EDITORIAL

The safety of patients undergoing anaesthesia depends on a number of factors, the most vital of which is a trained anaesthetist with the patient at all times.

Clinical monitoring by the anaesthetist includes movement of the reservoir bag, colour of the lips and tongue, observation of the airway and pattern of respiration, strength and nature of the pulse, quality of the heart sounds and the blood pressure reading. These signs have been used for many years, and in many countries continue to be the only monitoring available during anaesthesia and surgery.

Over the past ten years equipment for monitoring patients during anaesthesia has improved dramatically and at the same time, mortality resulting from anaesthesia has decreased substantially. It is clear that the information provided by these monitors supplements in a most valuable way the senses of the anaesthetist. Of all the monitors developed there is no doubt that the most useful is the pulse oximeter. Early detection of hypoxia and inadequate oxygen delivery has saved many lives and it is hoped that eventually every anaesthetist will have access to a pulse oximeter. Unfortunately, at present, most pulse oximeters cost over £1000, well beyond the budget of many hospitals. If prices fall, then their use should become more widespread.

It is clear that modern technology, when available, has the potential to increase the safety and quality of our anaesthetics. Nevertheless it is not the presence of monitoring machinery which makes an anaesthetic safe, but the presence of a well-trained alert anaesthetist - and such a person can produce high quality anaesthesia even in the absence of sophisticated machines. "Monitor" for us should be something we do, rather than something we have!

This article of Update contains advice on using monitoring equipment, along with a selection of other articles. We welcome letters in response to any of our articles and we are also delighted to receive requests for specific topics that readers would like to see covered. If you would like to contribute to Update please contact the editor by email (iain.wilson5@virgin.net) to discuss your suggestion.

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CARDIOVASCULAR PHARMACOLOGY FOR ANAESTHETISTS

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INTRODUCTION

The subject of cardiovascular pharmacology is diverse, and this article concentrates on areas relevant to the anaesthetist. The pathophysiology of common diseases of the heart and circulation is discussed before considering the drugs used for treatment.

In general, the action of drugs is either by (a) alteration of myocardial contractility or heart rate, (b) alteration of conduction of the cardiac action potential or, (c) vasodilatation or vasoconstriction of coronary and peripheral vessels. This article aims to build on subjects covered in previous issues, in particular: "The Autonomic Nervous System" 1995;5: 3-6, "Cardiovascular Physiology" 1999; 10: 2-8, and "Pharmacology of Vasopressors and Inotropes" 1999; 10: 14-17.

ANGINA

Pathophysiology

The normal myocardium accounts for 11 % of the total body oxygen consumption, but the coronary circulation receives only 4 % of the cardiac output. In other words, considering its metabolic demands, the myocardium is one of the more poorly perfused tissues in the body. As a consequence, any pathological disturbance that changes the *myocardial oxygen balance* can result in myocardial ischaemia. This balance of myocardial oxygen supply against demand is dependent on the following:

The most common cause of angina is the build up of *atheroma*, composed of cholesterol and other lipids, in larger coronary arteries. The obstruction to blood flow may become so severe that not enough blood can pass through the arteries to supply the myocardium when oxygen demand increases, for example during exercise. The ischaemic muscle produces the characteristic symptoms of *angina pectoris*, probably because metabolites produced by myocardial contraction accumulate in the poorly perfused tissue. These symptoms include retrosternal chest pain or tightness, which often radiates to the arms. The pain is usually caused or increased by exercise, and relieved by rest and treatment with nitrates.

Anti-anginal drugs

Treatment of patients with significant coronary artery disease aims to achieve an "even" myocardial oxygen balance. Drugs are used to; (a) reduce preload (nitrates), (b) reduce heart rate, myocardial work and oxygen consumption (beta-blockers), (c) maximise coronary vasodilatation (calcium channel blockers).

Nitrates are the drugs of first choice. Their main action is peripheral vasodilation, either venous (low doses), or both venous and arterial (higher doses). This vasodilation is mediated by production of nitric oxide (NO), and increased levels of intracellular guanosine 3',5'-monophosphate (cGMP) in vascular smooth muscle. The resulting pooling of blood in the capacitance vessels (veins) reduces venous return and decreases ventricular volume. This reduction in distension of the heart wall decreases oxygen demand and

Myocardial oxygen supply	Myocardial oxygen demand
Heart rate	Heart rate
Coronary perfusion pressure	Ventricular preload and afterload
Arterial oxygen content	Contractility
Coronary artery diameter	

the pain of angina is relieved quickly. At the same time nitrates improve myocardial oxygen supply by increasing the coronary blood flow to the endocardium. Cardiac output is usually unchanged or decreased slightly, and a reflex tachycardia occurs in normal subjects. However in patients with heart failure, who have a high systemic vascular resistance, cardiac output may increase and there is little change in the heart rate.

Glyceryl trinitrate (GTN) is a short-acting nitrate, with a duration of action of 30 minutes. It is more useful in preventing attacks of angina than in stopping them once they have begun. It may be given by a sublingual, transdermal or intravenous route. In the latter case, between 40-80% of the dose may be absorbed by the plastic giving set. Longer acting nitrates are more stable and may be effective for several hours, depending on the drug and formulation used. (eg. isosorbide dinitrate, isosorbide mononitrate).

Unwanted effects of nitrates include dilation of cranial vessels causing headaches, which can limit the dose used. More serious side effects are tachycardia and hypotension. These result respectively in increased myocardial oxygen demand and decreased coronary perfusion, both of which have an adverse effect on myocardial oxygen balance. Another well recognised problem is the development of tolerance to nitrates. Blood vessels become hypo- or non-reactive to the drugs, particularly when large doses, frequent dosing regimes and long acting formulations are used. To avoid this, nitrates are best used intermittently, allowing a few hours without treatment in each 24 hours period.

Beta blockers These are used to prevent angina as well as to treat hypertension, by blocking beta 1 receptors in the heart. In addition to this blocking action, some beta-blockers can actually stimulate the receptors at a low level. This so called intrinsic activity can be a disadvantage when treating angina. Drugs such as atenolol and metoprolol are the drugs of choice, because they are cardioselective, i.e. only work on beta-1 receptors, and not the beta-2 receptors elsewhere in the body.

Most side effects of beta blockers are the consequence of their blocking action. *Bronchoconstriction* (mediated by beta-2 receptors) is normally of little importance. However in asthmatics it can be life-threatening, and beta blockers should not be used in these patients. *Cold extremities, worsening of peripheral vascular disease, hypoglycaemia* and impotence are also caused by blockade of beta-2 receptors. Some patients with heart disease need a sympathetic "drive" to the heart to maintain adequate cardiac output, and blockade of beta-1 receptors in these patients can cause heart failure.

Calcium antagonists These drugs act by blocking the calcium channels which open in response to depolarisation of the cell membrane (voltage sensitive channels). Such channels occur in many different cells of the body, but the important pharmacological effects of these drugs are restricted to cardiac and smooth muscle. Calcium antagonists are divided into three sub-groups based on their chemical structure;

    (a)   papaverine derivatives e.g. *verapamil*

    (b)   benzothiazepines e.g. *diltiazem*

    (c)   dihydropyridines e.g. *nifedipine, amlodipine*

Calcium antagonists reduce afterload (by arterial vasodilation), dilate coronary arteries, and reduce cardiac work, thus improving myocardial oxygen balance. Verapamil and diltiazem also have an anti-arrhythmic effect, whereas the dihydropyridines predominantly affect vascular smooth muscle. Adverse effects of calcium antagonists include postural hypotension, flushing, peripheral oedema and constipation. All calcium antagonists have a negative inotropic effect, especially verapamil, and should not be used in patients in cardiac failure.

CARDIAC FAILURE

Introduction

Cardiac failure is common and the incidence is increasing in many countries. Despite adequate ventricular filling, the failing heart is unable to deliver as much blood as the tissues require, even when myocardial contractility is increased by the sympathetic nervous system. The causes of heart failure are numerous, but can be classified as follows: (a) endocardial disease, (b) myocardial disease, (c) pericardial disease and, (d) congenital heart disease. Cardiac failure may be precipitated by the factors listed below:

Disturbance of heart rate or rhythm

- Drugs
- Anaemia
- Infection or fever
- Pulmonary embolism
- Fluid overload
- Hypertension
- Pregnancy
- Hyper- or hypothyroidism

Pathophysiology

Cardiac output is the product of heart rate and stroke volume (CO = HR x SV). Stroke volume is determined by three factors; *preload, afterload and contractility*. The effect of changes in any of these variables can be illustrated graphically by the "Starling curve". In cardiac failure the Starling curve is displaced downwards, meaning that increases in end-diastolic ventricular filling volumes (preload) are required to maintain performance.

There are adaptive mechanisms to improve cardiac output, including sodium and water retention by activation of the renin-angiotensin-aldosterone system. This increases blood volume, and thereby preload, but may in turn cause unwanted oedema in both peripheral tissues and the lung. A rise in catecholamine release leads to increased heart rate and contractility, but also an increase in systemic vascular resistance (afterload). In long standing cardiac failure there is hypertrophy of the muscle mass and enlargement of the ventricles.

Drugs used in heart failure

Where possible, underlying disease and the precipitating factors mentioned above should be investigated and treated. The treatment of heart failure aims to:

Diuretics increase the excretion of sodium and water. By reducing the circulating volume they reduce preload and oedema. Loop diuretics such as frusemide are often used in acute as well as chronic heart failure. Low serum levels of potassium, calcium and magnesium are a common side-effect. At high parenteral doses frusemide may cause renal failure (interstitial nephritis) and deafness (auditory nerve damage), especially when it is given rapidly. Toxic effects are more common when frusemide is given in combination with aminoglycoside antibiotics or to patients with impaired renal function.

Nitrates These are used in the overdistended, acutely failing heart to reduce preload, pulmonary venous pressure and oedema. Their mode of action is discussed above.

Inotropic drugs

Digoxin is used to treat heart failure, especially when associated with atrial fibrillation. It is discussed in detail below. Sympathomimetic agents such as dopamine and dobutamine are given by intravenous infusion in severe, acute heart failure. Phosphodiesterase inhibitors such as milrinone improve contractility by increasing myocardial intracellular calcium. They are given in severe heart failure unresponsive to other drugs.

Angiotensin converting enzyme (ACE) inhibitors Drugs such as **captopril** are potent arterial and venous vasodilators. They block the renin-angiotensin system and reduce both preload and afterload, with a resulting increase in cardiac output. Despite the fall in arterial pressure the sympathetic system is not activated, and the decrease in heart rate improves the myocardial oxygen balance. Increased renal blood flow and reduced release of aldosterone causes an increased excretion of sodium and water, thus reducing circulating volume. ACE inhibitors are the most appropriate vasodilators for the long term treatment of heart failure.

(a)	optimise ventricular filling pressures	*Diuretics* *Nitrates*
(b)	improve contractility (inotropic drugs)	*Digoxin* *Sympathomimetic agents* *Phosphodiesterase inhibitors*
(c)	reduce cardiac work by reducing afterload (vasodilators)	*ACE inhibitors* *Prazosin*

Prazosin dilates coronary arteries, peripheral arterioles and veins by blocking alpha-1 receptors. Afterload is reduced, cardiac output increases and there is little reflex tachycardia.

ARRHYTHMIAS

Pathophysiology

Cardiac arrhythmias are common in the peri-operative period (see also page 16) but most are benign and cause no harm to the patient. Arrhythmogenic factors include drugs, ischaemia and altered biochemical and physiological states. Disturbances of cardiac rhythm, although complex, can be classified on the basis of electrophysiology as follows:

(a) **Arrhythmias of sinus origin:** electrical conduction follows the normal pathway but can be either too fast, too slow or irregular.

(b) **Ectopic rhythms:** these can be due to either abnormal automaticity (spontaneous discharge) or re-entry. In the former, single or multiple groups of myocardial cells take over as the pacemaker, giving either atrial or ventricular ectopic rhythms. Re-entry tachycardias involve the cardiac impulse being transmitted to a functional "ring" of conducting tissue, one side of which has a unidirectional (one-way) block. In this situation the impulse is conducted in a circular fashion in the ring, re-exciting the neighbouring myocardium immediately after its refractory (unresponsive) period. Re-entry mechanisms are implicated in atrial flutter and both supraventricular and ventricular tachycardias.

(c) **Conduction blocks:** these can occur at the level of either the atrioventricular node (first, second and third degree blocks) or the His-Purkinje fibres (bundle branch blocks).

(d) **Pre-excitation syndromes:** in this situation the normal delay at the atrioventricular node is bypassed by an accessory pathway, leading to premature ventricular depolarisation. Wolf-Parkinson-White and Lown-Ganong-Levine are the two most common pre-excitation syndromes.

Anti-arrhythmic drugs

The Vaughan-Williams classification of anti-arrhythmic drugs is on the basis of mechanism of action. Although widely used, it does not include digoxin and adenosine, which both have useful anti-arrhythmic actions.

Class I

Membrane stabilising drugs: fast depolarisation of the cell membrane is inhibited by blocking the inward flux of sodium ions. This class is further sub-divided into three groups based on the effect on duration of the action potential. Lignocaine is the most widely used drug in this class, and can be used for all forms of ventricular arrhythmia. Other membrane stabilising drugs include quinidine, procainamide, mexiletine and flecainide.

Class II

Beta-blockers: these drugs reduce automaticity, and increase the action potential duration in the ventricles as well as the refractory period at the atrioventricular node. Examples include propranolol, metoprolol and the shorter acting esmolol.

Class III

Prolongation of action potential and refractory period: amiodarone is the drug of choice, although bretylium is also used. They have an anti-fibrillatory effect by increasing electrical stability.

Class IV

Calcium antagonists: verapamil is the only useful agent, and slows conduction at the atrioventricular node in the treatment of supraventricular arrhythmias.

Digoxin is a cardiac glycoside, and sometimes classified as a Class V drug. It is the only anti-arrhythmic drug available for the treatment of atrial fibrillation that does not have negative inotropic or vasodilator effects. It is therefore used widely for heart failure as well as arrhythmias. Its direct action is to block the sodium/potassium ion exchange pump in the cell membrane. This eventually causes a rise in calcium ions within the cell, which increases contractility. Digoxin slows the conduction of the action potential, mostly at the atrioventricular node, but also in other parts of the heart. Digoxin also acts indirectly by increasing parasympathetic activity via the vagus nerve, further slowing atrioventricular node conduction. The drug is used to control the heart rate in atrial fibrillation, by limiting the ventricular response to atrial discharge.

High doses of digoxin can cause serious arrhythmias, such as complete heart block and ventricular ectopic beats,

which may lead to ventricular tachycardia or fibrillation. Less serious side-effects are nausea, vomiting, visual disturbance and headache, but these may precede toxicity. Plasma levels can be measured to ensure therapeutic levels are not exceeded. Some factors enhance toxicity, such as hypokalaemia, hypomagnesaemia, hypercalcaemia, hypoxia, acidosis and myocardial ischaemia. The dose of digoxin must be reduced in renal failure, and if there is a known drug interaction (e.g. amiodarone, verapamil, quinidine).

Adenosine is a naturally occurring molecule which is a metabolite of adenosine monophosphate. It acts via specific adenosine receptors to cause coronary vasodilation and reduced conduction at the sino-atrial and atrioventricular nodes. It is particularly useful in treating re-entry supraventricular tachycardias, but has no effect on ventricular tachycardia. It has a very short half-life (10 seconds), and is given by rapid intravenous injection. The short duration of action and low incidence of adverse effects makes adenosine useful in diagnosing broad complex tachycardias as either supraventricular or ventricular in origin.

HYPERTENSION

Pathophysiology

In most patients with raised blood pressure there is no obvious cause; this is called *essential hypertension*. The pathogenesis of hypertension is complex but the following factors have been implicated: (a) increased sympathetic activity, (b) sodium retention and an increased circulating volume, (c) increased vascular rigidity and reactivity, (d) increased circulating catecholamines and activation of the renin-angiotensin-aldosterone system, and (e) abnormal baroreceptor responses. High blood pressure is associated with a reduced life expectancy, because of an increased risk of stroke and coronary artery disease; also other end-organ disease, such as retinopathy and renal failure.

Anti-hypertensive drugs

Arterial pressure increases with age and therefore there is no absolute value at which treatment should be started. Untreated hypertension leads to increased perioperative morbidity and mortality. In the presence of other cardiovascular risk factors such as diabetes, hyperlipidaemia or smoking, the threshold for treatment should be lower. As a general rule elective surgery should be delayed if the resting diastolic pressure is greater than 110 mm Hg.

Based on the pathogenesis of essential hypertension described above, there are different classes of anti-hypertensive drugs. They are used alone or in various combinations. *Diuretics* (either thiazide or loop diuretics) reduce sodium and extracellular volume and are often the first-line drugs. Another important group are the vasodilators, such as *ACE-inhibitors, calcium antagonists,* and the direct vasodilator *hydralazine*. Adrenergic blocking agents such as *beta-blockers* and the alpha 1-receptor antagonist *prazosin* are also widely used.

Treatment of severe hypertension

Although severe hypertension (e.g. diastolic pressure above 140 mmHg) can often be managed with oral treatment, this may not be suitable for the peri-operative patient, or when there are life threatening complications such as encephalopathy or heart failure. In these situations blood pressure can be controlled with the intravenous agents described below, which have the advantage of a rapid onset of action. The dose should be titrated against the response, because rapid falls in blood pressure can cause reduced cerebral perfusion and infarction. Before starting such treatments in the peri-operative patient, factors which may be aggravating the hypertension should be identified and treated. These include inadequate analgesia, hypothermia, hypoxia and withdrawal of normal anti-hypertensive drugs.

Labetalol This drug is both an alpha-1 and beta adrenergic receptor blocker, with a ratio of alpha:beta activity of about 1:5. As well as emergency treatment of severe hypertension, it is also used in the treatment of pre-eclampsia and to provide hypotension for certain surgical procedures. Labetalol has a half-life of between three and six hours depending on the dose given, and can cause hepatic damage, even after short periods of treatment. It is given intravenously in increments of 5-10mg up to a maximum of 200mg.

Hydralazine is an arteriolar vasodilator and thus reduces systemic vascular resistance, causing a reflex tachycardia. It is widely used in hypertension associated with pre-eclampsia, but its onset of action may be up to 20 minutes. Headache, nausea, vomiting, and flushing are common side effects, and it can cause angina in patients with ischaemic heart disease. In the obstetric patient the aim is to keep the blood pressure below 170/110 mmHg, using doses of 5-10mg which can be repeated after 30 minutes if necessary.

Sodium Nitroprusside (SNP) This acts directly on vascular smooth muscle and causes arteriolar and venous dilation. As a consequence blood pressure falls and a reflex tachycardia occurs. SNP acts very rapidly and the duration of action is only a few minutes. The drug can produce toxicity by production of cyanide, and there are maximum recommended doses for both acute and longer term use. GTN, which is discussed elsewhere, can also be used for rapid control of high blood pressure.

CARDIOVASCULAR EFFECTS OF ANAESTHETICS

Inhalational agents

All volatile agents depress myocardial contractility, but this effect is most marked with *halothane* and *enflurane*. With the exception of halothane they all decrease systemic vascular resistance, contributing further to the fall in blood pressure and resulting in a reflex tachycardia. During halothane anaesthesia systemic vascular resistance is unchanged and, due to vagal stimulation, bradycardias and nodal rhythms are common. Unlike other volatile agents halothane sensitises the heart to the arrhythmogenic effects of catecholamines, and ventricular ectopics are often seen. High levels of circulating catecholamines can cause ventricular tachycardia or ventricular fibrillation, especially in the presence of hypercarbia, which can occur in a patient spontaneously breathing halothane. *Ether* causes sympathetic stimulation, catecholamine release and, to a certain degree, vagus nerve blockade. As a result there is an increase in cardiac output, heart rate and systemic vascular resistance, so blood pressure is well maintained.

Intravenous induction agents

Most induction agents are cardiovascular depressants. The greatest effect is seen with *propofol*, which may cause a marked fall in blood pressure, systemic vascular resistance and heart rate, the latter due to central vagal stimulation. *Thiopentone* has similar effects, although less pronounced, and there is a reflex tachycardia mediated by the baroreceptor reflex. This can result in increased myocardial oxygen consumption and a consequent increase in coronary blood flow. Benzodiazepines such as *midazolam* and *diazepam* are associated with cardiovascular stability, and only high doses will cause cardiovascular depression. *Etomidate* provides the most cardiovascular stability, with only slight changes in haemodynamic variables. Etomidate has little effect on myocardial oxygen balance. *Ketamine*, in contrast to other induction agents, is a potent cardiovascular stimulant by increasing sympathetic nervous discharge, although its *direct* effect on the myocardium is negatively inotropic. On induction there is a marked rise in heart rate and blood pressure caused by central nervous stimulation and an increase in circulating catecholamines.

ANAESTHESIA AND CHRONIC RENAL FAILURE

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Chronic Renal Failure (CRF) may be caused by primary renal disease or by systemic diseases which also affect the kidney. A decrease in nephron function occurs and can lead to a typical clinical pattern. CRF only becomes biochemically evident when less than 40% of the nephrons are functioning. Dialysis (either peritoneal or haemodialysis) is generally not required until less than 10% of nephrons are functioning. Patients with CRF are more likely to have associated atheroma formation and hypertension.

Preoperative Assessment and Treatment of Medical Problems in Renal Failure

The following factors should be considered when assessing a patient for anaesthesia prior to either an elective or emergency procedure.

Fluid balance In CRF sodium and water excretion is relatively fixed and often reduced. The kidneys can have difficulty handling both large fluid loads and dehydration. The degree of hydration should be assessed in the usual way using skin turgor, examination of the mucous membranes, jugular venous pressure, presence of dependent oedema and presence of pulmonary oedema on auscultation. Invasive measurement of central venous pressure may occasionally be indicated. Many patients on dialysis regimens will know their normal hydrated weight and their fluid allowance per day.

The patient must be normovolaemic prior to surgery. Fluid resuscitation should normally be with normal saline but if there has been blood loss this might also have to be replaced.

Biochemical balance Although numerous biochemical abnormalities can exist and the potassium can be low, the most significant biochemical problems related to severe uncorrected renal disease are hyperkalaemia and acidosis.

Hyperkalaemia is defined as a serum potassium of more than 5 mmol/l. ECG changes become apparent at 6-7 mmol/l and immediate treatment is needed if the serum potassium is over 7 mmol/l. ECG changes include tall peaked T waves, shortened QT intervals, widened QRS complexes and loss of P waves. Eventually the QRS complexes merge into the T waves to produce a sine wave pattern. Ventricular fibrillation may occur at serum concentrations over 10 mmol/l.

Methods of treating a high serum potassium in an emergency include:

a) Administration of 0.5ml/kg of 10% calcium gluconate (max 20 ml). This has an immediate but transient stabilising effect on the myocardial cells.

b) 50mls of 50% glucose as an intravenous bolus or infusion. Glucose and insulin will produce an immediate migration of potassium into the cells thus reducing the serum level. Blood glucose levels should be closely monitored but unless the patient is diabetic, endogenous insulin will be secreted and maintain normal glycaemia. Alternatively 5-10 units of soluble insulin may be added to the infusion. Apart from the risk of errors which may occur, the patient may also become hypoglycaemic as secretion of endogenous insulin is also stimulated.

c) Administration of 1-2 mmol/kg sodium bicarbonate intravenously over 5-10 minutes. This provides a large sodium and fluid load which may not be desirable.

d) Nebulised salbutamol 2.5 - 5mg will assist in moving K^+ into the cells.

Total body potassium levels can then be reduced:

a) By dialysis.

b) With calcium resonium (0.5 g/kg) 8 hourly either rectally or orally. This takes approximately 12 hours to produce an effect.

c) By the introduction of a low potassium diet.

Acidosis can best be improved by dialysis. Administration of bicarbonate solution should only be considered when the pH is <7.2. Side effects of bicarbonate solutions include hypernatraemia and volume overload.

Cardiovascular status *Hypertension* may be a primary problem, secondary to chronic salt and water retention or to excess renin production. Blood pressure must be controlled preoperatively. *Ischaemic heart disease* is more common and should be assessed preoperatively. *Pulmonary oedema* may occur with fluid overload or with left ventricular failure. *Pericarditis* can occur in uraemic conditions.

Respiratory function *Pulmonary oedema* and *pleural effusions* both cause a decrease in lung compliance, functional residual capacity and increased ventilation/ perfusion mismatch. All these increase the likelihood of hypoxia and are best treated by fluid removal with diuretics or dialysis.

Haematological function *Chronic anaemia* is common in patients with CRF who are not being treated with erythropoeitin and is usually well tolerated. Unless the patient has ischaemic heart disease the haemoglobin level may be maintained at around 7-8 g/dl. Uraemic patients may have a *bleeding tendency* due to a decrease in platelet adhesion and fragility of the vessel walls.

Gastrointestinal system Anorexia, nausea, vomiting, bleeding from stress ulceration, diarrhoea and hiccups are all common symptoms. These can exacerbate dehydration. Nutrition is often poor and this can impair wound healing.

Central nervous system Uraemia causes malaise, fatigue, decreased mental ability and eventually coma. Severe uraemia or fluid or electrolyte imbalance may cause convulsions.

Endocrine system *Hyperparathyroidism* leads to demineralisation of bone making patients more susceptible to fractures. Diabetic control may be difficult because of decreased sensitivity to insulin.

Multiple medications Patients may be taking corticosteroids or other immunosuppressants which cannot be stopped. Other treatments may have been prescribed for associated diseases.

Dialysis regimen Those patients with end stage renal failure who are maintained on peritoneal dialysis should continue dialysing until they go to theatre. Haemodialysis should be ideally undertaken with minimum heparinisation up to 12 hours prior to elective surgery.

Pharmacology of Anaesthetic Agents in Renal Failure

The *excretion* of water soluble drugs and their active metabolites will be impaired. For drugs which are renally

excreted the half life increases slowly with deteriorating renal function until severe nephron loss at which point the half life increases sharply with further reductions in renal function. Dialysis can only usually replace a small part of the excretory capacity of the healthy kidney.

Induction agents Their effect is terminated by redistribution. All of these agents are myocardial depressants and should be administered cautiously in patients with heart disease.

Muscle relaxants *Suxamethonium* should be avoided if hyperkalaemia is present.

Some non-depolarising muscle relaxants depend on the kidney for elimination. *Atracurium* is the agent of choice as it undergoes spontaneous Hoffman degradation at body temperature.

Vecuronium and *mivacurium* are safe to use in renal failure as only small percentages are excreted renally.

Gallamine should be avoided and *pancuronium, alcuronium, pipecuronium, curare and doxacurium* should be used with caution. Potentiation of neuromuscular blockade may occur in the presence of a metabolic acidosis, hypokalaemia, hypermagnesaemia, or hypocalcaemia and with medications such as aminoglycosides. Monitor neuromuscular blockade whenever possible.

Opioids *Morphine* is metabolised in the liver to morphine-6-glucuronide which has about half the sedative effect of morphine with a markedly prolonged half life. Pethidine is partially metabolised to norpethidine which is less analgesic and has excitatory and convulsant properties. Both of these metabolites may accumulate in renal failure after repeated doses or with infusions. Standard intraoperative use will not usually cause problems. When available, morphine is preferable to pethidine.

Fentanyl and **alfentanil** can be used as normal.

Benzodiazepines can be used in renal failure.

Inhalational agents There is decreased elimination of the fluoride ions which are significant metabolites of enflurane, sevoflurane and methoxyflurane which can worsen renal function, so these inhalational agents should be avoided especially if used at low flows.

Non steroidal anti inflammatory agents (NSAIDS) should be avoided as all decrease renal blood flow and may precipitate complete renal failure.

Conduct of Anaesthesia

Premedication Oral sedatives such as diazepam or temazepam may be used. H_2 antagonists or non particulate antacids (e.g. sodium citrate) should be given if oesophageal reflux is a problem.

Anaesthesia *Venous access* may be difficult. If future haemodialysis is planned it is important to preserve AV fistulas and potential fistula sites. Forearm and antecubital veins should be avoided if possible in these patients.

Full monitoring must be established prior to induction of anaesthesia, with special attention being paid to the ECG and blood pressure. The patient should be kept well oxygenated and haemodynamically stable. Hypovolaemia and hypotension worsen renal function therefore blood and other fluid losses should be carefully replaced. If possible the shorter acting sedative agents should be used.

If spinal or epidural anaesthesia is being performed fluid preloading should be kept to a minimum and vasoconstrictors used to maintain the blood pressure. Otherwise postoperative fluid overload may necessitate dialysis.

Postoperatively *Postoperative fluid balance* must be meticulous and prompt action taken to limit vomiting and replace any fluids lost. Some patients may require *haemodialysis* for fluid overload postoperatively but this should be delayed if possible as the patient will have to be heparinised. Some patients may become drowsy on relatively low doses of analgesics.

Oxygen (2-3 litres/minute nasally or 3-4 litres/minute via face mask) should be administered for 48 hours after major abdominal or thoracic surgery and 24 hours after intermediate surgery.

PREVENTING ACUTE RENAL FAILURE

Previously healthy patients most at risk of developing acute renal tubular necrosis are those with massive haemorrhage, multiple trauma, sepsis, extensive burns and crush injuries, especially if they already have some degree of renal impairment. Renal failure is diagnosed when urine output is persistently <0.5ml/kg/hour or the serum creatinine rises.

The maintenance of normovolaemia and an adequate renal perfusion pressure are the two most important factors in avoiding acute renal failure. The underlying clinical problem should be controlled and treated as far as possible and adequate hydration guided if necessary by central venous pressure measurement. The urine output should be

measured hourly and should be maintained above 1 ml/kg/hr.

Only after the patient is well resuscitated with fluid should vasoactive drugs be used to maintain an adequate mean arterial blood pressure for the patient (this will depend on the patients preoperative blood pressure). If the patient becomes oliguric (urine output < 0.5 ml/kg/hr) despite adequate hydration and blood pressure administration of frusemide can be considered, up to 240 mg intravenously over 1 hour. If no diuresis develops further administration of frusemide is useless. *Dopamine and mannitol* both increase urine output but also increase the oxygen demand of the kidney so frusemide is preferred. Low dose dopamine has not been shown to have any protective effect on the kidney.

All nephrotoxic drugs should be avoided if possible. These include *NSAIDS* and *ACE inhibitors*. If *aminoglycosides* are essential their serum levels must be monitored.

Electrolytes including potassium, sodium and bicarbonate must be measured at least daily during the perioperative period. Adequate calorie intake is essential and must be established as soon as possible postoperatively.

Table 1:   Preoperative assessment of the patient in chronic renal failure

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Fluid balance
Biochemistry and acid base status
Associated illnesses
Associated medications
Dialysis regimen

Table 2:   Common biochemical and haematological abnormalities in chronic renal failure

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Hyper- (or hypo-)kalaemia
Hypo- (or hyper-)natraemia
Hyperphosphataemia
Hypocalcaemia
Metabolic acidosis
Normochromic normocytic anaemia

Table 3:   Treatment of the acutely oliguric patient

Control the underlying cause if known
Ensure the patient is well hydrated using invasive monitoring if necessary
Ensure the blood pressure is normal or above normal for that patient
After fluid resuscitation try frusemide 240 mg over 1 hour
Avoid all non-essential nephrotoxic drugs
Adjust doses of renally excreted drugs
Measure sodium, potassium, bicarbonate and urea and/or creatinine twice daily
Establish low potassium nutrition as soon as possible

PRACTICAL APPLICATIONS OF PULSE OXIMETRY

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INTRODUCTION

Pulse oximetry is a useful method of monitoring patients in many circumstances,and in the face of limited resources, the pulse oximeter may represent a wise choice of monitor, as with training it allows for the assessment of several different patient parameters.

Pulse oximeters are now a standard part of perioperative monitoring which give the operator a non-invasive indication of the patient's cardio-respiratory status. Having been successfully used in intensive care, the recovery room and during anaesthesia, they have been introduced in other areas of medicine such as general wards apparently without staff undergoing adequate training in their use[1]. The technique of pulse oximetry does have pitfalls and limitations and it is possible that patient safety may be compromised with untrained staff. This article is therefore intended for the 'occasional' user of pulse oximetry.

Pulse oximeters measure the arterial oxygen saturation of haemoglobin. The technology involved[2] is complicated but there are two basic physical principles. First, the absorption of light at two different wavelengths by haemoglobin differs depending on the degree of oxygenation of haemoglobin. Second, the light signal following transmission through the tissues has a pulsatile component, resulting from the changing volume of arterial blood with each pulse beat. This can be distinguished by the microprocessor from the non-pulsatile component resulting from venous, capillary and tissue light absorption.

The function of a pulse oximeter is affected by many variables, including: ambient light; shivering; abnormal haemoglobins; pulse rate and rhythm; vasoconstriction and cardiac function. A pulse oximeter gives no indication of a patient's ventilation, only of their oxygenation, and thus can give a false sense of security if supplemental oxygen is being given. In addition, there may be a delay between the occurrence of a potentially hypoxic event such as respiratory obstruction and a pulse oximeter detecting low oxygen saturation. However, oximetry is a useful non-invasive monitor of a patient's cardio-respiratory system, which has undoubtedly improved patient safety in many circumstances.

What does a pulse oximeter measure?

1. The oxygen saturation of haemoglobin in arterial blood - which is a measure of the average amount of oxygen bound to each haemoglobin molecule. The percentage saturation is given as a digital readout together with an audible signal varying in pitch depending on the oxygen saturation.

2. The pulse rate - in beats per minute, averaged over 5 to 20 seconds.

A pulse oximeter gives no information on any of these other variables:

●   The oxygen content of the blood

●   The amount of oxygen dissolved in the blood

●   The respiratory rate or tidal volume i.e. ventilation

●   The cardiac output or blood pressure

Systolic blood pressure can be estimated by noting the pressure at which the plethysmograph trace reappears during deflation of a proximal non-invasive blood pressure cuff.
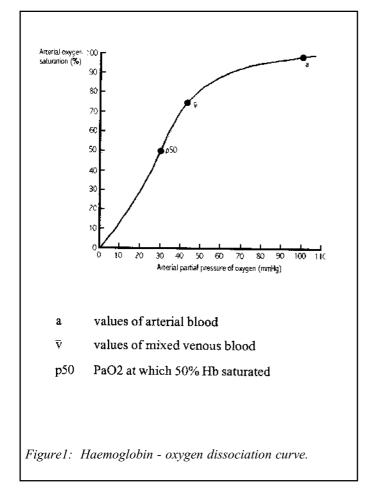
Principles of modern pulse oximetry

Oxygen is carried in the bloodstream mainly bound to haemoglobin. One molecule of haemoglobin can carry up to four molecules of oxygen, which is then 100% saturated with oxygen. The average percentage saturation of a population of haemoglobin molecules in a blood sample is the oxygen saturation of the blood. In addition, a very small quantity of oxygen is carried dissolved in the blood, which can become important if the haemoglobin levels are extremely low. The latter, however, is not measured by pulse oximetry.

The relationship between the arterial partial pressure of oxygen (PaO_2) and the oxygen saturation is described by the haemoglobin-oxygen dissociation curve (see figure 1). The sigmoid shape of this curve facilitates unloading of oxygen in the peripheral tissues where the PaO_2 is low and oxygen is required for respiration. The curve may be shifted to the left or right by various patient characteristics e.g. recent blood transfusion, pyrexia.

A pulse oximeter consists of a peripheral probe, together with a microprocessor unit, displaying a waveform, the oxygen saturation and the pulse rate. Most oximeters also have an audible pulse tone, the pitch of which is proportional to the oxygen saturation - useful when one cannot see the oximeter display. The probe is placed on a peripheral part of the body such as a digit, ear lobe or the nose. Within the probe are two light emitting diodes (LED's), one in the visible red spectrum (660nm) and the other in the infrared spectrum (940nm). The beams of light pass through the tissues to a photodetector. During passage through the tissues, some light is absorbed by blood and soft tissues depending on the concentration of haemoglobin. The amount of light absorption at each light frequency depends on the degree of oxygenation of haemoglobin within the tissues.

The microprocessor can select out the absorbance of the pulsatile fraction of blood, i.e. that due to arterial blood, from constant absorbance due to non-pulsatile venous or capillary blood and other tissue pigments. Several recent advances in microprocessor technology have reduced the effects of interference on pulse oximeter function. Time division multiplexing, whereby the LED's are cycled: red on, then infrared on, then both off, many times per second,

helps to eliminate background 'noise'. Quadrature division multiplexing is a further advance in which the red and infrared signals are separated in phase rather than time and then recombined in phase later. In this way, an artefact due to motion or electromagnetic interference may be eliminated since it will not be in the same phase of the two LED signals once they are recombined.

Saturation values are averaged out over 5 to 20 seconds. The pulse rate is also calculated from the number of LED cycles between successive pulsatile signals and averaged out over a similar variable period of time, depending on the particular monitor.

From the proportions of light absorbed at each light frequency, the microprocessor calculates the ratio of the two. Within the oximeter memory is a series of oxygen saturation values obtained from experiments performed in which human volunteers were given increasingly hypoxic mixtures of gases to breath. The microprocessor compares the ratio of absorption at the two light wavelengths measured with these stored values, and then displays the oxygen saturation digitally as a percentage and audibly as a tone of varying pitch. As it is unethical to desaturate human volunteers below 70%, it is vital to appreciate that oxygen saturation values below 70% obtained by pulse oximetry are unreliable.

Reflection pulse oximetry uses reflected rather than transmitted light on a single-sided monitor. It can therefore be used more proximally anatomically e.g. forehead, bowel, although it may be difficult to secure. Other than using specific reflection spectra, the principles are the same as for transmission oximetry.

Practical tips to the successful use of pulse oximetry:

- Plug the pulse oximeter in to an electrical socket, if available, to recharge the batteries.

- Turn the pulse oximeter on and wait for it to go through its calibration and check tests.

- Select the probe you require with particular attention to correct sizing and where it is going to go. The digit should be clean (remove nail varnish).

- Position the probe on the chosen digit, avoiding excess force.

- Allow several seconds for the pulse oximeter to detect the pulse and calculate the oxygen saturation.

- Look for a displayed waveform.
  Without this, any reading is meaningless.

a   values of arterial blood

v̄   values of mixed venous blood

p50   PaO2 at which 50% Hb saturated

Figure1: Haemoglobin - oxygen dissociation curve.

- Read off the displayed oxygen saturation and pulse rate.

* Be cautious interpreting figures where there has been an instantaneous change in saturation - for example 99% falling suddenly to 85%. This is physiologically not possible.

- If in doubt, rely on your clinical judgement, rather than the value the machine gives.

Alarms

- If the Low Oxygen Saturation alarm sounds, check that the patient is conscious if that is appropriate. Check the airway and make sure the patient is breathing adequately. Lift the chin or apply other airway manoeuvres as appropriate. Give oxygen if necessary. Call for help.

- If the Pulse Not Detected alarm sounds, look for the displayed waveform on the pulse oximeter. Feel for a central pulse. If there is no pulse, call for help, start the procedures for Basic and Advanced Life Support. If there is a pulse, try repositioning the probe, or put the probe on a different digit.

- On most pulse oximeters, the alarm limits for oxygen saturation and pulse rate can be altered according to your needs. However, do not alter an alarm just to stop it sounding - it could be telling you something important!

Uses of pulse oximetry

- Simple, portable "all-in-one" monitor of oxygenation, pulse rate and rhythm regularity, suitable for "field" use.

- As a safe, non-invasive monitor of the cardio-respiratory status of high-dependency patients - in the emergency department, during general and regional anaesthesia, postoperatively and in intensive care. This includes procedures such as endoscopy, where often frail patients are given sedative drugs such as midazolam. Pulse oximeters detect the presence of cyanosis more reliably than even the best doctors when using their clinical judgement.

- During the transport of patients - especially when this is noisy - for example in aircraft, helicopters or ambulances. The audible tone and alarms may not be heard, but if a waveform can be seen together with an acceptable oxygen saturation, this gives a global indication of a patient's cardio-respiratory status.

- To assess the viability of limbs after plastic and orthopaedic surgery and, for example, following vascular grafting, or where there is soft tissue swelling or aortic dissection. As a pulse oximeter requires a pulsatile signal under the sensor, it can detect whether a limb is getting a blood supply.

- As a means of reducing the frequency of blood gas analysis in intensive care patients- especially in paediatric practice where vascular (arterial) access may be more difficult.

- To limit oxygen toxicity in premature neonates supplemental oxygen can be tapered to maintain an oxygen saturation of 90% - thus avoiding the damage to the lungs and retinas of neonates. Although pulse oximeters are calibrated for adult haemoglobin, HbA, the absorption spectra of HbA and HbF are almost identical over the range used in pulse oximetry, so the technique remains reliable in neonates.

- During thoracic anaesthesia - when one lung is being collapsed down - to determine whether oxygenation via the remaining lung is adequate or whether increased concentrations of oxygen must be given.

- Fetal oximetry- a developing technique that uses reflectance oximetry, using LEDs of 735nm and 900nm. The probe is placed over the temple or cheek of the fetus, and needs to be sterile and sterilisable. They are difficult to secure and the readings are variable, for physiological and technical reasons. Hence the trend is more useful than the absolute value.

Limitations of pulse oximetry

- **Not a monitor of ventilation** A recent case report[3,4] highlighted the false sense of security provided by pulse oximetry. An elderly woman postoperatively in the recovery room was receiving oxygen by face mask. She became increasingly drowsy, despite having an oxygen saturation of 96%. The reason was that her respiratory rate and minute volume were low due to residual neuromuscular block and sedation, yet she was receiving high concentrations of inspired oxygen, so her oxygen saturation was maintained. She ended up with an arterial carbon dioxide concentration of 280 mmHg (normal 40 mmHg) and was ventilated for 24 hours on intensive care. Thus oximetry gives a good estimation of adequate

oxygenation, but no direct information about ventilation, particularly as in this case, when supplemental oxygen is being administered.

- **Critically ill patients** It may be less effective in very sick patients, because tissue perfusion may be poor and thus the oximeter probe may not detect a pulsatile signal.

- **Waveform presence** If there is no waveform visible on a pulse oximeter, any percentage saturation values obtained are meaningless.

- **Inaccuracies** Bright overhead lighting, shivering and motion artefact may give pulsatile waveforms and saturation values when there is no pulse.

(i) Abnormal haemoglobins such as methaemoglobinaemia, for example following overdose of prilocaine, cause readings to tend towards 85%.

(ii) Carboxyhaemoglobin, caused by carbon monoxide poisoning, causes saturation values to tend towards 100%. A pulse oximeter is extremely misleading in cases of carbon monoxide poisoning for this reason and should not be used. CO-oximetry is the only available method of estimating the severity of carbon monoxide poisoning.

(iii) Dyes and pigments, including nail varnish, may give artificially low values.

(iv) Vasoconstriction and hypothermia cause reduced tissue perfusion and failure to register a signal.

(v) Rare cardiac valvular defects such as tricuspid regurgitation cause venous pulsation and therefore venous oxygen saturation is recorded by the oximeter.

(vi) Oxygen saturation values less than 70% are inaccurate as there are no control values to compare them to.

(vii) Cardiac arrhythmias may interfere with the oximeter picking up the pulsatile signal properly and with calculation of the pulse rate.

NB. Age, sex, anaemia, jaundice and dark-skin have little or no effects on oximeter function.

- **Lag monitor** This means that the partial pressure of oxygen can have fallen a great deal before the oxygen saturation starts to fall. If a healthy adult patient is given 100% oxygen to breathe for a few minutes and then ventilation ceases for any reason, several minutes may elapse before the oxygen saturation starts to fall. A pulse oximeter in these circumstances warns of a potentially fatal complication several minutes after it has happened. The pulse oximeter has been described as "a sentry standing at the edge of the cliff of desaturation." because of this fact. The explanation of this lies in the sigmoid shape of the haemoglobin / oxygen dissociation curve (figure 1).

- **Response delay** due to signal averaging. This means that there is a delay after the actual oxygen saturation starts to drop because the signal is averaged out over 5 to 20 seconds.

- **Patient safety** there have been one or two case reports of skin burns or pressure damage under the probe because some early probes had a heater unit to ensure adequate skin perfusion. The probe should be correctly sized, and should not exert excessive pressure. Special probes are now available for paediatric use.

The penumbra effect re-emphasises the importance of correct probe positioning. This effect causes falsely low readings and occurs when the probe is not symmetrically placed, such that the pathlength between the two LEDs and the photodetector is unequal, causing one wavelength to be "overloaded". Repositioning of the probe often leads to sudden improvement in saturation readings. The penumbra effect may be compounded by the presence of variable blood flow through cutaneous pulsatile venules. Note that the waveform may appear normal despite the penumbra effect as it measures predominantly one wavelength only.

Alternatives to pulse oximetry?

- Bench CO-oximetry is the gold standard - and is the classic method by which a pulse oximeter is calibrated. The CO-oximeter calculates the actual concentrations of haemoglobin, deoxyhaemoglobin, carboxyhaemoglobin and methaemoglobin in the sample and hence calculates the actual oxygen saturation. CO-oximeters are much more accurate than pulse oximeters - to within 1%, but they give a 'snapshot' of oxygen saturation, are bulky, expensive and require constant maintenance as well as requiring a sample of arterial blood to be taken.

- Blood gas analysis - requires an invasive sample of arterial blood. It gives the 'full picture', including arterial partial pressure of oxygen and carbon dioxide, arterial pH, actual and standardised base excess and actual and standardised bicarbonate concentrations. Many blood gas analysers report a calculated saturation which is less accurate than that provided by the pulse oximeter.

- Lag monitor - time delay between potentially hypoxic event such as respiratory obstruction and a pulse oximeter detecting low oxygen saturation.

- Inaccuracies: ambient light; shivering and vasoconstriction; abnormal haemoglobins; and alterations in pulse rate and rhythm.

- Advances in microprocessor have led to improved signal processing.

SUMMARY POINTS

- Pulse oximeters give non-invasive estimation of the arterial haemoglobin oxygen saturation.

- Useful in: anaesthesia, recovery, intensive care (including neonatal), patient transport.

- 2 principles involved:
  (1) Differential light absorption by haemoglobin and oxyhaemoglobin.

  (2) Identification of pulsatile component of signal.

- No direct indication of a patient's ventilation, only of their oxygenation.

Further Reading

1. Stoneham MD, Saville GM, Wilson IH. Knowledge about pulse oximetry among medical and nursing staff. Lancet 1994:334:1339-1342.

2. Moyle JTB. Pulse oximetry. Principles and Practice Series. Editors: Hahn CEW and Adams AP. BMJ Publishing, London, 1994.

3. Davidson JAH, Hosie HE. Limitations of pulse oximetry: respiratory insufficiency - a failure of detection. BMJ 1993;307:372-373.

4. Hutton P, Clutton-Brock T. The benefits and pitfalls of pulse oximetry. BMJ 1993;307:457-458.

ECG MONITORING IN THEATRE

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Cardiac arrhythmias during anaesthesia and surgery occur in up to 86% of patients. Many are of clinical significance and therefore their detection is of considerable importance. This article will discuss the basic principles of using the ECG monitor in the operating theatre. It will describe the main rhythm abnormalities and give practical guidance on how to recognise and treat them.

The continuous oscilloscopic ECG is one of the most widely used anaesthetic monitors, and in addition to displaying arrhythmias it can also be used to detect myocardial ischaemia, electrolyte imbalances, and assess pacemaker function. A 12 lead ECG recording will provide much more information than is available on a theatre ECG monitor, and should where possible, be obtained pre-operatively in any patient with suspected cardiac disease.

The ECG is a recording of the electrical activity of the heart. It does not provide information about the mechanical function of the heart and cannot be used to assess cardiac output or blood pressure. Cardiac function under anaesthesia is usually estimated using frequent measurements of blood pressure, pulse, oxygen saturation, peripheral perfusion and end tidal CO_2 concentrations. Cardiac performance is occasionally measured directly in theatre using Swan Ganz catheters or oesophageal Doppler techniques, although this is uncommon.

The ECG monitor should always be connected to the patient before induction of anaesthesia or institution of a regional block. This will allow the anaesthetist to detect any change in the appearance of the ECG complexes during anaesthesia.

Connecting an ECG monitor

Although an ECG trace may be obtained with the electrodes attached in a variety of positions, conventionally they are placed in a standard position each time so that abnormalities are easier to detect. Most monitors have 3 leads and they are connected as follows:

- **Red** - right arm, (or second intercostal space on the right of the sternum)

- **Yellow** - left arm (or second intercostal space on the left of the sternum)

- **Black** (or Green) - left leg (or more often in the region of the apex beat.)
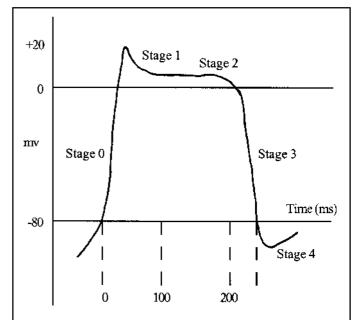

Figure 1. The Cardiac Muscle Action Potential

Stage O = depolarisation, opening of voltage gated sodium channels

Stage I = initial rapid repolarisation, closure of sodium channels and chloride influx.

Stage 2 = plateau - opening of voltage gated calcium channels.

Stage 3 = repolarisation, potassium efflux.

Stage 4 = diastolic pre potential drift.

This will allow the Lead I, II or III configurations to be selected on the ECG monitor. Lead II is the most commonly used. (See page 18 for other lead positions and their uses).
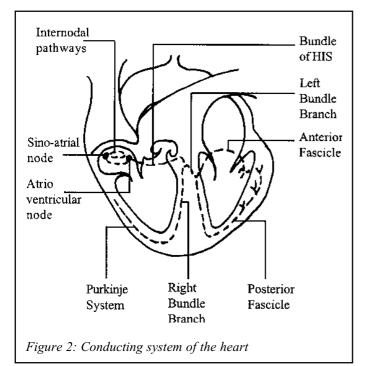
The cables from the electrodes usually terminate in a single cable which is plugged into the port on the ECG monitor.

A good electrical connection between the patient and the electrodes is required to minimise the resistance of the skin. For this reason gel pads or suction caps with electrode jelly are used to connect the electrodes to the patients skin. However when the skin is sweaty the electrodes may not stick well, resulting in an unstable trace. When electrodes are in short supply they may be reused after moistening with saline or gel before being taped to the patient's chest. Alternatively, an empty 1000ml iv infusion bag may be cut open to allow it to lie flat (in the form of a flat piece of plastic) on the patient's chest. If 3 small

holes are made in 3 of the corners electrodes may be stuck on one side of the plastic allowing the electrode gel to make contact with the skin. This device can be cleaned at the end of the operation and laid on the next patient allowing electrodes to be used repeatedly.

Principles of the ECG

The ECG is a recording of the electrical activity of the heart. An electrical recording made from one myocardial muscle cell will record an action potential (the electrical activity which occurs when the cell is stimulated). The ECG records the vector sum (the combination of all electrical signals) of all the action potentials of the myocardium and produces a combined trace.

At rest the potential difference across the membrane of a myocardial cell is -90mv (figure 1). This is due to a high intracellular potassium concentration which is maintained by the sodium/potassium pump. Depolarisation of a cardiac cell occurs when there is a sudden change in the permeability of the membrane to sodium. Sodium floods into the cell and the negative resting voltage is lost (stage 0). Calcium follows the sodium through the slower calcium channels resulting in binding between the intracellular proteins actin and myosin which results in contraction of the muscle fibre (stage 2). The depolarisation of a myocardial cell causes the depolarisation of adjacent cells and in the normal heart the depolarisation of the entire myocardium follows in a co-ordinated fashion. During repolarisation potassium moves out of the cells (stage 3) and the resting negative membrane potential is restored.

THE CONDUCTING SYSTEM OF THE HEART

The specialised cardiac conducting system (figure 2) consists of :

The Sinoatrial (SA) node, internodal pathways, Atrioventricular (AV) node, bundle of HIS with right and left bundle branches and the Purkinje system. The left bundle branch also divides into anterior and posterior fascicles. Conducting tissue is made up of modified cardiac muscle cells which have the property of automaticity, that is they can generate their own intrinsic action potentials as well as responding to stimulation from adjacent cells. The conducting pathways within the heart are responsible for the organised spread of action potentials within the heart and the resulting co-ordinated contraction of both atria and ventricles.

In pacemaker tissue, after repolarisation has occurred, the membrane potential gradually rises to the threshold level for channel opening, at which point sodium floods into the cell and initiates the next action potential (figure 3). This gradual rise is called the pacemaker (or pre-potential) and is due to a decrease in the membrane permeability to potassium ions which result in the inside of the cell becoming less negative. The rate of rise of the pacemaker potential is the main determinant of heart rate and is increased by adrenaline (epinephrine) and sympathetic stimulation and decreased by vagal stimulation and hypothermia. Pacemaker activity normally only occurs in the SA and AV nodes, but there are latent pacemakers in other parts of the conducting system which take over when firing from the SA or AV nodes is depressed. Atrial and ventricular muscle fibres do not have pacemaker activity and discharge spontaneously only when damaged or abnormal.
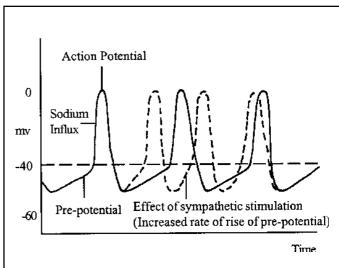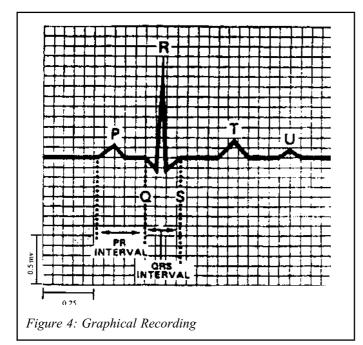

Figure 2: Conducting system of the heart

Figure 3: Action Potential in Pacemaker Tissue

GRAPHICAL RECORDING

On a paper trace the ECG is usually recorded on a time scale of 0.04 seconds/mm on the horizontal axis and a voltage sensitivity of 0.1mv/mm on the vertical axis (figure 4). Therefore, on standard ECG recording paper, 1 small square represents 0.04seconds and one large square 0.2 seconds. In the normal ECG waveform the P wave represents atrial depolarisation, the QRS complex ventricular depolarisation and the T wave ventricular repolarisation.

Figure 4: Graphical Recording

- The P- R interval is taken from the start of the P wave to the start of the QRS complex. It is the time taken for depolarisation to pass from the SA node via the atria, AV node and His - Purkinje system to the ventricles.

- The QRS represents the time taken for depolarisation to pass through the His - Purkinje system and ventricular muscles. It is prolonged with disease of the His - Purkinje system.

- The Q -T interval is taken from the start of the QRS complex to the end of the T wave. This represents the time taken to depolarise and repolarise the ventricles.

- The S - T segment is the period between the end of the QRS complex and the start of the T wave. All cells are normally depolarised during this phase. The ST segment is changed by pathology such as myocardial ischaemia or pericarditis.

LEAD POSITIONS

The ECG may be used in two ways. A 12 lead ECG may be performed which analyses the cardiac electrical activity from a number of electrodes positioned on the limbs and across the chest. A wide range of abnormalities may be detected including arrhythmias, myocardial ischaemia, left ventricular hypertrophy and pericarditis.

During anaesthesia, however, the ECG is monitored using only 3 (or occasionally 5) electrodes which provide a more restricted analysis of the cardiac electrical activity and cannot provide the same amount of information that may be revealed by the 12 lead ECG.

The term 'lead' when applied to the ECG does not describe the electrical cables connected to the electrodes on the patient. Instead it refers to the positioning of the 2 electrodes being used to detect the electrical activity of the heart. A third electrode acts as a neutral. During anaesthesia one of 3 possible 'leads' is generally used. These leads are called bipolar leads as they measure the potential difference (electrical difference) between two electrodes. Electrical activity travelling towards an electrode is displayed as a positive (upward) deflection on the screen, and electrical activity travelling away as a negative (downward) deflection. The leads are described by convention as follows:
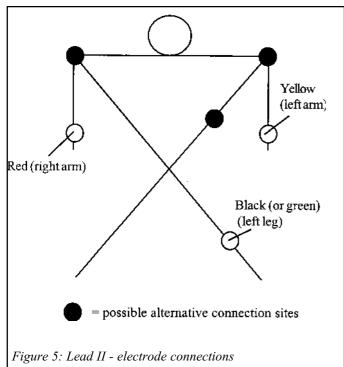

ECG Normal Values

P - R interval	0.12 - 0.2 seconds (3-5 small squares of standard ECG paper)
QRS complex duration	less than or equal to 0.1 seconds (2.5 small squares)
Q - T interval corrected for heart rate (QTc) QTc = QT/ RR interval	less than or equal to 0.44 seconds

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- **Lead I** - measures the potential difference between the right arm electrode and the left arm electrode. The third electrode (left leg) acts as neutral.

- **Lead II** - measures the potential difference between the right arm and left leg electrode.

- **Lead III** - measures the potential difference between the left arm and left leg electrode.

Most monitors can only show one lead at a time and therefore the lead that gives as much information as possible should be chosen. The most commonly used lead is lead II (figure 5) - a bipolar lead with electrodes on the right arm and left leg as above. This is the most useful lead for detecting cardiac arrhythmias as it lies close to the cardiac axis (the overall direction of electrical movement) and allows the best view of P and R waves.

For detection of myocardial ischaemia the CM5 lead is useful (figure 6). This is a bipolar lead with the right arm electrode placed on the manubrium and left arm electrode placed at the surface marking of the V5 position (just above the 5th interspace in the anterior axillary line). The left leg lead acts as a neutral and may be placed anywhere - the C refers to 'clavicle' where it is often placed. To select the CM5 lead on the monitor, turn the selector dial to 'lead I'. This position allows detection of up to 80% of left ventricular episodes of ischaemia, and as it also displays arrhythmias it can be recommended for use
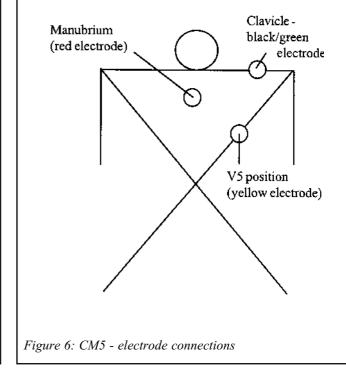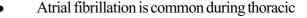
in most patients. The CB5 lead is another bipolar lead which has one electrode positioned at V5 and the other over the right scapula. This results in improved QRS and P wave voltages allowing easier detection of arrhythmias and ischaemia. Many other electrode positions have been described including some used during cardiac surgery, for example oesophageal and intracardiac ECG's.

CARDIAC ARRHYTHMIAS

The detection of cardiac arrhythmias and the determination of heart rate is the most useful function of the intraoperative ECG. Anaesthesia and surgery may cause any type of arrhythmia including:

- Transient supraventricular and ventricular tachycardias due to sympathetic stimulation during laryngoscopy and intubation.

- Bradycardias produced by surgical manipulation resulting in vagal stimulation. Severe bradycardia and asystole may result. It is more common in children because the sympathetic innervation of the heart is immature and vagal tone predominates. Bradycardias are most commonly seen in ophthalmic surgery due to the oculocardiac reflex. Generally the heart rate will improve when the surgical stimulus is removed.

- Atrial fibrillation is common during thoracic

Figure 5: Lead II - electrode connections

Figure 6: CM5 - electrode connections

surgery.

Drugs may also cause changes in cardiac rhythm eg;

- Halothane and nitrous oxide may cause junctional rhythms - (these will be detailed later). Halothane has a direct effect on the SA node and conducting system leading to a slowing in impulse generation and conduction and predisposes to re-entry phenomena. Catecholamines also have potent effects on impulse conduction, so the interaction of halothane and exogenous or endogenous catecholamines may cause ventricular arrhythmias. Ventricular ectopic beats are common. However rhythm disturbances such as ventricular tachycardia or rarely ventricular fibrillation may occur. The presence of cardiac disease, hypoxia, acidosis, hypercarbia (raised CO_2 level) or electrolyte disturbances will increase the likelihood of these arrhythmias.

- Arrhythmias occurring during halothane anaesthesia can often be resolved by reducing the concentration of halothane, ensuring adequate ventilation thereby preventing hypercarbia, increasing the inspired oxygen concentration and providing an adequate depth of anaesthesia for the surgical procedure. Tachyarrhythmias in the presence of halothane anaesthesia are uncommon if ventilation is adequate, and the use of adrenaline infiltration for haemostasis is limited to solutions of 1:100,000 or less and the dose in adults is not greater than 0.1mg in 10 minutes or 0.3mg per hour).

- Drugs increasing heart rate include ketamine, ether, atropine and pancuronium. Drugs decreasing heart rate include opioids, beta blockers and halothane.

Action Plan - when faced with an abnormal rhythm on the ECG monitor

Assess the vital signs - A.B.C.

- Check the airway is patent

- Check the patient is breathing adequately or is being ventilated correctly

- Listen for equal air entry into both lungs

- Circulation - check pulse, blood pressure, oxygen saturation. Is there haemodynamic compromise? Does the abnormal rhythm on the monitor match the pulse that you can feel?

Consider the following:

- Increase the inspired oxygen concentration

- Reduce the inspired volatile agent concentration

- Ensure that ventilation is adequate to prevent CO_2 build up. Check end tidal CO_2 where this measurement is available

- Consider what the surgeon is doing - is this the cause of the problem? Eg: traction on the peritoneum or eye causing a vagal response. If so ask them to stop while you treat the arrhythmia.

- If the arrhythmia is causing haemodynamic instability, rapid recognition and treatment is required. However, many abnormal rhythms encountered in every day practice will respond to the above basic measures - sometimes even before identification of the exact rhythm abnormality is possible.

PRACTICAL INTERPRETATION AND MANAGEMENT OF ARRHYTHMIAS

When interpreting arrhythmias a paper strip is often easier to read than an ECG monitor. Where this is not possible from the theatre monitor it may be possible to obtain a paper trace by connecting a defibrillator, most of which have a facility for printing a rhythm strip. The following basic points should be considered:

Examining an ECG strip:

1. What is the ventricular rate?

2. Is the QRS complex of normal duration or widened?

3. Is the QRS regular or irregular?

4. Are P waves present and are they normally shaped?

5. How is atrial activity related to ventricular activity?

1.   What is the ventricular rate? Arrhythmias may be classified as fast or slow:

- Tachyarrhythmias - rate greater than 100/min

- Bradyarrhythmias - rate less than 60/min

- Calculate approximate ventricular rate on a paper strip by counting the number of large squares between each QRS complex and dividing this number into 300 which will give the rate in beats/minute.

2.   *Is the QRS complex of normal duration or widened? Arrhythmias may be due to abnormal impulses arising from the:*
- atria = a supraventricular ryhthm
- AV node = a nodal or junctional rhythm
- or the ventricles = a ventricular arrhythmia

Supraventricular and nodal rhythms arise from a focus above the ventricles. Since the ventricles still depolarise via the normal His-Purkinje system the QRS complexes are of normal width (< 0.1sec - 2.5 small squares) - and are therefore termed 'narrow complex' rhythms. Arrhythmias arising from the ventricles will be 'broad complex' with a QRS width of >0.1sec. The QRS complexes are widened in these patients since depolarisation is via the ventricular muscle rather than the His-Purkinje system and takes longer. In a few cases where there is an abnormal conduction pathway from atria to ventricles a supraventricular rhythm may have broad complexes. This is called 'aberrant conduction'.

3.   *Is the QRS regular or irregular?*
The presence of an irregular rhythm will tend to suggest ectopic beats (either atrial or ventricular), atrial fibrillation, atrial flutter with variable block or second degree heart block with variable block - see page 29.

4.   *Are there P waves present and are they normally shaped?*
The presence of P waves indicates that the atria have depolarised and gives a clue to the likely origin of the rhythm. Absent P waves associated with an irregular ventricular rhythm suggest atrial fibrillation whilst a saw tooth pattern of P waves is characteristic of atrial flutter. If the P waves are upright in leads II and AVF they have originated from the sinoatrial node. However, if the P waves are inverted in these leads, it indicates that the atria are being activated in a retrograde direction ie: the rhythm is junctional or ventricular.

5.   *How is atrial activity related to ventricular activity?*
Normally there will be one P wave per QRS complex. Any change in this ratio indicates a blockage to conduction at some point in the pathway from the atria to the ventricles.

CLASSIFICATION OF ARRHYTHMIAS

Arrhythmias may be divided into narrow complex and broad complex for the purpose of rapid recognition and management.

Narrow complex arrhythmias - arise above the bifurcation of the bundle of His. The QRS duration is less than 0.1s (2.5 small squares) duration

Broad complex arrhythmias - usually arise either from the ventricles or less commonly are conducted abnormally from a site above the ventricles so that delay occurs (this is called aberrant conduction). The QRS duration is greater than 0.1s (2.5 small squares).

NARROW COMPLEX RHYTHMS:
- Sinus arrhythmia
- Sinus tachycardia
- Sinus bradycardia
- Junctional / AV nodal tachycardia
- Atrial tachycardia, atrial flutter
- Atrial fibrillation
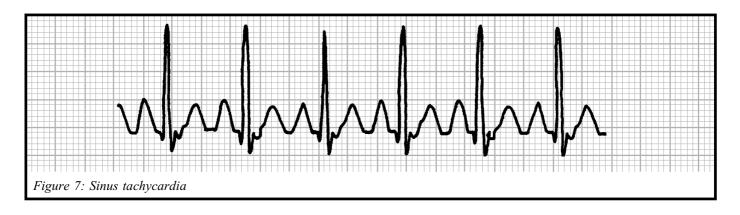- Atrial ectopics

BROAD COMPLEX RHYTHMS
- Ventricular ectopics
- Ventricular tachycardia
- Supraventricular tachycardia with aberrant conduction
- Ventricular fibrillation

NARROW COMPLEX ARRHYTHMIAS

Sinus arrhythmia This is irregular spacing of normal complexes associated with respiration. There is a constant P-R interval with beat to beat change in the R-R interval. It is a normal finding especially in young people.

Sinus tachycardia (figure 7). There is a rate greater than 100/min in adults. Normal P-QRS-T complexes are evident. Causes include:
- Inadequate depth of anaesthesia
- Pain / surgical stimulation
- Fever / sepsis
- Hypovolaemia
- Anaemia

Figure 7: Sinus tachycardia

- Heart failure

- Thyrotoxicosis

- Drugs eg atropine, ether, ketamine, catecholamines

Management : correction of any underlying cause where possible. Beta blockers may be useful if tachycardia causes myocardial ischaemia in patients with ischaemic heart disease, but should be avoided in asthma and used with caution in patients with heart failure.

Sinus bradycardia (figure 8).

This is defined as a heart rate of less then 60 beats/minute in an adult.

It may be normal in athletic patients and may also be due to vagal stimulation during surgery - see above.

Other causes include:

- Drugs eg; beta blockers, digoxin, anticholinesterase drugs, halothane, second dose of suxamethonium (occasionally first dose in children)

- Myocardial infarction

- Sick sinus syndrome

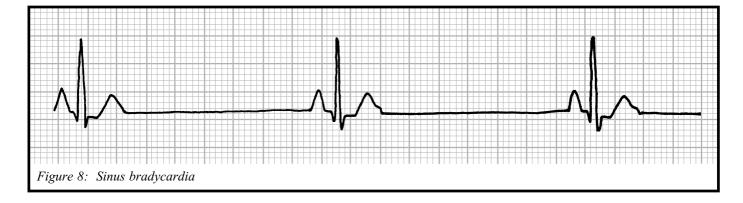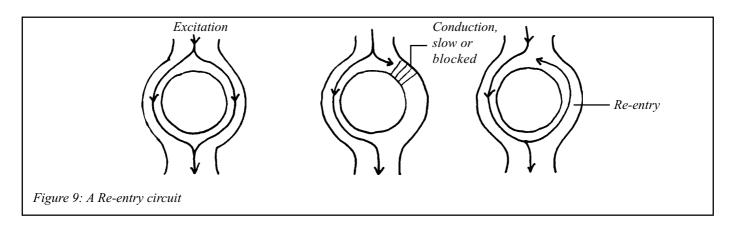- Raised intracranial pressure

- Hypothyroidism

- Hypothermia

Management It is often not necessary to correct a sinus bradycardia in a fit young person, unless the rate is less than 45 - 50 beats per minute, and / or there is haemodynamic compromise. However consider:

- Correcting the underlying cause eg: stop the surgical stimulus

- Atropine up to 20 mcg/kg iv or glycopyrolate 10 mcg/kg iv. (Atropine works more rapidly and is usually given in doses of 300-400mcg and repeated if required).

- Patients on beta blockers may be resistant to atropine - occasionally an isoprenaline infusion may be required. Alternatively glucagon (2-10mg) can be used in addition to atropine.

ARRHYTHMIAS DUE TO RE- ENTRY (Circular movement of electrical impulses).

These arrythmias occur where there is an anatomical branching and re-joining of a conduction pathway. Normally conduction would occur down both limbs equally. But if one limb is slower than the other, an impulse may pass normally down one limb but be blocked in the other. Where the pathways rejoin the impulse can then

Figure 8: Sinus bradycardia

Figure 9: A Re-entry circuit

spread backwards up the abnormal pathway. If it arrives at a time when the first pathway is no longer refractory to activation it can pass right round the circuit repeatedly activating it and resulting in a tachycardia (figure 9.) The classical example of this is the Wolf Parkinson White syndrome where there is a relatively large anatomical 'accessory' conduction pathway between the atria and the ventricles. This is called a 'macro re-entry' circuit. Other macro re-entry circuits can occur within the atrial and ventricular myocardium and are responsible for paroxysmal atrial flutter, atrial fibrillation and ventricular tachycardia. In junctional or AV nodal tachycardia there are 'micro re-entry'circuits within the AV node itself.

JUNCTIONAL / AV NODAL TACHYCARDIA (figure 10)

The term Supraventricular Tachycardia (SVT) applies to all tachyarrhythmias arising from a focus above the ventricles. However it is often used to describe junctional (AV nodal) tachycardias arising from micro re-entry circuits in or near the AV node, or as in the Wolf Parkinson White syndrome from an accessory conduction pathway between the atria and the ventricles. The ECG appearance is of a narrow complex tachycardia (QRS<0.1s ie 2.5 small squares of standard ECG paper), with a rate of 150 -200 bpm (figure 10).

The typical features seen on a 12 lead ECG taken when the patient is in sinus rhythm are:

- A short P-R interval

- A slurred upstroke on the R wave (the delta wave - best seen in V4)

- Inverted T waves in V2-5 are characteristic.

Management:

This arrhythmia may be associated with severe circulatory disturbance and needs to be managed as an emergency if it occurs during anaesthesia.

1. In the presence of hypotension, especially where the patient is anaesthetised in theatre, the first line treatment is synchronised direct current cardioversion with 200 - 360 joules.

2. Carotid Sinus Massage - this rarely converts to sinus rhythm but slows the rate and will reveal the underlying rhythm if there is any doubt. It is helpful in differentiating it from atrial flutter and fast atrial fibrillation. (The carotid sinus is a small dilatation of the proximal part of the internal carotid artery at the level of the superior border of the thyroid cartilage. It is vagally innervated and is involved in the control mechanism for causing a fall in heart rate and cardiac output in response to a rise in arterial pressure.

Figure 10: AV Nodal Tachycardia

Gentle pressure on the internal carotid artery at this level may result in a slowing of the heart rate and occasionally termination of a re-entry supraventricular tachycardia. It should NEVER be attempted on both sides at once as this may result in asystole and occlusion of the main arterial blood supply to the brain.) It is contra-indicated in patients with a history of cerebrovascular disease.

3. Adenosine - this slows AV conduction and is especially useful for terminating re-entry SVTs of the Wolf Parkinson White type. Give 3mg iv rapidly preferably via a central or large peripheral vein - followed by a saline flush. Further doses of 6mg and then 12mg may be given at 2 min intervals if there is no response to the first dose. The effects of adenosine last only 10 -15 seconds. It should be avoided in asthma.

4. Verapamil, beta blockers or other drugs such as amiodarone or flecainide may control the rate or convert to sinus rhythm.

- Verapamil 5 -10mg iv slowly over 2 minutes. A further 5mg may be given after 10 minutes if required. Avoid giving concurrently with beta blockers as this may precipitate hypotension and asystole.

- Beta blockers eg: propranolol 1 mg over 1 minute repeated if necessary at 2 minute intervals (maximum 5mg), or sotalol 100mg over 10 minutes repeated 6 hourly if neccesary. Esmolol - a relatively cardio-selective beta blocker with a very short duration of action may be given by infusion at 50 - 200 mcg/kg/minute.

Digoxin should be avoided - it facilitates conduction through the AV accessory pathway in the Wolf Parkinson White syndrome and may worsen the tachycardia. Note that atrial fibrillation in the presence of an accessory pathway may allow very rapid conduction which can degenerate to ventricular fibrillation.

ATRIAL TACHYCARDIA AND ATRIAL FLUTTER (figure 11)

This is due to an ectopic focus depolarising from anywhere within the atria. The atria contract faster than 150 bpm and P waves can be seen superimposed on the T waves of the preceding beats. The AV node conducts at a maximum rate of 200 bpm, therefore if the atrial rate is faster than this, AV block will occur. If the atrial rate is greater than 250 beats/min and there is no flat baseline between P waves, then the typical 'saw tooth ' pattern of atrial flutter waves will be seen.
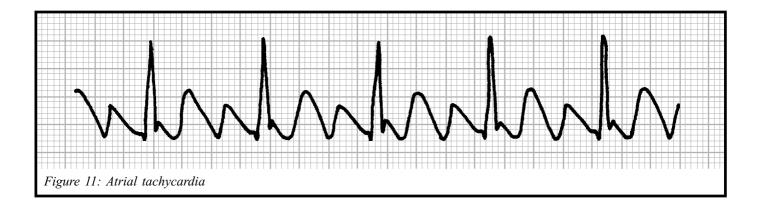
Atrial tachycardia and flutter may occur with any kind of block:

Eg: 2:1, 3:1, or 4:1.

Atrial tachycardia is typically a paroxysmal arrhythmia, presenting with intermittent tachycardia and palpitations, and may be precipitated by anaesthesia and surgery. It is associated in particular with rheumatic valvular disease as well as ischaemic and hypertensive heart disease and may be seen with mitral valve prolapse. It may precede the onset of permanent atrial fibrillation. Atrial tachycardia with 2:1 block is characteristic of digitalis toxicity.

Management

- This arrhythmia is very sensitive to synchronised direct current cardioversion - there is a nearly 100% success rate. Therefore in the anaesthetised patient with any degree of cardiovascular compromise this should be the first line treatment.

- Carotid sinus massage and adenosine will slow AV conduction and reveal the underlying rhythm and block where there is doubt.

- Other drug treatment is as for atrial fibrillation. (see page 25).

Figure 11: Atrial tachycardia

ATRIAL FIBRILLATION (AF - figure 12)

A common arrhythmia encountered in anaesthetic and surgical practice. There is chaotic and unco-ordinated atrial depolarisation, an absence of P waves on the ECG, with an irregular baseline and a completely irregular ventricular rate. Transmission of atrial activity to the ventricles via the AV node depends on the refractory period of the conducting tissue. In the absence of drug treatment or disease which slows conduction, the ventricular response rate will normally be rapid ie: 120 - 200 beats/min.

Common causes of AF include:

- Ischaemia

- Myocardial disease / pericardial disease / mediastinitis

- Mitral valve disease

- Sepsis

- Electrolyte disturbance (particularly hypokalaemia or hypomagnesaemia)
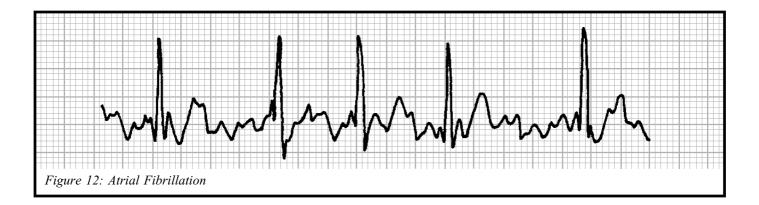
- Thyrotoxicosis

- Thoracic surgery

Since contraction of the atria contributes up to 30% of the normal ventricular filling, the onset of AF may result in a significant fall in cardiac output. Fast AF may precipitate cardiac failure, pulmonary oedema and myocardial ischaemia. Systemic thrombo-embolism may occur if blood clots in the fibrillating atria and subsequently embolises into the circulation. There is a 4% risk per year of an embolic cerebro-vascular episode (CVE = stroke). The treatment of AF is aimed at the restoration of sinus rhythm whenever possible. Where this is not possible, the aim is control of the ventricular rate to <100/ minute and prevention of embolic complications. The management of this arrhythmia will vary depending on how long it has been

present. In acute AF restoration of sinus rhythm is often possible, whereas in longstanding AF control of the ventricular rate is the usual aim of therapy.

Management:

1.   **Acute AF** - Occuring in theatre or of recent onset (less than 48 hours):

- Correction of precipitating factors where possible, especially correction of electrolyte disturbances.

- Synchronised DC cardioversion - for recent onset AF. If AF has been present for more than several hours there is a risk of arterial embolisation unless the patient is anticoagulated. Shock at 200J then at 360J.

- Digoxin can be used acutely to slow the ventricular rate - in the presence of a normal plasma potassium concentration. An intravenous loading dose of 500mcg in 100mls of saline over 20 minutes may be given and repeated at intervals of 4 - 8 hours if necessary until a total of 1 - 1.5mg has been given. This is contraindicated if the patient is already taking digoxin when lower doses are required. There is no evidence that digoxin is useful for converting AF to sinus rhythm or maintaining sinus rhythm once established.

- Amiodarone may be used to restore sinus rhythm - it is especially useful in paroxysmal atrial fibrillation associated with critical illness, and where digoxin or beta blockers cannot be used. A loading dose of 300mg iv via a central vein is given over 1 hour and then followed by 900mg over 23 hours.

- Verapamil 5 -10 mg slowly iv over 2 minutes can be used to control the ventricular rate. Where there is no impairment of left ventricular function or coronary artery disease, the subsequent

Figure 12: Atrial Fibrillation

administration of flecainide 50 - 100mg slowly iv may restore sinus rhythm. However flecainide should only be used where the arrhythmia is life threatening and no other options are open. It should be avoided if left ventricular function is poor or there is evidence of ischaemia.

- Beta blockers are sometimes used to control the ventricular rate but may precipitate heart failure in the presence of an impaired myocardium, thyrotoxicosis or calcium channel blockers, and should be used with caution.

2. **Chronic AF** with a ventricular rate of greater than 100/min. Aim to control the ventricular rate to less than 100/minute. This allows time for adequate ventricular filling and helps maintain the cardiac output.

- Digitalisation - if patient not already taking it. Consider extra digoxin if not fully loaded - beware signs of digoxin toxicity, nausea, anorexia, headache, visual disturbances etc, and arrhythmias especially ventricular ectopics and atrial tachycardia with 2:1 block.

- Beta blockers or verapamil

- Amiodarone

When AF has been present for more than a few hours anticoagulation is necessary before DC cardioversion to prevent the risk of embolisation. Usually patients should be warfarinised for 3 weeks prior to elective DC cardioversion, with regular monitoring of their prothrombin time. An INR of 2 or more is a satisfactory value at which to proceed with cardioversion. Warfarin should then be continued for 4 weeks afterwards. Occasionally when a patient develops AF and is compromised by it, DC cardioversion has to be considered even where anticoagulation is contraindicated (eg recent surgery).

ATRIAL ECTOPIC BEATS (figure 13)

An abnormal P wave is followed by a normal QRS complex. The P wave is not always easily visible on the ECG trace. The term 'ectopic' indicates that depolarisation originated in an abnormal place, ie not the SA node hence the abnormal shape of the P wave. If such a focus depolarises early the beat produced is called an extrasystole or premature contraction and may be followed by a compensatory pause. If the underlying SA node rate is slow, sometimes a focus in the atria takes over and the rhythm is described as an atrial escape, as it occurs after a small delay. Extrasystoles and escape beats have the same QRS appearance on the ECG, but extrasystoles occur early whereas escape beats occur late.

Causes:

- Often occur in normal hearts

- May occur with any heart disease

- Ischaemia, hypoxia

- Light anaesthesia

- Sepsis

- Shock
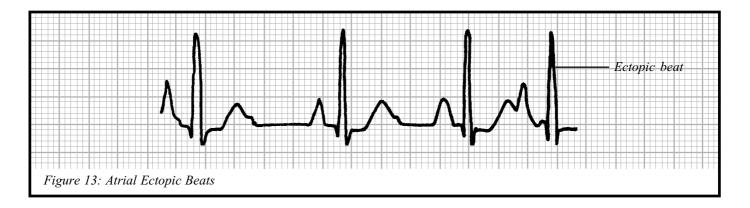
- Anaesthetic drugs are common causes

Management:

- Correction of any underlying cause.

- Specific treatment of atrial ectopic beats is unnecessary unless runs of atrial tachycardia occur - see above.

BROAD COMPLEX ARRHYTHMIAS

Ventricular Ectopic Beats (figure 14)

Depolarisation spreads from a focus in the ventricles by an abnormal, and therefore slow, pathway so the QRS

Figure 13: Atrial Ectopic Beats

Ectopic beat

complex is wide and abnormal. The T wave is also abnormal in shape.

In the absence of structural heart disease these are usually benign. They may be related to associated abnormalities especially hypokalaemia. They are common during dental procedures and anal stretches particularly with halothane, or whenever there is raised CO_2, light anaesthesia or no analgesia associated with halothane anaesthesia. In fit young patients under anaesthesia, they are often of little significance and respond readily to manipulation of the anaesthetic as described in 'first line management'. Small doses of intravenous beta blockers are very commonly effective in this situation.

However they may herald the onset of runs of ventricular tachycardia, and should be taken more seriously where:

- There is a bigeminal rhythm (one ectopic beat with every normal beat).

- If they occur in runs of 2 or more, or where there are more than 5/minute.

- Where they are multifocal (arising from different foci within the ventricles and hence having different shapes).

- Those where the R wave is superimposed on the T wave ('R on T 'phenomenon).

The value of prophylactic treatment has been questioned as it is not known whether this influences the final outcome. However most would recommend treatment in the above four situations or where ventricular tachycardia has already occurred.

Management:

- Correction of any contributing causes identified with the anaesthetic ensuring adequate oxygenation, normocarbia and analgesia. A small dose of beta blocker is worth trying as mentioned above.

- If the underlying sinus rhythm is slow <50 bpm, then increasing this rate using intravenous atropine or glycopyrrolate may be effective as the ventricular ectopics may be a form of escape rhythm.

- Lignocaine is the drug of first choice. An initial loading dose of 50 - 100mg iv over 2 minutes is given followed by infusion of: 4mg/minute - for 30 minutes, then 2mg/minute - for 2 hours and then 1mg/minute. The dose should be reduced in the elderly, in liver disease and where there is bradycardia or hypotension.

- Alternatives include amiodarone 300mg iv (preferably via a central venous catheter) over1 hour, followed by infusion of 900mg over 23 hours. Occasionally bretyllium or procainamide may be used.
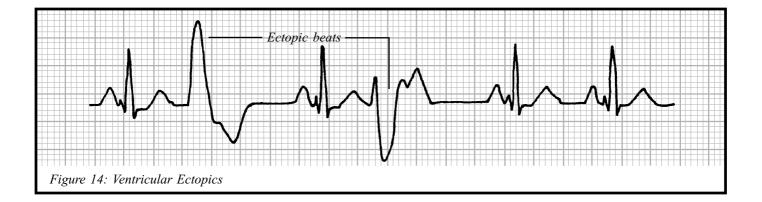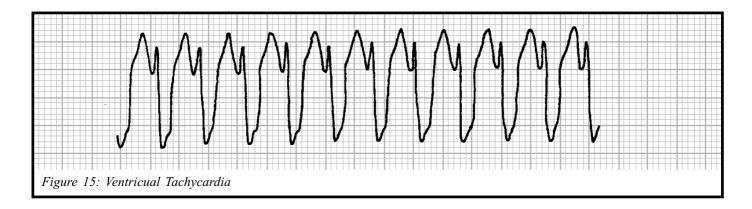
Ventricular tachycardia (VT - figure 15)

In this rhythm a focus in the ventricular muscle depolarises at high frequency. Excitation spreads through the ventricles by an abnormal pathway and therefore the QRS complexes are wide and abnormal.

The appearance is characterised by absent P waves, wide QRS complexes which may be slightly irregular or vary in shape.

VT is a serious, potentially life threatening arrhythmia. It may be triggered intraoperatively by:

- Hypoxia

- Hypotension

- Fluid overload

- Electrolyte imbalance (low K^+, Mg^{2+} etc)

- Myocardial ischaemia

- Injection of adrenaline

Figure 14: Ventricular Ectopics

Figure 15: Ventricual Tachycardia

Management:

- Synchronised direct current cardioversion is the first line treatment if the patient is haemodynamically unstable. This is safe and effective and will restore sinus rhythm in virtually 100% of cases. If the VT is pulseless or very rapid, synchronisation is unnecessary. But otherwise synchronisation is used to avoid a 'shock on T ' phenomenon which may initiate VF. If the patient lapses back into VT, drugs such as lignocaine or amiodarone may be given to sustain sinus rhythm.

- Lignocaine given as a 100mg bolus restores sinus rhythm in up to 60% and may be followed by a maintenance infusion as above.

- Verapamil is ineffective in ventricular tachycardia and may worsen hypotension and precipitate cardiac failure .

Other drugs which may be used if lignocaine fails:

- **Amiodarone** 300mg iv - via a central venous catheter over 1 hour followed by infusion of 900mg over 23 hours.

- **Procainamide** 100mg iv over 5 minutes followed by one or two further boluses before commencing infusion at 3mg/min.

- **Mexiletine** 100 - 250mg iv at 25mg/min followed by infusion 250mg over 1 hour, 125mg/hour for 2 hours, then 500mcg/min.

- **Bretylium tosylate** 400 - 500 mg diluted in 5% dextrose over 10 minutes

- **Propranolol** 0.5 - 1.0mg iv and repeated if necessary particularly if the underlying pathology is myocardial ischaemia or infarction.

- **Sotalol** 100mg iv over 5 minutes. This was shown to be better than lignocaine for acute termination of ventricular tachycardia.

- Overdrive pacing can be used to suppress VT by increasing the heart rate.

Supraventricular tachycardia with aberrant conduction

When there is abnormal conduction from the atria to the ventricles, a supraventricular tachycardia (SVT) may be broad complex as discussed above. This may occur for example if there is a bundle branch block. Sometimes the bundle branch block may be due to ischaemia and may only appear at high heart rates. SVTs may be due to an abnormal or accessory pathway (as in the Wolf Parkinson White syndrome), but during the tachycardia the complex is of normal width as conduction in the accessory pathway is retrograde, ie; it is the normal pathway that initiates the QRS complex. Adenosine may be used diagnostically to slow AV conduction and will often reveal the underlying rhythm if it arises from above the ventricles. In the case of SVT it may also result in conversion to sinus rhythm. In practice however the differentiation of the two is not important, and all such tachycardias should be treated as ventricular tachycardia if there is any doubt.

Ventricular Fibrillation (figure 16)

This results in cardiac arrest. There is chaotic and disorganised contraction of ventricular muscle and no QRS complexes can be identified on the ECG.

Management

Immediate direct current cardioversion as per established resuscitation protocol. (See Update 10).

Figure 16: Ventricular Fibrillation

DISTURBANCES OF CONDUCTION

The wave of cardiac excitation which spreads from the sinoatrial node to the ventricles via the conduction pathways may be delayed or blocked at any point.

First Degree Block (figure 17)

There is a delay in the conduction from the sinoatrial node to the ventricles, and this appears as a prolongation of the PR interval ie greater than 0.2 seconds. It is normally benign but may progress to second degree block - usually of the Mobitz type I. First degree heart block is not usually a problem during anaesthesia.

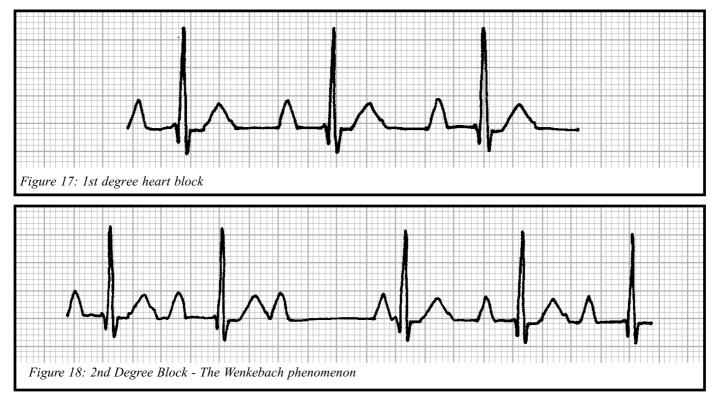### Second Degree Block - Mobitz Type I (Wenkebach) (figure 18)

There is progressive lengthening of the PR interval and then failure of conduction of an atrial beat. This is followed by a conducted beat with a short PR interval and then the cycle repeats itself. This occurs commonly after an inferior myocardial infarction, and tends to be self limiting. It does not normally require treatment although a 2:1 type block may develop with haemodynamic instability.
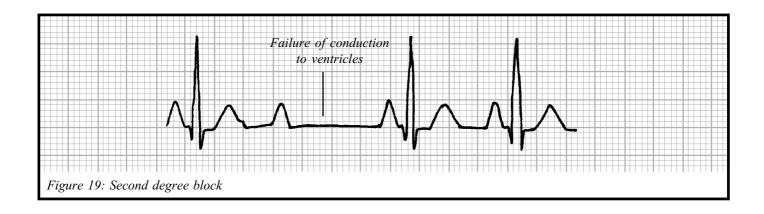
Second Degree Block - Mobitz Type II (figure 19)

If excitation intermittently fails to pass through the AV node or the bundle of HIS, this is the Mobitz type II phenomenon. Most beats are conducted normally but occasionally there is an atrial contraction without a subsequent ventricular contraction. This often progresses to complete heart block and if recognised preoperatively will need expert assessment..

Second Degree Block - 2:1 Type

There may be alternate conducted and non-conducted

Figure 17: 1st degree heart block

Figure 18: 2nd Degree Block - The Wenkebach phenomenon

Figure 19: Second degree block

beats, resulting in 2 P waves for every QRS complex - this is 2:1 block. A 3:1 block may also occur, with one conducted beat and two non-conducted beats. This may also herald complete heart block, and in some situations the placing of a temporary transvenous pacing wire pre-operatively would be recommended.

Complete Heart Block (figure 20)

There is complete failure of conduction between the atria and the ventricles. The ventricles are therefore excited by a slow escape mechanism from a focus within the ventricles. There is no relationship between the P waves and the QRS complexes, and the QRS complexes are abnormally shaped. This may occur occasionally as a transient phenomenon in theatre as a result of vagal stimulation, in which case it often responds to stopping surgery and intravenous atropine. When it occurs in association with acute inferior myocardial infarction, it is due to AV nodal ischaemia and is often transient. Very rarely it may be congenital! However if it occurs with anterior myocardial infarction it indicates more extensive damage including to the HIS - Purkinje system. It may also occur as a chronic state usually due to fibrosis around the bundle of HIS.
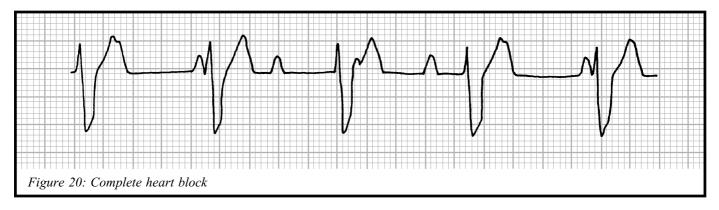
Management

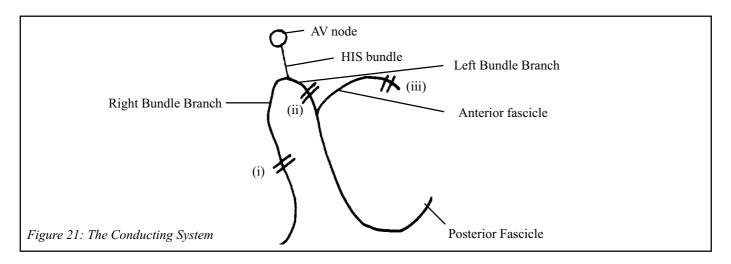● Isoprenaline given by intravenous infusion can be used to increase the ventricular rate

● In the acute situation a temporary transvenous pacing wire may be required. A permanent pacemaker will be required in the longer term if the block is chronic and before contemplating elective surgery.

Bundle Branch Block (figure 21)

If the electrical impulse from the SA and AV nodes reaches the interventricular septum normally the PR interval will be normal. However if there is a subsequent delay in depolarisation of the right or left bundle branches, there will be a delay in depolarisation of part of the ventricular muscle and the QRS complex will be wide and abnormal.

A wide complex rhythm which is present at the start of surgery on initial attachment of the ECG monitor is usually due to bundle branch block (BBB), and is not an indication for cancelling the operation. However this does indicate the importance of attaching the ECG monitor before induction of anaesthesia, particularly where a pre-operative ECG is not available. Any changes on the ECG during anaesthesia and surgery can then easily be compared to the patients ' normal' ie pre-anaesthetic ECG tracing. The definition of which bundle is blocked can only be achieved by analysing a full 12 lead ECG. Two types of BBB are recognised.

Figure 20: Complete heart block

Figure 21: The Conducting System

- **Right Bundle Branch Block** (i). This may indicate problems with the right side of the heart, but a right bundle branch block type pattern with a normal axis and QRS duration is not uncommon in normal individuals.

- **Left Bundle Branch Block** (ii). This often indicates heart disease and makes further interpretation of the ECG other than rate and rhythm impossible.

Other forms of BBB

Bi-Fascicular Block (i and iii). This is a diagnosis which can only be made on a formal 12 lead ECG, and is included for completeness. It is the combination of right bundle branch block and block of the left anterior or posterior fascicle and appears on the ECG as a RBBB pattern with axis deviation. This progresses to complete heart block in a few patients.

Tri-Fascicular Block This is the term sometimes used to indicate the presence of a prolonged PR interval together with a bi-fascicular block.

PRE - OPERATIVE PROPHYLACTIC PACEMAKER INSERTION

Where facilities allow, pacemakers are sometimes inserted prior to surgery in patients who are at risk of developing complete heart block perioperatively. Those at risk of this complication have recently been described by the American college of cardiology and the American heart association. A pacemaker should be considered for:
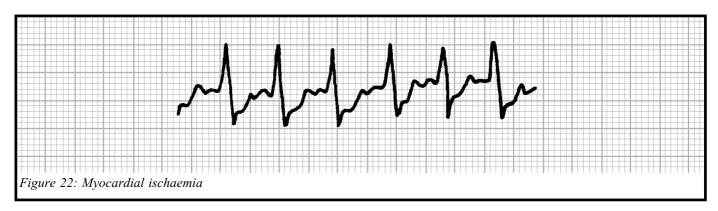
- 3rd degree AV block which is symptomatic or has a ventricular escape rate of less than 40 beats per minute. Where the rate is greater than 40, there is conflicting evidence of benefit but the weight of opinion is in favour of pacing.

- 2nd degree AV block of any type if there is symptomatic bradycardia.

- Asymptomatic 2nd degree heart block or first degree block with symptoms suggestive of sick sinus syndrome (intermittent tachycardia and bradycardia) plus documented relief of symptoms with a temporary pacing wire. In both of these cases the weight of current opinion favours pacing.

- Any type of bundle branch block with intermittent second or third degree block, or syncope should be paced.

- Bifascicular block is relatively common in the elderly and does not require pacing.

DETECTION OF MYOCARDIAL ISCHAEMIA (figure 22).

Cardiac events are the main cause of death following anaesthesia and surgery. Perioperative myocardial ischaemia is predictive of intra and post operative myocardial infarction. The likelihood of detection of ischaemia intraoperatively on the ECG is increased by the use of the CM5 lead as discussed above. This lead has the highest probability of detecting ischaemia, particularly in the lateral wall of the left ventricle which is the zone at greatest risk. Lead II is more likely to detect infero-posterior ischaemia and is therefore useful in those patients whose pre-operative ECG shows evidence of inferior or posterior ischaemia or infarction.

The ECG should always be recorded from before the start of the anaesthetic so that any subsequent changes can be observed. ST segment depression of 1mm or more below the isoelectric line with or without T wave changes indicates myocardial ischaemia. The magnitude of ST depression correlates with the severity but not the extent of the

Figure 22: Myocardial ischaemia

ischaemia. The ST segment depression moves progressively from up-sloping to horizontal to down-sloping as ischaemia worsens. Down-sloping ST segment depression may indicate transmural ischaemia (through the full wall thickness).

On a 12 lead ECG full thickness myocardial infarction results in ST segment elevation often with the subsequent development of pathological Q waves (greater than 1 mm thick and 2mm deep). In subendocardial infarction - typically there is deep symmetrical T wave inversion. In subepicardial infarction - there is loss of R wave amplitude without development of Q waves.

Management

● If ST segment depression develops during anaesthesia, 100% oxygen should be given, the volatile agent decreased and the blood pressure and heart rate normalised as far as possible. It is important to maintain diastolic blood pressure and systemic vascular resistance, in order to maintain coronary atery perfusion. In this situation methoxamine (if available) in 2mg iv increments titrated to effect may be useful.

● Postoperative management in a high care environment should be considered where possible, with oxygen therapy, adequate analgesia and correction of fluid and electrolyte balance being of great importance. Monitoring should be continued into the postoperative period as this is the time when further (often silent) ischaemia and infarction may occur. Oxygen should be given to all high risk patients post operatively, ideally for at least 48 hours.

OTHER ECG CHANGES SEEN IN THEATRE

Occasionally the ECG changes shape slightly with a change in position of the patient or during different phases of mechanical ventilation. This usually causes a slight change in the position of the heart and results in the ECG being recorded from a different angle. It is not usually of importance.

ECG APPEARANCE OF ABNORMAL POTASSIUM CONCENTRATIONS.

The ECG trace may develop characteristic changes with alterations in the concentration of various electrolytes. It is rarely possible to diagnose these from the ECG alone but the reading may give arise to a suspicion which should be confirmed by the laboratory.

Hyperkalaemia

● Tall peaked T waves

● Reduced P waves with widened QRS complexes

● Ultimately a sine wave pattern - pre-cardiac arrest

● Cardiac arrest in diastole

Hypokalaemia

● Increased myocardial excitability - any arrhythmia may occur

● Prolonged PR interval

● Prominent U waves

● Enhancement of digitalis toxicity

FURTHER READING AND REFERENCES

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MEASURING THE BLOOD PRESSURE

Dr Peter Hambly, Consultant Anaesthetist, Oxford, UK

Adequate blood pressure is essential to maintain the blood supply and function of vital organs. Measurement of blood pressure is therefore a key part of the monitoring of patients during anaesthesia and critical care.

What is normal blood pressure?

'Normal' or 'acceptable' blood pressure varies with age, state of health and clinical situation. At birth, a typical blood pressure is 80/50 mmHg. It rises steadily throughout childhood, so that in a young adult it might be 120/80 mmHg. As we get older, blood pressure continues to rise and a rule of thumb is that normal systolic pressure is age in years + 100. Blood pressure is lower in late pregnancy and during sleep.

From this, you can see that a systolic pressure of 160mmHg for an elderly man or 90 mmHg for a pregnant woman may be quite normal. To judge whether any particular reading is too high or too low, we must compare the reading with the 'normal' for that patient.

Techniques of measurement

Rough estimates without using any equipment at all

It is not possible to derive a numerical value for blood pressure without some equipment, but a crude assessment of the circulation can still be obtained. If you can feel a radial pulse the systolic blood pressure is usually at least 80 mmHg. The character of the pulse, i.e. bounding or thready, gives a further clue. In most cases, shocked patients have cold hands and feet. (The most important exception to this is a patient who is shocked because of severe sepsis). Capillary refill time is another simple test of circulatory adequacy: press firmly on the patient's nail bed with your thumb; release your thumb and see how long it takes for blood to return. A refill time of greater than 2 seconds suggests an inadequate circulation.

Manual non-invasive blood pressure measurement

This requires, at the very least, an inflatable cuff with a pressure gauge (sphygmomanometer). Wind the cuff round the arm (which should be at about heart level) and inflate it to a pressure higher than the expected blood pressure. Then deflate the cuff slowly. With a stethoscope, listen over the brachial artery. When the cuff reaches systolic pressure, a clear tapping sound is heard in time with the heart beat. As the cuff deflates further, the sounds become quieter, but become louder again before disappearing altogether. The point at which the sounds disappear is the diastolic pressure. If you have no stethoscope, the systolic blood pressure can be found by palpating the brachial artery and noting the pressure in the cuff at which it returns.

The sounds heard while measuring blood pressure in this way are called the Korotkoff sounds, and undergo 5 phases:.

I   initial 'tapping' sound (cuff pressure = systolic pressure)

II   sounds increase in intensity

III   sounds at maximum intensity

IV   sounds become muffled

V   sounds disappear

Most inaccuracies result from the use of the wrong size of cuff. A narrow cuff wrapped round a fat arm will give an abnormally high reading, and vice versa. The World Health Organisation recommends a 14cm cuff for use in adults. Smaller cuffs for infants and children are available. In occasional patients, the reading obtained from one arm can be different from that obtained from the other arm. An appropriate size of cuff can be applied to the calf, and pressure estimated by palpation of the posterior tibial pulse.

Oscillotonometry

The Von Recklinghausen Oscillotonometer is a device which allows both systolic and diastolic blood pressure to be read without a stethoscope. It consists of two overlapping cuffs (one large, one small) a large dial for reading pressure, a bleed valve and a control lever. The large cuff performs the usual function of the sphygmomanometer cuff. The job of the smaller cuff is basically to amplify the pulsations which occur as the larger cuff is deflated, so that instead of listening for the Korotkoff sounds, they are seen as oscillations of the needle on the pressure gauge. The lever simply switches the dial between the two cuffs.

Wrap the cuff round the arm in the usual way, and inflate it. Adjust the bleed valve so that the pressure falls slowly. Pull the control lever towards you. The needle will jump slightly in time with the pulse. As the cuff pressure approaches systolic, the needle suddenly starts to jump

more vigorously. At this point, let go of the lever, and the needle will display systolic pressure. Pull the lever forward again. As the pressure is reduced, the needle jumps more vigorously. If the lever is released at the point of maximum needle oscillations, the dial will read the mean arterial pressure. If it is released at the point when the needle jumps get suddenly smaller, the dial reads diastolic pressure.

Automatic non-invasive blood pressure measurement

Automatic devices which essentially apply the same principle as the oscillotonometer have been produced (e.g. the 'Dinamap' made by Critikon). They require a supply of electricity. A single cuff is applied to the patients arm, and the machine inflates it to a level assumed to be greater than systolic pressure. The cuff is deflated gradually. A sensor then measures the tiny oscillations in the pressure of the cuff caused by the pulse. Systolic is taken to be when the pulsations start, mean pressure is when they are maximal, and diastolic is when they disappear. They can produce fairly accurate readings and free the hands of the anaesthetist for other tasks. There are important sources of inaccuracy, however. Such devices tend to over-read at low blood pressure, and under-read very high blood pressure. The cuff should be an appropriate size. The patient should be still during measurement. The technique relies heavily on a constant pulse volume, so in a patient with an irregular heart beat (especially atrial fibrillation) readings can be inaccurate. Sometimes an automatic blood pressure measuring device inflates and deflates repeatedly "hunting" without displaying the blood pressure successfully. If the pulse is palpated as the cuff is being inflated and deflated the blood pressure may be estimated by palpation and reading the cuff pressure on the display.

Invasive arterial pressure measurement

This technique involves direct measurement of arterial pressure by placing a cannula in an artery (usually radial, femoral, dorsalis pedis or brachial). The cannula must be connected to a sterile, fluid-filled system, which is connected to an electronic monitor. The advantage of this system is that pressure is constantly monitored beat-by-beat, and a waveform (a graph of pressure against time) can be displayed. Patients with invasive arterial monitoring require very close supervision, as there is a danger of severe bleeding if the line becomes disconnected. It is generally reserved for critically ill patients where rapid variations in blood pressure are anticipated.

RESPIRATORY GAS ANALYSIS IN THEATRE

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CAPNOGRAPHY

Capnography is the measurement of carbon dioxide (CO_2) in each breath of the respiratory cycle. The capnograph displays a waveform of CO_2 (measured in kiloPascals or millimetres of mercury) and it displays the value of the CO_2 at the end of exhalation, which is known as the end-tidal CO_2.

It is useful to measure CO_2 to assess the adequacy of ventilation, to detect oesophageal intubation, to indicate disconnection of the breathing system or ventilator, and to diagnose circulatory problems and malignant hyperthermia.

Applications of Capnography

Provided the patient has a stable cardiac status, stable body temperature, absence of lung disease and a normal capnograph trace, end-tidal carbon dioxide ($ETCO_2$) approximates to the partial pressure of CO_2 in arterial blood ($PaCO_2$.) Normal $PaCO_2$ is 5.3kPa (40mmHg).

In these patients, $ETCO_2$ can be used to assess adequacy of ventilation i.e. hypo-, normo-, or hyperventilation. $ETCO_2$ is not as reliable in patients who have respiratory failure. The increased V/Q mismatch is associated with a widened P(a-ET) gradient, and can lead to erroneous $ETCO_2$ values.

The capnograph is the gold standard for detecting oesophageal intubation. No or very little CO_2 is detected if the oesophagus has been intubated.

The capnograph is also useful in the following circumstances:

- As a disconnection alarm for a ventilator or a breathing system. There is sudden absence of the capnograph trace.

- May detect air embolism as a sudden decrease in $ETCO_2$, assuming that the arterial blood pressure remains stable.

- To recognise sudden circulatory collapse as a sudden decrease in $ETCO_2$.

- To diagnose malignant hyperthermia as a gradual increase in $ETCO_2$.

Techniques of Measurement

Most analysers in theatre work using 2 principles:

Infrared absorption spectroscopy is the most commonly used technique in anaesthesia. Gases of molecules that contain at least two dissimilar atoms absorb infrared radiation. Using this property, carbon dioxide concentration can be measured continuously throughout the respiratory cycle to give a capnograph trace.

CO_2 absorbs infrared radiation particularly at a wavelength of $4.3\,\mu m$. A photodetector measures radiation reaching it from a light source at this wavelength. The amount of infrared radiation absorbed is proportional to the number of CO_2 molecules (partial pressure of CO_2) present in the chamber, according to the Beer-Lambert Law. This allows the calculation of CO_2 values.

Photo-acoustic spectroscopy The sample gas is irradiated with pulsatile infrared radiation, of a suitable wavelength. The periodic expansion and contraction produces a pressure fluctuation of audible frequency that can be detected by a microphone. The advantages of photo-acoustic spectrometry over conventional infrared absorption spectrometry are:

- The photo-acoustic technique is extremely stable and its calibration remains constant over much longer periods of time.

- The very fast rise and fall times give a much truer representation of any change in CO_2 concentration.

Other techniques for measuring CO_2 include Raman scattering and mass spectrometry.
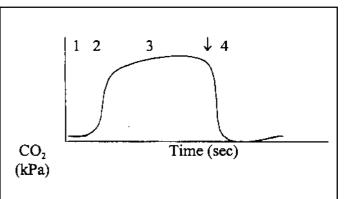
Position of Sampling

Gas from the breathing system can be sampled either by a sidestream or a mainstream analyser.

Sidestream: Gas is drawn from the breathing system by a 1.2mm internal diameter tube. The tube is connected to a lightweight adapter near the patient's end of the breathing system. It delivers the gas to the sample chamber. It is made of Teflon so it is impermeable to CO_2 and does not react with anaesthetic agents. Only the precise tubing recommended by the manufacturer should be used, and only of the recommended length. Typical infrared instruments sample at a flow rate between 50 and 150 ml/minute. When low fresh gas flows are used the sampled gas may be returned to the breathing circuit. It is important that the tip of the sampling tube should always be as near as possible to the patient's trachea, but the sampled gas mixture must not be contaminated by inspired gas during the expiratory phase.

Mainstream: The sample chamber is positioned within the patient's gas stream near the patient's end of the breathing system. Although heavier and more cumbersome, it does have advantages over sidestream sampling: there is no delay in the rise and fall times of gas composition changes, no gas is lost from the attachment, no mixing occurs along the capillary tube before analysis, and there are fewer problems with water vapour condensation.
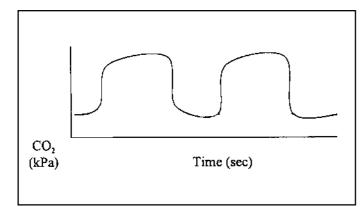
The capnograph trace

1: Inspiratory baseline

2: Expiratory upstroke

3: Expiratory plateau

4: Inspiratory downstroke

↓ End-tidal CO_2 ($ETCO_2$)

The first phase occurs during inspiration. The second phase is the onset of expiration, which results in a rapid increase in CO_2. The third phase, the expiratory plateau, occurs as the CO_2 is exhaled from all the alveoli. The highest point of the plateau is known as the end-tidal CO_2 ($ETCO_2$). This marks the end of expiration. Phase four is the onset of inspiration.
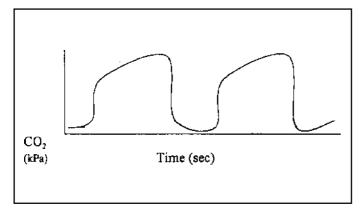
Abnormal Traces

1. Rebreathing

A waveform that does not return to the baseline during inspiration indicates that rebreathing of exhaled gas is occurring.

Causes:
- Fresh gas flow too low in non-rebreathing system.
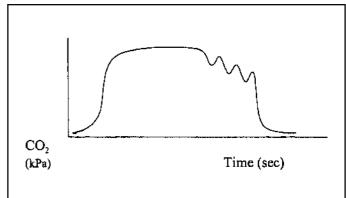- Soda lime depleted in circle system.

2. Sloping Plateau

Cause:

- Obstructive airways disease, because of impairment of V/Q ratio.

Explanation In patients with obstructive airways disease, the lungs are perfused with blood as normal, but the alveoli are unevenly ventilated. CO_2 that is transferred to the alveoli from the bloodstream may take longer to exhale because of the narrowed bronchi. This delayed emptying of the alveoli varies in different parts of the lungs. This results in the sloping plateau on the capnograph trace, as the CO_2 from those parts of the lungs with more severe bronchial narrowing is exhaled later than those parts with less severe narrowing.
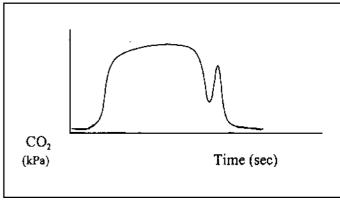
3. Cardiac oscillations

Cause:

- Cardiac impulses transmitted to capnograph.

Explanation The oscillations reflected on the capnograph trace result from transmission of cardiac impulses to the airways.

4. "Curare cleft"

Cause:

- Reversal of neuromuscular blockade in a ventilated patient.

Explanation When a paralysed patient starts taking small breaths as the neuromuscular blocking agent reverses, deep "clefts" are seen on the capnograph trace.

OXYGEN CONCENTRATION ANALYSERS

It is important to measure the oxygen concentration in the gas mixture delivered to the patient during anaesthesia. There are three techniques of measuring the inspired oxygen concentration (FiO_2): galvanic, polarographic, and paramagnetic techniques. The paramagnetic method is most widely used in anaesthesia. These analysers measure the oxygen partial pressure in a gas sample but they display

a percentage. Regular calibration of oxygen analysers is vital.

Paramagnetic oxygen analysers Oxygen possesses the property of paramagnetism, which means that it is attracted to a magnetic field. This is because it has two electrons in unpaired orbits. Most of the gases used in anaesthesia are repelled by a magnetic field (diamagnetism).

The sample gas is delivered to the analyser via a sampling tube, which should be placed as close as possible to the patient's trachea. The analyser has two chambers separated by a sensitive pressure transducer. The sample gas is delivered to one chamber. Room air is delivered to the reference chamber. An electromagnet is rapidly switched on and off creating a changing magnetic field to which the sample gas is subjected. The magnetic field causes the oxygen molecules to be attracted and agitated. This results in changes in pressure on either side of the pressure transducer. The pressure difference across the transducer is proportional to the oxygen partial pressure difference between the sample gas and the reference gas (room air).

Paramagnetic oxygen analysers are very accurate and highly sensitive. The analysers should function continuously without any service breaks. They have a rapid response allowing measurement of inspiratory and expiratory oxygen on a breath-to-breath basis. They are affected by water vapour and have a water trap incorporated into their design.

The Galvanic Oxygen Analyser (Fuel Cell) The galvanic analyser is placed on the inspiratory limb of the breathing system. Oxygen molecules diffuse across a membrane and an electrolyte solution to a silver cathode, which is connected through an electrolyte solution to a lead anode. An electrical current is generated which is proportional to the partial pressure of oxygen in the inspired gas.

The galvanic analyser has a response time of approximately 20 seconds. It is accurate to within 3%. Calibration is achieved using 100% oxygen and room air (21% O_2). Water vapour does not affect its performance. It is depleted by continuous exposure to oxygen due to exhaustion of the cell, so limiting its life span to about one year.

The Polarographic Oxygen Analyser (Clark Electrode) The polarographic analyser has similar principles to the galvanic analyser. Oxygen molecules diffuse across a Teflon membrane. Current flows between a silver cathode and a platinum anode, which is proportional to the partial pressure of oxygen in the inspiratory gas. A battery powers it. Its life expectancy is limited to about three years because of deterioration of the Teflon membrane.

Further reading

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MONITORING DURING CAESAREAN SECTION

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Recommendations for monitoring during Caesarean section (CS) have been developed by the American Board of Anesthesiologists and the Obstetric Anaesthetists Association (OAA) in the UK. The OAA's recommendations are reproduced in full in Box 1. Not all anaesthetists have access to complex equipment, but every anaesthetist should be aware of the potential problems that may be encountered and make appropriate use of the monitors they do have. The requirements for regional and general anaesthetics are different and so considered separately. **All obstetric patients undergoing CS should be positioned with left lateral tilt to avoid aorto-caval compression.** (See Update in Anaesthesia 1999).

Regional anaesthesia

Most of the monitoring is clinical since awake mothers are excellent monitors of their own physiology. The anaesthetist should be continuously present from the start of anaesthesia to the completion of surgery.

Assessment of analgesia

A major cause of maternal complaint is pain during CS under regional anaesthesia. For CS, a block should extend from S4 to the upper thoracic dermatomes. One common reason for inadequate pain relief is a failure of the block to spread to the sacral dermatomes. Although this happens more frequently with epidural than spinal anaesthesia,

whichever technique is used, always test the back of the legs (S2 and S3) to confirm that the sacral dermatomes are blocked before surgery starts.

How high a regional block must extend into the thoracic dermatomes to achieve intraoperative analgesia, remains controversial. Recommendations from T10 to T4 have been made, although the method of testing the block is often unspecified and the need for supplemental analgesics not mentioned. The three most commonly used methods of assessment are:

- loss of temperature sensation
- loss of pinprick sensation
- loss of light touch sensation.

These may differ by as much as 10 dermatomes, with temperature sensation lost first and light touch sensation last. Experimental data suggests that intraoperative analgesia is most reliably predicted by blocking light touch sensation (the hub of a needle lightly applied to the skin) to T5 (just beneath the nipples).

Haemodynamic consequences of regional anaesthesia

Extensive epidural and spinal blocks cause a temporary sympathectomy which makes the patient susceptible to hypotension. In pregnant women, this is made worse by the uterus compressing the aorta and inferior vena cava (aorto-caval occlusion). Hypotension may develop rapidly. Therefore, blood pressure should be measured at least every two minutes from starting a regional block until

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Box 1: Recommendations for monitoring during caesarean section

For operative delivery under regional block

Continuous pulse oximetry, non invasive blood pressure and continuous ECG during induction, maintenance and recovery.

The fetal heart rate should be recorded during initiation of regional block and until abdominal skin preparation in emergency caesarean section.

During general anaesthesia

Continuous inspired oxygen and end-tidal carbon dioxide concentration should be monitored, as well as pulse oximetry, non-invasive blood pressure and ECG.

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delivery. Nausea during onset of a regional block is usually an indication of hypotension.

Blocks above T4 cause a loss of sympathetic innervation to the heart which may be associated with bradycardia particularly if aorto-caval occlusion is present. Because of this continuous monitoring of the pulse is essential.

Respiratory consequences of regional anaesthesia

Pregnant women are prone to hypoxia because of a reduction in functional residual capacity (FRC) of the lungs and an increased oxygen consumption. This is compounded during regional blocks by abdominal and intercostal muscle weakness which causes a further reduction in FRC. Pulse oximetry not only monitors the pulse but also provides a continuous non-invasive monitor of the saturation of arterial haemoglobin. It is simple and accurate; always use it if you can.

When the thoracic dermatomes are blocked, patients often complain of a strange sensation when breathing, usually as they realise that they cannot produce a forceful cough. This is normal and a result of intercostal paralysis and the patient can be reassured. However difficulty in speaking represents diaphragmatic paralysis developing and needs very careful assessment of the level of block. Further spread of local anaesthetic must be minimised. If hyperbaric local anaesthetic has been used, this can be done by careful elevation of the head and neck. However be prepared to intubate and support these patient's ventilation.

Unexpected high blocks

"Total spinals" or very high blocks may follow excessive spread of a deliberate intrathecal injection of local anaesthetic or be caused by an epidural catheter that is misplaced in the subarachnoid space. Misplaced epidural catheters can be detected by attempting to aspirate CSF through the catheter and carefully assessing the effect produced by a test dose. An appropriate test dose will produce detectable changes in sensory and motor function within five minutes of injection if the catheter is in the subarachnoid space and no significant effect if the catheter is in the epidural space.

The spread of deliberate intrathecal injections of hyperbaric (heavy) local anaesthetics can be controlled by keeping the upper thoracic and cervical spine elevated. As spinal blocks sometimes extend very rapidly, you must check the spread of the block within 4 minutes of injection and reposition the patient if necessary.

Symptoms of high blocks are predictable. As the block extends the hands become warm and dry, then loss of hand and arm movement follows. Loss of abduction of the shoulder may be rapidly followed by diaphragmatic paralysis. At the same time sensation is lost over the upper chest, hands, arms, shoulder and neck. If the block extends further, consciousness may be lost and the pupils may become fixed and dilated. However all these signs will reverse provided cardiovascular and respiratory support are provided.

Regional blocks may continue to extend for at least 30 minutes after local anaesthetic has been injected, so the anaesthetist must remain vigilant for symptoms of high blocks even after surgery has started.

Monitoring the injection of local anaesthetic

Accidental intravenous injection of local anaesthetics may occur with epidural anaesthesia and although deaths are rare, convulsions occur in 1 in 500 - 9000 patients. This risk can be minimised by carefully aspirating before each injection, by assessing the effect of a small initial test dose of local anaesthetic and by splitting all large doses of local anaesthetics into several small portions. Every dose must be assessed for symptoms of intravenous injection (Table 1) even when previous doses have been uncomplicated.

Table 1: Symptoms of intravenous injection of local anaesthetic

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Tingling around the mouth

Tinnitus (ringing in the ears)

Visual disturbance

Confusion

Slurred speech

Altered conscious state

Convulsions

Coma

Cardiovascular collapse

Cardiac arrhythmias

General anaesthesia

Patients undergoing CS performed under general anaesthesia should be monitored in the same way as with any general anaesthetic. The obstetric anaesthetist should be particularly aware of airway problems and episodes of hyper- or hypotension.

Monitors of intubation

Failure of intubation and oxygenation remains one of the commonest causes of anaesthetic related maternal deaths. Confirmation of the correct placement of an endotracheal (ET) tube is crucial. Various monitors are available to help the anaesthetist, but seeing the ET tube pass through the glottis remains the most valuable. However the presence of bilateral breath sounds should always be checked and, when possible, the presence of expired CO_2 confirmed. Figure 1 shows ten simple clinical tests of correct placement of a tracheal tube.

The oesophageal detection device is a useful additional monitor. It is cheap and easily constructed using a 50 ml syringe or a self inflating bulb. If a negative pressure is applied by the syringe to a correctly positioned endotracheal (ET) tube, gas can be aspirated because the trachea is supported by rigid cartilage. However if the ET tube is misplaced in the oesophagus and a negative pressure applied, the oesophagus will obstruct the tip of the ET tube and gas cannot be aspirated. (see Update number 1997;7)

Monitors of ventilation

As with regional anaesthesia the pregnant mother is vulnerable to hypoxia; look at the patient's colour and at movement of the chest wall. If you are ventilating by hand feel for any changes in resistance to ventilation - if you are using a lung ventilator look regularly at and make a note of the inflation pressure. Always use a pulse oximeter if you have one.

Haemodynamic consequences of general anaesthesia

Aorto-caval occlusion means that mothers are vulnerable to hypotension, while hypertension may occur with laryngoscopy and surgical stimulus. Pre-eclamptic mothers are particularly vulnerable to hypertension on laryngoscopy. So, as with regional anaesthesia, blood pressure must be measured at least every two minutes until delivery and the pulse continuously.

Monitors for awareness

To reduce fetal depression and uterine relaxation, anaesthetists have sometimes used low doses of anaesthetic agents in a paralysed mother during CS. This has resulted in some mothers being awake and in severe pain. No single monitor reliably predicts awareness, although signs of sympathetic stimulation - sweating, tachycardia, hypertension and pupillary dilation - should always be regarded with concern.

The most reliable method of ensuring the mother is asleep, is to give adequate doses of induction agents and an initial overpressure of inhalational agents (ie twice MAC for 5 min, 1.5 times MAC for the next 5 min and 0.8 times MAC thereafter).

Neuromuscular blockade

With modern short acting muscle relaxants, reversal of neuromuscular block at the end of caesarean section is rarely a problem. The exception is if the mother has been treated with magnesium sulphate. Magnesium enhances the action of non-depolarising muscle relaxants. So in these patients, assessing neuromuscular function is important, ideally with a nerve stimulator but alternatively clinical methods may be used, such as assessment of hand grip or head lift.

Fetal monitoring

Various monitors of fetal condition are available. Fetal heart rate (FHR) monitoring is the most common. The FHR may be recorded intermittently with a stethoscope, by abdominal ultrasound, or with a fetal scalp electrode. A normal FHR has a 95% association with good fetal condition, and a prolonged and continuing bradycardia is almost always associated with severe fetal distress.

During CS, the FHR should be monitored from the start of anaesthesia until abdominal skin preparation especially if the fetus is already distressed. Knowing that the FHR is not critical, may allow time for a regional technique to be used, when otherwise a general anaesthetic might have to be performed. Knowledge of the FHR is also useful if a failed intubation occurs during general anaesthesia. The FHR can influence the decision to either wake the mother

Figure 1. 10 Clinical tests of tracheal intubation

Test	Result	Significance	Reliability
Look with laryngoscope	Tube passes between cords	Correct tracheal intubation	Certain
Listen/feel	Breathing through tube	Correct tracheal intubation	Probable
Tap sternum	Air comes out through tracheal tube	Correct tracheal intubation	Probable
Inflate with SIB*	Chest rises & falls	Correct tracheal intubation	Probable
Inflate with SIB*	Gurgling noise	Oesophageal intubation (REMOVE TUBE)	Probable
Pass catheter down inside tube	Patient coughs (if not paralysed)	Correct tracheal intubation	Probable
Look	Patient remains pink after intubation	Correct tracheal intubation	Probable
Look	Patient becomes cyanosed after intubation	Oesophageal intubation (REMOVE TUBE)	Certain
Stethoscope	Air entry at both apices both axillae & both bases	Correct tracheal intubation	Probable
Stethoscope	Air entry over stomach	Oesophageal intubation (REMOVE TUBE)	Probable

* = self inflating bag.

The capnograph or an oesophageal detection device (see above) are the most useful pieces of equipment to confirm intubation

and perform a regional technique, or continue surgery with a face mask.

Special problems.

While the monitoring requirements for uncomplicated Caesarean deliveries are straight forward, additional monitors may be required if other pathologies are present. As haemorrhage, embolism, hypertensive disorders of pregnancy and maternal cardiac conditions are associated with more than 50% of maternal deaths in the UK, these conditions deserve special mention.

Major haemorrhage

Major haemorrhage may be life threatening. Whenever major haemorrhage occurs, invasive cardiovascular monitoring should be used if available. This should include hourly urine output measurement, temperature monitoring and central venous pressure and invasive arterial pressure monitoring.

Embolism

The triad of hypocapnia, hypoxia and hypotension should alert the anaesthetist to the possibility of an embolism. Air embolism, thromboembolism and amniotic fluid embolism may all occur. Minor air embolism can be detected in almost every caesarean section. However it is extremely unusual for this to have any clinical significance. Thromboembolism causes approximately 25% of UK maternal deaths, but rarely presents during surgery. Perioperatively, amniotic fluid embolism is the greatest risk. If embolism is suspected then invasive cardiovascular monitoring should be considered and the clotting cascade assessed. Amniotic fluid embolism is often associated with a coagulopathy.

Hypertensive disorders of pregnancy

Severe pre-eclampsia is associated with a reduced plasma volume, while total body water is increased. Laryngeal oedema may make intubation difficult and hypertensive responses to intubation may be greatly increased. Treatment with magnesium may prolong the action of muscle relaxants. (see Update in Anaesthesia 1998;9) Renal failure may be present. Monitoring should be tailored to detect these problems and particular consideration given to invasive monitoring of central venous pressure, arterial blood pressure and hourly urine output.

Maternal cardiac conditions

Pregnancy stresses the cardiovascular system, particularly at delivery, when large fluid shifts and rapid changes in the pre- and after-load of the heart occur. These changes may be compounded by anaesthesia. Patients with cardiac disease, especially significant shunts or stenotic valvular lesions, are vulnerable to these changes. Some patients will require invasive cardiac monitoring throughout the perioperative period.

Conclusions

Caesarean sections are so common that the risks are often ignored. However in a recent survey, 82% of anaesthetic related deaths occurred during Caesarean section. The obstetric anaesthetist can reduce the risk to his patients by careful monitoring. The monitors should be tailored to detecting the problems that may be encountered so that they can be corrected before mother or fetus are harmed.

MONITORING IN THE RECOVERY ROOM

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The recovery room is the area where patients recover from the immediate effects of anaesthesia and surgery and provides a setting for the detection and treatment of early post-operative complications. These areas vary considerably in the level of staffing and monitoring available from the "ideal" fully staffed, fully equipped modern recovery facility to the somewhat less than perfect dimly lit corridor just outside theatre.

The commonest causes of recovery room mishaps all result from unidentified changes in a patient's airway, breathing or circulation and these can almost always be rectified if identified at an early stage.

In 1993 the Association of Anaesthetists of Great Britain and Ireland published recommendations for the provision of equipment necessary for a modern day recovery area. These are shown in table 1.

These facilities may not all be available in many recovery areas throughout the world and some thought is necessary to determine the relative merits of each item on this long list.

Certainly the two most important pre-requisites of any recovery area should be the provision of good lighting together with a suitably trained recovery nurse available to recover each unconscious patient on a one-to-one basis. The nurse must be available to stay with the patient constantly until awake and able to maintain their own airway.

Clinical patient monitoring

Clinical monitoring can be divided into assessment of airway, breathing and circulation.
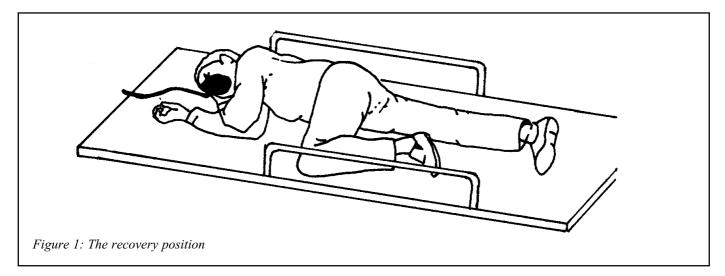

Table 1: **Association of Anaesthetists of Great Britain and Ireland Guidelines 1993**

Position of recovery	situated as close as possible to the operating theatre to minimise the risks of transporting unstable patients
Size and temperature	an average of 1.5 recovery bays per operating theatre (9.3m^2 per bay)
	room temperature 21-22 °C, relative humidity 38 - 45% and fifteen changes of air per minute
	gas scavenging system and six 13 ampere electricity outlets per bay
	well lit with lighting approximating to the daylight spectrum
Equipment in each bay	oxygen outlets, face masks and breathing systems
	pulse oximetry
	availability of blood pressure monitoring and ECG
	suction units with Yankaur ends
	a fully equipped anaesthetic machine with ventilator
	drugs and intravenous fluids
	a paediatric equipment trolley containing facemasks, airways, endotracheal tubes and connectors in a range of paediatric sizes

The patient's airway can be assessed by observing for signs of obstruction such as chest wall or supraclavicular inward movement on inspiration and/or the presence of noisy breathing. A patent airway is most easily maintained with the patient in the left lateral ('recovery') position (figure 1) as this allows the tongue and soft palate to fall forwards away from the oropharyngeal opening. This will also prevent any blood or secretions pooling in the oropharynx and causing aspiration or laryngospasm. In patients at risk of further oropharyngeal soiling, drainage of secretions can be increased by placing a pillow beneath the patient's chest ('tonsillar position'). If the patient has to remain supine for any reason then the use of jaw thrust or an oropharyngeal (Guedel) airway can be invaluable in airway maintenance. **Maintenance of a patent airway is probably the single most important aspect of immediate post-operative care.** Although few patients in the UK are now taken to recovery with an endotracheal tube in-situ this depends to some extent on the anaesthetic agents available. Where slowly eliminated agents are used (ether and halothane) the endotracheal tube is sometimes best left in-situ until full return of the laryngeal reflexes. When to extubate is a matter of clinical judgment, but as a rule it is safer to leave the tube as long as possible as this ensures airway patency. In children it is common practice to wait until they can remove it themselves. Suctioning of the oropharynx should be performed before tube removal to avoid aspiration of any blood or mucus present. The anaesthetist should always be on hand until a patient is awake and maintaining their own airway in case of severe laryngospasm necessitating re-intubation.

Respiration can be assessed by monitoring abdominal excursion, chest movement or by feeling for expiration with a cupped hand at the patient's mouth or nose. Oxygenation can also be assessed to some degree by examining the patient's colour. A dusky, bluish hue suggests hypoxia, and is often most easily noted around the lips or tongue. This does, however, require natural daylight or good quality artificial lighting as some systems produce monochromatic light which makes the appreciation of colour difficult. Bradypnoea will usually be due to intra-operative opioid use and if so will be associated with pinpoint pupils. This may resolve spontaneously as the other anaesthetic agents are eliminated and the patient wakes up. If treatment is indicated (respiratory rate less than 8 bpm or hypoxia) then first try to rouse the patient and if this fails consider naloxone (400 microgram diluted into 10 ml saline administering 2 ml boluses intravenously). Where available doxapram (1 mg/kg) is a useful respiratory stimulant and will not reverse the analgesic effects of opioids. Tachypnoea can be associated with certain volatile agents (particularly ether), acidosis, hypovolaemia, pain, hypoxia or other respiratory problem.

Circulation can be assessed by palpating the pulse (thready pulse or tachycardia suggesting volume depletion) and by feeling the peripheries (cold poorly perfused hands also suggest hypovolaemia or hypothermia following long operations). Heart rate should normally be between 60-90 bpm. Bradycardia is usually associated with deep anaesthesia or vagally stimulating surgery and may need treating if the heart rate is less than 40-50bpm or if associated with hypotension (give atropine 200-400mcg). Tachycardia is likely to be due to poor pain control or hypovolaemia, but may rarely be due to atrial fibrillation or a supraventricular tachycardia. Primary treatment should be directed at the cause (morphine or a 250ml fluid challenge). As with respiratory monitoring it is useful to chart the heart rate and blood pressure so that trends over time can be more easily seen. A developing tachycardia is often an early sign of unrecognised blood

Figure 1: The recovery position

loss. The wound site must also be observed every few minutes to ensure that any bleeding or haematoma formation is noted early. Drainage from surgical drains should also be charted.

Conscious level should be monitored by observing the return of reflexes such as the eyelash reflex, swallowing and the start of vocalisation and response to commands. Where the patient has undergone regional anaesthesia (spinal or epidural) the height of the block must be assessed until it is seen to be regressing. This is most easily tested by measuring the point at which cold can no longer be appreciated (using ethyl chloride or ice). It is safer not to sit these patients up too early as marked postural hypotension can occur.

Once the patient is vocalising and is reasonably awake pain levels should be assessed. Recovery nurses should be capable of administering intravenous analgesia and achieving adequate analgesia should be a primary goal once airway reflexes have returned. Pain is most easily treated by administering morphine 1-2 mg aliquots every 3-5 minutes until comfortable. It is very unusual to overdose patients using this regime, but intravenous naloxone should be available. See also Update in Anaesthesia 1997; 7.

Supplemental oxygen therapy

Whenever possible all patients recovering from anaesthesia should be given supplemental oxygen (4l/min by face mask). Where facilities are limited, provided the airway and breathing is monitored closely, young, fit individuals having relatively minor procedures can often recover without supplemental oxygen. However it should be given whenever possible in the less fit population having major surgery. Anaesthesia, particularly halothane, obtunds, or in some cases abolishes, the hypoxic respiratory drive so that hypoxia no longer stimulates increased ventilation. Coupled with this is a much increased tendency for hypoxaemia to occur due to a variety of reasons including airway obstruction due to an obtunded conscious level, hypoventilation secondary to opioids and anaesthetic agents, diffusional hypoxia caused by nitrous oxide diffusing into the lungs faster than nitrogen can diffuse in the opposite direction and many different causes of ventilation/perfusion mismatching. These include atelectasis (absorption collapse, mucus trapping, prolonged hypoventilation), a decrease in functional residual capacity with anaesthesia and supine posture, poor mucus clearance (absent/impaired cough reflex and poor ciliary function), and possibly hypovolaemia or pulmonary oedema. Patients particularly at risk of postoperative hypoxia include those

who have received nitrous oxide or opioids, and those with pre-existing pulmonary disease. Where oxygen can be administered, even 2 litres per minute via nasal spectacles or face mask may be sufficient to prevent desaturation associated with most of the causes listed above. It is also important to realise that the tendency for hypoxaemia extends long into the postoperative period and is particularly likely to occur on the first postoperative night. If possible, high risk patients undergoing major surgery should receive supplemental oxygen for 48-72 hours.

Monitoring Equipment

The most useful monitors in the recovery area are the *pulse oximeter* and the *sphygmomanometer*. The latter is obviously considerably cheaper, more widely available and doesn't need electricity to function. It provides valuable information about a patient's cardiovascular status. Postoperatively patients are often mildly hypotensive due to the sedative effects of drugs and the likelihood of blood loss or intraoperative fluid redistribution (coupled with some degree of dehydration due to preoperative fasting). More marked levels of hypotension (or even more seriously a downward trend in blood pressure) often herald an unrecognised blood loss, an adverse cardiac event or may follow spinal anaesthesia. For these reasons it is prudent to monitor blood pressure every five minutes or so until it is stable and within normal limits. This also demonstrates why it is important to document the vital signs over a period of time, so that these trends can more easily be spotted and acted upon.

Since its widespread introduction in the 1980's the pulse oximeter has become one of the mainstays of post anaesthetic monitoring. Where available these machines will give a fairly reliable indicator of systemic oxygenation, together with some indication of cardiovascular status. Oxygen saturation levels should remain above 93% and desaturation below this level in recovery is most commonly caused by airway obstruction and poor or inadequate ventilation. The presence of a good quality pulsatile signal usually denotes adequate peripheral circulation, although vasodilatation and hypotension can still be present, so the blood pressure should still be monitored. See p11 for further reading regarding the use of pulse oximeters.

Apart from the above minimal monitoring, further assessment must be tailored to a patient's particular needs. Urine output should be assessed where an indwelling urinary catheter is sited and ECG monitoring may be necessary where a patient is at risk of arrhythmias.

Table 2 Routine postoperative observations on patients undergoing major surgery

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- oxygen administration
- oxygen saturation
- respiratory frequency
- blood pressure and heart rate
- conscious level
- pain score
- operation site review
- temperature and urine output where necessary
- any drugs or fluids administered

Temperature should also be checked following long operations (particularly in the elderly) where hypothermia is a risk. Lack of attention to temperature maintenance in theatre can lead to major problems in recovery. Patients tend to lose heat rapidly under anaesthesia due to obtunding of homeothermic mechanisms and prolonged surgical exposure. Hypothermia (even a small reduction to 35 degrees centigrade) can have a major impact on postoperative recovery. As temperature decreases drug metabolism and excretion also decreases thereby prolonging recovery. Poor metabolism of neuromuscular blocking agents can be a particular problem. Blood clotting is also affected early, with a much increased tendency to postoperative bleeding. Shivering also causes increased oxygen utilization, increasing the tendency to hypoxaemia, and is best treated with body surface warming or intravenous pethidine (10-20 mg).

Where possible the observations in table 2 should be routinely charted on all patients who have undergone major surgery:

Discharge Criteria

The patient should continue to be observed until fit for discharge to the ward area. Discharge criteria vary, but should include the return of preoperative conscious level and protective reflexes, maintenance of a clear airway, satisfactory breathing and oxygenation (oxygen saturation > 93% on air), stable pulse and blood pressure, acceptable temperature and adequate analgesia (table 3).

Table 3 Discharge criteria

- patient conscious and maintaining a clear airway
- return of protective airway reflexes
- satisfactory breathing and oxygenation (oxygen saturation > 93% on air)
- stable pulse and blood pressure
- good peripheral perfusion
- acceptable temperature
- adequate analgesia

Reference

Immediate Postanaesthetic Recovery, 1993. The Association of Anaesthetists of Great Britain and Ireland, 9 Bedford Square, London WC1B 3RA.

ANAESTHESIA FOR EMERGENCY EYE SURGERY

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Introduction

Anaesthesia for emergency eye surgery can present special problems to the anaesthetist. An understanding of some basic principles and techniques of eye anaesthesia have been discussed in previous issues of Update (Nos. 6 & 8).

This article discusses the specific problems of emergency anaesthesia for eye surgery. We try and answer the common questions concerning these patients and provide a practical guide.

Indications for emergency eye surgery

An emergency is defined as an event that has to be dealt with immediately, usually within the first hour after presentation. The commonest eye emergencies that fall into this category are chemical burns of the eye and retinal artery occlusion. Neither of these requires surgery as part of the initial management. The majority of cases presenting as emergencies can therefore be defined as urgent cases.

Trauma is by far the commonest indication for urgent surgery. Traumatic injuries can be blunt or penetrating ("open eye"). The incidence is highest in young adult males and children. Trauma is often associated with industrial or motor vehicle accidents. Eye protection in the work place and car safety belts have lowered the incidence of eye trauma in many countries. Eye trauma is usually confined to one eye. Some patients may present with trauma to both eyes or with multiple injuries.

Non-traumatic surgical "emergencies" include spontaneous retinal detachment, infections, and complications of previous surgery. One of the factors which determines the degree of urgency for retinal detachment surgery is the condition of the macula. The risk of a detachment progressing and resulting in loss of the macula increases the sense of urgency. There is usually enough time however to allow for fasting prior to surgery.

Timing of surgery

Ideally all patients should be fasted before undergoing general anaesthesia to minimise the risk of aspiration and subsequent lung injury. This obviously has to be weighed against the risk to the eye that delaying surgery may cause. It is essential to liase closely with the surgeon to establish the degree of urgency. Most cases involving blunt trauma can usually be delayed to allow for patient fasting.

Penetrating injuries may need to be dealt with more urgently due to the risk of infection and endophthalmitis. If the patient has an open eye injury there is also the risk of vitreous loss and retinal detachment. Even with open eye injuries many ophthalmic surgeons are willing to delay surgery until a patient is adequately fasted prior to anaesthesia. This is especially the case where there is severe damage to the eye and surgery is not going to improve sight. This group of patients are usually admitted for bed rest and have an eye shield covering the injured eye until they are ready for primary closure of their eye wounds. Open eye injuries in which the eye is still largely intact and the visual prognosis is good need to be dealt with more urgently. Decision making needs to be made on a case by case basis. The degree of urgency will depend on the size of the laceration and commensurate risk of loss of ocular contents, how dirty the wound is and the risk of infection.

A fast of six hours is normally suggested in the uncomplicated patient. It is now common practice to allow patients to drink clear fluids (water, non-fizzy fruit drinks) up to two to four hours prior to the time of surgery. In patient's who have had trauma or received opioids, it can take up to 24 hours for gastric emptying to take place. The most important time interval is that between the last meal and the time of the injury. If trauma occurs soon after a large meal the patient may still have a full stomach after the standard six hour fast. Alcohol also delays gastric emptying. If surgery is necessary in a patient with a full stomach then a rapid sequence induction technique should be used (see below).

How long patients should be fasted for prior to surgery with a local anaesthetic block is controversial. We feel that in the patient undergoing emergency eye anaesthesia the above principles regarding fasting should be used irrespective of the anaesthetic technique chosen.

Does the patient have other medical problems ?

Eye trauma requiring surgery may be associated with other injuries that may or may not require surgery. In the multiply injured patient normal trauma principles must always be applied. Life-threatening problems should be dealt with before sight-threatening problems. The principles of managing the patient with major trauma have been discussed in Update 1996;6. Patients with other disease

processes such as diabetes or ischaemic heart disease should have these optimised prior to surgery if time allows.

Choice of a local or general anaesthetic technique

The choice of technique will depend on patient factors as well as local facilities and surgeon preferences. In many countries extra-ocular, anterior segment and vitreo-retinal eye surgery is routinely performed using local anaesthetic techniques. However there are many practical reasons why a general anaesthetic is often preferable for emergency cases. Firstly, the patient must be able to lie flat, still and protect his or her own airway safely for the duration of the procedure. Thus, children, uncooperative or intoxicated patients are usually better candidates for a general anaesthetic. An uncooperative patient with an open eye is extremely difficult to manage. Spread of local anaesthetic agents is poor in patients with eye and orbital infections. Some procedures such as scleral banding (scleral buckling) for retinal detachment can be extremely uncomfortable even with a good local anaesthetic block. In our experience younger adults tend to tolerate surgery with a local anaesthetic technique poorly compared with elderly patients.
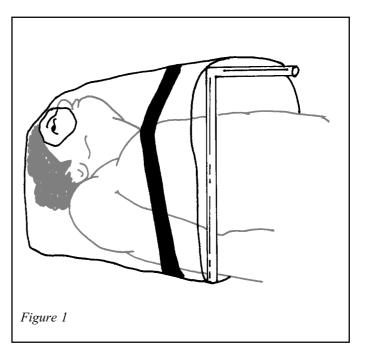
In open eye injuries local anaesthetic techniques are usually avoided. Injection of local anaesthetic using peribulbar and retrobulbar techniques is associated with an increase in intra-ocular pressure which may lead to vitreous loss. Oculocompression after the block is also not an option if the patient has an open eye injury. In some patients it may be possible to operate on small open eye injuries using topical anaesthesia, sub-tenon blocks or a careful peribulbar or retrobulbar block.

Is sedation an option ?

Sedation should be used cautiously. Oversedation can easily turn a cooperative patient into a difficult to manage patient due to airway problems and patient confusion. Sedation should not be used as an alternative to a general anaesthetic in a patient with a full stomach. If a patient develops pain during surgery using a local anaesthetic technique the patient requires analgesia and not sedation. The surgeon should supplement the block using local anaesthesia or small doses of intravenous analgesia should be given.

If sedation is to be used then small doses of a short acting agent such as midazolam should be given. Diazepam in small doses may also be an option. Propofol in small 10mg increment doses can also be used especially prior to performing a local anaesthetic eye block. Some anaesthetists use small doses of alfentanil or fentanyl. The

key to good sedation is to maintain verbal contact with the patient.

Careful surgical draping is also important. Patients become claustrophobic if their faces are draped. Use of a bar to hold up the drapes can allow a tent to be made to allow better ventilation (figure 1). Oxygen should be given to the patient, especially if sedation is to be used. Patients may find a face mask or nasal oxygen cannulae uncomfortable. Oxygen can be insufflated under the drapes using a breathing circuit. This also improves air circulation under the drapes.

Figure 1

Many of the problems associated with local techniques can be avoided with a clear explanation of the procedure to the patient prior to commencing surgery, having a comfortable operating table, and somebody to hold the patient's hand throughout. Allowing patients to empty their bladders prior to surgery also helps.

Choice of drugs for general anaesthesia

The choice of intravenous induction agent will depend on local availability and user familiarity. Most intravenous induction agents reduce intra-ocular pressure therefore preventing further damage to the injured eye.

Ketamine possibly raises intra-ocular pressure although the literature is conflicting. Most textbooks state that it should be avoided in open eye injuries. If it is to be used it is best to use it in combination with small doses of a benzodiazepine (midazolam, diazepam) to blunt its excitatory effects. The majority of problems with ketamine and intra-ocular pressure seem to occur when it used as a

sole agent in a patient with an unprotected airway breathing spontaneously. Ideally ketamine should be used with a muscle relaxant and controlled ventilation if intra-ocular pressure control is important.

All the non-depolarising muscle relaxants can be used without adverse effects on the eye so choice will depend on availability. Suxamethonium (scoline) increases intra-ocular pressure. The exact mechanism is unclear but it is not thought to be solely due to contraction of the extra-ocular muscles. Suxamethonium also causes an increase in the intra-ocular blood volume and this may contribute to the rise in intra-ocular pressure. The rise in intra-ocular pressure occurs after one to two minutes and wanes after six to ten minutes. The extent of the rise in intra-ocular pressure will depend on the other drugs used and the response to laryngoscopy and intubation. Its use in penetrating eye injury anaesthesia is controversial. The majority of eye surgeons prefer if it is not used. Adequate fasting prior to surgery will allow suxamethonium to be avoided for the majority of urgent cases. This obviously presents a dilemma in the patient with a full stomach as suxamethonium is used as part of a 'rapid sequence induction' to enable an airway to be secured quickly. In this situation the relative risks need to be weighed, i.e.

prevention of aspiration (potentially life threatening) verses ocular damage (potentially sight threatening). Suxamethonium avoiding techniques include the use of large doses of vecuronium or pancuronium to speed up its onset of action as part of a modifed rapid sequence induction technique. The non-depolarising neuromuscular blocker rocuronium has a rapid onset of action with a duration of 30 to 40 minutes. It can be used for a rapid sequence induction technique but can only be recommended to those who have gained experience in its use and for patients in whom airway problems are unlikely to occur.

On balance there are no case reports of ocular damage with suxamethonium use, and no good evidence that suxamethonium-avoiding techniques are any better or safer.

Airway management and mode of ventilation

It is considered good practice to intubate and ventilate the patient to ensure a secure airway (the surgical field is in close proximity) and to facilitate mild hypocarbia (this reduces intra-ocular pressure). The laryngeal mask airway is a popular choice for airway management for elective eye surgery in the UK. Laryngeal mask insertion avoids the pressor response to laryngoscopy and intubation causing raised intra-ocular pressure. The laryngeal mask

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Analgesic Drugs

Drug	Dose	Comments
Paracetamol (Acetominophen)	Children: 90 mg/kg total per 24 hours orally or rectally in 4-6 divided doses	Avoid if liver dysfunction. Decrease dose to total of 60 mg/kg per 24 hours if treatment for more than 48 hours.
	Adults: 1g orally or rectally. 4g total per 24 hours	
Ibuprofen	Children: 10mg/kg orally. 4 doses maximum in 24 hours.	Ibuprofen has the lowest side effect profile of the non-steroidal anti-inflammatory drugs. Avoid in renal and peptic ulcer disease. Use with care in asthma. Not in children <7kg.
	Adults: 400 mg orally. 4 doses maximum in 24 hours.	
Diclofenac	Children: 1mg/kg orally or rectally. 3 doses in 24 hours.	Cautions as for Ibuprofen.
	Adults: 150 mg total by any route in 24 hours	
Codeine Phosphate	0.5 mg/kg orally 6 hourly	Use with care when co-administered with other opioids

Drugs for nausea and vomiting

Drug	Dose	Comments
Droperidol	0.5 to 1 mg in adults. Up to 3 times a day	Cheap and effective but causes drowsiness, sedation, anxiety and restlessness. Risk of extrapyramidal effects.
Cyclizine	Children 1mg/kg iv	Up to 3 times a day
	Adults 50 mg iv	Anti histamine and anti-cholinergic effect.
Ondansetron	Children 0.1 mg/kg iv	3-4 doses per 24 hours
	Adults 4 mg iv	Expensive but effective with low side effect profile.

does not protect against aspiration of gastric contents. Its use in emergency anaesthesia is therefore limited.

Analgesia and control of nausea and vomiting

It is possible to manage pain in the majority of patients after eye surgery with oral analgesia. Avoiding opioids if possible helps prevent nausea and vomiting. Regular doses of paracetamol (acetominophen) and a non steroidal anti-inflammatory drug (ibuprofen, diclofenac, ketoprofen) should be prescribed. Codeine phosphate can also be added. These drugs are best accepted by children if given as an elixir (syrup). Some analgesic drugs are listed in the table below. In patients having surgery with general anaesthesia it is a good idea to ask the surgeon to perform a local anaesthetic block before waking up the patient. If stronger analgesia is required this is best given as small intravenous doses of morphine or pethidine.

Nausea and vomiting after emergency eye anaesthesia can be a major problem in some patients. Anti-emetic prophylaxis may help prevent this. Some patients may benefit from a regular anti-emetic in the post-operative period. There is a vast number of anti-emetic drugs available. Most have a limited efficacy. Using a combination of small doses of anti-emetic drugs from different pharmacological classes may enhance efficacy and reduce side effects. Some anti-emetic drugs are listed in the table below.

A practical approach to emergency eye anaesthesia

1) Assess the indication for emergency anaesthesia in discussion with the surgeon. Can surgery be deferred until normal working hours and to allow adequate fasting?

2) Carry out a full preoperative assessment including a history and examination.

3) Are there any medical/trauma issues that need addressing first?

    Decide on choice of anaesthetic technique. Provide the patient with a full explanation. Tell the patient what to expect if a local anaesthetic technique is to be used.

4) If a general anaesthesia is chosen decide if the patient has a full stomach and is at risk of aspiration.

5) If the patient has a full stomach a rapid sequence induction technique should be used. They should be preoxygenated with 100% oxygen. Pressure on the affected eye from the mask must be avoided. The patient should then be induced with an intravenous anaesthetic agent (eg thiopentone 4-7mg/kg) and a rapid onset muscle relaxant (suxamethonium 1-1.5mg/kg is currently the only realistic option). While the patient is being induced cricoid pressure should be applied by an assistant (Sellick's manouvre) thus occluding the oesophagus behind. The patient's trachea should be intubated after which the cricoid pressure can be removed. Note that the endotracheal tube tie should not be tight around the neck as this impedes venous drainage and raises intra-ocular pressure.

6) Choice of maintenance depends on local availability e.g. 40% O_2, 60% N_2O and an inhalational agent. Note that all inhalational agents reduce intra-ocular pressure.

7)   Control ventilation during the procedure aiming for low to normal end-tidal carbon dioxide. This may require the use of a longer acting muscle relaxant (e.g. vecuronium 0.1mg/kg). A slight head up tilt helps reduce intra-ocular pressure.

8)   At the end of the procedure the patient should be extubated on their side and once airway protective   reflexes have returned. In patients not deemed at risk of aspiration extubation with the patient deep and breathing spontaneously may prevent coughing. Severe coughing and straining needs to be avoided as this increases the risk of ocular haemorrhage.

9)   If the patient does not have a full stomach and is not deemed at risk of aspiration, general anaesthesia should proceed as for an elective patient. Pre-oxygenate the patient for safety and induce with an intravenous agent. Give a long acting muscle once ability to hand ventilate is established. Laryngoscopy should be performed gently. Consider spraying the vocal cords with lignocaine to minimise the pressor response to intubation. This may also decrease the risk of coughing on intubation. Intubate, ventilate and maintain anaesthesia as above.

10)   Post operatively nausea, vomiting and pain should be kept to a minimum as they can cause rises in intra-ocular pressure. Prescribe regular oral analgesia and an anti-emetic. Some patients may need stronger analgesia early after surgery. Titrate small doses of intravenous opioid (morphine, pethidine) to control pain.

References

1.   Mcgoldrick KE. The open globe: is an alternative to succinylcholine necessary ? J Clin Anaesth 1993, 5: 1-4.

This article argues that suxamethonium is probably still the best muscle relaxant for the real emergency. It also discusses the use of pretreatment.

2.   Libonati MM, Leahy JJ, Ellison N: The use of succinylcholine in open eye surgery. Anaesthesiology 1985, 62: 637-40.

This paper studied 100 patients needing "open eye" surgery with suxamethanium.

MANAGEMENT OF A HEAD INJURY - Case Report

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This is a report of a patient who has suffered a head injury. The purpose is to illustrate the practical application of the basic physiological and pharmacological principles explained before (Neurophysiology-intracranial pressure and cerebral blood flow, Update in Anaesthesia 1998;8:18-23 and Neuropharmacology-Intracranial pressure and cerebral blood flow. Update in Anaesthesia 1998;9:29-37). The problem is presented with the management and a range of anaesthetic techniques.

The Case

Cycling to work in the morning, a fit 30 year old man has an accident which causes severe damage to his head. Initially he is conscious but confused, and is taken to the local Accident Department. When he is admitted it is found that he has become unconscious.

Initial Management

An initial assessment is performed urgently, in the sequence described below.

A   Airway control including cervical spine immobilisation with a stiff collar.

B   Breathing

C   Circulation

D   Dysfunction or Disability

E   External Examination

His **airway** is clear. He is **breathing** adequately. His blood pressure is 180/90mmHg and he has a regular pulse with a rate of 55 bpm. There was no report of blood loss at the scene. He is warm and well perfused. Thus his **circulation** is adequate.

Neurological dysfunction is assessed by looking at **conscious level**, **pupils** and **posture**. Conscious state is assessed using the **G**lasgow **C**oma **S**core (**GCS** score Table 1) or the **AVPU** system. Glasgow Coma score range is 3-15, if it is less than 8, the patient has serious damage with raised intracranial pressure (ICP) more than 20 mmHg (normal 5-13 mmHg).

The **AVPU** is simple to carry out and offers a rapid method of assessment.

Alert	Yes/No
Verbal - response to verbal command	Yes/No
Pain - response to painful stimulus	Yes/No
Unresponsive	Yes/No

Patients who are not alert and are not responding to command (P or worse) are equivalent to a GCS of around 8 which indicates a severe injury.

On examination of the pupils the right pupil is found to be fixed and dilated, the left pupil is small and reacting. He is unresponsive to pain (GCS less 8, AVPU less than P). This is a neurological emergency where delay may result in a fatal outcome or major disability. A rapid secondary survey is carried out to exclude other life threatening injuries.

Although the risk of neck injury is low, it cannot be excluded and therefore the neck is kept stabilised with a semi-rigid collar and sand bags or blocks joined with tape with straps across the forehead (figure 1). An IV infusion is started with normal saline (0.9%).

Table 1: The Glasgow Coma Scale

Glasgow Coma Scale for Assessment of Level of Consciousness

Eye Opening:

Spontaneous	4
To speech (not necessarily a request for eye opening)	3
To pain (stimulus should not be applied to face)	2
None	1

Best Motor Response:

Obeys commands	6
Localise (purposeful movement towards the stimulus)	5
Normal flexion (withdraws from painful stimulus)	4
Abnormal flexion (decorticate posture)	3
Extension (decerebrate posture)	2
No movement	1

Verbal Response:

Oriented (knows name, age)	5
Confused (still answers questions)	4
Inappropriate words (recognisable words produced)	3
Incomprehensible sounds (grunts/groans, no actual words)	2
None	1
TOTAL SCORE	**/15**

COMMENT

As soon as the patient is admitted to hospital the basic ABCDE sequence described above is rapidly carried out to detect any problems such as airway obstruction or respiratory arrest which will rapidly cause death unless treated. With head injuries, respiratory obstruction will cause hypoxia and raised carbon dioxide and will lead to increased intracranial pressure causing severe secondary damage. An adequate blood pressure is vital in a patient with raised intracranial pressure.

*The patient has suffered a primary head injury from trauma. The signs indicate a rapidly rising intracranial pressure, with coning of the temporal lobe probably due, in this case to an extradural haematoma. This is a rapidly fatal condition if it is not treated urgently. When treated quickly, a good recovery may result. It is essential that further brain damage from ischaemia (due to low blood pressure and cerebral swelling) resulting from factors such as hypoxia, high carbon dioxide levels and venous congestion does not occur. **Hypotension and hypoxia will increase mortality of a patient with a severe head injury by 70-80%.***

Anaesthetic Management

Whenever possible the patient should be reviewed by an anaesthetist in the receiving room or accident department. As the patient has low AVPU and GCS scores, he needs to be intubated and ventilated to ensure a clear airway, full
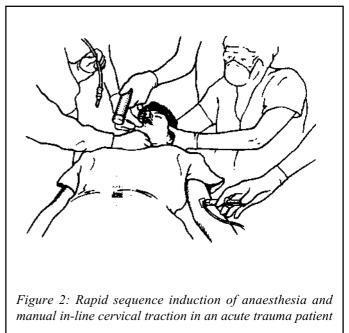

Figure 1: Cervical spine immobilisation with a long spine board, rigid collar, lateral blocks and straps.

oxygenation and low normal carbon dioxide levels before going to the CT scanner. Since there is still concern regarding a possible neck injury, further movement of the neck during intubation could cause injury to the cervical cord. Therefore intubation is carried out with the head in the neutral position and **manual in-line** traction to prevent neck movement (figure 2). To perform this manoeuvre, an assistant grasps the mastoid processes and the front part of the collar is removed to allow adequate mouth opening. Do not apply excessive traction as this can cause further damage to the cervical spine.

The mouth is clear, but as he may have eaten breakfast recently, a rapid sequence induction is necessary. He is pre-oxygenated and given an intravenous narcotic fentanyl 150 mcg, (pethidine 50 mg or morphine 5 mg would be reasonable alternatives) followed by a slightly reduced dose of thiopentone 150-200 mg to ensure anaesthesia,

Figure 2: Rapid sequence induction of anaesthesia and manual in-line cervical traction in an acute trauma patient

without causing hypotension. Many places today are now using a reduced dose of propofol 90 - 100 mg, for induction and continuing anaesthesia with a propofol infusion. Cricoid pressure is applied, he is paralysed with 100mg of suxamethonium, and intubated once fasciculation has stopped.

It is important to maintain intermittent positive pressure ventilation for neurosurgical patients to produce a moderately low normal arterial CO_2 ($PaCO_2$ 35 mmHg - 4.7 kPa) which will help to reduce cerebral swelling and hence intracranial pressure.

After tying in the endotracheal tube the blood pressure is measured again. It has fallen to 80/55 mmHg. 500mls of normal saline (0.9%) is rapidly given, and a 6mg dose of ephedrine is administered IV which restores the blood pressure to 180/90 mmHg.

IV fluid therapy

When anaesthesia is started, the cardiovascular system is depressed, particularly the ability to compensate for a reduction in blood volume (Cardiovascular Physiology Update in Anaesthesia 1999;10:2-8). Therefore part of the treatment of a fall in blood pressure is a rapid infusion of fluid intravenously.

Anaesthesia is now maintained using a further dose of fentanyl 150 mcg or iv morphine 10mg slowly, vecuronium 10mg and the patient ventilated with oxygen enriched air. He is monitored with a pulse oximeter, an ECG and BP. An infusion of 150mls 20% mannitol (0.25 - 0.5 g/kg) is commenced, followed by a litre of 0.9% saline, and a urinary catheter inserted.

He is transported to the CT scanner which shows a large extradural haematoma (figure 3) and therefore is immediately taken to the operating theatre for craniotomy.

Figure 3: CT scan of a patient with an acute extradural haematoma

Teaching Point
Hypotension following induction

Reasons for concern - hypotension will reduce perfusion pressure in the brain. This has two adverse effects:

1 Blood flow through the brain will fall dramatically, reducing oxygenation of the brain and cause ischaemia. The brain tolerates this badly and will suffer major neurological damage (stroke, paralysis, death).

2 The arteries in the brain will sense a reduction in flow and will try to compensate (**autoregulation**). They will dilate in an attempt to reduce their resistance and thus increase flow. Dilatation will increase their volume and cause a further increase in brain volume and intracranial pressure, making the situation worse.

Saline vs Colloid vs Dextrose

The brain is surrounded by a membrane separating it from the vascular space - the **blood-brain barrier.** This membrane will only allow water to pass through it. **Therefore only fluid with the same concentration of sodium as plasma should be given intravenously.** Otherwise, the plasma will become more dilute and water will pass from it into the brain, making the brain swell, and thus increase pressure further.

Normal Saline (0.9%) has a similar concentration of sodium and therefore is the fluid of choice for the brain. Colloid can be given if required to treat hypovolaemia due by major blood loss.

When Dextrose solutions in water (5% Dextrose, Dextrose 4%-Saline 0.18%) are given, the dextrose is metabolised leaving just the water or a very dilute saline solution. This "dilutes" the blood, reducing the concentration of sodium in the plasma. The water then passes into the brain where the concentration of sodium is higher. The brain then swells, and intracranial pressure will rise.

Vasopressors As explained, the blood pressure must be raised quickly. Therefore a small dose of cardiovascular stimulant drug can also be given intravenously to raise the blood pressure while the fluid is being run in. Suitable drugs include ephedrine, 3-6 mg, methoxamine 1-2 mg, or adrenaline 25-100 mcg.

Theatre

There is a wide range of anaesthetic drugs and hence techniques available. Anaesthesia may be maintained with an inhalational agent - isoflurane would be the first choice and halothane the second choice. If only ether is available then use ether. If available, increments of morphine, pethidine or preferably fentanyl should be used as narcotics reduce the risk of coughing and the concentration of inhalational agent required. Neuromuscular blockade is maintained with non-depolarising muscle relaxants, to avoid coughing and straining with the minimum concentration of the inhalational agent required. However, if no long acting muscle relaxant is available then continue with intermittent positive pressure ventilation using the inhalational agent to suppress normal ventilation. In some units where propofol is available, anaesthesia can be maintained with a propofol infusion (2-6 mg/kg/hour), oxygen enriched air, and small increments of narcotics. The infusion rate is adjusted to ensure that the blood pressure does not fall.

The circulation is monitored by observing the peripheral circulation, pulse rate, blood pressure and urine output. Blood pressure should be monitored either invasively or frequently with a cuff. Continuous measurement of the patient's blood pressure is very helpful as it allows blood pressure changes to be treated accurately and efficiently. Do not delay surgery to insert an arterial line. However there is usually time during preparation of the patient to attempt radial artery cannulation. If this proves difficult then a cannula can be put into the femoral artery to provide monitoring for the duration of surgery.

Operation

At operation, a large clot is found under high pressure. Once released, the blood pressure falls to normal levels. The bleeding point is identified and secured. The brain which was compressed, is pulsating with each heart beat and respiration. If an Intensive Care (ICU) bed is available and it is decided to ventilate the patient for a period postoperatively, an ICP monitor is inserted. As described below, a catheter is inserted subdurally and connected to an arterial transducer.

Postoperative care

The patient is now taken to ICU to allow the anaesthetic drugs to wear off with an intracranial pressure monitor in situ. After some hours he is opening his eyes, coughing on the ET tube and breathing well. His ICP is 12 mmHg and he is allowed to wake up and is extubated.

An inexpensive ICP monitor

If an arterial pressure transducer is available this can be done simply with a neonatal umbilical catheter. The catheter is inserted either subdurally or intraventricularly and filled with saline by the surgeon. A 1-2 cm subcutaneous tunnel is formed to reduce the risk of the catheter being pulled out. Care must be taken to avoid kinking the catheter. Under aseptic conditions it is connected to a transducer without the usual pressure heparin flush system. A dampened looking pressure trace is seen with pressure fluctuations with each cardiac cycle. It will rise with coughing and will be flat if it is blocked or ICP is very high. It does not often block, but if this happens only the surgical team should attempt to unblock it. Intraventricular catheters are less likely to block but have greater risk of infection, their use being limited to 5 days

Conclusion

1   The initial assessment and resuscitation is vital and must be carried out: a scheme is described.

2   A method of anaesthesia is described based on simple physiological and pharmacological principles which, when used, will reduce the risk of added damage.

An unconscious patient with an extradural haematoma will die or be permanently severely damaged unless treated quickly and correctly. Urgent surgical decompression is required. It ALONE is not enough. Attention to basic simple clinical details will prevent additional irreversible damage occurring before the intracranial pressure can be reduced by releasing the haematoma.

Teaching Point

At the end of surgery the anaesthetist should achieve the following aims

1   Prevent further damage from physiological factors which will cause brain swelling.

2   Observe the patient to detect any deterioration.

3   Provide adequate analgesia.

Deterioration postoperatively may occur from brain swelling or accumulation of a further haematoma. The best method of detecting any deterioration in neurological status postoperatively is observation of the conscious patient, looking for a change in conscious level and new or changed neurological signs. Therefore when a simple haematoma without bruising or contusion to the brain is removed early from an otherwise fit patient who has no other trauma, as in this case, it is better to wake the patient up immediately after surgery. Anaesthesia is stopped and the patient extubated. In contrast, when there is significant brain contusion or other systems are damaged by the accident, waking and extubation are delayed if an ICU bed is available.

ICU available

The patient is taken to the ITU where ventilation and sedation are continued. BP, pulse and ICP are monitored. When the ICP remains normal, the patient is allowed to wake up and be extubated.

ICU not available

Anaesthesia is stopped in theatre and the patient allowed to wake up. Neuromuscular block is reversed and spontaneous respiration allowed to start. Extubation should be carried out when the endotracheal tube is seen to be irritating the trachea. Initially the patient lies in the recovery (lateral) position until airway reflexes have returned. The patient is then sat up at about 30° to reduce cerebral venous congestion. The patient should be given the best nursing care that the hospital can provide with Glasgow Coma Score or AVPU recordings started, recorded and repeated at 15 minute intervals. Any deterioration in conscious level or appearance of new neurological signs is reported to the surgical team immediately.

NERVE BLOCKS FOR ANAESTHESIA AND ANALGESIA OF THE LOWER LIMB - A PRACTICAL GUIDE: Femoral, Lumbar plexus, Sciatic

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Introduction

The purpose of this guide is to provide a detailed, step by step description of how to safely and reliably perform nerve blocks for surgery and pain relief on the lower limb, for the non-specialist anaesthetic practitioner. It covers femoral nerve blockade, lumbar plexus blockade using the inguinal paravascular approach and sciatic nerve blockade. No description is given of more distal blocks, since the majority of the limb is readily anaesthetised with the techniques described and as a group they are relatively simple, reliable and commonly used.

Conduct of nerve blocks

It is recommended that all blocks on major nerves be carried out on patients that are awake or only lightly sedated, as it is believed that this decreased the risk of serious nerve damage, and will not hide the signs of unexpected local anaesthetic toxicity. Sedated patients must still be able to communicate with the operator during the nerve block procedure. It is also recommended that a nerve stimulator be used if one is available as this increases the success rate of the blocks especially in inexperienced hands.

Local anaesthetic drugs and dosages

For fast onset and short duration blocks, lignocaine (maximum 4mg/kg) or lignocaine with adrenaline 1:200,000 (6mg/kg) can be used. When injected into a plexus or near a large nerve such as the sciatic nerve the block will come on at about 10-20 mins and last for 4-8 hours. The adrenaline will prolong the block but may possibly increase the risk of nerve damage through ischaemia. Bupivacaine in a maximum dose of 3mg/kg will give a block of a major nerve that will start at 20-30 minutes and last as long as 18 hours. There is no value in adding adrenaline to bupivacaine except for local skin infiltration.

Anatomy

The nerve supply of the lower limb is derived from the lumbar and sacral plexuses, a network of nerves composed of the anterior primary rami of all the lumbar and the first three sacral nerve roots (and sometimes with a contribution from the twelfth thoracic nerve root). Arising from these plexuses are the five main nerves that innervate the lower limb.

The lumbar plexus: This gives rise to the femoral nerve, obturator nerve and lateral cutaneous nerve of the thigh.

The femoral nerve runs in the groove between the psoas major and iliacus muscles, with a covering of these muscles' fascia. It enters the thigh passing under the inguinal ligament, where it is lateral to the femoral artery, whose pulsations are used to help locate the nerve. The femoral nerve block is performed at this point and there are two important features of the anatomy. Firstly, below the inguinal ligament, the femoral nerve divides into anterior and posterior branches, the anterior (superficial) branch supplying sensation to the skin of the anterior and medial thigh and a posterior (deep) branch that supplies the quadriceps muscles, the medial knee joint, and the skin on the medial side of the calf and foot (via the saphenous nerve). Therefore, the block should not be performed lower than just distal to the inguinal ligament, in order not to miss one of the branches. Secondly, as it enters the thigh, the femoral nerve has two fascial layers covering it, the fascia lata and the fascia iliaca. This is in contrast to the femoral artery, which is only covered by the fascia lata alone. This means that the nerve will lie in a different tissue plane than the artery and usually a little deeper. These coverings can be used in blocking the nerve. (See techniques).

The lateral cutaneous nerve of the thigh and the obturator nerve both have important sensory distributions (to the thigh and knee). This article does not cover blocks of these nerves as single entities. However, they can readily be blocked in conjunction with the femoral nerve, using the same technique, to produce a "3-in-1" block and this is described later. (see Blockade of the lumbar plexus using the inguinal paravascular approach).

The sacral plexus: This gives rise to the sciatic nerve and the posterior cutaneous nerve of the thigh. Although these nerves are formed separately within the plexus, they pass through the pelvis and buttock together and with the techniques described here are blocked with the same injection. Hence they are considered here as a single nerve trunk, and unless specifically stated, "sciatic nerve" will refer to both the sciatic nerve and the posterior cutaneous nerve of the thigh.

The sciatic nerve leaves the pelvis and enters the buttock through the greater sciatic foramen, and then passes slightly medial to the midpoint between the greater trochanter and the ischial tuberosity, lying just posterior to the hip joint. It can be blocked at several points along this course (see techniques). The sciatic nerve leaves the buttock, passing out from under the lower border of gluteus maximus muscle and runs distally down the thigh to the popliteal fossa.

Areas supplied by the individual nerves:

A pictorial illustration of the skin areas supplied is given in figure 1.

Femoral nerve: supplies skin over the anterior (front) of the thigh, the anterior knee, some of the medial (inner) thigh, via the saphenous nerve it also supplies the antero-medial aspect of the calf down to and including the medial malleolus. It has branches to the hip joint and knee joint and supplies much of the shaft of the femur. Sensation to the medial aspect of the big toe may come from the saphenous (femoral nerve).

Lateral cutaneous nerve of the thigh: supplies sensation to the skin over the lateral (outside) thigh, from the greater trochanter to the knee, and on to the anterior thigh.

Figure 1: Cutaneous nerve distribution of the lower limb.

Obturator nerve: supplies a small, variable amount of skin on the medial aspect of the knee and lower thigh. More importantly, it has a branch to the knee joint. There is also a small branch to the hip joint.

Posterior cutaneous nerve of the thigh: supplies skin over the posterior (back) thigh, the popliteal fossa, the lower buttock and some of the genital area. Note that this nerve is blocked with the posterior approaches and is often missed with the anterior approach to the sciatic nerve.

Sciatic nerve: via its branches supplies all the skin of the leg below the knee and all the foot, except for the medial calf and ankle, which is supplied by the saphenous (femoral) nerve. The sciatic nerve also has a small branch to the hip joint, a branch to the knee joint and fully innervates the ankle joint.

Surface anatomy markings

When it comes to performing the nerve blocks, it is crucial to be able to palpate and accurately locate bony landmarks, since these are the reference points we use for determining the correct site for needle insertion. The following is a description of the bony landmarks used for femoral and sciatic nerve blocks. They are shown in the diagrams of the nerve block techniques.

Anterior superior iliac spine following the iliac crest (ridge of the pelvic bones) from the flanks forwards, it ends in an obvious bony prominence, at the side of the lower abdomen. This is the anterior superior iliac spine.

Pubic tubercle is the bony prominence that can be felt at the inner (medial) end of the groin crease. It is about 2 - 4 cm from the midline, at the top of the genital area.

Posterior superior iliac spine is the bony prominence at the posterior end of the iliac crest. It is directly caudal to the "sacral dimple" - that depression in the skin visible cranial to (above) the buttocks, on each side, close to the midline.

Greater trochanter this bony landmark is part of the lateral femur, just below the hip joint. It is easy to find at the top of the thigh, protruding directly laterally. With the patient on their side, it represents the highest point on the upper thigh. In obese patients try internally and externally rotating the hip, as this makes the greater trochanter more visible.

The sacral cornu are two bony prominences either side of the midline just at the top end of the natal cleft. One can readily palpate a narrow depression between them - the sacral hiatus. (see figure 3)

The ischial tuberosity is that part of the pelvic bone structure that can be felt posteriorly, on the medial side of the base of the buttock. It is the bony structure that we "sit on."

Indications for specific nerve blocks

From the outline of the areas covered by each nerve, the reader should know which blocks would be useful in a given situation. Two points are worth emphasising. The knee joint has significant contributions from femoral, obturator and sciatic nerves and significant injury or surgery to this joint will require that all these be blocked. (For the hip, it is nearly always sufficient to perform a 3-in-1 lumbar plexus block even though there is a small contribution from the sciatic nerve.) Secondly, the area covered by the different nerves may vary considerably and if in doubt, it is best to block both main nerve trunks.

The following are some examples of the possible uses.

Femoral nerve blocks:

- operations on the anterior thigh, such as repair of large lacerations.

- pain relief for fractures of the shaft of the femur, particularly more proximal fractures.

Lumbar plexus (3-in-1) block:

- all the uses of a femoral nerve block, plus the following:

- pain relief and anaesthesia for hip injuries such as dislocations and fractures of the neck of the femur. (Major hip surgery will also require a sciatic nerve block.)

- anaesthesia for operations on the lateral thigh such as harvesting of skin grafts, or muscle biopsies.

- pain relief for injuries and operations on the knee; extensive injuries and full knee anaesthesia require a sciatic nerve block also

- this block extends the field of a simple femoral nerve block considerably and is no more difficult to perform.

Sciatic nerve block:

- pain relief or anaesthesia for injuries or operations on the sole of the foot or any of the toes, such as toe amputation (amputation of the big toe may require supplementation at the medial maleolus as well, because the

distribution of the saphenous nerve occasionally extends down the medial side of the big toe).

- the distribution of the sciatic nerve means that it has fairly limited application as a block on its own and is most often combined with a femoral or 3-in-1 block.

Combined sciatic and femoral or 3-in-1 block:

- with this combination pain relief and anaesthesia can be provided for almost any injury or operation from the upper thigh downwards.

- one area sometimes not covered is the upper, inner thigh, and possibly the posterior thigh. This may be a problem with tourniquets applied high on the leg and in this situation some supplementary parenteral analgesia or sedation can be useful.

- it may be difficult to provide adequate anaesthesia for major hip surgery, although the blocks described will provide good postoperative analgesia.

Planning the dose of local anaesthetic and dealing with possible side effects

The above discussion will indicate that there are often situations in which one wishes to perform a combined sciatic and 3-in-1 block at the same time. This will necessitate using large volumes of local anaesthetic and the total dose administered may often be at the limit of recommended safe doses. It is important to be able to adjust the concentration of the solution injected when using large volumes, in order to keep the total dose at an acceptable level. See local anaesthetic, drugs and dosage page 56.

Local complications of local anaesthetic blocks:

The most important is damage to the nerve. Permanent nerve damage is very rare. It may be caused by accidentally injecting local anaesthetic within the nerve itself (intraneural) or by traumatising the nerve with the needle point. Two signs of intraneural injection are severe pain on attempted injection and marked resistance to injection. (For the patient to respond to the pain of intraneural injection he or she must be awake, or only slightly sedated.) Either of these warning signs should prompt the operator to stop injecting and reposition the needle. Intraneural injection may also be less likely if a short-bevel needle is used [1]. Paraesthesia is the "electric shock-like" feeling felt as the

nerve is touched by the needle. It should be a warning sign that nerve damage may occur if the needle is inserted further.

It is also possible to cause a haematoma by puncturing an artery with the needle - most commonly this will be the femoral artery. This is rarely of any significance. If the femoral artery is punctured then firm pressure applied to the site for 5 minutes will minimise the haematoma.

Performing the nerve blocks - patient preparation and techniques

When performing any of the blocks that are described here, the steps taken to safely prepare the patient should be carefully followed.

Preparing the patient

- **Consent** - explain the entire procedure to the patient. This will help to relieve any anxiety and increase co-operation.

- **Fasting** - if an elective procedure is planned, then the patient should be fasted similar to having a general anaesthetic. This increases safety in the event that a general anaesthetic or resuscitation is required.

- **Monitoring** - the potential complications described in the preceding section mean that monitoring is essential. If available, ECG and blood pressure monitoring should be used. If sedation is planned then a pulse oximeter should also be used. In every case, the most useful monitor is to maintain careful, continuous observation of the patient throughout. An assistant can be invaluable in helping with this.

- **Intravenous access** - because of the possible complications, should be intravenous access secured before any block is performed. This also allows administration of intravenous fluids, sedative agents and resuscitation drugs if required.

- **Positioning** - take care with positioning the patient for the block and make sure they are as comfortable as possible as this will make the block easier to perform.

- **Identify the bony landmarks** - these are described in the anatomy section.

- **Clean the site** - the skin over the block site should be cleaned with an antiseptic agent and surrounded with sterile drapes. The operator should wash their hands and wear sterile gloves.

- **Perform the block!**

- **Allow time for the local anaesthetic to take effect** - at least 15 - 20 minutes will be required for surgical anaesthesia. With the weaker concentrations of bupivacaine, 30 - 45 minutes may be required.

Techniques

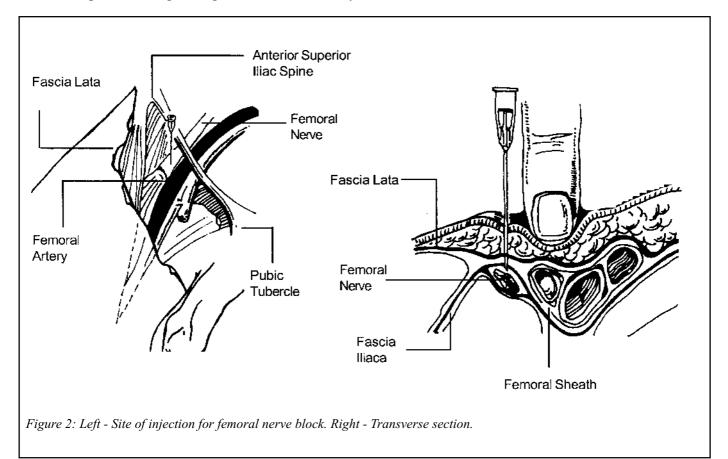### *The femoral nerve and lumbar plexus*

Anatomy The femoral nerve has contributions from the second, third and fourth lumbar nerves. It is derived from the lumbar plexus and in fact lies within the same fascial envelope as the lumbar plexus. This important fact may be utilised to block most of the nerves originating in the lumbar plexus with a single injection distally, as local anaesthetic can be made to spread proximally within this plane. (See anatomy)

Technique (figure 2) The patient lies supine with the leg extended, lying flat on the bed. The operator stands on the side of the patient that is to be blocked. Firstly, identify the point of injection, using the surface landmarks. For the femoral nerve, this is just below (distal to) the inguinal ligament. Palpate both the anterior superior iliac spine and the pubic tubercle. The line between these two overlies the inguinal ligament. It is often helpful to draw the lines that are described on the skin. The femoral artery should lie at the midpoint of the inguinal ligament and it is necessary

to locate this by feeling for the pulse at this point. The site for injection is 1cm lateral to (outside of) the pulsations of the femoral artery and 1 - 2cm below (distal to) the line of the inguinal ligament. Having identified the site, it will be more comfortable for the patient if a small amount of local anaesthetic is used to create a skin wheal ("bleb") at the injection point.

An ordinary needle of length 3 - 4 cm and 21 - 23g in width is suitable for performing this block. It should be inserted perpendicular to the skin, but aiming slightly towards the head of the patient.

The following are two ways of carrying out the block. In the first technique, the operator attempts to locate the nerve by eliciting paraesthesiae, or failing this by depositing the local anaesthetic over a range of areas (the classical technique of Labat [5]). In the second technique, use is made of the fascial layers overlying the nerve and a single injection only is employed (Khoo and Brown[2]). In both cases, it is very important to remember the anatomy. The femoral nerve lies adjacent to but slightly deeper than the structures contained within the femoral sheath (the artery, vein and femoral canal). This is because the nerve lies deep to the fascia iliaca, while the contents of the femoral sheath lie on top of it.

Figure 2: Left - Site of injection for femoral nerve block. Right - Transverse section.

The classical approach The needle is advanced through the skin, as described above, until the patient feels paraesthesiae in the distribution of the femoral nerve. If a depth of 4 - 5cm is reached and no paraesthesiae are found, then it should be withdrawn to just below the skin and advanced again in a slightly medial or lateral direction, repeating this until the patient feels paraesthesiae. Once this occurs, the needle should be fixed in position with one hand, resting this hand on the patient to try and minimise movement. With the other hand, the syringe containing local anaesthetic is then connected to the needle and gentle aspiration performed. If no blood is seen then 15 - 20 mls of local anaesthetic is injected (aspirating again regularly to check for the presence of blood).

The presence of paraesthesiae is the best indicator for correct positioning of the tip of the needle, but it is often not easy or even possible to locate the femoral nerve in this manner.

Alternatively as the needle is advanced alongside the artery, its pulsations may cause lateral (side to side) movement of the hub of the needle. If this is the case, the needle is slowly advanced and frequent observations made until the point is reached when the lateral movements are at their greatest. This generally represents a depth where the tip of the needle is just deep to the artery and should be in the correct plane. The needle is then fixed, the syringe connected and aspiration performed as before. However, only 10 ml of local anaesthetic is injected at this site. This injection is then supplemented by withdrawing the needle slightly redirecting the tip outwards (laterally), inserting to the same depth as before and injecting 3 - 4 mls of local anaesthetic (after aspiration). This process is repeated 2 or 3 times, moving progressively further laterally, such that a total of 20 - 25mls of local anaesthetic is deposited in a "fan-shaped" area lateral and deep to the femoral artery.

The single injection technique[2] This method has the virtue of simplicity and generally involves less probing with the needle. For this reason it is popular but requires some practice for a high success rate.

The site for injection is the same as already described. However, the needle is inserted directly perpendicular to the skin. If the needle is held gently between thumb and forefinger, then a slight resistance is encountered at the fascia lata, followed by a definite loss of resistance, or "pop" as the needle penetrates this layer. The same thing is felt as the needle penetrates the fascia iliaca and comes into the proximity of the femoral nerve. Therefore, immediately on feeling this second loss of resistance, or "pop", the tip of the needle should be in the correct position. The needle is then fixed in position with one hand, the other hand again being used to connect a syringe, aspirate to check for blood and inject 20 ml of local anaesthetic.

This technique is entirely dependent on being able to detect the two points of loss of resistance as each of the fascial layers is penetrated. This is much easier if a short-bevel needle is available to use, as it does not pierce the fascia quite as easily as an ordinary needle, making the feel of the layers more obvious. If a short-bevel needle is not available, then the same effect can be achieved using an ordinary needle but blunting it prior to insertion. The "blunting" may be cleanly achieved by piercing the side of the protective plastic sheath (that comes with the needle) several times with the needle tip before performing the block. The detection of paraesthesiae is not to be recommended when using this technique as the "blunted" needle is more likely to cause nerve damage.

Use of a nerve stimulator If an insulated stimulating needle is available for use, then it is necessary to obtain contraction of the quadriceps muscle group. This is most reliably seen by movement of the patella and extension of the knee joint. (The movement of the knee is not normally obvious, as the patient's leg is usually flat on the bed and fully extended anyway.) The contractions should still be visible at a stimulating current of 0.3 - 0.5 mA indicating adequate proximity to the nerve. Exactly the same technique will be used if one wishes to perform a lumbar plexus block using this approach.

Blockade of the lumbar plexus using the inguinal paravascular approach

This technique is also referred to as the "Winnie 3-in-1 block" after the author who first described it[3]. It is so called, because it aims to block three nerves with the one injection: the femoral nerve, the lateral cutaneous nerve of the thigh and the obturator nerve.

Anatomy For most operations on the thigh and knee it is not sufficient to block the femoral nerve alone. The lateral cutaneous nerve of the thigh supplies all the outside of the thigh and the obturator nerve supplies a variable amount of skin on the inner thigh just above the knee and contributes to the innervation of the knee joint.

All three nerves are derived from the lumbar plexus. The plexus lies on the quadratus lumborum muscle and behind

the psoas major muscle and is invested in a fascial sheath derived from these two muscles. This sheath forms a continuous covering around the plexus, extending down to the femoral nerve just below the inguinal ligament. Therefore if local anaesthetic injected around the femoral nerve at the inguinal ligament can be made to spread proximally, then the other two nerves can be simultaneously blocked at their origins from the lumbar plexus.

Technique The injection point and method for correct needle placement are exactly as described for the femoral nerve. It is most practical to use the single injection technique, as this will enable placement of the large volumes required.

There are two differences; the main difference comes with the actual injection of the local anaesthetic. Having aspirated on the needle to check that the tip is not intravascular, the hand is then moved to apply firm pressure on the thigh (with the thumb) about 2 - 4 cm. below the insertion point of the needle. The injection is then performed, all the while maintaining the pressure. The pressure can be released about thirty seconds after the injection has been completed. (The injection will require an assistant, as the operator will have one hand immobilising the needle and the other applying pressure.) This procedure encourages spread of the local anaesthetic upwards, towards the lumbar nerve roots.

The second difference is that larger volumes of local anaesthetic are used to achieve the necessary spread. The minimum volume to block all three nerves is 20 mls. However, many texts suggest larger volumes such as 25 - 30 mls. When using these volumes, particularly in combination with a sciatic nerve block, it may be necessary to dilute the concentration of local anaesthetic solution used in order to limit the total dose given.

Use of a nerve stimulator: as for the femoral nerve.

The Sciatic Nerve

Anatomy The sciatic nerve is the largest nerve in the body, measuring about 2 centimetres in thickness in its proximal portion. In this portion it is actually made up of the sciatic nerve and the posterior cutaneous nerve of the thigh. This "double nerve" contains contributions from lumbar nerve roots 4 and 5 and sacral nerve roots 1,2 and 3. In the techniques that are described here, this large "double nerve" is considered effectively as a single nerve and blocked with the one injection. For simplicity, it is referred
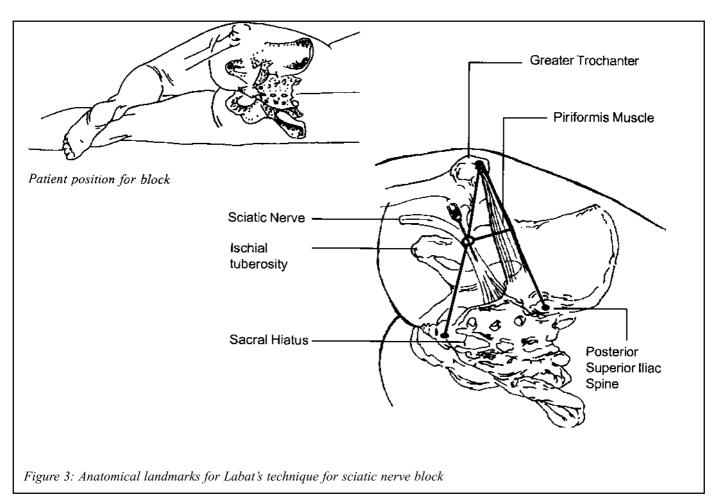
to just as the sciatic nerve.

The important bony landmarks that one needs to be able to identify for blocking the sciatic nerve via the two posterior approaches described are the greater trochanter, posterior superior iliac spine, the ischial tuberosity and the sacral hiatus. For the anterior approach, the landmarks are the anterior superior iliac spine and the pubic symphysis on the pelvis and the greater trochanter on the femur.

Technique It will be appreciated from the description of these landmarks that there are several possible routes to block the sciatic nerve. Three of the most common approaches are described here. The first is the classical posterior approach of Labat[5] performed with the patient in the lateral position. The second is another posterior approach, but the patient is supine and the leg is flexed at the hip and at the knee. Finally, the anterior approach is described where the patient is supine with the legs lying naturally extended. The choice of technique will to some extent be influenced by the position that is easiest for the patient to assume. However, the success rate is higher with the posterior approaches unless a nerve stimulator is used[4]. Furthermore, the anterior technique tends to be technically more difficult[4] and therefore it is suggested that every effort should be made to position the patient for one of the posterior approaches.

Posterior approach of Labat[5] (figure 3) The patient is first placed in the lateral position with the side to be blocked uppermost. While the lower leg is kept straight, the upper leg is flexed at the knee so that the ankle is brought over the knee of the lower leg. Another way of achieving the correct degree of hip and knee flexion is to have the posterior superior iliac spine, the greater trochanter and the knee in a straight line.

The point of injection is identified as follows:

A line is drawn between the greater trochanter and the posterior superior iliac spine (the line lies approximately over the upper border of the piriformis muscle). From the midpoint of this line, at right angles to it, draw a second line passing down over the buttock. The point of injection is 3 - 5 centimetres along this perpendicular line. It can be more precisely identified by drawing a third line between the greater trochanter and sacral hiatus, the point of injection being where this third line intersects with the second, perpendicular line. Having identified this point, place a small wheal of local anaesthetic at the site.

Figure 3: Anatomical landmarks for Labat's technique for sciatic nerve block

The needle that is required for this block needs to be quite long. A standard adult lumbar puncture needle is usually sufficient (9cm, 22G). In a very large person, an extra long needle, (10 - 12cm) may make the location of the nerve an easier task.

The needle is inserted perpendicular to the skin and slowly advanced until either bone is encountered, or paraesthesiae are elicited. (For the block to be successful, paraesthesiae below the knee should be felt.) If bone is encountered, the needle is withdrawn approximately 1-3cm and redirected slightly, either medially or laterally. Gentle probing within this single (transverse) plane should enable the nerve to be located by producing paraesthesiae as described. If the needle has been inserted as far as possible and neither paraesthesiae elicited nor bone encountered then the tip may have entered the greater sciatic notch. Should this occur, then the needle should be withdrawn almost fully, until the tip is just beneath the skin and then redirected in a slightly medial or lateral plane as described above.

Having located the nerve by paraesthesiae, the needle should be fixed in position and a syringe containing approximately 20ml of local anaesthetic connected. Aspiration is performed to exclude intravascular placement of the needle and the local anaesthetic is then injected. Repeat aspiration half way through the injection, in case the tip of the needle has moved). It is important that if severe pain occurs or if there is significant resistance to injection then the operator should stop immediately and reposition the needle, as these may be signs of intraneural injection.

Alternative posterior approach (of Raj) If it is not possible to have the patient lying on their side, then a variation of the posterior approach may be performed with the patient supine, although the hip is still manipulated.

To perform this block, the operator stands by the patient's bed, on the side to be blocked. The hip is then flexed as much as possible with knee bent. This position can be held stable by bracing the foot against the front of the operator's shoulder as they face towards the head of the bed. Alternatively, an assistant can hold the leg steady. The greater trochanter is palpated on the outside of the leg and the ischial tuberosity is also located, being the main prominence at the base of the buttock. It is often possible

to palpate these two bony protuberances at the same time using the thumb and middle finger of the same hand. The midpoint between these two landmarks is the injection point. It is sometimes possible to identify this line joining the greater trochanter and ischial tuberosity as a depression between two muscle bellies - semitendinosus and biceps femoris.
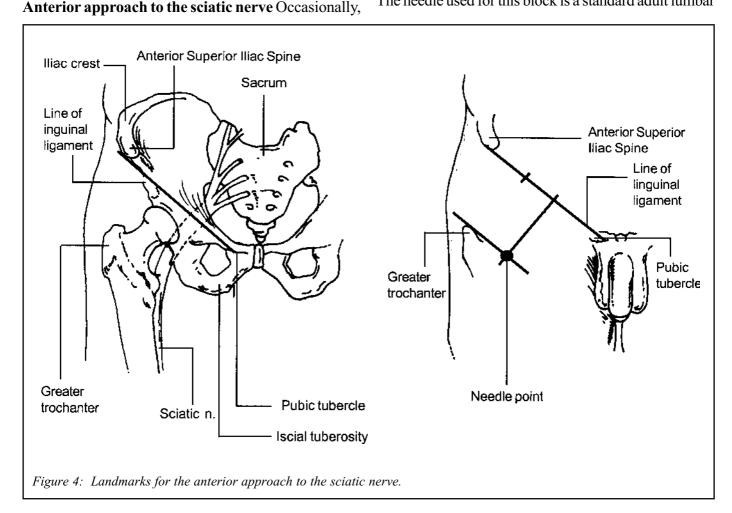
Having located the injection point a small skin wheal is raised and the same needle as above is inserted at right angles to the skin. Once again the aim is to elicit paraesthesiae below the knee and this is achieved exactly as described above - by gentle probing in the transverse ("side to side") plane. The sciatic nerve is most likely to lie slightly medial to the path of the needle if these landmarks are used. The injection of local anaesthetic is then also performed in exactly the same manner as for the classical technique of Labat.

It should be noted that this alternative approach would block the sciatic nerve several centimetres more distally than the first approach described. However, it is rare to miss the posterior cutaneous nerve of the thigh, even with the alternative approach.

Anterior approach to the sciatic nerve Occasionally,

it will be not be possible to move the patient's leg from the neutral position with them lying supine. In this case a sciatic nerve block can be performed using an anterior approach although as already stated, this tends to be more of a technical challenge! This technique will also result in the sciatic nerve being blocked at a relatively distal point (just beyond the hip joint) and hence it is possible to miss the posterior cutaneous nerve of the thigh, which becomes separated from the sciatic nerve by the hamstring muscles just below the buttock.

The landmarks for the point of injection are as follows: (see figure 4) firstly, trace a line over the inguinal ligament (from the anterior superior iliac spine to the pubic tubercle) and divide it into thirds. From the junction of the inner and middle thirds draw another line at right angles to the first, going down the leg. The next step is to find the greater trochanter and from this draw another line parallel to the line over the inguinal ligament (the first line drawn). Where this last line crosses the perpendicular line (the second line) is the point of injection. A small skin wheal of local anaesthetic is then injected at this site.

The needle used for this block is a standard adult lumbar

Figure 4: Landmarks for the anterior approach to the sciatic nerve.

puncture needle (9 cm long). However, the depth of insertion required for this block is commonly greater than for the posterior approaches and a longer needle is often required.
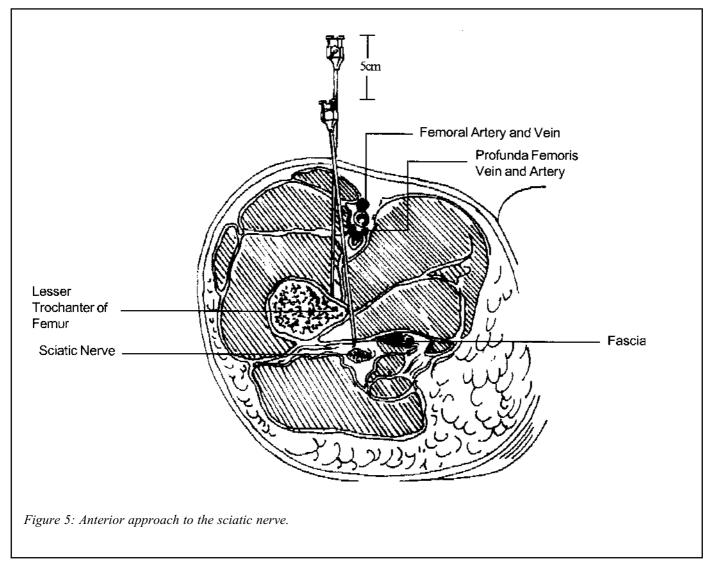
The needle is then inserted perpendicular to the skin, which means aiming in a slightly lateral direction. The operator should aim to strike bone, close to the medial edge of the femur. This will be at about the level of the lesser trochanter. The needle is then withdrawn slightly, redirected more medially (it should be more towards the vertical) and advanced, this being repeated until the needle is "walked off" the bone, the aim being to just slip past the medial edge of the femur. The operator should note the depth at which the needle initially strikes the femur. Normally this is done by carefully observing the portion of needle shaft remaining at the skin. Once the needle has been directed off the bone, as described above, it should be inserted a further 5 cm (2 inches). The tip of the needle should now be in the region of the neuromuscular bundle. (See figure 5)

Aspiration to check for intravascular needle placement is particularly important if this approach is used, as there is a higher likelihood of vascular puncture. Having aspirated, the needle is immobilised in the usual fashion and approximately 20 ml of local anaesthetic injected.

If there is resistance to injection, then the tip of the needle may still be within muscle substance. If this occurs then the needle should be slowly advanced until injection is easily accomplished.

This technique does not require that paraesthesiae are found, but if they are elicited this is a positive sign of correct needle placement.

If one wishes to be positive about the needle placement and it is not possible to elicit paraesthesiae using the anterior approach as just described, then it is sometimes helpful to use a more medial injection point (about 1 - 2cm inside of the one described). This means that the needle will pass the medial edge of the femur at more of an angle than

Figure 5: Anterior approach to the sciatic nerve.

before and the tip will end posterior to the femur. This may help find the nerve, which tends to lie slightly behind the femur at this level. (When using this more medial injection point, it may help to place the free hand under the buttock and palpate the ischial tuberosity. The needle is then aimed at a point estimated to be 1 - 2cm lateral to the ischial tuberosity.)

Performing a sciatic nerve block using a nerve stimulator A nerve stimulator may be used in conjunction with any of the approaches to the sciatic nerve that have been described above. The techniques for determining the point of injection and locating the nerve are no different, except that one will look for muscle contraction. The best indicator of proximity to the nerve is dorsiflexion of the foot at the ankle and one should aim to achieve this at a stimulating current of 0.3 - 0.5 mA. However, when using the posterior approaches, one may also see contraction of the "hamstring" muscles down the back of the thigh, which may be taken as a sign of proximity to the sciatic nerve. Having achieved muscle contraction at the required stimulating current, injection of local anaesthetic is performed in the usual manner.

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CLINICAL MANAGEMENT OF DIABETES MELLITUS DURING ANAESTHESIA AND SURGERY

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INTRODUCTION

Diabetes is a condition where the cells of the body cannot metabolise sugar properly, due to a total or relative lack of insulin. The body then breaks down its own fat, proteins and glycogen to produce sugar, resulting in high sugar levels in the blood (hyperglycaemia) with excess by-products called ketones being produced by the liver.

There are two main types of diabetes (table 1) which classically affect different age groups. In reality there is a huge overlap between age groups.

Diabetes causes disease in many organ systems, the severity of which may be related to how long the disease has been present and how well it has been controlled. Damage to small blood vessels (diabetic microangiopathy) and nerves (neuropathy) throughout the body results in many pitfalls for the unwary anaesthetist. The following

guidelines should help to identify these problems and cope with them.

Preoperative assessment. The general preoperative assessment has been reviewed in a previous article. Update in Anaesthesia in 1997;7.

Specific problems arise:

Cardiovascular- diabetics are more prone to hypertension, ischaemic heart disease, cerebrovascular disease, myocardial infarction which may be silent and cardiomyopathy. Damage to the nerves controlling the heart and blood vessels (autonomic neuropathy) may result in sudden tachycardia, bradycardia or a tendency to postural hypotension. A history of shortness of breath, palpitations, ankle swelling, tiredness and of course chest pain should therefore be sought and a careful examination

Table 1.	Classification of diabetes mellitus *	
	Insulin Dependent (Type I)	**Non Insulin Dependent (Type II)**
Age of onset	Infancy to twenties	Sixties onwards, occasionally younger
Pathology	Pancreas unable to produce insulin (autoimmune disorder)	Body unable to use insulin properly
Treatment	Insulin	Diet and oral hypoglycaemics.

* Note. This is a general classification and there is considerable overlap. Obesity is a common cause of Type II- the pancreas cannot make enough insulin for the body size. Diet /oral hypoglycaemics may initially be enough but eventually insulin may be required.

for heart failure (distended neck veins, ankle swelling, tender swollen liver, crackles heard on listening to the chest) made. A preoperative ECG should be performed. Heart failure is a very serious risk factor and must be improved before surgery with diuretics. Table 2 describes how to test clinically for autonomic neuropathy.

Renal - kidney damage may already be present, often indicated by the presence of protein (albumin) in the urine. Urine infections are common and should be treated aggressively with antibiotics. The diabetic is at risk of acute renal failure and retention postoperatively. Blood electrolyte measurement (if possible) may reveal a raised urea and creatinine. If the potassium is high (> 5 mmol/l) then specific measures should be taken to lower it before surgery.

Respiratory - diabetics, especially if obese and smokers, are particularly prone to chest infections. Chest physiotherapy pre and postoperatively are indicated, with nebulised oxygen and regular bronchodilators (salbutamol 2.5-5mg in 5ml saline) if wheeze is heard. A chest X-ray, blood gases and spirometry are the gold standard investigations, but careful repeated clinical assessment will usually reveal when a patient is as good as they are going to get. Non-emergency surgery should be delayed until this point.

Airway - thickening of soft tissues occurs eg ligaments around joints. If the neck is affected there may be difficulty extending the neck, making intubation difficult. To test if the patient is at risk, ask them to bring their hands together as in praying. If they cannot have the fingers of each hand flat against the other hand, then they probably have ligament thickening of the finger joints, and difficult intubation should also be anticipated.

Gastrointestinal - the nerves to the gut wall and sphincters can be damaged. Delayed gastric emptying and increased reflux of acid make them more prone to regurgitation and at risk of aspiration on induction of anaesthesia. A history should be sought of heartburn and acid reflux when lying flat; if present they should have a rapid sequence induction with cricoid pressure, even for elective procedures. If available, prescribe an H_2 antagonist and metoclopramide as a premedication. Ranitidine 150mg or cimetidine 400mg plus metoclopramide 10mg orally 2 hours preoperatively to reduce the volume of stomach acid.

Table 2:	Detecting autonomic neuropathy		
	Tests for autonomic neuropathy	**Normal response**	**Abnormal response**
Sympathetic System	Measure systolic blood pressure lying down then standing.	Decrease < 10 mm Hg	Decrease > 30 mm Hg
Parasympathetic system	Measure heart rate response to deep breathing	Increase rate > 15 beats /min	Increase < 10 beats /min

Note: if above detected, patient at risk of unstable BP, myocardial ischaemia, arrhythmias, gastric reflux and aspiration, inability to maintain body temperature under anaesthesia.

Eyes - cataracts are common, as is an abnormal growth of blood vessels inside the eye (retinopathy). The anaesthetist should try to prevent sudden rises in blood pressure that might rupture them, further damaging the eyesight. Ensure an adequate depth of anaesthesia, especially at induction.

Infection - diabetics are prone to getting infections that can upset their sugar control. If possible, delay surgery until these are treated. Wound infections are common. Great care should be paid to aseptic techniques when any procedure is undertaken.

Miscellaneous - diabetes may be caused or worsened by treatment with corticosteroids, thiazide diuretics and the contraceptive pill. Thyroid disease, obesity, pregnancy and even stress can affect diabetic control.

Blood and urine glucose monitoring - meter analysis (most accurate) or reagent strips (which employ a visual colour comparison with a pre-printed chart) are commonly available. It is vital that the instructions are properly followed for whatever method is used. Out-of-date strips will give an inaccurate reading. If strips are cut in half for economy (not recommended), then the unused portion must be carefully stored in a dry place. When using meters, ensure that the testing strips are properly matched for the meter. Remember, false readings could lead to the wrong, even life threatening treatment being given. Strips or tablets can also be used to test the urine for glucose or ketones. The same precautions apply.

Anaesthetic management:

Many of the operations diabetic patients face are a direct result of their disease. Skin ulcers, amputations and abscesses are amongst the commonest.

Preoperative assessment-

Timing - diabetic patients should be placed first on the operating list. This shortens their preoperative fast. Badly controlled diabetics need to be admitted to hospital one or two days before surgery if possible to allow their treatment to be stabilised.

Hydration - Glucose in the urine (glycosuria) causes a diuresis which makes the patient dehydrated and even more susceptible to hypotension. Check for dehydration (Table 3) and start an intravenous infusion.

Medication - all medications should be continued up until surgery. Surgery causes a stress response which will change the patient's insulin requirements. Treatment will need to be adjusted according to:

-      the extent of the anticipated surgery

-      whether the patient is insulin dependant (IDDM) or non-insulin dependant (NIDDM)

-      the quality of their blood sugar control.

In general, if the patient can be expected to eat and drink within 4 hours of surgery, then it is classified as MINOR. All surgery other than minor is classified as MAJOR. Figures 1-4 give regimes for major and minor surgery and for NIDDM and IDDM.

The aim is to keep the blood glucose level within the range 6 -10 mmol/l at all times.

Special problems.

Low Blood Sugar (hypoglycaemia)-

The main danger to diabetics is low blood sugar levels (blood glucose < 4mmol/l). Fasting, alcohol, liver failure, septicaemia and malaria can cause this. The characteristic signs and symptoms of early hypoglycaemia are tachycardia, light-headedness, sweating and pallor. If hypoglycaemia persists or gets worse then confusion, restlessness, incomprehensible speech, double vision, convulsions and coma will ensue. If untreated, permanent brain damage will occur, made worse by hypotension and hypoxia.

Anaesthetised patients may not show any of these signs. The anaesthetist must therefore monitor the blood sugar regularly if possible, and be very suspicious of any unexplained changes in the patient's condition. If in doubt, regard them as indicating hypoglycaemia and treat.

Treatment - diabetic patients learn to recognise the early signs and often carry glucose with them to take orally. If unconscious, 50ml of 50% glucose (or any glucose solution available) given intravenously and repeated as necessary is the treatment of choice. If no sugar is available, 1mg of glucagon intramuscularly will help.

High Blood Sugar (hyperglycaemia)-

This is defined as a fasting blood sugar level > 6 mmol/l. It is a common problem found in many conditions other than diabetes eg - pancreatitis, sepsis, thiazide diuretic therapy, ether administration, glucose infusions, parenteral nutrition administration and most importantly, any cause of stress such as surgery, burns or trauma. Slightly elevated levels are thus commonly found after routine major surgery. It is usual to treat this only if the level is above 10 mmol/l. At this level, sugar is present in the urine and causes a diuresis which may result in dehydration, loss of potassium

(hypokalaemia) and sodium (hyponatraemia) ions. The blood thickens and this may cause clotting problems such as thrombosis, and could precipitate a crisis in a patient with sickle cell disease.

Assess the patient, rehydrate them and delay surgery if necessary. Remember the aim is a sugar level of 6-10 mmol/l. If the sugar is below 10 mmol/l, observe and recheck it hourly throughout the operation. Should it be above 10 mmol/l, then follow the regimes in figures 1 - 4, according to the extent of the surgery planned.

After surgery, the insulin requirements fall as the stress response subsides. Newly diagnosed diabetics need further investigation to establish whether they will need insulin therapy, oral hypoglycaemics or indeed just diet control.

Sometimes when the blood sugar has become very high, the patient becomes comatose (diabetic coma). It is vital to correct this by adhering to the general guidelines and regimes already mentioned. Aim to reduce the sugar levels to below 10 mmol/l. When this has happened over a few days, the body uses its own fat to produce energy, and this results in high levels of waste products (ketones) in the blood and urine - this is called **diabetic ketoacidiosis** and is a medical emergency with a significant mortality.

Diabetic ketoacidosis

This may be triggered by infections or other illnesses such as bowel perforations and myocardial infarction. The patient will be drowsy or even unconscious with fast, deep breathing due to acid in the blood. The ketones make their breath smell sweetly, like acetone. Ketones can also be detected by the use of urine and blood testing strips.

Diarrhoea, vomiting, gastric dilatation (insert a nasogastric tube) or even severe abdominal pain may be present which can be misinterpreted as an acute surgical problem! As severe dehydration is usually present, surgery must be delayed until fluid resuscitation has commenced in order to avoid disastrous hypotension with induction agents. A urinary catheter will help monitor fluid balance, and an ECG and CVP line (to estimate the fluid deficit) are helpful. The aim is to slowly return the body chemistry to normal.

Give high flow oxygen therapy.

Although the blood potassium level is usually high, the body has actually lost large amounts in the urine, and extra potassium is required intravenously. It is important to lower the blood sugar level slowly, as reducing it too fast can result in further complications such as brain oedema and convulsions. Search for infections (chest X-ray, blood and urine cultures) and treat with antibiotics. Blood gases and electrolyte measurements may also help management. figure 5 gives a regime for treatment.

Anaesthetic technique.

Intraoperative monitoring - record blood pressure and pulse every 5 minutes during the operation, and watch skin colour and temperature. If the patient is cold and sweaty, then suspect hypoglycaemia, check the blood glucose and treat with intravenous glucose

General anaesthesia - if gastric stasis is suspected then a rapid sequence induction should be used. A nasogastric tube can be used to empty the stomach and allow a safer awakening. There are no contraindications to standard anaesthetic induction or inhalational agents, but if the patient is dehydrated then hypotension will occur and should be treated promptly with intravenous fluids. Hartmanns solution (Ringers lactate) **should not** be used in diabetic patients as the lactate it contains may be converted to glucose by the liver and cause hyperglycaemia.

Sudden bradycardias should respond to atropine 0.3mg iv, repeated as necessary (maximum 2 mg). Tachycardias, if not due to light anaesthesia or pain, may respond to gentle massage on one side of the neck over the carotid artery. If not then consider a beta-blocker (propanolol 1mg increments: max 10mg total or labetalol 5mg increments: max 200mg in total).

IV induction agents normally cause hypotension on injection due to vasodilatation. If a patient has a damaged autonomic nervous system (and many diabetics do), then they cannot compensate by vasoconstricting, and the hypotension is worsened. Reducing the dose of drug and giving it slowly helps to minimise this effect.

Regional techniques - are useful because they get over the problem of regurgitation, possible aspiration and of course difficult intubation. However, the same attention should be paid to avoiding hypotension by ensuring adequate hydration. It is a wise precaution to chart any pre-existing nerve damage **before** your block is inserted.

With spinals and epidurals, autonomic nerve damage means the patient may not be able to keep their blood pressure in a normal range. Intervene early with ephedrine (6mg boluses) when the systolic pressure falls to 25% below normal.

Postoperative therapy regimes are also given in figures 1 - 4. It is not unusual to find that insulin requirements are reduced once the patient begins to recover from surgery.

Figure 1.	**Which regime for my patient ?**

1.   Decide on the type of surgery

> **Minor** - patients expected to eat and drink within 4 hours of operation
> **Major** - all other patients

2.   Then, is the patient **Insulin** or **Non-insulin dependent ?**

3.   Finally, are they   -   **poorly controlled**: delay surgery and change to soluble insulin three times daily
>   *but* if surgery urgent, use **Major surgery regime**
>   -   **well controlled**: use the appropriate regime from the **Major** or **Minor**

General Measures for all diabetics:
Measure random sugar preoperatively -   4 hourly for IDDM
                                    -    8 hourly for NIDDM

Test urine 8 hourly for ketones and sugar
Place first on operating list
Aim for a blood glucose of 6 - 10mmol/l

Figure 2.	**Minor Surgery**	
Non insulin Dependent Diabetics	**Preoperatively** - random blood sugar on admission < 10 mmol/l > 10 mmol/l	Normal medication until day of op Follow as for MAJOR SURGERY
	Day of operation	Omit oral hypoglycaemics Blood glucose- 1 hour preop and at least once during op (hourly if op > 1 hour long) postop - 2 hourly until eating then 8 hourly
	Postoperatively	Restart oral hypoglycaemics with first meal
Insulin dependent Diabetics This regime only suitable for patients whose random sugar is < 10 mmol/l on admission, will only miss one meal preop & are first on the list for very minor surgery eg cystoscopy	**Preoperatively**	Normal medication
	Day of operation	No breakfast, no insulin, place first on list. Blood glucose- 1 hour preop and at least once during op (hourly if op > 1 hour long) postop - 2 hourly until eating then 4 hourly
	Postoperatively	Restart normal S/C insulin regime with first meal

Figure 3.	Major surgery

- All insulin dependent and non-insulin dependent who are poorly controlled (blood glucose >10mmol/l) (many NIDDM become insulin dependent during major surgery and will need managing as such. Regular glucose measurements will detect this).
- Normal medication until day of operation

Day of operation - Omit oral hypoglycaemics and normal subcutaneous (S/C) insulin

Blood glucose - check blood sugar(and potassium) 1 hour preop
then 2 hourly from start of infusion
at least once during operation
(hourly if op > 1 hour long)
at least once in recovery area
2 hourly post operatively

Regime 1 - no infusion pump available.

Start intravenous infusion of 5 or10 % dextrose (500 ml bags) over 4 - 6 hours and add Insulin and Potassium Chloride (KCl) to each 500 ml bag as below. Change bag according to blood sugar level readings:-

Blood glucose (mmol/l)	Soluble insulin (units) to be added to bag	Blood potassium (mmol/l)	KCl (mmol) to be added to bag
< 4	No insulin		
4 - 6	5	<3	20
6 - 10	10	3 - 5	10
10 - 20	15	> 5	None
> 20	20		

* If blood potassium level not available, add 10 mmol KCl

Postoperatively

- *Non-insulin dependent* - stop infusion and restart oral hypoglycaemics when eating and drinking
- *Insulin dependent* - stop infusion when eating and drinking
  - calculate the total daily dose (units) of insulin the patient was taking preoperatively
  - give this as S/C Soluble insulin (Actrapid), divided into 3 - 4 doses in 24 hours
  - this may need to be adjusted up or down until blood sugar levels stable.
  - once stable restart normal regime

Remember that the patient may need additional fluids depending on surgery, blood loss etc.

Figure 4:	Major surgery - alternative regime

Regime 2 - for use with infusion pumps

The insulin and dextrose infusions are given via separate infusion pumps. This allows better control than regime 1, but care is needed to ensure the separate lines do not become blocked, or that one infusion runs out leaving the other infusing alone.

Insulin infusion - 50 units insulin made up to 50 ml with saline (i.e. concentration is 1 unit per ml)

Blood glucose (mmol / l)	Insulin infused at (units / hour)
< 5	0
5.1 - 10	1
10.1 - 15	2
15.1 - 20	3
> 20	6 & review *

- If it is proving difficult to reduce the blood sugar level, then consider increasing the rate of insulin for each glucose levelor also giving a bolus of Actrapid of 3 - 5 units.
- Patients normally on higher doses of insulin will need higher rates of insulin infusion.
- Dextrose infusion - 5 or 10 % dextrose infused at 100 ml per hour. Add 10 mmol KCl to each 500 ml of solution.
- Post op - follow instructions as in figure 3.

Summary

The diabetic patient presents the anaesthetist with many challenges. Careful attention to clinical signs and rapid action to prevent even suspected hypoglycaemia peroperatively should see them safely through their surgery. The goal is to keep things as normal as possible. Regional techniques are often safer than general anaesthesia, but require the same vigilance.

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Figure 5.	**Treatment of Diabetic Ketoacidosis**

Aims -   rehydration (water and salt)
                                     lower blood sugar
                                     correction of potassium depletion

Start an intravenous infusion of 0.9 % saline as follows-

                          1 litre over 30 minutes
               then   1 litre over 1 hour
               then   1 litre over 2 hours.
               Continue 2 - 4 hourly until the blood glucose is below 15 mmol / l,
               then change to 5% glucose, 1 litre 2 - 4 hrly

Up to 6 -8 litres of fluid may be required or more. Use clinical signs BP, heart rate, CVP, conscious level to judge the amount.

Give soluble insulin (Actrapid) intramuscularly (IM) as follows-

               -        20 units IM first dose then 6 units IM hourly
               -        measure the blood glucose hourly
               -        when the blood glucose is below 15 mmol/l, change to 6 units IM every 2 hours.

Once the patient has recovered and is eating/drinking, change to S/C insulin.

Potassium (K^+) supplementation will be required-
There may be a high blood potassium initially, but this will fall as the sugar level comes down.
Measure the potassium hourly. Put 10 mmol K^+ in the first litre of saline then 10 - 40 mmol in subsequent litres of fluid, depending on the plasma level (normal 3.5 - 5.0 mmol/l).
If potassium measurements are unavailable then put 10 mmol KCl in each litre of fluid.

Other measures- 100 % O_2. Blood gas estimation-if pH < 7.10, give 50 mmol of 8.4% bicarbonate. Usually acidosis will correct itself slowly as the sugar comes down. Emergency surgery can start once the rehydration and lowering of blood sugar is underway.

Table 3	**Clinical signs of dehydration**

Sign	**Water loss (% body weight)**
Thirst	< 5 %
Dry mouth	
Capillary refill > 2 seconds *	5 - 10 %
Decreased skin turgor *	
Orthostatic hypotension *	
Low intraocular pressure (soft eyes)	
Reduced urine output	
Low CVP/JVP	
Shock	> 10 %
Unconscious	

* Capillary refill - lift limb above level of heart, press on skin for 5 seconds, release and observe colour returning to area. Normal is < 2 seconds. Skin turgor - pinch skin on back of hand and release. Normally the fold of skin quickly falls back flat but if dehydrated stays folded or returns slowly. Orthostatic hypotension - a severe fall in BP when patient stands up causing fainting.

THE APPLICATION OF BASIC SCIENCE TO PRACTICAL PAEDIATRIC ANAESTHESIA

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This paper will attempt to show how a sound knowledge of anatomy, physiology, pharmacology and psychology of infants and children and how they differ from adults helps to improve their care during anaesthesia and the perioperative period.

A baby can be taken from the parent without undue distress up to 6 to 7 months of age while an older infant or young child will become very distressed and hence they should be accompanied by a parent to induction of anaesthesia provided the parent doesn't exhibit undue anxiety. In such cases it may be better to give the child some premedication. Older children can cope but many like to be accompanied by a parent. Sometimes children, particularly boys of 8 to10 years old, appear well adjusted when seen beforehand but show signs of extreme apprehension when they reach the induction room - this presents as almost invisible veins so that venepuncture is difficult. Again, premedication should be considered. Midazolam 0.2-0.3 mg/kg given orally 30 to 45 minutes before anaesthesia usually has a tranquilising effect. If a child is very distressed on arrival at the operating theatre 0.2mg/kg can be squirted into the nose. It may sting but it usually has an effect within about 10 minutes. In older children diazepam (0.3 mg/kg) or temazepam can be given an hour before induction. This may be accompanied by an analgesic such as paracetamol (30mg/kg orally). An apprehensive patient has an increased cardiac output, which is largely redistributed to muscle so that the injected or inhaled induction agent does not reach the target organ - the brain - unless substantially increased doses are given. The increased ventilation through crying does not necessarily speed induction - the increased uptake merely compensates for the drug which has been redistributed to muscle.

It is often said that children are like small adults. This is not so - for a start their proportions differ. Infants have a relatively large head and therefore brain. It must receive a greater proportion of cardiac output as a consequence. The surface area is larger and thus heat loss is increased when it is exposed, especially in neurosurgery.

The **surface area: body weight ratio is double** in infants compared to adults resulting in greater heat loss. **Oxygen consumption relative to body weight is also double** (6-7 mls/kg/min). This is another key to working out important differences because if they need double the volume of oxygen then they have to double the amount taken in and transported. This means the alveolar ventilation must be increased which is largely achieved by increasing respiratory rate. Cardiac output must also be doubled to carry the oxygen around the body - this is achieved by increasing heart rate as babies have a very limited ability to increase stroke volume. Thus heart rates of 120 - 160 are common. The increased work of doing this is minimized by having a lower vascular resistance so that babies systolic blood pressures are lower (70 - 80 mmHg).

The fixed stroke volume in infants is also important because anything that causes bradycardia such as hypoxia, deep halothane anaesthesia, or reflex bradycardia due to vagal stimulation, such as occurs during laryngoscopy, will result in a decrease in cardiac output. When combinations of these occur serious decreases in output can result.

Cardiac output can be assessed clinically with a stethoscope because heart sounds become softer as the output decreases. Normally blood flow into the ventricles or into the aorta and pulmonary artery causes expansion followed by an elastic recoil which slams the valves closed resulting in loud heart sounds. If the volume decreases the recoil is diminished and the resulting heart sounds become soft. When the cause is corrected, such as by giving blood or fluids in hypovolaemia, one can hear the sounds becoming louder. The stethoscope is thus a very useful and sensitive monitor with which it is also easy to differentiate between patient and equipment problems when a monitor such as an oximeter gives abnormal readings.

Ventilation is greatly influenced by the anatomical differences, especially the structure of the chest wall. The ribs in neonates are more horizontal limiting antero-posterior expansion of the chest and they lack the bucket handle movement of the middle ribs that allows lateral expansion of the thoracic cage in older patients. The consequence is that ventilation is much more dependent on diaphragmatic movement and hence anything that restricts it (abdominal distention or compression) will cause respiratory difficulties. This includes inflation of the stomach with gas which can occur during ventilation with a mask when too high a pressure is applied or the bag is squeezed too fast thereby forcing gas down the oesophagus as well as the trachea. In patients with oesophageal atresia stomach distention is more likely with positive pressure ventilation when there is a large fistula. This can been assessed beforehand with a lateral chest X ray which shows the air containing fistula. Beware if this is more than 2.5mm in diameter. Patients in the lithotomy position have their abdominal contents compressed forcing the diaphragm up and restricting ventilation.

Intubation technique is important because infants have a higher oxygen consumption (6-7ml/kg/minute compared to 3 in an adult). This results in there being a shorter time before hypoxia begins to develop when a paralysed baby is not being ventilated. There are anatomical differences in the airway which are relevant. The larynx is situated at a higher level relative to the vertebrae - C3 in the infant compared to C6 in the adult; the epiglottis is U shaped and relatively longer, the angle of the mandible is greater (120 degrees) and the trachea has an anterior inclination. In addition the relatively large head does not need to be on a pillow but needs to be stabilized. This can be done by slightly extending the neck, rolling the thenar eminence of the right hand on to the forehead to stabilize it, then opening the mouth with the index finger and inserting the laryngoscope with the left hand down the right hand side of the mouth so that the tongue is kept out of the way. If the laryngoscope is held between the thumb and index finger the little finger of the left hand can reach to press the larynx backwards thus bringing the larynx into view (figures 1 - 3). The tube can then be passed from the right corner of the mouth so that it does not obstruct the view of the larynx. The important anatomical points in relation to the tube are that the cricoid cartilage forms the narrowest part of the larnyx before puberty and because it is circular an uncuffed tube can be used until 10 -12 years of age. Another convenient point is that the nose accommodates the same size of tube as the larynx before puberty. Tracheal length is often quoted to be 4cms but Anneke Meursing showed that the mean length is 4.5cms in a 3 kg baby. The importance of tracheal length is to appreciate how far the tube can be passed without going into the bronchus. The problem is that there are occasional babies who have short tracheas. It is thus important always to check after intubation that both lungs are being ventilated.

Figure 1

Figure 2

Figure 3

Blood volume and haemoglobin are greater in newborns. The volume is about 80-85 ml.

Haemoglobin at full term is about 180-200gm/l decreasing to about 110gm/l at 3-6 months. This haemoglobin is predominantly fetal with alpha and gamma chains which enable it to take up oxygen at low tensions such as exist in the placenta but do not release it as readily to the tissues. Gradually it changes to adult haemoglobin (alpha and beta chains). As lowering $PaCO_2$ shifts the oxygen dissociation curve to the left, hyperventilation further reduces oxygen delivery to the tissues so that excessive hyperventilation should be avoided even by increasing dead space in the circuit - not shortening the endotracheal tube will help.

In neonates with a high haemoglobin, albumin rather than blood can be used for early transfusion, when needed. In premature infants haemoglobin tends to be low because most of the iron stores are laid down in the last three months of pregnancy.

Several factors should be considered in deciding that it is necessary to start a blood transfusion. The haemoglobin should be at a level above that which supplies minimal oxygen requirements for metabolism. In infants metabolic rate is higher, the haemoglobin level may be higher in full term babies but lower in prematures and between 3-6 months so that the tolerated blood loss will vary. A 20% loss is usually well tolerated provided fluids are given to maintain the circulating volume. At that point one might consider whether blood loss is likely to continue and, if haemoglobin was low to start with, then blood may be started. On the other hand, clinical signs such as a rising pulse, when apparently adequate fluids have been given or a bolus does not reduce the pulse in a patient who also looks pale, will usually suggest that it is time to start blood.

The total **body water** is about 80% of body weight at birth, gradually decreasing with age to 60-65 % in adults. Premature infants have relatively more, making fluid loss an even more critical problem to them. When neonates and infants become dehydrated they initially lose extracellular water. Because the extracellular space is relatively larger at this age (about 50% of body weight) the losses will be proportionately greater. The relatively smaller intracellular compartment then has less fluid to shift to the extracellular space when losses occur resulting in a much sicker infant than an adult might be in similar circumstances.

The other consequence of the relatively **large extracellular** space is that drugs predominantly distributed in the extracellular space will have to be given with a larger loading dose. Also, extracellular electrolytes such as chloride will have to be given in larger amounts to correct deficits which occur, for example in pyloric stenosis, because of the larger extracellular compartment.

Kidney function is immature at birth and although the various functions develop at different rates there is a rapid improvement in the first few weeks of life. The relevance is that fluids ,electrolytes and drugs excreted by the kidney are handled more slowly during the early days and weeks of life. Glomerular filtration is less, the cortical tubules which are important in sodium excretion are not fully developed, and the interstitial urea concentration in the loops of Henle is low (because the amino acids are being utilized to build cells) and hence water reabsorption is reduced.

The **brain** is immature. Centrally acting drugs such as morphine and barbiturates have a greater depressant effect and thus have to be used in reduced doses, if at all.

The **temperature** regulating centres are also immature so that body temperature control is less efficient. This problem is aggravated in neonates because they have a relatively large surface area (2-2.5 times relative to weight), thin skin and subcutaneous fat so that they are poorly insulated and their body mass is less so that the body stores less heat. Neonates do not shiver so that they cannot respond to a cold environment. During anaesthesia the temperature control mechanisms are depressed so that methods of maintaining body heat must be instituted. These include overhead heaters, warming blankets, warming inspired gases and fluids and covering parts of the body not being operated upon.

Other **anatomical points** are important in regional and local anaesthesia. The spinal cord and dura mater reach lower levels in neonates (L3 and S3), the iliac crests are not fully developed so that the line between them is one vertebral space lower. Fascia and aponeurosis are thinner and therefore not as easily detected when used as depth markers during nerve blocks. They can be located more easily moving the needle up and down until a scratching sensation is felt or by angling the needle so that the traverse through the layer is thicker.

An understanding of the basic sciences is helpful in optimally managing our smallest patients during anaesthesia. In the next section anaesthesia for some common operations will be considered highlighting the application aspects of basic sciences to the clinical management.

Inguinal hernia repair is a common operation in young children, especially ex-premature infants. In the latter patients the abdominal wall is weak and the normal obliteration of the sac has not occurred. The infant born prematurely has deficient iron and glycogen stores because these are laid down mainly in the last three months of pregnancy. Thus they tend to be anaemic and susceptible to hypoglycaemia unless glucose is administered. In addition, the factors which increase heat loss are exaggerated so that particular care is necessary to maintain body temperature.

There are several options for anaesthesia. **General anaesthesia** for hernia repair in infants can be used if there is no history of apnoea. Even if there is this complication can be largely avoided postoperatively if the patient is ventilated with air instead of nitrous oxide and PEEP of 2-3cm water is applied.

The use of air prevents denitrogenation of the lungs and, together with PEEP, prevents atelectasis which results in increased work of breathing and fatigue in ex-prematures and is a major cause of postoperative hypoxaemia in many patients. The anaesthetic consists of muscle relaxation, ventilation with an inhalation agent and preferably a local anaesthetic block rather than opioids so that respiratory depression is avoided.

In prematures **spinal** analgesia in advocated by some anaesthetists because there are fewer respiratory problems if they are immobilised rather than being anaesthetised. The fact that the iliac crests are level with one intervertebral space lower is fortuitous as the spinal cord also ends one space lower. A 25 needle is often used to administer bupivacaine 0.5%. An alternative is to use **caudal** anaesthesia aiming to reach at least T10.

The **ilioinguinal block** as originally described involved placing local anaesthetic under the external oblique aponeurosis thus blocking the ilioinguinal and iliohypogastric nerves as they approach the skin. This provides adequate surface anaesthesia but does not anaesthetise the area around the inguinal sac. This can be achieved by placing local anaesthetic in the layer between the internal oblique and transversus abdominis muscles. *If a short bevelled needle is available it makes it easier to feel the loss of resistance as the aponeurosis is penetrated* 1-2 cm medial to the anterior superior iliac spine depending on the size of the patient. If one is not available the aponeurosis can be located by moving the needle horizontally as it is gradually advanced until a grating or rough sensation is felt. The needle is then advanced through the aponeurosis and a pop may be felt especially with a short beveled needle. 0.25 ml/kg of 0.25% bupivacaine can be injected to produce surface analgesia. The needle is then advanced slowly with gentle pressure on the plunger of the syringe. *It is difficult to inject into*

muscle but as soon as the needle emerges into the space between them it becomes easy to inject and a similar volume should be injected as above.

Circumcision is usually performed under light general anaesthesia with a local anaesthetic block. The reason is that under halothane alone laryngeal spasm often occurs. An understanding of the anatomy is important. **Caudal** anaesthesia is commonly used. The key points in locating the caudal canal are to feel the sacral cornua and then pull the skin cephalad (upwards) until it is just above the apex of the sacral hiatus. The needle can then be inserted just distal to the finger tip so that it passes through skin which has not been touched since being prepared with antiseptic. At this point the sacrococcygeal membrane is thickest and so more easily felt as the needle is inserted. It also enters the deepest part of the sacral epidural space and so it is not necessary to angle the needle into the canal although many people do this to ensure that they are in the correct space. 0.5 ml/kg of 0.25% bupivacaine will provide an adequate block.

The alternative is to perform a **dorsal nerve** of penis block. The skin is put on a stretch and the needle is inserted in the midline below the symphysis pubis. It is safer to angle it 10 degrees from the entry point and to advance and make injections on both sides of plain bupivacaine 0.5% 1ml + 0.1ml /kg. The needle has to penetrate the superficial fascia which can be felt with a short beveled needle or by scratching up and down as the needle is advanced until a rough sensation is felt. The fascia divides to form the suspensory ligament of the penis in the midline. This divides over the body of the penis but the nerves and blood vessels lie in the midline deep to it. It is to avoid puncturing these vessels that it is recommended to inject at an angle. As the needle is advanced under the symphysis gentle pressure on the syringe plunger will be met at first by resistance. When it becomes easy to inject the needle tip has entered a potential space which is pear shaped when filled and lies close to the nerves. Injection should be made here where it is easy to inject. The local anaesthetic diffuses easily through the fascial layer separating it from the nerves and vessels. It is important to fill this space between the symphysis and the corpora cavernosa so that the dorsal nerve and its ventral branch are both blocked as they come forward under the symphysis. The volume suggested usually achieves this.

There are some important **consequences of hypovolaemia** which may occur after trauma, burns, or as a result of post-operative bleeding such as occasionally happens after tonsillectomy. The redistribution of cardiac output, as well as its reduction has important consequences when anaesthesia is induced or analgesia is given. Students and young doctors must have the principles instilled into them because the consequences of not knowing that a greater proportion of the cardiac output goes to the brain and heart in hypovolaemia may result in dangerous myocardial or respiratory depression if depressant drugs are injected in usual doses. In addition, it is important to know that analgesics injected intramuscularly will not be effective until muscle perfusion is improved after correction of hypovolaemia .

An understanding of the application of basic sciences is important in the provision of high quality anaesthesia and care of children undergoing surgery.

VOLATILE ANAESTHETIC AGENTS

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One of the prominent features of anaesthetic practice in developing countries is the widespread use of volatile anaesthetic agents. This is surprising, as they are relatively expensive. Even modest supplies of halothane, for example, can cost several times more than the salary of the person using it but despite this burden on limited budgets, in most government hospitals cases are done using general anaesthesia with halothane and no other drug. However, many mission hospitals favour spinal anaesthesia for reasons of cost.

The demise of inhalation anaesthesia is sometimes predicted, partly because of cost and partly because of pollution of the atmosphere. Total intravenous anaesthesia may one day replace it. This event is probably far away and volatile agents will remain a central part of anaesthesia practice for many years to come.

An important safety feature of all volatile agents is that most of what goes into the patient via the lungs should come out the same way. Therefore the anaesthetic effect is reversible, as long as the patient is breathing. Also, with spontaneous breathing, the patient adjusts his or her own "dose " and respiratory depression will reduce the amount of vapour taken up and help prevent overdose. With controlled ventilation it is very easy to give an overdose.

A typical general anaesthetic (GA) using halothane or ether and nothing else is "bumpy", often unpleasant for the patient during induction and recovery, but reasonably safe.

The cost of some of the newer agents is very great and they are not generally used in developing countries. The cheaper, older agents, like ether, though widely used in poorer countries, are hardly ever used in the west. Most anaesthetists in the western world today have never given ether anaesthesia.

How do volatile agents work?

An agent breathed into the lungs will dissolve first in the blood and then be carried to all parts of the body and dissolve in the tissues. The agent that dissolves in the brain produces the state of anaesthesia. The brain, being mostly fat, absorbs a lot of the agent. Many theories have been considered to explain how anaesthesia is produced. One suggests that the fat in the cell wall swells up. This reduces the ability of the nerves to conduct impulses to each other and activity is reduced, or stopped altogether if you give an overdose. Fortunately, the higher centres controlling consciousness are the first affected and the vital centres such as the respiratory and vasomotor centres are more resistant to this effect. Thus we take it almost for granted that the anaesthetised patient will go on breathing with a near-normal pulse and blood pressure.

There are four broad physical properties of any agent that will tell the anaesthetist how it behaves in and out of the body and, therefore, how to use it to best advantage.

1. Solubility and Uptake. The blood solubility of an agent is related to its blood-gas partition coefficient. The partition coefficient is a simple ratio of amounts: eg. the blood/gas coefficient is the ratio of the amount dissolved in blood to the amount in the same volume of gas in contact with that blood. The more blood-soluble the agent (high blood-gas partition coefficient), the slower the onset of effect and the slower the patient goes to sleep. Thus a very soluble agent eg. ether will dissolve in large quantities in blood before the brain levels can rise sufficiently to produce anaesthesia. To understand this concept, think of the circulating blood volume as a large pool, soaking up agent and not allowing the brain to have any.

An anaesthetic agent does not "target" the brain: it dissolves in all tissues according to the tissue/gas partition coefficient for the particular agent in a tissue type. The blood flow to that tissue and the mass of tissue present will also determine the amount of agent reaching it and accumulating there. Fat stores, like the brain, have a very high affinity for anaesthetic agents. Luckily for the induction of anaesthesia, body fat has a very poor blood flow and during a short or medium length operation, only a limited amount of agent will have dissolved there.

Similarly, a high cardiac output such as may be found in fever or fear will cause more agent to be dissolved in blood and tissues other than brain, thus delaying the onset of CNS effects. In all these instances, there is said to be a high **uptake** of the agent into the body, i.e. the venous blood returning to the heart has a low concentration of the agent and there is room for lots more. Paradoxically, though a **high uptake** means a lot of agent is disappearing into the body, blood levels rise slowly and the patient takes a **long time** to go to sleep by inhalation.

High uptake will also mean slow recovery because during the process of induction and maintenance, a large reservoir of the agent will have accumulated in blood, fat and other tissues like muscle. At the end of a long operation, this reservoir will slowly give up its stores of anaesthetic agent and thus act like a depot, delaying recovery. As ether is very blood soluble, it leaves the blood slowly and therefore circulates for a long time, before it is finally excreted out from the lungs. Blood levels fall slowly delaying a return to consciousness. Halothane, being fat soluble, also remains for hours in the fat of an obese patient at sub-anaesthetic levels, slowly being washed out long after the operation is over. But, the blood solubility is lower than ether and therefore blood levels fall more quickly. Thus the level in the brain falls more quickly, as the blood is able to "wash" the agent out. The patient therefore recovers consciousness more quickly than when ether has been used. Tissue blood flow and cardiac output are important determinants in the elimination of highly soluble agents.

The opposite happens in shock, with a low cardiac output: in this case blood levels rise quickly, induction is fast and uptake is low.

It can now be understood what happens when an agent with a very low blood solubility is used (low blood/gas partition coefficient). Blood levels rise very rapidly, leading to a rapid induction of anaesthesia. When the agent is stopped the reverse happens: blood levels fall very quickly and recovery occurs after a short interval, no matter how long the agent has been used. Changes in cardiac output have little effect on the speed of induction of anaesthesia. The gas, nitrous oxide and newer agents, sevoflurane and desflurane are examples of very insoluble drugs.

2. Volatility. An agent with a low boiling point will evaporate easily and therefore be more available than one that has a high boiling point. Ether is highly volatile and thus there is almost no limit to the concentration that a vaporiser can give. Ether is really too volatile to be convenient and sometimes new, sealed bottles arrive with no agent inside, but at least it means we can give plenty of it to counteract its slow onset. Trichloroethylene, on the other hand, only reluctantly becomes a vapour and we have difficulty in getting it into the patient in sufficient quantities. Halothane is in between and has a near-perfect profile of physical properties.

Another index of volatility is the Saturated Vapour Pressure or SVP. It indicates the maximum proportion of atmospheric pressure which can be occupied by the vapour of an agent. Ether has an SVP of 425 mmHg and theoretically will allow a maximum concentration of 56% (425/760 x 100). SVP is dependant only on the temperature and not on atmospheric pressure.

3. Potency. Regardless of solubility and boiling point, each agent will have its own potency value. This is called the MAC - the Minimum Alveolar Concentration. This is the concentration at equilibrium required to prevent a reflex response to a skin incision in 50% of patients. Thus the potency of different agents can be compared by showing how much you need to produce the effect you want, expressed as a percentage vapour strength.

An agent with a low MAC, is a potent agent because only a small amount is required to produce anaesthesia. A high MAC means the agent is weak because a lot of agent is required to produce anaesthesia. Ether has a high MAC, is a weak agent, while trichloroethylene has a very low MAC, is potent and produces its effects at a fraction of the concentration of that needed for ether. Once again, halothane has the ideal MAC, somewhere in between. If the agent is being used alone with spontaneous breathing in a fit patient, you will need to set your vaporiser to at least three times the MAC to keep the average patient settled during surgery.

The MAC of any agent is broadly determined by its fat solubility: the more fat soluble, the greater the potency.

4. Pharmacological effects. Although we say that ether is weak, it is difficult to believe this statement if you see a patient totally unrousable after ether anaesthesia, a common occurrence. To explain this, one has to think of the different ways an agent works: the anaesthetic effect, the analgesic effect. the volatility and correlate these with the properties outlined above. Ether is very volatile, has good anaesthetic and analgesic effects and these, with the large reservoir effect and slow recovery, make it an effective anaesthetic, despite its low potency.

Halothane is a good anaesthetic, but a poor analgesic. Thus the combination of low solubility, a small blood reservoir and postoperative pain causes the patient to wake up quickly.

Trichloroethylene is a good analgesic but the patient breathing this alone will never get to the state of anaesthesia at all unless he is given the agent for several hours because it does not evaporate enough to give a sufficient inspiratory concentration and is rather blood soluble.

The side effects of the individual agents are mentioned in more detail below. All volatile agents trigger Malignant Hyperthermia.

What agents are available?

The inhalation agents that are commonly used in Africa and other places where resources are limited are ether and halothane. When it is available, trichloroethylene is also used.

In the West halothane has been displaced by newer agents: isoflurane and sevoflurane. (Halothane is still widely used in paediatric anaesthesia.) These are far more costly than halothane and will not be considered in detail, though if you get the chance to use isoflurane you will be impressed how good the recovery is compared to halothane. Ether, of course, is never used in the western world and trichloroethylene has a diminishing number of users world-wide and is hard to get. Laboratory grade is still available.

The individual agents that are used all over Africa and elsewhere will be described now in detail:

ETHER (diethyl ether)

This is a very cheap agent as it is non-halogenated, made from sugar cane via ethanol using recycled sulphuric acid. With suitable fire precautions, it could easily be made locally in any country with the will to be self-sufficient. W.T.G.Morton demonstrated its effects on a famous occasion in Boston, USA, in 1846 and this event has become recognised world-wide as the "first anaesthetic".

Physical properties: Low boiling point: 35 deg C. High SVP at 20 deg C : 425 mm Hg. Blood/Gas partition coefficient: 12 (high), MAC: 1.92% (low potency). Cost: from US$10/litre, according to supplier. Ether is highly volatile and inflammable. In oxygen, it is explosive. It has a very strong and characteristic smell.

Advantages: stimulates respiration and cardiac output, maintaining blood pressure and causes bronchodilatation, all due to its sympathomimetic effect mediated by

Table 1	**Volatile agents and their physical properties.**			
AGENT	**SVP in mmHg**	**MAC**	**Blood/Gas coefficient**	**Comments**
Ether	425	1.92%	12	High volatility Low potency High solubility
Trichloroethylene	60	0.17%	9	Low volatility High potency High solubility
Halothane	243	0.75%	2.3	Volatile Low potency Low solubility
Sevoflurane	160	1.7 - 2%	0.6	Volatile Low potency Insoluble
Isoflurane	250	1.15%	1.4	Volatile Potent Low solubility

adrenaline release. A good sole anaesthetic agent because of its analgesic effect. Does not relax the uterus like halothane but gives good abdominal relaxation. A safe agent.

Disadvantages: flammable, slow onset, slow recovery, secretions +++ needing atropine. Bronchial irritation, so inhalation induction of anaesthesia by mask is very difficult because of coughing. PONV (postoperative nausea and vomiting) is sometimes seen in Africa but is a major disadvantage in the West, where patients vomit much more.

Indications: Any general anaesthetic, but especially good for Caesarean section (because the baby tolerates it and the uterus contracts well), and major cases with intubation. It is life saving for poor risk cases using a low dose. Also indicated when no supplementary oxygen is available.

Contra-indications: There are no absolute contra-indications for ether.

Scavenging should be carried out (where possible) to avoid contact between heavy inflammable ether vapour and diathermy apparatus or other electrical devices that may spark and also to prevent exhaled vapour blowing at the surgeon.

Practice points: The best method is to give a high concentration to a paralysed, intubated patient. Thus after atropine, thiopentone, suxamethonium and intubation, generous IPPV is commenced with ether 15-20% and then according to the patient's needs, the ether is reduced after about 5 minutes to 6-8%. Remember vaporiser performance is variable. Poor risk, septic or shocked patients may need only 2%. Switch off well before the end of the operation to avoid a prolonged recovery. With skill you can have your patients almost awake as they move off the table. If you have a big strong man for a hernia repair, save yourself a lot of embarrassment and give him a spinal instead!

It seems to be purely fortuitous, but the patients that benefit most from ether anaesthesia, such as Caesarean section and emergency laparotomy (which comprise over 90% of all major surgery in Africa[2]) do not need diathermy. Where diathermy is essential, eg. in paediatric surgery, halothane is a better drug, so the conflict between ether and diathermy rarely arises. **At our hospital, we do not allow ether to be used with diathermy.**

HALOTHANE ("Fluothane")

Physical properties: Boils at 50°C, SVP at 20°C: 243mmHg. Blood /Gas partition coefficient:2.3, MAC 0.75%. Cost: US$ 140/litre.

Advantages: Well tolerated, non-irritant, potent (low MAC) agent, which is relatively insoluble in blood, giving rapid induction, low dose maintenance and rapid recovery. There is predictable, dose-related depression of the respiratory and cardiovascular systems. The ideal inhalation induction agent.

Disadvantages: Perhaps too potent, and overdose is easy. Poor analgesic properties necessitating deep planes of anaesthesia before surgery and especially intubation can be tolerated. No post-operative analgesia. Uterine relaxation and haemorrhage at concentrations above 2%. Hypotension, dysrhythmias and especially dangerous with adrenaline where cardiac arrest in VF readily occurs. Post-operative shivering. "Halothane hepatitis" may very rarely occur (I have never seen a case in Africa). It is extensively metabolised in the body and is best avoided within three months of a previous halothane anaesthetic unless the indications to use halothane are considered to override the risk of this rare condition.

Indications: almost all general anaesthesia, especially paediatrics. Inhalation induction especially in upper airway obstruction.

Contra-indications: simultaneous administration with adrenaline, especially during spontaneous breathing. High dose for Caesarean section or uterine evacuation. History of unexplained hepatitis following a previous anaesthetic.

Dosage: Induction with 3%, reducing to 1.5% for maintenance. Children need 2% for maintenance. Over 4% for more than a few minutes will produce an overdose.

Practice Points: Halothane alone is not ideal because it has no analgesic properties. You need high concentrations to abolish reflex activity, eg. straining on the endotracheal tube. This becomes expensive and may also be unsafe. The common clinical situation of an intubated patient breathing spontaneously high concentrations of halothane in oxygen and air is potentially hazardous in the presence of heart disease. Many anaesthetists get away with it in ignorance, but only because heart disease is uncommon in Africa.

A common arrangement is to have two draw-over vaporisers in series containing halothane and trichloroethylene. Where available, nitrous oxide is commonly used for analgesia; opioids or regional blocks are alternatives.

Supplementary oxygen is mandatory when using halothane to avoid hypoxia.

TRICHLOROETHYLENE ("Trilene")

Physical properties: Boils at 87°C (high), SVP at 20°C: 60 mmHg. Blood/Gas partition coefficient: 9 (high), MAC 0.17%

Advantages: Non-irritant. Safe. Good analgesia during and after surgery. Cardiovascularly stable. Very cheap, because one uses so little.

Disadvantages: Low volatility, slow onset of effect because of high blood solubility and low boiling point making it impossible to get concentrations that are high enough. It is a potent agent because you need little to produce an effect, BUT it is a weak anaesthetic because, despite this, vaporisers cannot produce high enough concentrations because the volatility is so low. Tachypnoea used to be reported in the West but we don't see it in Malawi. Dysrhythmias may occur with adrenaline. Prolonged recovery, because of high blood solubility.

Indications: analgesic supplement to halothane or used on its own for minor procedures such as fracture manipulation, debridement etc.

Contraindications: overdosage in the elderly. Closed circuit with soda-lime. Best avoided in very small babies.

Dosage: 0.5 - 1% initially, reducing to 0.2 - 0.5%.

Practice Points: Switch off 20-30 minutes before the end of a long operation to avoid prolonged sedative effects. Its ideal function is to give background analgesia for long cases using halothane as the main anaesthetic but it is also very good given with halothane for a fast turn-over of short cases using inhalation induction. We give it from a Goldman halothane vaporiser in series with a halothane vaporiser, using a gauze in the bowl to increase evaporation. This gives about 0.7%.

The newer agents:

Enflurane: was a replacement for halothane, now used infrequently.

Isoflurane: Boils at 48°C. SVP at 20°C: 250mmHg, Blood/Gas partition coefficient: 1.4, MAC: 1.15. In general use, good recovery because of relatively low blood solubility, but induction difficult because of irritating bad smell, minimal metabolism, no arrhythmias but causes hypotension, six times the cost of halothane. Big cost reductions when used in a low flow system.

Desflurane: Boils at 23.5°C, SVP at 20 °C: 673 mmHg, Blood/Gas partition coefficient 0.4 (low), MAC: 5-10%. Replacement for enflurane, requires a specially designed vaporiser, has come and gone without me ever seeing it!

Sevoflurane: Boils at 58.5°C, SVP at 20°C: 160 mmHg, Blood/Gas partition coefficient 0.6 (low), MAC: 1.7-2%. Fabulously expensive ($1000/litre), but costs can be reduced if used in a low flow system. There may be problems with sevoflurane and carbon dioxide absorbers, baralyme in particular, but these are currently being investigated. Ultra low solubility resulting in ultra rapid induction and recovery especially as it is non-irritant and sweet smelling. High volatility and high percentage required.

How should volatile agents be used?

One way is to use them for inhalation induction of anaesthesia followed by maintenance with the same or another agent as your sole anaesthetic. The patient puts him or herself to sleep by breathing via a close-fitting mask and provided the smell is accepted and the stage II excitement effects are not excessive, this is a very satisfactory method of inducing general anaesthesia for minor cases without gastric aspiration risk. Lung disease, smoking or drinking habit, obesity and high uptake situations (see above) will make this method slower and prolong stage II effects. Loss of airway in an obese patient may be dangerous. Ideal for a fast turn-over of lots of short procedures on thin patients.

The other way is to put up a drip and give an intravenous induction followed by the volatile agent for maintenance of anaesthesia. Very often the intravenous induction will include intubation of the trachea as well. All general anaesthesia for major cases will be done this way.

Further reading

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EXTRACTS FROM THE JOURNALS

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Pulmonary Artery Catheterization.

Over the past decade, there has been vigorous debate concerning the indication for, and clinical utility of, the Pulmonary Artery Catheter (PAC). Recently, Connors et al.(1) reported an observational study of PAC use conducted in five teaching hospitals in the United States between 1989 and 1994. In this study, the PAC was associated with both increased mortality and utilisation of resources when compared with case-matched control patients who did not undergo pulmonary artery catheterisation. Despite the paper's shortcomings (not prospective, nor randomised, nor a controlled trial) it served as a catalyst to again intensify heated debate over the use of the PAC in the critically ill patient. Complications directly associated with the catheter itself does not seem to cause the problems but incorrectly collected haemodynamic data may lead to improper therapeutic strategies. Another factor is the documented significant interobserver variability in interpretation of pulmonary artery pressure tracings. Modification of care that PACs seem to provoke, like increasing the use of vasopressors, inotropes and intensity of care, may, at times do more harm than good. Also, disturbing evidence exists suggesting that knowledge of basic principles of pulmonary artery catheterization by physicians and nurses engaged in routine use of these devices is suboptimal.

The study of Connors et al. led to a consensus conference that recommended guidelines for the use of the PAC (2). Some of the final recommendations, which reflect the collective opinion of the participants, are:

- Clinicians should continue to carefully weigh the risks and benefits of the PAC.

- Clinician knowledge about the use of the PAC and its complications should be improved.

- The indications and contraindications for PAC use, where clinical evidence is lacking, should be determined.

To summarise, most clinicians believe that the PAC is useful in guiding intravascular volume expansion and pharmacological intervention in selected critically ill patients, however finding clear evidence to substantiate this belief is difficult, despite 25 years of PAC use!

1. Connors Jr AF, Speroff T, Dawson NV, Thomas C, Harrell Jr FE, Wagner D, et al. The effectiveness of right heart catheterization in the initial care of critically ill patients. JAMA 1996;276:889-897

2. Pulmonary Artery Catheter Consensus Conference Participants. Pulmonary artery catheter consensus conference: consensus statement. Crit Care Med 1997;25:910-925

Management of Dural Puncture

Unintended dural punctures continue to occur during the attempted insertion of epidural needles. Berger et al. (1) reported in a survey of 46 North American tertiary care obstetric centres on the management of dural punctures occurring with epidural analgesia during labour. The incidence of inadvertant dural puncture was 0.4%-6%. Following accidental dural puncture, 86% of patients experienced headache, in 63% of these patients it was severe.

Resiting the epidural catheter at another level was the most common initial step (90%) after dural puncture. If another epidural catheter was successfully placed, most centres used their standard top up or infusion regimes. Some centres (mainly in the USA) considered continuous intrathecal catheters as an alternative if the epidural catheter proved difficult to place.

Following delivery 86% of centres allowed unrestricted mobilisation. If headache occurred lying in bed was found to be useful for pain relief.

Enhanced hydration, either orally or intravenously, to increase CSF production, has not been shown to decrease the risk of headache, but may lessen its severity. The beneficial effects of caffeine are transient in many patients. 17% of centres employed epidural saline boluses or infusions.

Prophylactic epidural blood patch (EBP) was recommended by 37% of centres, with twice as many US as Canadian centres doing so. The EBP is still the most efficacious treatment for a post dural puncture headache with a reported success rate of greater than 90%. The resolution of symptoms are thought to be caused by an increase in CSF pressure from the injection of an epidural blood volume and formation of a clot at the site of the dural hole that seals and prevents further CSF leakage.

1 Berger CW, Crosby ET, Groecki W. North American survey of the management of dural puncture occurring during labour epidural analgesia. Can J Anaesth 1998; 45: 110-114

SELF ASSESSMENT SECTION

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1.   In significant aortic stenosis:
A   there is an early diastolic murmur
B   the ECG often shows evidence of left ventricular hypertrophy
C   can be caused by rheumatic fever
D   collapse may be the first manifestation
E   cannot be present if the patient is hypertensive

2.   With aortic stenosis:
A   a pulse rate of 40 per minute is good for haemodynamics
B   ketamine is a useful anaesthetic agent
C   antibiotic prophylaxis is necessary for surgical procedures on the bladder
D   neuromuscular relaxation is contraindicated
E   hydralazine is a safe drug to use

3.   In cardiac tamponade there may be:
A   hypotension
B   distended neck veins
C   Kussmaul's sign
D   pulsus paradoxus
E   cardiac arrest

4.   Concerning cardiac tamponade:
A   may be caused by penetrating trauma
B   may be secondary to a pericardial effusion
C   general anaesthesia is required to drain the pericardium
D   can follow blunt trauma
E   ECG complexes may be small

5.   Constrictive pericarditis:
A   may be caused by TB
B   causes abdominal swelling
C   may lead to hepatomegaly
D   peripheral oedema may be minimal
E   may cause peritonitis

6.   Rheumatic fever:
A   affects only the heart valves
B   occurs after streptococcal infection
C   may cause a large joint polyarthritis
D   does not affect the mitral valve

E   may cause elevated ST segments on the ECG

7.   Elevated blood urea may occur:
A   in renal failure
B   after bleeding into the gut
C   in dehydration
D   in liver failure
E   in overhydration

8.   Concerning tuberculosis:
A   miliary TB may be associated with a negative mantoux test
B   the X-ray of miliary TB may mimick stapylococcal pneumonia
C   primary TB can cause hilar adenopathy on chest X-ray
D   the primary complex causes upper lobe cavitation
E   may present in conjunction with malnutrition

9.   The Apgar score:
A   should be measured at 0 and 3 minutes
B   has a maximum score of 8
C   includes assessment of respiration
D   includes assessment of colour
E   each measure is scored 0-1

10.   The GCS (Glasgow Coma Scale):
A   has a minimum score of 0
B   scores pupil size
C   a confused patient would score 13
D   if reduced in presence of a skull fracture is a worrying sign
E   if 15 in presence of a skull fracture is reassuring

11.   After head injury:
A   a lucid interval may occur with an extradural haematoma
B   craniotomy will take preference over surgery for abdominal bleeding
C   depressed skull fracture always requires operative repair
D   wound toilet may be safely accomplished under local anaesthetic
E   hypertension and bradycardia may occur with

raised intracranial pressure

E   low blood pressure and slow pulse

12. *Features of base of skull fracture include:*
A   Battle's sign
B   "raccoon" or "panda" eyes
C   a haemotympanum (blood behind the ear drum)
D   normal GCS
E   lowered GCS

13. *After head injury treatment of raised ICP (intracranial) pressure can include:*
A   1000ml of 20% mannitol
B   20mg of frusemide
C   intravenous colloid or 0.9% saline solution
D   maintenance of nutrition with 5% dextrose solution
E   positioning the patient in slight head up posture

14. *After head injury treatment of raised ICP (intracranial pressure) can include:*
A   surgical decompression of space occupying lesion
B   slight hyperventilation
C   applying atropine to the conjunctiva in order to see optic discs
D   using tight bandages round the neck to secure an endotracheal tube
E   ketamine infusion

15. *Instruments can be sterilised:*
A   by thorough cleaning in hot soapy water
B   by immersion in chlorhexidine for one hour
C   by boiling in water for 20 minutes
D   by autoclaving even if they are still dirty
E   by autoclaving if they have been cleaned thoroughly beforehand

16. *Spinal injury may be associated with:*
A   flaccid paralysis
B   priapism
C   urinary retention
D   loss of anal tone

17. *After blunt thoracic trauma:*
A   fractured ribs 9-12 may be associated with liver or spleen injury
B   myocardial injury may occur in association with a fractured manubrium
C   pulmonary contusion may be complicated with ARDS
D   flail segment can be diagnosed clinically
E   consider thoracotomy if there is > 1500ml blood loss from chest drain

18. *Concerning failed intubation:*
A   never remove cricoid pressure if the stomach is full
B   if you cannot intubate or ventilate a surgical airway is indicated
C   don't worry how long it takes- try, try, try again until you are successful at intubating
D   the safest plan is to wake the patient up before using an alternative approach
E   repositioning the patient's head and neck may be useful manoevers

19. *Low serum potassium (hypokalaemia):*
A   may occur in kwashiorkor
B   can occur after high dose salbutamol
C   may cause cardiac arrhythmias
D   reduces digoxin toxicity
E   may cause ileus

20. *High serum potassium (hyperkalaemia):*
A   may cause cardiac arrest
B   should be treated with 50mls 50% dextrose plus 50u of soluble insulin
C   can be treated with sodium bicarbonate
D   may be caused by rhabdomyolysis
E   can occur with spironolactone

TECHNIQUES FROM AROUND THE WORLD

This is a new section of Update in Anaesthesia in which anaesthetists from different countries and different hospitals explain how they anaesthetise for certain types of surgery. Techniques vary widely from one facility to the next and we hope to illustrate the different methods of anaesthesia which are used. In this edition, anaesthetists from South Africa, India and Indonesia describe how they would anaesthetise a previously fit patient for bowel resection.

Anaesthesia for a patient scheduled for an elective bowel resection

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The anaesthetic course would vary depending on the type of surgery to be undertaken. This patient is scheduled for a simple laparotomy for a bowel resection, and not a major procedure such as an anterior- posterior resection.

Preoperative preparation and investigations: Routine preoperative investigations include a finger prick haemoglobin. Any further investigations would depend on the case scenario and the patient's pre-morbid state.

Premedication: Temazepam 10-20 mg depending on the patients weight. DVT prophylaxis (for prolonged procedures) with 5000 units of heparin administered subcutaneously.

Pre-induction: Venous access is established by placing a large bore intravenous catheter and the administration of modified Ringers lactate

Induction: In the absence of any indication for a rapid sequence induction, anaesthesia would be induced with 3-4mg/kg of thiopentone and 0.1mg/kg of vecuronium. The patient is then manually ventilated via a facemask with 50% oxygen in air and 1.5% halothane for 3 minutes after which an oral endotracheal tube is inserted. A rapid sequence induction would be performed if there was any risk of reflux and aspiration.

Maintenance: The patient is ventilated with an oxygen/ air/ halothane mixture. If the patient had received halothane within the last 6 months, isoflurane would be used instead. A circle system is used with a total flow of about 1 litre / minute.

A nasogastric tube would be inserted. Intermittent boluses of vecuronium to maintain surgical relaxation. Analgesia would be provided by 10-15mg of morphine intravenously.

Monitoring: 3 lead ECG, non-invasive blood pressure at 3 minute intervals, pulse oximetry, capnography, urinary catheter and peripheral nerve stimulator. Nasopharyngeal temperature probe is used with prolonged surgery. An internal jugular CVP line would be placed if large volumes of fluid shifts were anticipated.

Fluids: Modified Ringers lactate at approximately 6-8 mls/kg/hr. Additional colloids and crystalloids administered as required to replace fluid and blood loss and for third space losses.

Other measures: Temperature is maintained with forced air warming blanket for a prolonged procedure. Dynamic calf compressors are used to prevent DVT. Antibiotics would be given i.v. in theatre - benzyl penicillin 2 million units, gentamicin 6mg/kg and metronidazole 500mg.

End of anaesthesia: Discontinuation of volatile agent. Reverse muscle relaxation with 0.4mg glycopyrrolate and 2.5mg of neostigmine. Extubate patient and transfer to the recovery room, with 40% oxygen via a Venturi facemask.

Recovery Room*:* Patient nursed in the recovery position and given 40% oxygen by facemask. Monitor non-invasive blood pressure and pulse oximetry.

Pain Management: Further intravenous boluses of morphine as required to ensure adequate analgesia before transfer to ward.

Recovery discharge criteria for ward:

- Patient awake and able to cough
- Sustained head lift for 5 seconds
- Pain free
- Haemodynamically stable
- No nausea or vomiting
- Haemostasis as assessed via surgical dressing

Postoperative Instructions

Monitoring: Routine postoperative monitoring to include heart rate, respiratory rate, blood pressure every 15 minutes for 2 hours and then 4 hourly if patient stable.

Fluids: 5 % dextrose in 0.45% saline; 1000mls each 8hrs

Analgesia: Morphine infusion 20mg of morphine in 200mls of saline @ 10-15mls/hr titrated to effect

How would you anaesthetise a 50 year old previously healthy patient scheduled for elective laparotomy and bowel resection?

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Patients scheduled for bowel resection invariably have some degree of bowel obstruction and malnutrition. Causes include malignancy, inflammatory bowel disease or more rarely cases of amoebiasis or extra-lumen abscess constricting the bowel.

Preoperative evaluation includes physical examination, vital signs evaluation and laboratory evaluation of Hb, Hct, albumin, creatinine, K^+ , Na^+ and blood glucose whenever posssible. A chest X-ray will provide important data for the lung conditions, possible lung metastases and heart configuration. ECG recording is useful in identifying arrhythmias, coronary ischemia and hypertrophy.

Premedication is determined by the patient's psychological state as assessed at the preoperative visit. A dose of midazolam 2.5 - 5mg i.m. will help relax the patient; promethazine 1mg/kg i.m. or diazepam 0.2mg/kg i.m. are alternatives. This sedation applies for both general or regional anaesthesia.

When ether anaesthesia is planned, atropine 0.25mg i.m is given preoperatively followed by 0.25mg i.v. on induction to prevent hypersecretion of the salivary and bronchial glands. Opioid analgesia should be provided when the plan includes halothane which has a low analgesic property (e.g. pethidine 1mg/kg or morphine 0.1 mg/kg).

Anaesthesia: Some surgeons are capable of performing bowel resection very quickly. With such a surgeon epidural anesthesia can sometimes be used for lower abdominal operations. A continuous lumbar epidural with the catheter inserted at the lumbar 2-3 intervertebral space usually works well. Lignocaine 1.5% to 2.0% with 1:100,000 adrenaline is used to produce anaesthesia up to the sensory level of thoracic segment 4-6th. However regional anaesthesia is not safer for this type of surgery and general anaesthesia is usually preferred.

Induction is usually with thiopentone or ketamine and suxamethonium followed by tracheal intubation. This is followed by an inhalational agent (halothane or ether) administered with a non-depolarising muscle relaxant such as pancuronium and controlled ventilation. Although deep ether may be used with spontaneous or assisted ventilation (stage III plane 2 or 3), light ether anaesthesia (stage II or I) with pancuronium is preferred because the patient will recover very quickly.

Basic vital sign monitoring includes blood pressure, pulse rate, temperature (usually rectal). A precordial stethoscope and a finger on the pulse is compulsory. Ventilation is usually manual, but when a simple ventilator is used chest movement is observed continuously.

Intravenous fluids: Preoperative hydration is 1000 ml of Ringer dextrose or Ringer's lactate starting before bowel preparation and continued up to the time of induction. During surgery Ringer's lactate or NaCl 0.9% is given at 10ml/kg/hour via a 16G or 18G i.v.catheter placed in the arm.

Blood loss in excess of 15% - 20% of estimated blood volume is replaced with blood transfusion. In my institution we try to delay transfusion until the postoperative period if the circulation is stable. This allows the patient to complain of any adverse effect from the transfusion. When transfusion is delayed, Ringer's lactate 2-3 times the measured loss is given.

Postoperatively: At the end of the procedure the patient is extubated and supplemental oxygen is given for 4-6 hours postoperatively. The patient stays in the recovery room before being transferred to the ward. In many hospitals there is a new trend of keeping these patients in a high dependency care area so that the vital signs, fluid balance and pain management can be optimized.

Postoperative instructions include pain management, which is often oversimplified and not effective. Opioids are frequently in short supply and this form of analgesia may be impossible. Alternatively, i.v. NSAIDS are more readily available but more expensive.

Ringer's lactate and dextrose 5% 40-50 ml/kg/day is given postoperatively taking into account the high ambient temperature in the ward. As soon as possible gradual oral alimentation is started and normal diet is resumed around day 5 with most patients.

Malnutrition occurs commonly in developing countries and increases the risk of surgery considerably. Although parenteral nutrition has not been proved to be beneficial in these circumstances, we believe that giving some nutrition is better than none. The cost of dextrose 10% is exactly the same as dextrose 5%, and some brands of amino acid

preparations are reasonable compared to the risk of dehiscence (wound breakdown). Many centres therefore give peripheral parenteral nutrition using dextrose 10% plus amino acids, particularly to those patients who are unlikely to be able to eat for more than 7 days.

Management of a case of small bowel obstruction for resection in India

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The management in India would vary widely, from the limited facilities in remote regions to standard anaesthetic techniques in the cities and towns. However a patient requiring such intervention is likely to be taken 20 - 200kms to the nearest large town or city where the facilities available would be either a Government hospital or a private nursing home.

Preoperative preparation

Investigations: Hb, urea, creatinine and electrolytes whenever available, blood grouping and crossmatching if indicated. An ECG is usually performed over the age of 40 years. A chest X-ray is only performed if there is clinical evidence of cardiac or respiratory disease.

Preoperative fluid resuscitation is usually with Ringer's lactate and organised by the surgeons. A nasogastric tube is usually inserted on the ward. It is common for patients coming from remote areas to be taken for surgery with inadequate resuscitation and investigations.

Anaesthetic technique: Intravenous access is secured using a cannula or Butterfly needle. Premedication, when given, is usually i.v. pethidine (25-50mg), buprenorphine or pentazocine. Morphine is rarely available. Intravenous atropine 0.6mg and metoclopramide 10mg are given by some anaesthetists.

Induction is carried out in the operating theatre using thiopentone and suxamethonium. If an assistant is available cricoid pressure is applied and the patient intubated using a red rubber endotracheal tube.

The patient is maintained on a mixture of oxygen, nitrous oxide and halothane and relaxation continued using pancuronium. Further doses of 10mg pethidine are given as required. In poor risk patients diazepam or midazolam may be used in small doses to supplement induction. Occasionally spinal anaesthesia is used where general anaesthetic techniques are not available.

Reversal: The patient is given neostigmine and atropine (2.5mg+1.2mg) and the patient extubated on the table when there is adequate respiratory effort. Once the anaesthetist is satisfied with the ability of the patient to maintain their airway and respiration the patient is moved to the recovery area.

Recovery: The pulse, respiration and BP is recorded every 15 minutes (manually) by a trained nurse or operating department assistant. Before moving the patient to the ward the anaesthetist is contacted and approval obtained.

Post Operative Instructions

Observations: Record pulse, respiration, BP every 15 minutes for 2 hours, then every 1/2 hour or as required.

Analgesia is i.m. pethidine 50 mg 6 to 8 hourly. This may be combined with i.m. diclofenac 50mg bd. Buprenorphine is also used and occasionally morphine. Tramadol is becoming popular.

Intravenous fluids are given as advised by the surgeon and is usually of the order of 2000mL Ringer's lactate solution over the next 12 hours.

Note that postoperative instructions are usually written up by the surgeon as they supervise the postoperative care, and the anaesthetist is usually not involved. The incidence of deep vein thrombosis (DVT) appears to be low therefore prophylaxis is unusual.

ANSWERS TO ASSESSMENT SECTION ON PAGE 83

1.   FTTTF

2.   FTTFF
The murmur is systolic. Blood pressure depends on cardiac output and systemic vascular resistance (SVR). In aortic stenosis there is often a reduced cardiac output but high compensatory vasoconstriction. Depending on the balance between the two, there may be normotension, hypotension (as is classically described) or even hypertension. Vasodilation can cause potentially fatal downward spiral of blood pressure, so vasodilating drugs (such as hydralazine) are dangerous. A fixed stroke volume means cardiac output cannot be maintained at very slow heart rates. By increasing heart rate and systemic vascular resistance, ketamine may prove useful. Voltage criteria for left ventricular hypertrophy is often seen on the ECG because the heart has been pumping against an increased resistance (or "afterload").

3.   TTTTT
Kussmauls sign is the rise in JVP (jugular venous pulsation) on inspiration, and is associated with impaired heart filling as occurs in constrictive pericarditis or cardiac tamponade. Normally the neck veins collapse and JVP falls on insiration.

4.   TTFTT
 Fluid in the pericardium should be drained before anaesthesia as severe hypotension can occur at induction. A fast heart-rate and adequate preload maximize cardiac output in cardiac tamponade.

5.   TTTTF
Constrictive pericarditis can be caused by a number of conditions including TB. The clinical features are essentially those of right sided heart failure, including elevated JVP, massive liver enlargement and ascites. Dependent oedema is also usually a feature of right sided heart failure but is often absent, minimal or much less pronounced than the hepatomegaly and ascites in this particular condition.

6.   FTTFT
Rheumatic fever causes a pan-carditis and can cause pericarditis which may be heard as a rub or seen on the ECG as concave elevated ST segments

7.   TTTFF
Blood urea level reflects three things: i) production (it is produced by the liver as proteins are broken down). Thus levels are low in liver failure and high after a gastro-intestinal bleed (effectively a large protein load). ii) clearance from the body, which is done by the kidneys. iii) body water levels - so simplistically the urea will be raised in dehydration and diluted or lowered in over-hydration.

8.   TTTFT
Post-primary TB may cause cavitation but the primary complex does not.

9.   FFTTF
The Apgar score includes assessment of 5 parameters with a maximum score of 10

10.   FFFTT
Minimum GCS score is 3. Noting pupil size is crucial in the assessment of head injury, but is not part of the GCS score. A reduced GCS in association with a skull fracture means that there is a significant possibility (about 1 in 4) of an intracranial haematoma.

11.   TFFTT
When dealing with a head injured patient, you need to consider the whole patient. If they are at risk of bleeding to death from another injury, then this should be treated first. "Secondary insults" such as hypovolaemia, hypotension and hypoxaemia should be corrected aggressively.

12.   TTTTT
With fractured base of skull the fracture may be difficult or impossible to see on X-ray but can be diagnosed clinically in the presence of bruising over the mastoid (Battle's sign), blood behind the tympanic membrane (or at the external auditory meatus)or periorbital bruising (Raccoon/Panda eyes). The fracture of bone alerts us to the possibility of damage to the underlying brain which needs to be assessed clinically by GCS measurement and neurological examination.

13.   FTTFT
Intracranial pressure (ICP) can be reduced with a dose of 100ml of 20% mannitol but 1000ml in one dose is too much. Frusemide augments the effects of mannitol in

reducing intracranial pressure. Head injured patients nearly always require intravenous fluids (especially after mannitol which will dehydrate them), but dextrose will worsen cerebral swelling and should not be used. ICP can also be reduced by placing the patient slightly head up (about 20-30 degrees) and avoiding compression or obstruction of the neck veins which will worsen intracranial congestion. (Having said that it is important to secure the endotracheal tube well - try taping it in)

14.   TTFFF

Noting pupil size is crucial in the assessment of head injury so do not dilate the pupils with atropine.

15.   FFFFT

Autoclaving does not work if the instruments have not been given a thorough "social" cleaning beforehand. Foreign matter must be removed from the instruments before autoclaving.

16.   TTTTT

17.   TTTTT

18.   FTFTT

Ventilation of the patient is more important than preventing aspiration - so if cricoid pressure is preventing ventilation, remove it! Decide early that intubation has failed and concentrate of ventilating the patient until they recover spontaneous ventilation.

19.   TTTFT

20.   TFTTT

High or low potassium will cause cardiac arrhythmias and disturbances in balance can occur after a variety of drugs and conditions.

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PAIN *Clinical Updates*

INTERNATIONAL ASSOCIATION FOR THE STUDY OF PAIN

Pain and Rehabilitation from Landmine Injury

International pacts such as the Geneva and Ottawa Conventions forbid weapons   that cause indiscriminate, unnecessary mutilation[1]. Still, these weapons are used worldwide and create victims long after peace agreements have been signed.

Pain, particularly phantom limb pain (PLP), is highly prevalent in landmine victims. These victims, often poor and rural, have much to lose from injury and disability. Relief agencies such as the Red Cross are ill equipped to deal with pain problems, and specialist pain relief organizations such as Douleur Sans Frontières have limited resources. The worst-hit countries lack IASP chapters. Health professionals, particularly members of IASP, have a responsibility not only to treat these victims but also, under the ninth item of the IASP constitution, to inform governments and the public about the suffering caused by these weapons and the measures needed to prevent their use[2–10].

Medical needs are divided between initial acute care and long-term rehabilitation and pain management, particularly of PLP. Many factors, however, may limit care. Among these are extremes of geography and terrain; dangers of travel during conflict; and looting of hospitals, sometimes with injury or death of workers. There may be great poverty, with poor education and social structures. Health care funding may be inadequate or limited by donor-derived constraints[3].

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Epidemiology

The lack of quantitative data precludes a precise and complete account of the health effects of landmines, but estimates can be made. The International Committee of the Red Cross (ICRC) has collected data on immediate injuries among mine survivors[5]. Several demographic surveys have tried to document the social consequences and frequency of mine-related injuries[8–10]. All available data suggest that the impact of landmines may be grossly underestimated, as only the fittest survivors reach treatment.

Mine injuries. Between 1995 and 1996 the ICRC registered 9384 landmine casualties[3,5]. That accounted for 27 % of surgical patients seen by the ICRC in three countries. Non-combatants (women, men >50 years, and children <15 years) accounted for 7.3%, 4.2%, and 19.8%, respectively.

Three distinct patterns of injury are[4,5,7]:
- I (30%) from standing on a buried blast mine. Victims sustain traumatic amputation of the lower limb and often injure the other lower limb or genitalia.
- II (50%) from fragmentation mines[7], which explode at waist height, have a

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killing zone of 25 m, and have an injury zone of 200 m. Injuries to head, neck, chest, or abdomen are often fatal.

- III (5%) from handling a mine. The victim, often a child, sustains severe upper limb injuries with associated face injuries.

The remaining 15% follow no particular pattern[5]. Coexisting long-term injuries may involve the eyes and peripheral nerves.

Social impact. A study of 206 communities in Afghanistan, Mozambique, Cambodia, and Bosnia found a heavy toll in physical, mental, and economic disability[8]. The WHO Global Burden of Disease—which assesses the impact of social, economic, and physical handicap on the individual, the family, and society—rates below-knee amputation as the midpoint of severity. Limb amputation impairs physical and hence earning capacity and may be accompanied by profound psychiatric problems and ostracism. Loss of income occurs through loss of land and livestock, and reduced access to food and water supplies. Agricultural production might be tripled in some areas by removal of landmines[8].

Numbers of amputees. Fatality rates average around 40%. For each person killed, 1.5 are injured[4,5,9]. Every year landmines kill 15,000 people, mainly civilians of whom 20% are children younger than 15 years[7,5]. Thus a decade from now, there will be about 250,000 documented landmine-related amputees. There may be many more, as there are 100,000 amputees in Angola already (J. Meynadier, personal observation). A retrospective analysis of 720 patients injured by mines suggests an overall amputation rate of 28 %. By combining ICRC data[5] about residual disability with the above-cited survey of landmine injury prevalence[8], one may estimate the number of amputees in the four countries studied. Further data on the numbers of mines per square mile in these and other countries[7] reinforce these weapons' potential health problems in terms of amputees per 1000 inhabitants (Table 1).

Medical Needs

Landmine injury victims are one group among many seeking medical care in those countries where mines have been used. Their relatively small annual needs are compounded over time because their long-term medical attention drains scarce resources, particularly as victims accumulate.

Acute care[3]. Evacuation of the injured from the minefield, control of bleeding by pressure dressing or tourniquet, and splinting of fractures are immediate needs. In wartime an epidemiological approach based upon first aid, tetanus vaccination, and antibiotic prophylaxis is more cost-effective than the traditional approach of urgent surgery. Basic nursing care saves more lives than heroic surgical interventions and is more easily available locally. A chest drain should be inserted if penetrating chest injuries are suspected. Antibiotic prophylaxis (benzylpenicillin) and tetanus prophylaxis should be administered. Delayed surgical intervention influences overall quality of survival[6]. Traumatic bilateral above-knee amputation and/or signs of intra-abdominal bleeding are ominous and justify an aggressive approach[4].
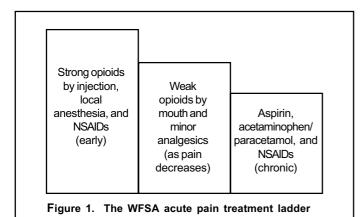
> Rehabilitation and pain control for landmine survivors have gained little attention so far. Instructions for the treatment of postamputation pain and PLP should be made available for use by relief agencies and local health care workers.

Evacuation may be slow. Only 25% of those treated by the ICRC arrived within six hours of injury; 15% traveled more than three days. In-hospital care is often limited by inadequate personnel and resources that can make surgery life-threatening. After excision of dead and contaminated tissue the wound should be left open for five days. Repeated operations and skin grafting may be necessary to achieve secondary closure. Sophisticated anesthetic practice may not be possible in areas where landmines are most common. Ketamine and local anesthetics are generally available in such settings and potentially offer effective postoperative pain relief. Spinal anesthesia can be administered safely by trained nonmedical personnel and is used frequently for subsequent operations. Adequate pain relief improves outcome by reducing complications and facilitating early recovery[11–14]. Routine pain assessment and organized provision of simple analgesic techniques will optimize postoperative analgesia[11].

Fig. 1, a modification of the WHO analgesic ladder for cancer pain, depicts suggestions for the treatment of acute postoperative pain, burns, and trauma from a review published jointly by IASP and the World Federation of Societies of Anaesthesiologists (WFSA)[12]. This review is available in French, Russian, and Arabic[15].

The WFSA "acute pain treatment ladder" uses well-known and simple techniques of regional anesthesia and a limited number of analgesics in a three-step approach. Its application depends upon on-site availability of these agents. Regional anesthesia provides excellent operating conditions and postoperative pain relief. Single-shot techniques or long-acting (>24 hour) blockade with dilute solutions of bupivacaine at plexus or peripheral nerves are alternatives when opioids are unavailable and pose less risk of hypotension, urinary retention, and immobilization than central axis blockade. Peripheral blockade requires less supervision postoperatively.

Table 1: Mine injuries per year (from Ref. 8). *Estimated numbers of amputees per 1000 population in parentheses*			
	Age (yrs)		Miles per
	<14	15-44	>45	square mile
Afghanistan				40
male	9.0 (2.5)	95.4 (26)	37.1 (10)	
female	8.0 (2.2)	4.5 (1.2)	18.1 (5.0)	
Bosnia				152
male	0.7 (0.19)	8.1 (2.2)	2.3 (0.64)	
female	0.0 (0)	0.0 (0)	0.5 (0.1)	
Cambodia				142
male	4.0 (1.1)	51.3 (14.6)	29.4 (8.23)	
female	0.4 (0.11)	2.3 (0.64)	3.7 (1.0)	
Mozambique				7
male	1.4 (0.39)	14.3 (4.0)	10.6 (2.9)	
female	1.0 (0.28)	3.2 (0.89)	5.6 (1.5)	

Figure 1. The WFSA acute pain treatment ladder

Patients with PLP may suffer from an exacerbation of their pain during regional anesthesia, but this problem subsides as the block wears off[14]. If this problem occurs during an operation on an amputee, it does not usually respond to opioids, but lignocaine, diazepam, or thiopentone have been successful[17].

Rehabilitation. Rehabilitation starts from day 1 with passive movement and active mobilization on crutches as soon as possible. In the case of lower limb amputation, restoration of function requires a prosthesis to regain mobility and make crutches unnecessary[18]. All too often PLP prohibits use of a prosthesis and creates a vicious circle of depression, isolation, and continued suffering. Psychological rehabilitation and recovery of self-esteem are dependent on social re-integration[19].

> If you give someone a leg - you are giving him hands.
> —Jacques Meynadier

The use of a prosthesis is vital to the rehabilitation process. Because of continued bone growth, prostheses for children need to be refitted every six months. Skin breakdown caused by growing bone may make reamputation necessary.

Phantom Limb Pain

Incidence and characteristics. It is helpful to distinguish between painless phantom sensations, stump pain, and pain in the amputated parts of the body as there are implications for pathophysiology, outcome, and treatment[20]. Few studies have looked at traumatic amputees and most trials are in elderly arteriopaths, but the reason for amputation does not seem to influence the long-term complication rate. Military casualties suffer the same type and frequency of problem as civilians[21].

Phantom sensations are experiences of the missing limb as though it were still present. Like PLP, they can start at the time of operation or much later. They can vary from vivid sensations moving in a complex fashion, to a vague and fixed awareness of fingers or toes attached to the stump ("telescoping"). *Stump pain* is pain felt in the stump only and not the absent limb. *Phantom limb pain* occurs commonly both in children[22,23] and in adults[20,23–25]. Patients may not mention it for fear of being ridiculed.

PLP varies greatly in frequency and intensity[21]. Emotional and autonomic influences can provoke or reduce it. The pain is generally felt in the more distal part of the amputated limb (toes, fingers) and has been described by Jensen et al.[20] as either exteroceptive (stabbing, burning) or proprioceptive (squeezing, cramp-like) in nature. It can be continuous or intermittent, and its intensity may be mild to excruciating. Phantom sensations, stump pain, and PLP are closely associated. PLP usually is less severe in amputees without phantom sensations or stump pain[24,25]. It seems to be less likely if the initial amputation is treated actively and a prosthesis promptly used[26].

A recent survey in 590 ex-servicemen found that PLP persisted in 47% of the amputees, disappeared in 16%, and required treatment in 55%. In this survey PLP was so severe (VAS 8.7) in 25% that they sought pain consultation. A large, older military survey found nearly identical figures[25].

Predisposing factors. Age, site of amputation, or pre-amputation pain intensity seem not to influence the persistence of late (>6 months) PLP[20,23–25,27]. No conclusive data link the type of anesthetic used during amputation and the incidence of PLP.

Despite earlier claims[28,29], a well-controlled, randomized trial did not show a reduction in the incidence of PLP by preemptive epidural analgesia[30]. This question is important as preamputation epidural analgesia is not without risk. The study did, however, show that active pain control decreased the incidence and severity of chronic pain problems.

Treatment. Treatments must reflect solid clinical experience or experimental evidence. No single form of treatment claims success[19].

Recently it has been suggested that transcutaneous electrical nerve stimulation (TENS), paracetamol (with or without a weak opioid), and nonsteroidal anti-inflammatory drugs (NSAIDs) are more effective for PLP than injections, "centrally acting" analgesics like tricyclics or anticonvulsants, and strong opioids[24]. Simpler methods of pain relief appear to be more effective and are more accessible in countries with landmine problems. Clinical experience[31] and that of the voluntary agency Douleur Sans Frontières in the developing world suggests that neurolytic blockade of neuromas may reduce stump pain and that TENS can reduce PLP[32].

Evidence for efficacy of second-line therapies for PLP usually is based on small numbers and limited follow-up[33–39]. These treatments include calcitonin, beta-blockers, neuroleptics, injection of local anesthetic drugs into the contralateral side, neurosurgery, and central stimulation. Other treatment methods may have been tried unsuccessfully and not reported, or not published owing to negative results.

There is increased interest in the use of NMDA antagonists in chronic pain conditions even though side effects limit their current use. They may also have a place in the preemptive management of postamputation pain problems. The wide use of ketamine in developing countries may yield data about the role of this NMDA antagonist to reduce PLP[40,41].

Sympathetic blockade has been used diagnostically and therapeutically. However, neurolytic block normally requires radiologic control and its effect gradually wears off[42].

Discussion

Those who produce and use armaments rarely consider their long-term effects upon health. From a military point of view landmines continue to be considered an effective weapon, due to their low cost and deterrent capabilities.

Implementation of a total ban on production, sale, stockpiling, and use of these weapons will prove difficult if not impossible, as has been the case with biological and chemical weapons. According to the World Health Organization (WHO), at current rates more than ten centuries would be required to remove the more than 100 million landmines already scattered around the globe.

Preventive measures in the countries afflicted with large numbers of mines include awareness programs on the risk of handling and efforts to clear or recover mines for commercial gain. Treatment and rehabilitation of victims will continue to be the principal humanitarian action needed. Rehabilitation and pain control for landmine survivors have gained little attention so far. Instructions for the treatment of postamputation pain and PLP should be made available for use by relief agencies and local health care workers.

The precise impact of PLP on the outcome of rehabilitation of minefield victims in the developing world must be assessed before we can estimate the response needed. However, data collection must not impede continued efforts by relief and medical agencies such as Douleur Sans Frontières. The incidence of severe PLP is at least 25 % in published surveys. PLP may prevent use of prostheses. In the case of single lower limb amputation, injury to the remaining limb may make weight-bearing more hazardous, further jeopardizing rehabilitation.

> The importance of pain control for optimal quality of life and long-term rehabilitation is increasingly obvious.

Treating the individual with relatively inexpensive and effective treatments is possible, and neurolytic blockade of neuromas and TENS have been shown to be effective under these circumstances (J. Meynadier, personal observation). The authors' observations support the multimodal treatment plan advocated by Sherman and colleagues[19,21]. They encourage a sympathetic discussion between health care worker and patient about phantom sensation and PLP and emphasize use of a prosthesis. They also advocate use of TENS and minor analgesics to disrupt the pain-anxiety-tension cycle. Their recommendation for referral to multidisciplinary pain treatment, however, is often difficult to carry out in practice.

Public discussion of landmines has taken place more as a political than a medical dialogue[43]. For other sources of pain such as cancer, burns, or operation, society's perspective is evolving from a view of the individual as an anonymous host

of a pathophysiological process toward a patient-centered focus. As this evolution advances, the importance of pain control for optimal quality of life and long-term rehabilitation is increasingly obvious. In parallel fashion, the crucial yet still unmet need for pain control among victims of landmine injury must now receive the attention of pain specialists worldwide.

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