an equivalent volume of saline preoperatively. The standardised anaesthetic used involved premedication with oral midazolam 0.5mg/kg (maximum 20mg), induction with sevoflurane and suxamethonium 1mg/kg to facilitate intubation. Maintenance was

with sevoflurane/N_2O/O_2 (FiO_2 0.4). All children received fentanyl 1 mcg/kg before surgery and 20mL/kg lactated Ringer's solution during the operation. Prior to awake extubation, gastric contents were suctioned via an orogastric tube. A standardised starving regime was implemented prior to the anaesthetic.

The incidence of early and late vomiting, need for rescue antiemetics (metoclopramide 0.15 mg/kg iv), time to first oral intake, time to first demand of analgesia and analgesic consumption (paracetamol 30mg/kg PR 6 hourly, as necessary or pethidine IV for rapid pain relief) were compared in both groups. Pain scores used included the Children's Hospital Eastern Ontario Pain Scale (CHEOPS), "faces" and a 0-10 visual analogue pain scale (VAS).

Compared with placebo, dexamethasone significantly decreased the incidence of early and late vomiting ($p < 0.05$, $p < 0.001$ respectively). The administration of dexamethasone reduced the overall incidence of postoperative vomiting from 56% to 20% when compared with saline ($p < 0.001$). Fewer patients in the dexamethasone group required antiemetic rescue ($p < 0.01$). The time to first oral intake was shorter and the time to first dose of analgesic was longer in the dexamethasone group ($p < 0.01$). Pain scores were significantly lower in the dexamethasone group at 30 minutes ($p < 0.05$), 4 hours ($p < 0.05$), 6 hours ($p < 0.05$), 12 hours ($p < 0.01$) and 24 hours ($p < 0.01$).

In conclusion, a prophylactic, pre-operative, single dose of dexamethasone (0.5 mg/kg) appears to reduce both postoperative vomiting and pain in children after electrocautery tonsillectomy.

Reprinted from 2002 CME lectures by kind permission of the Royal College of Anaesthetists and the Association of Anaesthetists of Great Britain and Ireland

HYPERTENSIVE DISORDERS OF PREGNANCY

Dr DM Levy, Queen's Medical Centre, Nottingham, UK.

This term, used in the *Reports on Confidential Enquiries into Maternal Deaths in the United Kingdom*,[1] covers the spectrum of disorders encompassing pre-eclampsia, eclampsia, and the syndrome of haemolysis, elevated liver enzymes and low platelets (HELLP). Acute fatty liver of pregnancy and other microangiopathies of pregnancy are related disorders that can arise simultaneously.[2]

Pre-eclampsia is a multisystem disorder of generalised vasospasm. It is thought that placental ischaemia might cause trophoblastic fragmentation. Widespread platelet aggregation on these fragments could release *serotonin*. This mediator would account for the widespread vasopasm and consequent endothelial cell dysfunction. Cardiovascular, central nervous, renal, respiratory, hepatic and coagulation systems are affected to variable extents.[3]

The circulating volume is expanded relative to the non-pregnant state, but less so than in normal pregnancy. The demonstration from pulmonary artery (PA) catheter data of flow-dependent oxygen consumption indicates that severe pre-eclampsia is associated with an oxygen extraction defect at the tissue level, similar to that found in critically ill patients with multi-organ failure.[4]

Pre-eclampsia is characterised by the variability of its presentation and rate of progression. The only definitive treatment is delivery of the fetoplacental unit. However, in the 24 hours following delivery, clinical and laboratory indices of the disease often continue to deteriorate before recovery begins.

Cardiovascular

Before delivery, the aim is to prevent intracerebral haemorrhage secondary to uncontrolled hypertension, whilst preserving uteroplacental blood flow and maternal renal perfusion. A systemic arterial pressure (BP) of >170/110mmHg is an indication for urgent treatment - although BP should not be reduced acutely to below 130/90mmHg.[5] For acute therapy, the following are suitable:

- A vasodilator such as **hydralazine** is effective given initially as 2.5 - 5mg boluses every 20 mins, thereafter by continuous infusion. Side effects of headache, tremor and vomiting can mimic impending eclampsia. Prior administration of a colloid bolus of no more than 500ml has been advocated to prevent acute fetal compromise secondary to vasodilatation.

- **Labetalol** has both vasodilating (αadrenergic blocking activity and (βblockade which mitigates tachycardia. 10 - 20mg increments (up to 100mg) at 5-10min intervals are usually effective.

- The calcium channel antagonist **nifedipine** has been used as an antihypertensive agent in pre-eclampsia, although there is a possibility of myocardial depression and excessive hypotension if it is given in conjuction with magnesium sulphate ($MgSO_4$), also a calcium antagonist.

- **Ketanserin**, a serotonin-2 receptor antagonist, has been shown to have an antihypertensive effect comparable to that of hydralazine, but with a lower incidence of headache, visual complaints, and nausea/vomiting. Notably, HELLP, oliguria, pulmonary oedema and placental abruption developed less frequently in women treated with ketanserin. This selective serotonin-2 blocker, currently unlicensed in the UK, appears to work at the level of the disturbed platelet-endothelial cell interaction (rather than acting merely as a vasodilator).[6]

Pre-eclampsia alone rarely causes cardiac failure in young, otherwise healthy women. Only a minority of pre-eclamptic women who develop pulmonary oedema have reduced systolic function and a dilated left ventricle.[7] Peripartum cardiomyopathy is unexplained heart failure associated with pregnancy in previously healthy women without detectable organic heart disease. The nature of its relationship with pre-eclampsia is unclear.8 Women with pre-eclampsia and pulmonary oedema should have echocardiography to establish whether there is evidence of cardiomyopathy, which might benefit from early treatment with ACE inhibition. Iatrogenic fluid administration, steroids given to promote fetal lung maturation, and (2 agonists (e.g. ritodrine) given for tocolysis, can all contribute to the development of pulmonary oedema.

Renal

Glomerular involvement causes proteinuria. The appearance of ≥300 mg protein over 24 hours was a traditional diagnostic feature of pre-eclampsia. However, proteinuria is no longer considered an *inevitable* feature of the disease.[9] Its degree and rate of increase are not - unlike serum uric acid concentrations - important predictors of maternal or perinatal outcome. Hyperuricaemia precedes significant proteinuria in pre-eclampsia and is thought to result from either enhanced tubular reabsorption of uric acid, or breakdown of nuclear (and therefore purine) rich syncytiotrophoblast.

Up to 6 hours of oliguria (urine output <30 ml/hr) after delivery is extremely common, and does not necessarily imply volume depletion. Acute tubular necrosis is exceptionally rare in the absence of a compounding factor such as major haemorrhage, or injudicious administration of a non-steroidal anti-inflammatory drug.

- There is little evidence upon which to base management of fluid balance in pre-eclampsia - no large prospective outcome studies have been performed.[10] No study has shown that crystalloid or colloid is superior. The use of crystalloid may reduce plasma colloid oncotic pressure, but the longer half-life of colloid may contribute to circulatory overload during the period of postpartum mobilisation of the increased extracellular fluid volume of pregnancy. Fluid input and output must be charted meticulously. If Syntocinon® is to be continued post-delivery, it should be administered in small diluent volumes by syringe pump (e.g. 40 units in 40ml Normal saline at 10ml/hr).

- Unless delivery has been complicated - for example by haemorrhage (e.g. abruption) or sepsis (e.g. chorioamnionitis), invasive monitoring is indicated only rarely. Measurement of CVP will help substantiate a diagnosis of *hypovolaemia*, and assist its correction. A brachial 'long' line is vastly safer than other approaches, particularly in the presence of coagulopathy. Airway obstruction secondary to inadvertent carotid puncture in the course of attempted jugular venous cannulation has been responsible for maternal mortality, and a number of near misses.

- Volume expansion can reasonably be undertaken if CVP (5mmHg. However, the circulating volume should be considered as *full* if CVP is >5mmHg. Minimal i.v. fluids (e.g. Normal saline or Hartmann's at 20ml/hr) will suffice.

- Dopamine 1-5μg/kg/min has been shown to increase urine output in oliguric postpartum pre-eclamptic patients who have not responded to a 300ml crystalloid challenge, although the benefit is questionable.

- There is a disparity between CVP and pulmonary artery wedge pressure (PAWP) at CVP measurements of greater than 6 mmHg, when PAWP may be *considerably higher*, as a result of left ventricular dysfunction.

Pulmonary artery (PA) catheterisation is warranted for situations where the benefits of knowing PAWP, cardiac output, and systemic vascular resistance (SVR) are judged to outweigh the risks. Potential indications are pulmonary oedema unresponsive to diuretic therapy, persistent severe oliguria, and hypertension refractory to standard therapy. Although oesophageal Doppler appears to underestimate cardiac output measured by PA catheter, the direction and magnitude of sequential changes are accurately reflected.[11]

CNS

Fits occurring in late pregnancy and labour should be regarded as eclamptic unless proven otherwise. Alternative diagnoses include epilepsy, intracerebral pathology (tumours, vascular malformations, haemorrhage), water intoxication, local anaesthetic toxicity, and amniotic fluid embolism (anaphylactoid syndrome of pregnancy).

Eclampsia complicates about 1:2000 maternities in Europe and developed countries. In 32 (60%) of 53 cases of eclampsia in two American centres over 10 years, seizures were the first manifestation of a hypertensive disorder of pregnancy.[12] When asked subsequently about prodromal symptoms, severe headache was noted by two thirds, and visual symptoms in one third. Only 7 women had a diagnosis of severe pre-eclampsia. This study suggests that eclampsia is not necessarily preceded by mild pre-eclampsia which has progressed to severe pre-eclampsia.

In the event of eclampsia

- Maintain airway patency and give 100% oxygen. Avoid aortocaval compression, and attempt to prevent trauma to the mother and fetus.

- Most initial fits will be self-limiting. If not, administer without delay a loading dose of 5g (10ml of 50%) **Magnesium sulphate** ($MgSO_4$) over at least 5 minutes. If i.v. Mg has already been started, treat with a further 2g bolus, unless a recent serum concentration was at the high end of the therapeutic range (see below).

- If $MgSO_4$ is not readily available, **diazepam** 10mg i.v. over 2 min is an appropriate alternative anticonvulsant. Phenytoin has been rendered obsolete in pre-eclampsia/eclampsia.

- After the convulsion has terminated, examine the mother for signs of pulmonary aspiration (tachypnoea, crackles/wheeze), and institue SpO_2 monitoring. Ensure that an obstetrician or midwife determines the fetal heart rate without delay. Fetal compromise secondary to maternal hypoxaemia or placental abruption will signal the need for emergency Caesarean section under general anaesthesia.

- In the rare event of a continuing seizure or difficulty maintaining maternal oxygenation, summon skilled anaesthetic assistance and transfer swiftly to theatre. Induce general anaesthesia with thiopental or propofol, ensure cricoid pressure is applied, and intubate the trachea following neuromuscular blockade with succinylcholine.

- Eclamptic patients who have an emergency GA Caesarean section should be transferred to ICU for a period of sedation and ventilation. Ideally, brain imaging should be performed *en route* in order to exclude intracranial haemorrhage and ascertain whether there is evidence of cerebral ischaemia.

- Treat as for a non-pregnant patient with cerebral ischaemia secondary to traumatic brain injury. If neuromuscular blockade is used, neurophysiological monitoring (e.g. cerebral function analysing monitor) will allow identification of further seizure activity.

- In the presence of therapeutic serum Mg concentrations, doses of non-depolarising neuromuscular blockers must be reduced, and the degree of block monitored with a peripheral nerve stimulator.

Magnesium sulphate

$MgSO_4$ has been shown to reduce the incidence both of eclampsia complicating severe pre-eclampsia, and further fits in eclamptic patients. In the Magpie study[13] over 10 000 women with hypertension and proteinuria were randomised to magnesium sulphate or placebo. Women allocated Mg had the incidence of eclampsia halved compared to those who had placebo. However, the number needed to treat was 91 (for each patient who had a seizure prevented, 91 patients received Mg).[14]

Mg reduces both systemic and cerebral vasospasm by antagonism of calcium. Increased oxygen delivery and consumption accompany systemic vasodilatation.[4] The anticonvulsant action of Mg is consistent with the theory that eclamptic seizures are caused by cerebral vasospasm. Nimodipine, a calcium channel antagonist (used extensively in neurosurgical practice to reduce cerebral ischaemia after subarachnoid haemorrhage) has also been used successfully to treat eclampsia. Resolution of cerebral ischaemia has been imaged by magnetic resonance. Nimodipine 1 mg/hr by i.v. infusion (increasing to 2mg/hr after 20 minutes) reduced SVR and BP in a small series of eclamptic women. Nimodipine 30mg 4-hrly, orally, has been shown to control BP effectively in pre-eclampsia.

The following is a suggested dose regimen for $MgSO_4$ used with laboratory support for estimations of serum magnesium concentrations.

1. Initial loading dose - **5g $MgSO_4$** (10 ml of 50% solution) either by i.v. bolus over (5 minutes or by adding to 40ml Normal Saline (total volume 50ml) and infusing over 20 minutes (150ml/hr).

2. Maintenance dose - infuse $MgSO_4$ at **2g/hr** e.g. add 20ml of 50% solution to 30 ml Normal Saline (total volume 50ml) and infuse at 10ml/hr. For women <50 kg, infuse at **1g/hr** (5ml/hr)

3. Check serum Mg concentration 60min after loading dose, then every 6hr. Adjust to maintain serum concentration in the therapeutic range **2 to 3.5mmol/l.**

4. If concentration is <2mmol/l, give an extra 2 g (increase rate to 40ml/hr *for 15 minutes only*).

5. If serum concentration is 3.5 - 4mmol/l, decrease rate to 5 ml/hr; if >4mmol/l, stop infusion until serum concentration has decreased.

6. Continue for 24hr, or as long as woman is symptomatic or has labile BP.

- ECG changes (widened QRS) may occur within therapeutic range. Renal impairment will reduce Mg clearance; nausea, vomiting and flushing are early signs of toxicity. Loss of deep tendon reflexes and respiratory muscle weakness occur at 5 - 7.5mmol/l, cardiac arrest at around 12.5mmol/l.

- In the event of toxicity, stop the $MgSO_4$ infusion and administer ventilatory and circulatory support as required. Calcium chloride or gluconate (10 - 20ml of 10% solution) will oppose the effects of magnesium on neural transmission.

Hepatic

Patients with the HELLP syndrome usually present pre-delivery, with malaise, nausea and vomiting, and epigastric or right upper quadrant pain and tenderness. Hypertension and proteinuria may be minimal or absent. As with eclampsia, presentation in the postpartum period is not uncommon.

- Haemolysis (seen on blood film) and a platelet count falling to <100 (10-9/l may be associated with DIC.

- Elevated bilirubin, alanine transaminase and lactate dehydrogenase concentrations are indicative of hepatocellular injury.

- Other associated maternal morbidities include placental abruption, subcapsular liver haematoma, acute renal failure, and pulmonary oedema.

Differential diagnoses include the related microangiopathies: thrombotic thrombocytopaenic purpura, haemolytic uraemic syndrome, and acute fatty liver of pregnancy. The treatment is delivery, with specific organ system support as necessary. There is evidence that postpartum i.v. administration of dexamethasone (10 mg 12-hrly) hastens recovery and reduces disease severity.

Whereas liver enzyme abnormalities are usually more pronounced in HELLP, acute fatty liver of pregnancy is more likely to result in marked hypoglycaemia, hyperammonaemia, and coagulopathy. In addition to coagulopathy, raised intracranial pressure (suggested by somnolence) dictates GA rather than a regional block for Caesarean section. In a recent case report, a serum Mg concentration at the upper limit of the therapeutic range was associated with respiratory depression necessitating mechanical ventilation. It was postulated that combined hepatic and renal dysfunction rendered the patient more susceptible to Mg toxicity.[2]

Analgesia and Anaesthesia

The risk of fetal compromise and placental abruption in labour is increased in pre-eclamptic women, therefore early establishment of good regional analgesia is recommended. BP should be controlled (e.g. with hydralazine) before the procedure is undertaken. The subsequent sympathetic blockade and relief of pain will prevent hypertensive surges during contractions. Early communication amongst midwives, obstetricians and anaesthetists should allow time for conversion to surgical anaesthesia if Caesarean section becomes necessary. The catheter will enable provision of optimal postoperative analgesia by continuous or patient-controlled infusion of a fentanyl/bupivacaine mixture in a high-dependency environment.

Concerns about regional anaesthesia for Caesarean section in pre-eclampsia include

- The risk of vertebral canal haematoma (VCH). Thromboelastography has shown that pre-eclamptic women with platelets >100 (10^{-9}/l are *hypercoagulable*. At platelet counts below 100 (10^{-9}/l there is a risk of *hypocoagulability*, and measurement of coagulation times (APTT/TCT) is indicated.[15] Thromboelastographic indices of coagulation in pre-eclampsia are not significantly altered by attainment of therapeutic serum magnesium concentrations. Logically, the risk of VCH should be less following single passage of a 26g pencil-point spinal needle as opposed to identification of the epidural space with a 16g Tuohy needle and insertion of a catheter.

- Haemodynamic instability. Prior vasodilatation by effective antihypertensive treatment (e.g. oral methyldopa or i.v. hydralazine) seems to prevent problematic hypotension following epidural or spinal anaesthesia. Judicious increments of ephedrine or phenylephrine do not cause arterial pressure overshoot.

A recent study demonstrated that women who have had an eclamptic seizure but were fully conscious and co-operative, treated with magnesium, and with platelet count >100 (10^{-9}/l could safely undergo regional anaesthesia.[16]

Concerns about general anaesthesia include

- The pressor response to laryngoscopy and intubation/extubation. The cerebral circulation must be protected from hypertensive surges at intubation and extubation - as in a neuroanaesthetic for cerebral aneurysm clipping. Despite pretreatment with Mg and labetalol, BP and middle cerebral artery velocity (measured by transcranial Doppler, and assumed to be indicative of cerebral blood flow) increased significantly after tracheal intubation in a series of pre-eclamptic women.[17]

- Laryngeal oedema. A real risk - particularly for the patient whose larynx was noted to be swollen at laryngoscopy, or in whom intubation was traumatic. Those undertaking postoperative care must be alert to the ominous significance of stridor.

- Interaction of Mg with neuromuscular blockers. Although the onset and duration of suxamethonium is unaffected by therapeutic Mg concentrations in pre-eclampsia, Mg affects the actions of all *non-depolarising* drugs. The onset time of vecuronium 0.1mg/kg is halved by prior bolus of Mg. Significant *recurarisation* has been demonstrated following administration of a bolus of Mg after recovery from vecuronium block to a train-of-four ratio of 0.7. The onset time of rocuronium 0.6mg kg^{-1} is not shortened by prior administration of magnesium, but the mean time to recovery of T1 to 25% during isoflurane anaesthesia is increased by 50%. In pre-eclamptic women treated with magnesium, mivacurium 0.15mg/kg given after recovery from suxamethonium has a mean duration of 35 min.

General anaesthesia is indicated if there is uncorrected coagulopathy or symptoms/signs consistent with impending eclampsia. Prior communication with a neonatal paediatrician is essential in order that preparation can be made for antagonism of opioid/provision of ventilatory support.

- Have a low threshold for direct arterial pressure monitoring.

- Attenuate the pressor response to intubation with alfentanil 10μg/kg or remifentanil 2μg/kg before rapid sequence induction with a generous dose of thiopental.

- Do *not* limit the end-tidal inhalational agent concentration on account of (spurious) concerns about neonatal depression.

- Before extubation, consider antihypertensive therapy (e.g. labetalol in 10-20mg increments) to avert a dangerous pressor response.

- A peripheral nerve stimulator is essential to indicate the degree of neuromuscular block.

Non-steroidal anti-inflammatory drugs (NSAID) are absolutely contra-indicated if pre-eclampsia has been complicated by haemorrhage, or there is concern about adequacy of haemostasis (e.g. uterine atony). In women with mild renal disease (good urine output and no serum indices of renal failure), there seems little reason to deny women the benefit of the morphine-sparing effect of NSAID. However, successive doses should not be given without repeated confirmation of sustained satisfactory urine output.

Maternal mortality

The need for clear delivery suite protocols and early consultant input, particularly to co-ordinate fluid balance, have been recurring themes in the Reports. In the '94-'96 triennium[1], 20 direct deaths were attributed to hypertensive disorders of pregnancy - the same number as over the previous 3 years. This is a mortality rate of 1 in 100 000 maternities. Pulmonary complications (e.g. ARDS) outnumbered intracranial haemorrhage as a cause of death.

The '97-'99 Report[18] saw fewer deaths[16], with the majority[7] swinging back to intracerebral haemorrhage. Only one death was attributed to ARDS.

References

1. Department of Health et al. Report on confidential enquiries into maternal deaths in the United Kingdom 1994-1996. London: HMSO, 1998

2. Holzman RS, Riley LE, Aron E, Fetherston J. Perioperative care of a patient with acute fatty liver of pregnancy. Anesthesia and Analgesia 2001; **92**:1268-70

3. Hutter C, Crighton IM, Smith K, Liu DTY. The role of serotonin in preeclamptic hypertension. A review and case report. International Journal of Obstetric Anesthesia 1996; **5**:108-14

4. Belfort MA, Anthony J, Saade GR et al. The oxygen consumption/oxygen delivery curve in severe preeclampsia: Evidence for a fixed oxygen extraction state. American Journal of Obstetrics and Gynecology 1993; **169**:1448-55

5. Mushambi MC, Halligan AW, Williamson K. Recent developments in the pathophysiology and management of pre-eclampsia. British Journal of Anaesthesia 1996; **76**:133-48

6. Bolte AC, van Eyck J, Kanhai HH, Bruinse HW, van Geijn HP, Dekker GA. Ketanserin versus dihydralazine in the management of severe early-onset pre-eclampsia: Maternal outcome. American Journal of Obstetrics and Gynecology 1999; **180**:371-7

7. Desai DK, Moodley J, Naidoo DP, Bhorat I. Cardiac abnormalities in pulmonary oedema associated with hypertensive crises in pregnancy. British Journal of Obstetrics and Gynaecology 1996; **103**:523-8

8. Cunningham FG, Pritchard JA, Hankins GDV, Anderson PL, Lucas MJ, Armstrong KF. Peripartum heart failure: idiopathic cardiomyopathy or compounding cardiovascular events? Obstetrics and Gynecology 1986; 67: 157-67

9. Moran P, Davison JM. The kidney and the pathogenesis of pre-eclampsia. Current Obstetrics and Gynaecology 1999; **9**: 196-202

10. Engelhardt T, MacLennan FM. Fluid management in pre-eclampsia. International Journal of Obstetric Anesthesia 1999; **8**:253-9

11. Penny JA, Anthony J, Shennan AH, de Swiet M, Singer M. A comparison of hemodynamic data derived by pulmonary artery flotation catheter and the esophageal Doppler monitor in preeclampsia. American Journal of Obstetrics and Gynecology 2000; **183**:658-61

12. Katz VL, Farmer R, Kuller JA. Preeclampsia into eclampsia: Towards a new paradigm. American Journal of Obstetrics and Gynecology 2000; **182**:1389-96

13. The Magpie Trial Collaborative Group. Do women with pre-eclampsia, and their babies, benefit from magnesium sulphate? The Magpie Trial: a randomised placebo-controlled trial. Lancet 2002; **359**:1877-90

14. Yentis SM. Editorial. The Magpie has landed: preeclampsia, magnesium sulphate and ratiomal decisions. International Journal of Obstetric Anesthesia 2002; **11**:238-41

15. Sharma SK, Philip J, Whitten CW, Padakandla UB, Landers DF. Assessment of changes in coagulation in parturients with preeclampsia using thromboelastography. Anesthesiology 1999; **90**:385-90

16. Moodley J, Jjuuko G, Rout C. Epidural compared with general anaesthesia for caesarean delivery in conscious women with eclampsia. British Journal of Obstetrics and Gynaecology 2001; **108**:378-82

17. Ramanathan J, Angel JJ, Bush AJ, Lawson P, Sibai B. Changes in maternal middle cerebral artery blood flow velocity associated with general anaesthesia in obstetrics. Anesthesia and Analgesia 1999; **88**:357-61

18. Report on confidential enquiries into maternal deaths in the United Kingdom 1997-1999. London: RCOG Press, 2001 http://www.cemach.org.uk

Further references on request from Dr DM Levy:

david.levy@mail.qmcuh-tr.trent.nhs.uk

ANAESTHESIA FOR CORRECTION OF STRABISMUS

Philip Hartwig, Pietermaritzberg, South Africa

Strabismus, often known as crossed eyes or squint, is a visual condition where gaze is misaligned. It is fairly common in children and affects roughly 2% - 5% of the population.

Strabismus can affect either one or both eyes, with an eye turning in, out, up or down. Although it is never too late to try correction, the earlier this is done the better. The best results are usually obtained in children less than six years old, and especially before the age of two.

Surgery is only one of the ways of treating strabismus; other methods include wearing spectacles and wearing eye patches. Strabismus surgery is extra-ocular surgery which involves repositioning of whichever ocular muscles are misaligned. This may be either unilateral or bilateral.

Anaesthesia

Correction of strabismus is the most commonly performed paediatric ophthalmic operation. Strabismus surgery is normally carried out under general anaesthetic (always so in children), although a local anaesthetic technique may occasionally be used in adults.

There are several ways of administering a general anaesthetic in strabismus surgery. Commonly a technique involving endotracheal intubation with the use of a neuromuscular blocking agent is used, although Laryngeal Mask Airways (LMA's) are also popular.

During surgery it is very important that the eye should be immobile, as the surgeon needs an absence of muscle tone to perform the forced duction test (FDT). This involves assessing mechanical restriction to movement of the eye by moving it into each field of gaze, done by grasping the sclera near the corneal limbus with a pair of forceps. This test allows the surgeon to differentiate between a paretic muscle and a mechanical restriction limiting eye movement.

Because muscle tone may vary with changing depths of anaesthesia, some surgeons may prefer neuromuscular blockade.

Preoperatively

Children may be premedicated with paracetamol, 20mg per kg, and it is wise to obtain consent for rectal NSAID suppositories. With older patients undergoing a general anaesthetic, routine investigations should be performed. A premedication of Glycopyrrolate (200mcg in adults, 5mcg per kg in children) will reduce the amount of saliva, especially useful if you are using an LMA. It also decreases the occurrence of the oculo-cardiac reflex (see below).

Induction

This will depend on whether the patient is to be paralyzed or allowed to breathe spontaneously on a laryngeal mask.

Intravenous induction performed with fentanyl or alfentanil combined with propofol or thiopentone is common. A gas induction with either halothane or sevoflurane may also be used, especially in younger children.

The choice of whether to use an LMA or to intubate the patient will depend on several factors. Given that LMA's have a greater potential for problems in small children, some anaesthetists prefer to use an endotracheal tube here. Generally speaking when an LMA is used the patient will be allowed to breathe spontaneously, although they may be used to ventilate patients. When this is the case, high airway pressures (more than 15cm water) should be avoided to minimise gastric insufflation. Armoured LMA's are often more satisfactory than conventional ones. Normal contra-indications to the use of LMA's such as uncontrolled reflux obviously apply.

It is also worth remembering that access to the airway is difficult during strabismus surgery, so be sure of your airway before the patient is draped.

If the patient is to be intubated (usually a RAE tube is used), non-depolarising agents are normally preferred to suxamethonium. This is for two reasons; Firstly, patients who have been given suxamethonium have a prolonged increase in the extra-ocular muscle tone, which interferes with the FDT. (This effect lasts roughly 15-20 minutes) Secondly, patients undergoing correction of strabismus may be at increased risk of developing malignant hyperthermia.

Maintenance of anaesthesia

Correction of strabismus typically lasts 60 to 90 minutes, with the patient lying supine. Anaesthesia may be maintained either with volatile agents (with or without nitrous oxide) or a propofol infusion. Since this type of surgery is not particularly painful, the combination of paracetamol/NSAID with fentanyl or alfentanil is usually adequate. Supplemental local anaesthesia may also be used.

With all ocular surgery comes the risk of the oculo-cardiac reflex (OCR). This is particularly common in children and adolescents undergoing correction of strabismus. The OCR is characterised by a marked slowing of the heart rate or the occurrence of dysrhythmias in response to traction on the extra-ocular muscles or pressure on the globe. It may even result in cardiac arrest in extreme circumstances. This reflex is mediated by the trigeminal-vagal reflex arc. It tends to be more marked with sudden and sustained traction compared to slow, gentle, progressive traction. Fatigue of the OCR usually occurs with subsequent stimulation.

Because of the importance of the oculo-cardiac reflex, much attention obviously needs to be paid to its prevention and treatment. Although a dose of glycopyrrolate given at the time of induction (200mcg in adults, 5mcg per kg in children) does offer

a degree of protection from the OCR, it does not completely prevent it in every patient. Generally, however, premedication with glycopyrrolate will abolish the need for any further anticholinergic agents to be given (e.g. atropine). If the patient does experience a significant OCR with bradycardia or dysrhythmias, atropine is the drug of choice for acute intervention. In such a situation the surgeon must be informed, as relaxation of any applied traction will help return the heart rate to normal levels.

The side effects associated with anticholinergic agents, such as a dry mouth and tachycardia, also need to be taken into account.

Simple manoeuvres such as using supplementary local anaesthetic and avoiding hypercapnia also decrease the incidence of the OCR.

Postoperative

As mentioned earlier, these procedures are not particularly painful and opioids may be avoided to decrease postoperative nausea and vomiting (PONV). This is particularly common with surgery to correct strabismus, and consideration should be given to including a prophylactic anti-emetic agent.

EARLY WARNING SCORES

JE Rees, Royal Devon and Exeter, UK

What is an Early Warning Score?

In the United Kingdom Early Warning Scores (EWS) are now commonly used for the assessment of unwell hospital patients. The Early Warning Score is a simple physiological scoring system that can be calculated at the patient's bedside, using parameters which are measured in the majority of unwell patients. It does not require complex, expensive equipment to measure any of the parameters. It is reproducible[1] and can be used to quickly identify patients who are clinically deteriorating and who need urgent intervention. EWS can be used to monitor medical, pre and post-operative surgical, and Accident and Emergency patients. Early warning scores are sometimes also referred to as Patient at Risk scores (PARS) or Modified Early Warning Scores (MEWS).

How do you calculate an Early Warning Score?

An EWS is calculated for a patient using five simple physiological parameters. Mental response, pulse rate, systolic blood pressure, respiratory rate and temperature. For patients who are post-operative or unwell enough to be catheterised a sixth parameter, urine output, can also be added (See Table 1). The idea is that small changes in these five parameters will be seen earlier using EWS than waiting for obvious changes in individual parameters such as a marked drop in systolic blood pressure which is often a pre-terminal event.

Of all the parameters, respiratory rate is the most important for assessing the clinical state of a patient, but it is the one that is least recorded. Respiratory rate is thought to be the most sensitive indicatory of a patients physiological well being.[2,3,4]. This is logical because respiratory rate reflects not only respiratory function as in hypoxia or hypercapnia, but cardiovascular status as in pulmonary oedema, and metabolic imbalance such as that seen in diabetic ketoacidosis (DKA).

When and why to use an Early Warning Score?

An EWS score should be calculated for any patient that nursing staff are concerned about. It gives a reproducible measure of how "at risk" a patient is. Patients who have suffered major trauma, or have undergone major surgery, can be started on an EWS observation chart (table 1) as soon as they arrive on the ward to monitor their clinical progress, and give early warning of any deterioration. Repeated measurements can track the patient's improvement with simple interventions such as oxygen or fluid therapy or further deterioration. Serial EWS readings are more informative than isolated readings as they give a picture of the patient's clinical progress over time.

The scoring system was developed because not all unwell patients can be monitored on intensive care or high dependency units. It allows deteriorating patients to be identified, before physiological deterioration has become too profound. Once an unwell patient has been identified, with an EWS score of 3 or more, this should stimulate a rapid assessment of the patient by a ward doctor or, if available, the intensive care unit (ICU) team. The result of the review should be the modification of patient management to prevent further deterioration. If deteriorating patients are identified early enough, simple interventions such as oxygen, or fluid therapy, may prevent further deterioration and imminent collapse. The use of EWS has been shown to be effective in reducing mortality and morbidity of deteriorating patients as well as preventing ICU admissions[5,6,7,8,9,10].

What should happen if a patient has an Early Warning Score of 3 or more?

Studies have indicated that score of 3 or more requires urgent attention[4,6]. The level of response is dependent on the facilities available. In many UK hospitals a score of 3 triggers an immediate review by a ward doctor. If no improvement is seen the most senior ward nurse can then call a senior doctor. This gives the ward nursing staff the authority to refer upwards to more senior members of staff if a patient's clinical situation is not improving. Some UK hospitals have gone further and a score of 3 results in an immediate call, by the nursing staff, directly to the Intensive care unit registrar for a ward review. Other hospitals have been more cautious and use a score of 4 or even 5 as a call out trigger[4].

A generic EWS flowchart is given in figure 1.

Case Histories

1. A 60-year-old man arrived in hospital with increasing shortness of breath. He had no chest pain. He had a past history of a myocardial infarction and was awaiting coronary artery bypass surgery; he was also a known asthmatic. On arrival in hospital he was alert with a respiratory rate of 30, a pulse rate of 130 and a blood pressure of 108/60, his temperature was 38.5°C. He therefore had an EWS score of 5. He was assessed by the emergency doctors. A salbutamol nebuliser and oxygen therapy were given. After 15 minutes, on clinical observation, he looked better. His respiratory rate had dropped to 24, his pulse rate was 124 bpm, temperature remained the same but his blood pressure had dropped to 95/55mmHg. Therefore despite looking better his EWS score had risen to 6, suggesting he was still deteriorating. The intensive care team were called and he was admitted to the high dependency unit for observation and treatment. He was found to be septic from a chest infection. This case shows that subjective judgements made on appearance only can be misleading. More objective judgements are often made on the basis of physiological parameters.

2. A 72 year old patient arrived in recovery after a Whipple's resection of his pancreas for a pancreatic tumour. He had lost 3 litres of blood intra-operatively and was receiving a blood transfusion in recovery. Initially in recovery he was alert with a heart rate of 70bpm, a respiratory rate of 15, a blood pressure of 110/70mmHg, and a urine output of 20ml/hr. His EWS was 1. Over the next 3 hours in recovery he became more tachycardic and hypotensive. He was alert with a heart rate of 105, a respiratory rate of 20, a blood pressure of 95/50 and a UO of 10ml/hr. His temperature was not recorded. Therefore his EWS can be calculated as having risen to 4. Despite this a doctor did not review him, and he was sent back to the ward. By midnight he was drowsy, had a respiratory rate of 30, temperature of 38.5°C, heart rate of 120bpm, blood pressure of 90/50mmHg and his urine output was negligible. This made his EWS 11. He was finally reviewed, actively resuscitated and taken immediately back to theatre for an exploratory laparotomy. Two litres of blood and clot were found in his abdomen from a bleeding artery. He was in hypovolaemic shock. He was sent intubated to the intensive care unit and remained there overnight. If the EWS protocol had been followed this patient should have never left recovery. All the signs were there from a very early stage that he was deteriorating. Early intervention would have prevented the development of hypovolaemic shock and possibly an ICU admission.

References

1. Stubbe CP., Kruger M., Rutherford P., Gemmell L. Validation of a modified Early Warning Score in medical admissions. Quarterly Journal of Medicine 2001;**94**:521-526

2. Fieselmann J et al. Respiratory rate predicts cardiopulmonary arrest for internal medicine patients. The Journal of General Internal Medicine. 1993;**8**:354-360.

3. Kenwood G, Hodgetts T, Castle N. Time to put the R back in TPR. Nursing Times. 2001;**97**:32-33.

4. Stubbe CP., Davies RG., Williams E., Rutherford P., Gemmell L. Effects of introducing the modified Early Warning Score on clinical outcomes, cardio-pulmonary arrests and intensive car utilisation on acute medical admissions. Anaesthesia, 2003;**58**:775-803.

5. Allen A, McQuillan P, Taylor B, Neilson M, Collins C, Short A, Morgan G, Smith G. Who sees the critically ill patient before ICU admission. Clinical Intensive Care, 1994;**5**:152.

6. Hourihan F, Bishop G, Hillman KM, Daffurn K, Lee A. The Medical Emergency team; a new strategy to identify and intervene in high-risk patients. Clinical Intensive Care 1995;**6**:269-72.

7. Goldhill D, White S, Sumner A. Physiological values and procedures in the 24 hours before ICU admission from the ward. Anaesthesia. 1999;**54**:853-860.

8. McGoin H, Adam S, Singer M. The quality of pre-ICU care influences outcome of patients admitted from the ward. Clinical Intensive Care; 1997;**8**:104.

9. McQuillan P, Pilkington S, Allan A, Taylor B, Short A, Morgan G, Nieslon M, Barrett M, Smith G. Confidential enquiry into quality of care before admission to intensive care. British Medical Journal 1997;**316**:1853-1857.

10. Buist D, Moore G, Bernard S, Waxman B, Anderson N, Nguyen T. Effects of a medical emergency team on the reduction of incidence of and mortality from unexpected cardiac arrests in hospital: preliminary review. British Medical Journal 2002;**324**:387-90.

Royal Devon and Exeter Healthcare NHS
NHS Trust

EARLY WARNING SCORE

For all emergency and compromised post ITU patients

This should be assessed on all emergency admissions, major surgery, all patients returning from ITU/HDU and any patient that you are concerned about. *If outside this range call cardiac arrest team.

Date of admission	Affix patient label here
Consultant	
Ward	

Date															
Time															
HR 30-180*															
BP <60*															
Resp. rate 8-40*															
Central Nervous System															
Temp.															
Urine															
Score															
Doctor Y / N															
Grade if called															

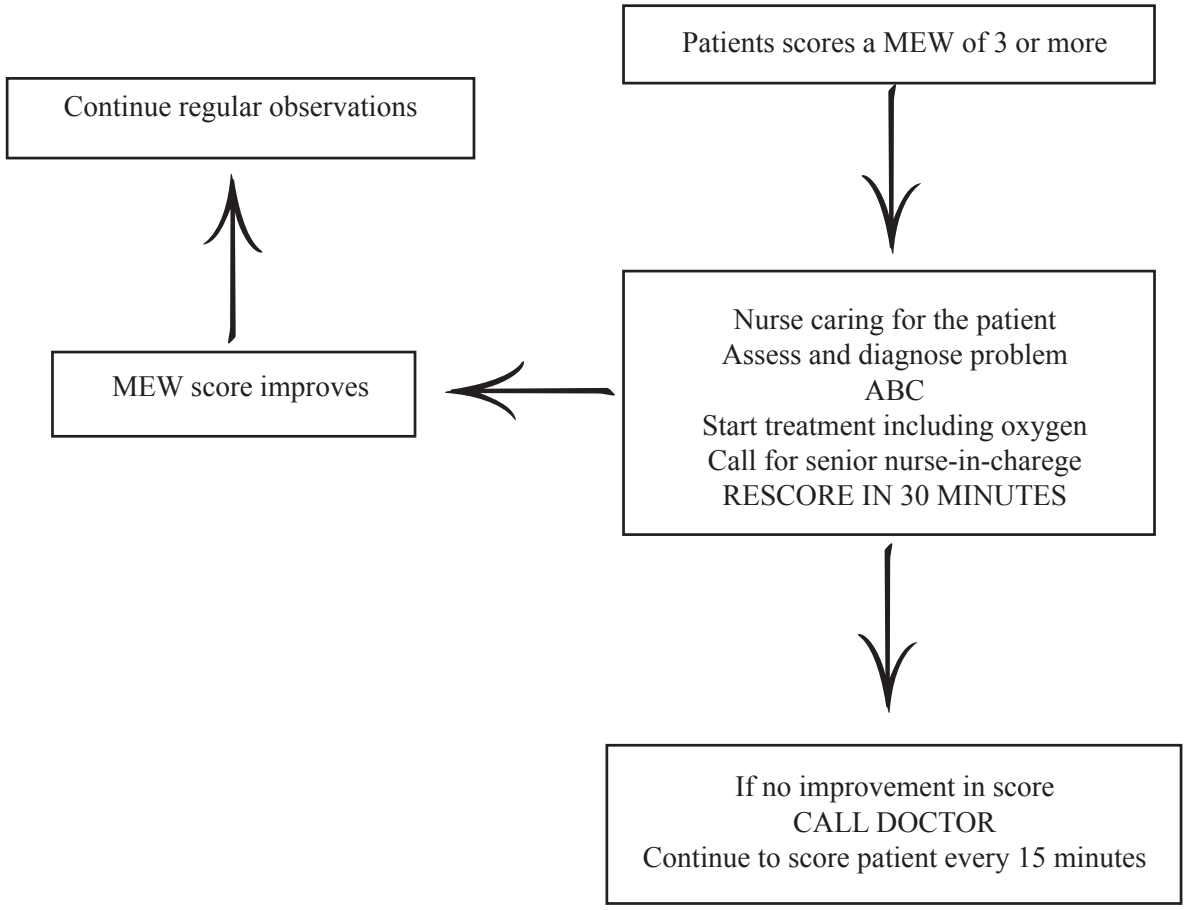
Score	3	2	1	0	1	2	3
HR per minute		<40	41-50	51-100	101-110	111-129	>130
BP systolic	<70	71-80	81-100	101-199		>200	
Resp per minute		<8		9-14	15-20	21-29	>30
Central Nervous System				Alert	Drowsy/ rousable to voice or newly confused	To pain	Un-responsive
Temperature		<35		35.1-37.5	>37.5		
Urine output	Nil	<20mls/2hrs or has not voided within 4hrs of admission	20-50ml/2hrs or has not voided within 4hrs of admission	>50ml/2hrs			

If the patient has a score of 3 or more follow the flowchart overleaf ❹

Page 1 of 2

Table 1.

EARLY WARNING SCORE GENERIC FLOWCHART
Any patient scoring more than 3 requires urgent attention



If **UNRESPONSIVE** patient has any of the following pre-arrest signs:

A - Severe airway compromise

B - Respiratory rate <8 or >40

C - Heart rate <30 or >120
Systolic BP <60
Oxygen Saturations <85%

CALL SENIOR HELP/DOCTOR IMMEDIATELY

Contact number for doctor on call:

Staff called as per flowchart

	Name	Grade	Time
1.			
2.			
3.			

Figure 1.

Reprinted from 2002 CME lectures by kind permission of the Royal College of Anaesthetists and the Association of Anaesthetists of Great Britain and Ireland

THE PATIENT WITH HEART DISEASE

Professor Pierre Foex, Nuffield Department of Anaesthetics, University of Oxford

Over the past six decades, mortality due solely to anaesthesia has decreased from approximately 1 in 1,500 to 1 in 150,000. However, death within 30 days of surgery remains a major issue. In the United Kingdom, over the past ten years, the number of such perioperative deaths has remained fairly constant at approximately 20,000 deaths per annum[1], of which 9,000 are due to cardiac causes. For each cardiac death there are between 5 and 20 major cardiac complications, such as myocardial infarction, unstable angina, life-threatening arrhythmias, or acute left ventricular failure. Thus, the UK number of cardiac complications is expected to range from 45,000 to 180,000 per annum. These complications occur in patients with a compromised cardiovascular system, most frequently because of underlying coronary artery disease. Indeed, 60% of patients who die within 30 days of surgery have evidence of coronary heart disease [2]. Pre-existing valvular heart disease, hypertensive heart disease, and congestive cardiac failure also play an important role.

Coronary heart disease

Angina causes only a moderate increase in perioperative morbidity provided it is well controlled and medication is continued throughout the perioperative period. Unstable, new and disabling angina are associated with a high post-operative morbidity. In such patients coronary angiography is usually necessary prior to major surgery in order to establish the severity of the disease and optimise treatment; this may include coronary angioplasty (with or without stenting) or coronary artery bypass surgery. When the angina is less severe and coronary bypass surgery is not indicated in its own right, prophylactic coronary artery revascularization may be performed, on occasion, in order to reduce the risk of postoperative myocardial infarction, but only in the face of major surgery[3]. However, where coronary revascularisation is indicated in its own right it should be carried out before non cardiac surgery.

Previous myocardial infarction. Myocardial infarction that has occurred less than three months before surgery is known to be associated with a very high risk of reinfarction[4]. In recent years this risk has become smaller[5]. The time that has elapsed between myocardial infarction and surgery is, therefore, only one of risk factors. Cardiologists consider that after uncomplicated myocardial infarction a delay of six weeks is acceptable. This view is endorsed in the American College of Cardiologists and American Heart association (ACC/AHA) guideline.[6]

Irrespective of the delay between infarction and surgery, risks remain high in patients presenting for major abdominal or thoracic surgery or for vascular surgery, and in patients who have suffered from acute left ventricular failure at the time of their infarction[7], exhibit poor left ventricular function, or continue to suffer from angina. Therefore evaluation of left ventricular function is essential especially before major surgery as there is an inverse relationship between left ventricular function and adverse cardiac outcome[8]. This evaluation includes clinical examination and exercise tolerance. Often, however, an objective test is needed as many patients minimise their disability, or are incapable of exercising for other reasons (arthritis, severe intermittent claudication) and, therefore, never "test" their cardiac reserve.

Silent myocardial ischaemia, as detected by ambulatory ECG (Holter) monitoring, is a feature of coronary heart disease. It is observed in patients who are totally asymptomatic (type 1), have suffered previous myocardial infarction (type 2), or suffer from angina (type 3). Up to 80% of ischaemic events are silent[9]. Silent myocardial ischaemia is associated with adverse prognosis[10]. Silent ischaemia occurs in up to 50% of adult surgical patients and is associated with postoperative cardiovascular complications[11,12]. It is more frequent and more prolonged during the postoperative period. This increased ischaemic burden (expressed as minutes of ischaemia per hour of monitoring) is responsible for the very strong association between postoperative silent ischaemia and adverse cardiac outcome[11]. Silent ischaemia may be caused by cardiovascular instability (tachycardia, hypertension, hypotension), coagulation disorders (microthrombosis and microlysis) and/or postoperative hypoxaemia[13]. A feature of perioperative silent myocardial ischaemia is that it is associated with short- and long-term adverse outcome thereby decreasing the event-free survival at two years from 90% to 76%[14].

Patients with coronary grafts. Many patients undergo non-cardiac surgery after previous coronary bypass graft operation. The risk of postoperative myocardial infarction is low in this group of patients[3,15] provided they are not operated on less than six weeks to two months after coronary surgery, and do not have other risk factors such as angina or poor left ventricular function. Hypotension during the perioperative period must be avoided as it may cause thrombosis of the grafts. In some patients, significant increases in left ventricular function are observed after coronary artery bypass graft[16] or after angioplasty and stenting, because previously underperfused myocardium (hibernating myocardium) becomes more contractile after reperfusion.

Patients with previous angioplasty and stenting. Some benefits in terms of risk reduction can be expected in patients who have undergone coronary angioplasty more than three months before elective surgery[17]. If a stent is inserted, strong antiplatelets drugs are always given and surgery within a short period of insertion of the stent is extremely dangerous[18]; waiting six weeks is regarded as essential.

Further investigations. Often the medical history alone underestimates the severity of coronary heart disease. Before coronary angiography is performed, screening tests are useful.

Their aim is to identify patients with reversible ischaemia who should undergo coronary angiography.

Ambulatory ECG monitoring is valuable, however, it is now regarded as inferior to formal exercise testing.

Reversible ischaemia may be elicited by exercise testing and detected by electrocardiography, echocardiography, radionuclide angiography (technetium) or myocardial scintigraphy (thallium). Where patients are unable to exercise, a dobutamine infusion is an excellent pharmacological substitute to exercise. Reversible ischaemia is identified as ST-segment depression on the ECG, new wall motion abnormalities and/or a reduction of the ejection fraction on echocardiography or radionuclide angiography, and by a reversible defect (decreased uptake of the radioactive isotope) on myocardial scintigraphy. Presence of reversible ischaemia, strongly predicts perioperative cardiac events.[19]

Exercise echocardiography or echocardiography with dobutamine infusion are more widely available than myocardial scintigraphy, and allow relatively non-invasive screening for coronary artery disease. If exercise or dobutamine infusion elicit reversible ischaemia this indicates the presence of areas with compromised blood supply and the need for coronary angiography, bearing in mind that a high proportion of patients, particularly those presenting for vascular surgery, have correctable coronary artery lesions. The management strategy depends, to a large extent, upon the severity of the coronary artery disease as seen on coronary angiography. In addition, coronary angiography may reveal lesions that justify revascularisation in their own right.

Recently, the cardiac troponins I and T have been found to be helpful in the diagnosis of perioperative myocardial damage, including myocardial infarction.[20] Postoperative troponin elevation predicts adverse outcome[21] and, in some patients, preoperative elevation of troponins has been shown to predict the risk of perioperative myocardial infarction.[22]

Arterial hypertension

Arterial hypertension is associated with an increase in the cardiovascular morbidity and mortality of anaesthesia and surgery[23,24], even though hypertension was not found to be a significant predictor of cardiac complications of anaesthesia and surgery in several indices of cardiac risk in non-cardiac surgery.[25,26] In patients diagnosed as hypertensive, and on anti-hypertensive medication, treatment of hypertension should be maintained throughout the perioperative period; often the morning dose of ACE inhibitors is omitted because of the risk of hypotension. However, this policy may cause an increase in the risk of perioperative hypertension.[27] Treatment with angiotensin receptor antagonists needs to be stopped the day before surgery because of the risk of refractory hypotension after induction of anaesthesia and during surgery.[28] Where hypertension is poorly controlled, management of the patients should follow the principles applicable to untreated hypertension.

In untreated patients, mild hypertension (Stage 1: 140-159/90-99mmHg), does not constitute a major threat. Moderate hypertension (Stage 2: 160-179/100-109mmHg), constitutes a threat especially where it is associated with target organ involvement (coronary, cerebrovascular or renal disease), in which case treatment prior to elective surgery is recommended. Severe hypertension (Stage 3: 180-201/110-119), and marked left ventricular hypertrophy (ECG and/or chest X-ray), increase the risk of complications. Such patients should be treated before surgery; this is also true of patients with malignant hypertension (Stage 4: > 210/>120).[29]

Professor Prys-Roberts, in an editorial published in 2001[30] took a different view of the management of hypertensive patients, suggesting that in untreated patients, postponement of surgery is unnecessary unless the diastolic pressure exceeds 120mmHg. For treated hypertension, cancellation in order to improve treatment may be justified if the diastolic pressure exceeds 110mmHg. Subsequently, Professor Prys-Roberts, in a letter[31], adopted a position that is more in keeping with the generally agreed principles. Similarly, the AHA/ACC guideline suggest that patients with a diastolic blood pressure above 110mmHg should be treated before surgery.[6]

Heart failure

Patients with heart failure are at risk of major postoperative cardiac events. Even incipient heart failure is a strong predictor of adverse outcome.[25,26,32] The number of patients with heart failure is increasing very rapidly because the mortality of myocardial infarction has been reduced and, therefore, more patients survive with impaired cardiac function.[33] Evaluation of cardiac function with echocardiography or radionuclide angiography is very useful because the risk of complications of anaesthesia and surgery is directly related to the severity of ventricular dysfunction. An ejection fraction less than 40% predicts adverse cardiac outcome.[34] The patient's drug therapy should be optimised before surgery. In some patients coronary bypass surgery[16] or coronary angioplasty and stenting improve left ventricular function to such an extent that even major surgery becomes considerably safer.

An increasing number of patients with heart failure are now receiving beta-blockers. The latter improve their long-term prognosis, especially where carvedilol is used. However, in all studies of beta-blockade in heart failure, treatment is initiated with extremely low doses, with increases in dosage over eight weeks or more.[35]

Recently the possible value of measuring natriuretic peptides has been emphasised.[36] In particular Brain Natriuretic Peptide (BNP) has been found to be elevated in patients with cardiac dysfunction.[37] It is a predictor of poor survival.[38] Measurement of BNP could be used as a screening test for cardiac dysfunction so that further tests would only be performed in selected patients.

Anaesthetic management of patients with coronary or hypertensive heart disease

A major requirement is to avoid haemodynamic changes that may precipitate myocardial ischaemia. Tachycardia increases myocardial oxygen consumption and decreases coronary flow because of the shorter duration of diastole. Hypotension may reduce coronary flow more than myocardial oxygen consumption because of low coronary perfusion pressure. Hypertension can cause increases in oxygen demand that exceed the coronary reserve. This adverse effect is worsened when tachycardia is

present. However, many episodes of myocardial ischaemia occur in the absence of marked haemodynamic changes.[39] These may be caused by coronary artery spasm, transient spontaneous coronary occlusion (microthrombosis), or coronary steal. The latter may develop during the administration of dilators of the coronary resistance vessels. This has been reported with isoflurane (40). Another important contributor to the safety of these patients is the prevention of post-operative hypoxaemia.[13]

In high risk patients, invasive monitoring, including monitoring of the pulmonary occluded pressure (pulmonary capillary wedge pressure) is useful for major surgery, particularly vascular surgery of the thoracic or abdominal aorta. Transoesophageal echocardiography (TOE) may be useful for the detection of ischaemia and, more importantly, the assessment of ventricular filling. The new generation of ECG monitors are capable of displaying ST-segment trends, thus allowing better detection of perioperative myocardial ischaemia than visual inspection on an ECG monitor.

A prerequisite in the management of patients with coronary or hypertensive heart disease is to protect the myocardium. The first step is to maintain their treatment throughout the perioperative period. However, not all drugs used in the chronic management of these patients are equally effective in preventing cardiac complications of anaesthesia and surgery. A more active approach to ischaemia prevention has developed.

Active drug prevention of ischaemia

Over the past five years it has become clear that the management of surgical patients with coronary heart disease could be improved by the prophylactic administration of drugs in order to decrease oxygen demand, make the circulation more stable, or improve the distribution of coronary blood flow. Drugs having such effects include calcium antagonists, adenosine modulators, alpha2 adrenoceptor agonists, and beta-blockers.

Systematic studies of the perioperative prophylactic administration of calcium antagonists are lacking. However, observational studies do not show patients on calcium antagonists to be protected against silent myocardial ischaemia[41,42] even though calcium antagonists cause coronary vasodilatation, relieve exercise-induced coronary vasoconstriction, reduce left ventricular afterload, and improve the oxygen balance.

Adenosine modulation causes a selective augmentation of adenosine levels in tissues under ischaemic conditions but not in the non-ischaemic myocardium. This results in improved left ventricular function, enhanced collateral blood flow, reduced risk of ventricular dysythmias, and attenuated risk of stunning. Five trials of the adenosine modulator acadesine (total of 4,043 patients) were analysed together.[43] They showed a significant reduction in myocardial infarction (-27%), stroke (-26%), and cardiac death (-50%). Unfortunately, the developpement of this agent has been stopped.

Alpha2-adrenoceptor agonists decrease sympathetic activity by a central mechanism, this results in better haemodynamic stability, and decreased risk of silent ischaemia. In addition, there is sedation, and reduction in anaesthetic and opioid requirements. Clonidine has been shown to reduce the risk of perioperative myocardial ischaemia.[44] In terms of cardiac outcome, a study of the alpha2-adrenoceptor agonist mivazerol showed significant reductions in cardiac death, myocardial infarction and cardiac death, and myocardial infarction and all causes of death, but only in vascular surgical patients.[45] Development of this promising agent has been stopped.

Beta-adrenoceptor blockers are known to reduce myocardial oxygen consumption, decrease the effects of sympathetic activation, and redistribute coronary blood flow. They may reduce overall sympathetic outflow. For more than twenty-five years, beta-blockers have been shown to minimise the risk of perioperative myocardial ischaemia.[46-48] More importantly, perioperative beta-adrenoceptor blockade has been shown to decrease the incidence of perioperative myocardial infarction.[49,50] More recently, atenolol given for one week perioperatively[51] was shown to result in lower mortality at two years by comparison with administration of a placebo (9% vs 20%).

In 1997, the American College of Physicians published a guideline for assessing and managing the perioperative risk from coronary artery disease associated with major non-cardiac surgery.[52] The important message was that for all patients, eligibility for beta-blocker use should be determined. Further evidence for beneficial effects of perioperative beta-blockade was obtained by Poldermans and colleagues.[53] They studied patients in whom coronary artery disease had been demonstrated by the presence of reversible ischaemia on dobutamine echocardiography. In their study, prolonged beta-blockade, started a week or more before surgery, was associated with a large reduction in cardiac death (3.4% vs 17% in the control group) and non-fatal myocardial infarction (0% vs 17% in the control group). The efficacy of beta-blockade was impressive. Moreover, as patients were maintained on beta-blockers, their long-term prognosis was also much improved.[54] However, as all patients had reversible ischaemia, they were at a particularly high risk for coronary events. Thus, the efficacy of beta-blockade cannot be extrapolated to patients at risk for, rather than with demonstrable coronary artery disease. However, based on published studies, and the efficacy of beta-blockade in patients with coronary heart disease, beta-blockers seem to be the logical answer to the perioperative drug management of patients with risk factors for, or with, coronary artery disease.

Why are they not used much more frequently? There are perceived risks to beta-blockade such as worsening of conduction disorders or airway obstruction in patients with reactive airway disease. There is also the risk of worsening of left ventricular dysfunction. Though beta-blockers are now used successfully in the treatment of patients with heart failure[55], their introduction shortly before surgery may not be well tolerated, unless the initial dosage is very low and doses are increased slowly over several weeks.

Before using beta-blockers routinely in all at risk patients, it is necessary to consider that the studies of Mangano and colleagues[51], and Poldermans and colleagues [53,54] were carried out in patients admitted to intensive care or high dependency units, not to ordinary wards. In ITU or HDU environments, any adverse effects can be easily prevented or treated. On the ward this may not be the case. Indeed, the most recent ACC/AHA

guideline[6] suggests that beta-blockers should be used in high risk patients and not necessarily in all patients at risk for coronary artery disease

Beta-blockers may still be the safest agents to use. The treatment should be started well ahead of surgery rather than just the day before surgery. More importantly, a prospective study of their safety on the ward is warranted. If they prove to be well tolerated then their use could be greatly increased.[56] Hopefully, this coupled with other measures could reduce substantially the number of cardiac complications of anaesthesia and surgery.

If acute beta-blockade is effective in reducing the risk of cardiac complications of anaesthesia and surgery, it is tempting to conclude that patients on chronic beta-bloker treatment are well protected. This is not the case. The incidence of perioperative silent myocardial ischaemia is not reduced in patients on chronic beta-blockade.[42] Similarly, chronic beta-blockade does not appear to reduce perioperative mortality.[57] Chronic beta-blockade may not offer the same degree of protection as acute beta-blockade because of beta-adrenoceptor up-regulation or other factors. Therefore, such patients must be considered to be at risk, and monitored especially carefully.

References

1. NCEPOD: Extremes of Age. The 1999 Report of the National Confidential Enquity into Perioperative Deaths, The National Confidential Enquiry into Perioperative Deaths, 1999.

2. NCEPOD: Then and Now: The 2000 Report of the National Confidential Enquiry into Perioperative Deaths. The National Confidential Enquiry into Perioperative Deaths. UK, 2001.

3. Eagle KA, Rihal CS, Mickel MC et al. Cardiac risk of noncardiac surgery: influence of coronary disease and type of surgery in 3368 operations. CASS investigators and University of Michigan Heart Care Program. Coronary Artery Surgery Study. Circulation 1997; **96**:1882-1887.

4. Steen PA, Tinker JH, Tarhan S, et al. Myocardial reinfarction after anesthesia and surgery. JAMA 1978; **239**:2566-2570.

5. Shah KB, Kleinman BS, Sami H, et al. Reevaluation of perioperative myocardial infarction in patients with prior myocardial infarction undergoing noncardiac operations. Anesth Analg 1990; **71**:231-235.

6. Eagle KA, Berger PB, Calkins H, et al. ACC/AHA guideline update for perioperative cardiovascular evaluation for noncardiac surgery—executive summary a report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines (Committee to Update the 1996 Guidelines on Perioperative Cardiovascular Evaluation for Noncardiac Surgery). Circulation 2002;**105(10)**:1257-67.

7. Dirksen A and Kjoeller E. Cardiac predictors of death after non-cardiac surgery evaluated by intention to treat. Br Med J 1988; **297**:1011-1013.

8. Lazor L, Russell JC, DaSilva J, Radford M. Use of the multiple uptake gated acquisition scan for the preoperative assessment of cardiac risk. Surg Gynecol Obstet 1988;**167**:234-238.

9. Deanfield JE, Maseri A, Selwyn AP, et al. Myocardial ischaemia during daily life in patients with stable angina: its relation to symptoms and heart rate changes. Lancet 1983; **2**:753-758.

10. Raby KE, Barry J, Treasure CB, et al. Usefulness of Holter monitoring for detecting myocardial ischemia in patients with nondiagnostic exercise treadmill test. Am J Cardiol 1993;**72**: 889-893.

11. Raby KE, Barry J; Creager MA, et al. Detection and significance of intraoperative and postoperative myocardial ischemia in peripheral vascular surgery. JAMA 1992;**268**:222-227.

12. Mangano DT, Browner WS, Hollenberg M et al. Association of perioperative myocardial ischemia with cardiac morbidity and mortality in men undergoing noncardiac surgery. The Study of Perioperative Ischemia Research Group.N Engl J Med 1990; **323**: 1781-1788.

13. Reeder MK, Muir AD, Foex P, et al. Postoperative myocardial ischaemia: temporal association with nocturnal hypoxaemia. Br J Anaesth 1991;**67**:626-631.

14. Mangano DT, Browner WS, Hollenberg M et al. Long-term cardiac prognosis following noncardiac surgery. The Study of Perioperative Ischemia Research Group. JAMA 1992;**268**:233-239.

15. Foster ED, Davis KB, Carpenter JA, et al. Risk of noncardiac operation in patients with defined coronary disease: The Coronary Artery Surgery Study (CASS) registry experience. Thor Surg 1986;**41**:42-50.

16. Louie HW, Laks H, Milgalter E et al. Ischemic cardiomyopathy. Criteria for coronary revascularization and cardiac transplantation. Circulation 1991; 84-III: 291-295.

17. Posner KL, Van-Norman GA, Chan V. Adverse cardiac outcomes after noncardiac surgery in patients with prior percutaneous transluminal coronary angioplasty. Anesth-Analg 1999;**89**:553-560.

18. Kaluza GL et al. Catastrophic outcomes of noncardiac surgery soon after coronary stenting. J Am Coll Cardiol 2000;**35**:1288-94

19. Vanzetto G, Machecourt J, Blendea D et al. Additive value of thallium single-photon emission computed tomography myocardial imaging for prediction of perioperative events in clinically selected high cardiac risk patients having abdominal aortic surgery. Am J Cardiol 1996;**77**:143-148.

20. Coudrey L. The troponins. Arch Int Med 1998; 158: 1173-1180.

21. Lopez-Jimenez F, Goldman L, Sacks DB et al. Prognostic value of cardiac troponin T after noncardiac surgery: 6-month follow-up data. J Am Coll Cardiol 1997;**29**:1241-1245.

22. Carrier M, Pelletier LC, Martineau R et al. In elective coronary artery bypass grafting, preoperative troponin T level predicts the risk of myocardial infarction. J Thor Cardiovasc Surg 1998;**115**: 1328-1334.

23. Howell SJ, Sear YM, Yeates D, et al. Risk factors for cardiovascular death after elective surgery under general anaesthesia. Br J Anaesth 1998;**80**:14-19.

24. Howell SJ, Sear JW, Foex P. Hypertension and perioperative cardiovascular complications: meta-analysis of observational studies. Br J Anaesth 1998;**80** (Suppl 1): A5.

25. Goldman L, Caldera DL, Nussbaum SR, et al. Multifactorial index of cardiac risk in noncardiac surgical procedures. N Engl J Med 1977;**297**:845-850.

26. Lee TH, Marcantonio ER, Mangione CM et al. Derivation and prospective validation of a simple index for prediction of cardiac risk of major noncardiac surgery. Circulation 1999;**100**: 1043-1049.

27. Piggott DW, Nagle C, Allman K et al. Effect of omitting regular ACE inhibitor medication before cardiac surgery on haemodynamic variables and vasoactive requirements. Br J Anaesth 1999;**83**:715-720.

28. Brabant SM, Eyraud D, Bertrand M, Coriat P. Refractory hypotension after induction of anesthesia in a patient chronically treated with angiotensin receptor antagonists. Anesth Analg 1999; **89**;887-888.

29. Howell S and FoÎx P. Pathophysiology of arterial hypertension: implications in surgical patients. Clin Anaesthesiol 1997/4,795-813.

30. Prys-Roberts C. Isolated systolic hypertension: pressure on the anaesthetist. Anaesthesia 2001;**56**:505-510.

31. Prys-Roberts C. Systolic hypertension. A reply. Anaesthesia 2002;**57(6)**:606-607.

32. Larsen SF, Olesen KH, Jacobsen E, et al. Prediction of cardiac risk in non-cardiac surgery. Eur Heart J 1987;**8**:179-185.

33. Braunwald E. Shattuck lecture—cardiovascular medicine at the turn of the millennium: triumphs, concerns, and opportunities. N Engl J Med 1997: 337: 1360-1369.

34. Halm EA, Browner WS, Tubau JF et al. Echocardiography for assessing cardiac risk in patients having noncardiac surgery. Study of Perioperative Ischemia Research Group. Ann Intern Med 1996;**125**:433-441.

35. Gottlieb SS, Fisher ML, Kjekshus J, et al. Tolerability of beta-blocker initiation and titration in the Metoprolol CR/XL Randomized Intervention Trial in Congestive Heart Failure (MERIT-HF). Circulation 2002;**105(10)**:1182-8.

36. Levin ER, Gardner DG, Samson WK. Natriuretic peptides. N Engl J Med 1998;**339**:321-328.

37. McDonagh TA, Robb SD, Murdoch DR et al. Biochemical detection of left-ventricular systolic dysfunction. Lancet 1998; **351**:9-13.

38. Tsutamoto T, Wada A, Maeda K et al. Attenuation of endogenous cardiac natriuretic peptide system in chronic heart failure. Circulation 1997;**96**:509-516.

39. Slogoff S, Keats AS. Further observations on perioperative myocardial ischemia. Anesthesiology 1986;**65**:539-542.

40. Hohner P, Nancarrow C, Backman C, et al. Anaesthesia for abdominal vascular surgery in patients with coronary artery disease (CAD), Part I: Isoflurane produces dose-dependent coronary vasodilation. Acta Anaesthesiol Scand 1994;**38**:780-792.

41. Slogoff S, Keats AS. Does chronic treatment with calcium entry blocking drugs reduce perioperative myocardial ischaemia? Anesthesiology 1988;**68**:676-680.

42. Sear JW, Foex P, Howell SJ: Effect of chronic intercurrent medication with beta-adrenoceptor blockade or calcium channel entry blockade on postoperative silent myocardial ischaemia. Br J Anaesth 2000;**84**:311-315.

43. Mangano DT: Effects of acadesine on myocardial infarction, stroke, and death following surgery. A meta-analysis of the 5 international randomized trials. The Multicenter Study of Perioperative Ischemia (McSPI) Research Group. Jama 1997; **277**:325-332.

44. Nishina K, Mikawa K, Uesugi T, et al. Efficacy of clonidine for prevention of perioperative myocardial ischemia: a critical appraisal and meta-analysis of the literature. Anesthesiology 2002;**96**:323-9.

45. Oliver MF, Goldman L, Julian DG, Holme I: Effect of mivazerol on perioperative cardiac complications during non-cardiac surgery in patients with coronary heart disease: the European Mivazerol Trial (EMIT). Anesthesiology 1999; **91**: 951-961.

46. Prys-Roberts C, FoÎx P, Biro GP, Roberts JG: Studies of anaesthesia in relation to hypertension. V. Adrenergic beta-receptor blockade. Br J Anaesth 1973;**45**:671-81.

47. Stone JG, Foex P, Sear JW, Johnson LL, Khambatta HJ, Triner L: Myocardial ischemia in untreated hypertensive patients: effect of a single small oral dose of a beta-adrenergic blocking agent. Anesthesiology 1988;**68**:495-500.

48. Wallace A, Layug B, Tateo I, Li J, Hollenberg M, Browner W, Miller D, Mangano DT: Prophylactic atenolol reduces postoperative myocardial ischemia. McSPI Research Group. Anesthesiology 1998;**88**:7-17.

49. Pasternack PF, Imparato AM, Baumann FG, Laub G, Riles TS, Lamparello PJ, Grossi EA, Berguson P, Becker G, Bear G: The hemodynamics of beta-blockade in patients undergoing abdominal aortic aneurysm repair. Circulation 1987;**76**: III 1-7.

50. Yeager RA, Moneta GL, Edwards JM, Taylor LM, McConnell DB, Porter JM: Reducing perioperative myocardial infarction following vascular surgery. The potential role of beta-blockade. Arch Surg 1995;**130**:869-72; discussion 872-873.

51. Mangano DT, Layug EL, Wallace A, Tateo I: Effect of atenolol on mortality and cardiovascular morbidity after noncardiac surgery. Multicenter Study of Perioperative Ischemia Research Group. N Engl J Med 1996;**335**:1713-1720.

52. Palda VA, Detsky AS: Perioperative assessment and management of risk from coronary artery disease. Ann Intern Med 1997;**127**: 313-328.

53. Poldermans D, Boersma E, Bax JJ, Thomson IR, van-de-Ven LL, Blankensteijn JD, Baars HF, Yo TI, Trocino G, Vigna C, Roelandt JR, van-Urk H: The effect of bisoprolol on perioperative mortality and myocardial infarction in high-risk patients undergoing vascular surgery. Dutch Echocardiographic Cardiac Risk Evaluation Applying Stress Echocardiography Study Group. N Engl J Med 1999;**341**:1789-1794.

54. Poldermans D, Boersma E, Bax JJ, Thomson IR, Paelinck B, van-de-Ven LL, Scheffer MG, Trocino G, Vigna C, Baars HF, van-Urk H, Roelandt JR: Bisoprolol reduces cardiac death and myocardial infarction in high-risk patients as long as 2 years after successful major vascular surgery. Eur Heart J 2001;**22**:1353-1358.

55. Gibbs CR, Davies MK, Lip GY: ABC of heart failure. Management: digoxin and other inotropes, beta blockers, and antiarrhythmic and antithrombotic treatment. Bmj 2000;**320**:495-498.

56. Howell SJ, Sear JW, FoÎx P. Peri-operative fl-blockade: a useful treatment that should be greeted with cautious enthusiam. Br J Anaesth 2001;**86**:161-164.

57. Sear JW, Howell SJ, Sear YM, Yeates D, Goldacre M, FoÎx P: Intercurrent drug therapy and perioperative cardiovascular mortality in elective and urgent/emergency surgical patients. British Journal of Anaesthesia 2001;**86**:506-512.

POTASSIUM AND ANAESTHESIA

Dr Michael Girgis, Royal Devon and Exeter Hospital, Exeter

Potassium is the major intracellular cation in the body and has several roles, the most important being the generation of the resting cell membrane potential and the action potential, as well as protein synthesis, acid-base balance and maintenance of intracellular osmolality. The total amount of potassium present in the body is approximately 3200mmol, 90% of which is intracellular, and this is regulated by a variety of homeostatic mechanisms.

Physiology of Potassium

Of total body potassium, approximately 135-150mmol/l is present intracellularly, compared to plasma levels of 3.5-5.5mmol/l. The daily requirement is about 1mmol/kg/day, which is absorbed from the small intestine by diffusion.

Potassium Homeostasis

The kidney is the main organ involved in potassium regulation. There is, however, some secretion of potassium in the colon, some of which is reabsorbed in exchange for H^+. Both of these are under the control of aldosterone which is the main hormone involved in potassium regulation. In the kidney the majority of filtered potassium is reabsorbed in the proximal tubules. The distal tubules then secrete potassium, and normally the amount secreted is equal to potassium intake so balance is maintained.

There are several factors that can alter the amount secreted in the distal tubules:

- The rate of flow of fluid in the tubules - potassium secretion is proportional to the rate of flow. Increasing the flow limits the build up of potassium in the tubule which would otherwise increase and inhibit further secretion. This is the main reason why the plasma potassium level is seen to fall in patients having a large diuresis.
- Electrical coupling in the distal tubules means that potassium secretion is also dependent on sodium reabsorption. The greater the amount of sodium reabsorbed, the more potassium is secreted. This means that potassium secretion will increase when there is decreased sodium in the distal tubular fluid. Potassium secretion will also increase when sodium absorption is increased, which is under control of aldosterone and Atrial Natriuretic Peptide and hence also dependent on intravascular volume and blood pressure.
- High potassium levels stimulate aldosterone secretion and act as negative feedback to maintain levels. Sodium is also reabsorbed in exchange for H^+, however, so the amount of potassium secreted is also affected by acid-base balance. Potassium secretion is decreased when there is increased H^+ secretion, for example when a patient is acidotic.
- Via a similar mechanism, acidosis also results in a shift of potassium from the intracellular compartment to the extracellular compartment.

Insulin and catecholamines are also involved in potassium regulation. They both reduce the extracellular potassium concentration by increasing uptake into cells by an action on Na^+/K^+ ATPase and β2 receptors respectively. These mechanisms can be quite potent and the release of adrenaline during a stress response can cause an acute decrease in plasma potassium by 0.5-0.6mmol/l.

Potassium and the Membrane Potential

One of the most important roles of potassium is the resting membrane potential and in the repolarisation phase of action potentials. The normal cell membrane is relatively permeable to potassium ions, and impermeable to sodium and anions. The anions generate a negative intracellular potential. Because of the relative permeability of the cell membrane to potassium compared to sodium, this results in holding potassium intracellularly against

its chemical gradient, although there is a small leak of potassium down its concentration gradient which is balanced by the action of the Na^+/K^+ ATPase pump which in turn maintains the resting membrane potential. The end result is a resting membrane potential, the size of which is primarily due to the relative intracellular to extracellular concentration of potassium. It is the ratio of theses concentrations rather than the actual potassium concentrations themselves that is more important for maintaining the membrane potential. As a result, chronic disorders of potassium balance that involve a total body deficit or excess of potassium where the ratio is relatively well maintained, are frequently better tolerated and result in less symptoms to patients than acute disorders of potassium balance where the total body potassium may be relatively normal and the ratio disturbed. This is important when deciding whether or not to operate on a patient with an abnormal potassium level and some thought must be given to whether the disorder is chronic or acute, as well as the absolute plasma potassium level.

Hypokalaemia

This is typically taken as being a potassium level of less than 3.5mmol/l, though symptoms may not occur until the level is less than 2.5mmol/l. The total body deficit may be up to 500mmol. On average plasma potassium decreases by 0.3mmol/l for each 100mmol reduction in total body stores.

Causes may be acute or chronic.

Acute causes can be divided into two groups:

- Those that cause a shift of potassium intracellularly such as alkalosis, excessive use of β2 agonists, administration of insulin and glucose and hypothermia
- Those that result in a loss of potassium such as vomiting, diarrhea and losses in bowel fistulae, IV fluid therapy without potassium, diuresis due to solutes such as glucose or mannitol and the diuretic phase of acute renal failure.

Chronic causes of hypokalaemia tend to be those that result in a decrease in total body potassium such as dietary insufficiency, malabsorbtion, diuretics, Cushing's syndrome and hyper-aldosteronism.

Effect of Hypokalaemia

An acute decrease in the extracellular potassium concentration will result in an increase in the intracellular/extracellular ratio. This causes the membrane potential to become more negative and results in muscle and nerve cells becoming less excitable. This results in weakness and increased sensitivity to non-depolarising neuromuscular blocking drugs. In the heart, atrial, ventricular and conduction cells are all affected to a different extent resulting in increased automaticity, increased excitability and arrhythmias such as tachyarrhythmias and extrasystoles, and decreased cardiac contractility. Cardiac arrest may also occur. Patients may also develop impaired renal concentrating ability, increased toxic effects of digoxin, muscle weakness, hypotonia and alkalosis.

ECG changes: S-T segment depression, P-R and Q-T prolongation, T wave inversion and U waves.

Hypokalaemia and Anaesthesia

It used to be thought that because of the risk of developing arrhythmias, a healthy patient undergoing surgery with a potassium level of less than 3.0mmol/l should have their operation postponed if possible and have replacement therapy to normalize the plasma potassium. But there is a morbidity and mortality associated with replacement therapy. Several studies have shown no increased incidence of intraoperative cardiac arrhythmias in asymptomatic patients with chronic hypokalaemia so this is no longer the current view. Some authors have suggested that levels as low as 2.6-2.9mmol/l may be acceptable in otherwise healthy patients. There is little evidence to suggest an absolute level at which replacement therapy should be undertaken before surgery. Preoperative arrhythmias rather than plasma potassium has been shown to be a stronger predictor of intraoperative arrhythmias.

More important is taking each patient on an individual basis and assessing the following:

- The urgency of the operation,
- The type of surgery being undertaken
- The cause and time course as well as the degree of hypokalaemia,

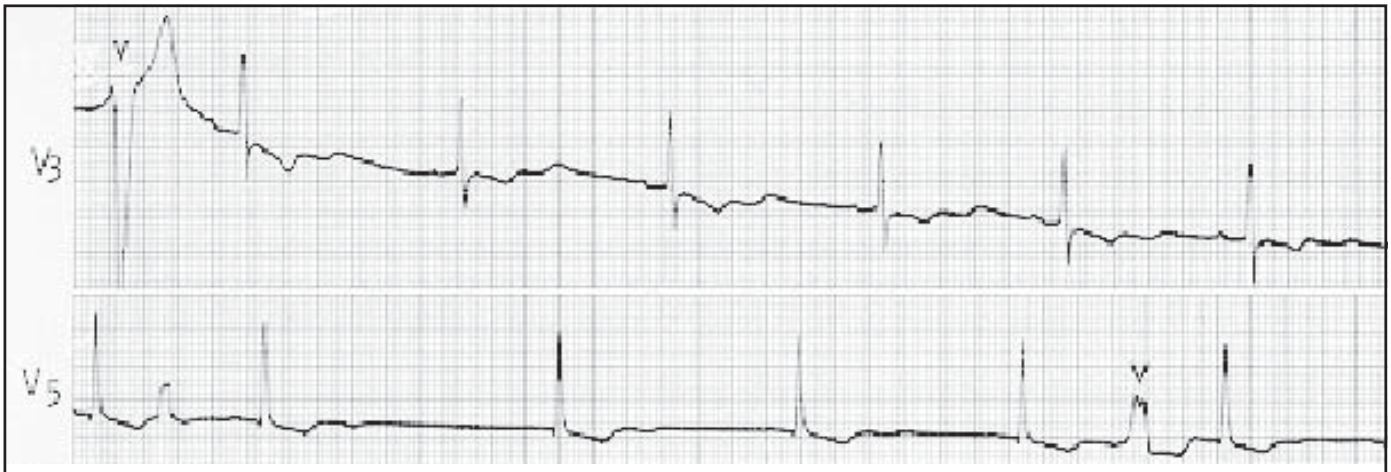


Figure 1. ECG showing T wave inversion and U waves

- Presence of arrhythmias, ECG changes or other symptoms,
- Cardiovascular risk factors such as myocardial ischaemia, heart failure or left ventricular hypertrophy
- Concurrent medications such as digoxin

Patients with acute or symptomatic hypokalaemia, preexisting cardiac disease and those taking digoxin are more likely to need replacement therapy if the level is less than 3.5mmol/l. Prior thought must be given to correction of the underlying disorder first, however, as replacement therapy may result in hyperkalaemia once the underlying cause has been corrected. Patients with chronic hypokalaemia may have a normal ICF/ECF ratio, and together with the lack of evidence that there is any increased risk due to chronic hypokalaemia, serious thought needs to be given as to whether there would be any benefit from delaying the operation and treating the hypokalaemia, which may cause problems by altering a normal membrane ratio.

The acute treatment involves first correcting the underlying disorder. If replacement therapy is deemed necessary it should be guided by estimation of total body deficit, and consists of:

- IV potassium chloride up to 40 mmol over 2 hours which needs to be given via a central vein and with full ECG monitoring, unless there is a metabolic acidosis in which case potassium bicarbonate is more suitable.
- If it does not need to be corrected quickly, oral supplementation can be used.

Caution must also be taken when correcting a chronic disturbance rapidly because this may result in an imbalance in the ICF/ECF ratio and arrhythmias. If possible, slow correction with oral potassium is better.

Anaesthetic management consists of preventing a further increase in hypokalaemia by allaying anxiety, avoiding dextrose solutions, maintaining a normal $PaCO_2$ and the use of a nerve stimulator to assess neuromuscular blockade.

Hyperkalaemia

This is defined as a plasma level of greater than 5.5mmol/l. Acute causes may be due to:

- potassium shift, such as in acidosis, rhabdomyolysis, trauma, malignant hyperpyrexia, suxemethonium (worsened by burns and nerve injury), familial periodic paralysis
- increased intake such as over-supplementation and blood transfusion.

The chronic causes are renal failure, Addison's disease and drugs such as potassium sparing diuretics, ACE inhibitors and cyclosporin.

Effect of hyperkalaemia

Acute hyperkalaemia causing a decrease in the ICF/ECF ratio will result in the resting membrane potential becoming less negative. The effect of this in muscle and nerve cells is that the membrane potential is closer to the threshold potential and so more excitable. If this continues, fatigue occurs resulting in muscle weakness. In the heart, excitability is decreased and as the activity decreases the threshold to ventricular fibrillation decreases so cardiac arrest in diastole may occur.

ECG changes: The earliest ECG change is a tall peaked T-wave, followed by prolongation of the P-R interval, widening of the QRS complex, absent P waves and slurring of S-T segments into T waves. Other symptoms include nausea and vomiting.

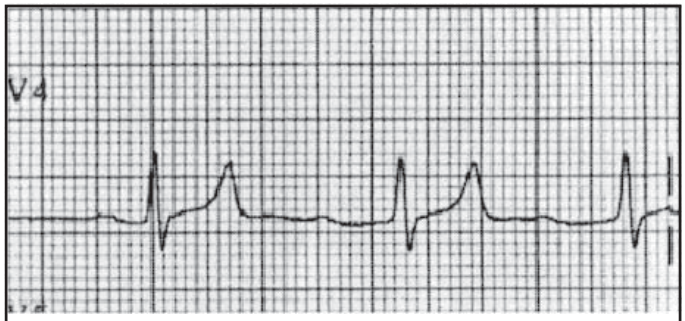


Figure 2. ECG showing peaked T waves

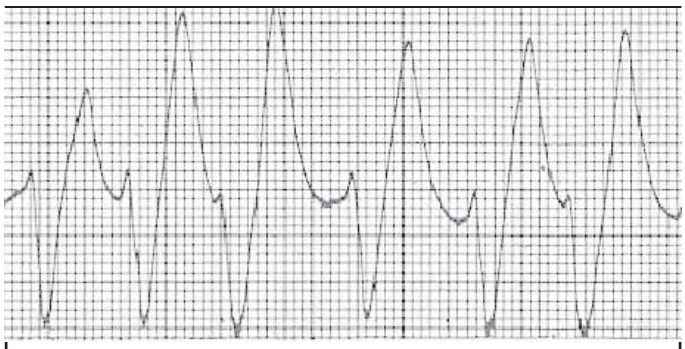


Figure 3. ECG showing wide QRS complexes

Hyperkalaemia and Anaesthesia

The decision to treat hyperkalaemia is easier and is based on the degree of elevation and the symptoms and signs present. If there are ECG changes or the concentration is greater than 6.5mmol/l the incidence of serious cardiac compromise is high and rapid intervention is necessary. A plasma potassium of less than 5.9mmol/l has been suggested before an elective operation. The cause should be investigated and corrected if possible.

Acute treatment may include the following:

- Insulin 5-10 units in 100ml of 10-20% dextrose IV over 30-60 minutes
- Salbutamol either 5mg nebulised or 50mcg bolus followed by 5-10mcg/min IV infusion
- Causing an alkalosis either by giving 50mmol IV bicarbonate or by increasing minute ventilation to cause a respiratory alkalosis if patient is ventilated.

The above all act by causing a shift of potassium intracellularly.

- Potassium exchange resins such as calcium resonium 15gPO/ 30gPR tds
- Dialysis/haemofiltration - these treatments act to remove potassium from the body.

- Calcium 5-10ml of 10% calcium gluconate IV if severe, or if there are cardiac manifestations. Calcium antagonizes the reduced conduction and improves myocardial contractility.

Further anaesthetic management consists of maintaining a normal or low $PaCO_2$ and avoidance of suxamethonium if possible.

Summary

Potassium is the major intracellular cation and is intimately involved in maintenance of the resting membrane potential. Hyper- and hypokalaemia can result in serious cardiac compromise, and treatment should be guided not only on the absolute levels but also on the presence of symptoms and other risk factors.

References

1. Gennari FJ. Hypokalaemia. New England Journal of Medicine 1998;**339**:451-8

2. Reddy VG. Potassium and Anaesthesia. Singapore Medical Journal 1998;**39**:511-6

3. Wong KC, Schafer PG, Schultz JR. Hypokalaemia and Anesthetic Implications. Anesthesia and Analgesia 1993;**77**:1238-60

4. Rastergar A, Soleimani M. Hyokalaemia and Hyperkalaemia. Postgraduate Medical Journal 2001;**77**:759-64

5. Tetzlaff JE, O'Hara JF, Walsh MT. Potassium and anaesthesia. Canadian Journal of Anaesthesia 1993;**40(3)**:227-46Hhhh

SELF ASSESSMENT

Dr Rob Law, Shrewsbury , UK

Question 1

A 70 year-old patient who is a heavy smoker with chronic obstructive pulmonary disease presents to the emergency department. He has had no previous surgery and his only medication is inhalers for his chest. He manages to walk about 2 miles a day and is otherwise well excepting for recent loss of weight. He gives a four-day history of mild abdominal pain, anorexia and constipation with increasing abdominal pain and vomiting over the last two days.

On examination:

General examination:	Pale. Apyrexial. Dry mouth. 70kg.
Cardiovascular:	Pulse 120 (sinus) B.P. 100/60 Cold peripheries.
Respiratory:	Slightly tachypnoeic. Chest clear.
Abdominal:	Very distended. Localised tenderness. No hernia. Increased bowel sounds.

Special investigations:

Chest Xray:	Chronic obstructive pulmonary disease. No air under the diaphragm.
Abdominal Xray:	Dilated colon and small bowel.
Full blood count:	Hb 9.0g/dl, WCC 13 x 10^9/l, Plts 600 x 10^9/l.
Biochemistry:	Creatinine 100 μmol/l, urea 15 mmol/l, Na 130mmol/l, K 3.5mmol/l
Arterial blood gas:	pH 7.3, pCO_2 4, pO2 8, BE-8 (breathing air)

The surgeon notifies you of this case, saying that he needs to do a laparotomy and that he suspects bowel obstruction due to a colonic malignancy. Discuss in detail the anaesthetic approach to this case and explain the pathophysiology and physiology likely to be responsible for the blood gas result.

ANSWER TO SELF - ASSESSMENT

Pre-operative Management

Although this patient has already undergone a number of special investigations and been in hospital a number of hours it is unfortunate that he has not yet received any treatment. Much of the initial resuscitation and treatment that he requires can occur without a specific diagnosis, and the sooner it is started the better. It may be useful to consider an A, B, C approach to his initial management.

Airway assessment: Conscious and maintaining his own airway. Start facemask oxygen. This patient has acute on chronic respiratory compromise, signs of poor perfusion and a low haemoglobin concentration (see Circulation) which will all result in a decreased oxygen delivery. His oxygen consumption is also likely to be raised due to the local and systemic effects of the bowel obstruction. Facemask oxygen will increase his arterial PO_2 and go some way to improving his oxygen delivery.

Oxygen delivery (mls O_2/min) = Hb (g/l) x 1.31 (mlsO_2/g Hb) x arterial O_2 saturation/100 x cardiac output (l/min). (See Update 10 for a full explanation)

Breathing assessment: The patient is hypoxic due to venous admixture. The cause is pulmonary shunt and V/Q mismatch secondary to chronic lung disease, and pulmonary atelectasis caused by abdominal distension and diaphragmatic splinting. The respiratory drive is increased and the arterial blood gas shows a partially compensated metabolic acidosis. Management**: Facemask oxygen and NG tube to decompress the bowel**. There are a number of potential causes for the metabolic acidosis. (base excess of -8). Anaerobic metabolism with lactic acid production secondary to a global reduction in oxygen delivery (shock), anaerobic metabolism in ischaemic bowel, acidosis due to decreased renal perfusion (shock) and loss of bicarbonate into the gut (balanced to some extent by a loss of acidic gastric secretions). Chemoreceptors in the carotid bodies sense the decrease in pH and respond by increasing ventilation. This results in a fall in pCO_2 and a respiratory alkalosis. Ventilation will also be increased in response to a low pO_2 stimulating the aortic and carotid bodies, however pO_2 values less than 8 kPa are necessary to produce the maximum response. Remember that the receptors that are normally the most important for the control of ventilation are those situated in the medulla. These respond to a high CO_2 level in the blood because of the effect that this has on reducing the cerebrospinal fluid pH. The Henderson-Hasselbach equation describes how the pH of the blood is due to a combination of respiratory and metabolic components. Tight control of pH is vital for metabolic processes and therefore the respiratory alkalosis (low CO_2) in this case is a physiological response designed to counteract the effect on pH of a metabolic acidosis (low HCO_3).

Circulation assessment: Dehydration (based on the history you know the patient will be dehydrated but this is confirmed by the dry mouth and the urea/creatinine ratio). Additional signs might include decreased skin turgor, sunken eyes, absence of sweating and decreased urine output. Poor perfusion/shock - increased heart rate, low BP, cold peripheries and acidosis. This is caused by fluid loss into the bowel and peritoneum, and losses due to vomiting. Septic shock due to bacterial translocation across the gut wall or due to local complications such as perforation or strangulation may also be the cause. Anaemia (the haemoglobin will be lower than this when rehydrated). Probably due to chronic blood loss into the gut and an 'anaemia of chronic disorders'.

Correct the intravascular deficit immediately. Remain with the patient and assess the response to repeated fluid boluses. In this patient a colloid should be used initially but blood should be used when it is available. Send blood for cross-match (4 units) and coagulation tests. A decrease in heart rate, increase in blood pressure and improvement in peripheral perfusion are the changes that one would hope to see at the bedside.

Insert a urinary catheter and monitor the urine output.

Correct dehydration. Intravenous crystalloid should be prescribed to replace lost water and electrolytes. (see explanation below).

Consider inserting a CVP line. Useful to guide fluid therapy especially in the elderly and those with impaired cardiac function and to give inotropes if necessary. There is no need for this to be inserted immediately. It may also be useful to aspirate mixed venous blood and determine its oxygen saturation. Patients with a mixed venous oxygen saturation of less than 70% have a high global oxygen extraction due to a low oxygen delivery compared to oxygen consumption. They may benefit from attempts to increase oxygen delivery with further fluids and inotropes. An acidosis that persists in patients with a high mixed venous oxygen saturation (implying an adequate oxygen delivery) may be due to renal failure or ischaemic bowel. It is also sometimes seen in the late stages of septic shock and is thought to be due to microcirculatory abnormalities or 'sick' cells that are unable to utilise the oxygen supplied to them.

Inotropes as necessary.

The fluid and electrolyte abnormalities that occur in bowel obstruction depend on the site of the obstruction. It is important to understand how the various biochemical abnormalities arise and how to treat them. **Pyloric obstruction** causes a loss of H^+ and Cl^- (and Na^+ and K^+) due to vomiting acidic gastric secretions. Alkaline pancreatic and duodenal secretions are retained and the result is a hypochloraemic metabolic alkalosis. This affects the renal handling of Na^+. Na^+ is normally reabsorbed in the proximal tubule for which Cl^- needs to be available, as HCO^{3-}, the only other significant anion, cannot pass easily through the proximal

tubular cell wall. Initially HCO_3-, and Na+ are lost in the urine. Subsequently an increase in aldosterone caused by a reduction in circulating plasma volume causes Na+ to be reabsorbed distally in the tubule in exchange for K+ and H+ making the alkalosis worse and causing hypokalemia. The result is a hyponatremic, hypokalemic, hypochloremic metabolic alkalosis.

Mid or high small bowel obstruction presents a different picture. Large volumes of fluid are lost (Na+, K+ and water) as the absorption of saliva, bile, gastric, pancreatic and duodenal secretions are impaired (the gastrointestinal tract secretes 8 litres of fluid a day). The loss of a combination of alkaline intestinal secretions and acidic gastric secretions prevents the development of a metabolic alkalosis.

In **low small bowel obstruction and large bowel obstruction** fluid loss tends to be less initially as much of the water and solute secreted into the gut can be absorbed above the obstruction. In all the above situations if the obstruction is not relieved and intravenous fluid replacement does not take place the combined effects of decreased fluid intake, vomiting, fluid loss into the bowel and peritoneum, bowel perforation, bowel ischaemia, peritonitis and sepsis leads to circulatory collapse and metabolic acidosis.

In all cases of dehydration due to bowel obstruction there is a total body deficit of Na+ and water and therefore whatever the Na+ concentration (i.e. whether the patient is hyponatremic or hypernatremic) replacement needs to be with a fluid with a high Na+ content (0.9% saline or Hartmanns solution). K+ should be added to the fluid as necessary (usually 20-40mmol/l provided the patient is not anuric or hyperkalaemic). The aim should be to correct the dehydration over 24 hours, giving half the calculated amount in the first 8 hours and the second half over the following 16 hours. If the patient is very hypernatremic (Na+ > 155mmol/l) rehydration should be over 48 hours because of the risk of cerebral oedema. It is often helpful to crudely classify the extent of dehydration as 5%, 10% or 15% rather than as mild, moderate or severe because this enables one to estimate roughly what the fluid deficit might be. A patient that is 5% dehydrated has lost 50 ml/kg of fluid and a patient that 10% dehydrated has lost 100ml/kg of fluid etc. This calculation serves as a useful starting point when prescribing rehydration fluid but it needs to be emphasised that the rate of fluid administration should be increased or decreased depending on the results of future assessments.

Returning to the example above, the patient will need intravenous fluids to be administered to take account of the following:

- **Maintenance fluid** - 2000 - 3000mls/day
- **Ongoing losses.** Initially difficult to quantify but includes NG loss and loss into bowel. A conservative estimate would be 2000 mls/day
- **Fluid deficit** - If 7.5% dehydrated then the deficit is 75mls/kg or 5250mls
- **Therefore the total fluid requirement for the first day is about 10 litres. Rehydration should therefore be started with 0.9% saline + 20mmol/l KCl at 600mls/hour for the first 8 hours**. Frequent reassessment will be necessary and the rate of fluid administration adjusted accordingly.

Once the above treatment has been commenced the **surgeon and anaesthetist should decide together when they think that the patient will be in an optimum condition to undergo an operation.** Ideally the patient would go to theatre when fully rehydrated but surgical urgency may dictate otherwise. The natural history of bowel obstruction is that the bowel above the obstruction initially increases its blood supply and peristalsis in an attempt to overcome the obstruction. If the obstruction is not relieved the bowel dilates and then becomes flaccid. The distension is caused by gas (nitrogen and hydrogen sulphide) due to bacterial overgrowth and unabsorbed digestive juices. The intramural vessels become stretched and the bowel wall becomes oedematous and ischaemic. Eventually fluid leaks out into the peritoneum and perforation or necrosis of the bowel wall may occur. Patients with necrosed or perforated bowel will need to proceed to operation as early as possible and it may not be possible to rehydrate them fully before operation. A very high WCC (>25 $x10^9$/l) or peritonitis may signify these complications and the localised tenderness in this may also increase the surgical urgency. A reasonable compromise in this case may be to plan to do the laparotomy after 8 hours of resuscitation when hopefully the shock and anaemia will have been corrected and the patient will have received about 5000mls of rehydration fluid. Other factors that should be considered are the need for **thromboembolism prophylaxis and antibiotics.**

Intra-operative and postoperative management

After adequate preparation and monitoring the NG tube should be aspirated and anaesthesia should be induced using a **rapid sequence induction** technique with **cricoid pressure. Large bore venous access** should be obtained if this is not already in place. A **CVP line** will be useful to guide intra-operative fluid therapy and an **arterial line** will allow beat-by-beat assessment of blood pressure and facilitate the intraoperative monitoring of haemoglobin concentration, acid-base balance and electrolyte concentrations. If the patient's coagulation status is normal an **epidural** may help provide high quality postoperative pain control, and will decrease the likelihood of postoperative pulmonary morbidity. It may be prudent not to use the epidural immediately, but rather to establish the block cautiously during the operation. The use of epidural anaesthesia combined with general anaesthesia in a partially rehydrated, elderly patient having emergency surgery with the potential to develop a systemic inflammatory response and intra-operative fluid shifts may cause profound hypotension, and therefore caution is essential. Intra-operative hypothermia should be prevented by the use of **fluid warmers** and a **warming mattress**.

Intra-operative fluid requirements include the pre-operative requirements and in addition replacement of blood loss and 'third space' losses. 'Third space' loss is the loss of extracellular fluid that occurs during a laparotomy due to the trauma, manipulation and retraction of the abdominal contents. It is not possible to measure this but it usually amounts to about 10mls/kg/hour of surgery of this magnitude.

Measurement of pulse rate, CVP and urine output will help to guide fluid therapy intra-operatively. An **oesophageal doppler** monitor provides an additional non-invasive measure of cardiovascular output and filling in some centres. At the end of the procedure a decision will need to be made as to whether it is appropriate to wake the patient up and extubate him. Patients who are hypothermic, cardiovascularly unstable or very acidotic should be ventilated postoperatively, and managed on an intensive care unit.

If this patient is extubated, **good analgesia** will enable him to cough and breath deeply, and chest **physiotherapy** will help to prevent pulmonary atelectasis and infection. NSAIDs should be used with caution because of their propensity to precipitate acute tubular necrosis in elderly, dehydrated patients. Nutritional support is very important in this patient because of the period without adequate nutritional intake pre-operatively and because of the catabolic effect of cancer and surgical trauma. NG feeding should be started as soon as the surgical procedure allows. Postoperative oxygen, fluids, DVT prophylaxis and antibiotics should be prescribed as indicated. A bed on the **High Dependency Unit**, if available, is the most appropriate place to care for this patient.

SELF ASSESSMENT - Questions

Dr Rebecca Appleboam and Dr Ed Hammond

Question 1

The following statements about suxamethonium are true

A. It is the muscle relaxant most frequently implicated in allergic reactions
B. It is metabolised to two molecules of acetylcholine by the action of plasma pseudocholinesterase
C. The rise in intraocular pressure caused by its administration lasts for up to half an hour
D. It may cause a bradycardia due to stimulation of nicotinic receptors
E. The Phase II block seen after repeated administration or infusion is reliably reversed using neostigmine

Question 2

The following statements are true about digoxin

A. 75% of the oral dose is absorbed
B. 50% is bound to plasma proteins
C. Most is excreted unchanged in the urine
D. Hyperkalaemia may cause raised serum levels of digoxin
E. Toxicity may result in complete heart block

Question 3

According to the Hagen-Poiseuille equation

A. Flow varies inversely with the fourth power of the radius
B. Flow varies inversely with fluid density
C. Flow varies directly with pressure difference between the ends of the vessel
D. Haematocrit is likely to inversely affect blood flow
E. If the radius of a vessel is doubled, resistance will fall to less than 50% of its previous value.

Question 4

Diastolic filling of the left ventricle

A. Is aided by a modest tachycardia when the left ventricle is hypertrophied
B. Occurs mainly in later diastole, including the time of atrial systole
C. Active relaxation is improved by sympathetic stimulation in the setting of a steady heart rate
D. Is most commonly disturbed by hypertensive cardiac disease
E. Can be represented by a constant of ventricular stiffness

Question 5

Sevoflurane

A. Is a hexafluoroisopropyl fluromethyl ether
B. Undergoes minimal biotransformation in the liver to produce inorganic fluoride ions
C. Releases carbon monoxide when in contact with sodalime
D. Has a lower SVP than isoflurane
E. Has a blood:gas partition coefficient approximately half that of isoflurane

Question 6

Rocuronium

A. Is more potent than vecuronium
B. Is chemically related to vecuronium
C. In suitable doses produces good intubating conditions in 60-90's
D. Has a longer elimination half time than vecuronium
E. Possesses active metabolites

Question 7

The following are likely to cause serious complications during pregnancy

A. Mitral stenosis
B. Secundum atrial septal defect
C. Ventricular septal defect with normal pulmonary artery pressure
D. Isolated aortic regurgitation
E. Primary pulmonary hypertension

Question 8

Pulmonary hypertension

A. Causes wide splitting of S2
B. Is a cause of the Graham-Steele murmur
C. Can cause peripheral cyanosis
D. Is a cause of atrial fibrillation
E. Causes giant a waves in the JVP

Question 9

Respiratory failure

A. $PaCO_2$ is <8.0 kPa by definition
B. Lactic acidosis may occur
C. Is always due to pre-existing lung disease
D. Should always be treated with 100% oxygen
E. Is a recognised complication of diphtheria

Question 10

A small pupil is characteristic of

A. IIIrd nerve palsy
B. Horner's syndrome
C. Tabes dorsalis
D. Optic neuritis
E. Holmes-Adie pupil

Question 11

Regarding the physiology of glucose control

A. A protein meal stimulates glucagon release
B. A protein meal stimulates insulin release
C. Somatostatin infusion induces hypoglycaemia
D. Insulin-like growth factor-I (ILGF-I) secretion by the liver is stimulated by insulin
E. Ketone body synthesis is stimulated by insulin

Question 12

In paracetamol overdose

A. The toxic metabolite is N-acetyl-P-benzoquinonimine
B. Decreased conscious level is common on admission
C. A paracetamol level above 200 ng/L at 4 hours after ingestion requires treatment with acetylcysteine
D. Those taking enzyme-inducing drugs are at increased risk
E. After a severe overdose the patient should not take paracetamol again, even in normal therapeutic doses

Question 13

Albumin

A. Has a biological half life of 20 days
B. Has a molecular weight of 65,000 Daltons
C. Analbuminaemia presents with severe peripheral oedema
D. 60 % of the extracellular albumin is in the plasma compartment
E. Plasma levels vary with posture

Question 14

Pressure can be measured with the following

A. Aneroid gauge
B. Bourdon gauge
C. Rayleigh refractometer
D. Raman gauge
E. Displacement of a flexible diaphragm

Question 15

The following are true regarding breathing systems

A. The Bain circuit is an example of a Mapleson A system
B. The Bain circuit is more efficient than the Lack circuit during spontaneous breathing
C. In the Bain circuit, fresh gas flow occurs through the outer tube
D. During spontaneous breathing, the Lack circuit requires a fresh gas flow rate of twice the alveolar minute ventilation to prevent rebreathing
E. The Lack circuit may be used to ventilate the patient's lungs with the Penlon Nuffield 200 ventilator

Question 16

Adverse drug reactions

A. Most commonly affect the cardiovascular and respiratory systems
B. Are uncommon in patients taking digoxin and diuretics
C. Often affect the gastrointestinal tract and skin
D. Are particularly likely to occur in females over 60 years old
E. Cause up to 3% of admissions to acute medical wards

Question 17

The following are natural precursors of adrenaline

A. Noradrenaline
B. Glycine
C. Tyrosine
D. Phenylalanine
E. Dobutamine

Question 18

Warfarin

A. Prevents the carboxylation of vitamin K
B. Interferes with the synthesis of clotting factors II, V, X and XII
C. Has an anticoagulant effect delayed by about 12 hours following the first dose
D. Is teratogenic
E. Is excreted in the urine

Question 19

The following poisons are matched to the appropriate therapy

A. Carbon monoxide — hyperbaric oxygen
B. Organophosphates — atropine and pralidoxime
C. Beta-blocker — phentolamine
D. Methanol — ethanol
E. Tricyclic antidepressants — phenytoin

Question 20

Gastrointestinal motility is affected by

A. 5HT3 antagonists
B. Metoclopramide
C. H2 receptor blockers
D. Neostigmine
E. Opioids via the chemoreceptor trigger zone

SELF ASSESSMENT - Answers

Dr Rebecca Appleboam and Dr Ed Hammond

Question 1

A. true B. false C. false D. false E. false

It is composed of two molecules of acetylcholine, but is metabolised to the relatively inactive succinyl monocholine. The rise in intraocular pressure caused by suxamethonium alone is brief, lasting for a few minutes. The bradycardia is caused by activation of muscarinic receptors. Phase II block does exhibit the characteristics of non-depolarising block but is not reversed by anticholinesterases.

Question 2

A. true B. false C. true D. false E. true

There is insignificant binding to plasma proteins. Hypokalaemia may precipitate digitalis toxicity. All forms of heart block have been recorded in digitalis toxicity.

Question 3

A. false B. false C. true D. true E. true

Flow = [Pressure difference x Pi x (fourth power of radius)] / [8 x length x VISCOSITY]. Also don't forget that Flow = Pressure difference/RESISTANCE. Blood viscosity depends on haematocrit. If the radius of a vessel is doubled, resistance will fall to 6% of its previous value.

Ref: Ganong WF. Review of Medical Physiology. Lange,

Question 4

A. false B. false C. true D. true E. true

Diastole is divided into active relaxation, rapid filling, slow filling and atrial systole. Active relaxation is improved by sympathetic stimulation, increased inotropic state and increased heart rate. In the early part of diastole 70% of ventricular filling occurs. Especially when the left ventricle is hypertrophied any increase in heart rate will adversely affect left ventricular filling. Ventricular filling is most commonly disturbed by hypertensive heart disease and myocardial infarction. A modulus of chamber stiffness is the slope of the (dp/dv)/P relationship for the exponential curve of diastolic pressure against volume.

REF: Priebe & Skarvan. Cardiovascular Physiology. BMJ Publishing. Chapter 2. Ventricular performance.

Question 5

A. true B. false C. false D. true E. true

Sevoflurane is indeed a hexafluoroisopropyl fluromethyl ether. It is related to isoflurane, enflurance and desflurane which are all also ethers. Halothane is a hydrocarbon. About 5% undergoes biotransformation in the liver. It does not possess a -CF2H group and thus produces no CO when in contact with very dry sodalime (this property is shared with halothane). Its SVP at 20 degrees C is 160 mmHg (isoflurane 238 mmHg), its BP is 56 degrees C (isoflurane 48.5, enflurane 56.5) and its blood:gas partition coefficient is 0.69 (isoflurane 1.15).

Ref: British Journal of Anaesthesia 1996; 76: 435-445

Question 6

A. false B. true C. true D. true E. false

Rocuronium is an aminosteroid based neuromuscular blocker. It has a monoquaternary structure similar to pancuronium and vecuronium. It is much less potent with an ED 95 of 0.3 mg/kg (vec 0.056 mg/kg). The lack of potency is thought to be an important factor in determining the speed of onset of neuromuscular block. The less potent the drug, the greater the number of molecules there are available to diffuse into the NMJ. A more rapid onset is likely to be achieved with the less potent drug due to the increased diffusion gradient (due to the number of molecules) with the higher dose of the weaker agent. Rocuronium has no active metabolites because of the lack of a methyl group at the 3 carbon position. For vecuronium the metabolite 3 disacetylvecuronium is active.

Ref: British Journal of Anaesthesia 1996; 76: 481-483

Question 7

A. true B. false C. false D. false E. true

Pregnancy is associated with marked haemodynamic changes. The blood volume increases substantially. The heart rate, stroke volume and cardiac output increase while systemic blood pressure and vascular resistance fall (diastolic BP > systolic producing a wide pulse pressure). Patients with mitral stenosis may deteriorate significantly during gestation due to the fixed flow obstruction. The increased heart rate and cardiac output with the decrease in colloid osmotic pressure predispose to pulmonary oedema. Aggressive diuretic therapy is contraindicated as it may decrease uterine perfusion pressure. VSD and ASD are usually well tolerated in pregnancy even among patients with large left-right shunts. However, the degree of pulmonary hypertension should guide management as marked reduction in blood pressure during or after delivery may result in reversal of the shunt. Aortic regurgitation is also well tolerated (as is mitral regurgitation) probably because the systemic vascular resistance falls. Pregnancy in patients with primary pulmonary hypertension is associated with a high mortality, probably due to right ventricular ischaemia and failure, increased arrhythmias and pulmonary embolism.

Question 8

A. false B. true C. true D. true E. true

Clinical signs of pulmonary hypertension are raised JVP with prominent a waves and large v waves (especially with coexistent tricuspid regurgitation), left parasternal heave, sometimes a palpable pulmonary second sound, and on auscltation, loud P2 sometimes with an ejection click (P2 is closer to A2, not further apart), pansystolic murmur of tricuspid incompetence, right ventricular fourth heart sound and early diastolic murmur of functional pulmonary regurgitation (the Graham-Steele murmur). Atrial fibrillation may occur.

Question 9

A. false B. true C. false D. false E. true

Respiratory failure is defined as PaO2 <8kPa. Type I is characterised by ventilation perfusion mismatch and patients have a PaCO2 <6.5kPa while type II is characterised by alveolar hypoventilation and patients have a PaCO2 >6.5 kPa. Often both coexist. Hypoxia results in confusion, cyanosis and eventually coma. Hypercapnoea produces papilloedema, miosis, hypertension, flapping tremor, hyporeflexia, muscle twitching, sweating, headache, bounding pulse, retinal vein distension and eventually coma with extensor plantars. Lactic acidosis is a common finding due to anaerobic metabolism within tissues. Treatment should be directed at the precipitant as well as supportive therapy. 100% oxygen is unsafe in patients with COAD. Artificial ventilation or doxapram are the mainstay. Diptheria produces neuromuscular paralysis and can precipitate respiratory failure. Other causes of neuromuscular respiratory failure include: myasthenia gravis, motor neurone disease, polymyositis, muscle dystrophies (e.g. myotonic), polio, multiple sclerosis, stroke, encephalitis, etc.

Question 10

A. false B. true C. true D. false E. false

The pupil is dilated in IIIrd nerve palsy (compressive lesion), Holmes-Adie syndrome (myotonic pupil - often unilateral and poorly responsive to light; associated with reduced or absent ankle and knee reflexes), midbrain lesions, congenital syphilis, anticholinergic treatment (atropine), cocaine intoxication.

Causes of Horner's syndrome include Pancoast's tumour (apical lung carcinoma involving sympathetic chain), iatrogenic (sympathectomy), syringomyelia, lateral medullary syndrome, Shy Drager syndrome (causes Parkinsonism with postural hypotension and atonic bladder).

Other causes of small pupil include myotonic dystrophy, pontine lesions, acute iritis, opiates and organophosphates. The Argyll-Robertson pupil is seen in neurosyphilis - the pupil is unreactive to light but reacts to accomodation (a similar phenomenon may be seen in DM).

Question 11

A. true B. true C. true D. false E. false

A protein meal stimulates both insulin and glucagon secretion; the glucagon prevents the hypoglycaemia that would result from the increased insulin levels if there were no carbohydrate with the protein. Somatostatin infusion inhibits both insulin and glucagon secretion and produces hypoglycaemia, suggesting that glucagon is essential for the liver to release glucose. In addition, cortisol and growth hormone are required for normal glucose efflux from the liver. ILGF-I and ILGF-II are peptides that appear to function primarily as growth factors rather than influencing glucose uptake by tissues. ILGF-I is synthesised by the liver in response to growth hormone (not insulin). Beta-oxidation of free fatty acids to form acetyl CoA and ketone bodies provides the energy required for gluconeogenesis in starvation. This is inhibited by insulin.

Question 12

A. true B. false C. false D. true E. false

In a pure paracetamol overdose patients are normally fully conscious on admission. A decreased level of consciousness suggests another substance has been taken. A paracetamol level above 200 mg/L at 4 hours suggests treatment is indicated. Alcoholics and those taking enzyme-inducing drugs should be treated at half this level. Even after an overdose that causes severe hepatic damage long term sequelae don't develop, and normal therapeutic doses of paracetamol can be taken.

Question 13

A. true B. true C. false D. false E. true

Albumin , with a molecular weight of 65,000 Da and a plasma half life of 20 days is synthesised in the liver. Approximately 60% of albumin in the extracellular compartment is in the interstitial compartment though the concentration in the plasma compartment is very much higher. Albumin levels vary by as much as 5-10g/litre in the recumbent patient due to fluid redistribution. Analbuminaemia is a rare condition in which despite the complete lack of albumin there is only minimal ankle oedema following prolonged standing

Ref: Zilva JF, Pannall PR & Mayne PD. Clinical chemistry in diagnosis and treatment.

Question 14

A. true B. true C. false D. false E. true

High pressures commonly employed in anaesthetic practice can be measured using a Bourdon gauge. In this gauge, the gas at high pressure causes a tube to uncoil and in doing so moves a pointer over a scale on a dial. Bourdon gauges have the advantage over manometers that there is no liquid to spill, and they are sometimes called anaeroid gauges from the Greek 'a-neros' (without liquid). Another form of anaeroid gauge is based on a bellows or capsule which expands or contracts depending on the pressure across it. The strain gauge pressure transducer involves movement of a diaphragm with changes in pressure. This movement of the diaphragm alters the tension in the resistance wire thus changing its resistance. The change of current flow through the resistor can then be amplified and displayed as a measure of pressure on a scale. The Rayleigh refractometer and Raman spectrophotometer are techniques used for anaesthetic gas analysis.

Ref: P D Davis, G D Parbrook, G N C Kenny. Basic Physics and Measurement in Anaesthesia, 4th ed. Butterworth-Heinemann, 1995.

Question 15

A. false B. false C. false D. false E. false

The Bain circuit is the coaxial version of the Mapleson D system. Fresh gas flow (FGF) is supplied through a narrow inner tube. The patient's expired gases pass through the outer tube and are vented to atmosphere. This sytem is inefficient during spontaneous breathing but efficient during controlled ventilation. A FGF rate of between two to three times minute volume (200-250 ml/kg/min) may be required during spontaneous ventilation to prevent rebreathing. FGF of between 70 and 80 ml/kg/min is required during controlled ventilation to prevent rebreathing.

The Lack circuit is the coaxial version of the Mapleson A system. The outer tube supplies inspired gas from the reservoir bag and the patient exhales through the inner tube. This system is inefficient during controlled ventilation but efficient during spontaneous breathing. During controlled ventilation, the FGF rate must be at least three times alveolar minute volume to prevent rebreathing. If the system is functioning correctly and no leaks are present, a FGF rate equal to the patient's alveolar minute ventilation is sufficient to prevent rebreathing. In practice, a higher FGF rate (equal to the minute volume) is selected to compensate for leaks. Unlike the Bain circuit, the Lack circuit does not permit the use of ventilators to provide controlled ventilation.

Ref: A R Aitkenhead, G Smith. Textbook of Anaesthesia,

Question 16

A. false B. false C. true D. true E. true

The most common adverse drug reactions are gastrointestinal (nausea) and dermatological (rashes). Approximately 3% of hospital admissions are directly related to adverse drug interactions.

Question 17

A. true B. false C. true D. true E. false

Dopamine is a precursor of adrenaline. Dobutamine is a synthetic compound. The synthetic pathway is as follows:

Tyrosine-DOPA-Dopamine-Noradrenaline-Adrenaline

Question 18

A. true B. false C. true D. true E. false

Warfarin interferes with the activation of vitamin K, and thereby prevents the hepatic synthesis of the vitamin K dependent clotting factors II, VII, IX and X. It has serious teratogenic effects, one third of infants being still born, or born with severe abnormalities. It is 97% bound to albumin, and there is therefore negligible urinary excretion. Metabolites are conjugated with glucuronic acid and excreted in the bile and urine.

Question 19

A. true B. true C. false D. true E. true

For beta-blocker overdose, try atropine, glucagon infusion and temporary pacing. Tricyclic overdose may require iv neostigmine to counteract the anticholinergic effects and a beta blocker for treatment of SVTs. Phenytoin is useful for convulsions and VT in TCA poisoning. Other antidotes include desferrioxamine for iron, calcium EDTA and/or dimecaprol for lead poisoning, dimecaprol for heavy metal poisoning, ethanol for ethylene glycol, dicobalt edetate for cyanide, digoxin-specific antibody for digoxin, naloxone for opiates, N-acetylcysteine for paracetamol, Fuller's earth for paraquat, vitamin K for warfarin.

Question 20

A. false B. true C. false D. true E. false

Ondansetron (5HT3 antagonist) reduces nausea and vomiting by central activity. Opioids and metoclopramide have both central and local actions on gut motility. Neostigmine causes a rise in acetylcholine levels and will increase segmental contractions within the bowel.

ABSTRACTS FROM ANAESTHESIA

The journal of the Association of Anaesthetists of Great Britain and Ireland. Reprinted with permission of the editor Professor Harmer, and the publisher Blackwell Scientific

Anaesthesia 2002;57:61-5

Comparison of cyclizine and ondansetron for the prevention of postoperative nausea and vomiting in laparoscopic day-case gynaecological surgery

K. Grimsehl, J. B. Whiteside and Neil Mackenzie

Seventy-four patients undergoing laparoscopic gynaecological surgery were randomly allocated to two groups receiving cyclizine 50 mg or ondansetron 4 mg at induction of anaesthesia. Anaesthetic and postoperative analgesia regimens were standardised. Approximately half of the patients in each group experienced some degree of postoperative nausea and vomiting (cyclizine, 56%; ondansetron, 54%). There was no difference between groups in respect of pre- and postdischarge incidence. Mean (SD) time to eye opening was significantly prolonged in the cyclizine group [10 (4) min vs. 8 (2) min; $p < 0.001$], but this had no influence on discharge times. Cyclizine and ondansetron appear equally effective in preventing postoperative nausea and vomiting but the 10-fold price differential favours cyclizine.

Anaesthesia 2002;57:128-32

Comparison of times to achieve tracheal intubation with three techniques using the laryngeal or intubating laryngeal mask airway

J. J. Pandit, K. MacLachlan, R. M. Dravid and M. T. Popat

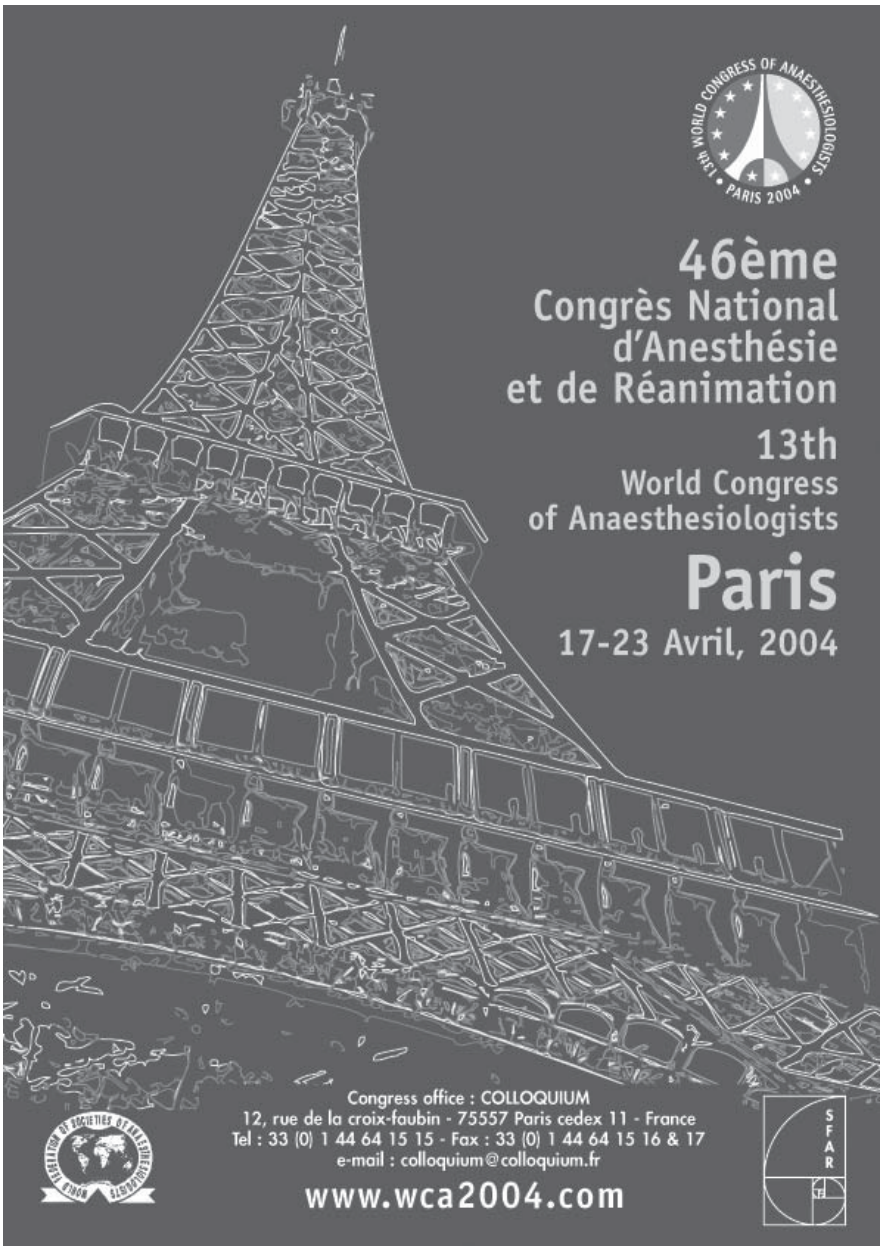
Summary. We compared the times to intubate the trachea using three techniques in 60 healthy patients with normal airways: (i) fibreoptic intubation with a 6.0-mm reinforced tracheal tube through a standard laryngeal mask airway (laryngeal maskfibreoptic group); (ii) fibreoptic intubation with a dedicated 7.0-mm silicone tracheal tube through the intubating laryngeal mask airway (intubating laryngeal maskfibreoptic group); (iii) blind intubation with the dedicated 7.0-mm silicone tracheal tube through the intubating laryngeal mask airway (intubating laryngeal maskblind group). Mean (SD) total intubation times were significantly shorter in the intubating laryngeal maskblind group (49 (20) s) than in either of the other two groups (intubating laryngeal maskfibreoptic 74 (21) s; laryngeal maskfibreoptic group 75 (36) s; $p < 0.001$). However, intubation at the first attempt was less successful with the intubating laryngeal maskblind technique (15/20 (75%)) than in the other two groups (intubating laryngeal maskfibreoptic 19/20 (95%) and laryngeal maskfibreoptic 16/20 (80%)) although these differences were not statistically significant. We conclude that in this patient group, all three techniques yield acceptable results. If there is a choice of techniques available, the intubating laryngeal maskblind technique would result in the shortest intubation time.

Anaesthesia 2002;57:338-47

REVIEW - Isoflurane and coronary heart disease

N. M. Agnew, S. H. Pennefather and G. N. Russell

Summary. Early studies indicated that isoflurane caused coronary steal and should therefore be avoided in patients with coronary heart disease. Subsequently, more detailed trials have disputed this and have shown that as long as coronary perfusion pressure is maintained, isoflurane does not cause coronary steal or myocardial ischaemia. There is now growing evidence, initially in animal work but more recently in human studies, that isoflurane has myocardial protective properties, limiting infarct size and improving functional recovery from myocardial ischaemia. The mechanism for this protection mimics ischaemic preconditioning and involves the opening of adenosine triphosphate-dependent potassium channels. The few studies comparing the myocardial protection offered by individual anaesthetic agents indicate that isoflurane represents the anaesthetic agent of choice for patients with coronary heart disease.



Dear Reader

If you would like to receive **Update in Anaesthesia** please write to: Mrs Christine Lethbridge, Department of Anaesthetics, Royal Devon and Exeter Hospital (Wonford), Exeter EX2 5DW, UK. The fax number is + 44 1392 402472. Alternatively contact her by email Christine.Lethbridge@rdehc-tr.swest.nhs.uk 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.

Sponsored by: World Federation of Societies of Anaesthesiologists, 21 Portland Place, London, W1B 1PY, United Kingdom. Tel: 020 7631 1650. E-mail: wfsa@compuserve.com

Typeset by: Angela Frost

Printed in Great Britain by: Media Publishing

Correspondence to editor: Dr I H Wilson, Anaesthetics Department, Royal Devon & Exeter Healthcare NHS Trust, Barrack Road, Exeter, EX2 5DW, UK. E-mail: iain.wilson@rdehc-tr.swest.nhs.uk