UPDATE IN ANAESTHESIA

WORLD ANAESTHESIA

Editorial

Welcome to Update in Anaesthesia No 16! We are delighted with the feedback we have been receiving about Update and always look forward to receiving your comments and ideas by mail or email. We have responded to a request for some articles about critical care topics and have included two papers in this edition.

Recently we have been fortunate to receive the support of the Royal College of Anaesthetists of Great Britain and the Association of Anaesthetists of Great Britain and Ireland who have allowed us to reprint some of their material published at National Meetings. We have also been given permission to use abstracts from the journal Anaesthesia and would like to thank Professor Harmer, Editor in Chief, for his support. The Association of Anaesthetists have also given us a generous educational grant which will allow us to print cdroms containing the back issues of Update in Anaesthesia, and a variety of other anaesthesia and critical care material. These will be sent out to readers later in the year. We would like to thank Dr Peter Wallace, the President of the Association, for their kind support.

Readers wishing to get paper copies of previous editions of Update in Anaesthesia can purchase issues 6 - 12 in a bound set from Teaching Aids at Low Cost (TALC). TALC PO Box 49, St Albans, Herts, AL1 5TX, UK. Telephone +44 (0) 1727 853869, Fax +44 (0) 1727 846852, e-mail: talc@talcuk.org

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ACID BASE BALANCE AND INTERPRETATION OF BLOOD GAS RESULTS
Dr DH Barrett, Principal Anaesthesiologist, Ngwelezana Hospital, Empangeni, South Africa.

Introduction

Acid base disturbances are indicators of serious underlying pathology, rather than being the pathology themselves. Arterial blood gas examination is a useful investigation in patients with suspected respiratory or metabolic disease and serial blood gas investigation can monitor the progress or treatment of the underlying disease. The blood gas should be considered in conjunction with the patient's clinical condition. It does have a limitation because we only measure the extracellular fluid and do not know what the intracellular pH and gas tensions are.

Many clinicians find it difficult to interpret the blood gas results. This overview is written to give a basic understanding of the blood gas and a step-wise approach to its interpretation. The section on physics is to give a more complete understanding but you can gloss over it and go straight to the clinical significance.

Some physics

pH is the negative log of the H^+ ion concentration.

When pH = 7, the H^+ concentration is 10^{-7} or $1/10^7$

This is neutral because the H^+ and OH^- concentration is the same.

$H_2O \times H^+ + OH^-$

When the pH = 1, the H+ concentration is 10^{-1} or $1/10$. This is a very strong acid.

- pH 7.00 = neutral
- pH >7 = alkaline
- pH <7 = acid
- pH 7.4 = physiological pH of extracellular fluid. (Range of normal 7.35 - 7.45.)

Because of the log function, a small change in the pH is a significant change in the H^+ concentration. If the pH drops from 7.4 to 7.0, the acidity is $2^1/_2$ times higher.

pH	H^+ concentration
7.0	1/10 000 000
7.1	1/12 589 254
7.2	1/15 848 931
7.3	1/19 952 623
7.4	1/25 118 864

Usually pH is measured directly by a special glass electrode that has a H^+ permeable membrane.

HCO_3^- is measured by a bicarbonate electrode or may be calculated.

CO_2 is usually measured directly by a CO_2 electrode.

There are numerous physiological buffers that help prevent sudden swings in the intracellular pH (such as bicarbonate, lactate, phosphate, ammonia, haemoglobin, proteins and others). The bicarbonate system is used to regulate the whole-body pH because it is possible to regulate it at two different sites: HCO_3^- is regulated by the kidneys and CO_2 is regulated by the lungs.

$$H^+ + HCO_3^- \times H_2CO_3 \times H_2O + CO_2$$

The exact pH can be calculated from the Henderson Hasselbach equation

$$pH = pK + \log \frac{[\, base \,]}{[\, acid \,]}$$

$$= pK + \log \frac{[\, HCO_3^- \,]}{[\, H_2CO_3 \,]}$$

pK is a constant for the specific buffer. (For the bicarbonate buffer system at 37°C it is 6.1)

Because HCO_3^- is controlled by the kidneys and CO_2 is controlled by the lungs, this equation becomes

$$pH = constant \frac{KIDNEY}{LUNG}$$

Abbreviations used in acid base notation	
p	Negative log (lower case "p")
P	Partial pressure (upper case "P")
PA	Alveolar partial pressure (upper case "A")
Pa	Arterial partial pressure. (lower case "a")
Pv	Venous partial pressure

Notes about terminology: acidosis/acidaemia and alkalosis/alkalaemia

The suffix "-aemia" means "in the blood."

The overall acid base status of the blood is correctly referred to as an acidaemia or alkalaemia. This is taken from the pH alone and does not consider if the primary defect is metabolic or

respiratory and if there is compensation or not. The metabolic or respiratory components in the blood or any other body fluid have the suffix "-osis". If there is (for example) a metabolic acidosis with incomplete respiratory compensation there will be with a low pH and therefore an acidaemia.

Clinical significance

The bicarbonate buffer system is the most important buffer system in the body and is the one measured with the blood gas. The lungs can adjust CO_2 exhalation, and the kidneys can adjust HCO_3^- excretion or retention, so the precise ratio of acid to base can be maintained and adjusted.

The respiratory system (CO_2) can make rapid adjustments within minutes.

The metabolic component (renal, bicarbonate) takes hours or even days to adjust.

These two systems work together to maintain a fine balance. They aim to keep the extracellular pH 7.4 as this is the optimal environment for most metabolic activity such as chemical reactions catalysed by enzymes and transport of substances across cell membranes.

Pathological processes such as tissue hypoxia, renal failure, hypoventilation will all disrupt the normal acid base balance. If there is an abnormality in one part of the system, the other part will attempt to compensate and correct the pH.

Acid base disturbances and some examples of how they may occur

Compensatory mechanisms will tend to restore the pH towards normal even though the $[HCO_3^-]$ and the PCO_2 are not restored until the primary disturbance is corrected. The compensatory mechanisms should not overshoot. For example a metabolic acidosis will drop the pH to <7.4. If there is respiratory compensation the pH will return towards normal but will not overshoot to become >7.4.

Tips about determining which is the primary defect and which is the compensatory effect.

The primary defect (metabolic or respiratory) will go in the same direction as the pH. That is towards an acidosis if the pH is low or towards an alkalosis if the pH is high.

The compensatory effect (respiratory or metabolic) will go in the opposite direction.

The compensation will bring the pH back towards normal but it will never overshoot and will seldom actually reach normal.

For example: if there seems to be a metabolic acidosis and a respiratory alkalosis, the pH tells you which one is primary and which one is compensatory. If the pH is low, the primary defect is metabolic acidosis with respiratory compensation. If the pH is high, the primary defect is respiratory alkalosis with metabolic compensation.

Further reading

1. Update in Anaesthesia No 13

2. Alan Grogono has produced a very good tutorial on acid base. It is at http://www.acid-base.com/

Acid base disturbances		
Respiratory acidosis	$PaCO_2$ increased	This occurs when there is inadequate ventilation and CO_2 production is greater than CO_2 elimination. It may occur with airway obstruction, respiratory depression due to drugs or head injury, lung diseases, etc
Respiratory alkalosis	$PaCO_2$ decreased	This occurs with hyperventilation. The hyperventilation may be in response to hypoxaemia and hypoxic respiratory drive. The lungs are more efficient at eliminating CO_2 than at absorbing O_2 so patients with diseased lungs frequently have hypoxaemia with a normal or low CO_2. Mechanical ventilation with a large minute volume also leads to respiratory alkalosis.
Metabolic acidosis	HCO_3^- decreased (base deficit)	Multiple aetiologies ● Loss of bicarbonate due to GIT losses or chronic renal disease (Normal anion gap) ● Addition of inorganic acids such as diabetic ketoacidosis, lactic acidosis associated with tissue hypoxia, salicylate, ethylene glycol and other toxins, decreased acid excretion in renal failure (increased anion gap)
Metabolic alkalosis	HCO_3^- increased (base excess)	Occurs with loss of gastric acid (e.g. pyloric stenosis) and diuretic therapy. Metabolic alkalosis is commonly associated with low serum chloride.
Mixed and respiratory acidosis	$PaCO_2$ increased metabolic and HCO_3^- decreased	This is very dangerous and may occur in severe diseases such as septic shock, multiple organ dysfunction, cardiac arrest

Blood gas normal values *You should remember the numbers in bold*			
Item	**Normal range***	**Units**	**Notes**
pH	7.35 - 7.4 - 7.45		(no units)
PCO_2	4.8 - 5.3 - 5.9	kPa	
	36 - 40 - 44	mmHg	
PO_2	11.9 - 13.2	kPa	at sea level, $FiO_2 = 21\%$
	90 - 100	mmHg	lower at high altitude, higher if supplemental oxygen
HCO_3 (actual bicarbonate)	22 - 24 - 26	mmol/l	normal values vary if the PCO_2 is abnormal
standard bicarbonate	22 - 24 - 26	mmol/l	the $[HCO_3^-]$ after the sample has been equilibrated with CO_2 at 40mmHg (5,3kPa)
base excess	-2, 0, +2	mmol/l	a negative number is a base deficit

What do the different numbers mean?	
pH	The total acidity or alkalinity of the sample. This indicates if the patient has an acidaemia or an alkalaemia.
PCO_2	The respiratory component
PO_2	Indicates the oxygenation status of the patient and must not be confused with the acid base status. In general it is an indicator of the severity of lung disease, but cannot really be interpreted without knowing the FiO_2. The PO_2 could be up to 650mmHg (85kPa) if the lungs are normal and the FiO_2 is 100%.
	The predicted PaO_2 for normal lungs can be calculated from the alveolar gas equation (which I am not going to discuss)
	A rough approximation of the predicted PaO_2 is percentage inspired O_2 x 6mmHg. (eg a patient breathing 40% oxygen should have a PaO_2 of 6 x 40 = 240mmHg. If it is less than that, it means there is a shunt. Blood is not passing a ventilated alveolus before getting to the aorta. The worse the lung disease, the lower the PaO_2 will be at any given FiO_2.
HCO_3 (Actual bicarbonate)	The renal component.
Standard Bicarbonate	Another measure of the renal (metabolic) component. More useful than the actual bicarbonate as it has been corrected for an abnormal PCO_2
Base Excess	The amount of strong acid (or base if there is a base deficit) needed to titrate 1litre of blood back to pH 7,4 at $PCO_2 = 5,3$kPa and Temperature = 37°C
	Another measure of the renal (metabolic) component.
	It gives the same information as the standard bicarbonate except that the normal value is 0 instead of 24.

	Stepwise interpretation of the blood gas	
1	Is the overall picture normal, acidaemia, alkalaemia?	pH < 7.35 = acidaemia [...go to step 2] pH > 7.45 = alkalaemia [... go to step 5]
2	If there is an acidaemia: is the primary defect metabolic or respiratory or mixed?	CO_2 high = respiratory acidosis [...3] Bicarbonate low or BE negative = metabolic acidosis. [...4] Both of the above = mixed metabolic and respiratory acidosis.
3	If there is respiratory acidosis: is there metabolic compensation?	The CO_2 is high (resp acidosis) but the metabolic component is going in the opposite direction (BE or SB high, towards metabolic alkalosis) then there is metabolic compensation.
4	If there is metabolic acidosis: is there respiratory compensation?	BE is negative (metabolic acidosis) but the respiratory component is going in the opposite direction (CO_2 low, towards resp alkalosis), then there is respiratory compensation.
5	If there is an alkalaemia, is the primary defect respiratory or metabolic?	The primary defect will go in the same direction as the pH (towards alkalosis): respiratory alkalosis will have low CO_2 metabolic alkalosis will have high SB and positive BE.
6	If metabolic or respiratory alkalosis, is there any compensation by the other one?	Same principles as above
7	Look at the oxygenation	Is the PO_2 consistent with the FiO_2? If it is lower than expected, it either indicates lung disease, right to left shunt, or venous sample. (A venous sample usually has $PO_2 < 40$mmHg, saturation < 75%). The lung is much more efficient at eliminating CO_2 than absorbing oxygen so lung disease will show in the low PO_2 but the PCO_2 is often normal or even low. If the CO_2 is very high, the O_2 will also be low.
8	Summarise the interpretation	eg. There is a metabolic acidosis (because the pH is low and BE is negative) with respiratory compensation (because the PCO_2 is low).
9	Try to establish the cause	

ANAESTHESIA FOR ELECTRO CONVULSIVE THERAPY

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Introduction

Electro convulsive therapy (ECT) is the electrical induction of a grandmal seizure. It is an effective therapy used as part of the treatment of several psychiatric conditions including depression, mania, catatonic schizophrenia and other psychosis. A short general anaesthetic is usually given for the procedure.

Anaesthetic Problems

Patient Population. Patients are often elderly with associated co-morbidity of other chronic medical and psychiatric disease.

Drug Interactions. Patients are frequently taking psychotrophic drugs. There are a number of important drug interactions which are listed below.

Monoamine oxidase inhibitors (MAOI) such as phenelzine and tranylcypromine. These may produce life threatening hypertensive crisis with precipitants such as ephedrine and pethidine.

Tricyclic antidepressants: increase cataholamine concentrations at central sites may lead to increased anaesthetic requirements and exaggerated response to catecholamines.

Lithium to be stopped at least 24hours before ECT as the drug is known to interact with suxamethonium and prolong apnoea.

Withhold anti convulsants

Repeat General Anaesthetics. ECT is usually given twice or three times a week over several weeks.

Location. In many hospitals ECT is administered at relatively isolated sites away from operating theatres. This may mean that help and backup to deal with unexpected problems or difficulties can be delayed or unavailable.

Of Any Anaesthetic. Nausea due to side effects of anaesthesia. Myalgia secondary to the use of suxamethonium.

Effects of ECT

Central Nervous System: The exact mode of action in the treatment of psychiatric illness is poorly understood. The ECT device delivers a brief pulse of current (0.5-0.8amps) through electrodes placed at specific locations on the head to induce a seizure or grand mal convulsion. There is an associated increase in cerebral blood flow, oxygen consumption, intracranial pressure and intraocular pressure.

There may be a post-ictal phase with confusion, agitation or amnesia. The patient may complain of headache after the procedure.

Musculoskeletal: Uncontrolled myoclonic-tonic contractions may cause bony or musculoskeletal injury to the patient (or their carers). The passage of current directly stimulates the jaw muscles and causes the teeth to clench which may lead to dental or oral injury. Because of increased muscular activity oxygen extraction is greatly increased and the patient may desaturate or become cyanosed.

Cardiovascular System: There may be initial parasympathetic stimulation with risk of bradycardia and hypotension followed by sympathetic stimulation with possible tachycardia, hypertension and dysrhythmias.

Gastrointestinal System: there is an increase in intra gastric pressure and there may be increased salivation and nausea and vomiting.

Anaesthetic Management

Aims

- Safety.

- Pleasant and stress free environment for the patient who may be returning for multiple treatments.

- Rapid loss of consciousness and attenuation of the hyperdynamic response.

- Reduction of seizure movements (with appropriately judged paralysis) to avoid gross movements and injury but at the same time allowing a visual assessment of the motor element of the fit.

- Minimal interference with seizure activity.

- Prompt recovery of spontaneous ventilation and consciousness

Preoperatively

Assess the history, physical examination, and investigations as appropriate.

Identify and optimise co-existing disease if time allows before ECT.

It is important to explain the procedure and gain informed consent from the patient to proceed. However the nature of the underlying condition may lead to patients refusing treatment. It is important when imposing treatment against a patient's will to do this within the appropriate psychiatric framework and regulations of the country or region in which you are practising.

The patient may be an unreliable historian. Take extra care to ensure that the patient is appropriately fasted.

Premedication

Rapid return of consciousness and recovery is ideal so avoid premedication unless specifically required. Benzodiazepines will increase the seizure threshold so should be avoided if possible. Anticholinergic (atropine 0.6mg im or iv or glycopyrrolate 0.2mg

im or iv) may be given to attenuate bradycardia and salivation but are not absolutely necessary.

Monitoring

Attach patient monitoring if available. Pulse oximeter is particularly useful to monitor cardiac rate and any desaturation that may occur during the fit. ECG and non invasive blood pressure recording are also useful if available. The psychiatric team may monitor the electroencephalogram (EEG) to track the progress of the fit.

Induction

Preoxygenate the patient if tolerated.

Use a sleep dose of one of the following intravenous induction agents: methohexitone, propofol, thiopentone, or etomidate. Maintain the airway with an anaesthetic facemask, hand ventilating with 100% oxygen.

Muscle Relaxation

In order to modify the fit give a low dose of suxamethonium calculated to cause incomplete muscular paralysis. Complete muscle paralysis obscures the muscular element of the fit and makes assessment of fit duration difficult. Start with a dose of 20-50mg ie 0.3 to 0.5mg/kg. Maintain the airway and ventilate with 100% oxygen throughout the procedure until the patient has resumed spontaneous and regular respiration. Check that the airway is clear and dentures are removed (if not already done so). Insert an oropharyngeal airway or bite block before allowing the psychiatrist to administer the stimulus when suxamethonium fasciculations has finished.

Carefully observe the patient for signs of a modified fit. Appropriate level of paralysis would be slight twitching of face and limbs but little more. If no movements are seen the dose of suxamethonium was probably too high. Signs to suggest evidence of successful passage of current include dorsi flexion of great toes, dilatation of pupils and goose-pimples.

Record the doses of induction agent and suxamethonium and the patient's response to them.

This allows appropriate dose adjustments to be undertaken on subsequent occasions if necessary. (for example increasing the dose of suxamethonium to increase the fit modification)

The adequacy of ECT is judged by duration of seizure. Seizure activity is monitored direct observation of the modified fit, isolated arm technique or EEG if available. An adequate seizure duration is defined as 25-30 sec of EEG activity or 15 sec of motor seizure duration. Motor seizure duration is 30% shorter than EEG seizure.

A prolonged seizure of 120seconds (on EEG) or 90 seconds of motor activity may be detrimental and should be terminated actively with drugs. Use one of the following intravenously: thiopentone, methohexitone, lorazepam, diazepam or midazolam.

Post ECT Care

Treat headache with simple analgesics or intra nasal sumatriptan.

Monitor the patient in recovery area until the patient is fully alert and able to ambulate.

Post ECT agitation, confusion and aggressive behaviour can be attenuated by

avoiding excessive stimulation during the recovery period. A small dose of benzodiazepine (eg midazolam) or haloperidol may be given if all else fails.

[I thank our Psychiatrists Dr. I. Selvamani & Dr. Kumanan who help me to prepare this article].

EDUCATION IN ANAESTHESIA - PART 1
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BASIC PRINCIPLES IN SETTING UP AN EDUCATIONAL PROGRAMME

When planning an educational program it is important to first **define the aims**. What do you want the students to learn? In anaesthesia the broad aim is to train people so that they have adequate, appropriate knowledge and skills to give anaesthesia safely to their patients and to enable them to keep the patient alive and unharmed during surgery. Perioperative care extends this to teach pre and postoperative care of the patient. This requires good knowledge of medical and surgical principles, including intensive care and pain management.

Having decided on the **aims** the next step is to work out what the students need to know, and how they are going to learn it - objectives. Anaesthesia requires both a **theoretical syllabus** and **practical training** to perform the various procedures that are undertaken.

The theoretical course is wide ranging. A **good basis of physiology and pharmacology** with their practical applications is invaluable, and really essential in a good program. Unfortunately in some parts of the world these basic sciences are not particularly well taught so that students learn the job by rote, without a clear understanding of why and what they do. Within this type of framework misconceptions are common, for example some students might believe that since hypovolaemia results in tachycardia, someone who is bradycardic is unlikely to be hypovolaemic. Knowledge of basic physiological principles allow the student to understand the basic mechanisms involved and to adapt their anaesthetic skills around the patient's physiology and pathophysiology. A good understanding of the physiology of cardiac output, and its distribution will help the student understand

why certain cardiovascular events occur and how best to manage them. The further implications of pathological processes and can be built on to sound basic knowledge.

Teaching complex topics to students of different backgrounds requires considerable teaching skills. Techniques vary from one country to the next, and it is hoped that within this short series some useful principles will be described. One technique when teaching large topics such as blood flow, cardiac output and systemic vascular resistance is to teach by breaking down the subject to components and building on prior knowledge. Often a complex topic may then be successfully taught. Where students have an understanding of secondary school physics teaching may be helpful. Pressure = Flow x Resistance tranforms to Blood Pressure = Cardiac Output x Peripheral Resistance. The latter can be further broken down to its components in Pouisseuille's equation which brings in the influence of length, viscosity (haematocrit) and the 4th power of the radius (vasodilated or constricted). These can also be applied to flow in the airways and in intravenous infusions. When these basics are understood more complex issues such as oxygen delivery can be introduced and mastered. The concepts of a high cardiac index associated with pregnancy, small babies and anaemic patients may be better appreciated, and the anaesthetic principles in looking after these patients developed with the student in the practical setting.

Understanding distribution of cardiac output also allows the student to understand why reduced doses of depressant drugs should be used in hypovolaemic patients (the amount of blood going to the brain and heart is kept the same, while the proportion to other non essential tissues, including muscle, is decreased). This means intramuscular administration is ineffective in shock and may even be dangerous because it forms a depot of drug which is released when the patient is resuscitated. Alternatively the apprehensive patient needs more induction agent to compensate for the increased proportion of cardiac output which goes to muscle where it has no effect. Premedication may counteract this by relieving apprehension. All of these theoretical physiological and pharmacological topics may not be considered as essential topics for anaesthesia training, but without them, the student is impoverished. The syllabus needs to cover aspects of many subjects, which should always be made relevant to clinical practice. Reinforcing these lessons in theatre is helpful.

Practical training is usually undertaken in the operating theatre, although some centers have access to clinical skills rooms where techniques such as intubation and airway care may be practiced. The importance of being taught correctly and consistently at the start of training cannot be overemphasised so that the student acquires good technique and understands the ergonomic principles that apply. When training a practical technique a good teacher will let the student watch while they demonstrate the method, explain the steps involved and then guide the student through the process. Unfortunately many who teach have not analysed the separate steps clearly, and therefore this valuable component of the process is lacking. The result is that the student will take longer to learn and may not learn to do the procedure well. This applies to common procedures such as intubation, ventilation and intravenous cannulation as well as regional and local anaesthetic techniques.

When detailed **Aims and Objectives of Training** have been drawn up, as they were by the Australian and New Zealand Faculty, now College, of Anaesthetists in 1976, there are **General Instructional Objectives** and much more detailed **Specific Instructional Objectives** which give a more detailed account of how the learning may be achieved. The important point is that the syllabus should be relevant, practical and define what the students should learn to accomplish the aims of the course. Some anaesthesia curriculums are available on the internet.

Finally there should be **assessment** to ensure that the students have achieved the learning that was planned. This may take the form of written examinations or practical assessments. If the course is well designed, the students are well taught, have sufficient ability to learn and have covered the course syllabus, the final examination should be passed. If many candidates fail, the students may not be up to standard required, the examination may not be assessing what was laid out in the syllabus, or the teaching has been ineffective. Ideally repeated assessments are better than one final examination. A logbook or portfolio of experience should be maintained by the student throughout their career and be inspected regularly by the teacher to ensure that each student has covered the practical elements of the course appropriately. Students should be encouraged to develop personal learning plans and develop the ability to learn independently from the teacher. Assignments are a useful way of encouraging this. Reflecting with the help of the teacher over their day to day work and particularly critical incidents will help the student TO mature in their practice.

Further information

Useful information regarding anaesthesia training is available from a variety of sources including:

- http//www.rcoa.ac.uk

- http//www.anzca.edu.au

SEDATION IN INTENSIVE CARE PATIENTS
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INTRODUCTION

Sedation is an essential component of the management of intensive care patients. It is required to relieve the discomfort and anxiety caused by procedures such as tracheal intubation, ventilation, suction and physiotherapy. It can also minimise agitation yet maximise rest and appropriate sleep. Analgesia is an almost universal requirement for the intensive care patient.

Adequate sedation and analgesia ameliorates the metabolic response to surgery and trauma. Too much or too little sedation and analgesia can cause increased morbidity e.g. oversedation can cause hypotension, prolonged recovery time, delayed weaning, gut ileus, DVT, nausea and immunosuppression; undersedation can cause hypertension, tachycardia, increased oxygen consumption, myocardial ischaemia, atelectasis, tracheal tube intolerance and infection.

Sedation in the ICU varies widely from producing complete unconsciousness and paralysis to being nursed awake yet comfortable. There are many components to the ideal regimen but key elements include recognition of pain, anxiolysis, amnesia, sleep and muscle relaxation.

Although the mainstay of therapy is pharmacological, other approaches are just as important:

- Good communication with regular reassurance from nursing staff
- Environmental control such as humidity, lighting, temperature, noise
- Explanation prior to procedures
- Management of thirst, hunger, constipation, full bladder
- Variety for the patient - e.g. radio, visits from relatives, washing/shaving
- Appropriate diurnal variation -gives pattern to days

ASSESSSSING THE LEVEL OF SEDATION

The dosage of commonly used sedative and analgesic drugs varies widely between patients because of variations in metabolism and pharmacodynamics. A valid method for monitoring sedation would allow sedation to be tailored to the individual.

Any scoring system needs to be simple, rapidly performed, non invasive and most importantly, reproducible.

Physiological variables, serum concentrations of drugs and neurophysiological tools such as EEG, CFAM and lower oesophageal contractility have all been used but are both expensive and unreliable.

The best systems are clinically based and the one used most commonly throughout the world is the Ramsay Scale. Six levels of sedation are used:

1. Anxious and agitated
2. Cooperative, orientated and tranquil
3. Responds to verbal commands only
4. Asleep but brisk response to loud auditory stimulus/light glabellar tap
5. Asleep but sluggish response to loud auditory stimulus/light glabellar tap
6. Asleep, no response

This should be completed hourly by the ward nurse but can be reduced in frequency as the patient stabilises. It is suggested levels 2 to 5 can be considered suitable for patients in the ICU.

An increase in the sedation score must prompt the physician to make a differential diagnosis between over sedation, decreased conscious level due to neurological/biochemical disease, or ICU-associated depression.

As a rule, the aim for the majority of patients is for them to be sleepy, although easily rousable and hence cooperative. It is preferable to allow the patient to breathe as soon as possible on SIMV or triggered ventilation, such as pressure support. Deep sedation with or without paralysis is reserved for severe head injury, critical oxygenation (reduces work of breathing and improves chest compliance) and diseases such as tetanus.

DRUGS USED IN SEDATION

The "Ideal Sedative Agent" should possess the following qualities:

- Both sedative AND analgesic
- Minimal cardiovascular side-effects
- Controllable respiratory side-effects
- Rapid onset/offset of action
- No accumulation in renal/hepatic dysfunction
- Inactive metabolites
- Cheap
- No interactions with other ICU drugs

Such a drug does not exist and therefore typically drug combinations are usually required. Sedative drugs may be given as boluses or infusions. As a rule, infusions for maintenance are preferable with boluses for procedures/PRN although continuous infusion results in higher cumulative doses.

Benzodiazepines

These are particularly useful because they are anxiolytic, anticonvulsant, amnesic and provide some muscle relaxation in addition to their hypnotic effect. Their effects are mediated by depressing the excitability of the limbic system via reversible binding at the gamma aminobutyric acid (GABA)-benzodiazepine

receptor complex. They have minimal cardiorespiratory depressant effects and are also synergistic with opioids. However rapid bolus doses can cause both hypotension and respiratory arrest. They are all metabolised in the liver. The common drugs used in this class are diazepam, midazolam and lorazepam.

Diazepam use has decreased because of concern about its active metabolites (esp. nor desmethyl diazepam) which has a long half-live and can accumulate particularly in the elderly and patients with hepatic impairment. It is safe to give in single boluses, if given sensibly.

Midazolam is water soluble at PH 4 yet fat soluble at PH 7 thus avoiding the unnecessary solvents required with the other 2 drugs and hence causing less irritation at the injection site. It has 3 metabolites, one of which (1-hydroxymidazolam) can accumulate in the critically ill. The normal elimination half life is 2 hours but can be as long as a few days in the long term sedated, critically ill patient.

Lorazepam undergoes glucuronidation and has metabolites thought to be inactive, and may become more widely used in time especially in hepatic disease.

Overdose or accumulation can be reversed by flumazenil, the benzodiazepine receptor antagonist. It should be given in small aliquots as large doses can precipitate seizures. It has a half-life of only 1 hour so may need to be given as an infusion.

There is wide inter-patient variability in the potency, efficacy and pharmacokinetics of benzodiazepines so the dose must be titrated to the level of sedation.

After long term administration the dose should be ideally reduced gradually or a lower dose reinstated if there is withdrawal (symptoms include insomnia, anxiety, dysphoria and sweating.)

Propofol (2,6-diisopropylphenol)

The mode of action is via the GABA receptor but at a different site to the benzodiazepines. It was first developed as an intravenous anaesthetic agent and has a rapid onset of action yet because it is metabolised rapidly, both hepatically and extrahepatically, it is ideal for continuous infusion. Recovery usually occurs within 10 minutes but it can accumulate with prolonged use, particularly in the obese patient. It is solubilised as an emulsion and the formulation can cause thrombophlebitis and pain so ideally it should be infused via a large or central vein. Prolonged infusions can lead to increased triglyceride and cholesterol levels and indeed its use is not licensed is children because of associated deaths attributable to this fat load. A theoretical maximum recommended dose is thus 4mg/kg/hr.

Disadvantages also include cardiorespiratory depression, particularly in the elderly, septic or hypovolaemic patient. Infusions may cause the urine to colour green.

Ketamine

Ketamine acts at the N-methyl-D-aspartate (NMDA) receptor. In subanaesthetic doses ketamine is sedative and also analgesic. However it is generally not used because of the rise in blood pressure, ICP and pulse rate that may result. It also causes hallucinations but these can be avoided if administered

concomitantly with a benzodiazepine. It appears not to accumulate and sometimes has a role in severe asthma given its bronchodilatory properties.

Etomidate

Historically was used in ICU as an infusion but is now no longer used as it has been shown to cause adrenal suppression, even after a single dose.

Barbituates

These, for example thiopentone, have been used especially in the management of patients with head injuries and seizure disorders. They cause significant cardiovascular depression and accumulate during infusions leading to prolonged recovery times. Thiopentone is still used occasionally in severely raised ICP to induce a "barbituate coma', and in intractable seizure activity.

Butyrophenones and phenothiazines

Strictly these are classed as Major Tranquillizers but they remain useful in ICU, particularly in agitated/delirious patients. A "sliding scale" of haloperidol may be particularly useful in a patient with delerium to promote calmness i.e. increasing doses if no effect after 15 minutes until the desired response is achieved. Haloperidol in particular causes minimal respiratory depression and has less alpha blocking tendency than chlorpromazine and hence less hypotension. Other side effects include prolongation of the QT interval (caution when given with erythromycin), extrapyramidal effects or neuroleptic malignant syndrome.

Clonidine

This is the most well known of the alpha-2 agonists but also has alpha-1 agonistic properties. A more specific agonist is dexmedetomidine but this is expensive and rarely available at present. It is particularly useful in patients with sympathetic overactivity such as alcohol withdrawal and tetanus as it inhibits catecholamine release. It also is synergistic with opioids and acts at the spinal cord to inhibit nociceptive inputs thus imparting analgesia. It is contraindicated in hypovolaemia and can cause hypotension, bradycardia and dry mouth.

Chlormethiazole

This is a vitamin B derivative widely used for treatment of delirium tremens. It is not a respiratory depressant and is an anticonvulsant.

Chloral Hydrate

This is used in paediatric intensive care as an adjunct usually to a benzodiazepine such as midazolam. It is metabolised in the liver to the active compound trichloroethanol. Metabolites can accumulate in renal dysfunction

Volatile agents

Isoflurane has been used in concentrations of up to 0.6% and produces good long term sedation with minimal cardiorespiratory side effects and yet rapid awakening. Scavenging and pollution are a problem as is incorporating the vaporiser into the ventilator. Free fluoride ions from metabolised methoxyfluorane can cause renal failure. More recently desflurane has been shown to be effective in sedation with rapid offset of effects.

DRUGS USED FOR ANALGESIA (In combination with sedation)

Opioids are the mainstay of treatment and possess sedative, antitussive (cough suppressant) and hypnotic effects besides the obvious analgesic effects. They work at the opioid receptors, reclassified in the late 80's to OP1 (old delta), OP2 (old kappa), OP3 (old mu). Most of the recognised effects are mediated via the OP3 receptor. Unfortunate effects include gastrointestinal stasis and respiratory depression. Newer opioids have fewer side effects and accumulate less. It is equally important however to remember other analgesic techniques such as non-steroidal anti-inflammatory drugs NSAIDs, paracetamol, regional techniques (esp. epidural infusions for post op patients/lower limb trauma)

Morphine

This is the most commonly used drug. All other opioids are measured against morphine, although some newer agents have specific advantages. The dose required for analgesia is very variable and it can be delivered as intermittent boluses (problems with peak and trough effects but less accumulation) or as a continuous infusion.

Morphine is metabolised mostly in the liver to two main products, morphine-3-glucuronide and morphine-6-glucuronide. Both are excreted renally and will accumulate in renal dysfunction. The M-6-G metabolite also has independent longlasting, sedative activity. Morphine has minimal cardiovascular side effects unless given as a large bolus to hypovolaemic patients or secondary to histamine release. It is relatively contraindicated in asthma and renal failure and should be given in small increments in uncorrected hypovolaemia. However its use in renal failure is acceptable as long as the dosing interval is increased or the infusion rate reduced. Normal duration of action after a single dose is about 2 hours. Care should be taken, as with all opioids, in hepatic failure

Fentanyl

Fentanyl is a potent synthetic opioid derived from pethidine. It is presented as a short acting opioid with a rapid onset but after prolonged infusion the duration of action approaches that of morphine, although it does not accumulate in renal failure. It does not cause histamine release and is suitable for analgesia in the haemodynamically unstable patient.

Alfentanil

Alfentanil is one of the newer synthetic opioids, and has an onset of action about five times faster than fentanyl due to the small volume of distribution but is less lipid soluble so is not prone to accumulation. The duration of action is about a third of fentanyl and it too is safe in renal failure. It has minimal cardiovascular effects and is a potent antitussive agent. Although it is not particularly sedative, it does posses many of the qualities desired of the ideal ICU analgesic. It is a relatively expensive drug.

Other drugs to mention include **pethidine**, which is not suitable for use in infusions as the metabolite, norpethidine, may accumulate and cause convulsions. **Remifentanil** is an ultra short acting opiod metabolised by non-specific tissue and blood esterases. It has a rapid onset of action and does not accumulate after infusions even in organ dysfunction. It is however, very expensive and can cause significant bradycardia.

Naloxone is a specific receptor antagonist working at the OP3 (old mu) receptor. It completely abolishes the effects of all opioids at this site. The dose should be titrated slowly at the risk of unmasking arrhythmias or seizures in certain patients.

DRUGS USED FOR MUSCULAR RELAXATION

In some patients muscle relaxation may be needed in addition to sedation and analgesia. Such indications include:

- Early resuscitation (including intubation)

- Refractory hypoxaemia e.g ARDS - will decrease oxygen consumption and optimise chest wall compliance

- Raised intracranial pressure - stops coughing and patients resisting ventilation

- Status epilepticus and tetanus

- During patient transfer

- To allow inverse ratio/prone ventilation

It is vital to remember that relaxants have no effect of conscious level or comfort and should be avoided if possible. There are no standard clinical techniques to monitor conscious level in the paralysed patient so it is necessary to give generous doses of sedative drugs. Use of relaxants has fallen from about 90% of patients in the 80's to 10% of patients in the 90's in the UK.

Some relaxants used in anaesthesia are less suitable for use in the ICU such as curare because of the hypotension and histamine release. Suxamethonium is predominantly used during emergency tracheal intubation, but the resultant rise in serum potassium must be expected which makes it inappropriate for use in cases of renal failure. Excessive potassium release also occurs after 48hrs in extensive burns and spinal cord injury. Pancuronium is long acting but it may cause an undesirable tachycardia and it accumulates in renal failure. Vecuronium is an analogue of the aminosteroid pancuronium, but causes minimal cardiovascular side effects. It is suitable for intubation and infusion, dose 0.1mg/kg bolus, 1-2mcg/kg/min infusion but may accumulate in renal failure. Atracurium is a benzylisoquinolinium and is metabolised by ester hydrolysis and Hoffman (spontaneous) elimination. Its metabolites are inactive and it doesn't accumulate in renal or hepatic dysfunction. Histamine release occasionally occurs with boluses, but recovery occurs predictably within one hour regardless of duration of infusion. Intubating dose is 0.5mg/kg, infusion 4-12mcg/kg/min.

Monitoring should ideally be performed using a nerve stimulator (e.g. train-of-four count). Clinical monitoring such as cardiovascular reflexes to noxious stimuli should also be observed. Full "surgical" relation may not be necessary.

Problems with relaxants

- The patient may receive inadequate sedation and be aware. This can be checked by withdrawing muscle relaxants for a time to allow recovery of muscular function and assessment of sedation levels.

- Accumulation especially with aminosteroids in ARF

- Prolonged paralysis after discontinuation from accumulation

- Severe myopathy critcal illness polyneuropathy occasionally (esp. if steroids used as well)
- Loss of protective reflexes
- Tendency to perhaps oversedate
- Enhanced paralysis from other common ICU problems such as hypokalaemia, aminoglycoside antibiotics, hypophosphataemia

RECOMMENDATIONS

Non-ventilated patients

Pain should be titrated with opioids to the desired level. Co-operative patients may benefit from patient-controlled analgesia. Regional techniques in selected patients are ideal. Always use simple analgesics in combination, and consider other causes e.g. full bladder

Postoperative/short-term mechanical ventilation

If available then a combination of alfentanil and propofol allows a rapid wake up but is only really of benefit if used for less than 72 hours. Sometimes the high costs of short acting agents can be offset against the higher hidden costs of delayed weaning/ prolonged ICU stay. Alternatively a benzodiazepine/morphine combination is ideal.

Long term mechanical ventilation

There is little logic in using very short acting substances in these cases and longer acting drugs are as the weaning process will be prolonged anyway.

A recent randomised, blinded controlled trial has shown that daily interruption of sedative infusions reduced the duration of mechanical ventilation and intensive care stay in the critically ill. Infusions were interrupted until the patient was awake and could follow instructions or became agitated or uncomfortable.

Morphine plus midazolam or propofol were the agents used and the daily wake up procedure helped prevent too much of these agents being administered. This is a useful reminder that over sedation results in lengthened ICU stay, and that such a policy of interruption should be considered in all patients every day.

In some centres a newer technique of sedation is employed - patient controlled sedation - using increments of propofol, as opposed to morphine/fentanyl/pethidine used in patient controlled analgesia. This is a very effective technique in the awake, orientated patient. It minimises nursing time, is inherently safe and gives control to the patient. However it requires specialised, expensive equipment and is unsuitable for the majority of ICU patients.

SUMMARY

Good sedation can be achieved with simple combinations of drugs. Over sedation is widespread but use of sedation scoring and adequate nursing staff provision should reduce its frequency. Use of sedative drugs should be questionned daily, just as vasopressors/diatropes. Sedation should be prescribed on an individual basis as requirements wary widely and sometimes analgesia alone may suffice.

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DRUG	DOSE	COMMENTS
Propofol	0.5 - 4mg/kg/hr Bolus 5-50mg	Not licensed for children for ICU sedation Care in hypovolaemia. Rapid recovery
Midazolam	0.5-10mg/hr. Bolus 2-4mg Paeds: 5mg/kg dissolved in 50 mls. Infuse 1-2 mls/hr	Cheap. CVS stable. Good for prolonged sedation. May result in very prolonged sedation, particularly in the elderly
Morphine	1- 5 mg/hr. Bolus 2-5mg Paeds: 0.5mg/kg in 50 mls N/S. Infuse 1-4mls/hr	Accumulates esp. in renal failure. Histamine release.
Fentanyl	1-3mcg/kg/hr. Bolus 50 - 100mcg Paeds: 50mcg/kg to 50mls N/S. Infuse 1-4ml/hr	Less accumulation in renal failure. Less histamine release
Alfentanil	1-5mg/hr Bolus 0.5-1mg to supplement	Short acting and little accumulation. Expensive
Haloperidol	5-10mg boluses	Minimal effect on respiration.
Ketamine	Bolus 1-2mg/kg then infuse 10-45mcg/kg/min	Can be used in severe asthma. CVS stable. Emergence delirium
Thiopentone	50-250mg/hr	Use in epilepsy/raised ICP. Very prolonged wake up

MANAGEMENT OF SNAKE ENVENOMATION

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Introduction

Out of more than 3000 species of snake identifiable world wide, only one tenth of them are dangerous to human beings. There are three major families of venomous snakes:

- Elapidae (Land snakes like cobra, krait and coral snakes) - Snakes of this family have short & fixed fangs, which contain venom channels. Their tricolor bands (black red &yellow/white) encircle the body and they lack laureal shields (shield on the lateral aspect of head separating those shields bordering eyes from those bordering the nostril).

- Viperidae (Russell's viper, bamboo snakes) - These are further classified into pit vipers(crotalinae) and viperine vipers(Viperinae).Their fangs are long & movable. Their pupils are vertically elliptical. The ventral plates caudal to anus are in a single row. These snakes have a heat sensing pit as a small depression on the side of head for location of prey.

- Hydrophiladae (Sea snakes) - These snakes have a flattened tail.

Epidemiology

Although a major public health problem in many countries; the epidemiology of snakebite is still fragmentary, mainly due to lack of statistical data. This is compounded by the fact that the majority of victims come from rural areas, out of reach of the available medical facilities. It is estimated that snakebites may exceed 5 million per year, out of which approximately 100,000 develop severe sequelae. The incidence also shows a distinct seasonal pattern with a higher frequency in summers and during rains when the reptiles come out of their shelters. Epidemics of snake bite following floods owing to human & snake populations getting concentrated together have been noted in Pakistan, India & Bangladesh.

Snakebite is observed in all age groups, the majority (90%) affecting 11-50 year olds with males affected twice more often than females. Most bites occur between midnight and early morning and a large number of bites occur in fields, as most individuals are unable to spot the snake due to tall grass & crops. Fortunately every bite does not result in complete envenomation and more than half the victims escape without serious poisoning.

Table 1: Medically Important Snakes		
S.NO	REGION	TYPES
I	North America:	Eastern Diamond Rattlesnake (Crotalus adamanteus) Western diamond rattlesnake (C.atrox, C.viridis) Bothrops atrox (fer-de-lance)
II	Central & South America:	Bothrops jararaca & tropical rattlesnake (C.durissus, C.terrificus)
III	Britain:	European adder (Vipera berus)
IV	Europe:	Long nosed viper (V.ammodytes)
V	Africa:	Night adder (Causus species) Puff adder (Bitis arientan) Mambas (four species of Dendroaspis)
VI	Africa & Asia:	Cobra (Naja species) Saw-scaled viper (Echis carinatus)
VII	Part of Asia:	Russell's viper (V.russelli) Malayan Pit viper (Agkistrodon rhodstoma) Sharp-nosed pit viper (A.acutus) Mamushi Pit viper (A.halys) Haliu viper (Trimeresurus Flavoviridis) Kraits (Bungarus coeruleus, B.multicinctus)
VIII	Pacific- Australian area:	Tiger snake (Notechis scutatus) Death adder (Acanthophis antarcticus) Taipan (Oxyuranus scutellatus) Papuan black snake (Pseudechis Papuanus) King brown (Pseudechis australis)

However, if sufficient venom is injected during the bite to cause serious poisoning, the mortality can be high.

PATHOPHYSIOLOGY

Snake venom is a very complex chemical poison, containing approximately 5-15 enzymes and 3-12 non-enzyme proteins & peptides besides carbohydrates and metals, which exerts toxic & lethal effects on skin, hematological, nervous, respiratory and cardiovascular systems (Table 2). Different species have differing proportions of above mixtures. The picture may be further complicated by the release of endogenous mediators such as histamine, bradykinin & adenosine. Therefore snake venoms cannot be classified purely as 'neurotoxic' or 'cardiotoxic', although they may have some predominantly specific action. The effects however may conveniently, though arbitrarily, be classified into vasculotoxic for vipers, neurotoxic for elapids & myotoxic for sea snakes.

Viper venom. This is primarily vasculotoxic. It causes rapidly developing swelling of the bitten part. Local necrosis is mainly ischaemic as thrombosis blocks the local blood vessels and causes dry gangrene. Systemic absorption is via lymphatics. Some vipers such as Vipera berus (European Viper) cause vomiting, abdominal pain, explosive diarrhoea and shock within a few minutes of bite, which resolves spontaneously within half an hour. Persistence of the shock may however be fatal. Several viper venoms result in intracranial haemorrhage due to direct endothelial damage by 'haemorrhagin' (a venom component), which however does not affect the coagulation. In contrast other viper venoms (Crotalus, Bothrops) affect coagulation and a very small amount of venom can cause complete fibrinogen consumption. It can also differentiate various species of vipers, which can help in instituting appropriate antivenom therapy.

Elapid venom. Local necrosis causes a picture like 'wet gangrene' with a characteristic putrid smell due to direct cytolytic action of venom. Systemic absorption occurs through venous channels. These result in primarily 'neurotoxic features' causing selective neuromuscular blockade of the muscles of eyes, tongue, throat and chest leading to respiratory failure in severe poisoning.

Sea snake venom. The effects are both myotoxic and neurotoxic resulting in clinical and pathological changes typical of segmental myopathic lesions in the skeletal muscles. Muscle pains may be last for several months unless treated.

CLINICAL FEATURES OF SNAKEBITE

The clinical presentation of a snakebite victim varies with the size and species of snake, the number and location of bites, and the quantity of venom injected. As many 30% of Pit viper bites and 50% of elapid bites may result in no envenomation, sometimes referred to as "dry bites". The venom channel is recessed above the tip of the fang and the venom injected may be reduced by poor penetration or glancing blows, causing venom to be lost over skin & clothing surface. The volume of the venom available to a particular snake may also be reduced by previous bites. The age and health of the victim are also important determinants in the clinical presentation. However, whether the snake is poisonous or non-poisonous and regardless of the venom injected, the commonest symptom following snakebite is 'fright' which may lead to a vasovagal episode (faint).

Usually the minority of victims who receive a venom dose large enough to cause systemic poisoning will already have signs of this by the time they seek medical help, and differentiation of viperine from elapid systemic poisoning is usually obvious from simple clinical evaluation. A persistent bloody ooze from the fang marks may suggest the presence of snake venom anticoagulant. In difficult cases the presence of pain out of proportion to the size of the wound suggests snake envenomation whereas mild pain is more normally caused nonvenomous snakes, anthropod bites (centipedes, spiders), bacterial fascilitis or myonecrosis.

Local manifestations

After envenomation local swelling starts within few minutes.

Table 2: Snake venom components and their effects			
Component	**Pit viper**	**Coral snake**	**Effect**
Enzymes			
Proteinases	Heavy	Minimal	Tissue destruction, coagulation, anticoagulation
Hyaluronidase	Moderate	Moderate	Hydrolysis of connective tissue stroma
Cholinestrase	Minimal	Heavy	Catalyzes hydrolysis of acetylcholine
PhospholipaseA	Heavy		Haemolysis may potentiate neurotoxins
Phosphomesterase	Minimal	Heavy	Unknown
Phosphodisterase	Moderate	Moderate	Hypotension
Non-enzymes			
Neurotoxins	Minimal	Heavy	Flaccid paralysis
Cardiotoxins	Minimal	Heavy	Depolarizing

Fang marks may be difficult to see. Local pain with radiation and tenderness and a small reddish wheal are first to develop, followed by oedema, swelling and the appearance of bullae, all of which can progress quite rapidly and extensively. In most viper bites paraesthesia commences around the wound, and tingling and numbness over tongue, mouth & scalp can occur. The local bite may become necrosed & gangrenous. Russell's viper has been reported to cause Raynaud's phenomenon & gangrene in the limb other than the one bitten. Secondary infection including tetanus & gas gangrene can also result. Since the venoms are largely absorbed by the lymphatics, lymphangitis may appear early. Petichae or purpura may also be present due to the anticoagulant effect of some venom. These characteristic changes are useful clinically - for example if after a known Crotalid bite the victim demonstrates no local changes over next several hours of observation, he can be released from the hospital as significant envenomation is unlikely.

In contrast Elapid snakebites are associated with minimal local changes.

Systemic manifestations

Cobra and vipers produce symptoms within a few minutes to several hours after the bite. Sea snake bites almost always produce myotoxic features with 2 hours so that the bite can be reliably excluded if no symptoms are evident within this period. Although snakes are classified into predominantly neurotoxic, hemorrhagic and myotoxic types on the basis of their venoms, each species can result in any kind of manifestations.

● Viper bites - 75% cause envenomation, 35% mild, 15% severe. Pit viper venom can involve virtually every organ system. Nausea and vomiting are common and if present early suggest severe envenomation. Weakness, sweating, fever, chills, dizziness and syncope may occur. Some patients complain of a minty, rubbery or metallic taste in their mouths with increased salivation. Tingling or numbness in the tongue, scalp, face and digits are indications of moderate to severe envenomation as are fasciculations of the face, neck, back or the bitten extremity. Systemic anticoagulation can lead to gingival bleeding, epistaxis, hemoptysis, haematuria, haemetemesis and rectal bleeding or malena. Intra-abdominal or intracranial haemorrhages may occur. Visual disturbances may result from retinal haemorrhages. There may be tachycardia or bradycardia, often accompanied by hypotension. Delayed shock may occur due to excessive blood loss and hemolysis. Severe envenomation can result in pulmonary oedema as a result of destruction of the intimal lining of pulmonary blood vessels and pooling of pulmonary blood. The venom and associated hypotension along with haemoglobin, myoglobin and fibrin deposition in renal tubules can contribute to nephrotoxicity.

● **Elapid bites**. The venom of elapid bites is primarily neurotoxic. Neurotoxic features are a result of selective d-tubocurarine like neuromuscular blockade, which results in flaccid paralysis of muscles. Ptosis is the earliest manifestation of cranial nerve dysfunction followed closely by double vision. Paralysis usually then progresses to involve muscles of swallowing, but not strictly in that order.

Generally muscles innervated by cranial nerves are involved earlier. However pupils are reactive to light until terminal stages.

The muscles of the chest are involved relatively late with the diaphragm being most resistant. Respiratory paralysis is therefore often a terminal event. Even prior to respiratory failure, airway obstruction due to vomit or secretions can result in sudden death.

Reflex activity is generally not affected and deep tendon jerks are preserved until late. Symptoms that suggest severe envenomation include repeated vomiting, blurred vision, paraesthesiae around mouth and hyperacusis (increased sensitivity to sound), headache, dizziness, vertigo and signs of autonomic hyperactivity. Tachycardia, hypotension and ECG changes may occur. Tetanic contraction of heart following a large dose of cobra venom has also been documented.

● **Sea snakes**. Muscle pain is the most common presentation. Muscle necrosis may result in myoglobinuria and severe sea snake poisoning causes myoglobinuria and respiratory failure within a few hours. Coagulaopathy is not a feature of coral snake bites.

In severe systemic poisoning following either elapid or viper bites, the electrocardiogram may show T-wave inversion and ST segment deviation. In sea snake bites, an ECG is especially valuable in detecting hyperkalemia, which can result from damage to muscles. Tall, peaked T-waves in chest leads may appear within a few hours of bite and give early warning of impending death or acute renal failure.

Unusual presentations of snake envenomation

● A species Naja nigricollis (spitting cobra) can eject venom from a distance of 6-12 feet. The venom is aimed at victim's eyes resulting in conjuctivitis and corneal ulceration. It may result into anterior uveitis and hypopion. A dull headache may persist beyond 72 hours.

● Occasionally a recently killed snake or snakes with several heads can eject venom into those handling them.

● Rarely recurrence of snake envenomation manifestations may occur hours or even days after initial good response to the antivenom. This may be due to ongoing absorption of the venom.

MANAGEMENT OF SNAKEBITE

The management of snake envenomation is controversial. It can be divided into first aid and pre-hospital care, specific antivenom therapy and the supportive therapy.

First aid and prehospital care

Reassurance and immobilisation of the affected limb with prompt transfer to a hospital are of prime importance. The application of a "constriction band" to delay the absorption and venom spread has been advocated during transit to hospital for bites to the limb. A firm, but not tight, ligature may be applied just above the bite. The tension is correct if one finger can pass between the limb and the bandage. This will impede lymphatic drainage, but not arterial or deep venous flow. It should preferably not be released until the administration of anti-snake venom. If the limb becomes edematous the band should be advanced proximally. However, the band should not be left in place for too long due to the risk of venous thrombo-embolism and distal ischaemia. An increase in local envenomation has also been reported subsequent to release of the band. Venous or arterial tourniquets are contraindicated.

The site of bite should be wiped & covered with a handkerchief or dressing. Incision and mechanical suction of the bite (intended to open the puncture wound so that suction can be more effective) may be beneficial when performed by a health care worker within a few minutes of the bite in a victim who is more than 30 to 60 minutes from hospital. The incision should be parallel to the axis of the extremity and should be only approximately 6 mm long and 3mm deep and cross cuts or multiple cuts should be avoided.

Mechanical suction ("extractor" device found in Sawyer first aid kit) is preferable to mouth suction in order to avoid wound contamination with oral flora and to prevent possible envenomation of the rescuer through breaks in their oral mucosa. Suction should be maintained for about 30-60 minutes for maximal benefit, but due care should be taken as laceration of nerves, tendons & vessels has been reported following suction by untrained rescuers.

Application of cooling measures such as ice packs or cryotherapy, at the site of bite were initially advocated but have not been observed to be effective and this practice is not now recommended.

Anti tetanus toxoid should always be given following snakebite. There is controversy about use of drugs as part of first-aid care. It has been suggested that NSAIDS (Aspirin) may be beneficial to relieve local pain but it may precipitate bleeding especially if the venom is vasculotoxic. Paracetamol and / or codeine may be useful, however there are no clear-cut recommendations for the use of sedatives.

If the snake has been killed, it should be taken to hospital, otherwise it should be left alone, since attempts to find or kill it may result in further bites. The snake, even if judged to be dead, should be handled very carefully, since decapitated heads can bite for up to one hour!

Patient assessment

Evaluation should begin with the assessment of the airway, breathing and circulatory status. Oxygen should be administered to every envenomated patient and a large bore intravenous line with normal saline or Ringer's lactate established in the unbitten limb. Cardiac monitoring and pulse oximetry, if available, is indicated. Attempts should be made to determine whether a venomous snake has actually bitten the patient, and the severity of envenomation should be assessed. (Table 3)

During the initial evaluation, several locations on the bitten extremity (at the bite site and at least two sites more proximal) should be marked and the circumferences should be measured every 15 minutes until swelling is no longer progressing and every 1-4 hours thereafter. The extremity should be placed in a well-padded splint for at least 24 hours.

Laboratory Investigations

Although lab tests are of little value in the diagnosis of snake envenomation, nevertheless they are useful for monitoring the patient and deciding about specific interventions and prognosis. They should include a full blood count, electrolytes, glucose, creatinine, serum amylase, creatinine phosphokinase (CPK), prothombin time (PT), partial thromboplastin time (PTT), fibrinogen and fibrin degradation products (FDP's). Commonly hyperkalaemia and hypoxaemia with respiratory acidosis may be seen, particularly with neuroparalysis. Urine examination can reveal haematuria, proteinuria, haemoglobinuria or myoglobinuria. Arterial blood gases and urine examination should be repeated at frequent intervals during the acute phase to assess progressive systemic toxicity.

Blood changes include anaemia, lecuocytosis (raised white cell count) and thrombocytopenia (low platelet count). The peripheral blood film may show evidence of haemolysis especially in viperine bites. Clotting time and prothrombin time may be prolonged and a low fibrinogen may be present. Blood should be typed and crossmatched on the first blood drawn from the patient, as both direct venom and anti-venom effects can interfere with later cross matching. Some specialised centers can identify species of snake involved.

Non specific ECG changes such as bradycardia and atrio-ventricular block with ST,T segment changes may be seen.

Table 3: Assessment of severity of envenomation			
No envenomation	Absence of local or systemic reactions, Fang marks +/-		
Mild envenomation	Fang marks (, moderate pain, minimal local oedema (0-15cms), erythema+, ecchymosis +/-, no systemic reactions		
Moderate envenomation	Fang marks+, severe pain, moderate local edema (15-30cms), erythema & ecchymosis +, systemic weakness, sweating, syncope, nausea, vomiting, anemia or thrombocytopenia		
Severe envenomation	Fang marks+, severe pain, severe local edema (>30cms), erythema & ecchymosis+, hypotension, parasthesia, coma, pulmonary edema, respiratory failure		
	Phosphomesteras	Minimal	Heavy	Unknown
	Phosphomesteras	Minimal	Heavy	Unknown

Recently EEG changes have also been reported in many patients of snake envenomation. They may manifest within hours of bite without any clinical features suggestive of encephalopathy.

Anti venom therapy

Anti snake venoms (ASV) are prepared by immunising horses with venom from poisonous snakes and extracting serum & purifying it. The WHO has designated the Liverpool School of Tropical Medicine as the international collaborating center for anti-venom production and testing. Anti-venoms may be species specific (monovalent) or effective against several species (polyvalent) (Table 4).

The correct use of antivenom is the most important component of the hospital care and not every bite, even with a poisonous snake, merits its use. Administration of anti-venom should be selective and based on severity of clinical symptoms. The main concern about the empirical use of antivenom is the risk of allergic reactions, its relative scarcity in some centers and the cost factor. Moreover in a study of Elapid envenomation, all victims with neuromuscular paralysis survived without receiving any anti-venom. Shamesh et al did a preliminary evaluation of the possibility of reducing the dose of anti-venom or totally avoiding it in some viper species. They concluded that about half of the bitten patients in their study did not show systemic symptoms and therefore did not require antivenom treatment. They further observed that anti-venom treatment based on systemic symptoms was effective and the dose required was also less than the fixed amount advocated for each patient, thereby reducing the incidence of serum sickness.

Administration of Antivenom

Antivenom should be given within 4-6 hours of the bite and the dosage required varies with the degree of envenomation. Serum sensitivity should be tested by injecting 0.2 ml of antivenom subcutaneously. If a severe reaction occur within 15 minutes, anti-venom is contra-indicated. Adrenaline should be readily available in a syringe for moderate reactions that may occur despite negative tests for sensitivity. Initial dose should depend upon an estimate of amount of envenomation. (Table 5) However no upper limit has been described and up to 45 vials have been successfully used in a patient! In children and small adults (body weight <40 kgs) up to 50% higher dose of ASV should be administered to neutralise the relatively higher venom concentration.

ASV is administered intravenously either in an undiluted form at a rate of not more than 1ml per minute or diluted in 500ml of IV fluid & administered as rapidly as tolerated over 1-2 hours. Additional infusions containing 5-10 vials (50-100ml) should be repeated until progression of swelling in the bitten part ceases and systemic signs & symptoms disappear. However it is not advisable to infiltrate ASV at the local site. Delayed reactions may occur following anti-venom therapy and their frequency of occurrence is proportional to the amount of anti-venom administered. Therefore all patients receiving ASV should be observed for several days.

Role of Anticholinesterase Agents

Since Elapidae snakes result in primarily neurotoxic features as a result of selective d-tubocurarine like blockade, the post synaptic toxin of the venom leads to pathophysiological changes resembling those of myasthenia gravis; This prompted some of the workers to use anticholinesterase agents such as neostigmine in addition to a conventional antivenom therapeutic regimen with dramatic results. However the use of anticholinesterase drugs

Table 4: Types of anti-venoms	
Name of Antivenom	**Species**
Polyvalent Wyeth Labs [Antivenin (cortalidae) polyvenom] United States	All North American pit vipers
King cobra antivenom	King cobra (Ophiophagus hannah)
Polyvalent Naja naja serum (common cobra) antisnake venom CRI, Kausali, India	Vipra russelli(Russel's viper Bunqarus ceruleus(common krait) Echis carinatus (saw scaled viper)
Mono specific Echis carinatus antivenom, India	Indian species
Tiger snake antivenom, Australia	Sea snakebite & Afro-Asian elapids
Green pit viper antivenom	Trimeresurus albolabris, Trimeresurus monticola
Bothrops antivenoms, Brazil	
Monospecific antivenom from South African Institute for Medical Areas (SAIMR), Northern Nigeria	Echis pyramidum leakeyi
Poly specific German & French antivenoms	

Storage of ASV: Liquid -between +20 & +80C, Lyophilized - cool & dry place.

Table 5: Dose of anti venom	
Envenomation	Dose
Mild	5 vials (50ml)
Moderate	5-10 vials (50-100ml)
Severe	10-20 vials (100-200ml) or more

alone without ASV has also been recommended. Neostigmine can be given as 50-100 (g/kg 4 hourly or as a continuous infusion. Edrophonium can also be used in dose of 10mg in adult or 0.25mg/kg in children over 2 minutes & if the response is positive then one can switch over to long acting preparations like neostigmine. However prospective studies are required to fully establish the efficacy of neostigmine with or without ASV. Glycopyrrolate 0.2 mg preceding neostigmine can be given, as unlike atropine it does not cross blood brain barrier

Supportive therapy

The patient should be moved to an appropriate are of the hospital - ICU will be required for severe envenomation. Fasciotomy should be undertaken in patients with compartment syndrome and debridement should be performed for necrotic tissue. Coagulopathies should be corrected with fresh frozen plasma and platelets. Blood transfusion should be given to replace blood loss from haemolysis & bleeding. Ventilatory support and haemodialysis may be necessary for pulmonary and renal complications due to severe envenomation. Corticosteroids are of no proven value and in fact may interfere with the action of ASV. However, corticosteroids may be used for hypersensitivity reactions to ASV. Prophylactic antibiotics are of no proven value. If infection occurs broad-spectrum cover such as ciprofloxacin and clindamycin should be used.

Intravenous immunoglobin therapy has also been used for envenomation and it may improve coagulopathy but has no effect on neurotoxicity. Certain reports on the evaluation of intravenous immunoglobin suggest that it may reduce the need for repeat antivenom therapy for envenomations associated with coagulopathy.

A compound (2-hydroxy 4-methoxy benzoic acid) isolated and purified from anatamul (Hemidesmus indicus R.Br.), an Indian herb, has also been observed to have potent anti inflammatory, antipyretic and antioxidant properties, especially against Russel's viper venom.

Analgesia should be given - opioids may be required.

Other envenomations

Scorpion venom poisoning.

There are more than 1,400 species of scorpions in the world but the number of medically important species is limited. The venom of the Bark scorpion (C. exilicauda) contains at least five distinct neurotoxins that stimulate depolarization of the neuro muscular junction & autonomic nervous system via release of acetylcholine, norepineprine & epinephrine. It may also have cardiotoxic effects.

Most stings are minor though serious envenomations can occur in children. The sting is followed by the onset of intense local pain with hyperesthesia (increased skin sensitivity to touch) but local swelling and ecchymosis are absent. Systemic symptoms, when present, reflect sympathetic, parasympathetic and neuromuscular excitation. Tachypnoea, respiratory distress, wheezing, stridor, muscle fasciculations and spasm follow initial restlessness and anxiety. There may be convulsions, paralysis and involuntary voiding of stools/urine, priapism (persistent penile erection) and anxiety. Other systemic features may include hypertension, supraventricular tachycardia and hyperpyrexia.

The majority of stings can be treated with mild analgesics & cold compresses. In the event of severe envenomatiom, the patient should be resuscitated and appropriate symptomatic treatment should be instituted. A goat-derived anti-venom is available in Arizona. Most adults can be safely treated at home, but children should always be admitted and any child less than a year old or having neurological findings should be admitted to ICU.

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GUIDELINES FOR PERIOPERATIVE STEROIDS

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Introduction

It has been about 50 years since the first case reports of perioperative shock due to secondary corticosteroid insufficiency. Since then it has been recommended that patients have adequate steroid replacement therapy to avoid perioperative haemodynamic instability. However there is a continuing debate over the amount of steroid that should be given. Some feel that only physiological amounts of steroids are necessary while others give much larger doses.

Physiology of cortisol secretion

Long term steroid therapy for chronic diseases like asthma suppresses the hypothalamic-pituitary-adrenal (HPA) axis. Studies have shown that in normal patients with major stresses like trauma or surgery the HPA axis is activated, leading to a surge in systemic cortisol. This surge continues for up to 72 hours after the insult and is thought to be protective as cortisol has a number of anti-inflammatory effects and prevents hypotension and shock. Loss of this surge may precipitate intraoperative or postoperative haemodynamic instability.

It is estimated that adults secrete 75-150mg of cortisol in response to major surgery and 50mg a day for minor surgery, and secretion parallels duration and extent of surgery[1].

Side effects of excessive steroids

The documented adverse effects of excessive corticosteroid supplementation include hyperglycaemia, immunosuppression, protein catabolism, impaired wound healing, hypertension, fluid overload, psychosis and aseptic necrosis of the femoral head. Hence it is not advisable to prescribe supraphysiological amounts of steroids when current evidence shows that physiological amounts are sufficient.

Who needs additional steroids

Studies over the last 20 years have shown that many patients on long term glucocorticoid therapy have undergone uneventful major surgery with only their usual steroid doses.

In a review of perioperative haemodynamic instability less than 1% of the cases could be attributed to glucocorticoid insufficiency. However, the problem is real and does occur[2]. The risk of anaesthetising and operating on such patients depends on the duration and severity of the operation and dose of steroids taken.

The gold standard for assessment of HPA function is the insulin tolerance test but the short synacthen test is cheaper and less unpleasant.

In experimental studies evaluating the HPA axis almost all patients taking less than 10mg prednisolone have been shown to have a clinically normal response to HPA testing[1]. These patients do not need additional steroid cover other than their usual steroid dose. This should be taken preoperatively and continued as soon as oral intake is possible.

It is recommended that patients on long term steroids equivalent to more than 10mg prednisolone daily (or who have received

Table 1: Suggested steroid treatment regimen[1]	
PATIENTS WHOSE HAVE RECEIVED A REGULAR DAILY DOSE OF MORE THAN 10MG PREDNISOLONE OR EQUIVALENT IN THE LAST THREE MONTHS	
Minor Surgery (*hernias, hands*)	25mg Hydrocortisone at induction
Moderate Surgery (*hysterectomy*)	Usual pre-op steroids + 25mg Hydrocortisone at induction +100mg hydrocortisone/day
Major Surgery (*major trauma, prolonged surgery, or surgery where there is delayed oral intake*)	Usual pre-op steroids + 25mg Hydrocortisone at induction +100mg hydrocortisone/day for 2-3 days Resume normal oral therapy when gastrointestinal function has returned
ALL OTHER PATIENTS - no additional steroids required.	

Table 2: Equivalent drug doses (British National Formulary, March 2003)	
Prednisolone 10 mg is equivalent to	Betamethasone 1.5 mg
	Cortisone acetate 50 mg
	Dexamethasone 1.5 mg
	Hydrocortisone 40 mg
	Deflazacort 12mg
	Methylprednisolone 8 mg

An infusion is preferable as it avoids large increases caused by bolus injection[1]. However infusion may present practical difficulties. Some studies have shown that one quarter the daily dose administered six hourly may be adequate.

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such a dose within the last 3 months) receive a physiological replacement regimen. Alternatively adrenal suppression should be excluded by preoperative biochemical testing. However, in many hospitals, it may not be practical to conduct such assessments. In such situations, steroid cover will be appropriate according to the regimen in Table 1.

The regimen of replacement is based on the physiological requirements of stressed controls in human studies. In patients with proven adrenalcortical insufficiency a low dose physiological substitution regimen results in circulating cortisol values greater than in normal patients and is sufficient to prevent intra-operative haemodynamic instability[3].

ANAESTHESIA FOR TRANSURETHRAL RESECTION OF THE PROSTATE (TURP)
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INTRODUCTION

TURP is a cystoscopic procedure used to alleviate the symptoms of bladder outflow obstruction, usually caused by benign prostatic hypertrophy (BPH). BPH affects 50% of males at 60 years and 90% of 85-year-olds and so TURP is most commonly performed on elderly patients, a population group with a high incidence of cardiac, respiratory and renal disease. The mortality rate associated with TURP is 0.2-6%, with the commonest cause of death being myocardial infarction. Safe anaesthesia depends on the detection and optimisation of co-existing diseases, and on weighing up the relative risks and benefits of regional and general anaesthesia for each patient.

The operation is performed under direct vision using a diathermy current passed through a loop of wire at the tip of a resectoscope, which is inserted into the bladder through the patient's urethra. This enables the hypertrophied prostate to be resected in pieces and washed out using an irrigation solution. The most commonly used irrigation fluid is 1.5 % glycine solution, which has the advantages of being optically clear and non-electrolytic (and therefore does not conduct electric current). It has an osmolarity of 200mOsm/L which is much lower than that of blood, and large amounts of this hypotonic irrigation fluid, required to facilitate the procedure, may be absorbed systemically through the vascular prostate bed. This may cause several serious complications, which are discussed in this article.

PREOPERATIVE ASSESSMENT

Reduced functional reserve should be quantified, and the presence of any organ failure noted. If the patient has ongoing medical problems that can be improved before surgery, then TURP may need to be delayed.

A decision can then be made with the patient between regional and general anaesthesia, based on consideration of the advantages and disadvantages of each technique in their particular case. For some patients the risks of anaesthesia and surgery may outweigh the potential benefits of an elective procedure such as TURP.

History and examination

- **Cardiovascular** - Hypertension, ischaemic heart disease (IHD) and arrhythmias (particularly atrial fibrillation) are common. Patients with recent onset or poorly controlled heart failure have the highest perioperative mortality. Major risk factors for IHD (hypertension, diabetes, smoking, hypercholesterolaemia and family history) will raise the likelihood of silent perioperative myocardial ischaemia.

- **Respiratory** - Marked decrease in functional ability (e.g. inability to climb one flight of stairs) suggests severe disease. Inability to lie flat because of dyspnoea from cardiac or respiratory causes will make awake spinal anaesthesia poorly tolerated.

- **Neurological** - Confused patients may be not lie still during spinal anaesthesia.

- **Musculo-skeletal** - Degenerative changes in the vertebral column may make subarachnoid block (SAB) technically difficult. Arthritic joints or joint replacements are susceptible to damage or dislocation when the patient's legs are placed in the lithotomy position for the procedure.

- **Renal impairment** may occur due to obstructive uropathy

- **Airway** - Even if SAB is planned perform a full anaesthetic assessment (e.g. anticipated airway difficulties) in case the regional technique fails or is inadequate.

- **Drug history** - A high proportion of elderly patients take cardiovascular medications. Beta-blockers suppress the compensatory tachycardic response to hypotension associated with SAB or haemorrhage, but should generally be continued for prevention of perioperative myocardial ischaemia. ACE-inhibitors limit the renin-angiotensin mediated response to hypovolaemia that may be further impaired by SAB, and most anaesthetists omit them for 24 hours preoperatively. Alpha-blockers are commonly encountered as first-line medical treatment for BPH. The combined hypotensive effects of these drugs may precipitate severe hypotension after SAB. Warfarin has implications for both the anaesthetist (regarding SAB) and the surgeon (intra- and postoperative haemorrhage). If the INR is greater than 1.4 the procedure should be postponed until the INR is acceptable.

Investigations

Most patients are elderly and should have as routine:

- Full blood count or haemoglobin level

- Creatinine and electrolytes - this will detect renal impairment or overt renal failure, commonly secondary to obstructive uropathy.

- ECG for symptomatic patients, and routinely over 60 years

- Group and save - consider cross-matching blood for anaemic patients and those suspected of having large prostates on examination or ultrasound scan.

Other tests may be indicated in particular circumstances:

- Clotting studies (prothrombin time if on warfarin)

- Blood gas and pulmonary function tests (severe respiratory disease)

- Chest radiograph (worsening cardiac or chest disease / suspicion of metastases)

- Urinalysis (for glucose, protein, blood, white blood cells)

- Blood glucose

● Test for sickle cell disease or haemoglobinopathies in patients of African or Mediterranean extraction respectively.

CHOICE OF ANAESTHETIC

In the UK, 75% of TURPs are carried out under regional anaesthesia. Although regional anaesthesia in an awake patient has theoretical advantages, such as earlier detection of TUR syndrome (see below), the procedure can be equally successfully accomplished using a general anaesthetic technique. Short-term morbidity and mortality and long-term outcome are similar irrespective of the technique used. The decision is made after consideration of the individual's medical status and detailed discussion of the relative advantages and disadvantages of each technique.

The advantages of the regional technique include:

● Early detection of complications such as TUR syndrome and bladder perforation

● Possible reduced blood loss, requiring fewer transfusions

● Avoids effects of general anaesthesia on pulmonary pathology

● Good early post-operative analgesia

● Reduced incidence of post-operative DVT/PE

● Lower cost

The advantages of general anaesthesia are:

● Patients with chest disease may not tolerate lying flat or be able to suppress their cough

● No time constraints. Although the procedure should be kept as short as possible - see later.

● May be less haemodynamically challenging than SAB in patients with cardiac problems such as aortic stenosis (and other fixed output states) and IHD

● Allows better control of CO_2, which may reduce bleeding from the prostatic bed

● Patient preference.

Anaesthetic Technique

Premedication

Consider relevant premedication if indicated:

● *Analgesics* - give pre-emptive analgesia (paracetamol +/- NSAIDs if not contra-indicated)

● *Anxiolytics* - consider a short-acting benzodiazepine if clinically indicated. In the elderly these drugs may result in postoperative confusion.

All patients should be fully monitored with blood pressure, pulse oximetry and ECG for SAB, including capnography, volatile agent levels, and airway pressure for general anaesthesia. A reliable, large-bore intravenous cannulae (14-16G) should be placed.

Subarachnoid block / spinal anaesthesia. (See Update 12)

● Check for any contra-indications to SAB (see table).

Table 1. Contra-indications to SAB include:

● Patient refusal

● Infection - either localised or generalised (e.g. sepsis)

● Raised intracranial pressure

● Hypovolaemia or shock from any cause

● Coagulopathy - platelet count < 80-100 or INR < 1.5

● Pre-existing neurological disease - postoperative exacerbation of the disease may be erroneously attributed to the SAB

● A fluid preload with 500-1000ml of warmed saline 0.9% or Hartmann's is commonly given. Patients are likely to be dehydrated for a number of reasons including fasting and use of diuretics. Preloading assists compensation of the spinal-induced vasodilation and hypotension, and provides a small sodium load to counter the hyponatraemia that often occurs with TURP (discussed later).

● A confirmed block to at least T10 (level of the umbilicus) is required prior to the start of surgery. 2.5 to 3ml of plain or heavy bupivacaine 0.5% reliably achieves this, and provides up to 3 hours of dense motor and sensory blockade. This level of block does not usually cause severe hypotension, but vasopressors (ephedrine 3- 6mg, or metaraminol 0.5-1mg) should be immediately available. As a general guide, use ephedrine if the pulse is less than 60 per minute, and metaraminol if the pulse is over 60 per minute.

● Heavy lignocaine 5% 1.2-1.4ml can also be used, although the duration of block is unlikely to be reliable after 90min. Do not use lignocaine from multi-dose vials as these contain potentially harmful preservatives. Adding adrenaline 0.2mg to hyperbaric lignocaine will extend the block duration.

● Isobaric plain 2% lignocaine (with 0.2mg adrenaline to extend the block duration) in a dose of 2-2.5mL is also an appropriate choice.

● Consider intra-operative sedation for anxious or confused patients (e.g. IV midazolam 0.5-1mg as needed), but bear in mind that confusion may also be an early manifestation of the TUR syndrome (see later).

● Ideally a thermometer, warming blanket and fluid warmer should be available for the detection and prevention of hypothermia caused by the infusion of cold fluids and the effects of the irrigation fluid.

● All patients should be given supplementary oxygen.

General Anaesthesia

● Either a spontaneously breathing technique using a facemask or laryngeal mask, or a relaxant technique is appropriate, depending on the patient.

● Elderly patients are very susceptible to the hypotensive effects of induction and maintenance agents and have reduced requirements for volatile anaesthetic agents.

● Analgesic requirements can usually be met with pre-operative paracetamol and NSAID and increments of an opioid such as fentanyl, alfentanil or morphine. Further morphine in recovery is seldom required.

● Remember to consider the patient's renal function when using drugs that are excreted renally (e.g. morphine and non-depolarising neuromuscular blocking drugs other than atracurium).

Other considerations

● Following the initial fluid infusion, i/v fluids should be given to replace blood loss. Since irrigation fluid is continually absorbed during the procedure, maintenance fluids are not required.

● Urologists often request antimicrobial prophylaxis to cover the gram-negative bacteraemia. A single intravenous dose of gentamicin 3- 4mg/kg is suitable.

● Where available, consider invasive blood pressure monitoring in patients with severe cardiac disease.

INTRA-OPERATIVE COMPLICATIONS OF TURP

Hypotension

The major complication related to anaesthesia is hypotension following the sympathetic blockade of SAB. This is uncommon with blocks extending to T10, but inadvertently high blocks may cause resistant hypotension and bradycardia is seen if the cardioaccelerator fibres (from T1-4) are blocked. Treatment is with fluid, vasopressors and/or inotropes as detailed above.

TUR Syndrome

This occurs in up to 8% of cases in a mild form, but is severe in 1-2% of cases. Resection of prostatic tissue opens an extensive network of venous sinuses, which allows the irrigation fluid to be absorbed into the systemic circulation. Features may develop peroperatively or in the recovery room.

The volume of fluid absorbed depends on:

● the duration of the procedure (associated with large gland)

● the height of the irrigation fluid bag above the patient (increased height implies increased hydrostatic pressure driving the fluid intravenously)

● the vascularity of the diseased prostate

An average of 10 to 30mL of fluid is absorbed per minute of resection time, amounting to up to 1800ml per hour. The glycine-containing irrigation solution is slightly hypo-osmotic (200mosm/l) and therefore the classical triad of features that make up TUR syndrome are:

1. **Dilutional hyponatraemia**. Encephalopathy and seizures may develop when the sodium concentration falls below 120mmol/l. Cerebral oedema may occur.

2. **Fluid overload**. This causes pulmonary oedema and cardiac failure.

3. **Glycine toxicity**. This inhibitory neurotransmitter causes depression of the level of consciousness and visual impairment at toxic levels.

Symptoms and signs of TUR Syndrome:

● tachycardia

● nausea and vomiting - caused by hyponatraemia and cerebral oedema

● confusion / disorientation - hyponatraemia and cerebral oedema

● hypertension (fluid overload), then hypotension (cardiac insufficiency)

● transient blindness - glycine toxicity

● angina

● dyspnoea and hypoxia caused by pulmonary oedema

● cardiovascular collapse and arrhythmias (VT/VF)

● convulsions

● coma (Na <100mmol/l)

If the patient is under general anaesthesia all of the symptoms and some of the signs are masked, and only unexplained tachycardia and hypotension may be present.

Factors which increase the risk of TUR syndrome

● pre-existing hyponatraemia or pulmonary oedema

● prostate size larger than 60-100g

● inexperienced or slow surgeon

● procedures longer than 1 hour

● hydrostatic pressure > 60cm H_2O (height of bag above patient)

● reduced venous pressure (dehydration)

● use of large volumes of hypotonic intravenous fluids such as 5% dextrose

It is difficult to accurately assess the volume of irrigation fluid that has been absorbed. Early detection of the problem is therefore dependent on being aware of the high-risk situation and continuous observation for the signs and symptoms of TUR syndrome. Attempts should be made to keep the surgical time below one hour. In some countries, the irrigation fluid has a small amount of alcohol added, which allows an estimation of absorbed fluid from an alcohol measurement on the patients expired breath.

Investigations

A low serum sodium can confirm the diagnosis. Levels below 120 mmol/L are invariably symptomatic and a rapid fall is more likely to produce symptoms. ECG manifestations of hyponatraemia such as QRS widening, ST segment elevation and T wave inversion usually only occur below 115mmol/l. Hyperammonaemia is a common finding, being a by-product of glycine metabolism. A low serum osmolality and high anion gap (see Table 2), caused by the presence of glycine may be present.

Management

● Initial management should follow the airway, breathing and circulation (ABC) guidelines. Awake patients may need to be sedated and ventilated, whilst anaesthetised patients with mask airways may need intubation and positive pressure ventilation.

● Inform the surgeon and terminate surgery as soon as any bleeding points have been coagulated.

Table 2: Anion Gap

The term 'anion gap' is a diagnostic concept that is used to describe the concentration of all unmeasured anions in the plasma. The following simple formula is used to calculate the anion gap:

$$\text{Anion gap} = [Na^+] - [Cl^-] - HCO_3^-]$$
$$= 140 \quad - 105 \quad - 25$$
$$= 10$$

The normal range is 4 -12. In normal circumstances, negatively charged plasma proteins (such as albumin) make up the majority of this value. But any increase in unmeasured anions (such as glycine) will cause a 'raised anion gap'. A common example of such a situation is a metabolic acidosis, during which acidic anions such as lactates and ketones (which are not routinely measured) accumulate thus producing a surplus of unmeasured anions and a 'raised anion gap'.

Its major use is to signal the presence of an acidosis, and to differentiate between causes of a metabolic acidosis. The anion gap *increases* in acidosis caused by accumulation of organic anions such as in a lactic acidosis and ketoacidosis. It remains unchanged in acidosis due to loss of bicarbonate or increase in Cl$^-$.

● Initial management of fluid overload and hyponatraemia involves stopping IV fluids and commencing a fluid restriction (e.g. 800ml/24 hours can achieve a rise in the sodium level of up to 1.5mmol/l/24 hours). Give frusemide 40mg IV to promote a diuresis.

● Hyponatraemia causing encephalopathy requires more rapid correction than that achieved by fluid restriction and diuresis alone. Ideally these patients should be closely monitored on an intensive care unit. Hypertonic saline solutions (1.8%, 3% or 5%) should be used to increase the serum sodium level by about 1 mmol/l/hour, not exceeding an increase of 20mmol/l in the first 48 hours of therapy. Sodium levels should be checked every few hours. Therapy with hypertonic saline should be stopped when symptoms cease or the sodium level reaches 124-132mmol/l. Rapid correction has been implicated as a cause of central pontine myelinolysis, which causes irreversible brain damage.

● Convulsions should be acutely treated with a benzodiazepine (e.g. diazepam 5-10mg) or small doses of thiopentone (25 - 100mg). In the presence of intractable seizures, the sodium level may be corrected more rapidly at a rate of up to 8-10mmol/l/hour for the first 4 hours of therapy.

Haemorrhage

Blood loss is very difficult to quantify. The amount lost is related the mass of gland excised, the duration of the procedure and the experience of the operator. In practice, clinical judgement based on duration of procedure, mass of prostate excised, the patient's vital signs and communication with the surgeon are invaluable. If available, serial haematocrit levels are the most sensitive indicators of the need for transfusion. Haemoglobin and electrolytes should be measured the following day to exclude sub- clinical anaemia and hyponatraemia.

Severe blood loss during TURP occurs in less than 1% of cases. This may be the result of clotting abnormalities caused by the release of tissue plasminogen activator from the prostate and is possibly more common in malignant prostates. Anti-fibrinolytics such as intravenous tranexamic acid and aprotinin can be used to minimise active blood loss.

Bladder Perforation

This complicates about 1% of cases of TURP. Most perforations are extraperitoneal and result in suprapubic, inguinal or peri-umbilical pain in the awake patient. The surgeon may notice reduced return of irrigation fluid from the bladder. Intraperitoneal perforation is far less common, but more serious. In these cases the abdominal pain is generalised, and the patient may complain of shoulder-tip pain. Pallor, sweating, peritonism, nausea and vomiting, and associated hypotension may be present, depending on the size of the perforation. Perforation may present as sudden, unexpected hypotension under general anaesthesia. Management consists of immediate laparotomy and correction of the defect.

Hypothermia

The additive effects of general anaesthesia, the use of room-temperature IV fluids and large volumes of irrigation fluids leave elderly patients hypothermic. All irrigation fluid should be warmed to body temperature prior to use. Post-operative shivering can cause massively increased myocardial oxygen requirements, reduction in cardiac output and a coagulopathy.

Bacteraemia and sepsis

In as many as 6-7% of patients, a septicaemic picture may develop. Septic shock following TURP is rare but has a mortality rate up to 75%. Antimicrobial prophylaxis with a single dose of gentamicin 3 - 4mg/kg on induction is appropriate. Cephalosporins may also be used.

Positioning

The lithotomy position may cause nerve compression (especially involving the common peroneal nerve from pressure effects exerted by the stirrups), dislocation of hip prostheses, compartment syndrome in the lower legs and respiratory compromise in patients with pre-existing lung disease (reduction of functional residual capacity.)

CASE HSTORY - TURP Syndrome.

A 75 year old hypertensive male with a history of stable angina presented for TURP. Preopertaive assessment revealed a good effort tolerance with no symptoms of cardiac failure, and a history of smoking 20 cigarettes/day for 30 years. Blood results, ECG and chest X-ray were unremarkable.

In the operating room, routine monitoring was commenced, and a spinal anaesthetic inserted after a preload of 500mL saline. Oxygen was given via a Hudson mask. Surgery began after the block was confirmed to be at T8 level.

Sixty minutes into the procedure, the patient complained of nausea and was given ondansetron 4mg IV. The heart rate was noted to have increased to 106bpm. Blood pressure remianed within normal limits. After another 15 minutes, the patient became anxious, pulled his oxygen mask off and tried to get off the operating table. Oxcygen saturation levels dropped quickly without oxygen. An urgent blood gas showed pH 7.33, pCO_2 5.6, pO_2 8.8, BE-2.8, Na 109mmol/L, Hb 9.2g/dL. A diagnosis of TURP syndrome was made on the grounds on hyponatraemia. Severe respiratory distress ensued, with the patient unable to maintain adequate oxygen saturation on high flow oxygen. Pulmonary oedema was diagnosed clinically. The patient was intubated and ventilated and surgery terminated. Transfer to the ICU was arranged urgently and frusemide 40mg i/v was given.

On arrival in ICU, the patient's temperature was 33.8 degrees. Arterial and central venous catherters were inserted, and re-warming with a hot air blower commenced. CXR showed an enlkarged heart with pulmonary oedema. The hyponataemia was treated with 3% saline via the CVP line at an initial rate of 100mL/hr. One hour later the sodium level measured 116mmol/L. The 3% saline infusion was changed for a 1.8% saline infusion at 50mL/hr. Within 24 hrs the sodium level stabalised at 127mmol/L. Pulmonary oedema resolved with frusemide boluses. After 36 hours on the ventilator, blood gases showed pO_2 = 12.6 on 30% oxygen and he was extubated uneventfully. Forty-eight hours after admission to ITU the sodium level was 132mmol/L. The patient was discharged to the ward where he made a full recovery.

Learning point

- Rapid management of the patient's ABC allowed immediate treatment of the TUR syndrome

- Surgery was completed as soon as the complication developed

- Hypertonic saline was indicated for the very low sodium level associated with CNS signs.

- The hypertonic saline was used carefully with repeated estimates on the serum sodium being performed.

Erection

This may occur as a result of surgical stimulation when anaesthesia is insufficiently deep and makes cystoscopy technically difficult. The erection usually subsides with deepening of anaesthesia, although low-dose ketamine may be tried if it persists.

POST-OPERATIVE COMPLICATIONS

Hypothermia, hypotension, haemorrhage, septicaemia and signs and symptoms of TUR syndrome can all present in the recovery room. Patients may become acutely hypotensive after lowering their legs from the lithotomy position because the reduction in venous return. Persisting hypotension that is unresponsive to fluid therapy, in the absence of excessive blood loss, may indicate bladder perforation. The symptoms of this may be masked by residual sub-arachnoid block.

Bladder spasm is a painful, involuntary contraction of the bladder caused by stimulation of the bladder neck by the indwelling catheter. The flow of irrigation fluid via the catheter often reduces, preventing the bladder from draining completely and aggravating the pain. Low-doses of a benzodiazepine such as diazepam (2.5-5mg i/v), is often effective in relieving the spasm. Hyoscine butylbromide (Buscopan) 20mg i/v slowly may also be tried.

Clot retention. Bladder clot may block the catheter causing painful distension of the bladder. If the clot cannot be dislodged with aggressive bladder washouts using the three-way tap on the catheter, it may be necessary to insert a supra-pubic catheter.

ALTERNATIVE TECHNIQUES

Open prostatectomy is the operation of choice when the prostate is either malignant, or is considered too large (more than 100g) to be removed safely transurethrally. It is performed either by a retropubic (more common) or suprapubic approach through a Pfannenstiel incision. Blood loss is usually greater than in TURP and cross-matched blood should be readily available. Epidurals provide the best post-operative analgaesia. Duration of procedure, hospital stay, complication rates and recovery period are all greater for open prostatectomy, making TURP a more preferable option.

Laser prostatectomy is a new technique that uses a Holmium-YAG laser to resect the prostate instead of a diathermy current. Bleeding complications are thought to be less than with a normal resectoscope.

SUMMARY

● TURP is a procedure carried out on a predominantly elderly population with a higher incidence of coexisting disease. Consequently anaesthetising for the procedure may present a challenge to the anaesthetist, and carries a mortality risk of 0.2-6%.

● A thorough preoperative assessment is important in detecting at-risk patients, and helping to choose your anaesthetic technique. SAB is widely considered the most suitable technique for TURP, although GA has a similar morbidity and mortality profile.

● Subarachnoid block to T10 provides excellent anaesthesia without notable hypotension

● TUR syndrome is a rare but potentially fatal complication of TURP. Early recognition and prompt treatment are essential.

● Blood loss is difficult to quantify and may be significant. Close attention to the patient's clinical state and communication with the surgeon are vital.

Further Reading

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AN INTRODUCTION TO MECHANICAL VENTILATION IN THE INTENSIVE CARE UNIT
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Indications for mechanical ventilation

Categories of mechanical ventilation
- Types of ventilation
- Modes of ventilation

Initiating mechanical ventilation
- Ventilator settings
- Optimizing oxygenation and carbon dioxide elimination
- Sedation

Tracheostomy in the ICU

Trouble shooting
- Low tidal volumes or high airway pressure
- 'Fighting' the Ventilator

Weaning
- Indications to wean
- Modes of Weaning
- Failure to wean

One of the main functions of an Intensive Care Unit (ICU) is the provision of advanced respiratory support. An understanding of the indications and types of mechanical ventilation is therefore essential for anyone working in this environment.

INDICATIONS FOR MECHANICAL VENTILATION

The main indication for mechanical ventilation is respiratory failure. However, other clinical indications include a prolonged postoperative recovery, altered conscious level, inability to protect the airway or exhaustion when the patient is likely to proceed to respiratory failure. The aim of mechanical/artificial ventilation is to improve gas exchange, to reduce the work of breathing and to avoid complications while maintaining optimal conditions for recovery. Whatever the indication for respiratory support, the underlying condition of the patient must be reversible, otherwise subsequent weaning may not be possible.

Respiratory failure

This is the primary indication for respiratory support. It occurs when pulmonary gas exchange is sufficiently impaired to cause hypoxaemia with or without hypercarbia. The causes of respiratory failure are diverse and the problem may occur due to disease at the alveolar / endothelial interface (eg pulmonary oedema) or in the respiratory pump mechanism resulting in inadequate minute ventilation (eg flail segment accompanying fractured ribs).

Criteria for starting mechanical ventilation are difficult to define and the decision is often a clinical one. Indicators include:
- Respiratory rate >35 or <5 breaths/ minute
- Exhaustion, with laboured pattern of breathing
- Hypoxia - central cyanosis, SaO_2 <90% on oxygen or PaO_2 < 8kPa
- Hypercarbia - $PaCO_2$ > 8kPa
- Decreasing conscious level
- Significant chest trauma
- Tidal volume < 5ml/kg or Vital capacity <15ml/kg

Causes of Respiratory Failure

Inadequate gas exchange
- Pneumonia, pulmonary oedema, acute respiratory distress syndrome (ARDS)

Inadequate breathing
- Chest wall problems eg fractured ribs, flail chest
- Pleural wall problems eg pneumothorax, haemothorax
- Respiratory muscle failure eg myasthenia gravis, poliomyelitis, tetanus
- Central nervous system depression eg drugs, brain stem compression

Obstructed breathing
- Upper airway obstruction eg epiglottitis, croup, oedema, tumour
- Lower airway obstruction eg bronchospasm

Other indications for ventilation

Patients in this category are ventilated to assist in the management of other, non-respiratory conditions and may include:
- Control of intracranial pressure in head injury
- Airway protection following drug overdose
- Following cardiac arrest
- For recovery after prolonged major surgery or trauma

TYPES OF MECHANICAL VENTILATION

The most commonly used type of artificial ventilation is intermittent positive pressure ventilation (IPPV). The lungs are intermittently inflated by positive pressure generated by a ventilator, and gas flow is delivered through an endotracheal or tracheostomy tube. Tracheal intubation is usually achieved by the oral route although nasal intubation may be better tolerated by the patient during prolonged ventilation.

Although more secure, nasotracheal intubation is technically more challenging and has a higher incidence of bleeding and infective complications such as sinusitis.

Tracheal intubation not only allows institution of IPPV, but also reduces dead space and facilitates airway suctioning. However, it is also possible to deliver positive pressure ventilation to cooperative patients in a non-invasive manner through a tight-fitting face or nasal mask (NIPPV).

In general, there are two main types of ventilators commonly in use in ICU - those that deliver a preset tidal volume and those that deliver a preset inspiratory pressure during each inspiration. Modern ventilators allow different modes of ventilation and the clinician must select the safest and most appropriate mode of ventilation for the patient.

Types of Ventilation

● Volume-cycled ventilation occurs when the ventilator delivers a preset tidal volume regardless of the pressure generated. The lung compliance (stiffness) of the lungs determines the airway pressure generated, so this pressure may be high if the lungs are stiff, with the resultant risk of barotrauma (rupture of the alveoli resulting in pneumothoraces and mediastinal emphysema).

● Pressure-preset ventilation is where the ventilator delivers a preset target pressure to the airway during inspiration. The resulting tidal volume delivered is therefore determined by the lung compliance and the airway resistance.

Modes of ventilation

● Controlled Mechanical Ventilation (CMV). Ventilation with CMV is determined entirely by machine settings including the airway pressure/tidal volume, respiratory rate and I:E ratio. This mode of ventilation is not often used in ICU as it does not allow any synchronisation with the patient's own breathing. As a consequence, CMV is not well tolerated and patients require heavy sedation or neuromuscular blockade to stop them 'fighting' the ventilator, thereby resulting in inefficient gas exchange. CMV is normally used in theatre when the patient is receiving a full general anaesthetic to optimise surgical conditions.

● Assisted Mechanical Ventilation (AMV). There are several different modes of ventilation designed to work with the patients' own respiratory effort. The patient's inspiratory effort is detected and triggers the ventilator to 'boost' the inspiratory breath. These modes have two important advantages; firstly they are better tolerated by the patient and so reduce the requirement for heavy sedation, and secondly they allow patients to perform muscular work throughout the breath, thereby reducing the likelihood of developing respiratory muscular atrophy. The ventilator-assisted breaths can be supported either by a preset inspiratory pressure or by a preset tidal volume. There are several variations of assisted ventilation.

Intermittent mandatory ventilation (IMV) is a combination of spontaneous and mandatory ventilation. Between the mandatory controlled breaths, the patient can breathe spontaneously and unassisted. IMV ensures a minimum minute ventilation, but there will be variations in tidal volume between the mandatory breaths and the unassisted breaths.

Synchronised intermittent mandatory ventilation (SIMV). With SIMV, the mandatory breaths are synchronised with the patient's own inspiratory effort which is more comfortable for the patient.

Pressure-support ventilation (PSV) or Assisted spontaneous breaths (ASB). A preset pressure-assisted breath is triggered by the patient's own inspiratory effort. This is one of the most comfortable forms of ventilation. The preset pressure level determines the level of respiratory support and can be reduced during weaning. There are no mandatory breaths delivered, and ventilation relies on the patient making some respiratory effort. There is, however, no back up ventilation should the patient become apnoeic, unless this mode is combined with SIMV.

Positive End Expiratory Pressure (PEEP) is used with all forms of IPPV. A positive pressure is maintained during expiration expanding underventilated lung, and preventing collapse of the distal airways. This results in improved arterial oxygenation. However, PEEP causes a rise in intrathoracic pressure and can reduce venous return and so precipitate hypotension, particularly in hypovolaemic patients. With low levels of PEEP (5-10cmH$_2$O), these effects are usually correctable by intravenous volume loading.

Continuous Positive Airway Pressure (CPAP) is effectively the same as PEEP, but in spontaneously breathing patients.

Initiating Mechanical Ventilation

When initiating artificial ventilation the aim is to provide the patient with a physiological tidal volume and ventilatory rate adapted to meet the patient's underlying condition.

Initial ventilator settings:	
FiO$_2$	1.0 initially but then reduce
PEEP	5 cmH$_2$O
Tidal volume	7-10 ml/kg
Inspiratory pressure	20 cmH$_2$O (15cmH$_2$O above PEEP)
Frequency	10 - 15 breaths per minute
Pressure support (ASB)	20 cmH$_2$O (15cmH$_2$O above PEEP)
I:E Ratio	1:2
Flow trigger	2 l/min
Pressure trigger	-1 to -3 cmH$_2$O
'Sighs'	Nil - formerly thought to prevent atelectasis, but no longer considered effective

These settings should be titrated against the patient's clinical state and level of comfort.

OPTIMIZING OXYGENATION

When settling a patient on the ventilator, it is good practice to initially set FiO$_2$ at 1.0 and then wean rapidly to a FiO$_2$ adequate

to maintain SaO_2 of >93%. FiO_2 of greater than 0.6 for long periods should be avoided if possible because of the risk of oxygen-induced lung damage.

Strategies to improve oxygenation (other than to increase FiO_2) include increasing the mean airway pressure by either raising the PEEP to $10cmH_2O$ or, in pressure-preset ventilation modes, by increasing the peak inspiratory pressure. However, care should be taken to avoid very high inflation pressures (above $35cmH_2O$) as this may cause barotrauma to the lungs.

More complex strategies to improve oxygenation may be required in severely hypoxic patients eg acute respiratory distress syndrome (ARDS) or acute lung injury from a variety of causes. In severe hypoxia, it may be possible to improve oxygenation by increasing the PEEP further to 15 cmH_2O (or above) and using small (6-8mls/kg) tidal volumes more frequently. However, this may cause a reduction in blood pressure and may be poorly tolerated by the patient requiring intravenous fluid loading and inotropic or vasopressor therapy.

Another strategy is to prolong the inspiratory time. Normal inspiratory to expiratory ratio is 1:2 but oxygenation may be improved if this ratio is changed to 1:1 or even 2:1. However, these alterations are often not well tolerated by the patient who may require heavy sedation. Not infrequently, due to a reduced minute volume the $PaCO_2$ may rise. This is not usually a problem provided the patient does not have raised intracranial pressure and the arterial pH is above 7.2. In some patients this technique is used deliberately "Permissive Hypercapnia".

In severe ARDS the patient can be repositioned and ventilated in the prone (face down) position. This may improve oxygenation by re-expanding collapsed alveoli and improving the distribution of blood perfusion in the lung relative to ventilation. In this position, patient monitoring and care is obviously difficult, and this approach should be undertaken with careful monitoring and care.

Optimising carbon dioxide elimination

Carbon dioxide elimination is improved by increasing minute ventilation either by increasing the tidal volume or the respiratory rate.

Sedation (see page 000)

Most patients require sedation in order to tolerate the endotracheal tube. Ideally, only light sedation should be given so that the patient can understand and cooperate with ventilation as well as continue to make some respiratory effort reducing the risk of respiratory muscular atrophy.

PROBLEMS DURING MECHANICAL VENTILATION

"Fighting the ventilator"

When the patient starts to breathe out of phase with the ventilator or becomes restless or distressed during IPPV, there is a fall in the delivered tidal volume due to a rise in respiratory resistance. This results in inadequate ventilation and hypoxia. There are a number of causes including:

- Patient factors - Breathing against the ventilators inspiratory phase, breath holding and coughing.

- Decreased pulmonary compliance - pulmonary pathology, including oedema or infection and pneumothorax.

- Increased airway resistance - bronchospasm, aspiration, excess secretions

- Equipment - ventilator disconnection, leak, failure. ET tube blocked, kinked, dislodged

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Management of patient 'fighting' the ventilator

- Is the patient hypoxic? If yes - follow ABC:

   - Is the endotracheal tube patent and correctly positioned? Reintubate if necessary.

   - Give 100% O_2 by manual ventilation via self-inflating bag

   - Check chest expansion is adequate

   - Auscultate chest to assess bilateral air entry

   - Check heart rate and blood pressure

   - Check ventilator and apparatus for disconnection/leak/failure

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Diagnosing the problem

- High airway pressure due to blocked ET tube.

   - Patient may be biting - insert oral airway & sedate patient

   - Blocked with secretions - suction with catheter +/- 5mls saline flush. Reintubate if necessary

   - ET tube overinserted into right main bronchus - pull back tube

- High airway pressure due to intrapulmonary factors

   - Bronchospasm? (Expiratory and inspiratory wheeze). Ensure ET tube not overinserted stimulating the carina. Give bronchodilators

   - Pneumothorax, haemothorax, lung collapse or pleural effusion? (Unequal chest movements and breath sounds). Chest Xray and treat appropriately.

   - Pulmonary oedema? (Blood stained frothy secretions & crepitations). Diuretics, treat cardiac failure or arrhythmias, suction tube.

- Sedation/Analgesic Factors

   - Hyperventilating due to hypoxia or hypercarbia (cyanosis, tachycardia, hypertensive and sweating). Increase FiO_2 and the mean airway pressure with PEEP. Increase minute ventilation (if hypercarbic).

   - Coughing, discomfort or pain (raised HR & BP, sweating & grimacing). Look for causes of discomfort eg endotracheal tube irritation, full bladder, pain.

Review analgesia and sedation. Change ventilation mode to one better tolerated eg SIMV, PSV. Neuromuscular blockade - only if all other options explored.

Weaning

There are a number of complications associated with mechanical ventilation, including barotrauma, pneumonia and decreased cardiac output. For these reasons, it is essential to discontinue ventilatory support as soon as the patient improves.

Weaning is indicated when the underlying condition is resolving. Many patients are ventilated for a short period or time, for example those recovering from major surgery. Others undergoing many days of ventilation (eg ARDS). During long periods of prolonged ventilatory support, the respiratory muscles weaken and atrophy. As a consequence, the speed of weaning is often related to the duration and mode of ventilation. Assisted modes of ventilation and good nutritional support are important to prevent atrophy of the respiratory muscles.

Patients recovering from prolonged critical illness are at risk of developing 'critical illness polyneuropathy'. In this condition, there is both respiratory and peripheral muscle weakness, with reduced tendon reflexes and sensory abnormalities. Treatment is supportive. There is evidence that long-term administration of some aminosteroid muscle relaxants (such as vecuronium) may cause persisting paralysis. For this reason, vecuronium should not be used for prolonged neuromuscular blockade.

Indications for weaning

The decision to start weaning is often subjective and based on clinical experience. However, there are some guidelines that may be helpful:

- Underlying illness is treated and improving

- Respiratory function:

  - Respiratory rate < 35 breaths/minute

  - $FiO_2 < 0.5$, $SaO_2 > 90\%$, PEEP <10 cmH$_2$O

  - Tidal volume > 5ml/kg

  - Vital capacity > 10 ml/kg

  - Minute volume < 10 l/min

- Absence of infection or fever

- Cardiovascular stability, optimal fluid balance and electrolyte replacement

Prior to trial of weaning, there should be no residual neuromuscular blockade and sedation should be minimised so that the patient can be awake, cooperative and in a semirecumbent position. Weaning is likely to fail if the patient is confused, agitated or unable to cough.

Modes of Weaning

There is debate over the best method for weaning and no one technique has been found to be superior to others. There are several different approaches.

- Unsupported spontaneous breathing trials. The machine support is withdrawn and a T-Piece (or CPAP) circuit can be attached intermittently for increasing periods of time, thereby allowing the patient to gradually take over the work of breathing with shortening rest periods back on the ventilator.

- Intermittent mandatory ventilation (IMV) weaning. The ventilator delivers a preset minimum minute volume which is gradually decreased as the patient takes over more of the respiratory workload. The decreasing ventilator breaths are synchronised to the patient's own inspiratory efforts (SIMV).

- Pressure support weaning. In this mode, the patient initiates all breaths and these are 'boosted' by the ventilator. This weaning method involves gradually reducing the level of pressure support, thus making the patient responsible for an increasing amount of ventilation. Once the level of pressure support is low (5-10 cmH$_2$O above PEEP), a trial of T-Piece or CPAP weaning should be commenced.

Failure to wean

During the weaning process, the patient should be observed for early indications of fatigue or failure to wean. These signs include distress, increasing respiratory rate, falling tidal volume and haemodynamic compromise, particularly tachycardia and hypertension. At this point it may be necessary to increase the level of respiratory support as, once exhausted, respiratory muscles may take many hours to recover.

It is sensible to start the weaning process in the morning to allow close monitoring of the patient throughout the day. In prolonged weaning, it is common practice to increase ventilatory support overnight to allow adequate rest for the patient.

Tracheostomy in the intensive care unit

The commonest indication of tracheostomy in an ICU setting is to facilitate prolonged artificial ventilation and the subsequent weaning process. Tracheostomy allows a reduction in sedation and thus increased cooperation to the weaning process. It also allows effective tracheobronchial suction in patients who are unable to clear pulmonary secretions either due to excessive secretion production or due to weakness following critical illness.

Tracheostomy can be performed as a formal surgical procedure in theatre or at the bedside in the intensive care unit using a percutaneous method (see Update in Anaesthesia Number 15). The timing of conversion from an endotracheal tube to a tracheostomy remains controversial. In general, tracheostomy is considered if the patient is likely to need prolonged ventilation or when weaning will n ot be straightforward. Complications are few but include tube blockage, misplacement or dislodgement, infection and bleeding. Haemorrhage can occur either at the time of the procedure or at a later date caused by erosion of the tube into a major vessel such as the innominate artery.

Other indications for tracheostomy are to bypass an upper airway obstruction, protect the lungs from soiling if the laryngopharygeal reflexes are depressed or as part of a surgical or anaesthetic technique eg larygectomy.

EXPERIENCE WITH THE GLOSTAVENT ANAESTHETIC MACHINE

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In many parts of the world, anaesthetists have to work in difficult or isolated situations where medical supplies are erratic and servicing facilities are poor or non-existent. Most modern anaesthetic machines, however, are not designed to be used in these conditions, as they require high levels of maintenance and servicing by trained engineers and are dependent on continuous supplies of compressed gases and electricity. Consequently, when conditions are unfavorable, they are liable to malfunction or even fail completely.

An anaesthetist working in such difficult conditions requires an anaesthetic machine which has been specifically designed to overcome these problems. It should therefore be reliable, easy to understand and operate, economical to run, require minimal servicing which can be carried out locally, and be versatile, so that the same machine can be used in all patients both as an anaesthetic machine in the operating room and as a ventilator in a recovery room or I.C.U. Most important of all, it must continue to function if either the electricity or oxygen supplies fail, situations that are all too common in parts of the developing world and that have been responsible for many tragedies.

The Glostavent anaesthetic machine has been designed to fulfill these requirements precisely.

In the development of the Glostavent, four separate components have been incorporated, each of which has, in its own right, already proved valuable in difficult environments. These are the draw-over anaesthesia system, the oxygen concentrator, the Manley Multivent ventilator, and the air compressor.

1. The Draw-Over Anaesthesia System

Atmospheric air is used as the carrier gas, which is drawn over a low resistance vaporiser, in this case, the Oxford Miniature Vaporiser (O.M.V.), either by the negative pressure created during inspiration in spontaneously breathing patients, or by the action of bellows when breathing is controlled. IT CAN THEREFORE BE COMPLETELY INDEPENDENT OF THE SUPPLY OF COMPRESSED GASES. Oxygen from a cylinder or a concentrator can be added upstream of the vaporiser, to increase the inspired oxygen concentration.

2. Oxygen Concentrator

This is an electrically powered device which produces a continuous supply of oxygen from atmospheric air, by first compressing the air and then directing it through canisters containing zeolite granules where the nitrogen is absorbed and the residual oxygen delivered to the patient. The zeolite granules are continually being re-activated and do not require changing.

3. Manley Multivent Ventilator

This is a pneumatically driven version of the Oxford inflating bellows. It can be driven either by compressed air or oxygen and only requires a volume of driving gas equal to 1/10 of the patient's minute volume. When oxygen is used for driving the ventilator, it is automatically collected and returned to the breathing circuit. In other words the same oxygen is used twice, first to drive the ventilator and then for the patient to breathe. The bellows of the Multivent can also be operated manually.

4. Air Compressor

This is an integral part of the oxygen concentrator, which has been modified to allow some of the compressed air generated by the concentrator to be diverted for use as driving gas for the ventilator, so that the concentrator provides both the driving gas for the ventilator and oxygen for the patient. In the design of the Glostavent, the four components are mounted on a single trolley, together with 2 reserve oxygen cylinders (figures 1 & 2). It was initially described under its original name of Oxyvent[1] and subsequently as the Glostavent[2].
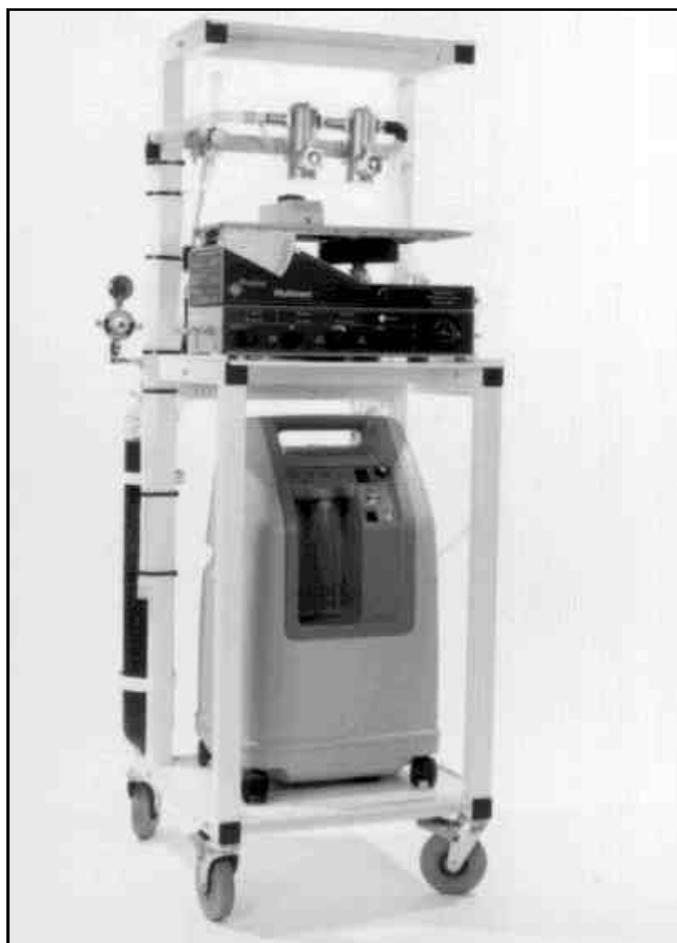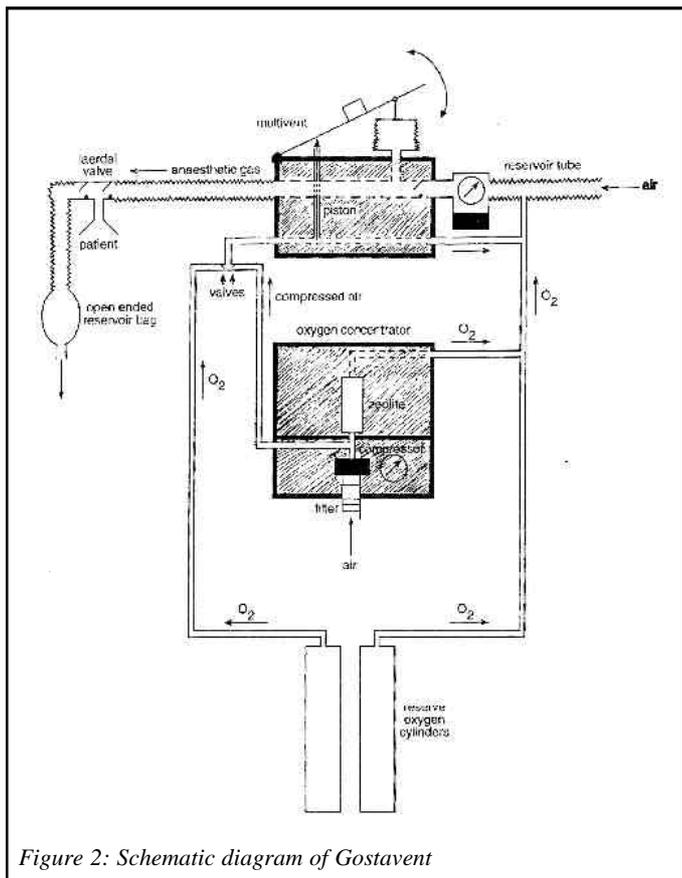

Figure 1: The Glostavent

Figure 2: Schematic diagram of Gostavent

Although it has now been in regular use in several hospitals throughout the world for the past 6 years[3 & 4] delivering anaesthesia safely to thousands of patients, few reports of its use have so far been published[5], and a full description of its operation illustrating its many advantages is not available.

Its potential as a safe, reliable and cost effective anaesthetic machine has been recognised by the Association of Anaesthetists of Great Britain & Ireland, the World Federation of Societies of Anaesthesiology and the Department for International Development, all of whom have contributed to its development.

However, anaesthetists practicing in difficult situations or in isolation need to be confident that it will perform predictably and reliably in their own environments, so that it can be used safely when monitoring facilities are limited or totally absent.

To illustrate this, the records of patients whose anaesthetic had been administered using the Glostavent in a district general hospital with full monitoring were scrutinised. A standard anaesthetic technique was used so that the performance of the Glostavent under various conditions could be analysed and recommendations made for its operation in adverse situations.

Anaesthetic Technique

After a period of pre-oxygenation, anaesthesia was induced intravenously with propofol 2.5mg kg[-1].

If intubation was indicated, this was achieved following vecuronium 0.1mg kg[-1] or rocuronium 0.6mg kg[-1]. In patients not requiring intubation, a laryngeal mask airway (LMA) or facemask was used.

A draw over technique with an inhalational agent in an air/oxygen mixture supplemented by opiates was used in all adult patients. In children under 25kg, the Glostavent was adapted for continuous flow use. (see below)

Either isoflurane or halothane was administered via the O.M.V., to give expired concentrations of 1-1.3 times the Minimum Alveolar Concentration. Opiate supplements (fentanyl 0.3mcg kg[-1] or morphine 0.03mg kg[-1]) were administered intravenously at 15 minute intervals in ventilated patients. In patients breathing spontaneously this rate of administration was adjusted as necessary to maintain a respiratory rate between 10-20 breaths per minute. In patients receiving I.P.P.V. the ventilator was set to deliver a tidal volume of 5ml/kg at a rate of 10 breaths per minute. Supplementary oxygen from either the oxygen concentrator or a cylinder was delivered into the side arm of the reservoir tube at flow rates ranging from 0.5 to 5 litres per minute in order to maintain the Sa O_2 above 95%.

The patients ranged in age between 1 and 92 years, in weight between 10 and 130kg and the duration of surgery between 10 minutes and 5 hours 30 minutes.

The Glostavent was used in any one of 4 different modes, depending on the type of breathing (spontaneous breathing or I.P.P.V.) and the source of the oxygen(concentrator or cylinder) [Table 1].

Table 1. Grouping of patients according to type of breathing and source of oxygen.

	Type of Breathing	Source of oxygen
Group 1	Spontaneous	Cylinder
Group 2	Spontaneous	Concentrator
Group 3	I.P.P.V	Cylinder
Group 4	I.P.P.V	Concentrator

Ventilation

In spontaneously breathing patients, provided the rate of administration of opiates was adjusted to maintain the respiratory rate between 10-20 breaths per minute, both respiratory depression on the one hand and light anaesthesia on the other could be avoided by using simple clinical observation alone [Table 2]. This is an important factor in situations where capnography is unavailable.

In patients receiving I.P.P.V., the ventilator settings gave levels of $FeCO_2$ which were satisfactory.

The minute volume was found to be similar in all four groups, although in both groups of spontaneously breathing patients (groups 1 & 2) this was associated with higher respiratory rates and lower tidal volumes and resulted in higher $FeCO_2$ values than in those receiving I.P.P.V. (groups 3 & 4).

Table 2 . Relationship between Tidal Volume, respiratory minute volume and FECO2.				
	Respiratory rate Breaths/min (Mean ± SD)	Tidal volume VT (ml)	Resp. minute volume (Litres/ min) (Mean ± SD)	FECO2 (mmHg) (Mean ± SD)
Group 1	16 ± 4.3	241	3.8 ± 4.9	49.3 ± 7.2
Group 2	15 ± 6	250	3.9 ± 5.1	48.6 ± 7.8
Group 3	10 ± 1	380	3.8 ± 0.9	39.8 ± 5.8
Group 4	10 ± 1	380	3.8 ± 1	39.5 ± 6.3

Oxygenation

No problems with oxygenation occurred in any of the patients. There was a clear relationship between the flow rate of added oxygen and the FiO_2 in all groups (figure 3). When the concentrator was the source of oxygen(groups 2 & 4), FiO_2's of 75-85% could be achieved with a flow of 5L/Min. However, when using cylinder oxygen (groups 1 & 3) which is not contaminated with nitrogen or inert gases, a corresponding FiO_2 was achieved with much lower flow rates. This is an important factor when cylinders are in short supply and oxygen needs to be conserved.

During I.P.P.V. (groups 3 & 4) the driving gas for the ventilator was either oxygen from the cylinder (group 3), or compressed air from the concentrator (group 4).

When this driving gas was added to the inspired mixture, FiO_2's were found to be 20% higher in the case of O_2. Indeed, in group 3, FiO_2's of over 35% were consistently achieved without the need for any supplementary oxygen whatsoever.

An important safety feature of the draw over system is that, in the absence of nitrous oxide, the accidental delivery of a hypoxic mixture is impossible. As the FiO_2 has been shown to be largely predictable for a given flow rate of supplementary oxygen from either concentrator or cylinder, the absence of an oxygen analyser need not preclude the use of the Glostavent. However, its use is recommended in situations where the FiO_2 may be critical, such as sickle cell disease, chronic bronchitis, or in the intensive care unit.

The O.M.V.

The O.M.V. proved entirely satisfactory both for draw over and continuous flow anaesthesia. In most patients, the target concentration of inhalational agents was achieved by using a dialed concentration of 2MAC for 15 minutes followed by 1.5MAC thereafter. Good correlation was seen between the concentration shown on the vaporisor setting and the inspired concentrations for both agents (figs. 4a & 4b).

Despite the absence of a full temperature compensating mechanism in the OMV as is found in more sophisticated

Fig 4 (a). Relationship between Vaporizer Settings and Inspired & Expired concentrations of Halothane (Mean and Standard error).
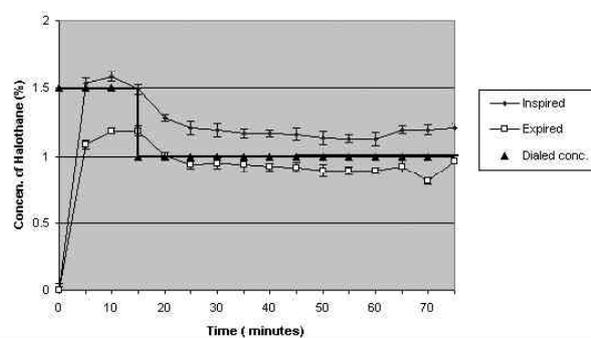

Fig 3 . Relationship between FiO_2 (Mean and Standard Error) and Supplementary Oxygen Flow (L/min).

Fig 4 (b). Relationship between Vaporizor Settings and Inspired & Expired concentrations of Isoflurane (Mean and Standard error).
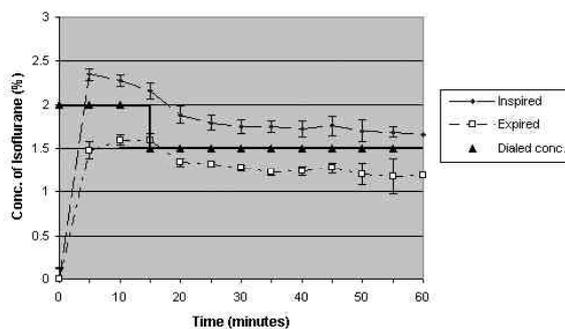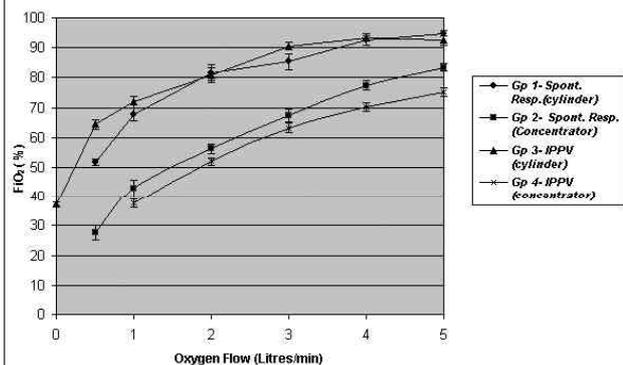
vaporisers, the fall in output concentration seen as the vaporiser cooled, was small and clinically insignificant. With the concentrations used, the chamber held sufficient volatile agent for approximately 2 hours of anaesthesia for isoflurane and 3 hours with halothane before requiring refilling.

Paediatric Use

The use of the draw over technique is not recommended in small children breathing spontaneously, because of the resistance of the circuit and the deadspace of the valves[6]. For this reason, in children under 25kg, the Glostavent was converted for continuous flow use. This was achieved simply by occluding the open end of the reservoir tube with a bung, in order to allow the gas flow to build up sufficient pressure to pass through the vaporiser. A Mapleson E circuit was then attached to the common gas outlet, as with any standard continuous flow anaesthetic machine and oxygen administered at a flow rate of 4L/Min from either the cylinder of the concentrator. The O.M.V was shown to function equally satisfactorily for continuous flow and draw over anaesthesia, so that no change of vaporiser was required.

Using the Glostavent

An important feature of the Glostavent is its simplicity, enabling first time users to master it quickly and easily. The same circuit is used for both I.P.P.V. and spontaneous respiration. Conversion from one to the other simply involves turning the ventilator off and bypassing the bellows, no other action is necessary. A handle is attached to the bellows to facilitate manual ventilation when this is required.

Under normal circumstances, when electricity is available, it is more convenient as well as more economical, to conserve cylinders of oxygen and to use the concentrator to provide both the oxygen for the patient and, when I.P.P.V. is required, the compressed air to drive the ventilator. In this mode, a flow rate of oxygen of 2 L/Min delivered into the side arm of the reservoir tube raised the FiO_2 to 50-55% in both spontaneously breathing and ventilated patients. This is satisfactory in most cases and can be recommended for routine use. Higher FiO_2 values, in the region of 75% can be obtained by increasing the oxygen flow to a maximum of 5L/Min. If still higher oxygen concentrations are required, oxygen from the reserve cylinders can be added.

In the developing world oxygen cylinders are expensive to purchase and to transport and they should normally be kept in reserve, to be used only if an electricity failure renders the concentrator inoperable or to increase the FiO_2 in an emergency.

When I.P.P.V. is used, the driving gas for the ventilator can either be oxygen from the cylinder (Group 3), or compressed air from the concentrator (Group 4). When the concentrator is in use, any interruption in the supply of electricity triggers an audible alarm. This alerts the anaesthetist that the concentrator has stopped. The reserve oxygen cylinders are then turned on and the anaesthetic can continue without interruption.

When cylinders are in use, conservation of supplies becomes extremely important. As has been clearly shown in groups 1 & 3, satisfactory FiO_2's were achievable with minimal flows of supplementary oxygen and indeed during I.P.P.V. without the need for any additional oxygen whatsoever.

Further conservation is possible because of the unique design of the Manley Multivent ventilator. With most other gas driven ventilators, the volume of driving gas required is equal to the patient's minute volume[7 & 8]. The Manley Multivent, however, was specifically designed for economy and the requirement for driving gas reduced to 1/10 of the minute volume[9]. With the minute volume for example set at 4 litres per minute, the driving gas is utilised at a rate of 0.4 litres per minute and an E size oxygen cylinder containing 680 litres should not only be able to drive the ventilator, but also supply the average oxygen requirement for a period of 28 hours. The Glostavent is therefore ideal for situations where cylinder supply is difficult and conservation is important.

Costs

1. Volatile Agents

Although other volatile agents can be used in draw-over anaesthesia, only halothane and isoflurane have so far been reported. Halothane is not only more readily available in the developing world than isoflurane, but is also cheaper and, because of its lower MAC value, can be used in lower concentrations. The mean rate of utilisation of halothane was found to be 16ml/hour and isoflurane 25ml/hour. At respective prices of 6.4p and 19p per ml[10], the cost of the inhalational agents were £1.02 and £4.75 per hour of anaesthesia.

2. Opiates

The mean rate of utilisation of the opiates was: fentanyl $100mcg/hr^{-1}$ and morphine $10mg\ hr^{-1}$. The corresponding costs per hour of anaesthesia were 24p and 66p respectively[11].

3. Oxygen

The cost of the electricity required to operate the concentrator calculated on present U.K. rates is only 2.5p per hour, regardless of the flow of oxygen produced. In contrast, when using cylinder oxygen, the cost varies in proportion to the flow rate. When using a standard 680L cylinder, (currently priced at approximately £2.00) the cost can vary from 10 pence per hour, at a flow rate of 1/2 Litre per minute, to £1.00 per hour at 5 Litres per minute.

The most economical way of providing anaesthesia with the Glostavent is therefore to use halothane as the volatile agent, with fentanyl supplementation and the concentrator as the source of oxygen. With this combination, general anaesthesia can be maintained for a cost of under £1.50 per hour [Table 3].

Conclusion

The Glostavent is much less expensive than the majority of continuous flow anaesthetic machines in current use and yet offers considerable advantages when used in difficult situations.

These include, not only the low cost of the anaesthesia, but much more importantly, the ability to maintain the delivery of an anaesthetic safely when cylinders of oxygen, nitrous oxide and compressed air and supplies of soda lime may be scarce and the electricity supply unreliable.

Regardless of the conditions in which they work, the aim of anaesthetists all over the world is the same, that is to provide an anaesthetic service which is both effective and safe at all times.

Table 3 . Cost of maintenance of anaesthesia per hour	
Halothane 16 ml (6.4 pence/ml)	£ 1.02
Fentanyl 100 mcg (24 pence/ 100 mcg ampoule)	£ 0.24
Electricity to drive the concentrator (per hour)	£ 0.03
Total cost	£ 1.29

To achieve this, considerably greater demands are placed on anaesthetists in the developing world because of the lack of drugs, equipment and facilities than those in wealthy environments with greater resources. In the attempt to make anaesthetic machines ever more foolproof for use when conditions are ideal, the basic problems that still confront the majority of anaesthetists throughout the world are easily forgotten. Modern sophisticated machines, however expensive, cannot be considered good enough for use in the developing world if they cannot be relied on when conditions become unfavorable. Only equipment which has been specifically designed to overcome their problems is adequate. The Glostavent can make a significant contribution towards meeting these requirements and is recommended for use in the developing world.

Acknowledgements

Summary

The Glostavent is an anaesthetic machine which has been designed to enable anaesthetists practicing in adverse conditions to overcome the difficulties they are likely to encounter. These include inadequate or non-existent monitoring and servicing facilities and frequent disruption in the supplies of oxygen, nitrous oxide, soda lime or electricity.

An examination of the records of patients who were anaesthetised using the Glostavent with full monitoring, demonstrates its predictability, reliability and economy over a wide range of clinical situations. Suggestions are made for its cost effective operation. It is recommended as an anaesthetic machine capable of providing a safe and reliable anaesthetic in adverse conditions.

Key Words

Equipment   - anaesthetic machine

                   - reliability in adverse situations

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SELF-ASSESSMENT QUESTIONS

Question 1:

1

An 11 month-old baby is brought to the emergency department. She has been completely well until a sudden bout of coughing while playing on the floor. Her parents report that in the intervening four hours she has been unhappy and refusing to eat or drink. On examination she is found to be irritable but apyrexial and not cyanosed. She is not in any respiratory distress but a soft inspiratory stridor is audible and she is drooling saliva. Examination of the rest of the respiratory system and the cardiovascular system is unremarkable. A chest X-ray is performed (shown).

A. What is the diagnosis?

B. How should this child be managed?

Question 2:

2

Interpret the following 3 electro-cardiograms.

3

Answers:

1a. Extrathoracic intratracheal foreign body

The sudden onset of cough and stridor in a previously well child of this age with no symptoms or signs of infection or allergic reaction make an inhaled foreign body the likely diagnosis. This is confirmed on the chest X-ray. A child with croup, epiglottitis or laryngeal oedema should not be sent for radiological investigation as this is unhelpful and dangerous. Both the diagnosis and the assessment of the severity of these cases are clinical. They were not considered likely in this case and the baby's condition was such that a x-ray was possible. This yielded valuable information as it confirmed the diagnosis and the level of the obstruction. Many foreign bodies are not radio-opaque but areas of collapse or hyperinflation (due to ball-valve type effect of the foreign body) may be seen. It needs to be emphasised that any child with a foreign body in the larynx or trachea is at risk of sudden, complete airway obstruction and if, as in this case, the decision is made that an x-ray is indicated, the x-ray needs to be taken in the emergency department or the child needs to be accompanied by an anaesthetist to the radiology department.

1b. This baby needs laryngoscopy and/or bronchoscopy and removal of the foreign body under general anaesthesia. Nothing should be done to distress the baby prior to anaesthesia as this could worsen the airway obstruction. Antisialogogue premedication is useful prior to laryngoscopy and bronchoscopy as it may make the induction smoother and makes the application of topical anaesthesia easier. This should be omitted in this case as the baby has dysphagia and the parenteral route will cause distress. An experienced ENT surgeon should perform the bronchoscopy as this can be a challenging procedure and he/she should be present during induction of anaesthesia as it is possible that a surgical airway may need to be created if the airway cannot be maintained after induction of anaesthesia. All equipment must be prepared and checked in advance. A wide range of tracheal tubes with introducers and bougies and equipment for transtracheal ventilation and emergency resuscitation drugs should be available. A range of laryngoscopes and a Stortz ventilating bronchoscope and forceps is the equipment usually required by the surgeon for these cases.

Anaesthesia should be induced by means of an inhalational induction with sevoflurane or halothane in 100% oxygen. This may be prolonged if the obstruction is severe. Spontaneous ventilation should be maintained and muscle relaxants should not be used. Intravenous access should be gained after induction and atropine can be given at this stage. Anaesthesia should be maintained with halothane in 100% oxygen as this has the advantage over sevoflurane that once deeply anaesthetised the mask can be removed and the airway examined/instrumented without a rapid decrease in the depth of anaesthesia. If the foreign body is in the lower respiratory tract and bronchoscopy is to be performed applying topical anaesthesia to the respiratory tract is advisable. 4mg/kg lignocaine can be used safely and it decreases the incidence of coughing, laryngospasm and breath-holding. The trachea can also be intubated with an endotracheal tube to ascertain the correct size and to assist in the sizing of the brochoscope.

In this case the foreign body is in the trachea and there is a constant risk of complete airway obstruction if its position changes. A careful laryngoscopy should be undertaken under deep halothane anaesthesia and the situation assessed. The surgeon and the anaesthetist should then make a decision on how best to proceed. If part of the foreign body is visible above the vocal cords it should be removed with forceps. There is the potential for it to be dislodged and to fall back and totally obstruct the airway. If the foreign body is not visible above the cords, bronchoscopic evaluation and removal should be undertaken with spontaneous ventilation/assisted spontaneous ventilation. Limited application of topical anaesthesia may be possible to the larynx. Care needs to be taken when removing it that it does not fall back and totally obstruct the airway. It may sometimes be necessary in this situation, if it cannot be rapidly removed, to temporarily push it further down past the carina to establish an airway.

When performing a rigid bronchoscopy the anaesthetic T-piece is connected to the side arm of the bronchoscope. The baby breathes through the bronchoscope and its breathing can be assisted as it is a closed system. The telescope does however narrow the lumen and greatly increases the resistance to airflow. Hypoventilation, hypoxia and too light or too deep anaesthesia are all potential problems and may cause arrythmias in the presence of halothane. Problems of this sort are particularly likely if the bronchoscope is introduced past the carina into one lung. Withdrawal of the bronchoscope into the trachea, temporary removal of the telescope, hyperventilation with oxygen and adjusting the level of anaesthesia may be necessary.

Postoperatively the baby should be kept nil by mouth until two hours after the application of the topical lignocaine and should be given humidified oxygen. If the procedure is prolonged and postoperative airway swelling is likely dexamethasone should be prescribed and stridor can be treated with nebulised adrenaline.

In this actual case the baby was anaesthesised as above and the tip of the foreign body was just visible between the vocal cords. Its widest part had lodged at the level of the cricoid cartilage with the rest of it lying in the trachea. The anaesthetist removed it at the initial laryngoscopy. There were no postoperative problems.

2a.   Atrial fibrillation, left bundle branch block (QRS complex greater than 0.12 seconds and of the LBBB pattern)

2b.   First degree heart block (PR interval greater than 0.2 seconds)

2c.   Ventricular pre-excitation of the Wolff-Parkinson-White type

In the normal heart only the AV node can conduct atrial impulses to the ventricle. Patients with ventricular pre-excitation have an additional connection between the atria and the ventricles. Unlike the AV node, the accessory pathway does not delay conduction between the atria and the ventricles and thus during sinus rhythm ventricular activation will occur via the accessory pathway before the impulse has traversed the AV node. However, because the accessory pathway is not connected to specialised conducting tissue the rate of ventricular activation will be slow, resulting in a slurred initial portion of the QRS complex (delta wave). Once

the atrial impulse has traversed the AV node further ventricular depolarisation will be normal. During sinus rhythm, therefore, the QRS complex is a fusion of the delta wave and a normal QRS complex. The diagnosis is made if the following are present on the ECG: a short PR interval (less than 0.12 seconds), a prolonged QRS duration (greater than 0.12 seconds) and a delta wave. In the presence of WPW type pre-excitation myocardial infarction and LBBB should not be diagnosed by the non-expert and is impossible without access to previous electrocardiograms.

These patients are prone to supraventricular taccyarrythmias. If they occur they are said to have the Wolff-Parkinson-White Syndrome. They can arise because the AV node and the accessory pathway differ in the time they take to recover after excitation and if an ectopic beat occurs during sinus rhythm this ectopic impulse may find one pathway refractory while the other is not. After conduction down the non-refractory pathway the other pathway may have recovered resulting in the impulse repeatedly circulating between the atria and the ventricles. The result is an atrioventricular re-entrant taccycardia. If the impulse travels from the atria to the ventricles via the AV node and back to the atria via the accessory pathway as occurs in 90% of cases the taccycardia is said to be orthodromic and the QRS complexes will be normal in appearance. If the opposite occurs, the taccycardia is said to be antidromic and the QRS complexes will show pre-excitation (broad complex). Treatment is as for any supraventricular taccycardia ie. vagal stimulation and/or adenosine initially and if necessary cardioversion or intravenous medication such as verapmil, β blockers or amioderone.

During atrial fibrillation conduction to the ventricles occurs via both the AV node and the accessory pathway. Most ventricular complexes will be broad with delta waves but some may be normal. This is a dangerous situation as very rapid ventricular rates may result. As most atrial impulses reach the ventricle via the accessory pathway drugs that slow conduction through the AV node (verapmil, digoxin) will not be effective as treatment and can increase the speed of conduction in the accessory pathway. They should be avoided. Flecainide, amioderone or sotalol can be used.

ANAESTHESIA AND LIVER DISEASE

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Liver disease can vary in severity from sub-clinical to end-stage liver disease (ESLD), with life threatening, multi-organ multi-system failure. Anaesthetic and operative risks are related to the severity of liver dysfunction, so thorough pre-operative assessment is essential for safe peri-operative care. A good understanding of the pathophysiology of liver dysfunction is vital for assessment of operative risk.

PATHOPHYSIOLOGY OF ESLD

While the severity of liver dysfunction may vary widely, a limited review of the pathophysiology of severe disease is appropriate. The range of symptoms depends largely upon whether the disease presentation is acute or chronic. If chronic, features may be super-imposed on a background of poor nutrition and chronic ill health. The acute presentations have been subject to re-classification in recent times in order to take into account differing survival rates with medical treatment alone. The first formal definition was proposed by Trey and Davidson who defined fulminant liver failure (FHF) as the appearance of encephalopathy within eight weeks of onset of jaundice. The King's College group now recognises three levels of acute presentation within FHF (Table 1). Contrary to expectation, survival rates are better in the acute and hyper-acute groups on medical management alone. Gimson has written a useful review of fulminant hepatic failure.

Impaired liver function

Impaired liver function gives rise to effects directly attributable to the failing liver itself and also to indirect effects expressed via other organ systems. Effects directly attributable include hypoglycaemia, lactic acidosis, hypermetabolism, azotemia and impaired urea synthesis. Jaundice appears when serum bilirubin exceeds 35 µmol/l and defects in cholesterol metabolism together with intra-hepatic cholestasis may lead to production of poor quality bile and malabsorbtion of fat and fat-soluble vitamins. There is reduced synthesis of proteins such as albumin, clotting factors, thyroid binding globulin and pseudo-cholinesterase. Impaired hormone biotransformation, reduced production of modulator proteins and reduced protein binding lead to increased circulating levels of hormones such as insulin, thyroxine, T_3, aldosterone and oestrogen.

Impaired hormone modulation, failure to clear by-products of metabolism, activation of cytokines and release of vasoactive substances from the damaged liver result in patho-physiological changes in many organ systems. These indirect effects include:

Cardiovascular changes

Vasodilatation and vascular shunting are almost invariable in ESLD. Low systemic vascular resistance (SVR) results in high cardiac output and high mixed venous oxygen saturations. Pulmonary hypertension may develop, while portal venous hypertension can lead to varices, variceal bleeding and porto-systemic shunting. Low flow in the portal vein can result in portal venous thrombosis. Variceal bleeding may be life threatening.

Pulmonary changes

Pulmonary problems are both vascular and mechanical. Intra-pulmonary shunt dilatation (hepato-pulmonary syndrome), impaired hypoxic vaso-constriction and ventilation perfusion mismatch lead to arterial desaturation and clubbing if chronic. Pleural effusions together with ascites can cause considerable mechanical embarrassment of respiration and a reduction in functional residual lung capacity.

Electrolytes and Renal

There are numerous causes of renal impairment in liver failure, including hepato-renal syndrome, sepsis and renin-angiotensin activation. Hyponatraemia due to water retention and inhibition of membrane bound Na/K ATPase, hypoalbuminaemia and oedema are common. Saline should be avoided but hypomagnesaemia and hypo-phosphataemia should be corrected.

Neurological problems

Mechanisms leading to deepening encephalopathy, loss of vascular auto-regulation, cerebral oedema and death are incompletely understood. A number of processes may act in parallel, but can be summarised as the accumulation of neurotoxic compounds penetrating an impaired blood-brain barrier. At the same time, lack of nutrients and substrates may impair brain metabolism and alter neuro-transmitter synthesis. Of particular interest are a group of endogenous benzodiazepine-like

Table 1: Liver Failure: mode of presentation and survival			
Presentation	**Encephalopathy**	**Jaundice** (Days)	**Survival Rate** (medical management only)
Chronic	No	+/-	-
FHF Sub-acute	Yes	29-72	14%
Acute	Yes	8-28	26%
Hyper-acute	Yes	<7	36%

substances that are thought to act at a site closely linked to the g-amino butyric acid (GABA) receptor. Drowsiness can be transiently reversed by flumazenil, but not in all cases. Symptoms can occur in chronic as well as in acute disease, may be rapid in onset and may be precipitated by a gastrointestinal bleed, dietary protein overload or sepsis. Somnolence can be exacerbated by sedative drugs and narcotics.

Rapid correction of hyponatraemia can lead to osmotic demyelination and central pontine myelinolysis and should be avoided.

Haematological

Anaemia may be the result of nutritional deficiency, toxic bone marrow depression or gastrointestinal bleeding from varices or erosions. Coagulation defects arise from thrombocytopenia, platelet dysfunction and decreased levels of circulating clotting factors. Clotting factor levels fall because of impaired synthesis, vitamin K malabsorbtion and intravascular consumption. The short half-life of clotting factors means that INR or Prothrombin Ratio (PTR) can reliably be used to evaluate residual hepatic function.

Susceptibility to infection

There may be a wide variety of defects in host defences that can contribute to a substantial risk of sepsis, with up to 80% of patients with FHF developing bacterial sepsis (frequently Gram positive organisms) and 30% fungal sepsis. Clearly, particular attention must be paid to aseptic technique when inserting lines.

Drug disposition

There may be considerable derangement of drug handling in the patient with liver dysfunction. Aetiology may influence pharmacokinetics and the nature and extent of hepatocellular damage may alter drug metabolism. Cholestasis will reduce absorbtion of fat-soluble drugs after oral administration, while other drugs with limited systemic availability due to high hepatic extraction, may achieve high peak plasma concentrations if there is porto-systemic shunting. Compartment changes and altered protein binding will affect volume of distribution, clearance and re-distribution. Patients with liver dysfunction may be particularly

sensitive to opiates and benzodiazepines due to altered end-organ sensitivity (see 'Neurological problems' above).

Causes of Liver Failure

The commonest causes of acute and chronic liver failure (ALF) are listed in Table 2. In the UK, paracetamol poisoning was until two years ago the most frequent cause of FHF. When a change in regulations reduced over-the-counter pack size of paracetamol to a maximum of 8 tablets, the incidence of paracetamol poisoning fell dramatically. Worldwide, by far the major cause of liver disease is viral infection, with Hepatitis B (HBV) and C (HCV) together accounting for 75% of all cases. The natural history of chronic infection with both HBV and HCV includes progression to cirrhosis and an increased risk of developing hepatocellular carcinoma (HCC).

Infection with HCV deserves special mention. The nature of HCV replication is such that during the course of a single infection, HCV frequently changes its antigenic signature. As a result of this and of other mechanisms, the virus is able to confuse host immune responses, with the result that 85% of HCV infections become chronic, as opposed to about 5% in the case of HBV. Chronic HCV infection is insidious and it may take up to 15 years for overt signs of liver failure to develop. Not only can apparently stable, asymptomatic patients decompensate acutely as a result of anaesthesia, but they can also represent a significant infection risk for the anaesthetist.

Risk and severity scoring

In 1964, Child and Turcotte classified risk for patients with liver cirrhosis undergoing porto-caval anastomosis for management of portal hypertension. Pugh et al at King's College Hospital published a severity scoring system for patients undergoing oesophageal transection for bleeding oesophageal varices. The two systems have been amalgamated and provide a disease severity assessment based on two clinical and three laboratory variables (Table 3).

Surgery in patients with liver dysfunction

The Child-Pugh classification is a useful method of staging the progress of liver decompensation. However, despite its surgical

Table 2: Causes of Liver Failure (UK)			
CHRONIC		**ACUTE**	
Infection	*Viral*	Infection	*Viral A-E, Non A-E*
Biliary obstruction	*1° : PBC*	Drugs	*e.g. paracetamol, rifampicin, phenytoin,*
	2°: Congenital, stone		*halothane*
Alcohol		Toxins	*Amanita phalloides*
Toxins		Miscellaneous	*Wilson's disease*
Drugs			*Fatty liver of pregnancy*
Auto-immune			*HELLP*
Metabolic diseases			*Lymphoma*
Wilson's Disease			*Sepsis*
a_1-Antitrypsin deficiciency			*Reye's syndrome*
Veno-occlusive	*Budd-Chiari*		*Heatstroke*

Table 3: Child-Pugh Score			
Clinical or biochemical measurement	**Points scored**		
	1	**2**	**3**
Encephalopathy grade Ascites Bilirubin Albumin PT (secs. prolonged)* [INR] *Score prothrombin time or INR	None Absent <35 µmol/l >35 g/l 1-4 secs [<1-7]	1-2 Mild 36-60 µmol/l 28-35 g/l 4-6 secs [1.7-2.3]	3-4 Moderate to severe >60 µmol/l <28 g/l >6 secs [>2.3]
Child-Pugh A Score ≤ 6, Child-Pugh B Score 7-9, Child-Pugh C Score ≥10			

pedigree, it is of limited predictive value in anaesthesia and surgery, because **Group B and C patients all represent a high perioperative risk**. In general surgical practice, only emergency or life-saving procedures should be undertaken in these patients. In a unit where liver transplantation is an option, other procedures can be considered, particularly those intended to 'buy time' until a suitable donor organ is available. **Group A patients are lower risk** and with sufficient care can be considered as candidates for most types of surgery.

Hepatocellular carcinoma is a recognised complication in those with chronic HBV or HCV infection. Rates are reported to run between 800 and 2000 cases per year per 100,000 chronically infected. Even in Group A patients, operative mortality for hepatic resection of tumour runs at 5%-10%.

Group B/C patients present an extremely high operative risk and surgical procedures in these patients should be avoided if possible. Considerable morbidity and a high mortality rate invariably accompany all but minor surgery. Procedures that might be performed in these patients include:

- Laparotomy for perforation or bleeding - often following previous surgery

- Porto-systemic anastomosis for portal hypertension: includes meso-caval and distal lieno-renal anastomosis. Encephalopathy is a common post-operative complication.

- Peritineo-venous shunting for intractable ascites where liver transplantation is not an option.

Hepatic resection of tumour in Group B/C patients carries an operative mortality of about 50%.

Anaesthesia for patients with liver dysfunction

For the purposes of this presentation, the basic principles of peri-operative care of three types of 'liver' patient will be considered. They are:

1.   Medium risk patients - Child-Pugh Group A

2.   High risk patients - Child-Pugh Group B/C

3.   Management of patients with bleeding varices

1)   Anaesthesia in Child-Pugh Group A Patients
In general terms, these patients can be considered as 'normal' and, for minor procedures, anaesthetic technique can be dictated by personal preference. However, major procedures should be planned with care and even though tests of coagulation may prove to be unremarkable, major bleeding is a possibility and adequate venous access and blood products should be available.

As with any patient, there should be careful pre-operative assessment, with appropriate history, examination and investigations. Issues relevant to Child-Pugh A patients are shown in Table 4. The anaesthetist should be aware of aetiology, particularly in the case of viral hepatitis or drug idiosyncrasy. It is useful to know also whether the condition is chronic or whether the patient is convalescing from an acute episode. Patients with chronic liver dysfunction can run a remitting / relapsing course and previous episodes of encephalopathy or bleeding are relevant

Table 4: Peri-operative considerations in Child-Pugh A patients

Pre-operative	Aetiology of condition – virology, drug idiosyncrasy Blood count and platelets Clotting screen Assess renal function Previous anaesthetics
Per-operative	Consider drug bio-availability issues ? avoid drugs excreted via liver Regional techniques acceptable if clotting normal
Post-operative	Monitor for post-operative hepatic decompensation Possible prolonged duration of action in opiates HDU / ITU care

to patient management. Patients recovering from an acute condition, such as acute hepato-cellular damage secondary to drug idiosyncrasy may continue to have an underlying coagulopathy or may have impaired drug clearance. Drugs dependent upon the liver for clearance, such as vecuronium and rocuronium will have a prolonged duration of action.

Regional techniques may be used in the absence of coagulopathy and are particularly helpful in post-operative pain management. Propofol is a useful induction agent as it undergoes considerable extra-hepatic metabolism, similarly, atracurium clearance is independent of liver metabolism. Isoflurane preserves hepatic blood flow, but both sevoflurane and desflurane are acceptable. Since many patients may be managed in intensive care post-operatively, the merit of using short and ultra short- acting narcotic analgesics is debatable. Morphine and fentanyl are entirely acceptable.

Postoperative HDU or ITU admission should be considered for all Group A patients undergoing major surgery, in order to forestall coagulation, fluid management, renal and respiratory complications.

2) Child-Pugh Group B/C patient undergoing major surgery

Patients in this group are very ill and present a considerable anaesthetic challenge. There can be profound derangement of nearly every physiological system and anaesthesia in such patients should not be embarked upon lightly. Aspects of pre-operative assessment are detailed in Table 5. Previous upper abdominal surgery, portal hypertension and coagulopathy dramatically increase the potential for per-operative blood loss, which can further complicate critical physiological derangements. 8-12 units of blood, together fresh frozen plasma and platelets should be

available. An exhaustive discussion of peri-operative management is beyond the scope of this paper, but the principal issues are listed below in note form.

Pre-medication

Sedative premedicants should be avoided in the encephalopathic patient. Other drugs may be needed pre-operatively and include antibiotics and H_2 receptor antagonists. Delayed gastric emptying is not uncommon. The oral or intravenous route should be used for administering drugs – intramuscular injections should be avoided. Coagulopathy may require correction with fresh frozen plasma and platelets and renal replacement therapy may need to be considered.

Induction and per-operative considerations

The comments on anaesthetic technique for Group A patients above apply equally in the case of Group B/C patients. Regional techniques need to be considered carefully as most patients will suffer some form of coagulopathy; and epidural varices can pose an additional risk. Other issues in the per-operative management of Group B/C patients include:

Vascular access with a multi-lumen central venous catheter together with at least one large bore central line (e.g., 8.5 FG Swan Sheath), is of paramount importance. Monitoring of arterial and central venous pressures is mandatory. Pulmonary artery, pulmonary capillary wedge pressure and cardiac output measurements may be necessary in the sick patient. The availability of techniques such as trans-oesophageal echocardiography and volumetric haemodynamic monitoring / pulse contour analysis can provide significant additional information for the strategic management of these patients.

Table 5: Pre-operative considerations in Child-Pugh B/C patients

History	Aetiology. Previous upper abdominal surgery. Anaesthetic history CVS: exercise tolerance, oedema, orthopnoea RS: dyspnoea, ascites, pleural taps GI: bleeding, haematemesis, maelaena, piles Sepsis / urine output Medication:diuretics / antibiotics
Examination	Usual PLUS muscle mass, cyanosis, clubbing, temperature CVS: pulse rate, venous pressure, BP, oedema RS: resp. rate, effusions, sputum Abdo: ascites, spleen, caput medusae CNS grade of consciousness (seeencephalopathy below) ICP: unconscious patient Venous access (existing + potential)
Encephalopathy	**Grade 1:** mild confusion, fully coherent when roused **Grade 2:** increasing confusion, rousable, able to be rational **Grade 3:** sleeping mostly, roused to command, may be agitated or aggressive **Grade 4:** unrousable, $\pm$ reacts to pain, ?signs of cerebral oedema
Investigations	ECG, CXR; Blood - electrolytes, sugar, albumin, creatinine, gas, SpO_2, lactate, Blood count, platelets, INR (PTR); Viral serology; Ultrasound - abdominal (portal flow, pressure, ascites) AND cardiac (myocard wall movement, pericardial effusion) ; endoscopy, microbiology

Although per-operative studies have yet to be undertaken, evidence is emerging that the use of these devices in ITU may significantly shorten ITU stay in the critically ill.

Coagulation and fibrinolysis are major concerns. The potential for large volume blood replacement means that temperature should be measured and a fluid warmer and warming mattress used. Cold patients do not clot. Regular per-operative estimation of INR/PTR may be necessary. Alternatively, thrombo-elastography provides useful intra-operative evaluation of coagulation.

Blood conservation Blood conservation with a 'cell saver' should be considered, particularly if the patient has had previous upper abdominal surgery or has portal hypertension and bleeding is likely to be heavy.

Preservation of hepatic function. N-acetylcysteine (NAC) is a sulphur-containing antioxidant that has been shown to benefit patients with fulminant hepatic failure. NAC appears to improve oxygen delivery and consumption, and reduce base deficit.

Renal Function

Renal function is often impaired in these patients, so peri-operative replacement therapy may be needed. Dopamine, in spite of doubts cast over its ability to influence long-term outcome, may be useful in preserving renal function. Appropriate inotropic support may also be needed.

Post-operative management

Patients will require post-operative admission to the intensive care unit for all but the most minor surgery, and should undergo elective ventilation until cardiovascular parameters are stable and there is no evidence of bleeding. Parameters monitored per-operatively should continue to be monitored in the post-operative phase, with regular review of blood count, clotting profile and blood gases, chemistry and sugar. Dopamine and inotropes should be continued as long as necessary. The principle complications are likely to be continued bleeding, sepsis and hepatic decompensation and a low index of suspicion should be entertained for all of these. Patients who continue to bleed despite adequate blood and blood product replacement must undergo urgent laparotomy to stop bleeding and evacuate clot. While intra-abdominal tamponade may be regarded as useful in reducing bleeding, there can be deleterious effects on the kidneys that may be only transiently reversible. Furthermore, a large clot can present an unacceptable protein load to an already impaired liver, precipitating encephalopathy, and can be a nidus for sepsis.

Analgesia and sedation

If neuraxial anaesthesia has been used, then appropriate analgesic regimes can be implemented, with light sedation while the patient is ventilated. Otherwise fentanyl or morphine can be given by infusion, together with propofol or midazolam for sedation

3) Bleeding oesophageal varices

The therapeutic pathway for the management of bleeding oesophageal varices is well established. As soon as the airway has been appropriately protected, adequate venous access and volume replacement obtained and blood cross-matched, the most common sequence is as follows:

1. Administration of a vaso-constrictor (e.g., vasopressin, glypressin or somatostatin) and balloon tamponade, for example, with a **Sengstaken Tube**. Balloon placement should be undertaken by an experienced clinician, as mal-positioning can result in oesophageal rupture, an invariably fatal complication.

2. Injection sclerotherapy can be used acutely, but is used also to prevent recurrence of bleeding.

3. If the preceding manoeuvres fail to arrest bleeding, then an aorto-portogram should be undertaken with a view to de-vascularisation or porto-systemic shunting.

4. In general, surgical de-vascularisation (oesophageal transection) is undertaken if the superior mesenteric vein or the portal vein is thrombosed, effectively ruling out shunt procedures. The procedure involves use of a staple gun and has a low operative mortality.

5. If the superior mesenteric vein or the portal vein is patent, then meso-caval or distal lieno-renal anastomosis can be attempted. Mortality is about 40%. Alternatively, the TIPS (Transvenous Intra-hepatic Porto-systemic Shunt) procedure may be used. This involves placement of an expandable metallic stent between the branches of the portal vein and systemic circulation within the liver parenchyma.

Principles of anaesthetic management

Bleeding oesophageal varices are a life-threatening complication of chronic liver disease and often occur against a background of abnormal clotting, thrombocytopenia, encephalopathy and ascites. Overall mortality is 30%. The anaesthetist may be involved in any or all of the stages listed above. The principles of anaesthetic management are as ever:

● Protect the airway.

● Establish good vascular access.

● Volume replacement - colloid, blood, fresh frozen plasma and platelets. Avoid saline.

● Check / correct clotting. Give Vitamin K, correct fibrinolysis and review blood chemistry.

Many aspects of management mentioned in connection with Child-Pugh Group B/C patients apply here, including post-operative care.

BURNS

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The Burn Injury

Burn injury can result from many causes, the majority by these mechanisms:
Thermal injury - Hot and Cold including iatrogenic heating and non-accidental injury
Chemical - Acids and Alkalis (e.g. Do-It-Yourselfers and building workers can be burnt by cement.)
Electrical - Mains, High Tension, Railways and Lightning

Incidence

Deaths in England and Wales from smoke or fire related injury

	Females	Males	Total
2001	146	185	331
1997	174	262	436
1977	278	359	637
1968	337	454	791

The commonest cause of death is by smoke inhalation

Age range: In infancy scalds are the commonest injury. 70% of burnt children are toddlers. In old age, medical co-morbidity compounds the tendency to more severe burns (E.g. Epilepsy Parkinson's immobility and falls)

Other social risk factors, especially poverty and psychiatric illness mean that the burden of burn injury falls on the already disadvantaged.

Assessment

ATLS & APLS: These authorities emphasise a 'safe' approach for the rescuer and immediate life saving manoeuvres for the victim: 'ABCDEFG', stopping the burning process, gaining IV access, starting fluid resuscitation and providing appropriate pain relief are the cornerstones of the initial approach. Potential airway compromise will be suggested by the history, facial burns, soot staining around the nostrils, singed nasal hairs etc.

Assessment of the percentage of total body surface area of skin affected (%TBSA) is an important guide to the severity of the burn. The 'Rule of nines', is used over 14 years of age. Modified Lund & Browder charts for babies and children are available in A&E departments and in the APLS handbook. The patients own palmar surface (excluding fingers) is about 1%TBSA.

The depth of the burn is important for the planning of treatment.

Erythema (1°) is not included in the estimate of the burned area. Nevertheless these areas, rather like sunburn, may be painful but will not be blistered and will heal normally without treatment.

Superficial partial thickness (2°), **deep partial thickness** (2°) and **full thickness** (3°) areas are included in the estimated area.

The distinction between superficial and deep is of importance as the former has a better prospect of healing without scarring. Under-resuscitation can cause deterioration of burned areas to a more severe grade.

Complex burns include destruction of tissues deep to the skin such as tendon muscle and bone. Burns to the face, airway and perineum would also be included as complex burns.

Smoke inhalation and poisoning due to Carbon Monoxide (CO) and Cyanide (CN) must also be suspected where relevant. The finding of high carboxy-haemoglobin levels (>25%) should suggest not only the need to treat with 100% Oxygen and ventilation but also the possible need to test for cyanide intoxication. There is no high quality RCT evidence to direct the use of Hyperbaric Oxygen in Carbon Monoxide poisoning, especially if the Hyperbaric chamber is distant from the burns treatment centre.

Compartment syndromes and need for Escharotomies / Fasciotomies

Full thickness burns can produce compromise of respiratory excursion or distal tissue perfusion, suggesting the need for escharotomy. Effective releasing incisions go down to vital and often sensate tissues-indicating the **need for** anaesthesia. Such work would usually be done at the accepting Burn centre and only rarely at the referring hospital, after discussion with the accepting Consultant surgeon. The need for fasciotomy is rarer still and is usually associated with the exit point to earth of an electrical burn and associated muscle necrosis.

Burns less than 10% TBSA do not normally require formal resuscitation, although admission for assessment, pain relief and investigation of circumstances may be required (especially when children are injured). Concurrent medical illness may make **Oxygen** and **fluids** necessary even for <10%, for example in children with renal disease or with complex burns.

Burns greater than 10% and less than 30% may be classified as moderate in severity and will require **Oxygen, fluid resuscitation, pain relief and naso-gastric feeding**. As severity approaches 30% TBSA, a systemic inflammatory response of pyrexia, raised white count and raised CRP in the absence of infection becomes more likely but only if the burn remains unexcised and only after 24-48 hours. For less severe burns, close to the time of injury, pyrexia and raised indices of infection may indicate true infection. Cultures should be taken and the wound inspected by a senior opinion prior to starting antibiotic therapy. If pre-existing skin disease is present infection may occur early. (Including Toxic shock syndrome from toxin generating Staphylococcus Aureus.)

Concomitant smoke inhalation and cutaneous 10%<burn<30% indicates **at least** HDU based management.

Burns>30% TBSA represent a major injury where a systemic response is universal. Mortality is high if management is not optimal, even in young patients. The management of these more major burns involves **Oxygen, fluids, feeding & pain relief** as before, but with the need for invasive monitoring to guide therapy and for ventilation in a higher proportion of cases. Prophylactic antibiotics are not usually given except as part of a 'Selective Decontamination of the Digestive Tract',regimen in the more severe burns expected to be ventilated long term (in some units).

Resuscitation

Aims to:

1) Preserve life

2) Maintain Organ function

3) Ameliorate the injury

4) Restrict surgery to necessity and functional restoration

5) Limit Psychological damage

Fluid Regimens

These are guides for appropriate replacement. They are not ends in themselves. All 'recipes', require monitoring and adjustment. The commonest now used is the Parkland formula. This is calculated having an accurate assessment of the burned area and the body weight, suggesting a volume of Ringer-Lactate (Hartmanns Solution) given by:

> **2-4 mls per %TBSA per Kg body weight**

This volume is given over the 24 hours following injury, half the (volume) being given over the first 8 hours from the injury. For example a 70 Kg Man with a 50% BSA Superficial partial thickness injury would require 7-14 (4x50x70/1000) litres over the first 24 hours. Experience suggests that those patients with smoke inhalation injury need still more fluids, as do those with extensive partial thickness burns.

If it has taken several hours for the patient to reach medical attention the first half may need to be given at a 'front loaded', accelerated rate for a couple of hours in order to catch up. It is important that under-resuscitation is avoided. The fluid should be warmed to ensure that the development of hypothermia doesn't complicate clotting function or cause inappropriate vasoconstriction.

Oliguria, haemoconcentration and hypotension are all signs of inadequate fluid administration under these circumstances.

Adequate cerebral function, brisk capillary refill, appropriate blood pressure and urine output in the range 0.5-1.0ml/kg/Hr output (1.0-2.0ml/kg/Hr urine output in children) suggest adequate resuscitation.

In larger burns and in patients with pre-existing impaired physiology, invasive monitoring with CVP or pulmonary artery flotation catheters may gauge adequacy of fluid replacement more effectively. These are best used early and removed before greater morbidity occurs due to infection. Experience is being gained with the oesophageal doppler monitor as a non-invasive guide to filling and in conjunction with CVP to guide SVR manipulation.

Big burns themselves appear to have cardio-depressant effects.

If **Myoglobinuria** is seen it is appropriate to aim for higher levels of urine output driven by osmotic diuresis with Mannitol. Up to 12.5 grams of Mannitol can be given per litre of resuscitation fluid (ATLS). This can give a sense of false security as urine output may be maintained while the patient remains dry overall. The overall fluid balance requires more careful monitoring under these circumstances.

Maintenance fluids appropriate to the age and weight of the patient are also required. In small children the use of Dextrose 4%/Saline 0.18% solutions will exaggerate the hyponatraemia due to use of Hartmanns solution (Na 131 mmol/litre) particularly if hypovolaemia is present. This will increase the likelihood of seizures. Significant hyponatraemia can also be part of a Toxic Shock presentation. In any significant burn injury the use of the enteral route for administration of maintenance fluid as feed will reduce the tendency to low Sodium and will minimise the loss of muscle to the catabolic response over the full duration of the injury.

The continuation of the Parkland formula involves a further 24 hours of fluid resuscitation again based upon Hartmanns solution with 2ml/%TBSA/kg plus 0.5ml/%TBSA/kg of colloid-originally Albumin. Since the Cochrane review of the evidence base of Albumin use, this component has been dropped by many centres and starches have been substituted. Similarly the Muir and Barclay formulas of Albumin based resuscitation have largely been dropped. FFP is still widely used by Paediatric Anaesthetists for fluid resuscitation and treatment of sepsis in burned babies and infants.

Involvement of Anaesthetists

Early surgical assessment is required for:

1) Accurate reassessment of %TBSA,

2) Burn wound cleansing and coverage,

3) Early debridement of full thickness injury (when indicated)

Repeated anaesthetic input may be required for debridement and dressings until stable wound coverage and healing is obtained

Long term input is required into the care of the survivors of the biggest burns, both in their initial care and during subsequent reconstruction/revision

Anaesthesia and Intensive Care

Initial care requires attention to detail in terms of pre-operative assessment of the patient. Both the history and events following the injury and the patients' personal history are important. The priorities are to maintain safety for the individual undergoing treatment, while maintaining an eye to the future, protecting vascular access and making appropriate airway care decisions.

The interaction of anaesthetic agents with the patient's physiology changes over time. At initial presentation the anaesthetist may be faced with a patient who is undergoing resuscitation but remains hypovolaemic; their airway may be compromised by the oedema of both burn and crystalloid resuscitation (or is becoming so); their vascular access may be compromised by the

burn itself and there may be significant problems with the acute pain of the injury, including an acute neuropathic element. Significant also, from the psychological point of view, is the possibility that the injury may be self-inflicted.

As the injury matures, airway difficulties may be worsened by scarring and contracture. This can render conventional laryngoscopy impossible. Gas inductions and the use of spontaneous breathing techniques may allow placement of laryngeal masks or fibre optic aided intubation to be perfomed. Blind intubation techniques have a place for those skilled in their use. Awake intubation may also need to be performed.

If there is both an obvious difficult route to the larynx and laryngeal compromise, expert judgement is needed to decide between gas induction or direct surgical access to the airway under local anaesthesia.

The use of Suxamethonium is known to be potentially dangerous due to exaggerated hyperkalaemia. This can develop as cholinergic sensitive ion channnels migrate and increase in muscle beyond the motor end plate. Similar considerations apply to any ITU patient who is denervated, immobilised, or has had repeated sepsis. Suxamethonium can be used for rapid sequence induction early on provided it is thought essential. The same proliferation of binding sites, with changes in metabolism, increase the requirement for non-depolarising agents, of all classes, for a given

duration of effect for many months after injury.

Outcomes

Advances in surgical techniques and dressings have meant that bigger burns can have an improved functional and cosmetic outcome – provided they survive. The management is aimed at excising dead tissue to minimise the inflammatory response. Areas that can recover are preserved and covered with dressings, including treated cadaver skin as a biological dressing. The priority is to gain coverage and avoid infection. That said, infection is universal as colonisation and is often tolerated without treatment provided that systemic sequelae are not occurring. Surveillance of the colonising flora gives a clue as to the appropriate treatment t should deterioration occur.

Grafting is taken from donor sites in unburned areas including the scalp. Donor sites can be revisited once they have healed well. The bigger the burned area the less donor site is available and the slower the native skin cover returns, despite meshing techniques etc.

Artificial dermal grafts and epidermal cell cultures offer the hope of reconstituting skin cover without scarring. However, all cell cultures and non immunologically active grafts dissolve in the presence of uncontrolled infection.

	Hours	**Days**	**Weeks**
Analgesics	Opiates IV as required. For example Morphine 30micrograms /kg repeated as necessary.	PCA +/_ Background Infusion Paracetamol, NSAIDs if stable and fed. Consider Co-Analgesics for neuropathic components and to reduce opiate impact on feeding.	Oral Opiates if required. Consider MST, Methadone, Oxycodone Paracetamol and NSAIDs as tolerated
Induction Agents	IV/ Inhalational as appropriate. Ketamine 1-2mg/kg sole agent. Propofol	Older agents less suitable for repeat administration. Newest agents give very rapid awakening. Desirable?	Patients become adept at their own chosen method of anaesthesia. Ask them where the veins are!
Maintenance	Inhalational /TIVA	Propofol contraindicated for ITU sedation under 16 yrs of age.	
Relaxants	Depolarizing agents OK if airway not currently compromised (RSI) Consider gas induction and no relaxant / awake intubation.	Depolarizers becoming dangerous for bigger burns >10% Non Depolarizers less effective and shorter acting.	Depolarizers usable once pt mobile and re-innervated Non Depolarizers return to normal patterns.
Co analgesics	Ketamine may help to avoid pain 'wind up'. 0.1-0.15mg/kg/Hr may help reduce opiate requirement.	Consider Gabapentin and /or tricyclics/SSRI for neuropathic components	Itch may be a prolonged problem. Due to nerve regrowth and Starches.
Starvation	Not starved	Do not starve intubated patients. Feed overnight with daytime rest. Feed during operations.	Early well established nutrition improves outcome and speeds rehabilitation.

What is achievable?

Number of Risk Factors	Guide mortality
0	1/300
1	1/30
2	1/3
3	19/20

The biggest area burns require the greatest effort and input. They also have the longest period, while they are healing, during which they may deteriorate and die after their injury. Late death occurs most frequently due to sepsis.There is no fixed point where the size of a burn dictates that recovery is not possible.

The traditional relationship:

%TBSA size of burn		Likely %Mortality
+ Age in years	=	is out of date.

Risk factors for Mortality include

1. Size of burned area,

2. Smoke Inhalation and

3. Age >65years (Co morbidity with physiological impairment)

Age >65years is often used as a measure although the younger patient with significant pre-existing physiological impairment will score this point and the Marathon winning 75 year old would not!

Results like these are only obtainable in the best centres worldwide. Most British institutions are working towards them as a goal rather than achieving them at present.

Decision making to treat or not to treat needs case-by-case consideration of the individuals' circumstances from their own point of view, that of their physiology, their injury(ies), and their potential for eventual rehabilitation.

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MCQ QUESTIONS
Dr Ed Hammond, Exeter

Question 1

Nerve stimulators

A. Double burst is of particular value in assessing blockade when there is no response to the train-of-four

B. A fading pattern to the train-of-four excludes prolonged action of suxamethonium

C. When used to locate nerves for regional blockade the positive electrode should be attached to the locating needle

D. The post tetanic count is particularly useful for assessing the patient's suitability for extubation

E. The ideal nerve for neuromuscular blockade assessment is generally considered to be the facial nerve

Question 2

The following potentiate the action of non-depolarising neuromuscular blockers

A. Diethyl ether

B. Quinidine

C. Enflurane

D. Lithium

E. Dantrolene

Question 3

When looking at an ECG

A. A bifid P wave may indicate that the patient has mitral stenosis

B. Peaked P waves are associated with right atrial hypertrophy

C. A short PR interval indicates that the patient has Wolff-Parkinson-White syndrome

D. Left bundle branch block is associated with a secondary R wave in V6

E. U waves are associated with hypothermia

Question 4

The following ECG changes are associated with the correct cause

A. U waves - Hypothermia

B. T wave flattening - Hypothyroidism

C. Short QT Interval - Hypercalcaemia

D. Biphasic P wave in V1 - Mitral stenosis

E. T wave inversion - Hypokalaemia

Question 5

The following ECG changes occur with hypokalaemia

A. Tall T waves

B. ST elevation

C. Loss of P waves

D. T wave inversion

E. Prominent U waves

Question 6

The CVP trace

A. The c wave follows the X descent

B. The X descent occurs in (ventricular) diastole

C. The v wave results from right atrial filling against a closed tricuspid valve

D. Cannon waves are seen in complete heart block

E. The a wave is of variable size in atrial fibrillation

Question 7

Anaesthesia breathing circuits

A. The Lack circuit is a Mapleson D system

B. The Magill attachment is more efficient for spontaneous breathing than for controlled ventilation

C. The recommended fresh gas flow rate for controlled ventilation through a Bain circuit is 70 ml/kg body weight

D. Minimum fresh gas flow to avoid rebreathing using a Mapleson A is equal to that patient's minute ventilation

E. The Mapleson F system is a modification of the Mapleson E system

Question 8

The following cause a rise in the end-tidal carbon dioxide level (assuming constant ventilation)

A. Hypothermia

B. Malignant hyperpyrexia

C. Pulmonary embolus

D. Disconnection of the inner tube of a Bain circuit

E. Failure of the endotracheal tube cuff

Question 9

The concentration of vapour in the gas mixture emerging from the outlet port of a vaporiser depends on

A. Saturated vapour pressure of the agent

B. Flow characteristics of the vaporiser

C. Duration of use

D. Surface area of the agent in contact with the gas mixture

E. Temperature

Question 10
Anaesthesia at high altitude (using a plenum vaporiser)

A. The concentration delivered by the vaporiser will be higher than the dialled value

B. The concentration dialled into the vaporiser will need to be higher for the same effect

C. The concentration dialled into the vaporiser will need to be lower for the same effect

D. The inspired oxygen concentration may need to be increased

E. The anaesthetic potency of 50% nitrous oxide will be reduced

Question 11
A low resistance to gas flow is a feature of the following vaporizers

A. Oxford minature vaporizer

B. Epstein Macintosh Oxford vaporizer

C. Copper Kettle Vaporizer

D. Goldman Vaporizer

E. Boyle's bottle

Question 12
The following are true concerning humidity and humidification of gases

A. Relative humidity is the ratio of absolute humidity to saturated humidity at a specified temperature

B. Operating theatre humididty should be maintained at no more than 30%

C. Heat and moisture exchangers can achieve 40% humidity

D. A nebuliser works on the poiseuille effect to entrain water across a pressure drop

E. The water trap for a simple bottle humidifier must be larger than the humidifier bottle

Question 13
During brachial plexus blockade

A. The interscalene approach commonly leads to inadequate blockade of the ulnar nerve

B. The axillary approach may lead to Horner's syndrome

C. The supraclavicular approach commonly leads to inadequate blockade of the axillary nerve

D. Bilateral interscalene blocks should be used for bilateral shoulder manipulation surgery

E. The axillary approach commonly leads to inadequate blockade of the median nerve

Question 14
Complications of retrobulbar blockade for cataract surgery include

A. Bradycardia

B. Retinal detachment

C. Brain stem anaesthesia

D. Vitreous haemorrhage

E. Optic nerve damage

Question 15
Effects of epidural blockade include

A. A greater degree of hypotension when adrenaline (epinepherine) containing local anaesthetics are used

B. Sympathetic blockade before sensory blockade

C. Anterior spinal artery syndrome

D. Reduced tidal volume with a normal block to T4

E. Reduced peristalsis

Question 16
A successful stellate ganglion block may cause

A. Ipsilateral miosis

B. Contralateral nasal congestion

C. Bilateral ptosis

D. Ipsilateral exomphalos

E. Horners syndrome

Question 17
Concerning brachial plexus blockade

A. The interscalene approach provides for anaesthesia to the ulnar border of the forearm

B. The supraclavicular approach is not reliable to produce anaesthesia to the hand

C. The axillary approach is least likely to cause pneumothorax

D. Diaphragmatic paralysis is a complication

E. Puncture of an artery may be deliberate

Question 18
Inguinal hernia field block

A. Blocks the ilioinguinal, iliohypogastric and genitofemoral nerves

B. May be employed for testicular surgery

C. Prilocaine 0.5% may be used

D. Depends on depositing local anaesthetic between internal and external oblique

E. Quadriceps weakness is a complication

Question 19
Serum Na^+ 120 mmol/l and K^+ 6.4 mmol/l are consistent with

A. Hyperaldosteronism

B. Renal failure

C. Hypopituitarism

D. Adrenocortical insufficiency

E. Cushings disease

Question 20
The following conditions are associated with a haemoglobin concentration of 7 g/dl and a mean corpuscular volume of 70 fl

A. Iron deficiency anaemia

B. Acute blood loss

C. Folate deficiency

D. Renal failure

E. Thalassaemia

Question 21

Obesity is associated with

A. An increase in the incidence of airway complications

B. Increased functional residual capacity (FRC)

C. Increased DO_2 (Oxygen delivery)

D. Pulmonary hypertension

E. Quetelet index of 24.5

Question 22

Diathermy safety

A. Ohm's law states that voltage = current x resistance

B. Diathermy works because there is a high current density at the active electrode

C. The heat energy produced by cautery is proportional to the current at the tip of the active electrode

D. Bipolar diathermy requires a passive electrode ('diathermy plate')

E. Poor contact of the passive electrode ('diathermy plate') may lead to inadvertent patient burns

Question 23

With regard to electrical safety and prevention of explosions

A. Nitrous oxide is not flammable at atmospheric pressure

B. Anaesthetic machines should be isolated from 'earth' to prevent completion of an electrical circuit

C. Currents of 10 microamps may initiate ventricular fibrillation

D. The neutral connection of a circuit is not at earth potential at the patient end of the circuit

E. In surgical diathermy the heat released depends on the square of the potential difference between electrodes

Question 24

Ephedrine is unlikely to be effective in reversing hypotension in patients chronically receiving the following medication

A. Reserpine

B. Alpha-methyl dopa

C. Phenoxybenzamine

D. Clonidine

E. Propranolol

Question 25

Heat loss

A. Conduction is the largest factor in patient heat loss

B. Radiation accounts for about 10% of the total heat loss

C. Convection is due to heating of the adjacent air layer which is replaced by cooler air from the surroundings

D. Heat lost in breathing dry gases is approximately 15% of total heat loss in the anaesthetised subject

E. Burns from faulty heating equipment are more likely in the vasoconstricted patient

Question 1

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

The pattern of peripheral nerve stimulation used for assessment of neuromuscular blockade most commonly is the train-of-four. This shows characteristic fade when recovery from non-depolarising neuromuscular blockade is occurring. However in prolonged blockade with suxamethonium there is a conversion to the non-depolarising pattern with fade appearing. This is called dual block and occurs if the prolonged block is due to either repeated doses of suxamethonium or impaired metabolism (suxamethonium apnoea). The train-of-four is of no value when there is no response to the first twitch, and in this case post-tetanic count is used. During recovery it is difficult to assess the ratio between the first (t1) and fourth twitch (t4) and this should be 100:70 for successful extubation. This comparison is easier using the double burst pattern. Stimulators used for regional blockade should be used with the locating needle attached to the negative electrode as this ensures depolarisation of the nerve at a lower applied current.

Question 2

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

All the volatile agents enhance the action of neuromuscular blockers by reducing the tone of skeletal muscle, an action mediated by an effect at the post junctional membrane. Quinidine is a membrane stabiliser and an isomer of quinine. Dantrolene disrupts excitation-contraction coupling and so does not directly potentiate the action of NMBs.

Question 3

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

Bifid P waves are associated with left atrial hypertrophy and may indicate the presence of mitral stenosis. Tall peaked P waves (>3mm) are associated with raised right atrial pressure and hypertrophy. The presence of a short PR interval suggests pre-excitation. If there is a delta wave present then this is known as the Wolff-Parkinson-White syndrome. The absence of a delta wave suggests that it is the Lown-Ganong-Levine syndrome. In left bundle branch block the left ventricle depolarises late and so there is a slurred or secondary R wave in V6 and S wave in V1 (M shaped complex in V6). U waves are associated with hypokalaemia and occur after the T wave. J waves are associated with hypothermia and are a characteristic deflection at the end of the QRS complex.

Ref: Yentis, Hirsch and Smith. Anaesthesia A to Z. Butterworth

Question 4

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

Hypothermia is associated with J waves which are positive deflections at the end of the QRS complex. Hypokalaemia is associated with U waves as well as T wave flattening, inversion, and prolongation of the PR and QT intervals. U waves may also be a normal finding and be associated with both hypercalcaemia and hyperthyroidism. A short QT interval is a recognised sign of hypercalcaemia. Biphasic P waves is V1 represent left atrial enlargment and are associated with P mitrale where the P wave

is bifid in lead II. Hypothyroidism is associated with low voltage complexes, bradycardia and T wave flattening or inversion.

Question 5

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

Hypokalaemia causes P-R prolongation, ST depression, T wave inversion and prominent U waves.

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

Question 6

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

The CVP trace consists of three main waves and two descents, (in chronological order):

The a wave - right atrial (RA) contraction. Absent in atrial fibrillation; enlarged with tricuspid stenosis, RV hypertrophy, pulmonary stenosis or pulmonary hypertension; cannon waves (giant A waves) occur if the RA contracts against a closed tricuspid valve (e.g. in complete heart block)

The c wave - bulging of the tricuspid valve at the onset of ventricular systole

The X descent - atrial relaxation during ventricular systole

The v wave - RA filling with a closed tricuspid valve

The Y descent - opening of the tricuspid valve with blood flow into the right ventricle.

Question 7

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

The Mapleson classification of breathing systems has six classifications A-F. The Lack circuit and the Magill attachment are examples of Mapleson A systems. They are very efficient for spontaneous breathing (requiring a fresh gas flow (FGF) equal to alveolar ventilation - less than minute ventilation (MV)). They are much less efficient for controlled ventilation (FGF = 3 x MV). The Mapleson D (including the coaxial Bain circuit) is most efficient for controlled ventilation. The Bain circuit requires a FGF of 70 ml/kg to maintain normocarbia. The Mapleson F (Jackson-Rees) is a modification of the Ayre's T-piece (Mapleson E) designed to minimise resistance to gas flow and ideal for paediatric use.

Ref: See Update No 7.

Question 8

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

A rise in end tidal carbon dioxide may be due to:

Inspired carbon dioxide

Rebreathing in the circuit (e.g. disconnection of the inner tube in a Bain circuit or increased dead space)

Increased production of carbon dioxide (e.g. malignant hyperpyrexia)

Inadequate ventilation

Leaks, reduced metabolic rate (due to hypothermia) and impaired

cardiac output (pulmonary embolus) all cause reduced end tidal carbon dioxide.

Question 9

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

All of these factors will determine the concentration of vapour in the gas mixture.

The more volatile the agent, the higher the SVP and hence the higher the concentration emerging from the vaporiser assuming the same splitting ratio. The temperature determines the SVP of the agent. Duration of use may alter the SVP via its effect on temperature. The surface area of the vaporiser must be sufficient to ensure full saturation of the mixture passing through the vaporising chamber. The flow characteristics are important to ensure complete mixing and hence full saturation once again. The splitting ration is also important as if the gas exiting the vaporising chamber is fully saturated it will determine the total amout of agent added to the final gas mixture.

Modern vaporisers are temperature compensated. The splitting ratio amount of gas passing through the bypass chamber is altered to reflect the change in SVP with temperature and thus ensure constant output of vapour at the required level.

Question 10

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

The amount of oxygen, nitrous oxide and anaesthetic agent required by the body for oxygenation and anaesthesia is related to the partial pressure of these in the trachea. For convenience the more easily measured concentration or fraction is used, and the barometric pressure is assumed to be constant. At high altitude the barometric pressure is reduced. The partial pressure of anaesthetic agent that is delivered by the vaporiser is constant at differing barometric pressures, so the concentration dialled into the apparatus will have the same. clinical effect as the SVP is unchanged if the temperature is the same. However the fraction (%) actually delivered will be higher than the dialled value as the absolute pressure in the enviroment is lower. Furthermore to get the same partial pressure of oxygen at lower barometric pressure requires a higher inspired fraction, and the same inspired fraction of nitrous oxide will provide a lower partial pressure and so a reduced effect.

This can be confusing and it is advisable to read the reference.

Question 11

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

Low resistance is a feature of the OMV,EMO and Goldman vaporizers.

The OMV is a small non-temperature compensated vaporizer used with portable anaesthetic equipment. It has a antifreeze filled sealed compartment that acts as a heat sink and so minimise temperature changes. The scale can be change for the use of the vaporizer with different volatile agents.

The EMO is used for ether. It is still used throughout the world and has a large water filled heat sink.

The Goldman vaporizer is a small cheap and simple device used for halothane. It is not temperature compensated and output depends somewhat on the gas flow through the vaporizer.

Both the Copper Kettle and Boyles bottle vaporizers are examples of plenum vaporizer with a high internal resistance to gas flow.

Ref: See Update No 14

Question 12

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

Absolute humidity is defined as the mass of water in a volume of air. Relative humidity is defined as in the question is usually presented as a %. Humidification devices can be defined as active or passive; vapour or droplet producing; hot or cold and finally functioning in a breathing system or in the atmosphere. Theatre humidity should be around 60% as a compromise between discomfort (if too high) and the increased risk of explosion due to static electricity (if too low). Heat and moisture exchangers can achieve 70% humidification. A nebuliser works on the venturi or bernouille effect. For a bottle humidifier the water trap should be at least the same size as the humidifier bottle.

Question 13

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

The brachial plexus can be approached from above or below during regional anaesthesia for surgery on the upper arm. The interscalene approach gives excellent anaesthesia to the shoulder and upper arm, but commonly leads to inadequate blockade of the ulnar nerve. Potential side-effects of this block are Horner's syndrome, phrenic nerve block, recurrent laryngeal nerve block and inadvertent extradural or intrathecal injection. Bilateral interscalene blocks should not be performed. The supraclavicular approach commonly leads to inadequate blockade of the median nerve. Horner's syndrome and pneumothorax are amongst the adverse events that can occur with this approach.The axillary approach commonly leads to inadequate blockade of the axillary nerve, and supplemental sub-cutaneous local anaesthetic will be required if the upper/outer aspect of the arm is involved in surgery, or if a tourniquet is to be used. Horner's syndrome does not occur with the axillary approach.

Question 14

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

Complications of retrobubar block may be systemic or local. Retrobulbar blockade requires the injection of local anaesthetic into the muscle cone behind the eye. The most common complication is retrobulbar haemorrhage due to puncturing the vessels within the retrobulbar space. The increased pressure in the globe can cause central artery occlusion. Other complications include bradycardia secondary to the oculocardiac reflex, posterior globe puncture with resultant retinal detachment and vitreous haemorrhage, penetration of the optic nerve, brain stem anaesthesia from local entering breeched dura around the optic nerve and subarachnoid blockade or inadvertant intraocular or intravascular injection; hence the preference of most anaesthetists for peribulbar blocks.

Question 15

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

More hypotension occurs when adrenaline containing local anaesthetics are used for epidural blockade. This may be due to the beta 2 effects of the absorbed adrenaline causing vasodilation in peripheral beds. It is countered by the chronotropic and inotropic effects on beta 1 receptors. However the more prolonged hypotension seen is probably due to the achievement of a more profound degree of sympathetic blockade. Sympathetic blockade occurs after sensory blockade. Small unmyelinated sensory fibres with no barrier to local anaesthetic diffusion are blocked before the larger autonomic B fibres. Anterior spinal artery syndrome is due to severe hypotension secondary to epidural blockade and not due to the technique itself. This leads to infarction of the spinal cord and results in a lower motor neurone paralysis at the level of the lesion and spastic paraplegia with decreased pain and temperature sensation below the level. Epidural blockade may cause lower intercostal muscle and abdominal muscle weakness resulting in impaired coughing and exhalation. However with a T4 block diaphragmatic innervation (C3-C5) is maintained and tidal volume and inspiratory pressure are maintained. Bowel contraction results from blockade of the sympathetic outflow and unopposed parasympathetic activity. Sphincters relax and peristalsis increases.

Question 16

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

Horners syndome is the triad of enopthalmos, ptosis and miosis. Nasal congestion and anhydrosis are common but ipsilateral. Remember that exomphalos is a neonatal condition!

Question 17

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

There are 3 common approaches to blocking the brachial plexus. The interscalene approach is ideal for shoulder and upper arm operations. It however frequently spares the C8 and T1 fibres which innervate the ulnar border of the forearm. Injection of local anaesthetic by this approach may produce cervical plexus block which may cause diaphragmatic paralysis. The phrenic nerve may also be blocked because of diffusion or inappropriate injection to the anterior side of the anterior scalene. The supraclavicular approach attempts to block the plexus at the first rib and is most reliable at producing anaesthesia of all four terminal nerves of the forearm and hand. It does however carry the greatest risk of pneumothorax. The axillary approach is simplest and has the least chance of pneumothorax. If paraesthesia cannot be elicited during this approach then one alternative is to deliberately pucture the axillary artery and advance the needle through the opposite wall where half the anaesthetic solution is deposited. The remainder is injected once the needle has been pulled back through the "anterior" wall of the artery.

Question 18

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

Local anaesthetic is deposited between the internal and external oblique muscle layers. If the internal oblique is penetrated, local anaesthetic may track back to the lumbar plexus and affect the femoral nerve, producing quadriceps weakness. Prilocaine may be used because of the larger amount of solution which may safely be injected, but most would use bupivacaine. The testicle is innervated by T10 and so this block is quite ineffective for testicular surgery.

Question 19

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

Aldosterone causes sodium retention and potassium loss. In chronic renal failure hyponatraemia or hypernatraemia can occur. In acute renal failure fluid retention can lead to hyponatraemia. Hyperkalaemia can also occur and is an indication for dialysis. Hypopituitarism leads to a reduced secretion from the anterior pituitary gland and hence ACTH insufficiency and reduced cortisol. Mineralocorticoid production remains largely intact as this is predominantly stimulated by angiotensin II. Destruction of the entire adrenal cortex reduces glucocorticoids, mineralocorticoids and sex steroids. As such hyponatraemia, hyperkalaemia and a raised urea result. Cushings results in excess cortisol which has some mineralocoticoid activity. This can lead to loss of potassium.

Question 20

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

These indices indicate a microcytic anaemia (normal MCV = 85 fl). The most common cause is iron deficiency. The red cells will also be hypochromic (MCH less than 27 pg). In thalassaemia there is a deficiency in the synthesis of the globin chains of haemoglobin. In addition the accumulation of abnormal chains within the red cell leads to its early destruction. This causes an anaemia with reduced MCV and MCH. The reticulocyte count is also raised. The anaemia of renal failure is normocytic and normochromic, in common with anaemias of chronic disease. In renal failure it is due to reduced erythropoietin production and in severe uraemia, > 30 mmol/l, a shortened red cell life and marrow toxicity. Folate and vitamin B12 deficiency cause macrocytic (high MCV) megaloblastic anaemia. Acute blood loss will cause an anaemia with a normal MCV and normal shaped existing red cells.

Question 21

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

The Quetelet index (or body mass index) is weight (kg) divided by the square of height (in m2). The upper limit of normal is 24.9. Overweight is 25-29.9, obese is 30-34.9, and morbid obesity over 35. Hypertension, ischaemic heart disease and increased oxygen consumption are characteristics, as are airway obstruction, right heart strain and, eventually, pulmonary hypertension and right-sided failure. The FRC is reduced with a tendency to V/Q mismatch and hypoxaemia. Hiatus hernia with reflux, and diabetes are other considerations.

Ref:Shenkman et al. Perioperative management of the obese patient. British Journal of Anaesthesia 1993;70:349.

Question 22

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

Diathermy relies on generation of heat energy from electrical energy. Ohm's law states that voltage = current x resistance (V=IR). The heat energy produced = (current)2 x resistance (E=I2R). At the active electrode there is a high current density due to the small area of the electrode. This leads to the heat energy and cautery. The passive electrode ('diathermy plate') used with unipolar diathermy has a large area and so the current density (and hence the heat produced) is small. However if the contact is poor then either there is a reduced area of contact (with a higher current density) or increased resistance. As E=I2R both these faults lead to an increased risk of burning. Bipolar diathermy does not have a passive electrode, and the current passes from an active electrode to a return electrode; these are the two blades of the diathermy forceps.

Question 23

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

Nitrous oxide is not flammable. An anaesthetic machine should be connected to a conducting floor of an operating theatre to prevent the production of static electrical sparks. A current of approximately 100 microamps is needed to produce a microshock to the heart, capable of producing ventricular fibrillation. Since all connections have some finite resistance the neutral connection is not exactly at earth potential at the patient end of the circuit. The heat released in surgical diathermy depends on the resistance and to the square of the current flowing.

Question 24

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

Indirectly acting sympathomimetics like ephedrine are unlikely to increase blood pressure in patients taking drugs which alter neuronal storage, uptake, metabolism or release of neurotransmitters. Reserpine depletes neuronal granules of noradrenaline. Alpha-methyl dopa acts as a false transmitter. Phenoxybenzamine and propranolol block peripheral receptors and industrial doses of directly acting sympathomometics may be required to overcome their blockade. Clonidine works on central adrenergic receptors and the peripheral effect of indirectly acting sympathomimetics is not decreased, in fact smaller doses may be required due to receptor up regulation.

Question 25

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

Patient heat loss is mainly due to radiation (40-50%) and, to a lesser extent, convection (defined as in the question) and evaporation. About 15% of heat loss in the anaesthetised patient is via the respiratory tract. The main part of this is due to the latent heat needed to vaporise water to humidify the gas within the trachea. This can be minimised by humidification. Warming equipment such as electric blankets and water-filled mattresses have caused thermal burns in anaesthetised patients. The risk of burns is greater if there is poor blood supply as blood can help conduct the heat away from the site of potential injury.
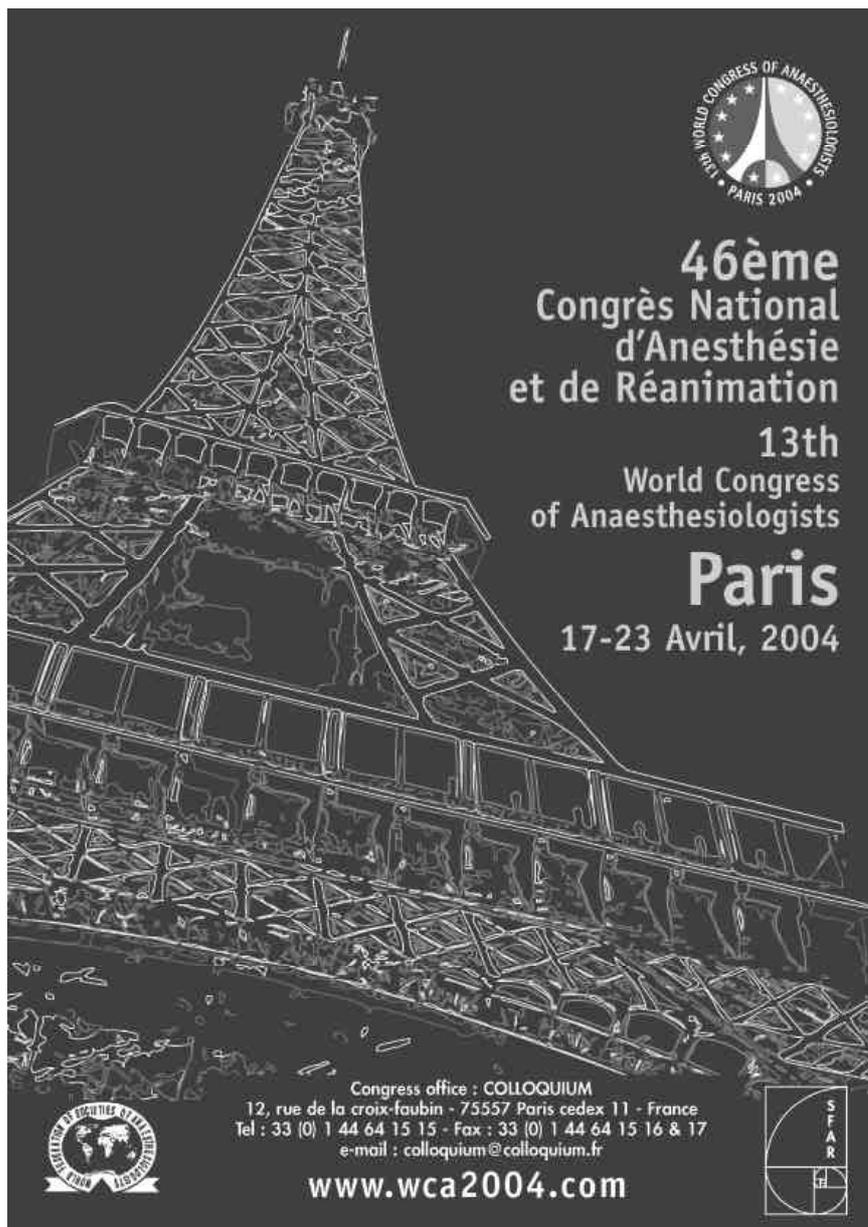
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