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Editorial

The World Congress of Anaesthesiology was held in Montreal, Canada in June of this year. The event, staged by the Canadian Society of Anaesthetists, was a fantastic success with over 6000 delegates from more than 90 countries attending. It was fascinating to hear and see presentations from many parts of the world, from various grades of anaesthetist concerning all aspects of anaesthesia. Thanks to generous donations, over 50 delegates from developing countries were able to attend and were warmly applauded at the opening ceremony.

Dr Kester Brown from Melbourne, Australia was elected as President of the World Federation of Societies of Anaesthesiologists. We congratulate Kester on his appointment and also thank him for his continuing support of Update in Anaesthesia.

We are delighted that the W.F.S.A. has decided to continue to fund Update in Anaesthesia, making it available free of charge to those working in developing countries who are unable to afford the subscription. Please note that we are revising our distribution list for Update in Anaesthesia and if you wish to continue to receive Update please register with us by following the instructions on the back cover of this edition. Alternatively contact the editor by email iain.wilson5@virgin.net.

Update is now produced in Russian, French, Spanish and Mandarin as well as English. Contact details to receive these are printed below.

The English version is produced in a paper format, CD Rom (email Michael.Dobson@nda.ox.ac.uk) and on the web www.nda.ox.ac.uk/wfsa. The demand for Update has increased dramatically and we hope it will continue to meet the needs of our readers.

We are always pleased to receive letters about any material in Update and would be grateful for ideas for future articles. If you wish to contribute to Update please contact the Editor for further information.

Dr Roger Eltringham *Chairman - Publications Committee WFSA*

Dr Iain Wilson *Editor - Update in Anaesthesia*

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Contacts:

Russian Edition:- Andrei Varvinski, Dept. of Anaesthesia, University of Wales School of Medicine, Heath Park, Cardiff, U.K.

Email: avarvinski@hotmail.com

Spanish Edition:- Oscar Gonzales, Rio Parana 445, Bo Felicidad - Lambare, Paraguay

Email: ojgam@conexion.com.py

French Edition:- Michel Pinaud, Service d'anaesthesia, Hotel Dieu, 44093 Nantes Cedex 1, France

Email: michel.pinaud@chu-nantes.fr

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

PREOPERATIVE FASTING GUIDELINES

J. Roger Maltby, MB, BChir, FRCA, FRCPC, Professor of Anesthesia, University of Calgary, Alberta, Canada

Clinically significant pulmonary aspiration during general anaesthesia is rare in healthy patients having elective surgery. The largest study reports an incidence of 1 in approximately 10,000 patients, with no deaths in more than 200,000.^[1] The majority of serious cases of pulmonary aspiration occur in emergency cases, particularly trauma, obstetrics and abdominal surgery in which delayed gastric emptying may be further prolonged by administration of opioid narcotic analgesics. If, in addition, tracheal intubation is difficult, anaesthesia is allowed to lighten and suxamethonium (succinylcholine) to wear off, repeated attempts at laryngoscopy may precipitate gagging, vomiting and aspiration.^[1]

Fasting guidelines

The purpose of fasting guidelines for healthy patients undergoing elective surgery is to minimize the volume of gastric contents while avoiding unnecessary thirst and dehydration. Dehydration is particularly important in hot countries. Guidelines should be based on clinical studies in surgical patients or, when this evidence is not available, on the physiology of digestion and gastric emptying. Although the earliest books on anaesthesia did not mention fasting, in 1883 the famous surgeon Lister^[2] recommended that there should be no solid matter in the stomach, but that patients should drink clear liquid about 2 hours before surgery. For the next 80 years until the 1960s most textbooks recommended a 6-hour fast for solids and 2-3 hours for clear liquids.

During the 1960s in North America the preoperative order 'nothing by mouth after midnight' was applied to solids as well as liquids. The change was widely accepted although the reasons for it have been lost in the mists of time. Pulmonary aspiration was known to be one of the leading causes of anaesthetic related mortality. Concern about the risk of pulmonary aspiration was fuelled by Roberts and Shirley's 1974 statement^[3] that patients with 0.4ml/kg (25ml in adults) of gastric contents, with pH <2.5 are at high risk of pulmonary aspiration. However, Roberts and Shirley did not establish a relationship between volume in the stomach and volume aspirated into the lungs.^[4,5] They later revealed that that they had drawn their conclusion after instilling 0.4ml/kg acid into the right mainstem bronchus in one experiment in one monkey.

The myth of 25ml in the stomach being a surrogate marker for high risk of aspiration is now discredited.^[6] Clinical studies show that 40-80% of fasting patients fall into that category,^[7] yet the incidence of pulmonary aspiration is 1 in 10,000. Raidoo et al^[8] have demonstrated that 0.8ml/kg in the trachea of monkeys (equivalent to >50ml in adult humans) is required to produce pneumonitis. For this volume to reach the lungs, the volume in the stomach must be greater, even if the lower and upper oesophageal sphincters are incompetent.

Gastric pressure

The human stomach is a very dispensable organ and can accommodate up to 1000ml before intragastric pressure increases.^[9] In cats, whose lower oesophageal sphincter mechanism is similar to that in humans, the minimum volume of gastric fluid required to overcome the sphincter varies from 8ml/kg to >20ml/kg.^[10] In humans, the lower figure is equivalent to approximately 500ml and the higher one 1200ml. The volume of gastric contents after an overnight (> 8 hours) fast averages 20 to 30ml, and varies from 0 to >100ml (Table 1). Therefore, unless the patient has an incompetent sphincter, reflux of gastric contents does not occur with the normal range of fasting gastric volumes. If we know how long the stomach takes to return to the fasting state, we can formulate appropriate fasting guidelines for elective surgery.

Gastric emptying

Modern physiological studies use a dual isotope technique in which solids and liquids are tagged with different radioactive isotopes.^[11] Clear liquids empty exponentially, 90% within 1 hour and virtually all within 2 hours. They do not contain particles >2mm and therefore pass immediately through the pylorus. The pylorus prevents passage of particles >2mm, so digestible solids (bread, lean meat, boiled potatoes) must be broken down to particles <2mm before they can pass into the small bowel. Total emptying of a meal normally takes 3-5 hours. Large particles of indigestible food, especially cellulose-containing vegetables, empty by a different mechanism, after the stomach has emptied liquid and digestible food, that may take 6-12 hours.

Gastric physiology therefore suggested that 'nothing by

Table 1. Preoperative oral fluid: Residual gastric fluid volume (RGFV) and pH

Year	Author	Oral Intake	Drink on day of surgery		Nothing by mouth from midnight	
			Mean Fast	RGFV (ml)	Mean Fast	RGFV (ml)
1983	Miller et al (UK)	toast and tea/coffee	3 $\frac{1}{4}$ h	11 (0-43)	9h	9 (0-42)
1986	Maltby et al (Canada)	water 150ml	2 $\frac{1}{2}$ h	18 (0-56)	16 $\frac{1}{2}$ h	27 (0-80)
1987	Sutherland et al (Canada)	water 150ml	2 $\frac{1}{2}$ h	21 (0-50)	13 $\frac{1}{2}$ h	30 (2-75)
1988	Hutchinson et al (Canada)	coffee/juice 150ml	2 $\frac{1}{2}$ h	24 (0-96)	14 $\frac{1}{2}$ h	23 (0-75)
1988	McGrady et al (UK)	water 100ml	2h	17 (4-52)	12h	35 (0-58)
1989	Agarwal et al (India)	water 150ml	2 $\frac{1}{2}$ h	21 (0-50)	12h	30(0-75)
1989	Scarr et al (Canada)	coffee/juice 150ml	2-3h	25 (0-90)	>8h	26(0-120)
1991	Maltby et al (Canada)	coffee/juice no limit	2-3h	22 (3-70)	>8h	25(0-107)
1991	Ross et al (USA)	water 225ml	$\frac{1}{2}$ h	21 \pm 18	>8h	30 \pm 2
1991	Mahiou et al (France)	Clear liquid 1000ml	2h	38 \pm 18	11h	35 \pm 15
1993	Lam et al (Hong Kong)	water 150ml	2-3h	26 (3-66)	11 $\frac{1}{2}$ h	22 (1-78)
1993	Phillips et al (UK)	clear liquid, no limit	2 $\frac{1}{4}$ h	21 (0-80)	13h	19(0-63)
1993	Søreide et al (Norway)	water 300-450ml	1 $\frac{1}{2}$ h	23 \pm 20	13h	31 \pm 30

Values are mean (range) or mean \pm SD

mouth after midnight' is logical for solid food but that patients could safely drink clear liquids on the day of surgery. Nevertheless, entrenched beliefs, those built on false premises, are difficult to dislodge. Double blind, randomized clinical trials in surgical patients were required.

Clinical Studies

In 1983, Miller et al reported no difference in gastric fluid volume or pH in patients who were 'nothing by mouth' after midnight' and those who had tea and toast 2-4 hours before surgery.^[12] Since then clinical studies with clear liquids in adults (Table 1) and children^[13,14] have confirmed those findings. Fasting guidelines at Foothills Medical Centre in Calgary were changed in 1988. Since then, 'nothing by mouth after midnight' has applied only to solids, and clear liquids are

encouraged until 3 hours before the *scheduled* time of surgery, or 2 hours before the *actual* time of surgery. Follow-up studies in more than 400 patients showed no difference in gastric fluid or pH at induction of anaesthesia between those who drank and those who fasted from midnight, nor did the volume ingested (50-1200ml) influence the residual volume in the stomach. This is not surprising because clear liquids empty within 2 hours. Gastric contents after that time consist of gastric secretions and swallowed saliva, as in patients who fast from midnight.

'Clear liquids' include water, apple juice, carbonated beverages, clear tea and black coffee. Sugar may be added to tea or coffee, and 10ml (two teaspoons) of milk. Milk, or tea or coffee made with milk, is treated as a solid because, with gastric juice, it forms a thick

flocculate that takes up to 5 hours to empty forms the stomach. Although chewing gum stimulates salivation; it does not significantly increase gastric fluid volume or acidity,^[15] but the gum must be removed from the patient's mouth before induction of anesthesia! Apart from Miller et al no investigators have allowed solid food on the day of surgery. When patients do eat solid food, the time of surgery should be decided according to the type of food ingested.

Delayed Gastric Emptying

Disorders of gastric motility, pyloric obstruction, gastroesophageal reflux and diabetic gastroparesis delay gastric emptying. Indigestible solids are the first to be affected, followed by digestible solids and finally liquids. Because the rate of gastric emptying of clear fluids is not affected until these conditions are far advanced, most patients may still be allowed to drink on the morning of surgery. Different investigators have found obese patients to have either a larger^[16] or smaller^[17] residual fasting gastric fluid volume than non-obese patients. These comments only apply to patients scheduled for elective surgery. All emergency cases, especially those involving trauma and women in labour, should always be assumed to have delayed gastric emptying.

Gastric emptying is normal in all three trimesters of pregnancy and beyond 18 hours post-partum, but is delayed in the first 2 hours post-partum.^[18] Labour

causes an unpredictable delay in gastric emptying that is markedly potentiated by opioids.^[19] Nevertheless, there is a move towards less rigid fasting guidelines during labour, especially in women who are not expected to require operative intervention.^[20]

Development of American Society of Anesthesiologists (ASA) fasting guidelines

The ASA formed a Task Force in 1996 to review relevant clinical human research studies published 1966 to 1996. Over 1100 citations were initially identified, of which 232 articles contained relationships between preoperative fasting and pharmacological prophylaxis of pulmonary aspiration. Expert opinion was also obtained from international anaesthesia and gastroenterologist consultants in preparing clinical guidelines for preoperative fasting (Table 2) and pharmacological prophylaxis in healthy patients undergoing elective (elective) surgery. These were approved by the House of Delegates at the 1998 ASA Annual Meeting and were published in the March 1999 issue of *Anesthesiology*.^[21] The Canadian Anesthesiologists' Society has published similar guidelines.^[22]

Implementation of new fasting guidelines

Cooperation of anaesthesia colleagues, surgeons and nurses are essential for implementation. In our hospital, we presented the evidence at a meeting of the anaesthesia department, then to a joint meeting of surgeons and

Table 2. American Society of Anesthesiologists fasting guidelines

Ingested material	Minimum fast ^a
Clear liquids ^b	2 hours
Breast milk	4 hours
Infant formula	6 hours
Non-human milk	6 hours
Light meal ^c	6 hours

a Fasting times apply to all ages.

b Examples: water, fruit juice without pulp, carbonated beverages, clear tea, black coffee.

c Example: dry toast and clear liquid. Fried or fatty foods may prolong gastric emptying time. Both amount and type of food must be considered.

The guidelines recommend no routine use of gastrointestinal stimulants, gastric acid secretion blockers or oral antacids.

anaesthetists, and also to a meeting of head nurses of our surgical wards. The clinical heads of anaesthesia and surgery then sent a joint letter to all consultant and trainee surgeons and anaesthetists, with copies to the head nurses to provide details of the revised guidelines. The nursing staff then used the guidelines to revise the fasting instructions in the hospital's nursing policy manual

Conclusion

The order 'nothing by mouth after midnight' should apply only to solids for patients scheduled for surgery in the morning. An early light breakfast of easily digested toast or similar food with clear liquid is permissible for afternoon cases. Clear liquids should be allowed until 3 hours before the scheduled time of surgery so that a change in the surgical schedule can be made and still allows 2 hours before the actual time of surgery. For patients with true gastroesophageal reflux, whether or not they drink, an H₂-receptor blocker (ranitidine) or proton pump inhibitor (omeprazole) may be advisable to minimize gastric acid secretion.

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ACUTE OXYGEN TREATMENT

Dr. Andrei M. Varvinski, Specialist Registrar in Anaesthesia and ICM, City Hospital N1, 1 Suvorova Str, Arkhangelsk, Russia, e-mail: avarvinski@hotmail.com

Dr. Sara Hunt, Specialist Registrar in Anaesthesia, Department of Anaesthesia, University Hospital of Wales Cardiff CF4 4XW

Table 1 - Key to terms used

PaO ₂	Tension or level of arterial oxygen
Bar	Unit of pressure, approximately 1 atmosphere (760mmHg or 101kPa)
kPa	Kilopascals = 1000 Pascals, a unit of pressure (7.5mmHg = 1 kPa)
Minute ventilation	The volume of gas breathed per minute
Peak Inspiratory Flow Rate	Maximum rate of air flow when breathing in (inspiratory breath)
< >	< = less than ; > = greater than

Introduction

Oxygen has been used in clinical practice for more than 200 years. It is probably the most widely prescribed medication in pre-hospital and hospital environments. If appropriately used it is life-saving and part of first-line treatment in many critical conditions. It is important that oxygen not only reaches the lungs but is delivered to the tissues. Therefore a good cardiac output, circulation and haemoglobin is vital and is why attention to the circulation is an early part of initial resuscitation (*The physiology of oxygen delivery, Update in Anaesthesia 1999; 10:8-14*). As with any drug, oxygen should be used when indicated, in appropriate dosage (concentration), and correctly administered for a planned duration.

OXYGEN MANUFACTURE AND STORAGE

When cooled to very low temperatures gases change to either solids, (carbon dioxide), or liquids (oxygen and nitrogen). Oxygen has to be cooled to below -118°C to change to a liquid. When the gas changes form to a liquid, it occupies a much smaller volume. Therefore when a small volume of liquid oxygen is warmed it will make a very large volume of oxygen gas. Oxygen can be stored as either a gas in cylinders or as a liquid in a special container. In the liquid form, a very large quantity of oxygen can be transported or stored in a low volume, although there are problems in keeping the liquid cold as explained below.

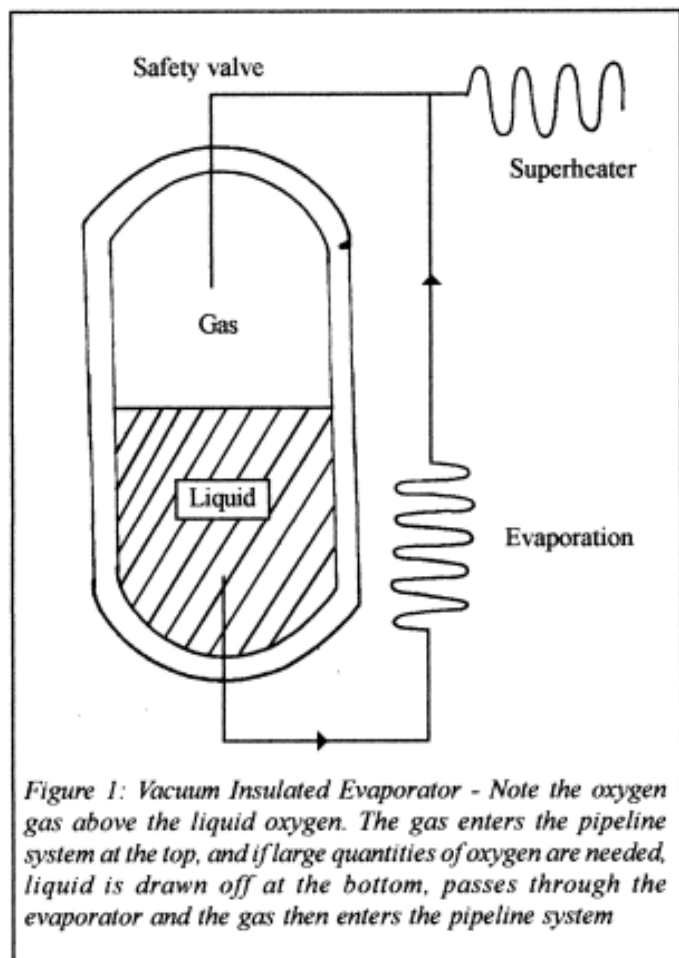
Vacuum Insulated Evaporator (VIE). A VIE is a container designed to store liquid oxygen. It has to be designed to allow the liquid oxygen inside to remain very

cold. It consists of two layers, where the outer carbon steel shell is separated by a vacuum from an inner stainless steel shell, which contains the oxygen (figure 1). The oxygen temperature inside is about -170°C and the container is pressurised to 10.5 atmospheres (10.5 bar). Gaseous oxygen above the liquid is passed through the superheater to raise the temperature to ambient (outside) levels. It then flows into the hospital pipeline system giving a continuous supply of piped oxygen to outlets on the wards and in theatre. Heat is always able to get into the container and provides the energy to evaporate the liquid oxygen, changing it into oxygen gas which is continuously drawn off into the pipeline system. This escape of gas into the pipeline system prevents the pressure inside the container from rising. If the pressure rises too much (above 17 bar), oxygen is allowed to escape via a safety valve into the atmosphere.

In contrast, if the pressure inside the container falls because of heavy demand in the hospital for oxygen, liquid oxygen can be withdrawn, passed through the evaporator and returned to the VIE in the gaseous form to restore the pressure. The amount of oxygen available in the container is estimated by weighing the container with an in-built device.

The VIE system is used in large hospitals which have a pipeline system, and where liquid oxygen can be supplied by road tanker.

Oxygen cylinders. Oxygen can be stored under pressure in cylinders made of molybdenum steel. Cylinders may be



combined to form a bank attached to a manifold. The advantages of combining large cylinders into a bank include a reduction in cost, transportation and constant change of exhausted cylinders. Oxygen cylinders come in several sizes (table 2). In UK oxygen cylinders are black with white shoulders. The pressure inside at 15°C is 137 bar.

Table 2 - Oxygen cylinder sizes

Size	C	D	E	F	G	J
Height (in)	14	18	31	34	49	57
Capacity (litres)	170	340	680	1360	3400	6800

Oxygen concentrators An oxygen concentrator is a device which extracts oxygen from atmospheric air using canisters of zeolite. Nitrogen is filtered out and oxygen produced. The function and successful economics were described in detail. (*Oxygen concentrators for district hospitals in Update in Anaesthesia 1999;10: 57-59*). When ether is used, the oxygen concentrator should be positioned 1.5m above the floor.

HYPOXIA

Hypoxaemia is when the oxygen tension in arterial blood is less than 80mmHg (10.6kPa). **Hypoxia** is a deficiency of oxygen at the tissue level. Traditionally, hypoxia has been divided into 4 types.

1. **Hypoxic hypoxia** in which oxygen tension of arterial blood is reduced
2. **Anaemic hypoxia** in which the arterial oxygen tension is normal but the amount of haemoglobin(Hb) available to carry oxygen is reduced.
3. **Stagnant or ischaemic hypoxia** in which blood flow to the tissues is so low that oxygen is not delivered to the tissues despite normal arterial oxygen tension and Hb concentration.
4. **Histotoxic hypoxia** in which oxygen is delivered to the tissues but a toxic agent prevents the cells using the oxygen.

Recognition of hypoxia. Recognition of tissue hypoxia is not always easy as there are a number of different signs and symptoms. Clinical signs and symptoms include:

- Altered mental status (agitation, confusion, drowsiness, coma)
- Cyanosis
- Dyspnoea, tachypnoea or hypoventilation
- Arrhythmias
- Peripheral vasoconstriction often with sweaty extremities
- Systemic hypotension or hypertension depending on the underlying diagnosis
- Nausea, vomiting and other gastrointestinal disturbance

Cyanosis means blueness of the tissues and is due to an excessive amount of deoxygenated Hb in the peripheral blood vessels. Cyanosis appears whenever the arterial blood contains more than 1.5grams of deoxygenated Hb in each 100mls of blood (normal Hb15g/100ml). Cyanosis can often be detected in a patient with a normal haemoglobin level when the oxygen saturation is less than 90%. When the oxygen saturation falls in anaemic patients, cyanosis is often absent.

As the clinical signs are non-specific, the best method of assessing oxygenation is to measure peripheral arterial oxygen saturation ($SaO_2 < 95\%$ is abnormal) and oxygen partial pressure in the arterial blood

($\text{PaO}_2 < 80 \text{ mmHg}$ (10.6 kPa)). Pulse oximeters and blood gas analysis have become more widespread throughout the world. Hypoxia at tissue level may still exist even when SaO_2 and PaO_2 are within normal limits, if there is a low cardiac output, anaemia or failure of tissues to use oxygen (e.g. cyanide poisoning). In this situation the blood lactate concentration rises due to anaerobic metabolism. Lactate can be measured in some laboratories.

OXYGEN DELIVERY SYSTEMS

Oxygen can be delivered to the patient using different devices. There are two main types of devices; *fixed and variable performance masks*.

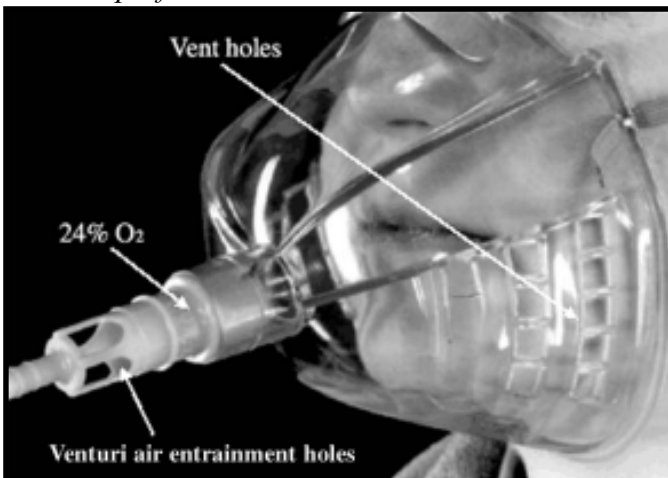


Figure 2: This is a fixed performance mask, with the Venturi entrainment device attached, and shown in detail inset. It indicates the oxygen flow rate and percentage of oxygen delivered (24%). Note the large volume of the mask and the holes which allow the gas flow to flush out expired gas

Teaching point

HAFOE masks use the Bernoulli effect to draw in or entrain a second gas via a side arm. This is the Venturi principle. Gas flowing through a tube is passed through a constriction or narrowing formed in the tube. The gas increases speed to pass through the narrowing, and therefore gains kinetic energy because of the increased velocity. The total energy of the system must remain the same, thus there has to be a fall in potential energy. The potential energy of a gas is the pressure it exerts. Therefore, if there is a fall in potential energy there will be a fall in pressure at that point. A second gas can be sucked in or entrained through a side arm into this area of low pressure (figure 3).

Fixed performance masks ensure that the patient receives a constant inspired oxygen concentration (FiO_2) despite of any changes in minute ventilation. These include:

- Closed or semi-closed anaesthetic breathing systems with a reservoir bag, attached to anaesthetic machine with pressurised gas supply.
- Head boxes for neonates - oxygen is piped into the box at a constant inspired oxygen concentration. Sufficient gas flow is needed to flush CO_2 out.
- HAFOE High Air Flow Oxygen Enrichment Devices e.g Ventimask

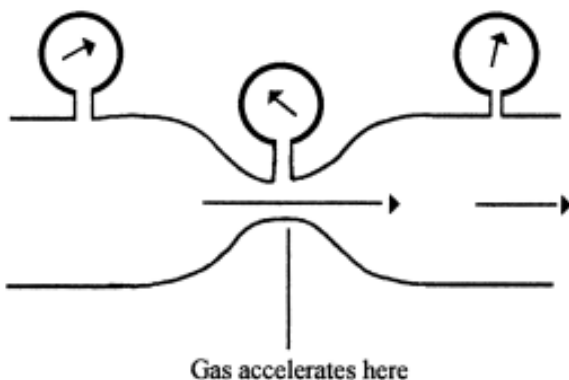


Figure 3a: The Bernoulli effect, with a flow of gas passing through a narrow tube. Note how the pressure falls at the narrow point.

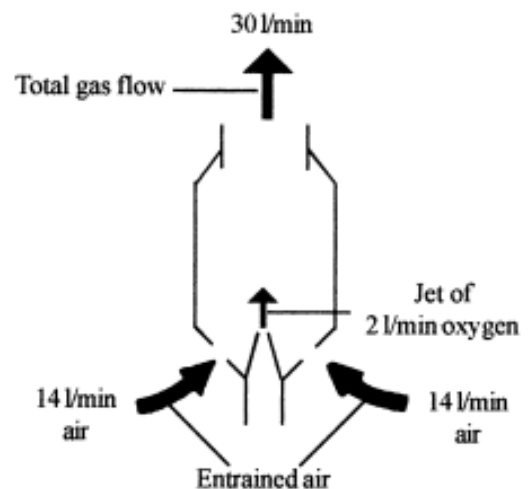


Figure 3b: Venturi valve - a low flow of oxygen, 2 l/min passing through a narrow tube draws in 28 l/min air, the hole size ensures the correct mixture of oxygen and air.

HAFOE masks (figure 2) are colour coded and each mask states the flow of oxygen in litres per minute required to achieve a specific inspired oxygen concentration. There are holes which allow entrainment of room air by the Venturi principle. Relatively high flows of oxygen are needed: e.g. 8 l/min to ensure an inspired oxygen concentration of 40% and 15 l/min to ensure an inspired oxygen concentration of 60%. The flows of 2, 4 and 6 l/min will provide 24, 28 and 31% oxygen respectively. The patient breathes a fixed concentration of oxygen enriched air because the gas flow is greater than the peak inspiratory flow rate of the patient. Thus there is minimal dilution from atmospheric air. The high gas flow flushes expired gas from the mask preventing rebreathing.

Variable performance masks/devices. The second type of oxygen delivery system includes those which deliver a variable concentration of oxygen. The oxygen concentration delivered depends on patient minute ventilation, peak inspiratory flow rate and oxygen flow rate. For example, when a patient is breathing with a **low** minute ventilation and is given a **high** oxygen

flow, oxygen concentration will be relatively high. If the patient breathes more without an increase in oxygen flow, there will be a fall in inspired oxygen concentration. Using these masks the oxygen concentration is not fixed or accurate, but in most situations a flow rate of 2l/min provides 25-30% O₂ and 4 l/min provides 30-40% O₂. Examples of these devices include:

- **Nasal cannula.** These do not increase dead space. Inspiratory oxygen concentration depends on the flow rate. No rebreathing occurs.
- **Nasal catheters,** 8FG, can be inserted into the nose as far as the pharynx, so that they can just be observed behind the soft palate. A gas flow of 150ml/kg/min gives an inspired oxygen concentration of 50% in children less than 2 years. No rebreathing occurs.

The same concept can be used in adults and the cannula may be fashioned from any soft tipped fine catheter (a fine nasogastric tube or urinary catheter may be used in emergencies).

When using nasal catheters they must be taped securely in place so that they cannot migrate down into the oesophagus.

- **Plastic oxygen masks** (figure 4) have a small dead space. The effect of the dead space depends on the patient's minute ventilation and oxygen flow. There is usually a small amount of rebreathing.

OXYGEN THERAPY

The American College of Chest Physicians and National Heart, Lung and Blood Institute published recommendations for instituting oxygen therapy. These include:

- Cardiac and respiratory arrest (give 100% oxygen)
- Hypoxaemia (PaO₂ < 59mmHg (7.8 kPa), SaO₂ <90%)
- Systemic hypotension (systolic blood pressure <100mmHg)
- Low cardiac output and metabolic acidosis (bicarbonate <18mmol/l)
- Respiratory distress (respiratory rate > 24/min)
- In anaesthesia, "added oxygen" should be used during and after anaesthesia as previously described, (*The physiology of oxygen delivery, Update in Anaesthesia 1999;10: 8-14*).

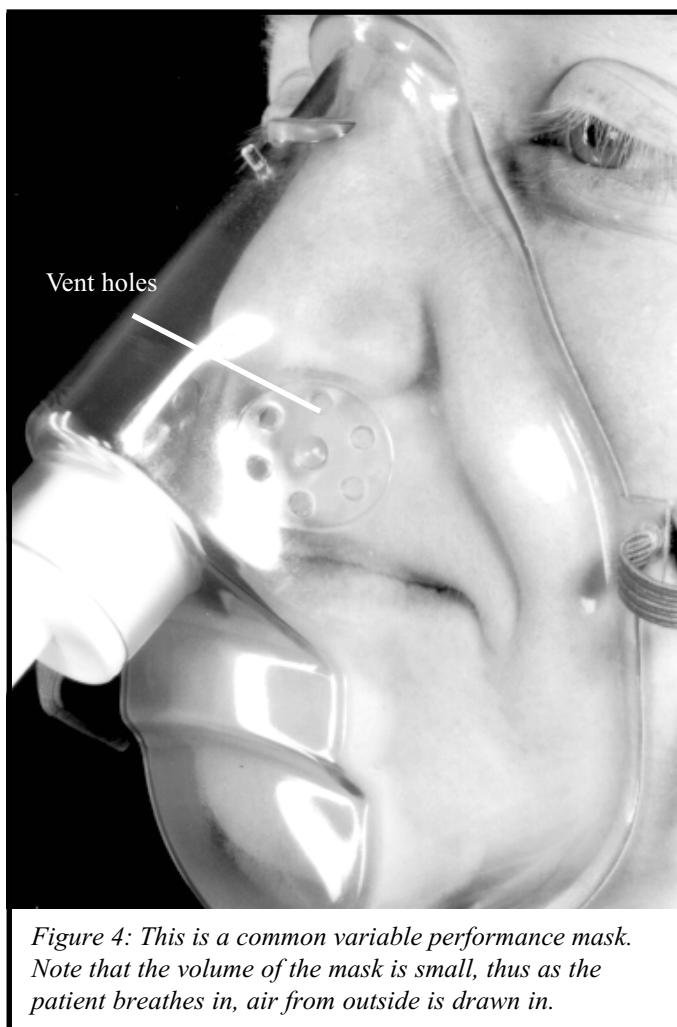


Figure 4: This is a common variable performance mask. Note that the volume of the mask is small, thus as the patient breathes in, air from outside is drawn in.

Table 1

Patients who do not require controlled oxygen therapy	Asthma Pneumonia Bronchiolitis Respiratory distress Cardiac or respiratory arrest Pulmonary embolus Shock - septic hypovolaemic cardiac failure myocardial infarction Carbon monoxide poisoning
Patients who require controlled oxygen therapy	Chronic obstructive pulmonary disease with hypoxic drive Premature infants

Teaching Point

Patients who can be harmed by high concentrations of oxygen are mentioned because they are encountered only occasionally. MOST patients benefit from uncontrolled oxygen and it should be given freely to those with cardiac or respiratory arrest, those with respiratory distress, asthma or hypotension.

28%, which is progressively increased, aiming to achieve an arterial oxygen tension, ideally, above 50mmHg (6.6kPa) or an SpO₂ of 85-90%. These patients are rarely encountered in anaesthetic practice, but the possibility of this situation should be considered in people with severe COPD. Unfortunately the risk of hypercapnia in patients with severe COPD is often overestimated, resulting in inadequate oxygen therapy and death from hypoxia.

Prescribing oxygen - controlled or uncontrolled?

As with any drug, oxygen should be prescribed. It may be prescribed as *controlled* oxygen therapy where the concentration is prescribed using a HAFOE device. However oxygen is more commonly prescribed at a recommended flow rate using a variable oxygen administration device - this is known as *uncontrolled* oxygen therapy.

A small group of patients with chronic obstructive pulmonary disease (COPD) have raised CO₂ levels and depend on hypoxia to stimulate respiration (hypoxic respiratory drive). This is in contrast to the normal patient where the blood level of CO₂ drives respiration. They have a long history of chest disease, are cyanosed, sleepy, have signs of cor pulmonale but are not breathless. In these patients high dose oxygen can reduce respiration and cause respiratory depression. They will develop increased CO₂ retention, respiratory acidosis and subsequently will require mechanical ventilation. These patients should receive carefully controlled oxygen therapy, starting at 24-

Monitoring of oxygen therapy

Clinical monitoring includes observation of conscious level, respiratory and heart rates, blood pressure, peripheral circulation (capillary refill, normally 1-2 sec.) and cyanosis. If available, additional monitoring can be provided by blood gas analysis and pulse oximetry. Check arterial oxygen blood tension and saturation before administering oxygen whenever possible. After starting oxygen, blood gases or oximetry should be repeated adjusting inspired oxygen concentration to achieve PaO₂ more than 59mmHg (7.8kPa) or SaO₂ more than 90%. Oximetry provides continuous monitoring of oxygen saturation and is especially helpful if blood gas analysis is difficult or unavailable.

However in the small group of patients with chronic lung disease who depend on their hypoxic drive, respiratory depression can be detected by seeing the patient become more drowsy and a rise in arterial CO₂ level. Note that oxygen saturation will not decrease until a late stage.

Risks of oxygen treatment

- **Fire** - oxygen supports combustion of other fuels. **Do not** smoke when on oxygen!
- **Absorption atelectasis.** Prolonged administration of high concentrations of oxygen can result in atelectasis particularly at lung bases. It is most common following chest or upper abdominal surgery and in those patients with poor lung function and sputum retention.
- **Retrolental fibroplasia.** High arterial oxygen tensions are a major factor in causing retrolental fibroplasias in neonates, which may result in blindness. The condition is caused by blood vessels growing into the vitreous, which is followed later by fibrosis. The low birth weight very premature infant is at risk up to 44 weeks postconceptual age. The level of PaO₂ required to cause retinal damage is not known, but an umbilical PaO₂ of 60-90mmHg (8-12kPa) is safe. Some doctors believe that the normal term infant is also at risk and that arterial saturation must not exceed 95%. However if the baby is hypoxic or requires resuscitation, oxygen **must** be given. Oxygen in normal concentrations is also safe for short periods during anaesthesia.
- **Patients on chemotherapy.** It is recognized that patients who have received bleomycin are at risk of developing pulmonary fibrosis if they are given excessive concentrations of oxygen during and after anaesthesia. In these patients controlled oxygen therapy should be prescribed to maintain SaO₂ 90-95%.

Teaching Point

The oximeter is a very useful instrument, but the clinician must not forget its limitations. It only measures oxygen saturation and therefore when interpreting the readings the shape and importance of the oxygen saturation curve must be remembered. The curve is flatter when the oxygen saturation is more than 93%. Therefore relatively large increases in oxygen tension (PaO₂) will cause small increases in saturation. In contrast, when the saturation falls below 90%, the oxygen tension will fall rapidly with falls in oxygen saturation.

Conclusion

Oxygen is widely used across all medical specialities. In many acute situations, it is the first drug to be given and is life saving. It should always be considered along with management of the airway, delivery system, the importance of the circulation, constant monitoring and reassessment of the treatment. Dangers of oxygen therapy should be always remembered but should **never** prevent oxygen from being given.

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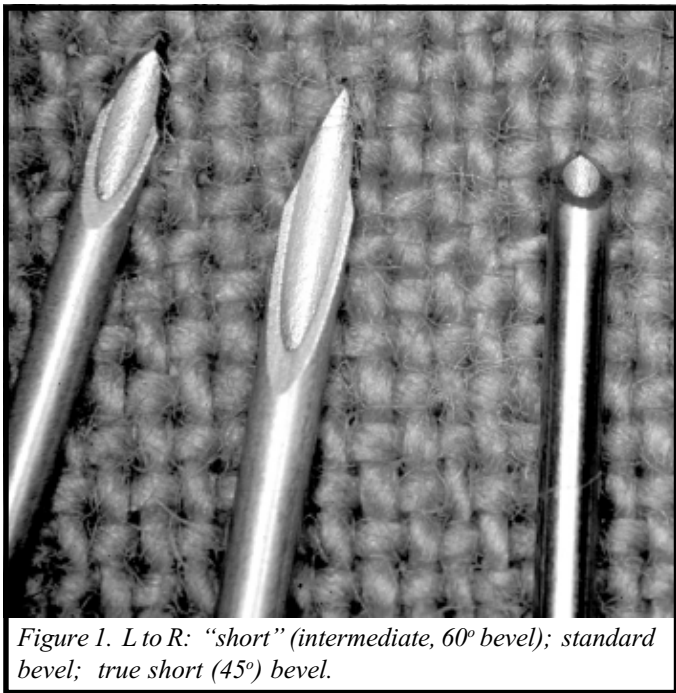
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REGIONAL BLOCKS AT THE WRIST

Hugh Spencer, Waikato Hospital, Hamilton, New Zealand

Regional blocks at the wrist are easy to perform and are useful for a number of situations:

- supplementing arm blocks and Bier's blocks particularly for postoperative pain relief
- minor surgery or dressings on the hand and fingers
- repairing hand trauma
- pain relief - such as burns to hand or fingers



General principles of regional anaesthesia applied to wrist blocks

- **Equipment.** A small "short bevel" is best. (figure 1) Monoject make an excellent 1/8 inch 27 gauge "intermediate" bevel needle which is good for wrist and other blocks, particularly in children and around the face. Long bevel ("standard") needles make blocking difficult as it is harder to feel the tissues because the long bevel may "straddle" two tissue planes. The true short bevel needles (45° are not suitable for this kind of block (and are usually too large). The 60° bevel (like those found on the needles of most intravenous cannula sets) are best.
- **Tissue planes and anatomy.** All regional blocks depend on a knowledge of the tissue planes in which nerves travel. As long as the local

anaesthetic is in the right plane reasonably near the nerve(s) it will spread to block the nerve.

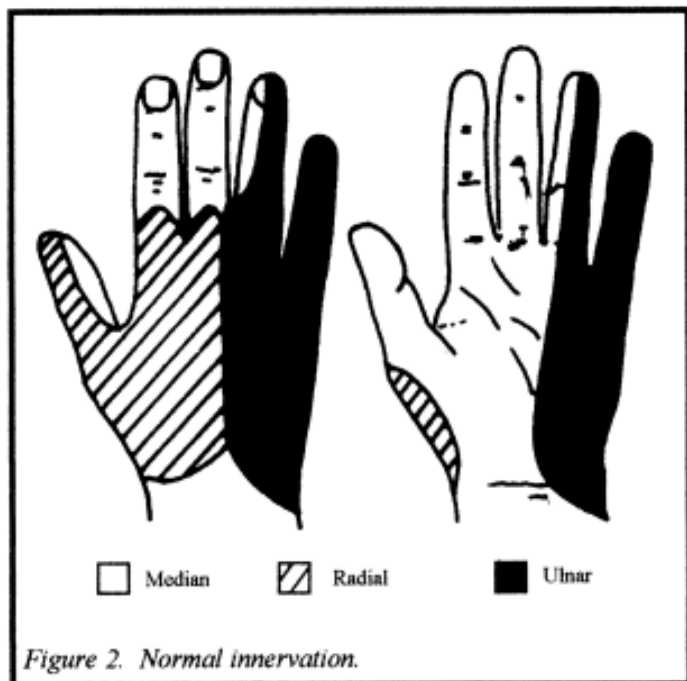
- **Raising the rate of success.** Thinking three dimensionally is vital, even in these simple blocks. "Feeling" the texture of the tissues with the tip of the needle using a very light finger-tip grip on the syringe and moving the tip slightly from side to side will identify the tissue - skin, subcutaneous fat, fascial layers, tendon sheath etc. They all have a distinctive feel. A needle tip in flexor carpi ulnaris does not help an ulnar nerve block.
- **Drugs and dosage.** There is practically no chance of toxic doses of local anaesthetic except for accidental intravascular injection in very small children. It is therefore of little use to interrupt the smooth flow of your technique by drawing back to look for blood. Use the smallest syringe possible for the dose required. The doses mentioned are for adults. Lignocaine 1-2% with adrenaline (1:100,000 or weaker) will give two hours' anaesthesia or more, and 0.5% bupivacaine plain will at least double that.
- **Technique - how not to hurt.** Many children and some adults respond poorly to regional anaesthesia. Demonstrating that the blocks themselves are not very painful is a good starting point for successful management. Advancing needles slowly with a little local is helpful, (and slow injection of the local). To perfect your technique, try blocking your own nerves - if there is someone you do not want to hurt, it's you!

BLOCKS AT THE WRIST

Note that variation of innervation may occur. Normal innervation is shown in figure 2.

The main nerves are radial, median and ulnar. But other descending cutaneous branches may need to be blocked with a subcutaneous "ring" (using the method described in blocking the radial nerve). Wrist blocks have the following characteristics:

- in most patients (except the very large) they can all be done with a very small needle.



- intraneural injection (direct injection into the nerve) is possible and must be avoided. Ask the patient to report sudden onset of pain or paraesthesia during the block immediately. If this is felt, stop, inject a very small quantity of local anaesthetic then continue if no further pain or paraesthesia occurs. Alternatively withdraw slightly, and try again.
- they are easy and reliable.
- in the vast majority of patients, the whole hand can be anaesthetised with three nerves blocks.

Radial nerve block at the wrist

Anatomy. The nerve divides into two major branches about two finger breadths proximal to the distal wrist crease (or anatomical snuff box)

- it is usually found just beside the cephalic vein (often anteriorly) and can be rolled under the finger in thin people
- it lies on the tough superficial fascia

Performing the block. Using the 27G needle (if available) three finger breadths from the distal wrist crease (or anatomical snuff box), carefully avoiding veins, gently feel for the tough fascia with the needle tip. Then, using 2-5ml of local anaesthetic use either of the following techniques to block the nerve:

- **method 1** - carefully holding the needle steady, straddle the needle with index and middle fingers (figure 3) and press them firmly against the

radius. Slowly inject the anaesthetic. It has no choice but to spread across the path of the nerve - and the veins are not punctured.

- **method 2** - deposit the dose in one “bleb” then press it with your thumb across the path of the nerve(s) first one way, then the other.



Median nerve block at the wrist

Anatomy. At the distal wrist crease it enters the carpal tunnel where it should not be blocked (neuritis may result). Usually the nerve is best found just under the tendon of palmaris longus (PL) - figure 4a. If this tendon is absent, it lies deep to the fascia just medial to flexor carpi radialis (FCR) - figure 4b. A superficial branch lies just in front of PL and supplies the proximal part of the front of the hand.

Performing the block. (figure 4) Using the 27G needle three finger breadths from the distal wrist crease, feel down from the groove between PL and FCR at an angle to take the tip of the needle directly under the PL tendon. Advance slowly, feeling to avoid tendon sheath and watching out for paraesthesia. Inject 3-5ml of local anaesthetic. For the superficial branch, on withdrawing the needle, place 2 ml subcutaneously immediately superficial to PL. Spread this across the line of the superficial branch with your thumb.

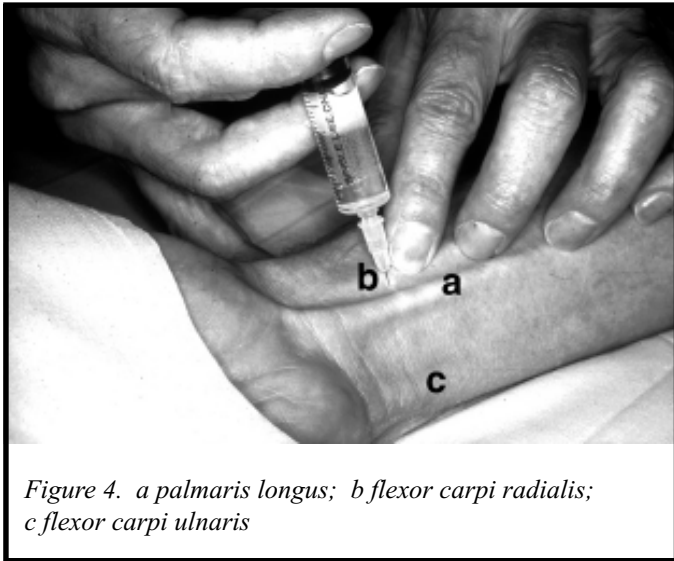


Figure 4. a palmaris longus; b flexor carpi radialis; c flexor carpi ulnaris

Ulnar nerve block at the wrist

Anatomy. By the time the ulnar nerve has reached the distal wrist crease it has divided into anterior and posterior branches. It is a mixed sensory and motor (small muscles of the hand) nerve and is found deep to the tendon of flexor carpi ulnaris (FCU) - figure 4c

Performing the block. Using the 27G needle three finger breadths from the distal wrist crease (to block the nerve before it branches) from either the anterior or the posterior aspect of FCU, advance slowly, feeling to avoid the FCU sheath, aiming to place the tip of the needle directly deep to the tendon. Look out for paraesthesia. Inject 3-5ml of local anaesthetic.

TOPICAL ANAESTHESIA FOR EYE SURGERY

James AL Pittman former Anaesthetic Fellow Project Orbis; Garry N Shuttleworth Ophthalmologist, Bristol, UK.

Local anaesthesia is currently performed for many ophthalmic procedures as it is associated with reduced morbidity and mortality when compared with general anaesthesia. Additional benefits include early patient mobilisation, improved patient satisfaction and reduced hospital stay. A variety of different methods of administration are described which may be broadly divided into 'injections' or 'topical applications'. 'Injection' techniques all involve needle perforation of the peri-orbital skin or conjunctiva and injection of local anaesthetic into the peri-orbital or orbital tissues. Orbital injections are occasionally associated with serious sight or even life-threatening complications. In contrast 'topical' anaesthesia, where local anaesthetic eye drops are applied to the surface of the eye, is non-invasive and has virtually no complications. It is becoming increasingly popular for phacoemulsification cataract surgery although many other procedures may also be performed topically (see Table 1).

Although topical anaesthesia is an extremely simple it may add to the complexities of surgery as operating conditions may be more challenging. Alterations in the practice of both ophthalmic anaesthetist and surgeon may be required. In addition, topical anaesthesia demands understanding and increased co-operation from the patient. This article aims to raise awareness and elaborate upon these changes.

Pre-assessment

Careful patient selection is essential if topical anaesthesia is to be used safely and effectively. Patients need to be co-operative, not unduly anxious, and scheduled for straightforward surgery. During the operation the patient must lie still and be comfortable in the supine position. They must also be able to co-operate and carry out instructions. The lack of akinesia (eye muscle paralysis) may be used by the surgeon as the patient can be asked to consciously fix or alter their gaze during the operation. In addition, because visual function is maintained the patient may be more aware of the operative procedures. Some patients find this stressful and often request sedation.

Short acting intravenous drugs such as midazolam and alfentanil are popular choices although pre-medication with oral benzodiazepines maybe just as effective. Sedation should only be given to help the patient to relax, and not to treat pain during surgery. Patients should be easily rousable and be able to respond when spoken to.

Unfortunately the administration of sedation may generate several problems such as confusion, disorientation and reduced co-operation which result in difficulties for the surgeon. Respiratory depression and a compromised airway can also occur. In the elderly where the sedative effects of these drugs are particularly unpredictable, airway obstruction and respiratory arrests have been reported.

Table 1.

Procedures that can be performed under topical anaesthesia in suitable patient.

- Application of surgical scrubs Povidine 2.5%-10%
- Conjunctiva: Excision of superficial lesions such as cysts or naevi.
- Cornea:
 - Removal of foreign bodies.
 - Removal of sutures.
 - Debridement of the corneal epithelium after recurrent erosions / herpetic keratitis.
 - Corneal scrapings / biopsy with infective keratitis.
 - Dissolution of Ca²⁺ salts in band keratopathy using EDTA
 - Removal of pterygia +/- conjunctival autografts.
 - Refractive surgery.
- Intra-ocular: Cataract surgery. Extra-capsular and Phacoemulsification
- Lid surgery with Eutectic Mixture of Local Anaesthetic (EMLA)

Intravenous access, supplementary oxygen and the presence of suitably trained personnel, usually an anaesthetist, are essential. Appropriate levels of monitoring must be available. As the effects of sedation persist postoperatively, hospital discharge policies need to be flexible. A full explanation of the technique and good communication with the patient often reduces the level of anxiety and keeps the administration of sedation to a minimum.

Perioperative care

Once a patient has been selected they are counselled and consented for their surgery under topical anaesthesia. There is no general consensus on which topical local anaesthetic eye drop provides the best analgesia. Tetracaine, amethocaine, proparacaine, lignocaine and bupivacaine have all been used successfully in a variety of different concentrations. Availability may determine the clinician's choice but it is essential that the preparation is *preservative free*. Manipulation of the preparation pH may alter the duration of action but is of little clinical benefit. Topical NSAIDs and pupil dilating agents are often co-administered.

Application of local anaesthetic eye drops to the cornea and conjunctiva should start in the patient holding area approximately 20-30 minutes before surgery. Different regimes are described but in general two or three drops are applied every five minutes. Sufficient absorption should occur over this period to render the surface of the eye anaesthetised. As the cornea is avascular, once absorbed the local anaesthetic remains for approximately half an hour.

Additional eye drops can be given at any stage during the operation if discomfort is experienced. The use of the Honan balloon to reduce intraocular pressure is not necessary with topical anaesthesia.

For surgery the patient should be positioned in a comfortable supine position. A pillow under the patient's knees will reduce their lumbar lordosis and help alleviate low back discomfort. Attempts should be made to keep the surgical drapes off the patient's nose and mouth during the procedure. Various devices exist that deliver oxygen to the patient during the operation and it is important that the patient is kept at a comfortable temperature (*see Update No. 11*).

Topical anaesthetic application alone produces detectable levels of local anaesthetic agent in the anterior chamber and provides good eye analgesia. However, certain manoeuvres such as iris manipulation, globe expansion and insertion of the intraocular lenses can be uncomfortable. To improve analgesia local anaesthetic can be injected intraoperatively into the anterior chamber of the eye. This 'intracameral' injection produces superior analgesia improving comfort and co-operation. Provided preservative-free solutions are used there are no undesirable effects. 0.5mls of 1% lignocaine is the most popular solution used. Although corneal toxicity has been reported in animal models, clinical studies have failed to demonstrate increased endothelial cell damage.

Patient satisfaction with intraoperative analgesia after topical anaesthesia appears comparable to that of 'injection' techniques. However, topical anaesthesia may

produce higher [but statistically insignificant] 'pain' scores than injection techniques. Fortunately any differences are small and relate to increased discomfort rather than pain. Any discomfort is better tolerated if the patient is fully informed pre-operatively. Intracameral injections reduce discomfort scores and improve patient satisfaction. The successful use of acupuncture to supplement topical anaesthesia in cataract surgery has also been described but is unlikely to become standard practice.

The demands on the surgeon and patient limit the use of topical anaesthesia to relatively short procedures. Occasionally a facial nerve block may be requested to reduce eyelid movements. A good surgeon-patient relationship facilitates the procedure. Communication with the patient whilst operating is a surgical skill that has to be acquired. Typically cataract extraction is performed via a temporal corneal incision with the surgeon sitting to the side of the patient. The surgeon should lower the brightness of the microscope light source to reduce photophobia and

limit patient distress. Surgically related complication rates under topical anaesthesia are similar to conventional orbital blocks. However, without akinesia an inexperienced surgeon may experience some difficulty with capsulorhexis and phacoemulsification.

In the event that the patient becomes distressed or the procedure complicated or lengthened a sub-Tenon's block can be administered by the surgeon to provide a retrobulbar analgesia with akinesia.

Post-operative care

Topical anaesthesia does not cause unwanted post-operative ptosis or diplopia nor does it affect the secretion of tears. As the protective mechanisms of the eye are preserved it is not essential that the eye be patched closed and the rapid return of visual function facilitates an early discharge from hospital. However, patients should be cautioned that vision remains sub-optimal for at least four hours post surgery and they should take additional care.

SELF ASSESSMENT

Michael Richards, Cheltenham Hospital, UK.

Multiple Choice

1. Cardiovascular physiology:

- a) The first heart sound indicates the start of isometric contraction.
- b) In heart failure increasing heart rate will improve myocardial oxygenation.
- c) Cardiac output (CO) = Heart rate (HR) x Systemic Vascular Resistance (SVR).
- d) Diabetes mellitus (DM) may give rise to an abnormal Valsalva response.
- e) Pulmonary artery (PA) catheter measurements are reliable in mitral stenosis.

2. Breathing circuits:

- a) The Mapleson A system is efficient during controlled ventilation.
- b) During spontaneous ventilation, the Mapleson A system requires a fresh gas flow (FGF) of 150ml/kg/min.
- c) During controlled ventilation a Bain circuit requires a FGF of 70-100ml/kg/min.
- d) During spontaneous ventilation a Lack circuit will conserve dead space gas.

- e) The Jackson-Rees modification of the Ayres T piece has a closed bag at the end of the expiratory limb.

3. Circle systems:

- a) Fresh soda lime contains mainly calcium carbonate.
- b) Fresh soda lime contains no water.
- c) Plenum vaporisers cannot be used in the circle due high internal resistance.
- d) A circle system contains 2 one way valves.
- e) Circle systems are economical because low flows can be used from the start of a procedure.

4. Trauma:

- a) All trauma patients should have their airway assessed and secured before having a long bone fracture reduced.
- b) A patient who opens their eyes, withdraws their arm and groans to pain has a Glasgow coma score (GCS) of 11.
- c) A patient with suspected extradural

haematoma and a positive diagnostic peritoneal lavage (DPL) should have an urgent neurosurgical opinion before any further intervention.

- d) All trauma patients who are intubated should have a nasogastric tube passed.
- e) Management of a tension pneumothorax should include chest X ray (CXR) before needle decompression.

5. *Ophthalmic anaesthesia:*

- a) The oculo-cardiac reflex is mediated by the sympathetic nerve supply.
- b) Suxamethonium is absolutely contraindicated in penetrating eye injury.
- c) An intra-ocular pressure (IOP) of 25mmHg is normal.
- d) Patients with myopia have greater risk of orbit puncture with peribulbar blocks.
- e) Ketamine is an appropriate induction agent in a penetrating eye injury.

6. *Renal physiology:*

- a) Renal blood supply is normally 10% of cardiac output.
- b) The juxtaglomerular complex produces angiotensin.
- c) Aldosterone promotes potassium (K⁺) excretion in the proximal tubule.
- d) Atrial natriuretic peptide (ANP) blocks the effect of aldosterone.
- e) Erythropoietin production is increased in hypoxia.

7. *Paediatric physiology:*

- a) Infants have a lower functional residual capacity (FRC) than adults.
- b) Stroke volume is relatively fixed.
- c) An appropriate maintenance fluid requirement for a 26kg child would be 46mls/hr.
- d) Infants have a lower closing volume than adults.
- e) Alveolar minute ventilation (MV) is approximately 60ml/kg/min.

8. *Paediatric anaesthesia:*

- a) In spontaneous respiration with an Ayres T piece, a FGF of 2-3 x minute volume is required.

- b) An appropriate resuscitation dose of fluid is 10mls/kg.
- c) Thiopentone can be given by intraosseous injection.
- d) Pyloric stenosis is a surgical emergency and should be operated on as soon as is possible.
- e) The maximum dose of bupivacaine is 2mg/kg per 4 hours.

9. *Pre-operative assessment:*

- a) A patient with 2:1 heart block should receive an atropine premed.
- b) A morphine infusion is the best form of post-op analgesia in a patient for a gastrectomy with TB bronchiectasis.
- c) Patients with oesophageal reflux should receive a rapid sequence induction (RSI).
- d) Patients having a thyroidectomy should have a CXR.
- e) Patients having non urgent surgery should be postponed by 6 weeks following an myocardial infarct (MI).

10. *Causes of electro-mechanical dissociation (EMD) include:*

- a) Tension pneumothorax
- b) Cardiac tamponade
- c) Pulmonary emboli
- d) Cardiac ischaemia
- e) Hyperthermia

11. *The following are correct doses with respect to paediatric resuscitation:*

- a) First dose adrenaline: 0.1ml/kg of 1:10,000
- b) Second dose adrenaline: 1.0ml/kg of 1:100,000
- c) Atropine: 40mcg/kg
- d) Initial defibrillation: 2 joules/kg
- e) Bicarbonate: 1ml/kg of 8.4% solution

12. *The following inotropes are correctly matched with their receptors:*

- a) Noradrenaline: $\alpha 1, \beta$
- b) Isoprenaline: $\alpha 1, \alpha 2$
- c) 1-2mcg/kg/min dopamine: β
- d) Salbutamol: $\beta 2$
- e) Adrenaline: $\alpha 2$

13. Ketamine:

- a) Acts at the NMDA receptor.
- b) Is related to phencyclidine.
- c) Acts in one arm brain circulation time.
- d) Is a trigger for malignant hyperpyrexia (MH).
- e) Increases post operative nausea and vomiting (PONV).

14. The following will give rise to an increase in body sodium (Na⁺):

- a) Angiotensin I
- b) Captopril
- c) Anti diuretic hormone (ADH)
- d) ANP
- e) Fludrocortisone

15. Concerning the neuromuscular junction (NMJ):

- a) Na⁺/K⁺ ATPase consumes 1/3 of the body's metabolic energy.
- b) The acetylcholine (Ach) receptor has 5 subunits.
- c) Ach binds to the β subunit of the Ach receptor.
- d) NMJ function is normal until 75-80% of receptors are blocked.
- e) The resting membrane potential is -70mV.

16. Immediate management of anaphylaxis should include:

- a) Oxygen
- b) Adrenaline
- c) Steroids
- d) Antihistamines
- e) Salbutamol

17. The following are safe to use in patients taking monoamine oxidase inhibitors (MAOI's):

- a) Metaraminol
- b) Pethidine
- c) Ephedrine
- d) Diclofenac
- e) Paracetamol

18. The following local anaesthetics are correctly paired with their maximum doses:

- a) Plain bupivacaine: 2mg/kg
- b) Bupivacaine with adrenaline: 4mg/kg
- c) Plain lignocaine: 6mg/kg
- d) Lignocaine with adrenaline: 7mg/kg
- e) Plain prilocaine: 6mg/kg

19. The following local anaesthetics are amides:

- a) Bupivacaine
- b) Lignocaine
- c) Cocaine
- d) Amethocaine
- e) Chloroprocaine

20. The following muscle relaxants are safe in a patient with a history of scoliosis apnoea:

- a) Atracurium
- b) Mivacurium
- c) Vecuronium
- d) Pancuronium
- e) Gallamine

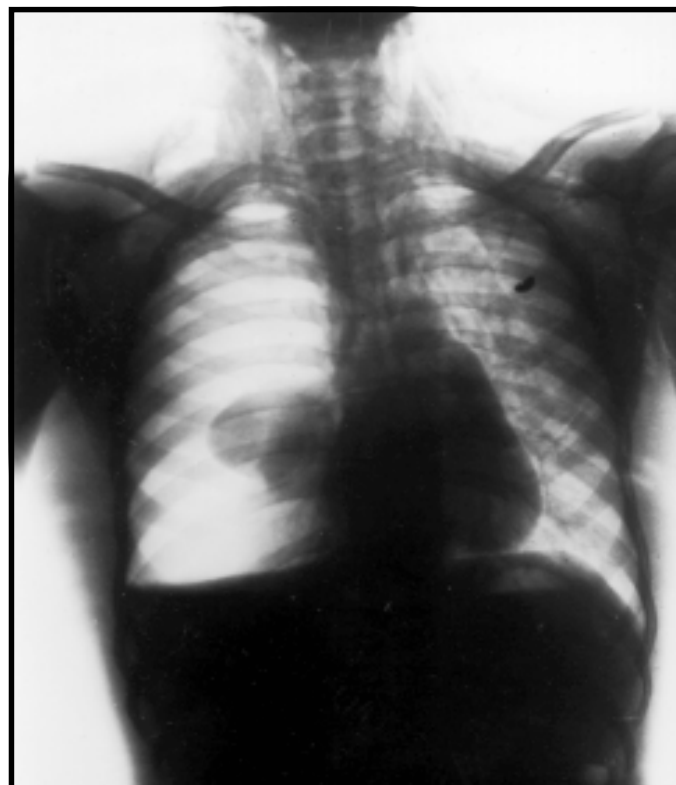
21. Pulse oximetry may be inaccurate in the presence of the following:

- a) Sick cell disease
- b) Methaemoglobinaemia
- c) Nail varnish
- d) Thalasaemia
- e) Carboxyhaemoglobin

Short Answers

1. You give a general anaesthetic for a patient involved in a road traffic accident. The patient has a fractured femur and has bruising to the anterior chest from a seat belt, initial chest X-ray shows no significant injury. Intraoperative blood loss is high so a central line is inserted.

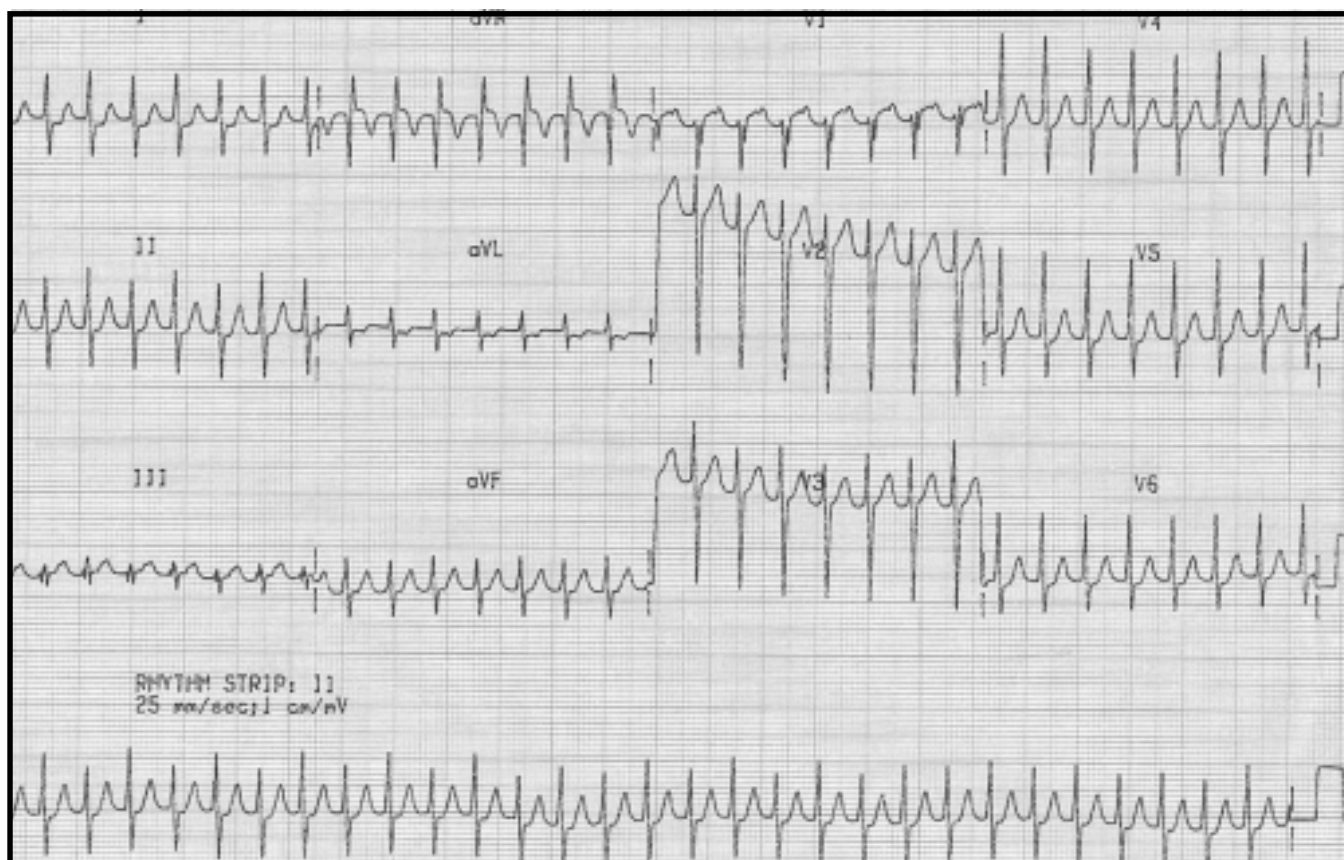
You are asked to see the patient in the recovery room with increasing shortness of breath. When you arrive the patient is dyspnoeic, hypotensive, tachycardic and when you examine the patient you notice decreased air entry unilaterally. A chest X-ray has already been taken:



- What is the diagnosis?
- What is your immediate management?
- What is your subsequent management?
- What are the possible causes of this pathology?

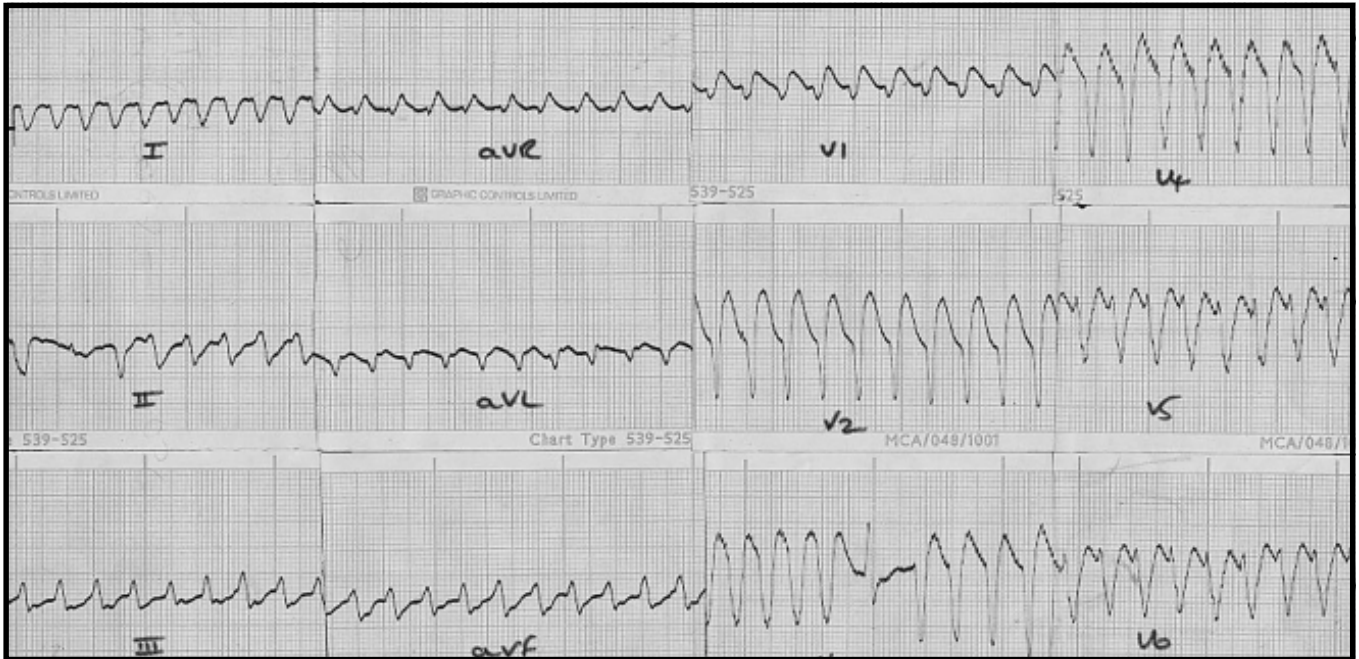
2. You are called to the recovery room to see a young patient who has had a minor ENT procedure. The recovery nurse has noticed a fast heart rate and an ECG has been performed:

- What does the ECG show?
- What physical manoeuvres can be performed to terminate this?
- What pharmacological methods can be used?
- If these fail what is the next step?



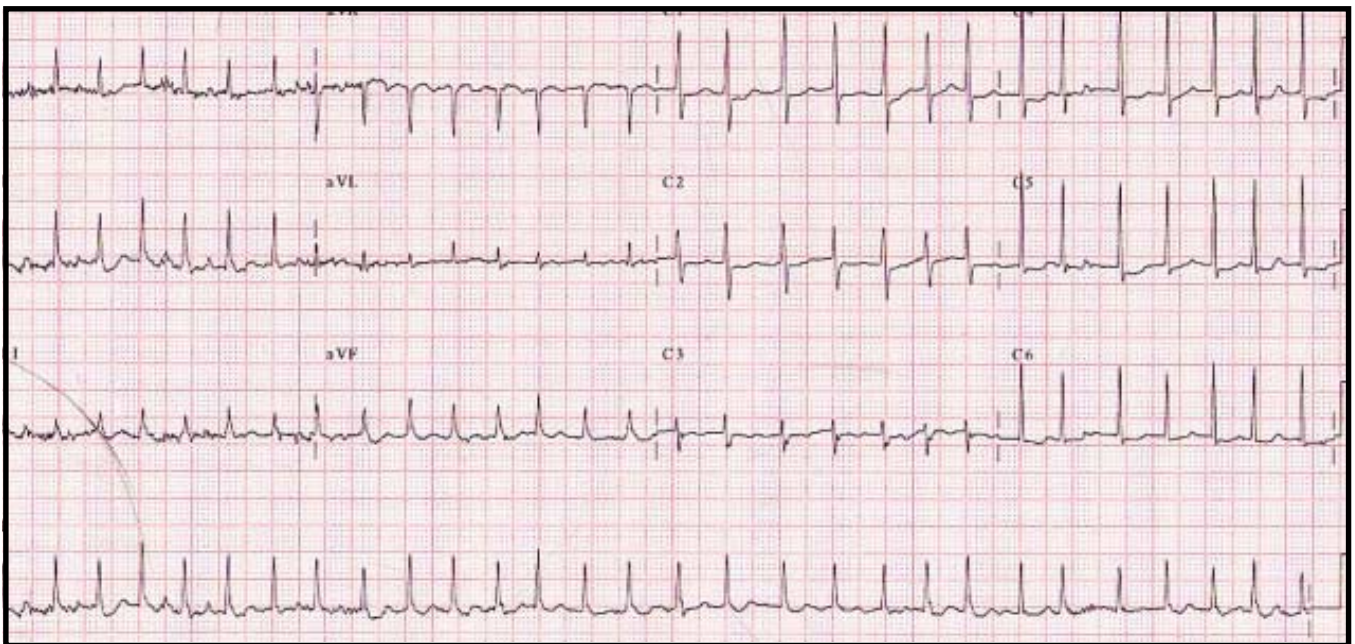
3. You are called to the recovery room to see a patient who has acutely deteriorated following peripheral vascular surgery. The patient is shocked and tachycardic, an ECG is performed:

- a) What is the diagnosis?
- b) What is the management?



4. You induce a patient for a cholecystectomy and following induction the patient develops a tachycardia, a 12 lead ECG is performed:

- a) What is the diagnosis
- b) What are the possible underlying causes
- c) What are the treatment options



SPINAL ANAESTHESIA - A PRACTICAL GUIDE

Dr W F Casey, Consultant Anaesthetist, Gloucestershire, U.K.

Spinal anaesthesia is induced by injecting small amounts of local anaesthetic into the cerebro-spinal fluid (CSF). The injection is usually made in the lumbar spine below the level at which the spinal cord ends (L2). Spinal anaesthesia is easy to perform and has the potential to provide excellent operating conditions for surgery below the umbilicus. If the anaesthetist has an adequate knowledge of the relevant anatomy, physiology and pharmacology; safe and satisfactory anaesthesia can easily be obtained to the mutual satisfaction of the patient, surgeon and anaesthetist.

The Advantages of Spinal Anaesthesia

Cost. Anaesthetic drugs and gases are costly and the latter often difficult to transport. The costs associated with spinal anaesthesia are minimal.

Patient satisfaction. If a spinal anaesthetic and the ensuing surgery are performed skilfully, the majority of patients are very happy with the technique and appreciate the rapid recovery and absence of side effects.

Respiratory disease. Spinal anaesthesia produces few adverse effects on the respiratory system as long as unduly high blocks are avoided.

Patent airway. As control of the airway is not compromised, there is a reduced risk of airway obstruction or the aspiration of gastric contents. This advantage may be lost if too much sedation is given.

Diabetic patients. There is little risk of unrecognised hypoglycaemia in an awake patient. Diabetic patients can usually return to their normal food and insulin regime soon after surgery as they experience less sedation, nausea and vomiting.

Muscle relaxation. Spinal anaesthesia provides excellent muscle relaxation for lower abdominal and lower limb surgery.

Bleeding. Blood loss during operation is less than when the same operation is done under general anaesthesia. This is because of a fall in blood pressure and heart rate and improved venous drainage with a resultant decrease in oozing.

Splanchnic blood flow. Because it increases blood flow to the gut, spinal anaesthesia may reduce the incidence of anastomotic dehiscence.

Visceral tone. The bowel is contracted during spinal anaesthesia and sphincters are relaxed although peristalsis continues. Normal gut function rapidly returns following surgery.

Coagulation. Post-operative deep vein thromboses and pulmonary emboli are less common following spinal anaesthesia.

Disadvantages of Spinal Anaesthesia

Sometimes it can be difficult to find the dural space and occasionally, it may be impossible to obtain CSF and the technique has to be abandoned. Rarely, despite an apparently faultless technique, anaesthesia is not obtained.

Hypotension may occur with higher blocks and the anaesthetist must know how to manage this situation with the necessary resuscitation drugs and equipment immediately to hand. As with general anaesthesia, continuous, close monitoring of the patient is mandatory.

Some patients are not psychologically suited to be awake, even if sedated, during an operation. They should be identified during the preoperative assessment. Likewise, some surgeons find it very stressful to operate on conscious patients.

Even if a long-acting local anaesthetic is used, a spinal is not suitable for surgery lasting longer than approximately 2 hours. Patients find lying on an operating table for long periods uncomfortable. If an operation unexpectedly lasts longer than this, it may be necessary to convert to a general anaesthetic or supplement the anaesthetic with intravenous ketamine or with a propofol infusion if that drug is available.

When an anaesthetist is learning a new technique, it will take longer to perform than when one is more practised. When one is familiar with the technique, spinal anaesthesia can be very swiftly performed.

There is a theoretical risk of introducing infection into the sub-arachnoid space and causing meningitis. This should never happen if equipment is sterilised properly and an aseptic technique is used. A postural headache may occur postoperatively. This should be rare (see later).

Indications for Spinal Anaesthesia

Spinal anaesthesia is best reserved for operations below the umbilicus e.g. hernia repairs, gynaecological and

urological operations and any operation on the perineum or genitalia. All operations on the leg are possible, but an amputation, though painless, may be an unpleasant experience for an awake patient. In this situation it may be appropriate to combine the spinal with a light general anaesthetic.

Spinal anaesthesia is particularly suitable for older patients and those with systemic disease such as chronic respiratory disease, hepatic, renal and endocrine disorders such as diabetes. Many patients with mild cardiac disease benefit from the vasodilation that accompanies spinal anaesthesia except those with stenotic valvular disease or uncontrolled hypertension (see later). It is suitable for managing patients with trauma if they have been adequately resuscitated and are not hypovolaemic. In obstetrics, it is ideal for manual removal of a retained placenta (again, provided there is no hypovolaemia). There are definite advantages for both mother and baby in using spinal anaesthesia for caesarean section. However, special considerations apply to managing spinal anaesthesia in pregnant patients (see later) and it is best to become experienced in its use in the non-pregnant patient before using it for obstetrics.

Contra-indications to Spinal Anaesthesia

Most of the contra-indications to spinal anaesthesia apply equally to other forms of regional anaesthesia. These include:

Inadequate resuscitation drugs and equipment. No regional anaesthetic technique should be attempted if drugs and equipment for resuscitation are not immediately to hand.

Clotting disorders. If bleeding occurs into the epidural space because the spinal needle has punctured an epidural vein, a haematoma could form and compress the spinal cord. Patients with a low platelet count or receiving anticoagulant drugs such as heparin or warfarin are at risk. Remember that patients with liver disease may have abnormal clotting profiles whilst low platelet counts as well as abnormal clotting can occur in pre-eclampsia.

Hypovolaemia from whatever cause e.g. bleeding, dehydration due to vomiting, diarrhoea or bowel obstruction. Patients must be adequately rehydrated or resuscitated before spinal anaesthesia or they will become very hypotensive.

Patient refusal. Patients may be understandably apprehensive and initially state a preference for general anaesthesia, but if the advantages of spinal anaesthesia are explained they may then agree to the procedure and be

pleasantly surprised at the outcome. If, despite adequate explanation, the patient still refuses spinal anaesthesia, their wishes should be respected. Likewise, mentally handicapped patients and those with psychiatric problems need careful pre-operative assessment.

Children. Although spinal anaesthesia has been successfully performed on children, this is a highly specialised technique best left to experienced paediatric anaesthetists.

Sepsis on the back near the site of lumbar puncture lest infection be introduced into the epidural or intrathecal space.

Septicaemia. If a patient is septicaemic, they are at increased risk of developing a spinal abscess. Epidural abscesses can, however, appear spontaneously in patients who have not had spinal/epidural injections especially if they are immuno-deficient: e.g., patients with AIDS, tuberculosis, and diabetes

Anatomical deformities of the patient's back. This is a relative contraindication, as it will probably only serve to make the dural puncture more difficult.

Neurological disease. The advantages and disadvantages of spinal anaesthesia in the presence of neurological disease need careful assessment. Any worsening of the disease post-operatively may be blamed erroneously on the spinal anaesthetic. Raised intracranial pressure, however, is an absolute contra-indication as a dural puncture may precipitate coning of the brain stem.

Controversies in spinal anaesthesia

Operator/anaesthetist. The same individual should not be responsible for over-seeing the anaesthetic and performing the surgery as "anaesthetic" problems can occur during the course of the operation. If such problems occur once surgery has started, the safety of the patient may be severely compromised. However, in many places doctors perform a spinal anaesthetic and then delegate intra-operative care of the patient to a suitably trained assistant while the surgery is performed.

Difficult airway. At first sight, spinal anaesthesia may appear to offer an ideal solution to the problem of a patient with a potentially difficult airway who requires lower abdominal surgery. However, the onset of a total spinal block or unforeseen surgical complications may make it imperative that the airway is secured. All the equipment necessary for intubation should, therefore, always be available before spinal anaesthesia is commenced. It is

always an extremely difficult to decide on whether to embark on a spinal anaesthetic when a patient is known to be difficult to intubate. The correct decision can only be made by the individual anaesthetist when all relevant clinical information is taken into account.

Spinal with sedation. Surgery is always stressful for the patient and most patients, even when totally pain free from a successful spinal, welcome some sedation. The optimal level of sedation can be difficult to judge as too much sedation can lead to hypoventilation, hypoxia or silent regurgitation of gastric contents. As a general rule, it should be possible to easily rouse the drowsy patient and be possible to maintain verbal contact with them.

In the event of an inadequate spinal anaesthetic it is much better to electively administer a light general anaesthetic and safeguard the airway, then to over-sedate a patient with benzodiazepines or narcotics.

Combined spinal/epidural block. There is currently much interest in combining the simplicity and speed of onset of a spinal anaesthetic with the flexibility of epidural anaesthesia by inserting an epidural catheter which allows the anaesthetic block to be extended. Furthermore, the epidural catheter can be used for post-operative analgesia. As well as combining the advantages of both techniques, the disadvantages are combined and new problems, peculiar to the technique are being described. Practitioners have to be skilled in performing and managing both spinal and epidural anaesthesia before considering the combined technique.

Physiology of Spinal Anaesthesia

Local anaesthetic solution injected into the subarachnoid space blocks conduction of impulses along all nerves with which it comes in contact, although some nerves are more easily blocked than others. There are three classes of nerve: motor, sensory and autonomic. Stimulation of the motor nerves causes muscles to contract and when they are blocked, muscle paralysis results. Sensory nerves transmit sensations such as touch and pain to the spinal cord and from there to the brain, whilst autonomic nerves control the calibre of blood vessels, heart rate, gut contraction and other functions not under conscious control.

Generally, autonomic and sensory fibres are blocked before motor fibres. This has several important consequences. For example, vasodilation and a drop in blood pressure may occur when the autonomic fibres are blocked and the patient may be aware of pressure or movement and yet feel no pain when surgery starts.

Practical implications of physiological changes. The patient should be well hydrated before the local anaesthetic is injected and should have an intravenous infusion in place so that further fluids or vasoconstrictors can be given if hypotension occurs.

Anatomy

The spinal cord usually ends at the level of L2 in adults and L3 in children. Dural puncture above these levels is associated with a slight risk of damaging the spinal cord and is best avoided. An important landmark to remember is that a line joining the top of the iliac crests is at L4 to L5. Remember the structures that the needle will pierce before reaching the CSF (figure 1).

The skin. It is wise to inject a small blob of local anaesthetic into the skin before inserting the spinal needle.

Subcutaneous fat. This, of course, is of variable thickness. Identifying the intervertebral spaces is far easier in thin patients.

The **supraspinous ligament** that joins the tips of the spinous processes together.

The **interspinous ligament** which is a thin flat band of ligament running between the spinous processes.

The **ligamentum flavum** is quite thick, up to about 1cm in the middle and is mostly composed of elastic tissue. It runs vertically from lamina to lamina. When the needle is within the ligaments it will feel gripped and a distinct “give” can often be felt as it passes through the ligament and into the epidural space.

The **epidural space** contains fat and blood vessels. If blood comes out of the spinal needle instead of CSF when the stylet is removed, it is likely that an epidural vein has been punctured. The needle should simply be advanced a little further.

The dura. After feeling a “give” as the needle passes through the ligamentum flavum, a similar sensation may be felt when the needle is advanced a further short distance and pierces the dural sac.

The **subarachnoid space.** This contains the spinal cord and nerve roots surrounded by CSF. An injection of local anaesthetic will mix with the CSF and rapidly block the nerve roots with which it comes in contact.

Local Anaesthetics for Spinal Anaesthesia

Local anaesthetic agents are either heavier (hyperbaric), lighter (hypobaric), or have the same specific gravity (isobaric) as the CSF. Hyperbaric solutions tend to

spread down (due to gravity) from the level of the injection, while isobaric solutions are not influenced in this way. Hypobaric solutions are rarely used. It is easier to predict the spread of spinal anaesthesia when using a hyperbaric agent. Isobaric preparations may be made hyperbaric by the addition of dextrose. Other factors affecting the spread of local anaesthetic agents when used for spinal blocks are described later.

Bupivacaine (Marcaine). 0.5% hyperbaric (heavy) bupivacaine is the best agent to use if it is available. 0.5% plain bupivacaine is also popular. Bupivacaine lasts longer than most other spinal anaesthetics: usually 2-3 hours.

Lidocaine/Lignocaine (Xylocaine). Best results are said to be obtained with 5% hyperbaric (heavy) lidocaine, which lasts 45-90 minutes. 2% lignocaine can also be used but it has a shorter duration of action. If 0.2ml of adrenaline 1:1000 is added to the lignocaine, it will usefully

prolong its duration of action. Recently concerns have been raised about the safety of 5% lidocaine (it is said to be potentially neurotoxic) despite it having been used uneventfully for over forty years. Lidocaine from multi-dose vials should not be used for intrathecal injection as it contains potentially harmful preservatives.

Cinchocaine (Nupercaine, Dibucaine, Percaine, Sovcaine). 0.5% hyperbaric (heavy) solution is similar to bupivacaine.

Tetracaine (Amethocaine, Pantocaine, Pontocaine, Decicain, Butethanol, Anethaine, Dikain). A 1% solution can be prepared with dextrose, saline or water for injection.

Mepivacaine (Scandicaine, Carbocaine, Meaverin). A 4% hyperbaric (heavy) solution is similar to lignocaine.

Pethidine/Meperidine. The 5% solution (50mg/ml) has local anaesthetic properties and is a versatile agent. The standard intravenous preparation is preservative-free and is isobaric. A dose of 0.5-1mg/kg is usually adequate for spinal anaesthesia.

Ropivacaine (Naropin) is a recently introduced long-acting local anaesthetic, similar to bupivacaine. It is not currently licensed for uses as a spinal anaesthetic.

It is generally thought that of the commonly used anaesthetic agents, lidocaine has a more rapid onset than bupivacaine, though some authors question this. Meperidine has a very rapid onset but can also wear off rapidly. It should also be remembered, especially when hyperbaric agents have been used, that patient movement, for example putting the patient "head-down" can cause the block to extend even some 20-30 minutes after it has been performed.

Spinal Anaesthesia and Common Medical Conditions

Respiratory disease. A low spinal block (below the umbilicus) has no effect on the respiratory system and is, therefore, ideal for patients with respiratory disease unless they cough a lot. Frequent coughing results in less than ideal conditions for the surgeon. A high spinal block can produce intercostal muscle paralysis, but this does not usually create any problems, unless the patient has a very limited respiratory reserve and is, for example, unable to lie flat.

Uncontrolled hypertension or severe valvular disease. Although moderate hypertension is not a contraindication to spinal anaesthesia, it should be remembered that there is an almost inevitable fall in blood pressure when spinal anaesthesia is induced. This can be particularly

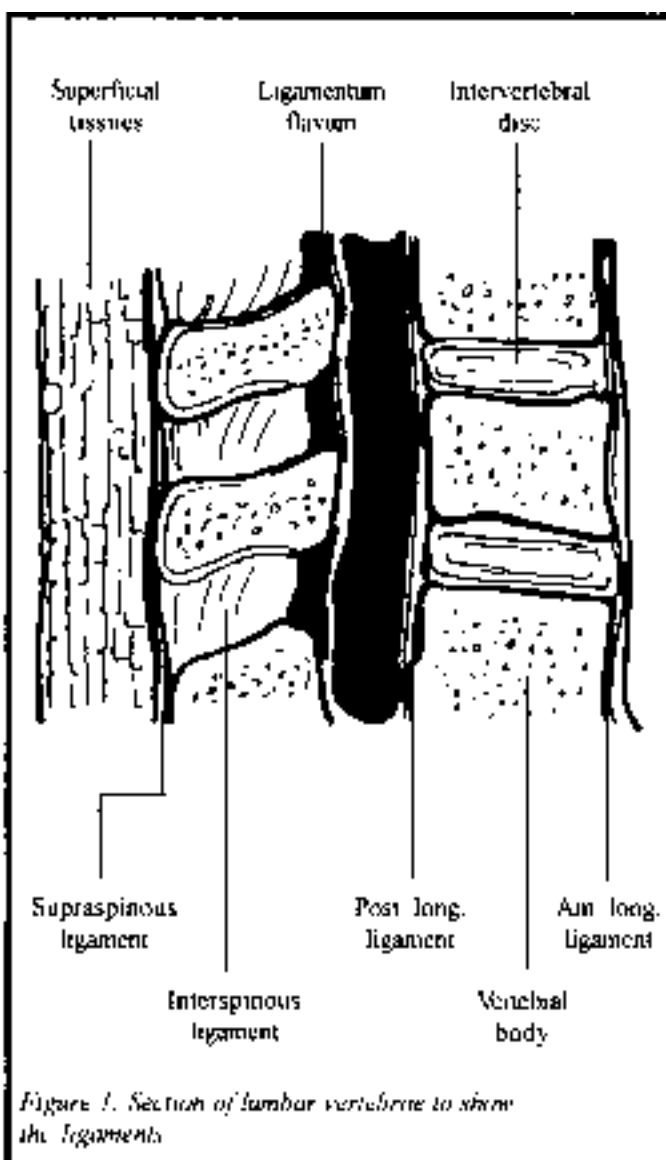


Figure 1. Section of lumbar vertebrae to show the ligaments

precipitous in patients with severe uncontrolled hypertension. Patients with aortic stenosis require a stable blood pressure (sustained after-load) to maintain their coronary perfusion. If they have a sudden fall in blood pressure, they may develop intractable cardiac arrest.

Sickle cell disease/trait. Spinal anaesthesia may be advantageous for patients with sickle cell disease. Follow the same rules as for general anaesthesia: ensure that the patient is well oxygenated, well hydrated and not allowed to become hypotensive. Consider warming the intravenous fluids and do not allow the patient to become cold. Avoid the use of tourniquets.

Preoperative Visit

Patients should be told about their anaesthetic during the preoperative visit. It is important to explain that although spinal anaesthesia abolishes pain, they may be aware of some sensation in the relevant area, but it will not be uncomfortable. It should also be explained that their legs will become weak or feel as if they don't belong to them any more. They must be reassured that these sensations are perfectly normal and that if, by any chance, they feel pain they will be given a general anaesthetic.

Premedication is often unnecessary, but if a patient is apprehensive, a benzodiazepine such as 5-10mg of diazepam may be given orally 1 hour before the operation. Other sedative or narcotic agents may also be used. Anticholinergics such as atropine or scopolamine (hyoscine) are not routinely required.

Intravenous Pre-loading

All patients having spinal anaesthesia must have a large intravenous cannula inserted and be given intravenous fluids immediately before the spinal. This helps prevent hypotension following the vasodilation which is produced. The volume of fluid given will vary with the age of the patient and the extent of the proposed block. A young, fit man having a hernia repair may only need 500mls. Older patients are not able to compensate as efficiently as the young for spinal-induced vasodilation and hypotension and may need 1000mls for a similar procedure. If a high block is planned, at least a 1000mls should be given to all patients. Caesarean section patients need at least 1500mls. Crystalloids such as 0.9% Normal Saline or Hartmans are most commonly used. Dextrose 5% should be avoided as it is not effective for maintaining the blood volume

Positioning the Patient for Lumbar Puncture

Lumbar puncture is most easily performed when there

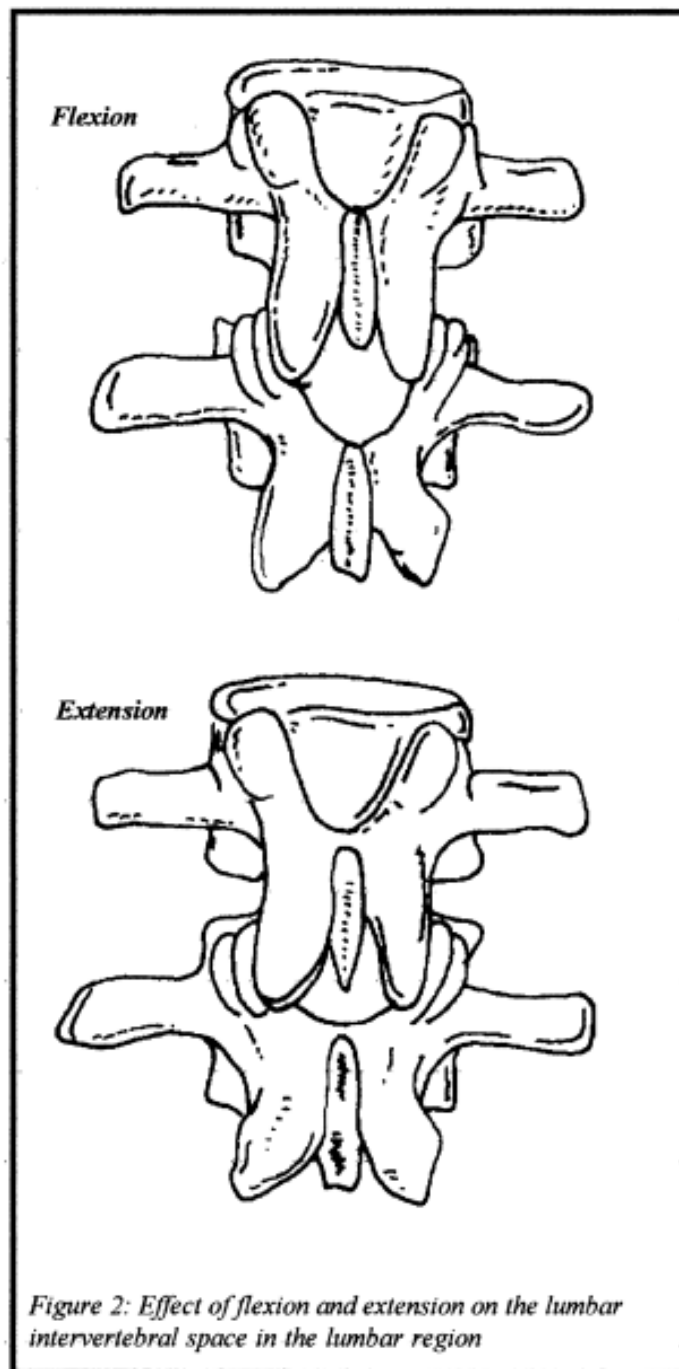


Figure 2: Effect of flexion and extension on the lumbar intervertebral space in the lumbar region

is maximum flexion of the lumbar spine (figure 2). This can best be achieved by sitting the patient on the operating table and placing their feet on a stool. If they then rest their forearms on their thighs, they can maintain a stable and comfortable position. Alternatively, the procedure can be performed with the patient lying on their side with their hips and knees maximally flexed.

An assistant may help to maintain the patient in a comfortable curled position. The sitting position is preferable in the obese whereas the lateral is better for uncooperative or sedated patients. Consider the consequences of sudden hypotension or a vaso-vagal

attack for a sitting patient. The anaesthetist can either sit or kneel whilst performing the block.

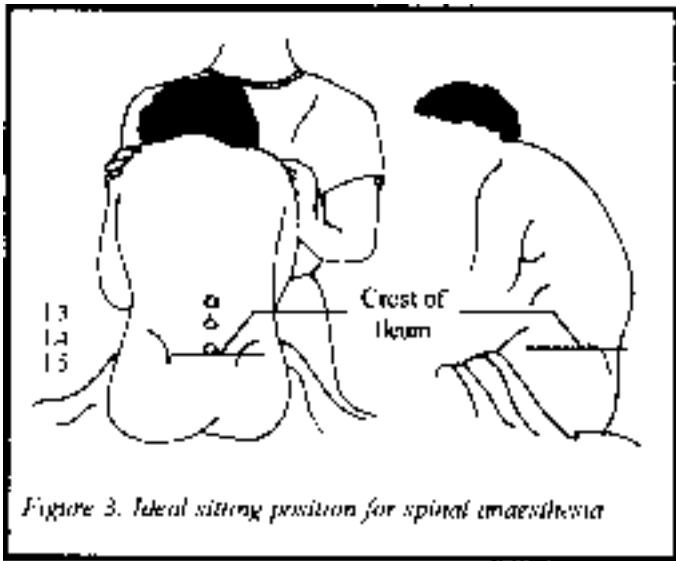


Figure 3. Ideal sitting position for spinal anaesthesia

Factors Affecting the Spread of the Local Anaesthetic Solution

A number of factors affect the spread of the injected local anaesthetic solution within the CSF and the ultimate extent of the block obtained.

Among these are:

- the baricity of the local anaesthetic solution
- the position of the patient
- the concentration and volume injected
- the level of injection
- the speed of injection

The specific gravity of the local anaesthetic solution can be altered by the addition of dextrose. Concentrations of 7.5% dextrose make the local anaesthetic hyperbaric (heavy) relative to CSF and also reduce the rate at which it diffuses and mixes with the CSF. Isobaric and hyperbaric solutions both produce reliable blocks. Injecting hyperbaric solutions and then altering the patient's position probably produces the most controllable blocks.

If a patient is kept sitting for several minutes after the injection of a small volume of a hyperbaric solution of local anaesthetic, a classical "saddle block" affecting only the sacral nerve roots will result.

The spinal column of patients lying on their side is rarely truly horizontal. Males tend to have wider shoulders than hips and so are in a slight "head up" position when lying on their sides, whilst for females with their wider hips, the opposite is true. Regardless

of the position of the patient at the time of injection and whatever the initial extent of the block obtained, the level of the block may change if the patient's position is altered within twenty minutes of the injection of a hyperbaric agent.

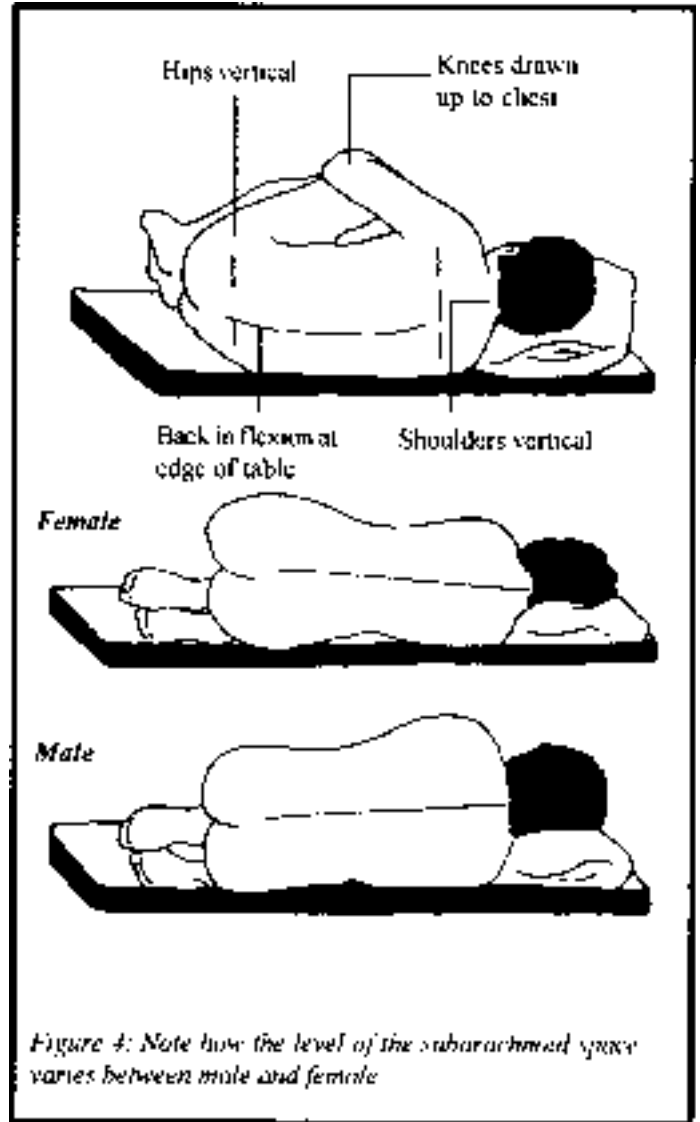


Figure 4: Note how the level of the subarachnoid space varies between male and female

The quantity of local anaesthetic (in milligrams) injected will determine the quality of the block obtained whilst its extent will also be determined by the volume in which it is injected. Large volumes of concentrated solutions will, thus, produce dense blockade over a large area. As spinal anaesthetics are generally only injected in the lumbar region, the extent of the block is influenced more by the volume and concentration injected and the position of the patient than the actual interspace at which the injection occurs.

The speed of injection has a slight effect on the eventual extent of the block. Slow injections result in a more predictable spread while rapid injections produce eddy currents within the CSF and a somewhat

less predictable outcome.

Finally, increased abdominal pressure from whatever cause (pregnancy, ascites etc.) can lead to engorgement of the epidural veins, compression of the dura and hence a reduction in the volume of the CSF. A given quantity of local anaesthetic injected into the CSF might then be expected to produce a more extensive block.

Quantities of Local Anaesthetics to Use

The degree of spinal blockade needed, as measured by the height of the block, will depend on the operation to be performed (see Table 1). For certain blocks, less local anaesthetic is needed when hyperbaric rather than plain solutions are used. Special considerations apply to obstetric patients and so the following chart does not apply to them (see later section).

The volumes of local anaesthetic shown in Table 1 should be considered only as a guideline. The lower volumes suggested should generally be injected in particularly small people. More may have to be given if the resultant block is not high enough for the proposed operation. Hyperbaric agents and appropriate positioning are the most reliable way of obtaining a mid-thoracic block.

Preparation for Lumbar Puncture

Assemble the necessary equipment on a sterile surface. It will include:

- **A spinal needle.** The ideal would be 24-25 gauge with a pencil point tip to minimise the risk of the patient developing a post-spinal headache.
- **An introducer,** if using a fine gauge needle as they are thin and flexible, and therefore difficult to direct accurately. A standard 19 gauge (white) disposable needle is suitable for use as an introducer.
- A 5ml syringe for the spinal anaesthetic solution.
- A 2ml syringe for local anaesthetic to be used for skin infiltration.
- A selection of needles for drawing up the local anaesthetic solutions and for infiltrating the skin.
- A gallipot with a suitable antiseptic for cleaning the skin, e.g. chlorhexidine, iodine, or methyl alcohol.
- Sterile gauze swabs for skin cleansing.
- A sticking plaster to cover the puncture site.
- The local anaesthetic to be injected intrathecally should be in a single use ampoule. Never use local anaesthetic from a multi-dose vial for intrathecal injection. Spare equipment and drugs should be readily available if needed.

Performing the Spinal Injection

It is assumed that the patient has had the procedure fully explained, has reliable intravenous access, is in

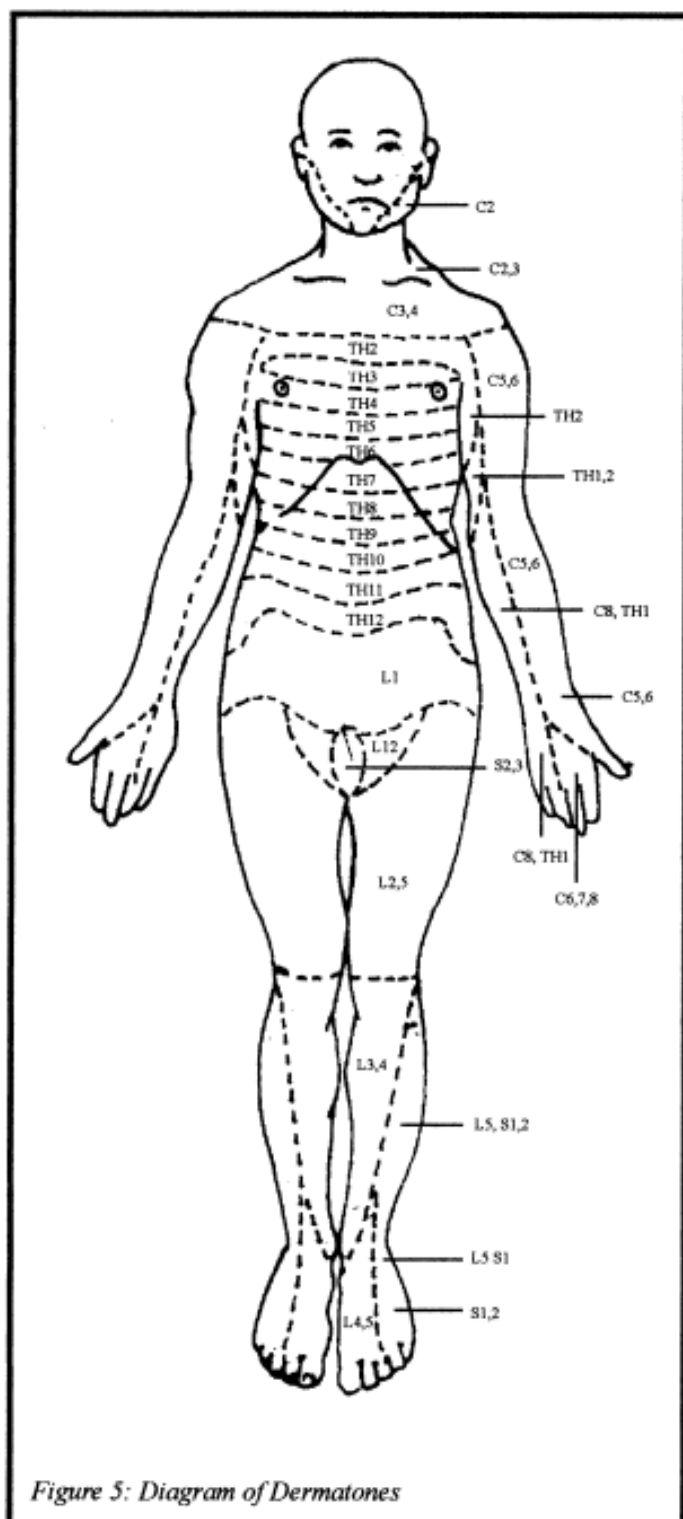


Figure 5: Diagram of Dermatomes

Table 1.

Type of block	Hyperbaric Bupivacaine	Plain Bupivacaine	Hyperbaric Lidocaine
Saddle block e.g. operations of genitalia, perineum	2ml	2ml	1ml
Lumbar block e.g. operations on legs, groin, hernias	2-3ml	2-3ml	1.5-2ml
Mid-thoracic blocks e.g. hysterectomy	2-4ml	2-4ml	2ml

a comfortable position and that resuscitation equipment is immediately available.

- Scrub and glove up carefully.
- Check the equipment on the sterile trolley.
- Draw up the local anaesthetic to be injected intrathecally into the 5ml syringe, from the ampoule opened by your assistant. Read the label. Draw up the exact amount you intend to use, ensuring that your needle does not touch the outside of the ampoule (which is unsterile).
- Draw up the local anaesthetic to be used for skin infiltration into the 2ml syringe. Read the label.
- Clean the patient's back with the swabs and antiseptic ensuring that your gloves do not touch unsterile skin. Swab radially outwards from the proposed injection site. Discard the swab and repeat several times making sure that a sufficiently large area is cleaned. Allow the solution to dry on the skin.
- Locate a suitable interspinous space. You may have to press fairly hard to feel the spinous processes in an obese patient.
- Inject a small volume of local anaesthetic under the skin with a disposable 25-gauge needle at the proposed puncture site.
- Insert the introducer if using a 24-25 gauge needle. It should be advanced into the ligamentum flavum but care should be exercised in thin patients that an inadvertent dural puncture does not occur.

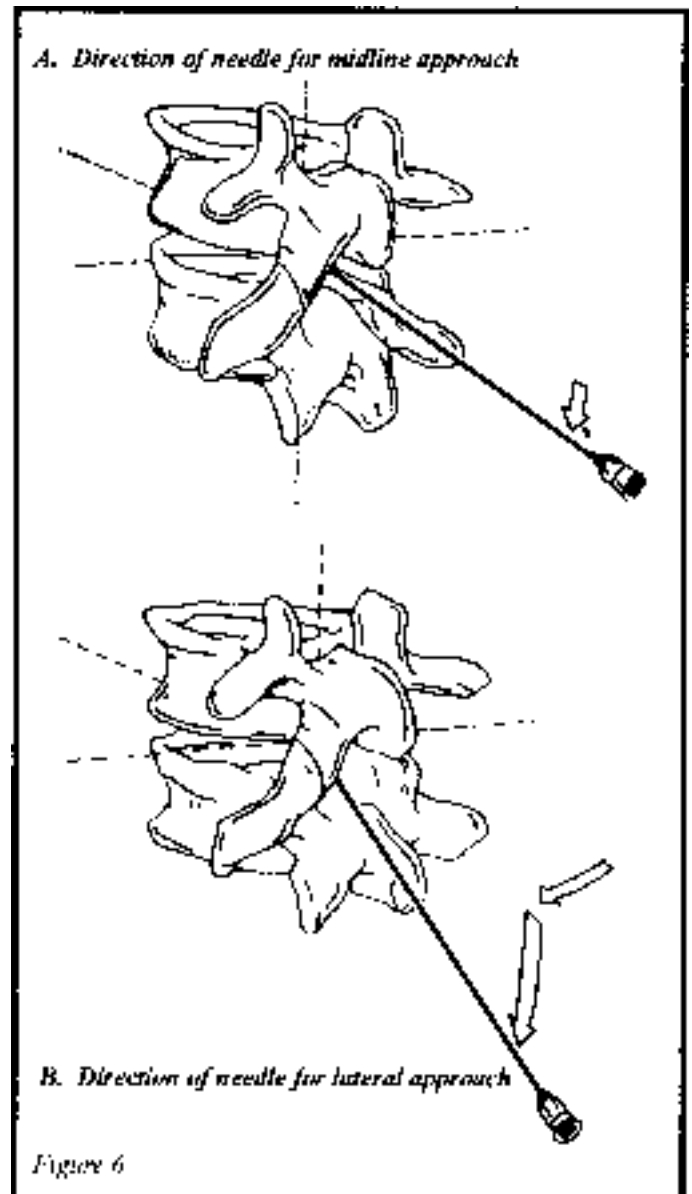


Figure 6

- Insert the spinal needle (through the introducer, if applicable). Ensure that the stylet is in place so that the tip of the needle does not become blocked by particles of tissue or clot. It is imperative that the needle is inserted and stays in the midline and that the bevel is directed laterally. It is angled slightly cephalad (towards the head) and slowly advanced. An increased resistance will be felt as the needle enters the ligamentum flavum, followed by a loss of resistance as the epidural space is entered. Another loss of resistance may be felt as the dura is pierced and CSF should flow from the needle when the stylet is removed. If bone is touched, the needle should be withdrawn a centimetre or so and then re-advanced in a slightly more cephalad direction again ensuring that it stays in the midline. If a 25 gauge spinal needle is being used, be prepared to wait 20-30 seconds for CSF to appear after the stylet has been withdrawn. If no CSF appears, replace the stylet and advance the needle a little further and try again.
- When CSF appears, take care not to alter the position of the spinal needle as the syringe of local anaesthetic is being attached. The needle is best immobilised by resting the back of the non-dominant hand firmly against the patient and by using the thumb and index finger to hold the hub of the needle. Be sure to attach the syringe firmly to the hub of the needle; hyperbaric solutions are viscous and resistance to injection will be high, especially through fine gauge needles. It is, therefore, easy to spill some of the local anaesthetic unless care is taken. Aspirate gently to check the needle tip is still intrathecal and then slowly inject the local anaesthetic. When the injection is complete, withdraw the spinal needle, introducer and syringe as one and apply a sticking plaster to the puncture site.

Practical Problems

The spinal needle feels as if it is in the right position but no CSF appears. Wait at least 30 seconds, then try rotating the needle 90 degrees and wait again. If there is still no CSF, attach an empty 2ml syringe and inject 0.5-1ml of air to ensure the needle is not blocked then use the syringe to aspirate whilst slowly withdrawing the spinal needle. Stop as soon as CSF appears in the syringe.

Blood flows from the spinal needle. Wait a short time. If the blood becomes pinkish and finally clear, all is well. If blood only continues to drip, then it is likely that the needle tip is in an epidural vein and it should be advanced a little further to pierce the dura.

The patient complains of sharp, stabbing leg pain. The needle has hit a nerve root because it has deviated laterally. Withdraw the needle and redirect it more medially away from the affected side.

Wherever the needle is directed, it seems to strike bone. Make sure the patient is still properly positioned with as much lumbar flexion as possible and that the needle is still in the mid-line. If you are not sure whether you are in the midline, ask the patient on which side they feel the needle. Alternatively, if the patient is elderly and cannot bend very much or has heavily calcified interspinous ligaments, it might be better to attempt a paramedian approach to the dura. This is performed by inserting the spinal needle about 0.5-1 cm lateral to the mid line at the level of the upper border of a spinous process, then directing it both cephalad and medially. If bone is contacted it is likely to be the vertebral lamina. It should then be possible to “walk” the needle off the bone and into the epidural space, then through it to pierce the dura. When using this technique inject some local anaesthetic into the muscle before inserting the spinal needle.

The patient complains of pain during needle insertion. This suggests that the spinal needle is passing through the muscle on either side of the ligaments. Redirect your needle away from the side of the pain to get back into the midline or inject some local anaesthetic.

The patient complains of pain during injection of the spinal solution. Stop injecting and change the position of the needle.

Assessing the Block

Some patients are very poor at describing what they do or do not feel, therefore, objective signs are valuable. If, for example, the patient is unable to lift his legs from the bed, the block is at least up to the mid-lumbar region. It is unnecessary to test sensation with a sharp needle and leave the patient with a series of bleeding puncture wounds. It is better to test for a loss of temperature sensation using a swab soaked in either ether or alcohol. Do this by first touching the patient with the damp swab on the chest or arm (where sensation is normal), so that they appreciate that the swab feels cold. Then work up from the legs and lower abdomen until the patient again appreciates that the swab feels cold.

If the replies are inconsistent or equivocal, the patient can be gently pinched with artery forceps or fingers on blocked and unblocked segments and asked if they feel pain. Using this method, there is rarely any difficulty in ascertaining the extent of the block.

Surgeons should be dissuaded from prodding the patient and asking, "can you feel this?" Surgeons and patients should be reminded that when a block is successful, a patient may still be aware of touch but will not feel pain.

Problems with the Block

No apparent block at all. If after 10 minutes the patient still has full power in the legs and normal sensation, then the block has failed probably because the injection was not intrathecal. Try again.

The block is one-sided or is not high enough on one side. When using a hyperbaric solution, lie the patient on the side that is inadequately blocked for a few minutes and adjust the table so that the patient is slightly "head down". When using an isobaric solution, lie the patient on the side that is blocked. (Moving a patient around in any way at all in the first 10-20 minutes following injection will tend to increase the height of the block).

Block not high enough. When using a hyperbaric solution, tilt the patient head down whilst they are supine (lying on the back), so that the solution can run up the lumbar curvature. Flatten the lumbar curvature by raising the patient's knees. When using a plain solution turn the patient a complete circle from supine to prone (lying on the front) and back to supine again.

Block too high. The patient may complain of difficulty in breathing or of tingling in the arms or hands. Do not tilt the table "head up". (See later under Treatment of a total spinal).

Nausea or vomiting. This may occur with high spinal blocks that may be associated with hypotension. Check the blood pressure and treat accordingly (see later).

Shivering. This occurs occasionally. Reassure the patient and give oxygen by mask.

Monitoring

It is essential to monitor the respiration, pulse and blood pressure closely. The blood pressure can fall precipitously following induction of spinal anaesthesia, particularly in the elderly and those who have not been adequately preloaded with fluid.

Warning signs of falling blood pressure include pallor, sweating, nausea or feeling generally unwell. A moderate fall in systolic blood pressure to say 80-90mm Hg in a young, healthy patient or 100mmHg in an older patient is acceptable, provided the patient looks and feels well and is adequately oxygenated.

Bradycardia is quite common during spinal anaesthesia particularly if the surgeon is manipulating the bowel or uterus. If the patient feels well, and the blood pressure is maintained, then it is not necessary to give atropine. If, however, the heart rate drops below 50 beats per minute or there is hypotension, then atropine 300-600mcg should be given intravenously. If the heart rate does not increase try ephedrine (see below).

It is generally considered good practice for all patients undergoing surgery under spinal anaesthesia to be given supplemental oxygen by facemask at a rate of 2-4 litres/minute, especially if sedation has also been given.

Treatment of Hypotension

Hypotension is due to vasodilation and a functional decrease in the effective circulating volume. The treatment is, therefore, to reverse the vasodilatation with vasoconstrictor drugs and increase the circulating volume by giving fluids. All hypotensive patients should be given oxygen by mask until the blood pressure is restored.

A simple and effective way of rapidly increasing the patient's circulating volume is by raising their legs thus increasing the return of venous blood to the heart. This can either be done manually by an assistant or by tilting the lower half of the operating table. Tilting the whole operating table head down will also achieve the same effect, but is unwise if a hyperbaric spinal anaesthetic has been injected as it will result in the block spreading higher and the hypotension becoming more severe. If an isobaric spinal solution has been used, tilting the table at any time will have very little effect on the height of the block.

Increase the speed of the intravenous infusion to maximum until the blood pressure is restored to acceptable levels and, if the pulse is slow, give atropine intravenously. Vasoconstrictors should be given immediately if the hypotension is severe, and to patients not responding to fluid therapy.

Vasopressors

Ephedrine is probably the vasopressor of choice. It causes peripheral blood vessels to constrict and raises the cardiac output by increasing the heart rate and the

force of myocardial contraction. It is safe for use in pregnancy, as it does not reduce placental blood flow. Ephedrine is generally available in 25 or 30mg ampoules. It is best diluted to 10mls with saline and then given in increments of 1-2ml (2.5-6mg) titrated against the blood pressure. Its effect generally lasts about 10 minutes and it may need repeating. Alternatively, the ampoule may be added to a bag of intravenous fluid and the rate of infusion altered to maintain the desired blood pressure. It can also be given intramuscularly but its onset time is delayed although its duration is prolonged. Larger doses are necessary when it is given intramuscularly.

Methoxamine (Vasoxine). It is available in 20mg ampoules and must be diluted before injection. A suitable adult dose is 2mg intravenously or 5-20mg by intramuscular injection. It is a pure peripheral vasoconstrictor and reflex bradycardia, needing treatment with atropine can occur. It is particularly useful to treat hypotension during spinal anaesthesia when the patient has a tachycardia.

Phenylephrine. A pure peripheral vasoconstrictor which is available in 10mg ampoules; it must be diluted before use. Suitable adult doses for intravenous use are 100-500mcg repeated after 15 minutes if necessary, or 2-5mg intramuscularly. It lasts about 15 minutes. A reflex bradycardia may occur.

Metaraminol (Aramine). It is supplied in 10mg ampoules and should be diluted and used incrementally (1-5mg) as with ephedrine. Alternatively, it can be added to 500ml of fluid and titrated against the blood pressure. It has a slower onset time (at least 2 minutes after intravenous injection) but lasts longer (20-60 minutes)

Epinephrine/Adrenaline. Available as 1mg/ml (1:1,000) and 1mg/10ml (1:10,000) ampoules. Dilute 1ml of 1:1,000 adrenaline to at least 10ml with saline and give increments of 50mcg (0.5ml of 1:10,000) repeating as necessary. Monitor the effect of epinephrine/adrenaline closely - it is a very powerful drug but only lasts a few minutes. It may be used during spinal anaesthesia if hypotension does not respond to first line drugs listed above or when they are not available.

Norepinephrine/Noradrenaline (Levophed). A powerful vasoconstrictor available in 2mg ampoules which must be diluted in 1000ml of intravenous fluid before use. It is then given at an initial rate of 2-3ml/minute and thereafter titrated against the blood pressure. Control the infusion with the utmost care taking particular care that to avoid extravasation.

Treatment of Total Spinal

Although rare, total spinal can occur with frightening rapidity and result in the death of the patient if not quickly recognised and treated. They are more likely to occur when a planned epidural injection is, inadvertently, given intrathecally. The warning signs that a total spinal block is developing are:

- **Hypotension** - treat as detailed above. Remember that nausea may be the first sign of hypotension. Repeated doses of vasopressors and large volumes of fluid may be necessary.
- **Bradycardia** - give atropine. If this is not effective give ephedrine or adrenaline
- **Increasing anxiety** - reassure.
- **Numbness or weakness of the arms and hands**, indicating that the block has reached the cervico-thoracic junction.
- **Difficulty breathing** - as the intercostal nerves are blocked the patient may state that they can't take a deep breath. As the phrenic nerves (C3,4,5) which supply the diaphragm become blocked, the patient will initially be unable to talk louder than a whisper and will then stop breathing.
- **Loss of consciousness.**

Call for help - several pairs of hands may be useful!

- ABC Resuscitation
- Intubate and ventilate the patient with 100% oxygen.

Treat hypotension and bradycardia with intravenous fluids, atropine and vasopressors as described earlier. If treatment is not started quickly the combination of hypoxia, bradycardia and hypotension may result in a cardiac arrest.

- Ventilation will need to be continued until the spinal block recedes and the patient is able to breathe again unaided. The time this will take will depend on which local anaesthetic has been injected.
- Once the airway has been controlled and the circulation restored, consider sedating the patient with a small dose of a benzodiazepine as consciousness may return before muscle power and the patient will find it distressing to be unable to breathe properly.

General Postoperative Care

The patient should be admitted to the recovery room as with any other anaesthetised patient. In the event of hypotension in the recovery room, the nurses should elevate the patients' legs, increase the rate at which intravenous fluids are being administered, give oxygen and summon the anaesthetist. Further doses of vasoconstrictors or fluids may be required, as previously discussed. Patients should be advised as to how long their spinal block will last and be told to remain in bed until full sensation and muscle power has returned.

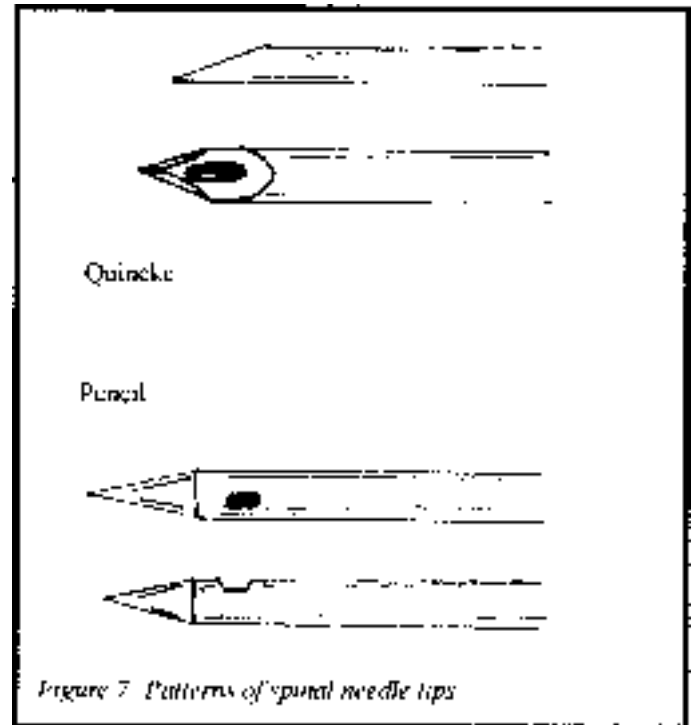
Complications of Spinal Anaesthesia

Headache. A characteristic headache may occur following spinal anaesthesia. It begins within a few hours and may last a week or more. It is postural, being made worse by standing or even raising the head and relieved by lying down. It is often occipital and may be associated with a stiff neck. Nausea, vomiting, dizziness and photophobia frequently accompany it. It is more common in the young, in females and especially in obstetric patients. It is thought to be caused by the continuing loss of CSF through the hole made in the dura by the spinal needle. This results in traction on the meninges and pain.

The incidence of headache is related directly to the size of the needle used. A 16 gauge needle will cause headache in about 75% of patients, a 20 gauge needle in about 15% and a 25 gauge needle in 1-3%. It is, therefore, sensible to use the smallest needle available especially in high-risk obstetric patients. As the fibres of the dura run parallel to the long axis of the spine, if the bevel of the needle is parallel to them, it will part rather than cut them and therefore, leave a smaller hole. Make a mental note of which way the bevel lies in relation to the notch on the hub and then align it appropriately. It is widely considered that pencil-point needles (Whiteacre or Sprotte) make a smaller hole in the dura and are associated with a lower incidence of headache (1%) than conventional cutting-edged needles (Quincke) (figure 7).

Treatment of spinal headache. Patients with spinal headaches prefer to remain lying flat in bed as this relieves the pain. They should be encouraged to drink freely or, if necessary, be given intravenous fluids to maintain adequate hydration. Simple analgesics such as paracetamol, aspirin or codeine may be helpful, as may measures to increase intra-abdominal and hence epidural pressure such as lying prone. Sumatriptan, normally used in the treatment of migraine, is said to

be effective. Caffeine containing drinks such as tea, coffee or Coca-Cola are often helpful. Prolonged or severe headaches may be treated with epidural blood patch performed by aseptically injecting 15-20ml of the patient's own blood into the epidural space. This then clots and seals the hole and prevents further leakage of CSF.



It used to be thought that bedrest for 24 hours following a spinal anaesthetic would help reduce the incidence of headache, but this is now no longer believed to be the case. Patients may get up once normal sensation has returned, if surgical considerations so allow.

Urinary retention. As the sacral autonomic fibres are among the last to recover following a spinal anaesthetic, urinary retention may occur. If fluid pre-loading has been excessive, a painful distended bladder may result and the patient may need to be catheterised.

Permanent neurological complications are extremely rare. Many of those that have been reported were due to the injection of inappropriate drugs or chemicals into the CSF producing meningitis, arachnoiditis, transverse myelitis or the cauda equina syndrome with varying patterns of neurological impairment and sphincter disturbances. Damage to an epidural vein can lead to the formation of an epidural haematoma that compresses the spinal cord. This is most unlikely in a patient with a normal clotting profile. If inadequate sterile precautions are taken, bacterial meningitis or an epidural abscess may result

although it is thought that most such abscesses are caused by the spread of infection in the blood. Finally, permanent paralysis can occur due to the “anterior spinal artery syndrome”. This is most likely to affect elderly patients who are subjected to prolonged periods of hypotension and may result in permanent paralysis of the lower limbs.

Spinal Anaesthesia in Obstetrics (See article in Update in Anaesthesia No. 9)

There are several reasons for preferring spinal anaesthesia to general anaesthesia for Caesarean sections. Babies born to mothers having spinal (or epidural) anaesthesia may be more alert and less sedated, as they have not received any general anaesthetic agents through the placental circulation. As the mother’s airway is not compromised, there is a reduced risk of aspiration of gastric contents causing chemical pneumonitis (Mendelson’s syndrome).

Many mothers also welcome the opportunity of being awake during the delivery and being able to feed their child as soon as the operation is completed. There are, however, also disadvantages. It may be difficult to perform the spinal injection as the pregnant uterus will impede lumbar flexion and, if labour has started, the mother may be unable to remain still when having contractions. Unless small gauge needles (25 gauge) are used, the incidence of post-spinal headache may be unacceptably high. Spinal anaesthesia for Caesarean section should not be attempted until the anaesthetist has accumulated sufficient experience in non-pregnant patients.

In the absence of hypovolaemia due to bleeding, spinal anaesthesia is a simple and safe alternative to general anaesthesia for manual removal of a retained placenta. It does not produce uterine relaxation and if this is required, a general anaesthetic with a volatile agent may be preferred.

Technique

Spinal anaesthesia is performed and managed in pregnant patients in the same way as in non-pregnant patients but with a number of special considerations.

- It is generally recommended that obstetric patients should be pre-loaded with not less than 1500mls of a crystalloid solution before the dural puncture is performed.
- Although spinal anaesthesia is not contra-indicated in the presence of mild pre-eclampsia, remember that such patients may have altered clotting function and are relatively

hypovolaemic. There is always a chance that a pre-eclamptic patient may suddenly fit and anticonvulsant drugs (diazepam or thiopentone) must be immediately available. (See Update in Anaesthesia No. 9).

- Pregnant women need smaller volumes of spinal anaesthetic solution than non-pregnant women in order to obtain a given height of block. For a Caesarean section, anaesthesia should extend to T6 (about the bottom of the sternum) to be completely successful. This can usually be achieved with the following regimes, although the hyperbaric agents are more predictable:
 - 2.0-2.5ml of a hyperbaric solution of 0.5% bupivacaine or
 - 2.0-2.5ml of an isobaric solution of 0.5% bupivacaine or
 - 1.4-1.6ml of a hyperbaric solution of 5% lignocaine or
 - 2.0-2.5ml of an isobaric solution of 2% lignocaine with added adrenaline (0.2ml of 1:1000).

If anaesthesia is required for a forceps delivery, 1.0ml of a hyperbaric solution injected with the mother in the sitting position is usually adequate. Anaesthesia to T10 is needed for removal of a retained placenta. This can be obtained by injecting 1.5mls of a hyperbaric solution with the patient sitting and then lying her down.

Positioning of the Pregnant Patient

Pregnant patients should never lie supine as the gravid uterus will compress the vena cava and, to a lesser extent the aorta (aorto-caval compression) resulting in hypotension. They should, instead, always lie with a lateral tilt. This can be achieved either by tilting the whole table or by inserting a wedge under the patients’ right hip. The uterus is displaced slightly to the left and the vena cava is not compressed (see Update in Anaesthesia No. 9).

As with all patients undergoing surgery under spinal anaesthesia, oxygen should be given during the operation. As hypotension commonly occurs despite fluid pre-loading, many anaesthetists routinely give a dose of vasopressor intravenously. Ephedrine is the favoured vasopressor, as it does not cause constriction of the uterine blood vessels. If it is not available, one of the other vasopressors discussed previously should

be used as untreated hypotension can seriously damage the unborn infant.

After delivery of the baby, syntocinon is the oxytocic of choice as it is less likely to produce maternal nausea and vomiting than ergometrine.

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POSTOPERATIVE ANALGESIA IN PAEDIATRIC DAY CASE SURGERY

Dr Manjushree Ray, MD, Associate Professor, N.R.S. Medical College, Calcutta-700 014 and Dr. S. M. Basu, MD, DA (London), Editor, Indian Journal of Anaesthesia

Paediatric day case surgery was first described in 1909 by James Nicoll, who performed 8988 operations as day case at the Royal Glasgow Hospital. Since then, day case surgery has continued to grow and now about 50% to 60% of paediatric surgery is performed as outpatients in most of the western countries like USA and UK. In India, the incidence of paediatric day case surgery is low, i.e., 35%. This is because of illiteracy, lack of proper transport facilities and unhygienic conditions at home.

Key to success in paediatric day case surgery is proper selection of patients, prevention of common postoperative complications and adequate pain management. Severe postoperative pain not only decreases the patients' functional capacity but also is associated with longer postoperative stay and higher incidence of unanticipated readmission. Pain may precipitate postoperative nausea vomiting (PONV) which is another cause of unanticipated readmission. Hence adequate pain management is mandatory in day case surgery.

Planning for postoperative analgesia must be done during the preoperative visit, keeping in mind the age, psychological and ASA status of the patient, and the type of surgery. Appropriate assessment of pain is essential for providing optimal analgesia.

ASSESSMENT

Numerous scoring systems are available for assessment of pain in paediatric patients. Each system has its own advantages and disadvantages. Selection of scoring systems mainly depends upon the age of the child.

Neonates. Day case surgery is not contraindicated in full term neonates - minor procedures like examination under anaesthesia and incision and drainage can be performed. Fortunately, these procedures do not produce much postoperative pain.

A variety of assessment tools have been developed for neonates. Observation of facial expression, body position and movement, crying, arterial pressure, heart rate, skin colour, ventilatory frequency and sleeplessness are used to find out the severity of pain in neonates. But these parameters can be altered by non-painful stimuli. Therefore a more rational approach is to assess the improvement of behavioural or physiological parameters in response to comfort, analgesia or sedation.

Infants and Children up to 3 years. Like neonates, assessment of pain in this age group of children is also based on behavioural and physiological response to comfort and analgesic therapy. Though exhibited behaviour may be more vigorous with an "all or nothing" type of response, sometimes the response is more precise and they can locate the pain. Objective pain scale (OPS) and toddler-preschooler postoperative pain scale (TPPPS) are commonly used to assess the intensity of pain.

Children aged 3 to 7 years. These patients can differentiate the presence or absence of pain and locate the pain. They can also express the intensity of pain in the form nil, mild, moderate and severe. The face scale or Oucher scale can be used in this age group. Children of five or more years old can operate visual or colour analogue scales for expression of pain.

Older Children. Like adults, children more than seven years old can express intensity, location and quality of pain. Any scoring system such as horizontal VAS, vertical colour analogue scale and self reporting are effective and reliable.

MANAGEMENT OF POSTOPERATIVE PAIN

Operative procedures associated with severe postoperative pain should not be performed as day surgery. For most patients postoperative pain should not be a major problem provided that local anaesthesia and NSAID have been used either as a part of the anaesthetic technique or after completion of surgery. Oral analgesics are the mainstay of pain relief at home.

Topical Anaesthesia. EMLA cream is an eutectic mixture of prilocaine and lignocaine and is very effective at providing dermal anaesthesia. Topical EMLA decreases the pain associated with circumcision, release of preputial adhesion, myringotomies and skin grafting. To obtain effective analgesia cream should be applied to the skin with an occlusive dressing about 45 to 60 minutes prior to surgery. Duration of analgesia is about 1^o hours. EMLA should be used with caution in infants less than 3 months of age or in patients who are taking sulphonamides or other methaemoglobin inducing medications because of potential of methaemoglobinaemia.

Lignocaine gel can be used to provide analgesia following circumcision and after repairs of lacerations. Parents can be taught to apply the gel for postoperative analgesia during first 24-36 hours. Application of bupivacaine and epinephrine (adrenaline) on the open wound towards the end of surgery provides excellent analgesia. Topical local anaesthetic eye drops can be used to provide analgesia following ophthalmic surgery.

Instillation. Bupivacaine instillation before closure of small wounds is very effective. Continuous infusion of 0.25% bupivacaine through a small cannula at a rate of 1-3mls/hour provides a simple, safe and effective method of analgesia at the donor site of skin graft or iliac crest bone graft.

Wound infiltration. Local anaesthetic agents may be administered intradermally or subcutaneously to block impulse conduction in local nerve fibres. Surgical wound infiltration can be used to provide analgesia following skin biopsies, muscle biopsies and virtually all procedures where other regional blocks are either inappropriate or contraindicated.

Caudal epidural block is widely used in paediatric patients to provide analgesia following surgery below the level of

the umbilicus. With a single injection, it provides long lasting postoperative analgesia in paediatric day case surgery.

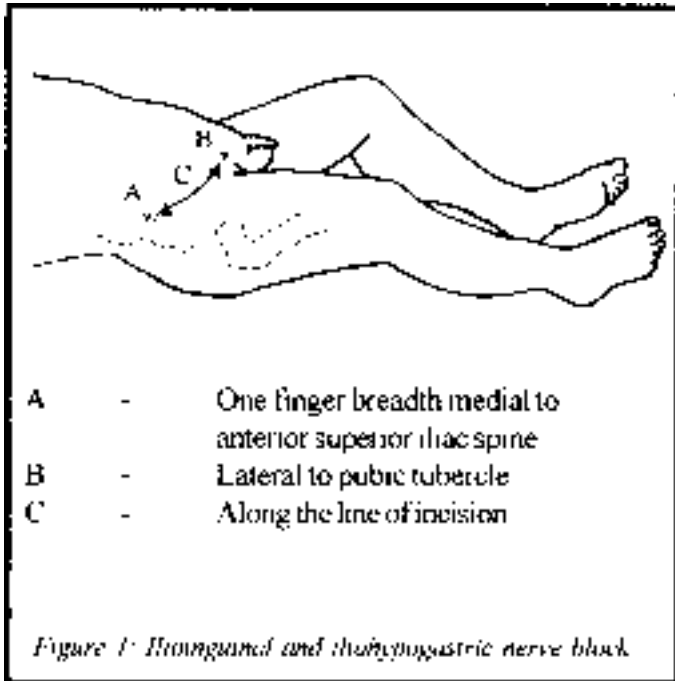
Caudal block is achieved by injecting local anaesthetic agents into the epidural space through the sacral hiatus, which is situated 1 to 2cm above the gluteal crease, superior to the coccyx and between the prominent sacral cornuae. The sacral hiatus can be located by drawing an equilateral triangle of which the two superior angles overlies the posterior superior iliac spines and third angle overlies the sacral hiatus (*see Update in Anaesthesia No. 9 1998*).

Under general anaesthesia the patient is placed in the lateral position. The skin is prepared using a standard sterile technique. The block is performed using a short bevelled needle of less than 3cm length to reduce the incidence of accidental dural puncture. The needle is inserted through the sacral hiatus at a 45 degree angle pointing rostrally (towards the head). Once the sacrococcygeal ligament is punctured the angle of the needle is decreased to 20 degrees. Approximately 0.75 to 1ml/kg of local anaesthetic agent is required for analgesia up to T10 level.

Weakness of the lower limbs associated with caudal block may delay the discharge of the patient. This can be minimised by using weaker local anaesthetic solutions such as 0.125% bupivacaine. Another drawback of single shot caudal block is its short duration. The duration can be prolonged by adding drugs such as clonidine an α_2 agonist, in a dose of 1-2mcg/kg or preservative free ketamine in a dose of 0.5mg/kg. Morphine and other spinal opioids are not recommended for paediatric day case surgery because of the risk of delayed respiratory depression.

Peripheral Nerve Block. Peripheral nerve blocks such as penile block, inguinal block, fascia iliaca block and sciatic nerve block have been demonstrated to be as effective as single shot caudal block. Moreover they produce longer lasting analgesia.

Penile block is performed to provide analgesia following circumcision, minor hypospadias surgery and other distal penile procedures. Different techniques have been described to block penile nerves including a midline and paramedian approaches. The paramedian approach is often preferred due to a lower incidence of complications such as intravascular injection, haematome and ischaemia. A short bevelled needle is inserted perpendicular to the skin at the inferior edge of the symphysis pubis at the 11 and 1o'clock positions. The needle is advanced until Bucks fascia is penetrated, which is determined by a loss of resistance. After careful aspiration plain 0.5% bupivacaine 1ml + 0.1ml/kg is administered. For better effect,



subcutaneous infiltration of local anaesthetics at the base of the penis from 3 to 9 o'clock position is recommended. However a full ring block should be avoided.

Ilioinguinal and iliohypogastric blocks provide effective analgesia after inguinal herniotomy and orchidopexy. The quality and duration of analgesia achieved by this block are comparable to caudal block.

A short bevelled 22 to 25 gauge needle is inserted, one patient's finger breadth medial to anterior superior iliac spine. After penetrating the external oblique aponeurosis and the internal oblique muscle fascia, a sudden loss of resistance is felt and the local anaesthetic can be deposited after a negative aspiration test. A dosage of 0.4 ml/kg of 0.25% bupivacaine with or without adrenaline is used for unilateral ilioinguinal and iliohypogastric nerve blocks. Another injection immediately lateral to pubic tubercle to block the nerves coming from the opposite side and local infiltration along the line of incision improve the quality of analgesia.

In about 50% patients the subcostal nerve accompanies the iliohypogastric nerve and may be responsible for inadequate pain relief. Therefore a more effective block can be achieved by an injection directed laterally to contact the inside wall of the ilium and infiltrating local anaesthetic as the needle is withdrawn slowly. For pain relief after orchidopexy ilioinguinal and iliohypogastric blocks must be combined with local infiltration of the scrotum. This is because the inferior aspect of the scrotum is innervated by the pudendal nerve.

Brachial plexus block may be used to provide postoperative analgesia following upper extremity surgery. The axillary approach is the safest, more reliable and most commonly used in children and may provide useful analgesia for operations below the elbow.

Positioning of the patient is very important to make the artery (which is surrounded by the nerve plexus) palpable. The child is placed supine and the arm is abducted to 90 degrees and rotated externally. The forearm is flexed to 90 degrees. A short bevelled needle is inserted perpendicular to skin at the most proximal part at which the artery can be palpated. The needle is advanced until a "fascial click" is felt. At this point arterial pulsation is usually transmitted to needle. These two signs indicate that the needle tip is within the fascial sheath. After a negative aspiration test, local anaesthetic agent may be injected. Bupivacaine 0.25%, 0.6ml/kg is usually adequate. A two point injection technique, i.e., one above and another below the artery improves the success rate.

Femoral nerve block and 3 in 1 blocks are indicated in day surgery to provide analgesia following skin grafting where the graft is taken from thigh and muscle biopsies. However due to the effect on the leg muscles, postoperative mobilisation is significantly affected which may delay discharge.

The femoral nerve is situated just lateral to the femoral artery below the inguinal ligament deep to the fascia lata and iliaca. Therefore when the needle is advanced, 2 losses of resistance must be felt. Usually 0.25% bupivacaine 0.3ml/kg is enough for adequate blocks of the femoral nerve (*see Update in Anaesthesia No. 11 2000*).

In a 3 in 1 block apart from the femoral nerve, the lateral cutaneous nerve of thigh and obturator nerve are also blocked. The volume of local anaesthetic should be doubled so that it can spread adequately between the iliacus fascia and muscle to reach the other nerves. Distal pressure on the femoral sheath during and after the injection improves the quality of nerve block.

Greater auricular nerve block. This nerve innervates most of the pinna and may be blocked to provide excellent analgesia after otoplasty. The block is performed by injecting 0.5% bupivacaine 1ml subcutaneously between the mastoid process and the descending ramus of the mandible.

Systemic Analgesics

Non-Steroidal Anti-Inflammatory Drugs (NSAIDs) along with local anaesthesia are the mainstay of postoperative pain relief in paediatric day case surgery. They have several advantages over opioid analgesics including a lack of respiratory depression and sedation. They do not cause nausea or vomiting.

NSAIDs have been found to be very effective analgesics in older children. However use of these agents are not recommended below one year of age due to the possibility of immature renal function and hepatic metabolism. Diclofenac, ibuprofen and ketorolac are the most commonly used agents. Administration of these agents before surgery as a premedicant provides optimal analgesia due to their anti-inflammatory activity.

Bronchospasm induced by NSAIDs is very rare in children and asthma is not a contraindication to the use of NSAIDs. However one should avoid them if the child has been recently or repeatedly hospitalised with asthma, or has required steroids systemically, or is known to be NSAID sensitive (Table 1).

Paracetamol (acetaminophen) is a very safe and effective analgesic in children including infants and neonates. Oral paracetamol 20mg/kg as a premedication is useful in achieving therapeutic plasma concentration postoperatively. The total daily dose of paracetamol can be up to 90mg/kg/day for the first 3 days in healthy children. This should be reduced to 60mg/kg/day in

Table 1: Doses of NSAIDs in Paediatric Patients

NSAID	Dose mg/kg	Maximum dose mg/kg/day
Ibuprofen	10	40
Diclofenac	1	3
Ketorolac	0.5	2
Naproxen	7.5	15
Indomethacin	1	3

Table 2: Dose of Paracetamol (Orally)

Loading dose	20 mg/kg
Maintenance dose	15 mg/kg
Maximum dose	90 mg/kg/day (Older children) 60 mg/kg/day (Neonates)

neonates (Table 2). The drug may be administered rectally but higher doses are necessary, due to poor and erratic absorption through the rectal mucosa.

Opioids are not ideal for paediatric day case surgery as they may produce ventilatory depression, excessive sedation and postoperative nausea and vomiting. With some procedures however opioids are required during and after surgery to control pain. Shorter acting opioids are ideal - fentanyl (1-2mcg/kg) is commonly used. Longer acting opioids (morphine / pethidine) may be required if postoperative pain is unexpectedly severe. Although the procedure may have been planned on a day case basis unexpected hospital admission may be required for control of severe pain.

Non Pharmacological Therapy may be helpful in some children. It includes distraction techniques like playing with toys, watching videos, music and hypnotic therapy. The child may be allowed to stay in a friendly atmosphere preferably with parents in the immediate postoperative period. All these measures reduce analgesic requirement and speeds recovery.

Conclusion

Postoperative pain following day case surgery in paediatric patients is usually not severe and diminishes within 3 to 5 days. Peripheral nerve blocks by local anaesthetic agents provide optimal analgesia in the immediate postoperative period. Patients should not be discharged until pain is well controlled with oral medications such as paracetamol, ibuprofen or diclofenac.

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INTRAOSSEROUS INFUSION

Eric Vreede, Anamaria Bulatovic, Peter Rosseel, and Xavier Lassalle, Medecins Sans Frontieres, France.

Key Points

- Intraosseous infusion is a temporary emergency measure
- Indicated in life-threatening situations when intravenous access fails (3 attempts or >90 seconds)
- Use the anteromedial aspect of the tibia
- Insert pointing caudal to avoid the epiphyseal growth plate
- Use an aseptic technique
- Crystalloids, colloids, blood products and drugs can be infused
- Remove as soon as the child has been resuscitated and intravenous access has been established

Introduction

The technique of intraosseous infusion was first described in humans in 1934 and it became increasingly popular in the 1940s. In recent years it has regained popularity especially in paediatric resuscitation. Unfortunately many doctors do not know this technique or do not employ it. However, intraosseous infusion is one of the quickest ways to establish access for the rapid infusion of fluids, drugs and blood products in emergency situations as well as for resuscitation. In many countries children are the victims of war trauma, road traffic accidents or severe dehydration and need good intravenous access, this technique can be life-saving. In these situations peripheral venous access can be difficult to obtain and alternatives such as central venous access can be difficult and/or dangerous.

Introduction to the technique

The marrow cavity is in continuity with the venous circulation and can therefore be used to infuse fluids and drugs, and to take blood samples for crossmatch, for example. The procedure must be performed under sterile conditions to avoid causing osteomyelitis. It is also recommended to limit the duration of the use of intraosseous infusion to a few hours until intravenous access is achieved. It is thus a temporary emergency measure. In experienced hands intraosseous access can be established within 1 minute.

It has been shown that the onset of action and drug levels during cardiopulmonary resuscitation using the intraosseous route are similar to those given intravenously.

Indications

Placement of an intraosseous needle is indicated when vascular access is needed in life-threatening situations in

babies, infants and children under the age of six years. It is indicated when attempts at venous access fail (three attempts or 90 seconds) or in cases where it is likely to fail and speed is of the essence. Although principally advocated for use in young children, it has been successfully used in older children where the iliac crest may also be used.

Contra-indications

- Femoral fracture on the ipsilateral side
- Do not use fractured bones
- Do not use bones with osteomyelitis

Equipment

1. Skin disinfectant
2. Local anaesthetic
3. 5 ml syringe
4. 50ml syringe
5. Intraosseous infusion needle or Jamshidi bone marrow needle. There are different needle sizes; 14, 16 and 18G. The 14 and 16G are usually used for children older than 18 months. However any size can be used for all ages.

It is possible but not ideal to use a 16 – 20G butterfly needle, spinal needle or even hypodermic needle. The chance that the needle gets blocked with bone marrow however, is much increased when not using a needle with a trochar.

Site

The best site to use is the flat anteromedial aspect of the tibia. The anterior aspect of the femur and the superior

iliac crest can also be used. The tibia is preferred since the anteromedial aspect of the bone lies just under the skin and can easily be identified. Avoid bones with osteomyelitis or fractures and do not use the tibia if the femur is fractured on the same side.

Technique

1. Palpate the tibial tuberosity. The site for cannulation lies 1 - 3cm below this tuberosity on the anteromedial surface of the tibia.
2. Use sterile gloves and an aseptic technique and a sterile needle.
3. Clean the skin. Placing a bone marrow needle without using a sterile technique obviously increases the chance of osteomyelitis and cellulitis.
4. Inject a small amount of local anaesthetic in the skin and continue to infiltrate down to the periosteum. When the child is unconscious it is not necessary to use local infiltration.
5. Flex the knee and put a sandbag as support behind the knee.
6. Hold the limb firmly above the site of insertion, usually at the level of the knee. Avoid putting your hand behind the site of insertion to avoid accidentally injuring your own hand.

7. Insert the intraosseous needle at 90 degrees to the skin (perpendicular) and slightly caudal (towards the foot) to avoid the epiphyseal growth plate.
8. Advance the needle using a drilling motion until a 'give' is felt – this occurs when the needle penetrates the cortex of the bone. Stop inserting further.
9. Remove the trochar. Confirm correct position by aspirating blood using the 5ml syringe. If no blood can be aspirated the needle may be blocked with marrow. To unblock the needle, slowly syringe in 10 ml of saline. Check that the limb does not swell up and that there is no increase in resistance.
10. If the tests are unsuccessful remove the needle and try the other leg.
11. Secure the needle in place with sterile gauze and strapping.

Correct placement is further confirmed by the following:

- A sudden loss of resistance on entering the marrow cavity (less obvious in infants who have soft bones).
- The needle remains upright without support (because infants have softer bones, the needle will not stand as firmly upright as in older children).
- Fluid flows freely through the needle without swelling of the subcutaneous tissue.

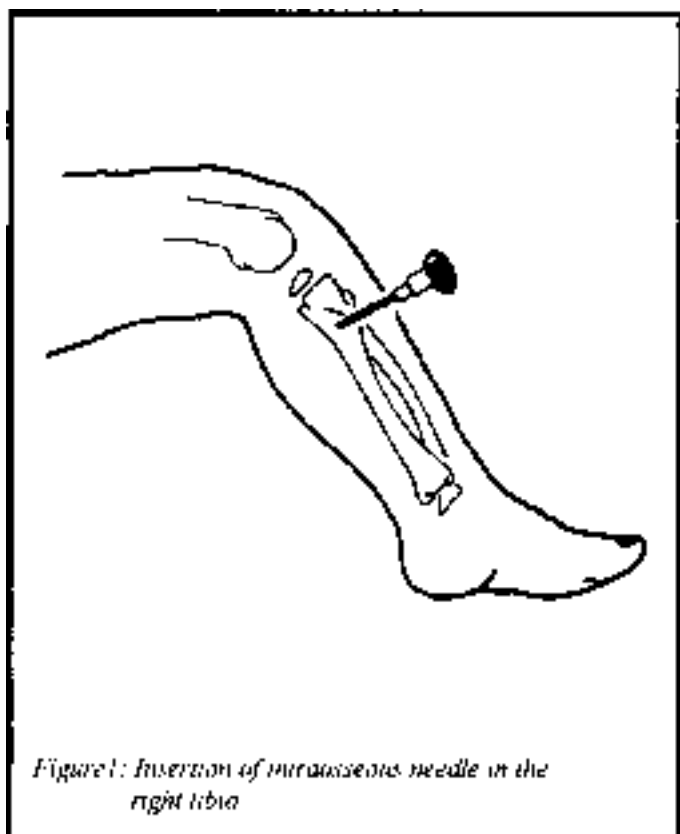
Complications

Important complications are tibial fracture especially in neonates, compartment syndrome, osteomyelitis and skin necrosis. When an aseptic technique is used, the incidence of osteomyelitis is less than 1%. Microscopic pulmonary fat and marrow emboli do not seem to be a clinical problem. Provided the correct technique is employed there does not seem to be any long-term effects on bone growth.

Infusion

Fluid can be infused under gentle pressure, manually by using a 50ml syringe or by inflating a blood-pressure cuff around the infusion bag. Crystalloids, blood products and drugs can be infused using this technique.

The intraosseous route should be replaced as soon as a normal vein can be cannulated and certainly within a few hours. The longer the period of use the greater the risk of complications.



Conclusion

In emergencies rapid intravenous access in children may be difficult to achieve. Intraosseous access is an easy, safe and life-saving alternative.

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The authors are members of the international medical aid organisation Médecins Sans Frontières (MSF).

Eric Vreede, Consultant Anaesthetist in London, anaesthetic advisor MSF Holland

Anamaria Bulatovic, attending Pediatrician in Baltimore, President of MSF USA

Peter Rosseel, Anaesthesiologist in Breda (Holland), Consultant Anaesthesiologist MSF Belgium

Xavier Lassalle, CRNA in Paris, anaesthetic advisor MSF France

ANSWERS - MULTIPLE CHOICE

1. TFFTF

Increasing HR will increase oxygen consumption. $CO = HR \times \text{Stroke Volume}$, and SV is proportional to preload, contractility and afterload. DM may cause an autonomic neuropathy, this can give rise to an abnormal Valsalva response. PA catheters assume a continuous column of blood from the catheter tip to the left ventri with no pressure gradients, in mitral stenosis there is a gradient between left atrium and the left ventricle.

2. FFTTF

In spontaneous ventilation the Mapleson A is extremely efficient and requires a FGF of approximately 70ml/kg/min. The Jackson Rees circuit has an open bag.

3. FFTTF

Soda lime: 94% calcium hydroxide, 5% sodium hydroxide and 1% potassium hydroxide, with a bit of silica. When fresh, soda lime contains 35% water. At the start of a case circle systems need to be denitrogenated with higher gas flows.

4. TFFFF

b) GCS 8. The patient in c) should have any haemodynamic instability resolved even if this requires laparotomy, i.e. ABC before all else. Avoid nasogastric tubes if there is a chance of skull fracture. Tension pneumothoracies should be decompressed as emergencies before an X-ray is taken.

5. FFTTF

The oculo-cardiac reflex is mediated by the parasympathetic nerve supply via the vagus. Normal IOP is 10-20mmHg.

Ketamine and suxamethonium are not ideal agents for use in induction in these cases but sometimes there are no alternatives e.g. patients with a full stomach.

6. FFFTT

Renal blood supply is 20% of CO. The juxtaglomerular complex produces renin, which via aldosterone promotes K^+ excretion in the distal tubule. ANP has an anti renin and anti angiotensin II effect as well as increasing GFR.

7. TTFFF

Maintenance fluid is 4/2/1mls/kg/hr respectively for the first, second and subsequent 10kgs of weight. Infants have a higher closing volume that encroaches upon tidal volumes. Alveolar MV is 100-150ml/kg/min due to higher oxygen demand.

8. TFTFT

Resuscitation fluid bolus is 20ml/kg. Children with pyloric stenosis should have their biochemistry and hydration corrected prior to surgery.

9. FFTTF

Patients with type II block should have a cardiological referral to consider pacing. Patients with chronic lung conditions are susceptible to hypoventilation and superadded infection, and where available an epidural could be an appropriate form of analgesia. MI patients should avoid all non-urgent surgery for >3months (ideally 6).

10. TTTTF

Other causes of EMD include; hypovolaemia, hypothermia, and electrolyte imbalance.

11. TFFTT

The second dose of adrenaline is 0.1ml/kg of 1:1000, and the resuscitation dose of atropine is 20mcg/kg.

12. TFFTF

Isoprenaline:	β_1 & β_2	
Dopamine	1-2mcg/kg/min:	D receptors
Dopamine	2-10mcg/kg/min:	D & β
Dopamine	>10mcg/kg/min:	D, β_1 & β_2
Adrenaline:		β_1 , β_1 & β_2 .

13. TFFTT

Ketamine induces dissociative anaesthesia by acting on the NMDA receptor hence its analgesic properties, however it takes greater than one arm brain circulation time to have its full effect. It is not a MH Trigger, unlike suxamethonium and the volatile agents.

14. TFFTT

Captopril is an angiotensin converting enzyme inhibitor hence it will decrease the amount of aldosterone present in the body (aldosterone normally promotes Na^+ retention). ADH will increase total body water not Na^+ . Fludrocortisone is a synthetic analogue of Aldosterone.

15. TFFTF

Ach binds to one of the two α subunits of the Ach receptor. The normal resting potential of the NMJ is -90mV.

16. TFFFF

The last three are all useful in the secondary management of anaphylaxis but will have no impact on the initial emergency.

17. FFFTT

MAOI's interact with opiates causing both excitatory and depressive effects, morphine is thought to be the most safe and pethidine the least. They also interact with indirectly acting sympathomimetic agents producing hypertensive crisis (directly acting drugs are thought to be safer).

18. TFFTT

Maximum dose of bupivacaine with adrenaline is the same as without adrenaline i.e. 2mg/kg.

Plain lignocaine can be used up to 3mg/kg.

19. TFFFF

The last 3 along with procaine are esters. They have a more frequent incidence of allergy and are metabolised by plasma and liver cholinesterases.

20. TFTTT

Mivcurium is metabolised by pseudocholinesterases hence deficiency of this enzyme will result in prolonged apnoea.

21. FTFTT

Nail varnish can absorb coloured light, carboxyhaemoglobin has a similar absorbance to oxyhaemoglobin and methaemoglobin has a similar

absorbance to deoxyhaemoglobin hence all interfering with pulse oximetry. Sickle cell and thalassaemia have no bearing on pulse oximetry.

Short Answers**Question 1**

- Tension Pneumothorax.
- Needle decompression in the 2nd intercostal space, mid clavicular line.
- Insertion of an intercostal drain with an underwater seal.
- There are a number of options as to the origin of the pathology however the most important issue is that the management is the same. There may have been an underlying subclinical pneumothorax, chest contusion is a risk factor for pneumothorax and complications of central line insertion include pneumothorax.

Question 2

- Narrow complex tachycardia.
- Vagotonic manoeuvres including, valsalva, carotid pressure and immersion in cold water.
- Adenosine causes transient complete a-v nodal block (for 5-10 seconds). It is given intravenously and is extremely effective in terminating supraventricular tachycardias. Adenosine has no effect in ventricular tachycardias and hence can also be used as a diagnostic tool.
- d) DC Cardioversion.

Question 3

- Ventricular tachycardia.
- The patient has cardiovascular compromise hence the treatment is DC cardioversion.

Question 4

- Atrial Fibrillation (AF)
- The causes of AF numerous however those that are likely to be significant in this case are anaesthetic drugs (both induction and maintenance agents), electrolyte imbalance (especially K^+) and underlying cardiac disease (especially ischaemic heart disease).
- Since the patient is now unconscious the obvious option is DC cardioversion. Correction of electrolyte disturbances is beneficial and if all this proves unsuccessful then chemical cardioversion can be attempted with drugs such as amiodarone and flecanide. Note digoxin is effective for rate control however will not cardiovert the patient into sinus rhythm.

RESPIRATORY PHYSIOLOGY

Dr Fred Roberts, Royal Devon & Exeter Hospitals, Exeter, UK.

INTRODUCTION

The main function of the lungs is to provide continuous gas exchange between inspired air and the blood in the pulmonary circulation, supplying oxygen and removing carbon dioxide, which is then cleared from the lungs by subsequent expiration. Survival is dependent upon this process being reliable, sustained and efficient, even when challenged by disease or an unfavourable environment. Evolutionary development has produced many complex mechanisms to achieve this, several of which are compromised by anaesthesia. A good understanding of respiratory physiology is therefore essential to ensure patient safety during anaesthesia.

ANATOMY

The respiratory tract extends from the mouth and nose to the alveoli. The upper airway serves to filter airborne particles, humidify and warm the inspired gases. The patency of the airway in the nose and oral cavity is largely maintained by the bony skeleton, but in the pharynx is dependent upon the tone in the muscles of the tongue, soft palate and pharyngeal walls.

Larynx

The larynx lies at the level of upper cervical vertebrae, C4-6, and its main structural components are the thyroid and cricoid cartilages, along with the smaller arytenoid cartilages and the epiglottis, which sit over the laryngeal inlet. A series of ligaments and muscles link these structures, which, by a co-ordinated sequence of actions, protect the larynx from solid or liquid material during swallowing as well as regulating vocal cord tension for phonation (speaking). The technique of cricoid pressure is based on the fact that the cricoid cartilage is a complete ring, which is used to compress the oesophagus behind it against the vertebral bodies of C5-6 to prevent regurgitation of gastric contents into the pharynx. The thyroid and cricoid cartilages are linked anteriorly by the cricothyroid membrane, through which access to the airway can be gained in an emergency.

Trachea and bronchi

The trachea extends from below the cricoid cartilage to the carina, the point where the trachea divides into the left and right main bronchus, with a length of 12-15cm in an adult and an internal diameter of 1.5-2.0cm. The carina

lies at the level of T5 (5th thoracic vertebra) at expiration and T6 in inspiration. Most of its circumference is made up of a series of C-shaped cartilages, but the trachealis muscle, which runs vertically, forms the posterior aspect.

When the trachea bifurcates, the right main bronchus is less sharply angled from the trachea than the left, making aspirated material more likely to enter the right lung. In addition, the right upper lobe bronchus arises only about 2.5cm from the carina and must be accommodated when designing right-sided endobronchial tubes.

Lungs and pleura

The right lung is divided into 3 lobes (upper, middle and lower) whereas the left has only 2 (upper and lower), with further division into the broncho-pulmonary segments (10 right, 9 left). In total there are up to 23 airway divisions between trachea and alveoli. The bronchial walls contain smooth muscle and elastic tissue as well as cartilage in the larger airways. Gas movement occurs by tidal flow in the large airways. In the small airways, by contrast, (division 17 and smaller) it results from diffusion only.

The pleura is a double layer surrounding the lungs, the visceral pleura enveloping the lung itself and the parietal pleura lining the thoracic cavity. Under normal circumstances the interpleural space between these layers contains only a tiny amount of lubricating fluid. The pleura and lungs extend from just above the clavicle down to the 8th rib anteriorly, the 10th rib laterally and the level of T12 posteriorly.

Blood supply

The lungs have a double blood supply, the *pulmonary circulation* for gas exchange with the alveoli and the *bronchial circulation* to supply the parenchyma (tissue) of the lung itself. Most of the blood from the bronchial circulation drains into the left side of the heart via the pulmonary veins and this deoxygenated blood makes up part of the normal physiological shunt present in the body. The other component of physiological shunt is from the thebesian veins, which drain some coronary blood directly into the chambers of the heart.

The pulmonary circulation is a low-pressure (25/10mmHg), low-resistance system with a capacity to accommodate a substantial increase in blood flowing through it without a

major increase in pressure. Vascular distension and recruitment of unperfused capillaries achieve this. The main stimulus which produces a marked increase in pulmonary vascular resistance is hypoxia.

MECHANISM OF BREATHING

A pressure gradient is required to generate flow. In spontaneous respiration inspiratory flow is achieved by creating a sub-atmospheric pressure in the alveoli (of the order of $-5\text{cmH}_2\text{O}$ during quiet breathing) by increasing the volume of the thoracic cavity under the action of the inspiratory muscles. During expiration the intra-alveolar pressure becomes slightly higher than atmospheric pressure and gas flow to the mouth results.

Motor pathways

The main muscle generating the negative intrathoracic pressure that produces inspiration is the *diaphragm*, a sheet separating the thorax from the abdomen. Its muscular part is peripheral, attached to the ribs and lumbar vertebrae, with a central tendon. Innervation is from the *phrenic nerves* (C3-5) with contraction moving the diaphragm downwards forcing the abdominal contents down and out. Additional inspiratory efforts are produced by the *external intercostal muscles* (innervated by their intercostal nerves T1-12) and the *accessory muscles of respiration* (sternomastoids and scalenes), although the latter only become important during exercise or respiratory distress.

During quiet breathing expiration is a passive process, relying on the elastic recoil of the lung and chest wall. When ventilation is increased, such as during exercise, expiration becomes active with contraction of the muscles of the abdominal wall and the internal intercostals. The same muscles are also used when producing a Valsalva manoeuvre (*Update in Anaesthesia, 1999, No. 10 p7*).

Central control

The mechanism by which respiration is controlled is complex. There is a group of *respiratory centres* located in the brainstem producing automatic breathing activity. This is then regulated mainly by input from *chemoreceptors* (see below). This control can be overridden by *voluntary control* from the cortex. Breath-holding, panting or sighing at will are examples of this voluntary control. The main respiratory centre is in the floor of the 4th ventricle, with inspiratory (dorsal) and expiratory (ventral) neurone groups. The inspiratory neurones fire automatically, but the expiratory ones are used only during forced expiration. The 2 other main

centres are the apneustic centre, which enhances inspiration, and the pneumotaxic centre, which terminates inspiration by inhibition of the dorsal neurone group above.

The *chemoreceptors* that regulate respiration are located both centrally and peripherally. Normally control is exercised by the central receptors located in the medulla, which respond to the CSF hydrogen ion concentration, in turn determined by CO_2 , which diffuses freely across the blood-brain barrier from the arterial blood. The response is both quick and sensitive to small changes in arterial CO_2 (PaCO_2). In addition, there are peripheral chemoreceptors located in the carotid and aortic bodies most of which respond to a fall in O_2 , but some also to a rise in arterial CO_2 . The degree of hypoxia required to produce significant activation of the O_2 receptors is such that they are not influential under normal circumstances, but will do so if profound hypoxia ($<8\text{kPa}$ or 60mmHg) occurs, for example at high altitude when breathing air (see later - Special circumstances). It also happens when the response to CO_2 is impaired, which can occur if the PaCO_2 is chronically elevated, leading to a blunting of the central receptor sensitivity. In this event the plasma bicarbonate (HCO_3^-) concentration will also be elevated.

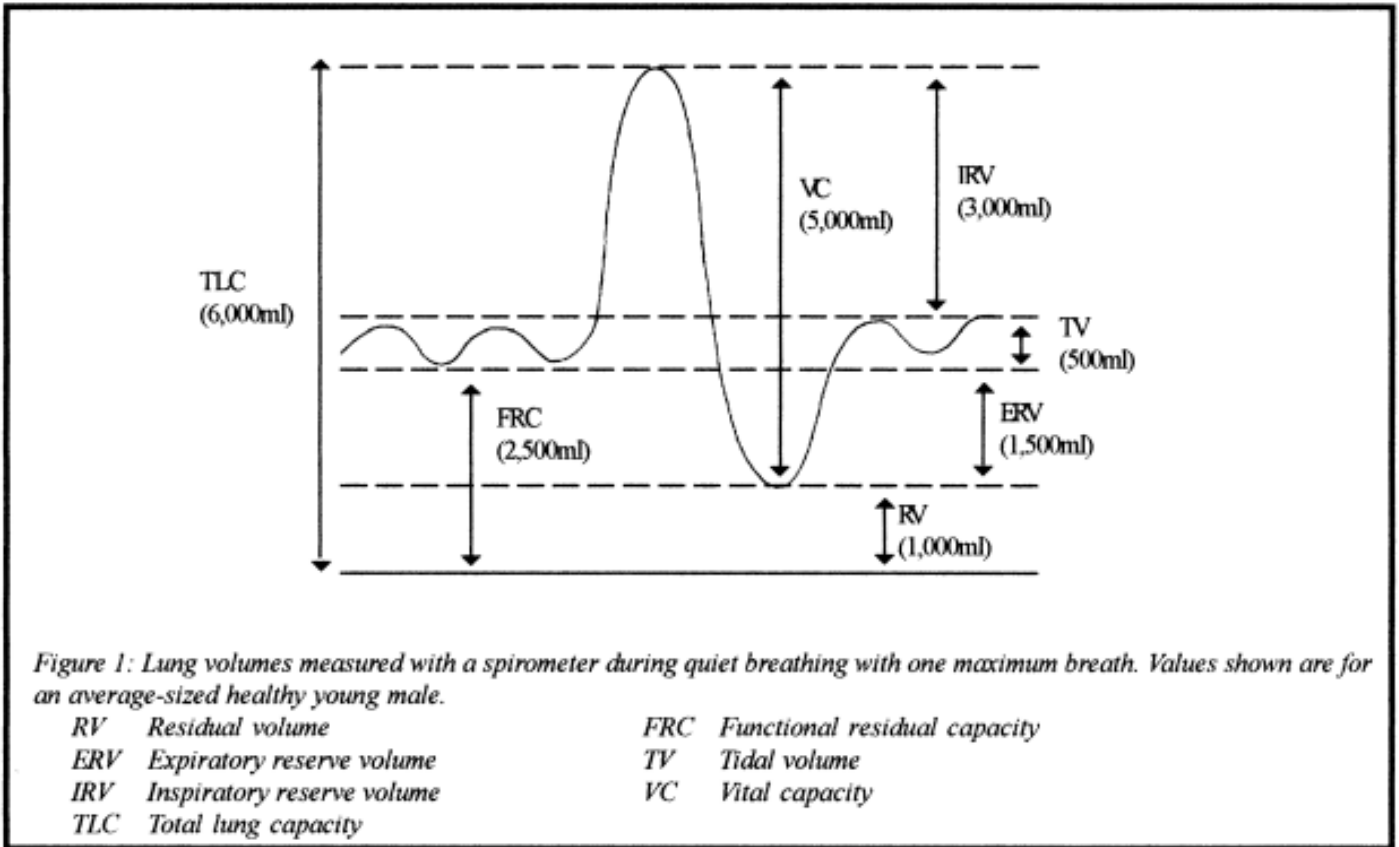
RESPIRATORY PROCESS

Respiratory values

The various terms used to describe lung excursion (movement) during quiet and maximal respiration are shown in figure 1:

The tidal volume (500ml) multiplied by the respiratory rate (14 breaths/min) is the *minute volume* (7,000ml/min). Not all of the tidal volume takes part in respiratory exchange, as this process does not start until the air or gas reaches the respiratory bronchioles (division 17 of the respiratory tree). Above this level the airways are solely for conducting, their volume being known as the *anatomical dead-space*. The volume of the anatomical dead-space is approximately 2ml/kg or 150ml in an adult, roughly a third of the tidal volume. The part of the tidal volume which does take part in respiratory exchange multiplied by the respiratory rate is known as the *alveolar ventilation* (approximately $5,000\text{ml/min}$).

Functional residual capacity (FRC) is the volume of air in the lungs at the end of a normal expiration. The point at which this occurs (and hence the FRC value) is determined by a balance between the inward elastic forces of the lung and the outward forces of the respiratory cage (mostly due to muscle tone). FRC falls with lying supine, obesity,



pregnancy and anaesthesia, though not with age. The FRC is of particular importance to anaesthetists because:

- During apnoea it is the reservoir to supply oxygen to the blood
- As it falls the distribution of ventilation within the lungs changes leading to mismatching with pulmonary blood flow
- If it falls below a certain volume (the closing capacity), airway closure occurs leading to shunt (see later - Ventilation/perfusion/shunt)

Resistance / compliance

In the absence of respiratory effort, the lung will come to lie at the point of the FRC. To move from this position and generate respiratory movement, two aspects need to be considered which oppose lung expansion and airflow and therefore need to be overcome by respiratory muscle activity. These are the airway resistance and the compliance of the lung and chest wall.

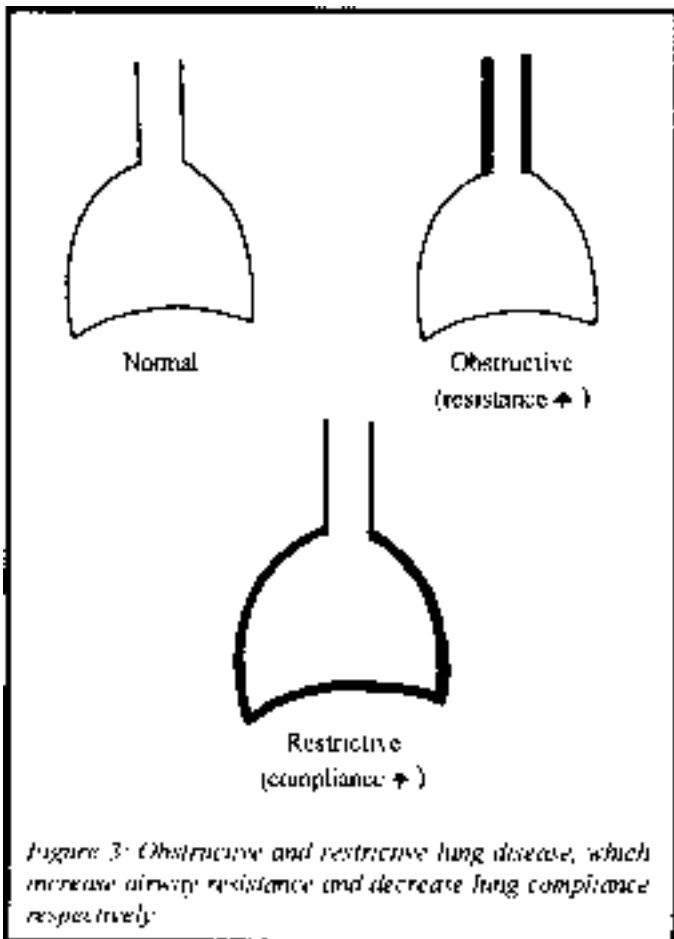
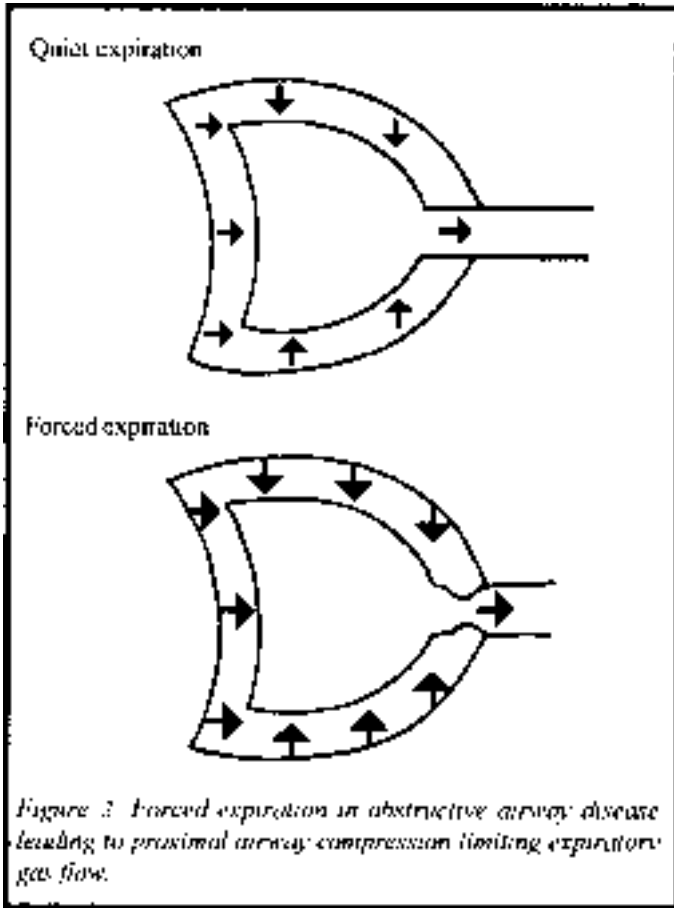
Resistance of the airways describes the obstruction to airflow provided by the conducting airways, resulting largely from the larger airways (down to division 6-7), plus a contribution from tissue resistance resulting produced by friction as tissues of the lung slide over each other

during respiration. An increase in resistance resulting from airway narrowing such as bronchospasm leads to obstructive airways disease.

Teaching point

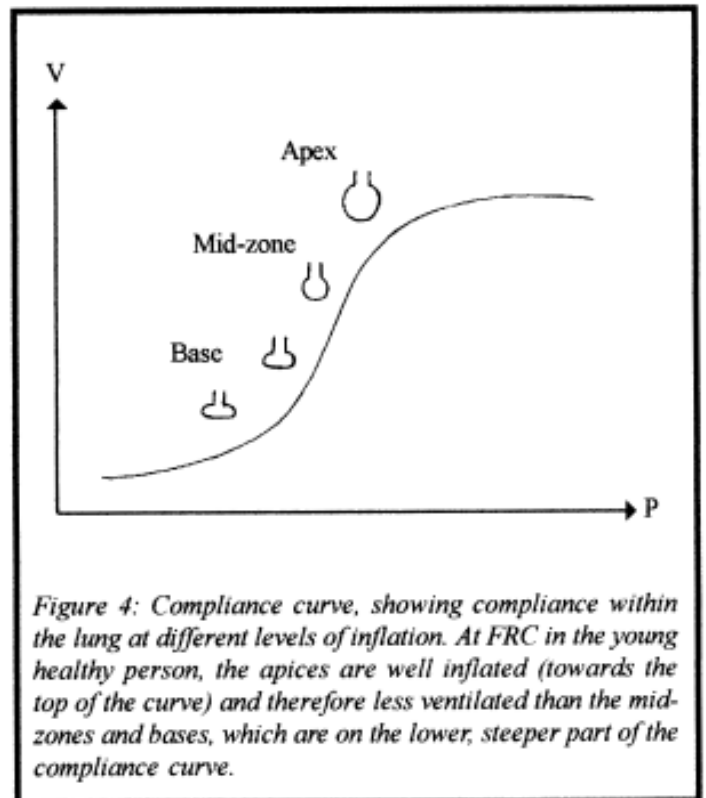
In obstructive airways disease, it might be expected that airflow could be improved by greater respiratory effort (increasing the pressure gradient) to overcome the increase in airways resistance. Whilst this is normally true for inspiration, it is not necessarily the case during expiration, as the increase in intrapleural pressure may act to compress airways proximal to the alveoli, leading to further obstruction with no increase in expiratory flow and air-trapping distally. This is shown in figure 2 and demonstrates why expiration is usually the major problem during an asthmatic attack.

Compliance denotes distensibility (stretchiness), and in a clinical setting refers to the lung and chest wall combined, being defined as the volume change per unit pressure change. When compliance is low, the lungs are stiffer and more effort is required to inflate



the alveoli. Conditions that worsen compliance, such as pulmonary fibrosis, produce *restrictive lung disease*.

Compliance also varies within the lung according to the degree of inflation, as shown in figure 4. Poor compliance is seen at low volumes (because of difficulty with initial lung inflation) and at high volumes (because of the limit of chest wall expansion), with best compliance in the mid-expansion range.

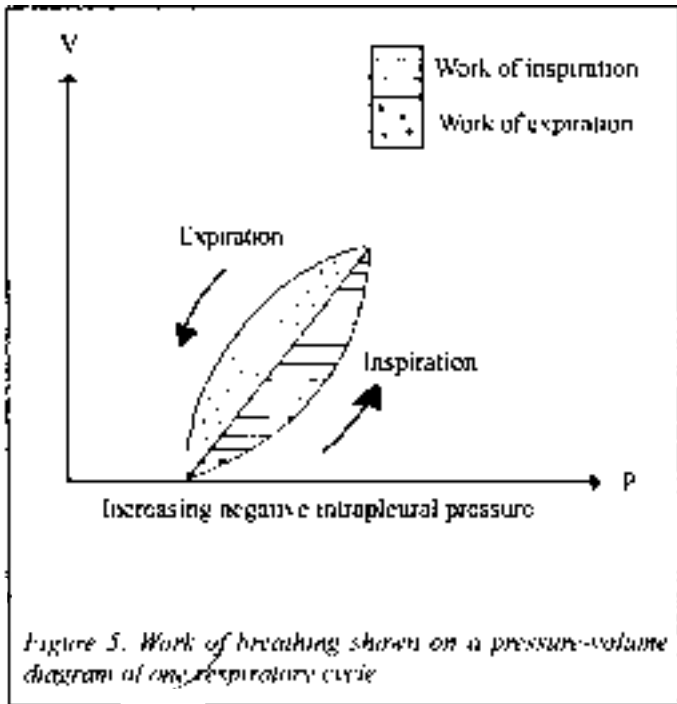


Work of breathing

Of the two barriers to respiration, airway resistance and lung compliance, it is only the first of these, which requires actual work to be done to overcome it. Airway resistance to flow is present during both inspiration and expiration and the energy required to overcome it, which represents the actual *work of breathing*, is dissipated as heat.

Although energy is required to overcome compliance in expanding the lung, it does not contribute to the actual work of breathing as it is not dissipated but converted to potential energy in the distended elastic tissues. Some of this stored energy is used to do the work of breathing produced by airways resistance during expiration.

The work of breathing is best displayed on a pressure-volume curve of one respiratory cycle (figure 5) which shows the different pathways for inspiration and expiration, known as *hysteresis*. The total work of breathing of the cycle is the area contained in the loop.



Teaching point

With high respiratory rates, faster airflow rates are required, increasing the frictional forces. This is more marked in obstructive airways disease - such patients therefore generally minimise the work of breathing by using a slow respiratory rate and large tidal volumes. In contrast, patients with restrictive lung disease (poor compliance) reach the unfavourable upper part of the compliance curve soon as the tidal volume increases. The pattern of breathing seen in such patients usually involves small tidal volumes and a fast respiratory rate.

Diffusion

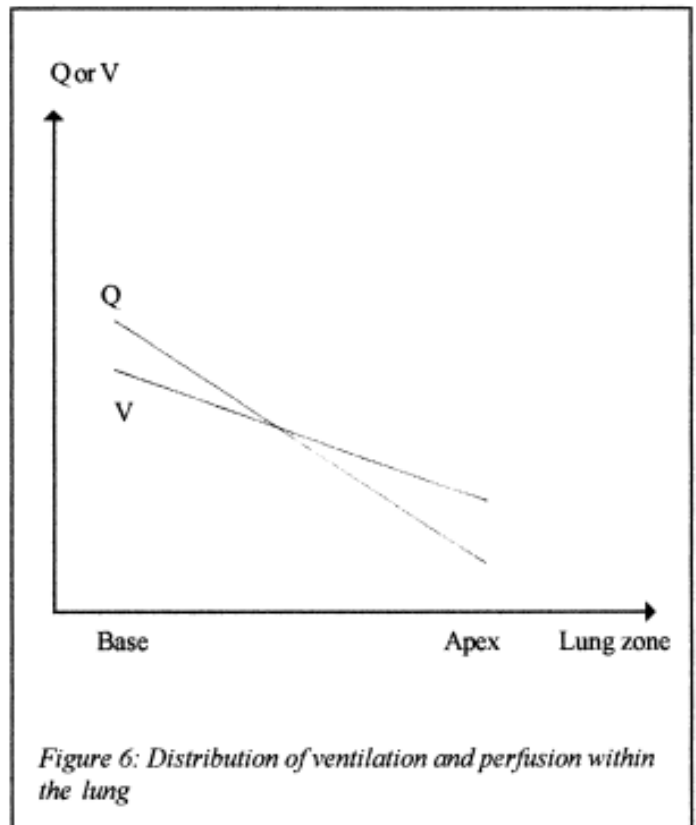
The alveoli provide an enormous surface area for gas exchange with pulmonary blood (between 50-100m²) with a thin membrane across which gases must diffuse. The solubility of oxygen is such that its diffusion across the normal alveolar-capillary membrane is an efficient and rapid process. Under resting conditions pulmonary capillary blood is in contact with the alveolus for about 0.75 second in total and is fully equilibrated with alveolar oxygen after only about a third of the way along this course. If lung disease is present which impairs diffusion there is therefore still usually sufficient time for full equilibration of oxygen when at rest. During exercise, however, the pulmonary blood flow is quicker, shortening the time available for gas exchange, and so those with lung disease are unable to

oxygenate the pulmonary blood fully and thus have a limited ability to exercise.

For carbon dioxide, which diffuses across the alveolar-capillary membrane 20 times faster than oxygen, the above factors are less liable to compromise transfer from blood to alveoli.

Ventilation / perfusion / shunt

In an ideal situation the ventilation delivered to an area of lung would be just sufficient to provide full exchange of oxygen and carbon dioxide with the blood perfusing that area. In the normal setting, whilst neither ventilation (V) nor perfusion (Q) is distributed evenly throughout the lung, their matching is fairly good, with the bases receiving substantially more of both than the apices (figure 6).



For *perfusion*, the distribution throughout the lung is largely due to the effects of gravity. Therefore in the upright position this means that the perfusion pressure at the base of the lung is equal to the mean pulmonary artery pressure (15mmHg or 20cmH₂O) plus the hydrostatic pressure between the main pulmonary artery and lung base (approximately 15cmH₂O). At the apices the hydrostatic pressure difference is subtracted from the pulmonary artery pressure with the result that the perfusion pressure is very low, and may at times even fall below the pressure in the alveoli leading to vessel compression and intermittent cessation of blood flow.

The distribution of *ventilation* across the lung is related to the position of each area on the compliance curve at the start of a normal tidal inspiration (the point of the FRC). Because the bases are on a more favourable part of the compliance curve than the apices, they gain more volume change from the pressure change applied and thus receive a greater degree of ventilation. Although the inequality between bases and apices is less marked for ventilation than for perfusion, overall there is still good V/Q matching and efficient oxygenation of blood passing through the lungs.

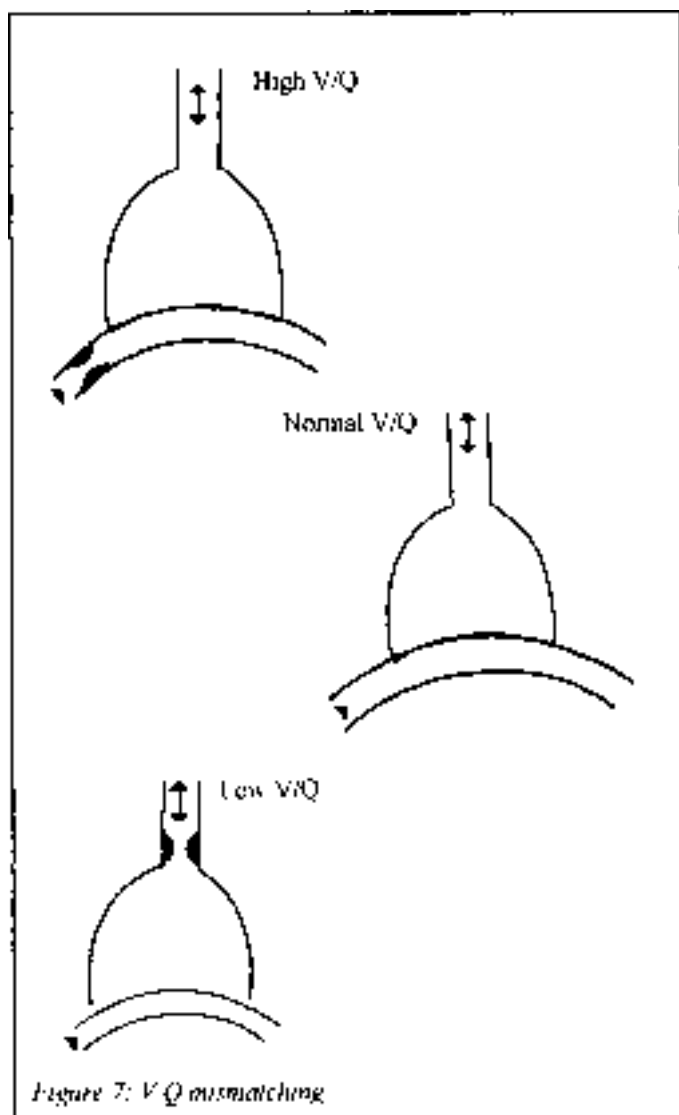
Disturbance of this distribution can lead to V/Q mismatching (figure 7). For an area of low V/Q ratio the blood flowing through it will be incompletely oxygenated, leading to a reduction in the oxygen level in arterial blood (hypoxaemia). Providing some ventilation is occurring in an area of low V/Q, the hypoxaemia can normally be corrected by increasing the FiO_2 , which restores the alveolar oxygen delivery to a level sufficient to oxygenate the blood fully.

V/Q mismatch occurs very commonly during anaesthesia because the FRC falls leading to a change in the position of the lung on the compliance curve. The apices, therefore, move to the most favourable part of the curve whilst the bases are located on a less favourable part at the bottom of the curve.

At the extremes of V/Q mismatch, an area of lung receiving no perfusion will have a V/Q ratio of ∞ (infinity) and is referred to as *alveolar dead-space*, which together with the anatomical dead-space makes up the *physiological dead-space*. Ventilating the dead space is, in effect, wasted ventilation, but unavoidable.

In contrast an area of lung receiving no ventilation, owing to airway closure or blockage, its V/Q ratio will be zero and the area is designated as shunt. Blood will emerge from an area of shunt with a PO_2 unchanged from the venous level (5.3kPa or 40mmHg) and produce marked arterial hypoxaemia. This hypoxaemia cannot be corrected by increasing the FiO_2 , even to 1.0, as the area of shunt receives no ventilation at all. The well-ventilated parts of the lung cannot compensate for the area of shunt because Hb is fully saturated at a normal PO_2 . Increasing the PO_2 of this blood will not increase the oxygen content substantially (*see Oxygen Carriage later*).

In the case of shunt, therefore, adequate oxygenation can only be re-established by restoring ventilation to these areas using measures such as physiotherapy, PEEP or CPAP, which clear blocked airways and reinflate areas of collapsed lung. Because closing capacity (CC) increases progressively with age, and is also higher in neonates, these patients are at particular risk during anaesthesia as the FRC may fall below CC and airway closure result.



Teaching point

A physiological mechanism exists which reduces the hypoxaemia resulting from areas of low V/Q ratio, by producing local vasoconstriction in these areas and diverting blood to other, better-ventilated parts of the lung. This effect, known as hypoxic pulmonary vasoconstriction (HPV), is mediated by unknown local factors. The protective action of HPV is, however, inhibited by various drugs, including inhalational anaesthetic agents.

Surfactant

Any liquid surface exhibits surface tension, a tendency for the molecules on the surface to pull together. This why, when water lies on a surface, it forms rounded droplets. If the surface tension is reduced, such as by adding a small amount of soap, the droplets collapse and the water becomes a thin film.

When a liquid surface is spherical, it acts to generate a pressure within the sphere according to Laplace's law:

$$P = \frac{4T}{R} \text{ if there are 2 liquid surfaces} \\ \text{(such as a bubble)}$$

$$P = \frac{2T}{R} \text{ if there is 1 liquid surface} \\ \text{(such as lining an alveolus)}$$

P	Pressure in sphere
R	Radius of sphere
T	Surface tension of liquid

The film of liquid lining the alveoli exhibits surface tension in such a manner to increase the pressure in the alveoli, with a greater rise in small alveoli than in large ones. Surfactant is a substance secreted by type II alveolar epithelial cells, which lowers the surface tension of this respiratory surface liquid markedly. Mainly consisting of a phospholipid (dipalmitoyl lecithin), its physiological benefits are:

- an increase (improvement) in overall lung compliance
- a reduction in the tendency for small alveoli to empty into large ones, leading to collapse
- a reduction in the fluid leak from pulmonary capillaries into the alveoli, as the surface tension forces act to increase the hydrostatic pressure gradient from capillary to alveolus

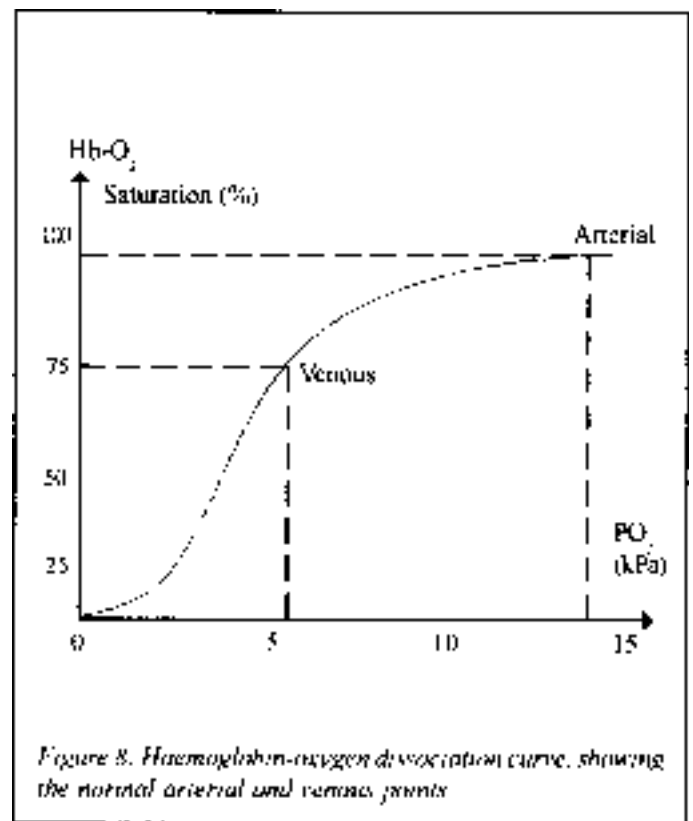
OXYGEN TRANSPORT

From an atmospheric level of 21kPa (21%), the partial pressure of oxygen falls in 3 stages before the arterial blood is reached. Firstly the inspired air is humidified by the upper respiratory tract, the saturated vapour pressure of water (6.2kPa or 47mmHg) reducing the PO₂ to around 19.7kPa (148mmHg) - *Update in Anaesthesia, 1999, No10, p.8.* In the alveoli the continuous exchange of carbon dioxide

for oxygen reduces the PO₂ to about 14.4kPa (108mmHg) and finally the small physiological shunt normally present reduces it to approximately 13.3kPa (100mmHg).

Oxygen carriage

After transfer of oxygen across the alveolar capillary membrane, an efficient carriage system is needed to transport it to the tissues for use in cellular respiration. The *oxygen content* in the blood is the sum of that bound to haemoglobin (Hb) and that dissolved in plasma, which is normally a minor contribution in patients breathing air. Hb is a large protein containing 4 subunits, each containing a ferrous (Fe²⁺) ion within a haem group. Up to 4 oxygen molecules can bind reversibly to each Hb molecule, one to each of the Fe²⁺ sites. The main factor that determines the extent of oxygen binding to Hb is the PO₂, the relationship between which is shown in figure 8.



The initial flat part of the curve occurs because the binding of the first oxygen molecule causes a small structural change to Hb facilitating the binding of subsequent oxygen molecules. The shape of the curve means that a fall in PO₂ from the normal arterial value will have little effect on the Hb saturation (and therefore oxygen content) until the steep part of the curve is reached, normally around 8kPa. Once the PO₂ has reached this level, however, a further decrease in PO₂ will result in a dramatic fall in the Hb saturation.

Several factors can change the affinity of Hb for oxygen, resulting in the curve moving to the right (acidosis, temperature \uparrow or 2,3-DPG \uparrow (2,3 diphosphoglycerate) or to the left (foetal Hb, alkalosis, temperature \downarrow or 2,3-DPG \downarrow). An index of the position of the Hb-O₂ dissociation curve is given by the P₅₀, the PO₂ at which Hb is 50% saturated.

Movement of the curve to the right decreases the affinity of Hb for oxygen. This is physiologically useful in the tissues, where the slightly acidic environment serves to improve oxygen unloading from the blood - the Bohr Effect. A left shift of the curve increases the affinity of Hb for oxygen, producing a higher saturation at a given PO₂. This acts to improve oxygen loading in the pulmonary capillary (slightly alkaline) and is greatly advantageous in the foetus, where the PO₂ is low (see later).

1g of Hb can carry 1.34ml of oxygen if fully saturated. At a PO₂ of 13.3kPa (100mmHg), Hb is normally about 97% saturated with oxygen. If the Hb concentration is 150gm/litre (15gm/100ml), arterial blood will therefore hold approximately 200ml/litre. With a cardiac output of 5 litre/min, the amount of oxygen available in the circulation is 1,000ml/min. Of this, approximately 250ml/min is used at rest, the Hb in venous blood being about 75% saturated.

The amount of oxygen dissolved in plasma is 0.23ml/litre/kPa (0.03ml/litre/mmHg). Whilst this is only about 3ml/litre when breathing air, it can be raised substantially by the use of hyperbaric pressure, reaching a level adequate to supply tissue requirements by breathing 100% oxygen at 3 atmospheres pressure. This can be used to sustain oxygenation if the patient's Hb is either insufficient or ineffective.

Special circumstances

It is useful to study the various specific physiological responses and adaptations which occur in response to changes in circumstances, in order to understand more clearly the different physiological mechanisms already described and the effects of anaesthesia and disease. These include:

Exercise

During exercise oxygen consumption can rise from 250 to over 3,000ml/min. Changes in response to this increased oxygen demand include:

- cardiac output \uparrow
- ventilation \uparrow
- extraction of oxygen from the blood \uparrow

Above a certain level, oxygen delivery cannot meet tissue demands, and anaerobic metabolism occurs, leading to lactic acid production.

Altitude

The acute response to the low arterial PO₂ resulting from high altitude is driven by the action of peripheral chemoreceptors to produce hyperventilation (as well as an increase in cardiac output). The resulting fall in the alveolar PCO₂ leads to an increase in the alveolar PO₂ (by the alveolar gas equation) which increases the arterial PO₂. The associated decrease in arterial PCO₂, however, reduces the drive at the central chemoreceptors, limiting the hyperventilation response. Metabolic compensation occurring over the next 2-3 days, involving an increase in renal HCO₃⁻ excretion and a subsequent fall in plasma and CSF HCO₃⁻, reduce this unwanted effect.

Later responses that improve oxygen carriage include:

- 2,3 DPG \uparrow , leading to a right shift of the dissociation curve
- polycythaemia

Foetus

Oxygenation of foetal blood comes from the maternal circulation via the placenta. Blood leaving the placenta in the umbilical vein has a PO₂ of only around 4.0kPa (30mmHg) and yet has an oxygen content of approximately 130ml/litre. The mechanisms by which this is achieved are:

- a left shift of the foetal Hb-O₂ dissociation curve, with a P₅₀ of 2.5kPa (19mmHg) [compared with a P₅₀ for adult Hb of 4.0kPa (30mmHg)]
- a raised Hb concentration (180gm/litre - 18gm/100ml - at term)

The increased Hb concentration increases the oxygen carrying capacity, whilst the left shift of the Hb-O₂ dissociation curve results in an increase in Hb affinity for oxygen (see earlier) and therefore a higher saturation at low PO₂.

Causes of hypoxia

Hypoxia indicates the situation where tissues are unable to undergo normal oxidative processes because of a failure in the supply or utilisation of oxygen. The causes of hypoxia can be grouped in to 4 categories:

Hypoxic hypoxia

Hypoxic hypoxia is defined as an inadequate PO₂ in arterial blood. This can result from an inadequate PO₂

in the inspired air (such as at altitude), major hypoventilation (from central or peripheral causes) or from inadequate alveolar-capillary transfer (such as shunt or V/Q mismatch).

Anaemic hypoxia

The oxygen content of arterial blood is almost all bound to Hb. In the presence of severe anaemia, the oxygen content will therefore fall in proportion to the reduction in Hb concentration, even though the PO₂ is normal. The normal compensatory mechanism to restore oxygen delivery is an increase in cardiac output, but when this can no longer be sustained tissue hypoxia results. Conditions in which Hb is rendered ineffective in binding oxygen, such as carbon monoxide poisoning, produce a reduction in oxygen carriage similar to anaemia.

Circulatory or stagnant hypoxia

If circulatory failure occurs, even though the oxygen content of arterial blood may be adequate, delivery to the tissues is not. Initially tissue oxygenation is maintained by increasing the degree of oxygen extraction from the blood, but as tissue perfusion worsens this becomes insufficient and tissue hypoxia develops.

Histotoxic hypoxia

This describes the situation where cellular metabolic processes are impaired to prevent oxygen utilisation by the cells, even though oxygen delivery to the tissues is normal. The best-known cause of histotoxic hypoxia is

cyanide poisoning, which inhibits cytochrome oxidase.

NON-RESPIRATORY LUNG FUNCTIONS

Whilst the main function of the lung is for respiratory gas exchange, it has several other important physiological roles.

These include:

- reservoir of blood available for circulatory compensation
- filter for circulation: thrombi, microaggregates etc
- metabolic activity: activation: angiotensin I \rightarrow II
inactivation: noradrenaline
bradykinin
5 H-T
some
prostaglandins
- immunological: IgA secretion into bronchial mucus

In summary, the article has outlined the many complex processes by which gas exchange in the body is maintained and regulated. With a fuller understanding of how these processes can be disturbed, the anaesthetist is better placed to manage the resulting problems logically and effectively. Readers are recommended to read this article with *The Physiology of Oxygen Delivery (Update in Anaesthesia, 1999, No. 10, pp 8-14)* and *Volatile Anaesthetic Agents (Update in Anaesthesia, 2000, No. 11, p79)*

ANAESTHESIA FOR THE PATIENT WITH RESPIRATORY DISEASE

Dr Michael Mercer, Bristol, UK and Sydney, Australia.

INTRODUCTION

Patients with respiratory disease have an increased chance of developing complications perioperatively. Most problems are seen postoperatively and are usually secondary to shallow breathing, poor lung expansion, basal lung collapse and subsequent infection. To minimise the risk of complications these patients should be identified preoperatively and their pulmonary function optimised. This involves physiotherapy, a review of all medications and may require the help of a respiratory physician. Elective surgery is postponed until the patient is ready.

In the general surgical population thoracic and upper abdominal procedures are associated with the highest risk (10-40%) of pulmonary complications. The benefits of the proposed surgery must therefore be weighed against the risks involved.

GENERAL CONSIDERATIONS

General health status

The American Society of Anesthesiologists classification (1 to 5) correlates well with the risk of post-operative pulmonary complications. Poor exercise tolerance also predicts those at risk.

Smoking

Active and passive smokers have hyper-reactive airways with poor muco-ciliary clearance of secretions. They are at increased risk of perioperative respiratory complications, such as atelectasis or pneumonia. It takes 8 weeks abstinence for this risk to diminish.

Even abstinence for the 12 hours before anaesthesia will allow time for clearance of nicotine, a coronary vasoconstrictor, and a fall in the levels of carboxyhaemoglobin thus improving oxygen carriage in the blood.

Obesity

The normal range for BMI (Body Mass Index - defined as weight (Kg) divided by the square of the height (m) is 22-28. Over 35 is morbidly obese. Normal weight (Kg) is height (cm) minus 100 for males, or height minus 105 for females.

Obese patients may present a difficult intubation and have perioperative basal lung collapse leading to postoperative hypoxia. A history of sleep apnoea may lead to post-operative airway compromise. If practical obese patients should lose weight preoperatively, and

co-existent diabetes and hypertension stabilised.

Physiotherapy

Teaching patients in the preoperative period to participate with techniques to mobilise secretions and increase lung volumes in the postoperative period will reduce pulmonary complications. Methods employed are early mobilisation, coughing, deep breathing, chest percussion and vibration together with postural drainage.

Pain Relief

Effective analgesia is important as it allows deep breathing and coughing and mobilisation. This helps prevent secretion retention and lung collapse, and reduces the incidence of postoperative pneumonia. Epidurals appear particularly good at this for abdominal and thoracic surgical procedures, although they are not available everywhere (*see Epidural analgesia section*).

The method of postoperative analgesia should always be discussed with the patient before surgery.

Effects of General Anaesthesia

These are relatively minor and do not persist beyond 24 hours. However, they may tip a patient with limited respiratory reserve into respiratory failure.

- Manipulation of the airway (laryngoscopy and intubation) and surgical stimulation may precipitate laryngeal or bronchial spasm.
- Endotracheal intubation bypasses the filtering, humidifying and warming functions of the upper airway allowing the entry of pathogens and the drying of secretions. Adequate humidification and warming of the anaesthetic gases with a Heat and Moisture Exchanger (HME) is ideal.
- Volatile anaesthetic agents depress the respiratory response to hypoxia and hypercapnia, and the ability to clear secretions is reduced. Functional residual capacity (FRC) decreases and pulmonary shunt increases; these are unfavourable changes leading to hypoxia and occur especially in lithotomy and head-down positions, and in the obese.
- Intermittent positive pressure ventilation causes an imbalance in ventilation and perfusion matching in the lung, and necessitates an increase in the inspired oxygen concentration.

- Excessive fluid therapy can result in pulmonary oedema in patients with cardiac failure.
- Neuromuscular blockade is reversed before extubation. In the recovery room residual effects of anaesthesia depress upper airway muscular tone, and airway obstruction may occur.

Anaesthetic Drugs

- The intravenous induction agents thiopentone, propofol and etomidate produce an initial transient apnoea. Ketamine preserves respiratory drive and is better at maintaining the airway, although secretions increase.
- Thiopentone increases airway reactivity.
- Volatile anaesthetics depress respiratory drive in decreasing order as follows:
Enflurane>Desflurane>Isoflurane>Sevoflurane>Halothane.
Ether however stimulates respiratory drive and increases minute ventilation. It is, however, irritant to the airway, stimulates saliva production and may induce coughing.
- Atracurium and tubocurarine release histamine and may result in bronchospasm. They are best avoided in asthma.
- Opioid drugs and benzodiazepines depress respiratory drive and response to hypoxia and hypercapnia. Morphine may result in histamine release and occasionally bronchospasm. Non-steroidal anti-inflammatory drugs (NSAIDs) may exacerbate asthma. Pethidine is a useful alternative analgesic for asthmatics.

Effects of Surgery

- To immobilise upper abdominal and thoracic incisions and limit pain, patients splint these areas postoperatively with their intercostal and diaphragmatic muscles. This limits their ability to take deep breaths and increases the risk of postoperative pulmonary complications. Surgery on the limbs, lower abdomen or body surface surgery has less effect.
- A laparotomy may remove fluid or masses that cause diaphragmatic splinting and respiratory difficulty. However, gas (especially nitrous oxide) and fluid may accumulate within the bowel and peritoneal cavity exacerbating post-operative distension and splinting.
- Surgery lasting more than 3 hours is associated with a higher risk of pulmonary complications.

- Postoperatively, return of lung function to normal may take one to two weeks.

PREOPERATIVE PREPARATION

General assessment

This involves history, examination and investigation.

History. Ask about symptoms of wheeze, cough, sputum production, haemoptysis, chest pain, exercise tolerance, orthopnoea and paroxysmal nocturnal dyspnoea. The diagnosis of chronic chest complaints such as asthma or bronchiectasis is often known. Present medication and allergies are noted, and a history of smoking sought. Previous anaesthetic records may be available and can help in planning care.

Examination. Inspect for cyanosis, dyspnoea, respiratory rate, asymmetry of chest wall movement, scars, cough and sputum colour. Percussion and auscultation of chest may suggest areas of collapse and consolidation, pleural effusions, pulmonary oedema or infection. Cor pulmonale may be evident as peripheral oedema and raised jugular venous pressure. A bounding pulse and hand flap may indicate carbon dioxide retention, and enlarged lymph nodes in the neck may suggest lung cancer.

Investigations. Leucocytosis may indicate active infection, and polycythaemia chronic hypoxaemia. Arterial blood gases should be performed in patients who are dyspnoeic with minimal exertion and the results interpreted in relation to the inspired oxygen concentration. Preoperative hypoxia or carbon dioxide retention indicates the possibility of postoperative respiratory failure which may require a period of assisted ventilation on the Intensive Care Unit.

Pulmonary function tests, if available, provide baseline pre-operative measurements. The chest clinic will have charts to compare these results against those predicted for the patients age, sex and weight. The results are also compared against the patient's previous records to assess current disease control.

- FEV1.0 (Forced Expiratory Volume in 1 second) and FVC (Forced Vital Capacity) are commonly measured. A reduction in the FEV1.0:FVC ratio indicates obstructive airways disease. (The normal is 0.75 (75%) or more). A reduction in FVC occurs in restrictive lung disease.
- An FEV1.0 or FVC less than 70% of predicted, or an FEV1.0:FVC ratio less than 65%, is associated with an increased risk of pulmonary complications.

Chest X-rays may confirm effusions, collapse and consolidation, active infection, pulmonary oedema, or the hyperinflated lung fields of emphysema.

An electrocardiogram may indicate P-pulmonale, a right ventricular strain pattern (dominant R waves in the septal leads) or right bundle branch block.

Pre-medication

In patients with poor respiratory function premedication (if used) must not cause respiratory depression. Opiates and benzodiazepines can both do this, and are best avoided if possible, or used with caution. Humidified oxygen may be administered (*see Oxygen therapy section*).

Anticholinergic drugs (e.g. atropine) may dry airway secretions and may be helpful before ketamine or ether.

Specific Respiratory Problems

Coryza (common cold)

Most patients with minor upper respiratory infections but without fever or productive cough can have elective surgery. However, patients with underlying respiratory disease or those having major abdominal or thoracic surgery should be postponed.

Respiratory tract infections

Patients with fever and productive cough should be treated before undergoing elective surgery as there is an increased risk of postoperative pulmonary complications. When these patients present for emergency surgery a course of antibiotics should be administered.

Asthma

Asthma causes hyper-responsive airways with oedema, inflammation and narrowing due to smooth muscle spasm. It is characteristically reversible, unlike chronic obstructive pulmonary disease. Elective cases should not be undertaken unless asthma is well controlled, and the anaesthetist will need to be informed of poorly controlled and severe asthmatics in advance. A consultation with a respiratory physician may be useful. In poorly controlled asthma a short course of steroids is often effective in improving control of the disease. Patients on preoperative steroids will need extra perioperative supplementation if they are taking more than the equivalent of 10mg of prednisolone a day.

Preoperative assessment

- The disease is assessed by the frequency and severity of attacks, including hospital and intensive

care admissions, and by drug history. The patient will be able to say how good (or bad) their asthma is. Examination may reveal expiratory wheezes, use of accessory muscles or an over-distended chest. Peak expiratory flow rates (PEFR) pre- and post-bronchodilator should be measured, although trends in PEFR are more useful (the patient may have their own PEFR records). Baseline spirometry, (FEV1.0 and FEV1.0:FVC ratio) is also helpful.

- Blood gas analysis is usually reserved for severe disease (breathlessness on minimal exertion).
- Before surgery, patients should be free of wheeze, with a PEFR greater than 80% of the predicted or personal best value. Severe asthmatics may require their inhalers being changed to nebulisers. Similarly inhaled steroid dose may have to be increased or oral steroids commenced (Prednisolone 20-40mg daily) one week prior to surgery - consult a respiratory physician early.

Perioperative management

- Consider converting inhaled beta 2 agonists such as salbutamol to the nebulised form. Give nebulised salbutamol (2.5-5.0mg) with premedication.
- Avoid aspirin or NSAIDs and any other allergens known to the patient. If applicable local or regional anaesthesia used alone will avoid the problems of general anaesthesia. However, if general anaesthesia is required, the addition of regional techniques can reduce operative volatile anaesthetic and post operative opioid requirements and the likelihood of respiratory complications.
- Ketamine and all the volatile agents are bronchodilators. Airway manipulation should be kept to a minimum and take place only under adequate anaesthesia.
- Controlled ventilation with the use of neuromuscular blocking drugs will be needed for major or long procedures. In cases with severe airways obstruction the ventilator rate may have to be sufficiently low to allow the slow expiration of asthma. Atracurium and tubocurare should be avoided as they release histamine. This is also true of morphine - pethidine is often preferred in patients who are wheezy on presentation.
- Residual neuromuscular blockade must be fully reversed, and extubation can occur when

spontaneous ventilation is resumed and oxygenation is adequate.

Postoperative care

- Adequate analgesia is vital.
- Humidified oxygen is continued for up to 72 hours following major abdominal or thoracic surgery (*see Oxygen therapy section*), together with regular physiotherapy until the patient regains mobility.
- Maintenance of hydration with intravenous fluids is required until oral intake is sufficient.
- Usual anti-asthmatic medications are resumed immediately. This may require intravenous steroids to temporarily replace oral (*see Steroid supplementation section*) and nebulised bronchodilators to replace inhalers if the patient cannot take a deep breath, or pulmonary function has deteriorated after surgery.
- Failure to ensure adequate postoperative oxygenation and ventilation may require admission to an intensive care area for a period of assisted ventilation.

Chronic obstructive pulmonary disease (COPD)

The main problems are airflow obstruction (usually irreversible), mucus hypersecretion and repeated infections. The ASA grade correlates with the risk of postoperative pulmonary problems.

If reversibility is demonstrated by spirometry (i.e. an increase in FEV_{1.0}:FVC ratio after bronchodilator), it is treated as for asthma. A trial of a week's course of systemic steroids (Prednisolone 20-40mg daily) is used if nebulisers fail to treat wheeze. Antibiotics are only used if a change in sputum colour suggests active infection. Right and left ventricular failure is treated with diuretics. Physiotherapy will clear chest secretions and the patient is encouraged to stop smoking.

Preoperative arterial blood gas estimation is required in the patient who has difficulty climbing one flight of stairs, or who has cor pulmonale. Postoperatively, these patients may need ventilating for 1-2 days on an intensive care unit following thoracic or high abdominal surgery. The best predictor of the need for postoperative ventilation is the arterial PaO₂, and whether the patient is dyspnoeic at rest.

Otherwise, perioperative considerations are the same as for asthma, except that the chances of post-operative pneumonia (pyrexia, purulent sputum) are high and

will require early treatment with amoxycillin, trimethoprim or clarithromycin.

Postoperatively, care is required with oxygen supplementation as some COPD patients rely on relative hypoxia for respiratory drive. (*see Oxygen therapy section*).

Restrictive pulmonary disease

Restrictive disease is either intrinsic, such as pulmonary fibrosis related to rheumatoid arthritis or asbestosis, or extrinsic, such as caused by kyphoscoliosis or obesity. Oxygenation may be impaired at the alveolar level and because of poor air supply to it. Steroids are the usual treatment for fibrotic disease.

Intrinsic Disease

- The anaesthetist should be alerted early. Preoperatively obtain spirometry, arterial blood gases, lung volume and gas transfer measurements, if not done in the previous 8 weeks. A reduced PaO₂ indicates severe disease. The chest physician may suggest an increase in steroid dose.
- Steroid supplementation will be required over the operative period (*see Steroid supplementation section*).
- Postoperatively, supplemental oxygen is given to keep SpO₂>92%, and respiratory infection is treated early.

Extrinsic Disease

- The restrictive deficit here leads to rapid, shallow breathing, often relying on diaphragmatic movement to be effective. This poses problems for breathing and sputum clearance postoperatively, especially following thoracic or upper abdominal incisions.
- Blood gases remain normal until disease is severe and PaCO₂ rises.
- Postoperatively, vigorous physiotherapy and adequate analgesia are vital. The patient may require ICU or HDU care if postoperative hypoxia, fatigue or carbon dioxide narcosis occur.

Bronchiectasis and Cystic Fibrosis

Prior to surgery therapy is maximised using a course of intravenous antibiotics, physiotherapy, nebulised bronchodilators and an extra 5-10mg/day of oral prednisolone, if taking long term steroids. This involves discussion with the patient's chest physician. Elective surgery is postponed if respiratory symptoms are present.

Postoperatively continue intravenous antibiotics and regular physiotherapy until discharge. The chest physician should be involved in any respiratory problems, and adequate nutrition is resumed as early as possible.

Tuberculosis

The patient with active pulmonary tuberculosis may be wasted, febrile and dehydrated. Production of sputum and haemoptysis may cause segmental lung collapse and blockage of the endotracheal tube. Humidification of anaesthetic breathing systems is therefore important, and inspired oxygen concentration will have to be increased. Appropriate intravenous fluids are given to rehydrate the patient. *Anaesthetic equipment must be sterilised after use* to prevent cross infection of tuberculosis to other patients.

ANAESTHESIA - TECHNIQUES

Perioperatively, continuous clinical observation of the patient is combined with monitoring appropriate to the case being undertaken. Hence, the patient's colour and respiratory rate and pattern is observed, and the pulse volume and rate palpated (during anaesthesia it may be easier to palpate the facial, superficial temporal or carotid artery). Monitoring involves pulse oximetry, electrocardiogram, non-invasive blood pressure recordings and, if available, end-tidal carbon dioxide measurement.

A preoperative pulse oximeter measurement of peripheral oxygen saturation *in air* is useful, and the perioperative inspired oxygen concentration must be sufficient to maintain this. Those patients at greatest risk of perioperative pulmonary complications will benefit from regular blood gas analysis using an indwelling arterial catheter.

The technique of anaesthesia chosen is the one considered to carry the lowest risk of perioperative pulmonary complications. The following points should be considered:

- **Regional anaesthesia** will avoid the pulmonary complications of general anaesthesia, but its use is limited by the duration of local anaesthetic activity, and to certain areas of the body, i.e. face, eyes and limbs.
- **Spinal/Epidural anaesthesia.** High spinal and epidural techniques impair intercostal muscle function and result in a decrease in FRC and an increased risk of perioperative basal atelectasis and hypoxia. There is no clear evidence that these techniques result in fewer respiratory complications than after general anaesthesia, although avoiding tracheal intubation may decrease the risk of postoperative bronchospasm.
- Low spinal and epidural techniques can be used for surgery below the umbilicus and on lower limbs without pulmonary impairment. However, under general anaesthesia, this kind of surgery has a low risk of pulmonary complications. As such there is little to choose between these two techniques. When planning to use spinal or epidural anaesthesia ensure that the patient will be able to lie flat for an extended period.
- **Ketamine anaesthesia** maintains some of the airway and cough reflex. Ventilation is not depressed, but there is an increase in salivation such that atropine premedication is required. Without muscular relaxation and endotracheal intubation, the airway remains vulnerable to aspiration of vomited or regurgitated gastric contents. Ketamine as the sole anaesthetic agent is therefore not used in the patient with a full stomach, and is most commonly used for surgery of the face, limbs or perineum, where muscular relaxation is not required.
- **Controlled ventilation.** Endotracheal intubation using muscle relaxants and controlled ventilation will be necessary during head, neck and ear, nose and throat surgery. Here the airway must be secured at the beginning of surgery as access to it will be difficult when surgery has commenced. Also, the trachea is at risk of soiling from blood. Similarly, during thoracic or abdominal surgery muscle relaxation will be required to enable controlled ventilation, and endotracheal intubation will protect against tracheal aspiration of gut contents. When patients require surgery in the prone position airway access will be difficult, and endotracheal intubation and controlled ventilation will be required.
- **Spontaneous ventilation** employing a facemask avoids airway instrumentation, although leaves the airway unprotected. If available, the laryngeal mask does not stimulate the larynx, but does offer some protection for the trachea. This technique is only used for minor surgery of the limbs or body surface of short duration (less than 2 hours), when the patient can breath comfortably in the supine position or on their side. Prolongation of spontaneous ventilation anaesthesia may lead to respiratory depression and delayed recovery.

POSTOPERATIVE CARE

The patient with underlying respiratory disease is at increased risk of postoperative pulmonary complications. This is particularly so in smokers, or after upper abdominal or thoracic surgery. The airway is vulnerable for up to 24 hours, and hypoventilation can occur for up to 3 days.

In the recovery room, the airway is kept patent, and adequate ventilation and oxygenation ensured before discharge to the ward. All neuromuscular paralysis must be reversed. If there are problems the patient will need to go to the HDU or ICU. Otherwise, the patient is kept warm and well hydrated, and fluid balance charts commenced.

Oxygen therapy

- Following minor surgery hypoxia may occur for the first hour or two after surgery. Oxygen should therefore administered until the patient is fully awake and recovered from the anaesthetic.
- After major surgery hypoxia can occur for up to for 3 days, particularly at night. This tendency is worse in patients who are receiving opioids by any route. This can put the patient with chest disease or ischaemic heart disease, at risk. Oxygen (2-4L/min by nasal cannulae) should be given over this period to those at risk.
- Some patients with very severe COPD depend on hypoxia to maintain ventilatory drive. It is difficult to diagnose but all affected patients will have had severe COPD for many years. This situation is very unusual, particularly in the developing world and hypoxia should always be treated immediately. If a patient like this presents, the inspired oxygen concentration should be monitored and repeat arterial blood gas estimations carried out to guide the level of oxygen treatment tolerated by the patient (*see Acute Oxygen Treatment*).

Pain Relief

Effective analgesia reduces the incidence of postoperative respiratory complications. Opioids may be required for 48-72 hours after major surgery.

- Intravenous opioid boluses can be titrated against pain in the recovery room, whilst observing for respiratory depression. This can be used as a guide to the dose of intramuscular opioid that may be given in relative safety on the ward. Continuous infusions of intravenous opioids demand very close supervision, as the risk of respiratory depression is significant.

- The combination of opioids with rectal or oral drugs, such as paracetamol or non-steroidal anti-inflammatory agents (NSAIDs), gives particularly good analgesia whilst reducing opioid side effects.
- If available, patient controlled analgesia (PCA) systems allow the patient to titrate opioid requirements to their pain. They give good quality analgesia and a fixed lockout period should prevent respiratory depression.

Epidural analgesia

The use of postoperative thoracic epidural analgesia for abdominal and thoracic surgical procedures results in good quality analgesia, with few respiratory complications. However, there is no clear evidence to demonstrate its superiority over other analgesic techniques, and it is not available everywhere.

The combination of low concentrations of local anaesthetic with low doses of opioids in an epidural infusion results in the best analgesia for the fewest side effects (i.e. opioid induced respiratory depression and local anaesthetic toxicity). For example give 50ml of 0.167% bupivacaine containing 5mg diamorphine given as an epidural infusion at a rate of 0-8ml/hr.

Whilst postoperative opioid analgesia is administered the patient must be observed in an area with sufficient medical and nursing skills, and staffing levels. Complications that may arise are then noted promptly and the appropriate help called. Regular observations are made of blood pressure, pulse, oxygenation (using a pulse oximeter), respiratory rate, conscious level and analgesia. Protocols may be used to guide nursing staff in the administration of analgesia, the observations required and when to call for help.

Physiotherapy

Teaching patients in the preoperative period to participate with techniques to mobilise secretions and increase lung volumes in the postoperative period, will reduce pulmonary complications. Methods employed are coughing, deep breathing, early mobilisation, and chest percussion and vibration together with postural drainage.

Steroid supplementation

The brittle asthmatic or severe COPD patient may benefit from a course of prednisolone (20-40mg daily) during the week before surgery. Seek the advice of the patient's chest physician.

Patients who have received a course of steroids in the 6 months before surgery, or who are on maintenance

therapy of greater than 10mg of prednisolone a day, are presumed to have adreno-cortical suppression. Perioperative steroid supplementation will be required.

- Intravenous hydrocortisone 100mg 8 hourly is given starting with the premedication. Over the next 5 days this is tapered to their normal daily dose, where 100mg intravenous hydrocortisone is equivalent to 25mg oral prednisolone.
- Intravenous steroids must replace oral whilst these cannot be taken.

Prophylaxis of venous thromboembolism

Prophylactic measures should begin before surgery in those at risk and continued until early mobilisation reduces the risk of postoperative deep vein thrombosis and pulmonary embolism. Regular subcutaneous heparin and anti-embolism stockings are commonly used.

Nutrition

Patients with severe respiratory disease are often malnourished and weak. This is associated with increased risk of postoperative infection, an increase in length of hospital stay and increased mortality. Therefore, early resumption of normal oral intake of food is important. If this is delayed postoperatively (more than 5 days) enteral feeding will be required.

POSTOPERATIVE RESPIRATORY PROBLEMS

Breathlessness usually indicates respiratory difficulty and hypoxia, but can be due to pain, anxiety, sepsis, acidosis and anaemia due to bleeding. Initial treatment is to ensure a patent airway and give a high percentage of humidified oxygen by facemask. This is guided by pulse oximetry to maintain a peripheral saturation adequate for the patient (usually > 92%). The cause is then sought by history, examination and chest X-ray, and treated.

Stridor

The postoperative airway may be compromised because of the residual effects of anaesthesia (in particular opioid or midazolam sedation), by vomit, or by surgical complications. After thyroid/parathyroid surgery recurrent laryngeal nerve palsy may cause vocal cord palsy, and cervical haematoma cause airway compression. The airway must be re-established using standard techniques. Laryngeal surgery may cause airway oedema. This may respond to nebulised adrenaline 2.5-5mg and intravenous dexamethasone 4-8mg.

Atelectasis and pneumonia

Wound splinting, pain, dehydration, and immobility lead to collapse and atelectasis of basal lung segments in the hours after surgery, which will be clearly seen on chest X-ray. A poor cough may lead to retention of secretions. The mainstays of treatment are good analgesia, humidified oxygen, careful fluid balance, physiotherapy and early mobilisation. Suctioning through a minitracheostomy can remove secretions.

Infection and pneumonia may intervene. The patient usually becomes febrile with purulent sputum (often not coughed up effectively) and a neutrophil leucocytosis. Consolidation is seen on the chest X-ray. (Widespread bronchopneumonia is much more common than lobar pneumonia).

Supplemental oxygen is given by facemask to maintain adequate peripheral saturations. Sputum and venous blood specimens are sent for microscopy, culture and sensitivity testing. Regular physiotherapy is required, and supplemental intravenous fluids to replace losses due to fever. Appropriate antibiotic therapy will depend on microbiological results and local prevalences of organisms, but cefotaxime or cefuroxime, and gentamicin are often used.

Bronchospasm

This may be due to an exacerbation of pre-existing asthma, perioperative pulmonary aspiration of blood or vomit, or a reaction to a drug. Pulmonary oedema or pulmonary embolus may mimic bronchospasm, and if suspected are treated (*see below*).

The patient will be dyspnoeic, tachypnoeic and using their accessory muscles. Speaking is difficult, and an expiratory wheeze is audible on auscultation. A quiet chest is ominous as it may represent very little airflow taking place. A chest X-ray is useful to exclude pneumothorax, but may show collapsed areas following pulmonary aspiration. Arterial blood gas hypercarbia indicates fatigue, and imminent respiratory collapse.

Treatment is high flow oxygen by mask, nebulised salbutamol 2.5-5mg (initially every 15 minutes, unless severe tachycardia occurs), nebulised ipratropium bromide (Atrovent) 250-500mcg 6 hourly, and intravenous hydrocortisone 200mg. For patients *not* usually taking oral theophyllines, consider addition of intravenous aminophylline (5mg/kg over 15 minutes, followed by an infusion at 0.5mg/kg/hr). Nebulised adrenaline 2.5mg may be used when salbutamol is not

available. Intramuscular adrenaline 0.5mg may be used with extremely severe bronchospasm, and may be repeated. Respiratory failure can occur rapidly, so equipment for sedation, re-intubation and assisted mechanical ventilation should be prepared. This will enable tracheal suction if aspiration is suspected.

Pulmonary oedema

Intraoperative fluid overload, ARDS and failure of the left ventricle (typically in patients with ischaemic heart disease), may all result in pulmonary oedema. ARDS is discussed below.

The patient is dyspnoeic, cyanosed, clammy, sweaty and tachycardic. Pink frothy sputum indicates severe pulmonary oedema. The jugular venous pressure is raised and there are coarse bibasal inspiratory crepitations on auscultation, or a “cardiac” wheeze. Chest X-ray shows bilateral, midzone, fluffy infiltrates and upper lobe blood diversion. The patient is sat up, and intravenous fluids stopped. Give high flow oxygen by facemask, intravenous diuretics (e.g. frusemide 50mg – consider a urinary catheter), and, if systolic blood pressure > 90mmHg, 2 puffs of sublingual GTN (glyceryl trinitrate). Intravenous diamorphine 2.5-5mg (or 5 –10mg of morphine) can also reduce preload, but observe for sedative side effects. If the above fail, application of CPAP (Continuous Positive Airways Pressure) 5-10cm H₂O, by tight fitting facemask, is often helpful. An ECG may show signs of perioperative myocardial ischaemia, and a new murmur may indicate acute cardiac valvular dysfunction.

Pneumothorax

This may occur as a complication of surgery, central line placement or follow positive pressure ventilation in a patient with asthma or COPD. The patient will be breathless and often complains of pain over the affected lung. There is diminished air entry and hyper-resonance on percussion, and if severe, tracheal deviation away from the pneumothorax, raised jugular venous pressure and cardiovascular collapse; these are signs of tension pneumothorax and require immediate life-saving treatment.

In stable patients the chest X-ray will confirm the diagnosis. There is no time to do this if tension pneumothorax is suspected. Immediate insertion of a large 14-gauge intravenous cannula into the pleural cavity at the second intercostal space, mid-clavicular line will be followed by a whoosh of air as the pneumothorax is decompressed.

Small pneumothoraces (<20% of the lung field), without patient compromise, may be observed until

resolution. Larger pneumothoraces will require placement of an intercostal chest drain attached to an under water seal.

Pulmonary embolus

This is rare in the recovery room. Embolus can be due to thrombus, air, fat or tumour. Massive embolus will result in cardiorespiratory arrest. A small embolus results in dyspnoea, pleuritic chest pain, haemoptysis and tachycardia. There may be signs of DVT (Deep Vein Thrombosis), and a pleural rub. Arterial blood gases typically show hypoxia, and hypocarbia of hyperventilation. A chest X-ray is more helpful in excluding other diagnoses (e.g. pneumothorax), but may show a wedge shaped pulmonary infarct. The commonest ECG finding is a sinus tachycardia, but right ventricular strain or an S1-Q3-T3 pattern (S wave in lead 1, Q wave in lead 3, and T wave in lead 3) may be seen.

Treatment is 100% humidified oxygen by facemask, and analgesia with intravenous morphine (5-10mg). Consideration is given to anticoagulation with intravenous heparin. This involves discussion with the surgical team as it may be contraindicated postoperatively. An alternative, to prevent recurrence of pulmonary embolus, is an inferior vena caval filter. If a V/Q (Ventilation/Perfusion) scan is available this will confirm the diagnosis.

ARDS (Acute Respiratory Distress Syndrome)

ARDS develops after a variety of major insults including shock, sepsis, pancreatitis, massive blood transfusion and multitrauma. Oxygenation is poor (and not the result of cardiac failure) and the X-ray shows widespread pulmonary infiltrates of alveolar oedema. Onset is generally within 24 hours of the insult. The patient may require mechanical ventilation on the ICU, with application of PEEP (Positive End Expiratory Pressure) and high inspired oxygen concentrations. Steroids are not useful in the early stages.

Further Reading

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CENTRAL VENOUS ACCESS AND MONITORING

Dr Graham Hocking, Frimley Park Hospital, Portsmouth Road, Frimley, Camberley, Surrey, GU16 5UJ

Central venous access is the placement of a venous catheter in a vein that leads directly to the heart. The main reasons for inserting a central venous catheter are:

- measurement of central venous pressure (CVP)
- venous access when no peripheral veins are available
- administration of vasoactive/inotropic drugs which cannot be given peripherally
- administration of hypertonic solutions including total parenteral nutrition
- haemodialysis/plasmapheresis

Which central vein to cannulate?

There are a number of central veins and for each of these there are a variety of techniques. It should be remembered that, with the exception of the external jugular, central veins are often deep and have to be located blindly. This is associated with risk to nearby structures, especially in the hands of the inexperienced operator. Veins commonly lie close to arteries and nerves, both of which can potentially be damaged by a misplaced needle. The subclavian vein also lies close to the dome of the pleura, damage to which can cause a pneumothorax. The choice of route will therefore depend on a number of factors as listed in table 1.

Types of central venous catheters

Catheters are available which differ in length, internal

diameter, number of channels (access ports), method of insertion (see below), material and means of fixation. Two useful lengths are 20cm catheters for subclavian and internal jugular lines, and 60cm catheters for femoral and basilic lines.

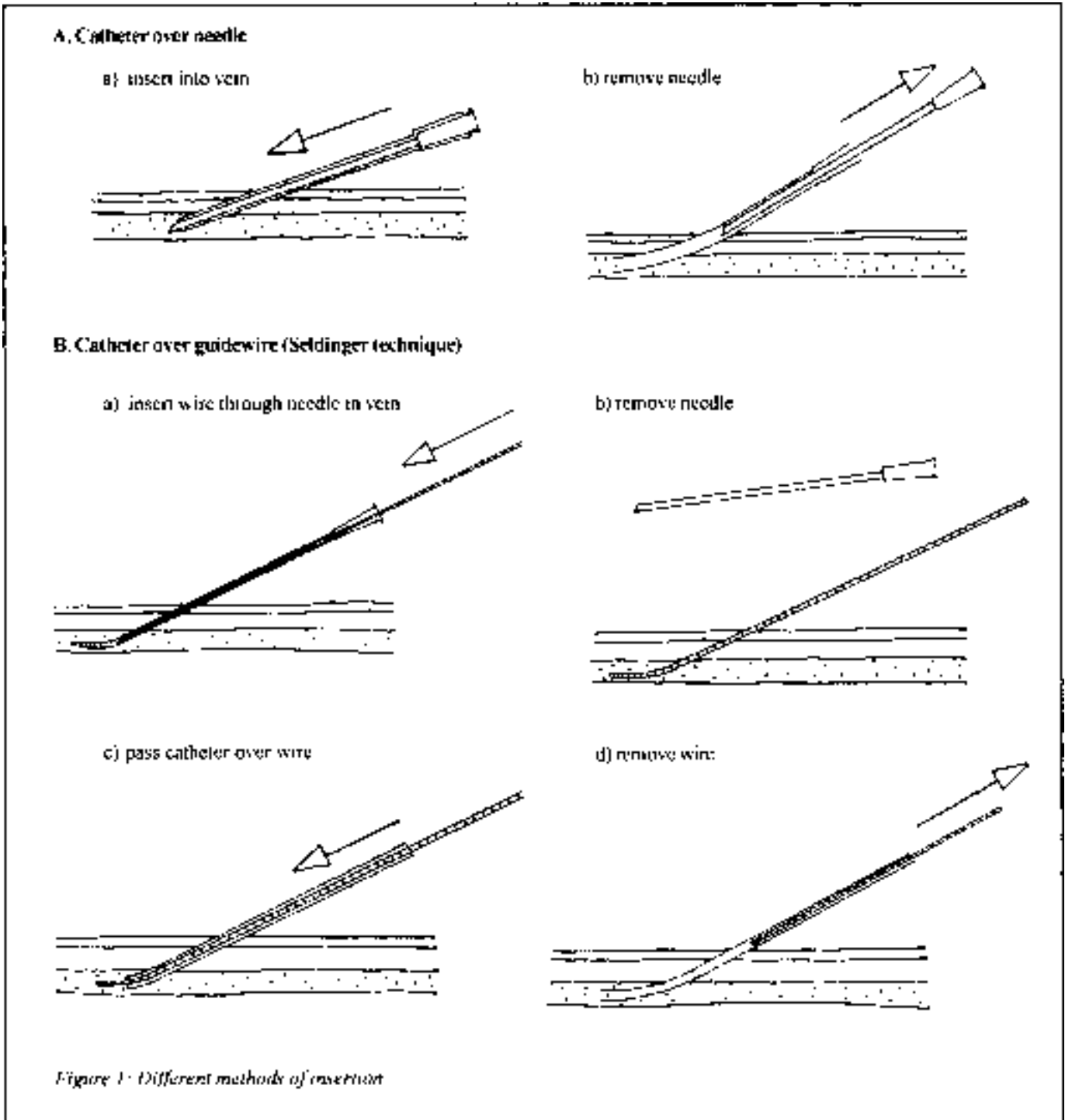
Different methods of insertion

There are several basic methods of inserting the catheter after the vein has been found:

- **Catheter over the needle.** This is a longer version of a conventional intravenous cannula and may be quickly inserted with a minimum of additional equipment. The catheter is larger than the needle, which reduces the leakage of blood from the insertion site, but using a larger needle to find the vein makes the consequences of accidental arterial puncture more serious. In addition it is easy to over-insert the needle.
- **Catheter over guidewire (Seldinger technique).** This is the preferred method of insertion. A small diameter needle (18 or 20 gauge) is used to find the vein. A guidewire is passed down the needle into the vein and the needle removed. The guidewire commonly has a flexible J-shaped tip to reduce the risk of vessel perforation and to help negotiate valves in the vein e.g the external jugular vein (EJV). Once the wire is placed in the vein, the catheter is passed over it until positioned in the vein. The wire should

Table 1. Factors which determine the choice of central vein

Patient:	How long will the catheter be required? ie. long term / intermediate / short term Suitability of the vein for technique chosen e.g. for CVP measurement the tip of the catheter must be within the thorax. A femoral route therefore needs a long catheter
Operator:	Knowledge and practical experience of the technique –it is better to have a few clinicians in each area who perform all the central venous cannulations and gain experience (a “central venous access team”)
Technique characteristics:	Success rate for vein cannulation Success rate of central placement Complication rate. Applicability to patients of different ages Ease of learning Puncture of a visible and/or palpable vein or ‘blind’ venepuncture based on knowledge of anatomy
Equipment available:	Availability of suitable apparatus Cost Suitability of material for long term cannulation



not be over-inserted as it may kink, perforate the vessel wall or cause cardiac arrhythmias. This technique allows larger catheters to be placed in the vein after the passage of appropriate dilators along the wire and a small incision in the skin at the point of entry.

- **Catheter through the needle or catheter through cannula.** The catheter is passed through a cannula or needle placed in the vein. The technique is

becoming less popular as the hole made in the vein by the needle is larger than the catheter that is passed leading to some degree of blood leakage around the site. If a problem is encountered during threading the catheter, withdrawal of it through the needle risks shearing part of the catheter off with catheter embolisation into the circulation. This technique is mainly reserved for the antecubital route.

General preparation to obtain central venous access

The basic preparation and equipment that is required for central venous cannulation is the same regardless of the route or technique chosen. Clinicians who insert central venous lines should be taught the technique by an experienced colleague. If this is not possible then the access routes associated with the fewest complications are the basilic vein or femoral vein.

General technique for all routes

- Confirm that central venous access is needed and select the most appropriate route. Explain the procedure to the patient
- Shave the needle insertion area if very hairy
- Using a strict aseptic technique, prepare and check all the equipment for use. Read instructions with the catheter.
- Sterilise the skin and drape the area
- Infiltrate the skin and deeper tissues with local anaesthetic. In cases where difficulty is anticipated use the small local anaesthetic needle to locate the vein before using the larger needle. This reduces the risk of trauma to other structures.
- Position the patient as for the specific route described – avoid long periods of head down, particularly in breathless patients
- Identify the anatomical landmarks for the chosen route and insert the needle at the recommended point. After the needle has penetrated the skin, aspirate gently whilst advancing the needle as directed until the vein is entered. If the vein is not found, slowly withdraw the needle whilst gently aspirating; often the vein has been collapsed and transfixed by the entry of the needle.
- If using a catheter over or through needle technique, thread the catheter into the vein, remove the needle, flush with saline and secure it in place (see checks below)
- If using a guidewire (Seldinger technique), pass this into the vein, flexible J-shape end first, then remove the needle. Small single lumen catheters may pass directly over the wire into the vein. In this case, thread the catheter over it until the end of the wire protrudes from the end of the catheter and whilst holding the wire still advance the catheter into the vein. TAKE CARE not to allow the wire to be pushed further into the vein whilst advancing the catheter
- It may be necessary to dilate up the hole in the vein when larger catheters are used. Make a small incision in the skin and fascia where the wire enters the patient. Thread the dilator over the wire into the vein with a twisting motion. Excessive force should not be needed. Remove the dilator taking care not to dislodge the guidewire. Thread the catheter over the wire as described above.
- Check that blood can be aspirated freely from all lumens of the catheter and flush with saline
- Secure the catheter in place with the suture and cover with a sterile dressing. Tape any redundant tubing carefully avoiding any kinking or loops which may snag and pull out the catheter.
- Connect catheter to a bag of intravenous fluid

Table 2. Equipment required for central venous access.

-
- Patient on a tilting bed, trolley or operating table
 - Sterile pack and antiseptic solution
 - Local anaesthetic – e.g. 5ml lignocaine 1% solution
 - Appropriate CV catheter for age/route/purpose
 - Syringes and needles
 - Saline or heparinised saline to prime and flush the line after insertion
 - Suture material – e.g. 2/0 silk on a straight needle
 - Sterile dressing
 - Shaving equipment for the area if very hairy (especially the femoral)
 - Facility for chest X-ray if available
 - Additional equipment required for CVP measurement includes: manometer tubing, a 3-way stopcock, sterile saline, a fluid administration set, a spirit level and a scale graduated in centimeters.
-

Checks before using the line

- Ensure fluid runs in freely and that blood flows freely back. To observe the latter place the infusion bag below the level of the bed
- If available, take a chest X-ray (ideally erect) to check the position of the catheter tip and to exclude a pneumo, hydro or haemothorax. An early radiograph may not show up abnormalities and it may be best to wait 3-4 hours unless symptoms develop. The tip of the CVP line should lie in the superior vena cava just above its junction with the right atrium.
- Ensure that the patient will be nursed where their CV line can be supervised. Give appropriate written instructions regarding how, and what it is to be used for, and who to contact if there is a problem

Practical problems common to most techniques of insertion

Table 3 lists some problems that can occur with any of the routes for central venous access.

Complications

The main complications that can arise from central venous cannulation are listed in table 4. The incidence of each complication varies for each route described.

The Subclavian Vein

The subclavian vein (SCV) has a wide calibre (1-2cm diameter in adults) and is thought to be held open by surrounding tissue. In severely shocked patients, however, it may be safer to perform a venous cutdown (eg. onto the long saphenous vein) or use the EJV which may be accessible. In conscious patients the subclavian route is often preferred (since head movement does not affect it) and also in trauma patients with suspected cervical spine injury. Subclavian cannulae are easier to secure which reduces subsequent movement and dislodgment. Whilst a high success rate for placement can be achieved, serious complications occur more commonly than with the other routes. Subclavian puncture should be avoided in patients with abnormal clotting since it is difficult to apply pressure to the subclavian artery following accidental puncture.

Table 3. Problems during CV cannulation

Arterial puncture	Usually obvious but may be missed in a patient who is hypoxic or hypotensive. If unsure, connect a length of manometer tubing to the needle / catheter and look for blood flow which goes higher than 30cm vertically or is strongly pulsatile. Withdraw the needle and apply firm direct pressure to the site for at least 10 minutes or longer if there is continuing bleeding. If there is minimal swelling then retry or change to a different route.
Suspected pneumothorax	If air is easily aspirated into the syringe (note that this may also occur if the needle is not firmly attached to the syringe) or the patient starts to become breathless. Abandon the procedure at that site. Obtain a chest radiograph and insert an intercostal drain if confirmed. If central access is absolutely necessary then try another route ON THE SAME SIDE or either femoral vein. DO NOT attempt either the subclavian or jugular on the other side in case bilateral pneumothoraces are produced.
Arrhythmias during the procedure	Usually from the catheter or wire being inserted too far (into the right ventricle). The average length of catheter needed for an adult internal jugular or subclavian approach is 15cm. Withdraw the wire or catheter if further than this.
Air embolus	This can occur, especially in the hypovolaemic patient, if the needle or cannula is left in the vein whilst open to the air. It is easily prevented by ensuring that the patient is positioned head down (for jugular and subclavian routes) and that the guidewire or catheter is passed down the needle promptly.
The wire will not thread down the needle	Check that the needle is still in the vein. Flush it with saline. Try angling the needle so the end of it lies more along the plane of the vessel. Carefully rotate the needle in case the end lies against the vessel wall. Reattach the syringe and aspirate to check that you are still in the vein. If the wire has gone through the needle but will not pass down the vein it should be very gently pulled back. If any resistance is felt then the needle should be pulled out with the wire still inside, and the procedure repeated. This reduces the risk of the end of the wire being cut off by the needle tip.
Persistent bleeding at the of entry	Apply firm direct pressure with a sterile dressing. Bleeding should usually stop unless there is a coagulation abnormality. Persistent severe bleeding may require surgical exploration if there is an arterial or venous tear

Anatomy. The SCV lies in the lower part of the supraclavicular triangle (figure 2.) and drains blood from the arm. It is bounded medially by the posterior border of the sternocleidomastoid muscle, caudally by the middle third of the clavicle, and laterally by the anterior border of the trapezius muscle. The SCV is the continuation of the axillary vein and begins at the lower border of the first rib. Initially the vein arches upwards across the first rib and then inclines medially, downwards and slightly forwards across the insertion of the scalenus anterior muscle into the first rib to enter the thorax where it joins with the IJV behind the sternoclavicular joint.

Anteriorly, the vein is covered throughout its entire course by the clavicle. It lies anterior to, and below the subclavian artery as it crosses the first rib. Behind the artery lies the cervical pleura which rises above the sternal end of the clavicle.

Preparation and positioning. The patient should be supine, both arms by the sides, with the table tilted head down to distend the central veins and prevent air embolism. Turn the head away from the side to be cannulated unless there is cervical spine injury. Normally the right SCV is cannulated since the thoracic duct is on the left and may occasionally be damaged during SCV cannulation.

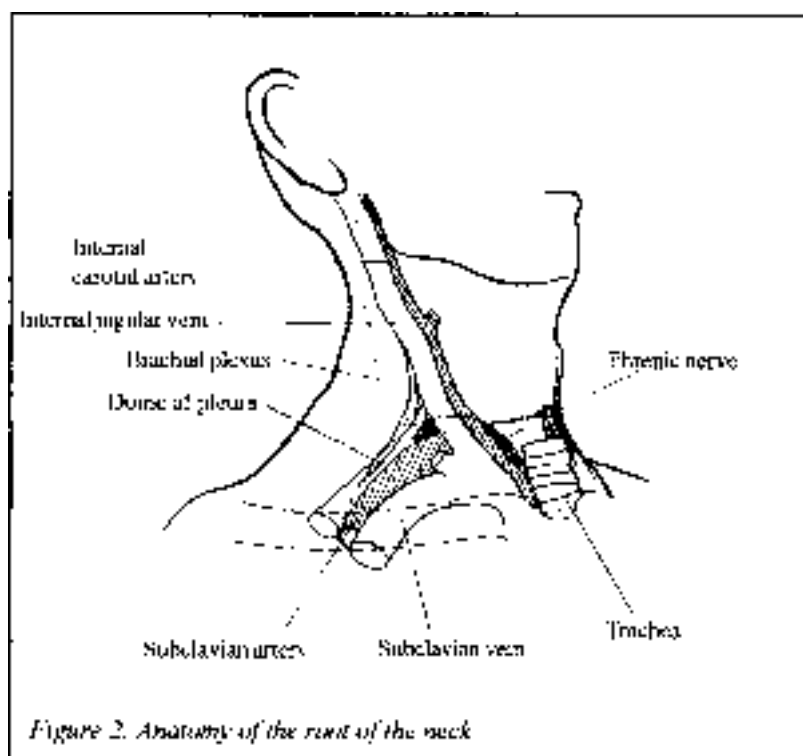
Technique. Stand beside the patient on the side to be cannulated. Identify the midclavicular point and the sternal

notch. The needle should be inserted into the skin 1cm below and lateral to the midclavicular point. Keeping the needle horizontal, advance posterior to the clavicle aiming for the sternal notch. If the needle hits the clavicle withdraw and redirect slightly deeper to pass beneath it. Do not pass the needle further than the sternal head of the clavicle.

Complications. Any of the complications described above can occur but pneumothorax (2-5%) or rarely haemothorax or chylothorax (fatty white fluid in the pleural cavity due to leakage of lymph from thoracic duct) are more common with this route than the others. Occasionally the catheter may pass up into either jugular or the opposite SCV rather than into the chest. This will not give reliable CVP readings and infusion of some drugs (hypertonic solutions/vasoconstrictors) may be contra-indicated.

Table 4. Potential complications.

Early	Late
Arterial puncture	Venous thrombosis
Bleeding	Cardiac perforation and tamponade
Cardiac arrhythmias	Infection
Injury to the thoracic duct	Hydrothorax
Injury to surrounding nerves	
Air embolism	
Catheter embolus	
Pneumothorax	



Practical problems specific to the subclavian route

- **Keep hitting the clavicle:** Check that you are starting from the correct position. Redirect the needle slightly more posterior whilst being careful not to enter the chest. Try bending the needle slightly to encourage it to pass beneath the clavicle. Try placing a pillow under the shoulders or getting someone to pull gently down on the arms.
- **Cannot find the vein:** direct the needle a little more cephalad
- **Fail after repeated attempts:** DO NOT PERSIST since the likelihood of complications increase. Try an alternative route ON THE SAME SIDE unless chest radiography is available to exclude any possible pneumothorax.

- **The catheter tip is not in the chest:** Usually detected on chest X-ray, or if the fluid level in the CVP manometer does not rise and fall with breathing. A simple test that may increase the suspicion of jugular placement is to rapidly inject 10ml of fluid into the catheter whilst listening with a stethoscope over the neck. An audible 'whoosh' or thrill under the fingers suggests the catheter has entered the jugular vein. If this is positive, in the presence of a CVP reading which does not change with respiration, then the position of the cannula must be questioned.

The Internal Jugular Vein

The internal jugular vein (IJV) is a potentially large vein commonly used for central venous access which drains blood from the brain and deep facial structures. Cannulation is associated with a lower incidence of complications than the subclavian approach. Unlike the subclavian route, failure on one side does not prevent the operator from trying the other side although this should be discouraged if arterial puncture had occurred. Many approaches have been described depending upon the level in the neck where the vein is entered. High approaches reduce the risk of pneumothorax but increase the risk of arterial puncture the opposite being true of a low approach. A middle level approach is described below.

Anatomy. The sigmoid venous sinus passes through the mastoid portion of the temporal bone, emerging from the jugular foramen at the base of the skull as the IJV. It passes vertically down through the neck within the carotid sheath. The vein initially lies posterior to the internal carotid artery, before becoming lateral and then anterolateral to the artery. It is able to expand laterally to accommodate increased blood volume. It joins the SCV behind the sternal end of the clavicle to enter the chest as the innominate vein (figure 2).

Preparation and positioning. The patient should be supine, both arms by the sides, with the table tilted head down to distend the central veins and prevent air embolism. Slightly turn the head away from the side to be cannulated for better access (turning it too far increases the risk of arterial puncture).

Technique. Stand at the head of the patient. Locate the cricoid cartilage and palpate the carotid artery lateral to it at this level. Keeping a finger gently over

the artery, insert the needle at an angle of 30-40° to the skin and advance it downward towards the nipple on the same side (in a woman guess where the nipple would be if she were a man). Always direct the needle away from the artery under your finger. The vein is usually within 2-3cm of the skin. If the vein is not found, redirect the needle more laterally.

Complications. With experience this route has a low incidence of complications. Arterial puncture is easily managed by direct pressure. Pneumothorax is rare providing the needle is not inserted too deeply.

Practical problems

- **Cannot feel the artery.** Check the patient! Try the carotid on the other side. It is safer to consider a different approach rather than 'blindly' try to find the jugular.
- **Arterial puncture.** Remove needle and apply firm pressure over the puncture site for 10 minutes.
- **Cannot find the vein.** Recheck your position. Ensure that you are not pressing firmly on the artery as this can compress the vein next to it. Try tipping the patient further head down if possible. If the patient is hypovolaemic, and central venous access is not immediately required to correct it, give intravenous fluids and wait until the veins are fuller. Try inserting the needle a little closer to the artery but beware of puncture.

The External Jugular Vein

Since the external jugular vein (EJV) lies superficially in the neck and is often visible or palpable, complications associated with 'blind' venepuncture of deep veins are avoided. The EJV is preferred when expertise is lacking, for emergency intravenous fluid administration and in cardiac arrests, when the carotid pulsation cannot be felt. However, because of the way the EJV joins the SCV there is a 10-20% chance that a cannula will not pass into the SVC. In this situation it will not be suitable for CVP measurements but can still provide central access for other purposes as described at the beginning.

Anatomy. The EJV is formed from the junction of the posterior division of the posterior facial vein and the posterior auricular vein, draining blood from the superficial facial structures and scalp. It passes down in the neck from the angle of the mandible, crosses the sternocleidomastoid muscle obliquely, and terminates behind the middle of the clavicle where it joins the SCV.

The vein is variable in size and has valves above the clavicle and just before its junction with the SCV which may obstruct the passage of CV catheters. If a guidewire with a J shaped tip is used the wire can often pass through these valves by rotating it at the lower end of the EJV. Natural variations and disease states are responsible for the wide range in the degree of prominence of the EJV.

Preparation and positioning. The patient should be supine, both arms by the sides, with the table tilted head down to distend the central veins and prevent air embolism. Turn the head away from the side to be cannulated for better access.

Technique. Stand at the head of the patient and identify the EJV as it crosses the sternocleidomastoid muscle. If it is not palpable or visible (see problems) then choose an alternative vein for catheterisation. Insert the needle in line with the vein where it is most easily seen or palpated. Thread the guidewire and then the catheter.

Complications

If the vein is easily seen or palpated this route carries a very low risk.

Practical problems

- **Cannot see the vein:** Ask the patient to take a big breath in and strain as if trying to go to the toilet (Valsalva manoeuvre). If mechanically ventilated briefly hold the lungs in inspiration. Press on the skin above the midpoint of the clavicle where the vein enters the chest. If none of these make the EJV visible then use a different vein.
- **Catheter will not pass into chest:** Press on the skin where the vein enters the chest. Try rotating the catheter or flushing it with saline as you insert it. If using a guidewire, rotate the wire when it reaches the bottom of the vein. Try slowly turning the head in either direction. It may be useful to insert a normal plastic cannula into the vein first, then thread the guidewire down this. By doing this, the wire can be pushed, pulled and rotated without the risk of it being cut which could occur if the wire is manipulated through a needle.

The Femoral Vein

This may be the safest and most accessible central vein in children requiring resuscitation where peripheral access has failed. It is also a preferred route for inexperienced operators, due to the minimal risk of serious

complications. The femoral vein (FV) should not be used for more than a few days due to the risk of contamination and infection from the groin area. With pelvic or intra-abdominal injury an alternative central vein is preferred. Remember that the femoral route is not a good choice for CVP monitoring since the value will be altered by the intra-abdominal pressure unless a long catheter is used to pass above the level of the diaphragm.

Anatomy. The FV starts at the saphenous opening in the thigh and accompanies the femoral artery ending at the inguinal ligament, where it becomes the external iliac vein. In the femoral triangle the FV lies medial to the artery. Here it occupies the middle compartment of the femoral sheath, lying between the artery and the femoral canal. The femoral nerve lies lateral to the artery. The vein is separated from the skin by superficial and deep fasciae.

Preparation and positioning. Abduct and externally rotate the thigh slightly.

Performance of the technique. Identify the pulsation of the femoral artery 1-2 cm below the inguinal ligament. Insert the needle about 1 cm medial to the pulsation and aim it towards the head and medially at an angle of 20-30° to the skin. In adults, the vein is normally found 2-4 cm from the skin. In small children reduce the elevation on the needle to 10-15° since the vein is more superficial.

Complications. Arterial puncture is possible if the needle is directed too lateral. Femoral nerve damage may follow incorrect lateral insertion of the needle. Infection is the commonest problem with femoral catheters and they are not recommended for long-term use.

Practical problems

- **Cannot feel the artery:** Try the other side. Check the blood pressure. Treat any hypotension and retry. If there is no other venous access then it may be acceptable to try to locate the FV with a small needle starting medially to avoid the femoral nerve. Once found, change to the normal needle and continue the procedure. If the artery is accidentally punctured, apply direct pressure with your fingers and insert the normal needle medial to the puncture site.
- **Cannot locate the vein:** Recheck the anatomical landmarks. It is possible for the femoral vein to be compressed by the fingers on the artery. Release pressure but leave the fingers resting on the skin over the artery and retry. Cautiously redirect the needle closer to the artery and in a more lateral direction.

The Antecubital Veins

A palpable vein in the antecubital fossa provides the safest route for central venous access. A long 60cm catheter is required. There are a number of veins in the antecubital fossa – use one on the medial side.

Anatomy. Venous blood from the arm drains through two intercommunicating main veins, the basilic and the cephalic as illustrated in figure 3.

Basilic vein. Ascends from the hand along the medial surface of the forearm draining blood from that area and medial side of the hand. Near the elbow the vein changes to a position in front of the medial epicondyle where it is joined by the median cubital vein. It then runs along the medial margin of the biceps muscle to the middle of the upper arm where it pierces the deep fascia to run alongside the brachial artery becoming the axillary vein.

Cephalic vein. Ascends on the front of the lateral side of the forearm to the front of the elbow, where it communicates with the basilic vein through the median

cubital vein. It then ascends along the lateral surface of the biceps muscle to the lower border of pectoralis major muscle, where it turns sharply to pierce the clavipectoral fascia and pass beneath the clavicle. It then usually terminates in the axillary vein although it can join the EJV. There are valves at the termination of the cephalic vein. The sharp angle and the valves frequently obstruct the passage of a catheter along the cephalic system.

Median cubital vein. The median cubital vein is a large vein that arises from the cephalic vein just below the bend in the elbow and runs obliquely upwards to join the basilic vein just above the bend in the elbow. It receives veins from the front of the forearm which themselves may be suitable for catheterisation. It is separated from the brachial artery by a thickened portion of the deep fascia (bicipital aponeurosis).

Preparation and positioning. Apply a tourniquet to the upper arm to distend the veins and select the best one. The order of preference for veins are:

- A vein on the medial side of the antecubital fossa – the basilic or median cubital vein. Even when not visible, these veins are often easily palpable when engorged
- A vein on the postero-medial aspect of the forearm – a tributary of the basilic vein. Rotation of the arm may be required.
- The cephalic vein

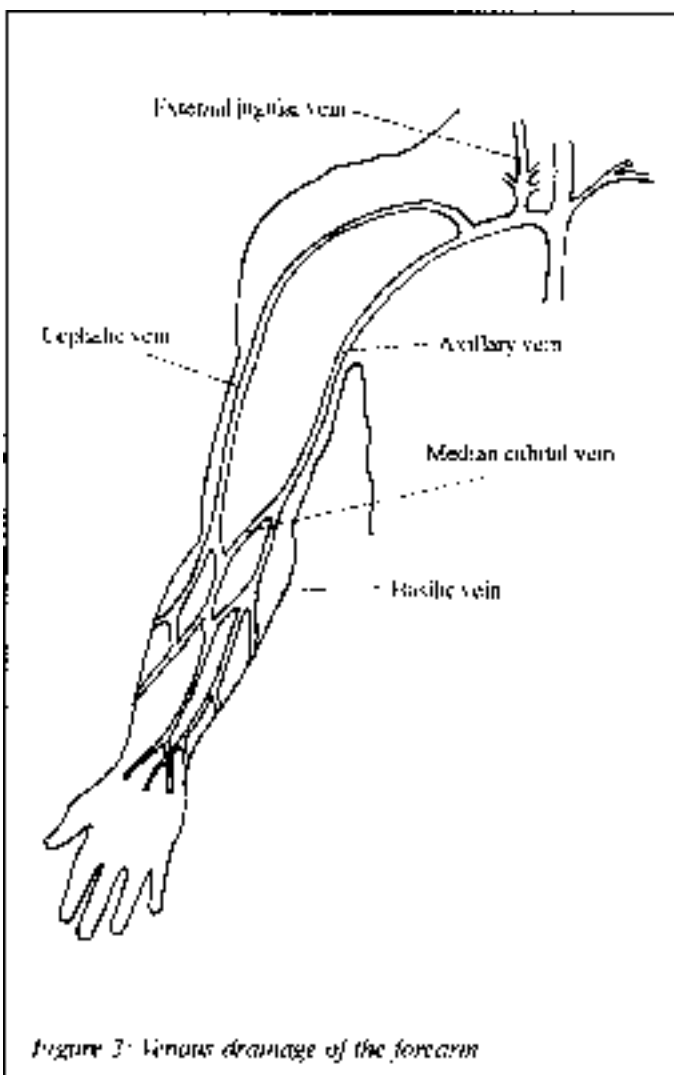
Lie the patient supine with the arm supported at 45° to the body and the head turned towards you (helps prevent the catheter passing into the IJV on that side).

Technique. Stand on the same side of the patient. Estimate the length of catheter needed to reach the SVC. Puncture the chosen vein with the needle and cannula and remove the needle. Insert the catheter through the cannula and advance it a short distance (2-4cm in adults, 1-2cm in children) then release the tourniquet. Steadily advance the catheter along the vein until it is estimated to be in the correct position.

Complications. Local bleeding since the catheter is smaller diameter than the needle used to puncture the vein. Apply direct pressure with a sterile swab.

Practical problems

- **Cannot thread the catheter along the vein.** Do not use force to pass it. If using a catheter-through-needle technique and you are sure that the catheter



is in the vein, remove the needle from the vein and slide it to the end of the catheter. This will allow you to advance and withdraw the catheter without risk of cutting it on the needle. Try flushing the catheter with saline whilst advancing. Try the arm in different positions. Rotate the catheter whilst inserting.

Care of the Central venous Catheter

- Use an aseptic technique when inserting the catheter and any subsequent injections or changing fluid lines
- Keep the entry site covered with a dry sterile dressing
- Ensure the line is well secured to prevent movement (this can increase risks of infection and clot formation)
- Change the catheter if there are signs of infection at the site.
- Remember to remove the catheter as soon as it is no longer needed. The longer the catheter is left in, the greater the risks of sepsis and thrombosis
- Some people suggest changing a catheter every 7 days to reduce the risks of catheter related sepsis and thrombosis. However, providing that the catheter is kept clean (sterile injections and connections) and there are no signs of systemic sepsis, routine replacement may not be necessary. Repeated cannulation to change lines on a routine basis, rather than based on clinical need, can increase the risks to the patient.

What is Central Venous Pressure ?

Blood from systemic veins flows into the right atrium; the pressure in the right atrium is the central venous pressure (CVP). CVP is determined by the function of the right heart and the pressure of venous blood in the vena cava. Under normal circumstances an increased venous return results in an augmented cardiac output, without significant changes in venous pressure. However with poor right ventricular function, or an obstructed pulmonary circulation, the right atrial pressure rises. Loss of blood volume or widespread vasodilation will result in reduced venous return and a fall in right atrial pressure and CVP.

The CVP is often used to make estimates of circulatory function, in particular cardiac function and blood volume. Unfortunately the CVP does not measure either of these directly, but taken in the context of the other physical

signs useful information can be gained. The supply of blood to the systemic circulation is controlled by the left ventricle. In a normal patient the CVP closely resembles the left atrial pressure and is usually used to predict it. However in patients with cardiac disease the right and left ventricles may function differently – this can only be detected clinically by measuring the pulmonary capillary wedge pressure (see later).

When should CVP be measured?

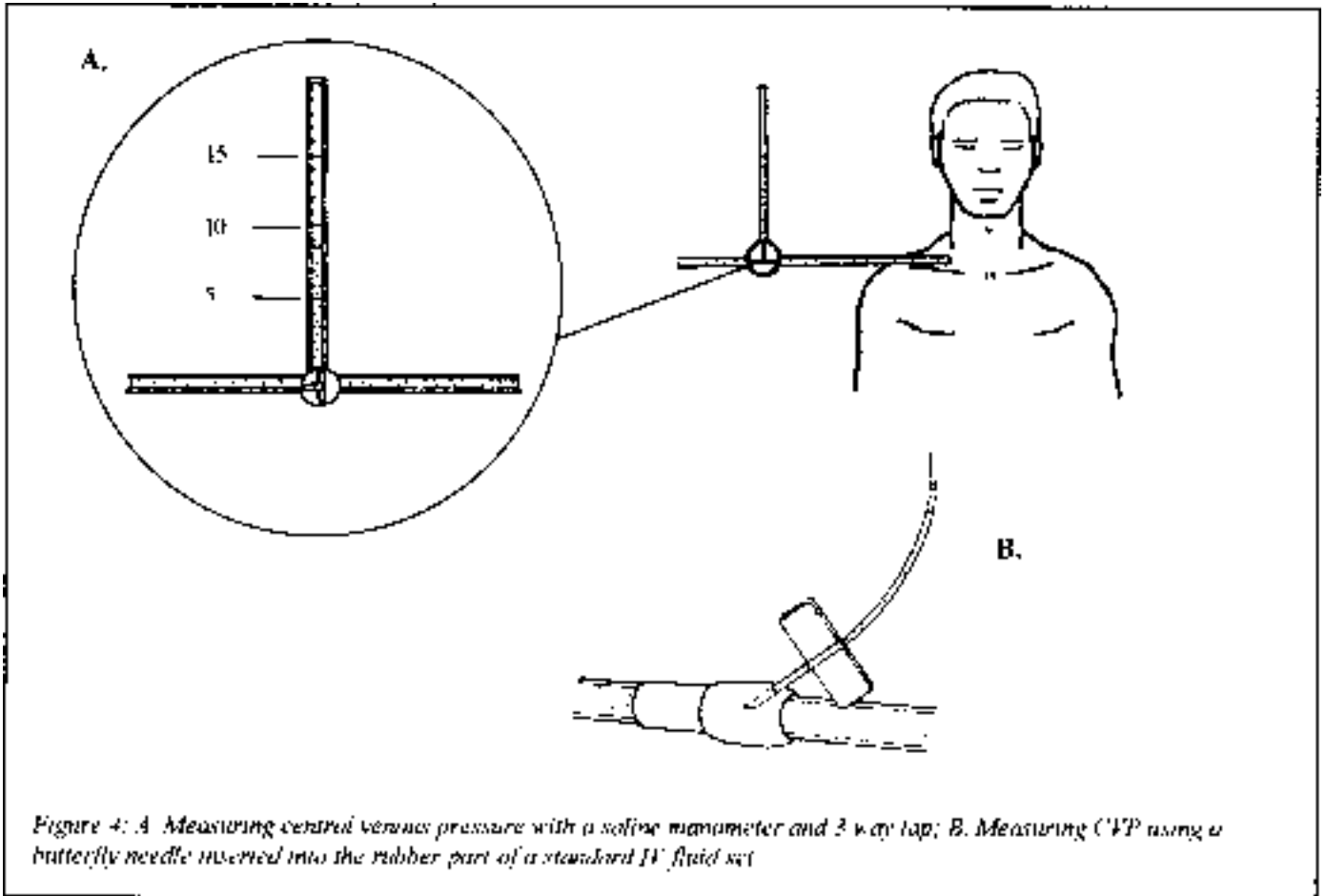
- Patients with hypotension who are not responding to basic clinical management.
- Continuing hypovolaemia secondary to major fluid shifts or loss.
- Patients requiring infusions of inotropes.

How to measure the CVP

The CVP is measured using a manometer filled with intravenous fluid attached to the central venous catheter. It needs to be 'zeroed' at the level of the right atrium, approximately the mid-axillary line in the 4th interspace supine. Measurements should be taken in the same position each time using a spirit level and the zero point on the skin surface marked with a cross. Check that the catheter is not blocked or kinked and that intravenous fluid runs freely in, and blood freely out. Open the 3-way tap so that the fluid bag fills the manometer tubing (check there is no obstruction to fluid flow and that the cotton wool in the top of the manometer is not blocked or wet). Turn the tap to connect the patient to the manometer. The fluid level will drop to the level of the CVP which is usually recorded in centimeters of water (cmH₂O). It will be slightly pulsatile and will continue to rise and fall slightly with breathing - record the average reading. An alternative to the manometer and 3-way tap is a butterfly needle inserted into the rubber injection port of ordinary intravenous tubing (figure. 4). In Intensive Care Units or theatres, electronic transducers may be connected which give a continuous readout of CVP along with a display of the waveform. Useful information can be gained by studying the electronic waveform. The CVP reading from an electronic monitor is sometimes given in mmHg (same as blood pressure). The values can easily be converted knowing that 10cmH₂O is equivalent to 7.5mmHg (which is also 1kPa)

Interpretation of the CVP

As previously stated, the CVP does not measure blood volume directly and is influenced by right heart function, venous return, right heart compliance, intrathoracic



pressure and patient positioning. It should always be interpreted alongside other measures of cardiac function and fluid state (pulse, BP, urine output etc.). The absolute value is not as important as serial measurements and the change in response to therapy. A normal value in a spontaneous breathing patient is 5-10cm water cmH_2O , rising 3-5 cmH_2O during mechanical ventilation. The CVP measurement may still be in the normal range even with hypovolaemia due to venoconstriction. A guide to interpretation is shown in table 5.

Short case examples of CVP interpretation

1. A 20 year old woman had a large post-partum bleed. Despite initial resuscitation her BP remained low and did not respond to large volumes of intravenous fluids. A CVP line was inserted. Her observations after insertion were: pulse 130/min, BP 90/70, and a CVP +1 cmH_2O . The CVP confirmed continuing hypovolaemia. After further IV fluid her pulse rate began to come down and her BP and CVP started to improve.
2. A 32 year old man was involved in a road accident sustaining chest and leg injuries. After initial resuscitation he was found to have a pneumothorax on the right which was drained with an underwater seal drain. Initially his respiratory function improved but despite fluid loading he remained hypotensive and a CVP line was inserted to guide fluid replacement for his leg injuries. After insertion he had a pulse of 120/min and BP 90/60 and a CVP reading of +15 $\text{cm H}_2\text{O}$. His neck veins were distended suggesting a high venous pressure. He was reassessed clinically and was found to have developed a tension pneumothorax on the left side which was drained with improvement in his condition.
3. A 19 year old man was admitted with an infected wound on his leg. His observations are: pulse 135/min, BP 80/30, CVP 7, hyperdynamic circulation. His pulse and BP does not respond to 2 fluid challenges so inotropes are started to support his circulation. His hypotension is due to septicaemia.

Table 5. Guide to interpretation of the CVP in the hypotensive patient

CVP reading	Other features that may be present	Diagnosis to consider	Treatment
Low	Rapid pulse Blood pressure normal or low Low urine output Poor capillary refill	Hypovolaemia	Give fluid challenges* until CVP rises and does not fall back again. If CVP rises and stays up but urine output or blood pressure does not improve consider inotropes
Low or normal or high	Rapid pulse Signs of infection Pyrexia Vasodilation/constriction	Sepsis	Ensure adequate circulating volume (as above) and consider inotropes or vasoconstrictors
Normal	Rapid pulse Low urine output Poor capillary refill	Hypovolaemia	Treat as above. Venoconstriction may cause CVP to be normal. Give fluid challenges* and observe effect as above.
High	Unilateral breath sounds Assymetrical chest movement Resonant chest with tracheal deviation	Tension pneumothorax	Thoracocentesis then intercostal drain
High	Rapid pulse Breathlessness Third heart sound Pink frothy sputum Oedema Tender liver	Heart failure	Oxygen, diuretics, sit up, consider inotropes
Very High	Rapid pulse Muffled heart sounds	Pericardial tamponade	Pericardiocentesis and drainage

*Fluid challenge. In hypotension associated with a CVP in the normal range give repeated boluses of intravenous fluid (250 – 500mls). Observe the effect on CVP, blood pressure, pulse, urine output and capillary refill. Repeat the challenges until the CVP shows a sustained rise and/or the other cardiovascular parameters return towards normal. With severe blood loss, blood transfusion will be required after colloid or crystalloid have been used in initial resuscitation. Saline or Ringers lactate should be used for diarrhoea/bowel obstruction/vomiting/burns etc.

When may the CVP reading be unreliable?

The use of CVP readings to estimate cardiac function and blood volume rely on the fact that there is no right ventricular disease and normal pulmonary vascular resistance. Table 6 lists some situations when CVP readings may be unreliable.

Catheter removal

Remove any dressing and suture material. Ask the patient to take a breath and fully exhale. Remove the catheter with a steady pull while the patient is breath holding and apply firm pressure to the puncture site for at least 5 minutes to stop the bleeding. Excessive force should not be needed to remove the catheter. If it does not come out, try rotating it whilst pulling gently. If this still fails, cover it with a sterile dressing and ask an

experienced person for advice.

Pulmonary artery flotation catheters (PAFC) catheters

A PAFC or Swan-Ganz catheter is a central venous catheter with a small inflatable balloon at the end. An introducing catheter is sited in a central vein and the catheter is then ‘floated’ along the central vein with the balloon inflated, through the right atrium and ventricle until it lies in a branch of the pulmonary artery. The position of the PAFC can be predicted as it moves through the circulation by the pressure waveform obtained by measuring the pressure at the tip of the PAFC. Once correctly positioned, when the balloon is inflated it occludes the branch of the pulmonary artery and measures the pressure distal to

Table 6

Problem	Effect on CVP
Pulmonary embolus High intrathoracic pressure	High pulmonary vascular resistance – left sided pressure and function may be normal. A higher than normal CVP may be needed to ensure adequate return of blood to the left side of the heart.
Left heart failure	Resulting rise in pulmonary venous pressure and right sided heart strain. Initially CVP may be normal but will increase with significant failure.
Constrictive pericardial disease	Paradoxical rise in CVP on inspiration and fall on expiration (opposite of normal in a spontaneously breathing patient). The absolute level will be higher due to impeded filling of the heart
Blocked cotton wool at top of manometer	Fluid will not move in the tube to give a correct reading
Complete heart block	'Cannon waves' on CVP reading – the reading will have a strong pulsatile element when the atrium contracts against a closed tricuspid valve sending the pressure wave back into the SVC
Tricuspid stenosis/regurgitation	Mean CVP will be higher

it (pulmonary artery occlusion pressure or 'wedge' pressure since it is 'wedged' in the artery). With the balloon inflated there is a continuous column of fluid between the tip of the PAFC and the left atrium, without interference from heart valves and lung pathology. It is therefore a better guide to the venous return to the left side of the heart than CVP. However, it is a more invasive monitor, requires more expertise to insert, has a greater complication rate and is more expensive.

PAFC are sometimes used in patients with significant right sided valve disease, right heart failure or lung disease as the CVP may be unreliable in predicting the left atrial

pressure. When connected to a computer a PAFC may be used to calculate the cardiac output using a thermodilutional technique and further guide patient management. However PAFC have not been shown to improve patient survival (see Further Reading).

Further Reading

Handbook of Percutaneous Central Venous Catheterisation. Rosen M, Latto IP, Shang Ng W. WB Saunders Company Ltd. 1981

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Connors AF et al. The effectiveness of right heart catheterization in the initial care of critically ill patients. *JAMA* 1996; 276(11): 889-97



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Contact Details:

SASA 2001/AAAC Congress Secretariat
Department of Anaesthetics
Private Bag 7, Congella 4013
Durban, South Africa
Tel/Fax: +27 31 260 4472

Email: aaac@nu.ac.za

Website: www.aaac.nu.ac.za



ANAPHYLAXIS

Dr. Christopher M, Dr. Albin Immanuel, Dr. Verghese Cherian, Dr. Rebecca Jacob, Vellore, India.

An anaphylactic reaction or anaphylaxis is an exaggerated immunological response to a substance to which an individual has become sensitised. When the patient is in contact with the substance, histamine, serotonin, tryptase and other vasoactive substances are released from basophils and mast cells. Anaphylactoid reactions are clinically indistinguishable from anaphylaxis, but are mediated by the drug or substance directly, and not by sensitised IgE antibodies.

Direct release of small amounts of histamine is commonly seen with drugs such as morphine and non-depolarising muscle relaxants (tubocurarine, alcuronium, atracurium). Clinical manifestations are usually minor and consist of urticaria (skin redness and swelling), usually along the line of the vein, flushing and occasionally mild hypotension.

Any drug can potentially cause an allergic reaction but agents used in anaesthetic practice that have been implicated in producing anaphylactic reactions include thiopentone, suxamethonium, non-depolarising muscle relaxants, ester local anaesthetics, antibiotics, plasma expanders (dextrans, starches and gelatins) and latex.

Clinical Presentation of Anaphylaxis

The commonest features are cardiovascular. Not all signs occur in every patient - one feature may be more obvious than others. Reactions range from minor to life-threatening. An awake patient will have a range of symptoms, but the diagnosis is more difficult in an anaesthetised patient.

Suspect anaphylaxis in an anaesthetised patient who suddenly becomes hypotensive or develops bronchospasm, particularly if this follows administration of a drug or fluid. Latex allergy may be delayed in onset, sometimes taking up to 60 minutes to occur.

- **Haematological.** Coagulopathy.
 - **Cutaneous.** Flushing, erythema, urticaria.
- ### MANAGEMENT
- #### Immediate Treatment of a Severe Reaction
- Stop administration of the causal agent and call for help.
 - Follow the ABC of resuscitation.
 - Adrenaline is the most useful drug for treating anaphylaxis as it is effective in bronchospasm and cardiovascular collapse.
- #### A - Airway and Adrenaline
- Maintain airway and administer 100% oxygen.
 - Adrenaline. If i/v access available give 1:10,000 adrenaline in 0.5-1ml increments, repeated as required. Alternatively give i/m 0.5 - 1mg (0.5 - 1ml of 1: 1000 solution) repeated each 10 minutes as required.
- #### B - Breathing
- Ensure adequate breathing. Intubation and ventilation may be required.
 - Adrenaline will treat bronchospasm and swelling of the upper airway.
 - Nebulised bronchodilators (e.g. 5mg salbutamol) or i/v aminophylline may be required if bronchospasm is refractory (loading dose of 5mg/kg followed by 0.5mg/kg/hour).
- #### C - Circulation
- Assess the circulation. Start CPR if cardiac arrest has occurred.
 - Adrenaline is the most effective treatment for severe hypotension
 - Insert 1 or 2 large bore i/v cannulae and rapidly infuse normal saline. Colloid may be used (unless it is thought to be the source of the reaction).
 - Venous return may be aided by lifting the patient's legs or tilting the patient head down.
 - If the patient remains haemodynamically unstable after fluids and adrenaline - give further doses of adrenaline or an intravenous infusion (5mg in 50mls saline or dextrose 5% through a
- **Cardiovascular.** Hypotension and cardiovascular collapse. Tachycardia, arrhythmias, ECG may show ischaemic changes. Cardiac arrest.
 - **Respiratory System.** Oedema of the glottis, tongue and airway structures may cause stridor and airway obstruction. Bronchospasm - may be severe.
 - **Gastrointestinal.** There may be abdominal pain, diarrhoea or vomiting.

syringe pump, or 5mg in 500mls saline or dextrose 5% given slowly by infusion. Uncontrolled intravenous boluses of adrenaline can cause dangerous surges in blood pressure and arrhythmias. Give the drug carefully, observing the response and repeating when required. Try to monitor the ECG, blood pressure and pulse oximetry.

Intramuscular dose of adrenaline in children

> 5 years	0.5ml of 1:1000
4 years	0.4ml of 1:1000
3 years	0.3ml of 1:1000
2 years	0.2ml of 1:1000
1 year	0.1ml of 1:1000

Further Management

- Give antihistamine agents. H₁ blockers eg chlorpheniramine (10mg i/v) and H₂ blockers ranitidine (50mg i/v slowly) or cimetidine (200mg i/v slowly).
- Corticosteroids Give hydrocortisone 200mg i/v followed by 100-200mg 4 to 6 hourly. Steroids will take several hours to work.
- Make a decision whether to cancel or continue with proposed surgery.

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- Transfer the patient to a high care area (eg intensive care or high dependency unit) for further observation and treatment. Anaphylactic reactions may take several hours to fully resolve and the patient must be closely observed during this time.

Less severe reactions

Anaphylaxis sometimes results in less severe reactions which are not life threatening. Treatment is similar to the regime above, but i/v adrenaline may not be required. Manage the ABC as described, and assess the response. Drugs such as ephedrine or methoxamine may be effective to treat hypotension along with i/v fluids. However, whenever the patient's appears to be worsening always use adrenaline.

Diagnosis and Investigations

Diagnosis is made on clinical grounds - though it may not be possible to define exactly which agent precipitated the attack. Make a record of events in the notes and when appropriate inform the patient and his/her general practitioner. If the patient requires further anaesthesia or surgery avoid the use of the suspected precipitating agents.

Some specialised laboratories can estimate Tryptase (a breakdown product of histamine) which can help to confirm the diagnosis. Take blood into glass tubes 60 minutes after the reaction. This test is unavailable in many places.

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Sponsored by: World Federation of Societies of Anaesthesiologists, Level 8, Imperial House, 15-19 Kingsway, London WC2B 6TH, United Kingdom. Tel: +44 171 836 5652. Fax: +44 171 8365616 E-mail: wfsa@compuserve.com

Typeset by: Angela Frost *Printed in Great Britain by:* Media Publishing

Correspondence to: Dr I H Wilson, Anaesthetics Dept., Royal Devon & Exeter Healthcare NHS Trust, Barrack Road, Exeter, EX2 5DW, UK. E-mail: jain.wilson5@virgin.net

Subscriptions to: Dr Charlie Collins, Anaesthetics Dept., Royal Devon & Exeter Healthcare NHS Trust, Barrack Road, Exeter, EX2 5DW, UK.

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